EQUINE PODIATRY

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PREFACE

Not too many years ago, as a general equine practitioner, I encountered my first case of laminitis. As fate would have it, this particular case was attached to owners who could not understand why laminitis should not be a death sentence. The horse was three years old and after extensive time, money, and "going where no one had gone before," we had saved the front feet and lost the right hind to a luxation of the second and third phalanges. This sent me down a path of exhaustive inquiry that stabilized the luxation. This year, with the horse having been a constant research model for 15 years, I let her slip gently into eternal sleep. I owe a great debt to Misty and her owners, Geri and Andy Olds, who passionately worked to save this mare's life, and in so doing began my career as an equine podiatrist.

We as veterinarians know so little about this second-highest killer of horses worldwide. General equine practitioners suffer from a lack of a viable working protocol for this disease process. We have learned so much, and we still have so far to go, but if we don't try we won't learn, and if we don't learn we cannot heal.

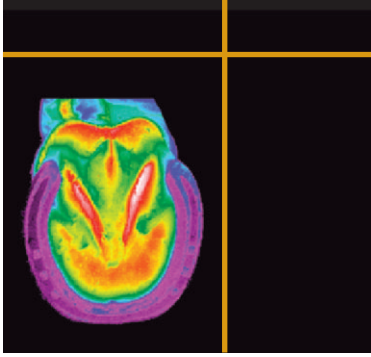
The only thing that kills horses with laminitis is ignorance and economics. Research is badly needed, not just in the university setting, but in the evidence-based practice. Horse owners must become proactive in their understanding of this disease process. Farriers and veterinarians must understand that any podiatry problem that is refractory in their hands may have a successful outcome in a referral facility, and referring these horses makes them a hero in the eyes of their clients.

Equine podiatry is a science that requires common sense, objectivity, intuitive skill, and team players. We now have very effective protocols for the diseased or lame foot, and we would like to share those with you.

We have designed this book for everyone to read, whether you are a farrier, horse owner, or veterinarian.

I would like to personally thank every author in this book. They have spent hundreds of hours in an exhaustive attempt to give you the latest information in an easily readable format.

Andrea E. Floyd, DVM
Richard A. Mansmann, VMD, PhD



Section I ANATOMY AND PHYSIOLOGY OF THE EQUINE DIGIT

1 GROSS ANATOMY OF THE EQUINE DIGIT

HELEN M.S. DAVIES and CHRISTOPHER PHILIP

GENERAL DEFINITIONS AND STRUCTURE

The digit of the horse is the region of the limb below the fetlock (metacarpophalangeal joint). Horses have only one digit in each limb. They stand on the tips of the digits, balanced on a modified fingernail (the hoof capsule). In each of these digits they have the same major bones (phalanges) as humans have in the middle finger, just a different shape and size (Figures 1-1, 1-2, 1-3). Box 1-1 contains definitions of some of the anatomical terminology that is used in this chapter.

Directional Terms

The surface of the digit that would be equivalent to the palm of the human hand is called the *palmar* surface (*plantar* in the hindlimb), and the opposite surface (which faces forward when the horse is bearing weight on the foot) is the *dorsal* surface. *Proximal* refers to regions that are located farther up the limb (i.e., closest to the body), and *distal* refers to regions that are located farther down the limb (i.e., farthest from the body). *Medial* refers to the side of the structure (bone, joint, etc.) that is closest to the midline of the body, and *lateral* refers to the side farthest from the midline.

Differences Between Limbs

The digits are very similar in all four limbs, although in most horses they differ slightly in shape between the forelimb and the hindlimb. Also, they are often slightly asymmetrical, so that the left digits tend to be mirror images of the right. For example, in the phalanges of the forelimb (and inconsistently in the hindlimb), the load-bearing portions of the bone at the joint (the condyles) tend to be slightly larger on the medial side than on the lateral side.

Nevertheless, there is a general similarity between the medial and lateral sides of each bone and between the fore and hind digits in any horse for a general description to be sufficient for most purposes. The following descriptions can be taken to cover all four limbs in a horse of normal conformation, except where indicated. Where the term *palmar* is used, the term *plantar* would be correct if applying the same description to the hindlimb.

This chapter focuses on the skin and hoof capsule that cover the column of bones and supporting soft tissues that comprise the digit. How these structures function in the living horse, both individually and as an integrated unit, is the focus of Chapter 2. Owing to their importance in disease conditions and diagnostics, the blood and nerve supply for the digit are

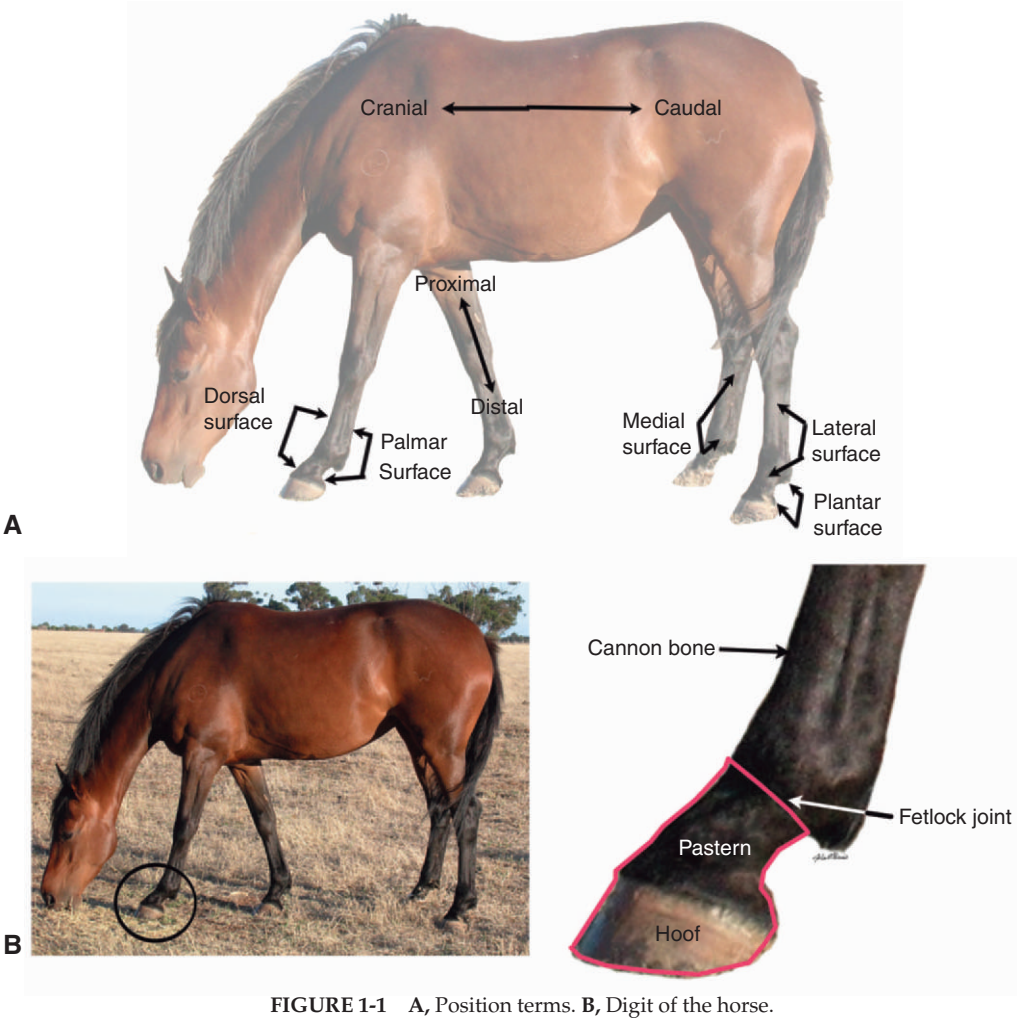


FIGURE 1-1 **A,** Position terms. **B,** Digit of the horse.

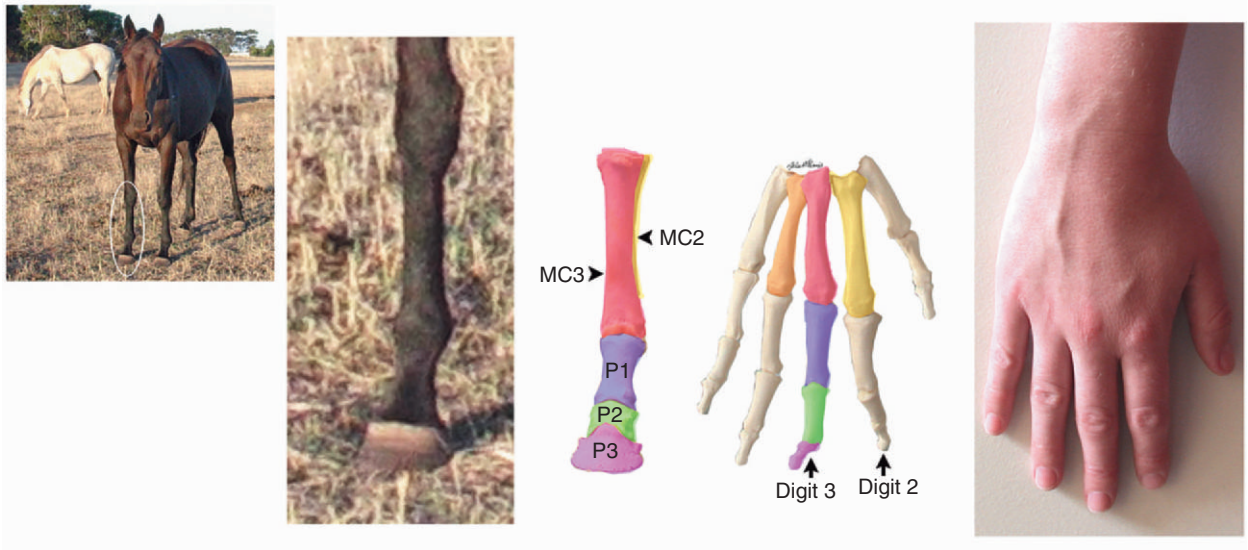


FIGURE 1-2 Equivalent bones in the human hand.

discussed in Chapters 5 and 17, respectively. The microscopic anatomy of the hoof also is discussed in Chapter 6.

SKIN

The skin (integument or cutis) covering the digit is a pliable sheet of tissue that is thickest on the dorsal surface and somewhat thinner on the palmar surface. The skin of the digit is completely covered by the hair that grows from it. The skin consists of two layers: the epidermis (outer layer) and the dermis (inner layer). The subcutis is the layer of connective tissue that lies between the dermis and the deeper structures (Figures 1-4, 1-5, 1-6).

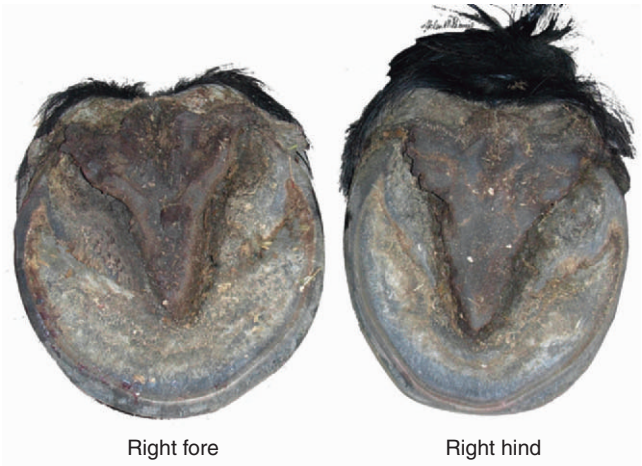


FIGURE 1-3 Untrimmed hooves of a yearling after 6 weeks of walking exercise on concrete.

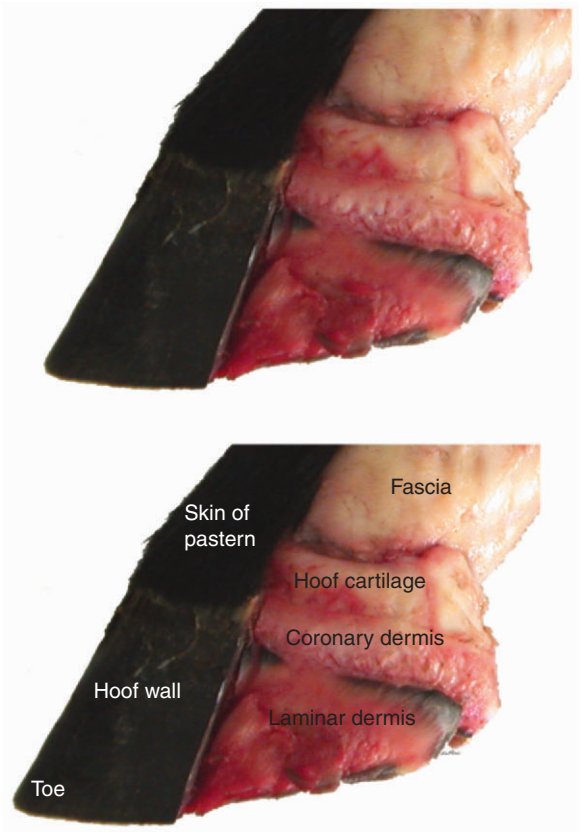


FIGURE 1-5 Hoof with part of the skin and hoof wall removed to show the dermis.

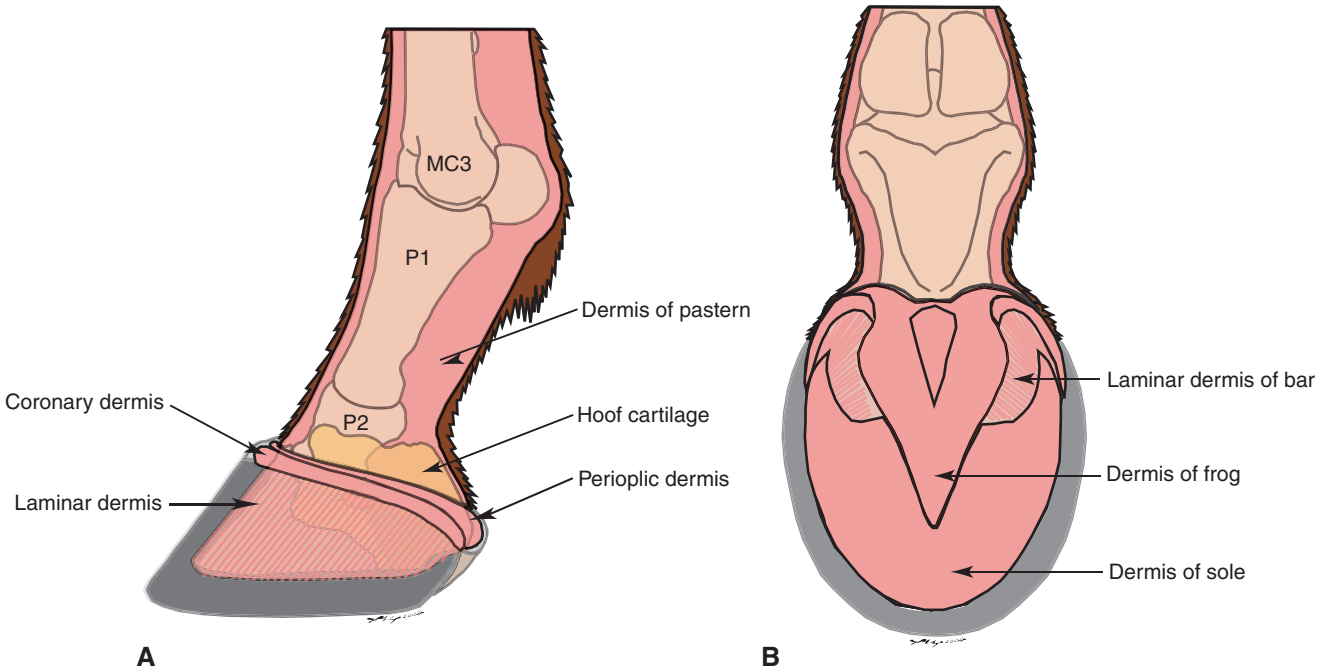


FIGURE 1-4 **A,** Left forefoot, standing, lateral view. **B,** Forefoot, ventral view.

Box 1-1 Glossary of Anatomical Terms

Term	Explanation
abaxial	Directional: farther away from the axis or related to the periphery of the limb; used when *medial* and *lateral* would not be decisive; relative to *axial*
abductor muscle	A muscle whose action is to abduct the limb (i.e., move the limb, or part of the limb, away from the central longitudinal vertical plane of the body)
adductor muscle	A muscle whose action is to adduct the limb (i.e., move the limb, or part of the limb, toward the central longitudinal vertical plane of the body)
articular	Pertaining to a joint
articular surface	The surface of a bone involved in forming a joint and in contact with the corresponding surface of the other bone(s) that makes up that joint
axial	Directional: closer to, or related to, the axis or center line of the limb; used when *medial* and *lateral* would not be decisive; relative to *abaxial*
bursa (*pl.* bursae)	A fluid-filled sac located between tissues to reduce friction
carpus	The joint between the radius (the bone in the forearm) and the cannon bone, otherwise known as the knee
cartilaginous	Containing cartilage (a special connective tissue comprising amorphous ground substance and some collagen fibers)
coffin bone	Third or distal phalanx of the horse's digit; also known as the pedal bone and P3
collagenous	Containing collagen fibers (fibrous structural proteins)
condyle	A rounded or expanded portion at the end of a bone; it forms part of the joint surface
digit	The portion of the limb distal to (i.e., below) the fetlock joint
distal	Directional: away from the body or toward the free end or extremity of the limb; relative to *proximal*
dorsal	Directional: the forward-facing aspect of the forelimb or hindlimb below the carpus or tarsus in the standing horse
epicondyle	A slightly raised area of bone adjacent to a condyle
extensor muscle	A muscle whose action is to move one bone relative to another such that the angle formed at the joint between the two bones is increased
fascia	Sheets or bands of fibrous tissue that cover or envelope structures beneath the skin
fetlock	The joint between the cannon bone and the digit in each limb; the metacarpophalangeal (forelimb) or metatarsophalangeal (hindlimb) joint
fibrous	Composed of collagen fibers
flexor muscle	A muscle whose action is to move one bone relative to another such that the angle formed at the joint between the two bones is reduced
fossa (*pl.* fossae)	A depression in the surface of a bone
joint capsule	A fibrous tissue sleeve, lined with synovial membrane, that joins the ends of the bones that form a joint
lateral	Directional: farther from the central longitudinal vertical plane of the body in the standing horse; relative to *medial*
ligament	A band of fibrous tissue that joins structures together
medial	Directional: closer to the central longitudinal vertical plane of the body in the standing horse; relative to *lateral*
palmar	Directional: the rear-facing aspect of the forelimb below the carpus in the standing horse; corresponds with *plantar* in the hindlimb
pastern	The region of the digit between the fetlock and the hoof
phalanges	The three major bones of the equine digit
plantar	Directional: the rear-facing aspect of the hindlimb below the tarsus in the standing horse; corresponds with *palmar* in the forelimb
proximal	Directional: toward the body or the attached end of the limb; relative to *distal*
sagittal	Directional: refers to any vertical plane passing through the limb and parallel to the long axis of the body in the standing horse
scutum (*pl.* scuta)	A plate of firm (yet nonbony) tissue that gives support to structures lying beneath or passing over a region
sesamoid bones	Small bones embedded in tendons, ligaments, or joint capsules; most often the term specifically refers to the proximal palmar/plantar sesamoid bones at the fetlock joint, but the navicular bone is also a sesamoid bone (the distal sesamoid bone)
synovial	Referring to the fluid found within a joint, bursa, or tendon sheath (e.g., the synovial membrane secretes synovial fluid); sometimes called *joint oil*
tarsus	The joint between the tibia (the bone in the gaskin) and the cannon bone, otherwise known as the hock
tendon sheath	A fluid-filled sleeve that surrounds certain tendons
tendon	Sheet, cord, or band of fibrous tissue that connects a muscle to a bone or other structure

Epidermis

This outermost layer of the skin is formed by a multilayered sheet of cells that become flattened and packed with the protein keratin as they near the skin surface.

Dermis

This thick feltwork of collagen, blood vessels, and nerves underlies the epidermis of the skin and the hoof capsule (where it is also referred to as the corium). It contains the blood vessels that supply the cells of the epidermis, including those that form the hoof capsule, as well as nerve endings, which provide for various sensations and control of blood flow. Within the hoof capsule, different regions of dermis are named according to the parts of the hoof capsule that they underlie; for example, the solar dermis (or solar corium) is the dermis underlying the sole.

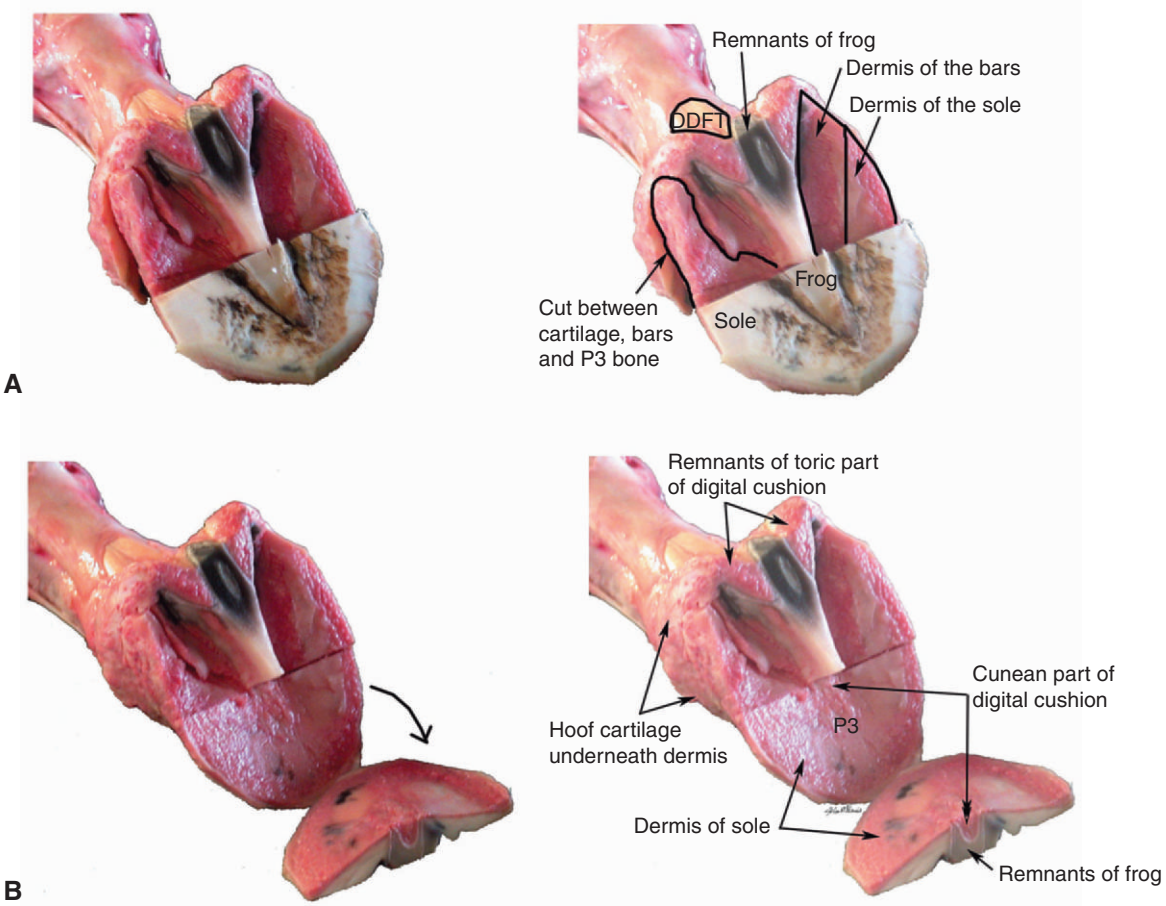


FIGURE 1-6 **A,** Underside of hoof with most of the hoof capsule removed. **B,** Piece of sole and frog cut through the underlying dermis to separate it from the third phalanx (P3). *DDFT,* Deep digital flexor tendon.

Coronet

The coronet (corona) is the region of the digit where the skin meets the hoof capsule. The contour of the digit bulges slightly at the coronet because of an accumulation of fat in the subcutis. This fatty connective tissue is called the coronary cushion, or pulvinis coronae.

HOOF CAPSULE

The hoof capsule is a skin derivative, like a modified fingernail, and it entirely encloses the bone (the third phalanx) at the extremity of the digit. It is made up of the wall and bars, the sole, the frog, the periople, and the bulbs of the heels. The hoof capsule thus forms a coherent, resilient boot, such that distortion of any particular part affects the remainder (unless there are tears or breaks in its form) (Figure 1-7).

Hoof Wall

The hoof wall is the part of the hoof capsule that is visible when the foot is squarely placed on the ground. It grows down from the coronary dermis to the ground and forms a truncated, incomplete cone that is folded in on itself on each side at the heels. It is thickest at the toe (dorsal surface) of the hoof and tapers off in thickness toward the heels.

The hoof wall can be divided into the toe, the quarters (medial and lateral), the heels (or angles), and the bars. The toe is the dorsal third of the hoof wall and the quarters form the sides, while the heels are the regions where the wall folds back on itself at the caudal boundary of each quarter.

The angle that the hoof wall makes with the ground at the toe ranges between about 45 degrees in very flat feet and 65 degrees in very upright feet. The overall shape of the hoof wall is generally more sloping at the toe, in the front hooves, and on the lateral side of each hoof. It is more upright at the heel of the hoof, in the hind hooves, and on the medial side of each hoof.

Coronary Groove

The coronary groove is a shallow groove that runs around the inner surface of the hoof wall at the coronet. In the living hoof, this groove is filled by the coronary dermis, which supports the epidermis that grows the hoof wall.

Insensitive Laminae

The epidermal, or insensitive, laminae line the internal surfaces of the hoof wall and the bars of the heels. They consist of about 600 thin folds of horn that extend from the coronary groove down to the sole. They interdigitate (interlock) with

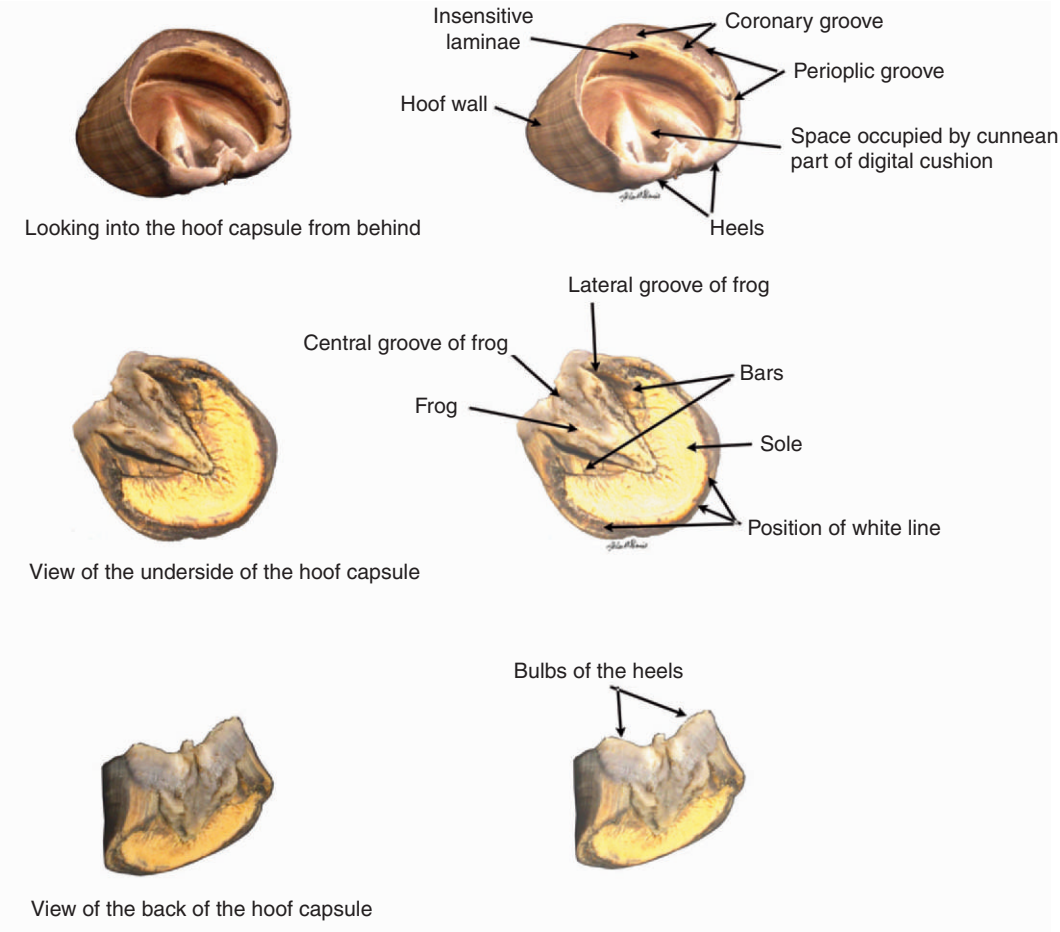


FIGURE 1-7 Hoof capsule.

the dermal, or sensitive, laminae underlying the hoof wall and the bars (see Chapter 6).

Bars of the Heels

The bars of the heels are those parts of the hoof wall that fold back toward the toe on the ground surface of the hoof. They contribute to the boundaries of a triangular space occupied by the frog. The lateral and medial bars blend with the sole as they converge toward the toe.

White Line

The white line is visible on the ground surface of the hoof capsule. It is the ring of horn of a lighter color and softer texture that indicates the boundary between the sole and the hoof wall.

Sole

The sole is a concave, crescent-shaped layer of horn that forms the surface on the underside of the hoof capsule. It is located between the ground-contacting surface of the hoof wall and the centrally located frog, bounded by the hoof wall and by the bars of the heels. The body of the sole lies in front of the apex of the frog and the two limbs of the sole extend caudally on either side of the frog.

Frog

The frog is a roughly triangular, wedge-shaped mass of keratinized tissue. It has a forward-pointing apex and a broad base caudally. There is a shallow central groove or cleft (central sulcus) that deepens caudally, toward the base. The frog has an appearance and consistency similar to hard rubber. It protrudes downward, toward the ground surface, from between the limbs of the sole, and occupies the triangular space between the bars of the hoof.

On each side of the frog there is a deep groove or cleft (paracuneal sulcus or commissure) between the frog and the bars of the hoof. Like the central groove, these lateral grooves are deepest toward the back of the hoof. The base of the frog is at the back of the hoof capsule, where it expands upward and sideways to blend with the bulbs of the heels, thus completing the back of the hoof capsule.

Periople

The periople is the narrow band of soft, pale horn that forms a ring around the top of the hoof wall. It widens over the heels to form the bulbs of the heels before blending into the frog. The perioplic horn continues distally as the stratum externum (or stratum tectorium) of the hoof wall. However, this thin, outermost layer of the wall is generally worn away,

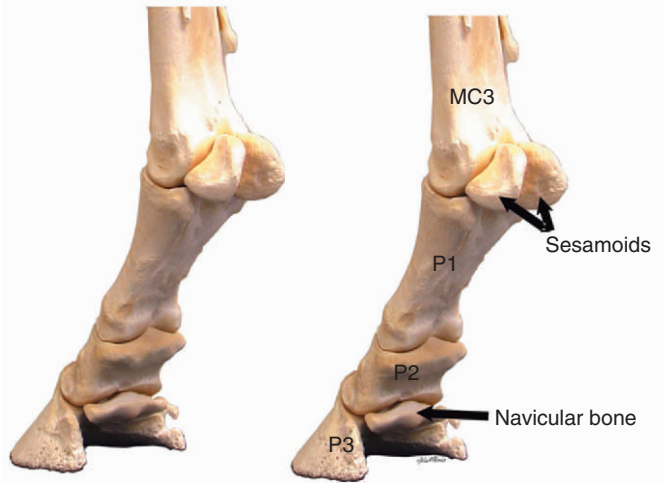


FIGURE 1-8 Palmaromedial view of the bones of the digit.

so it does not extend farther than the top 1 cm or so of the hoof wall in most hooves.

Bulbs of the Heels

The bulbs of the heels are the softer, rounded regions of horn immediately above the heels of the hoof wall, where the periople widens and thickens before blending with the frog to complete the back of the hoof capsule. NOTE: The term "bulbs of the heels" is used by some people to refer to the skin-covered bulges of the toric part of the digital cushion (see Figure 1-6) that lie immediately proximal to the classic bulbs of the heels.

Perioplic Groove

This narrow groove runs around the proximal edge of the coronary groove (see p. 6). In the living hoof, the perioplic groove is filled by the perioplic dermis, which supports the epidermis that grows the periople and stratum externum of the hoof wall. The perioplic groove widens toward the back of the hoof, where the periople becomes continuous with the bulbs of the heels.

BONES

The digit includes four bones: the first phalanx, the second phalanx, the third phalanx, and the distal sesamoid bone (navicular bone). The proximal sesamoid bones are included in this discussion because they are referred to in later sections on the soft tissues of the digit. The hoof cartilages also are included in this section because they can be considered to be extensions of the third phalanx (Figures 1-8 and 1-9).

Proximal Sesamoids

The sesamoids (proximal palmar sesamoids) are the pair of bones that lie on the palmar surface of the fetlock joint, embedded within a mass of ligamentous tissue that unites the suspensory (interosseous) ligament with the distal sesamoidean ligaments and the collateral ligaments of the fetlock joint.

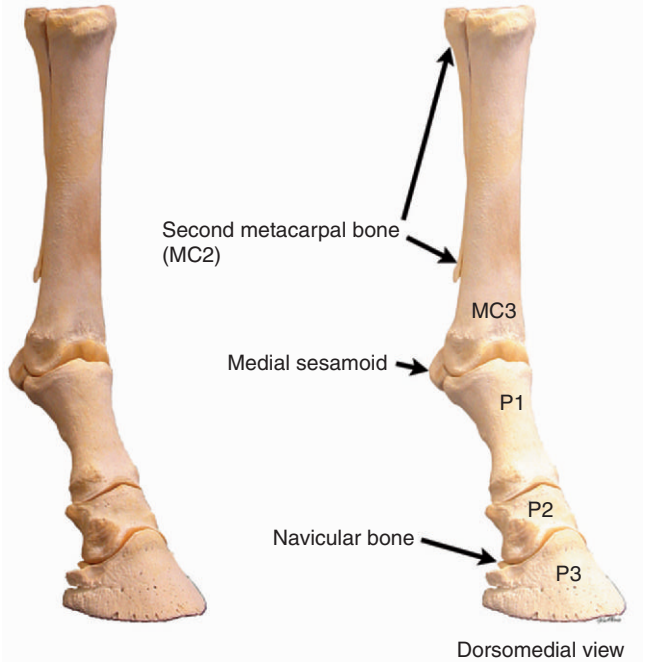


FIGURE 1-9 Dorsomedial view of the bones of the digit.

First Phalanx

The first phalanx (P1), also called the long pastern bone, is the more proximal of the two bones in the pastern. P1 has a flattened, cylindrical shape and is slightly flared at each end. It articulates with the third metacarpal bone (cannon bone) proximally, where it is widest and thickest, and with P2 distally. The dorsal surface of P1, along with the digital extensor tendon, determines the dorsal contour of the pastern, where it can be felt immediately under the skin and the extensor tendon.

The articular surfaces of P1 are concave proximally (at the fetlock joint) and convex distally (at the pastern joint). Both proximal and distal surfaces have a clearly defined central groove that runs front-to-back (a sagittal groove) that separates the slightly larger medial side from the slightly smaller lateral side of the joint surface.

Second Phalanx

The second phalanx (P2), also called the short pastern bone, is the middle one of the three bones that form the bony column in the digit. It has a similar appearance to P1, except that it is generally less than half the length of P1 and so is more cuboidal in overall shape. Like P1, the articular surfaces are concave proximally (at the pastern joint) and convex distally (at the coffin joint), but P2 has a sagittal ridge at its proximal end that fits into the sagittal groove on the distal end of P1, and it has a shallow indentation on its distal articular surface that fits with a median (midline) bulge in the articular surface of P3.

Third Phalanx

The third phalanx (P3), also called the coffin bone or pedal bone, is a semicircular, wedge-shaped bone. It is significantly

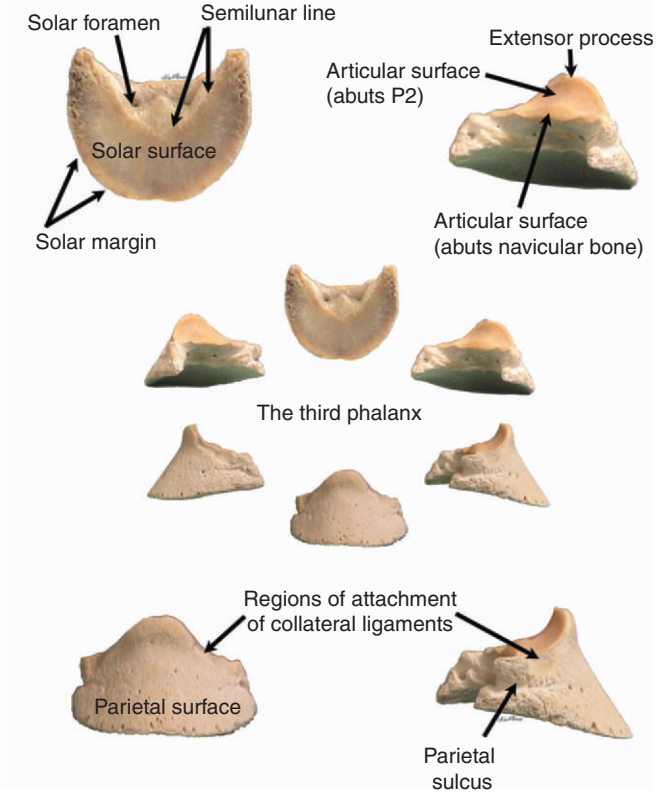


FIGURE 1-10 Third phalanx.

lighter than the other bones of the digit, primarily because it is perforated by numerous vascular channels. The slope and shape of the front and sides (the parietal surface, including the dorsal, medial, and lateral surfaces) of the bone are similar to the slope and shape of the overlying hoof capsule in this region (Figure 1-10).

Solar and Flexor Surfaces

On the underside of P3 is a crescent-shaped, relatively smooth and concave surface. It is called the solar surface, because it lies deep to much of the sole of the hoof. Caudal and proximal to this area is the flexor surface, a roughened region associated with the deep digital flexor tendon. Between these two areas is a roughened, semicircular ridge (the semilunar line), which defines the inner border of the solar surface and separates it from the more proximal flexor surface. The roughened region of the flexor surface immediately adjacent to the semilunar line is where the deep digital flexor tendon attaches to P3.

At its caudoproximal extremity, the flexor surface has a roughened border that forms the boundary between the articular surface of P3 and the flexor surface. The distal navicular impar ligament (Figure 1-11) attaches to P3 along this border. Between this attachment site and the semilunar line there is a pair of holes (the solar foraminae) in P3 through which blood vessels pass. These vessels meet within P3 to form the terminal arch (see Chapter 17).

Proximal Margins

The proximal-dorsal border of P3 forms a smooth peak, the extensor process, to which the digital extensor tendon attaches (see Figure 1-10). The extensor process of P3 fits neatly into a slight indentation in the dorsal edge of the distal articular surface of P2. On each side of the extensor process and the proximal edge of the lateral and medial surfaces of P3 are depressions where the collateral ligaments of the coffin joint (distal interphalangeal joint) attach to P3. These ligaments are discussed in more detail later.

Parietal Sulci and Palmar Processes

The third phalanx has two palmar processes, or wings (medial and lateral) that extend toward the heels. Each of these processes has a groove (the parietal sulcus) on its outer surface that runs forward a variable distance and contains blood vessels.

Solar Border

The semicircular, outermost distal edge of P3, where the parietal and solar surfaces meet, is called the solar border. When the hoof is placed squarely on the ground, the solar border is oriented close to the horizontal plane.

Navicular Bone

The navicular bone (distal sesamoid bone) is so called because it is shaped like a boat. (It is also called the shuttle bone because it is shaped like a weaver's shuttle.) The navicular bone hangs in a ligamentous sling on the palmar aspect of P3 (see p. 11). It articulates with P3 via a narrow, distal articular surface that may run the full width of the coffin joint. Caudodistal to this articular surface is a narrow ridge where the impar ligament attaches across to P3 (as described on p. 12 (Figure 1-11).

The main articulation of the navicular bone, however, is with P2, on the palmar region of the distal articular surface of P2. The entire dorsal surface of the navicular bone is dedicated to this articulation. This surface has a broad, midline ridge and slight concavities on each side. Altogether, the articular surfaces of the navicular bone account for between one quarter and one third of the entire coffin joint surface area.

Flexor Surface

The flexor surface of the navicular bone is the palmar surface over which the deep digital flexor tendon runs. It is situated on the side opposite the articular surface. The flexor surface faces distally, at a slight angle to the ground. It has a narrow median ridge with broad, shallow concavities extending on each side. There is a fluid-filled sac (the navicular bursa; see p. 11) that lies between the flexor surface of the navicular bone and the deep digital flexor tendon.

Hoof Cartilages

The hoof cartilages (also called the lateral, collateral, or ungual cartilages) are cartilaginous extensions of P3. They are variable in their extent, tending to be thicker and more extensive in the forelimbs than in the hindlimbs, and varying between breeds and types of horses, as well as between individuals of the same breed and type with different work histories (Figure 1-12). A cartilage extends from P3 on each side of the foot (medial and lateral), with each cartilage comprising two regions: basal and proximal.

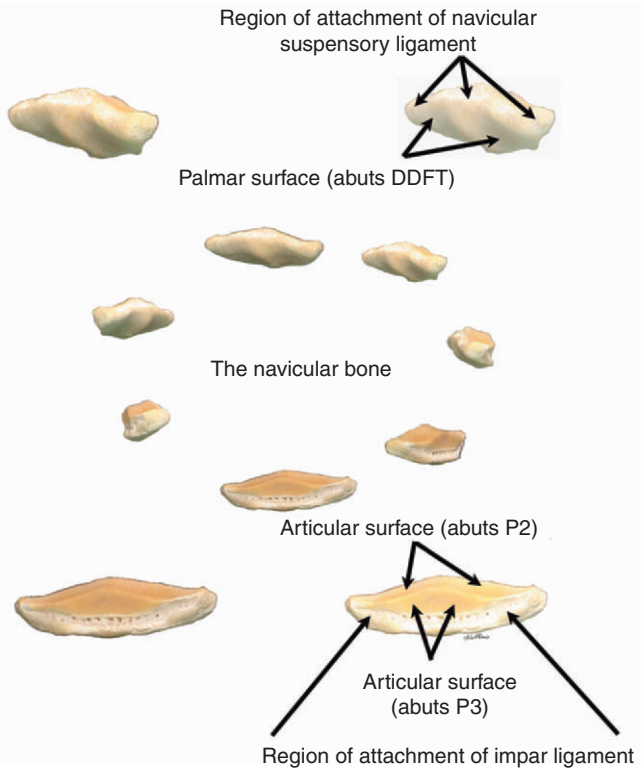


FIGURE 1-11 Navicular bone. *DDFT,* Deep digital flexor tendon.

Basal Region

The basal region forms an irregularly shaped, roughly rectangular mass of cartilage that extends caudally (i.e., toward the heels) and inward (i.e., axially) from P3. The irregular basal masses of each cartilage (medial and lateral) lie deep to the bars and the bulbs of the heels, and to the hoof wall behind the quarter line. The basal region of each hoof cartilage often extends into the basal parts of the digital cushion (see p. 10).

Proximal Region

The proximal region comprises a thin plate of cartilage that extends proximally and caudally from P3. The proximal edge of this platelike, springy cartilage can be palpated just above the coronet, caudal to the extensor tendon. As each plate extends caudally, it widens and forms the support for the bulbs of the heel, where the free edge of each cartilage can be palpated immediately under the skin. The soft material that can be felt beneath the skin between the two cartilages in the region of the heels is the digital cushion.

JOINTS, BURSAE, AND TENDON SHEATHS

Proximal Interphalangeal (Pastern) Joint

The proximal interphalangeal (PIP) joint, or pastern joint, is the articulation between P1 and P2. It lies about two thirds to three quarters of the way down the pastern and can be identified by the distinct bulge in the pastern where the distal end of P1 flares (Figure 1-13).

At the front and back of the joint, the joint capsule extends proximally to form dorsal and palmar pouches, or recesses (Figure 1-14). These irregular pockets of joint capsule lie against the dorsal and palmar surfaces of P1, respectively, beneath the overlying soft tissues (discussed later).

Distal Interphalangeal (Coffin) Joint

The distal interphalangeal (DIP) joint, or coffin joint, is the articulation between P2, P3, and the navicular bone. It is completely enclosed within the hoof. As described above for the PIP joint, the DIP joint capsule extends proximally to form both dorsal and palmar pouches, or recesses. These irregular pockets of joint capsule lie against the dorsal and palmar surfaces of P2, respectively.

Navicular Bursa

The navicular bursa is a synovial cavity (a fluid-filled sac) that lies between the flexor surface of the navicular bone and the deep digital flexor tendon (DDFT). Its connections with both of these structures and with the other structures in this region are rather complex (Figure 1-15).

T Ligament

The T ligament is a wide, thin, elastic sheet of fibrous material that is located at the proximal-palmar edge of the navicular bone. It forms a three-way attachment between the navicular bone, the palmar surface of P2, and the deep surface of the DDFT (i.e., the surface that faces the navicular bone and bursa). In the process, the T ligament separates the three synovial cavities in this area—the navicular bursa, the palmar pouch of the coffin joint (which extends proximally along the palmar surface of P2), and the flexor tendon sheath (see below).

The T ligament appears as a T-shaped structure only on sagittal sections of the digit (i.e., in sections cut in a front-to-back orientation). The two parts of the T (the crosspiece and the upright) connect the DDFT to either P2 or the navicular bone.

Pastern Branch of the Deep Digital Flexor Tendon

The crosspiece of the T is called the pastern branch of the DDFT in many texts. It is a thin, collagenous attachment between the DDFT and the palmar surface of P2. It originates from the DDFT as a sheet around the distal edge of the curve where the DDFT is thickened by fibrocartilage to form the distal scutum (see Figure 1-16). Thus, the crosspiece of the T is located at the distal boundary of the distal scutum (see Figure 1-15).

Attachment between Deep Digital Flexor Tendon and Navicular Bone

The upright of the T is formed by a collagenous attachment between the DDFT and the navicular bone. It is often considered simply as part of the coffin joint capsule.

These attachments (i.e., the components of the T ligament) are thin, often fat-filled, and covered by synovial membrane. They clearly are not load-bearing structures.

Impar Ligament

The impar ligament is a short, broad, strong ligament (also called the *distal sesamoidean* or *navicular impar ligament*) that attaches the distal margin of the navicular bone to P3 across

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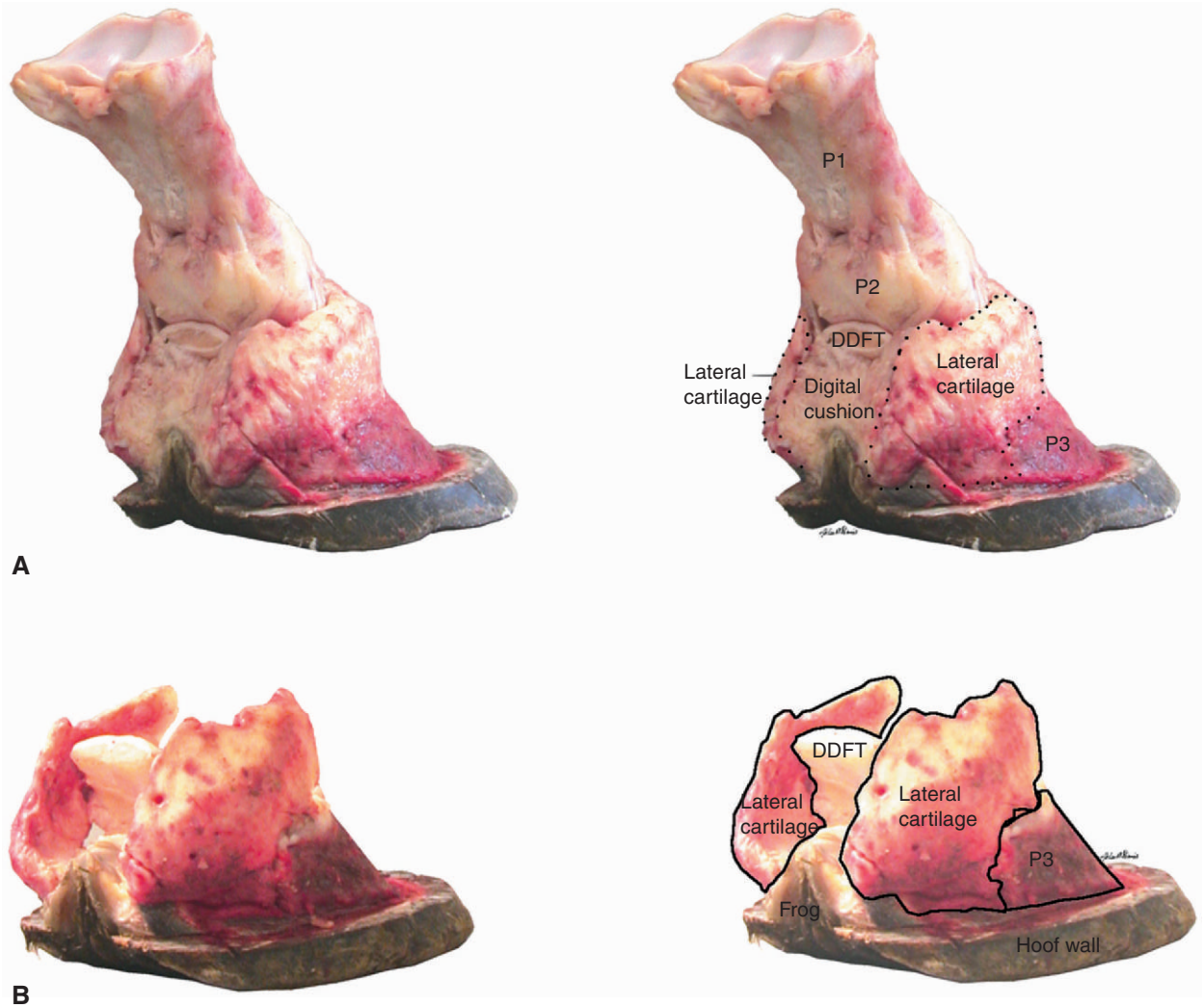

FIGURE 1-12 Hoof cartilages. **A,** Digit with skin, superficial digital flexor tendon (SDFT), and sesamoidean ligaments removed as well as some of the hoof wall, digital cushion, and deep digital flexor tendon (DDFT). **B,** Digit as above with the first phalanx (P1), the second phalanx (P2), and digital cushion removed.

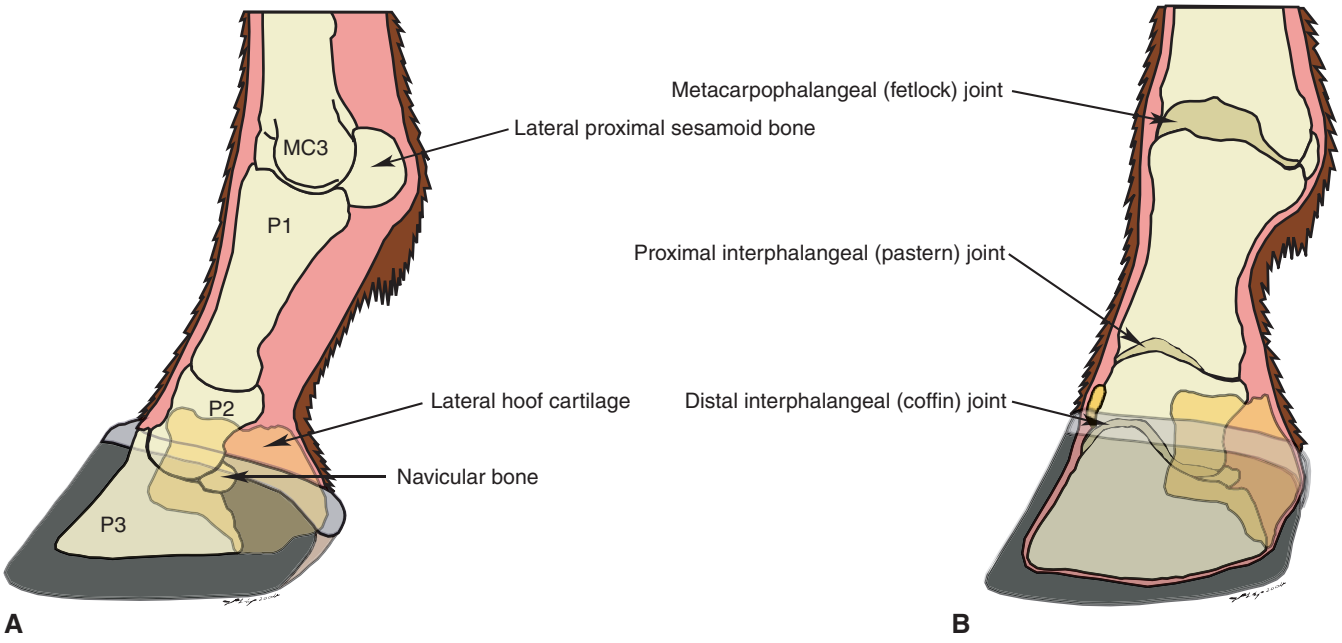

FIGURE 1-13 **A,** Left forefoot, standing, lateral view. **B,** Left forefoot, standing, craniolateral view.

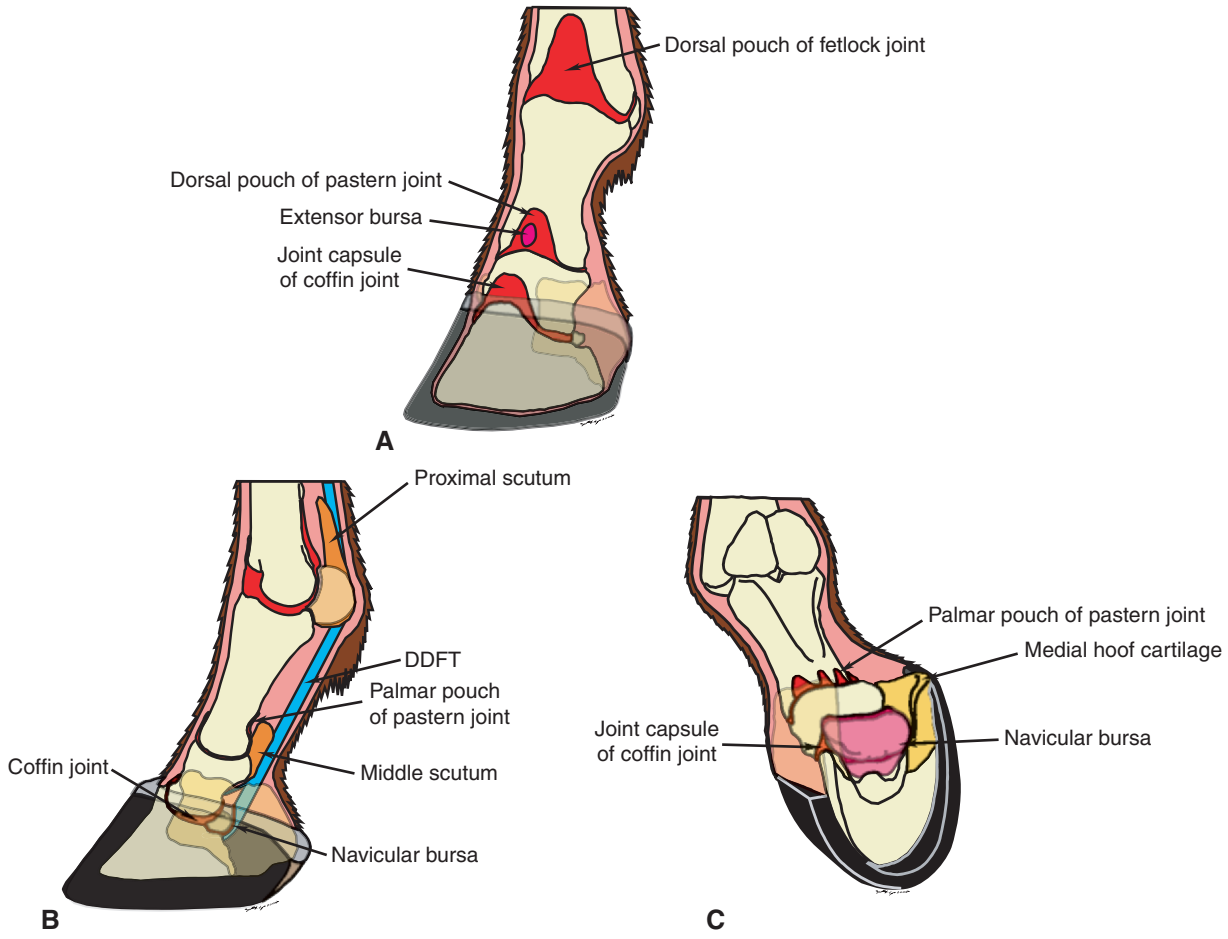

FIGURE 1-14 **A,** Left forefoot, standing, craniolateral view. **B,** Left forefoot, standing, lateral view. **C,** Left forefoot, elevated, caudolateral view. *DDFT,* Deep digital flexor tendon.

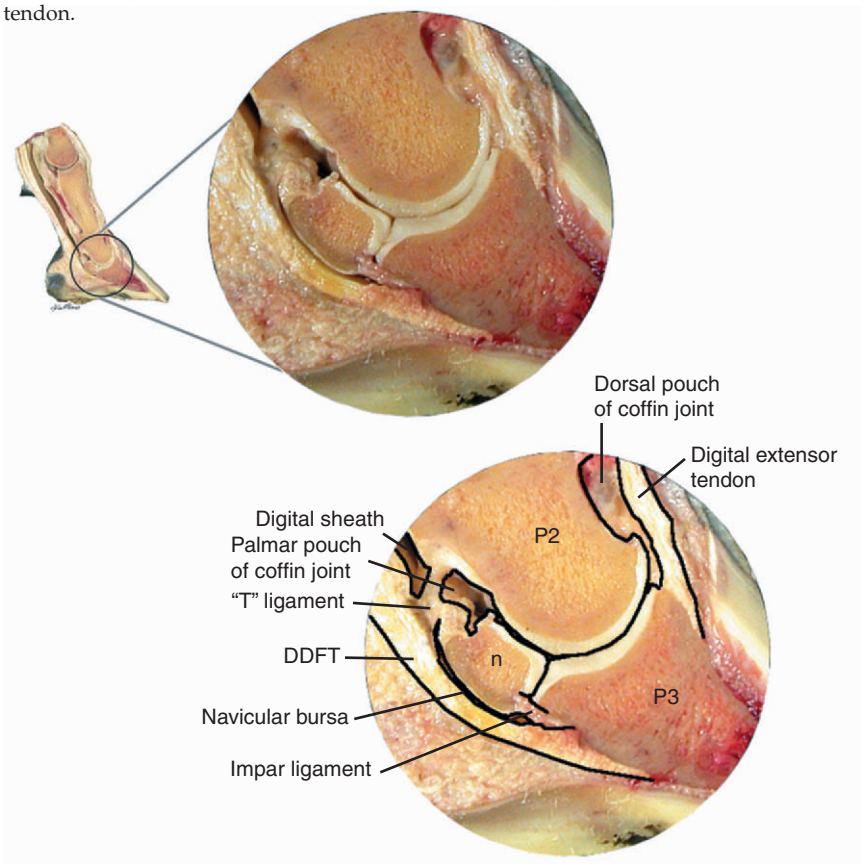

FIGURE 1-15 Sagittal section showing the coffin joint and navicular bursa. *DDFT,* Deep digital flexor tendon.

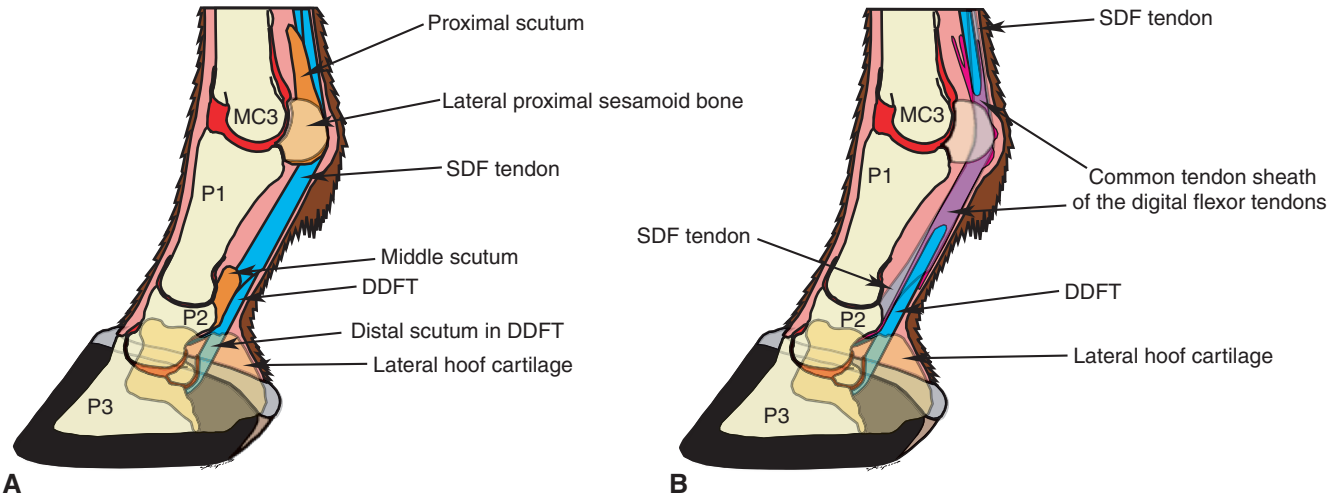

FIGURE 1-16 **A,** Left forefoot, standing, lateral view. **B,** Left forefoot, standing, lateral view.

the entire width of the joint surface at this level. The impar ligament separates the coffin joint capsule from the navicular bursa in this region. At its medial and lateral margins, the impar ligament is continuous with the chondronavicular ligaments (see Figure 1-21).

Other Connective Tissues

Proximally, the medial and lateral sides of the navicular bursa are formed by loose connective tissue that attaches to the edges of the DDFT and merges with the fascia in this region. More distally, the medial and lateral edges of the navicular bursa are formed by the suspensory ligaments of the navicular bone (see Figure 1-19). These edges are continued distally by the ligamentous attachments between the DDFT, the navicular bone, P3, and the lateral cartilages (discussed previously). This attachment finally blends into the insertion of the DDFT around the semilunar line of P3, thus completing the dorsal boundary of the navicular bursa.

Flexor Tendon Sheath

The common tendon sheath of the digital flexor tendons (flexor tendon sheath or fetlock tendon sheath) surrounds and lubricates the passage of the superficial and deep digital flexor tendons (SDFT and DDFT, respectively) from above the fetlock joint to the middle of P2. This tendon sheath is intimately associated with the fibrous tissue (fascia) that encases the digit (see Figure 1-16).

In the region of P1, the tendon sheath is formed largely by the SDFT itself as it forms a ring around the DDFT before splitting and attaching to the sides of P2 (discussed later). The tendon sheath at this level overlies the straight and oblique sesamoidean ligaments (see Figure 1-19)

The tendon sheath then continues distally, overlying the rest of the straight sesamoidean ligament and the middle scutum (a pad of fibrocartilage on the proximal-palmar surface of P2; see below). About halfway down P2, the sheath ends where it meets the pastern branch of the DDFT (the crosspiece of the T ligament; discussed above). Thus, the passage of the DDFT within the digit is lubricated by both the flexor tendon sheath and the navicular bursa.

Extensor Bursa

There is often a small bursa (or sequence of bursae) over the dorsal surface of the pastern joint, lying between the joint capsule and the extensor tendon, where fibrous strands from the deep face of the extensor tendon attach to the proximal-dorsal edge of P2. This bursa is much smaller than, separate from, and more superficial than the dorsal recess of the pastern joint capsule (Figure 1-17).

Variably, other minor bursae are found in the digit between tendons and ligaments and between these structures and the bones. These small bursae are most often associated with the attachments of the digital extensor tendons within the digit.

Other Specialized Soft Tissues

Middle Scutum

The middle scutum is a thickened region of fibrocartilage on the proximal palmar surface of P2 (see Figure 1-16). It is formed where the straight sesamoidean ligament and the axial palmar pastern ligaments coalesce with the superficial digital flexor tendon branches before their insertion into the proximal border of P2. (These tendons and ligaments are discussed in detail later.) The middle scutum thus forms a resilient extension of the proximal palmar surface of P2, over which the deep digital flexor tendon (in its tendon sheath) glides.

Distal Scutum

The distal scutum is a fibrocartilagenous thickening in the deep digital flexor tendon that forms a pad proximal to the navicular bone and immediately palmar and distal to the middle scutum (see Figure 1-16). Along with the intervening flexor tendon sheath, the middle and distal scuta allow the deep digital flexor tendon to slide easily against P2.

LIGAMENTS

Collateral Ligaments

The collateral ligaments of a joint are thick bands of collagen that are located on the medial and lateral sides of the joint.

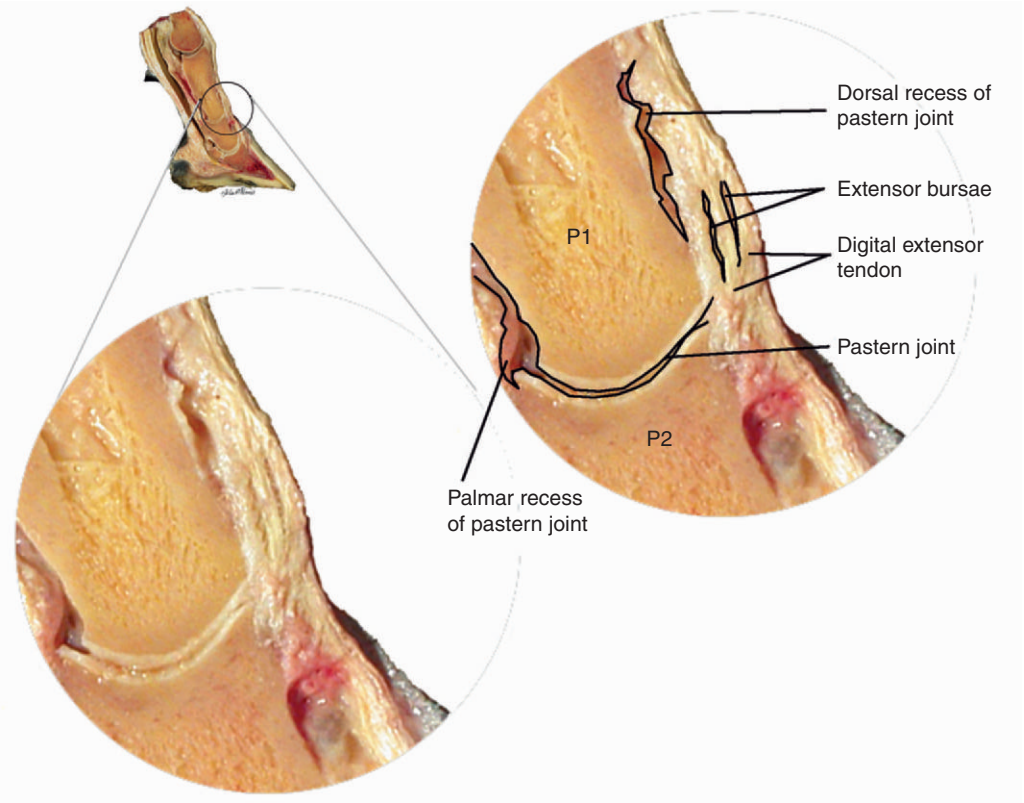

FIGURE 1-17 Extensor bursa.

Each collateral ligament attaches the adjacent ends of the bones that form the joint, on either the lateral or the medial side of the joint. They act to restrict medial and lateral bending (adduction and abduction) and rotational movements so that the joint is mainly limited to movement within a sagittal plane (i.e., flexion and extension).

The collateral ligaments of the pastern and coffin joints lie in a similar orientation, relative to the long axis of the digit. When looking at the left limb from the lateral side, in the normal standing position in a normally conformed horse, the lateral collateral ligaments of these joints lie at an angle between about 30 and 45 degrees counterclockwise from the axis of the bones (Figures 1-18 and 1-19). In horses with more upright pasterns, the angle tends to be closer to 30 degrees, and in horses with more sloping pasterns the angle tends to be greater. The orientation of the medial collateral ligaments is parallel to that of the lateral collateral ligaments.

Axial and Abaxial Palmar Ligaments

The connection between P1 and P2 at the pastern joint is further strengthened and stiffened by two extra pairs of ligaments called the axial and abaxial palmar ligaments. They are farther from the center of rotation of the joint than the collateral ligaments, so they reduce the range of movement in the pastern joint even more (see Figure 1-19).

The axial palmar ligaments are located at the back of the pastern, on each side of the straight sesamoidean ligament (see Figure 1-19). They originate on the palmar surface of P1, about midshaft, and insert onto P2 on each side of the attachment of the straight sesamoidean ligament. In the process, they form part of the middle scutum (see Figure 1-16).

The abaxial palmar ligaments are longer than the axial ligaments and are located farther around to the sides of the pastern. They originate in the middle of P1, directly below the insertion of the collateral ligaments of the fetlock joint, and they run distally, across the pastern joint, and insert onto the sides of P2, near the insertions of the SDFT (see Figure 1-19).

Suspensory Ligaments of the Navicular Bone

The complex attachments of the navicular bone to surrounding structures and the indistinct anatomical differentiation between the various components has led to considerable confusion in terminology of the structures in this region. The ligaments that support the navicular bone in its position on the palmar aspect of the coffin joint form a composite structure with potentially five components (Figures 1-20 to 1-22).

Suspensory Ligament of the Navicular Bone

The first component consists of a slinglike structure, comprising a thick horizontal band that runs along the palmar edge of the navicular bone and two vertical arms that extend from the extremities of this band upward on each side of P2. These vertical arms attach to the distal end of P1, just dorsal to the collateral ligaments of the pastern joint. Hereafter we will refer to this component as the *suspensory ligament of the navicular bone*. (In some texts, the vertical arms of this structure are referred to as the collateral ligaments of the navicular bone.)

Second and Third Components

The second and third components consist of ligamentous attachments between P2 and the medial and lateral extremities of the navicular bone. These structures can be considered as

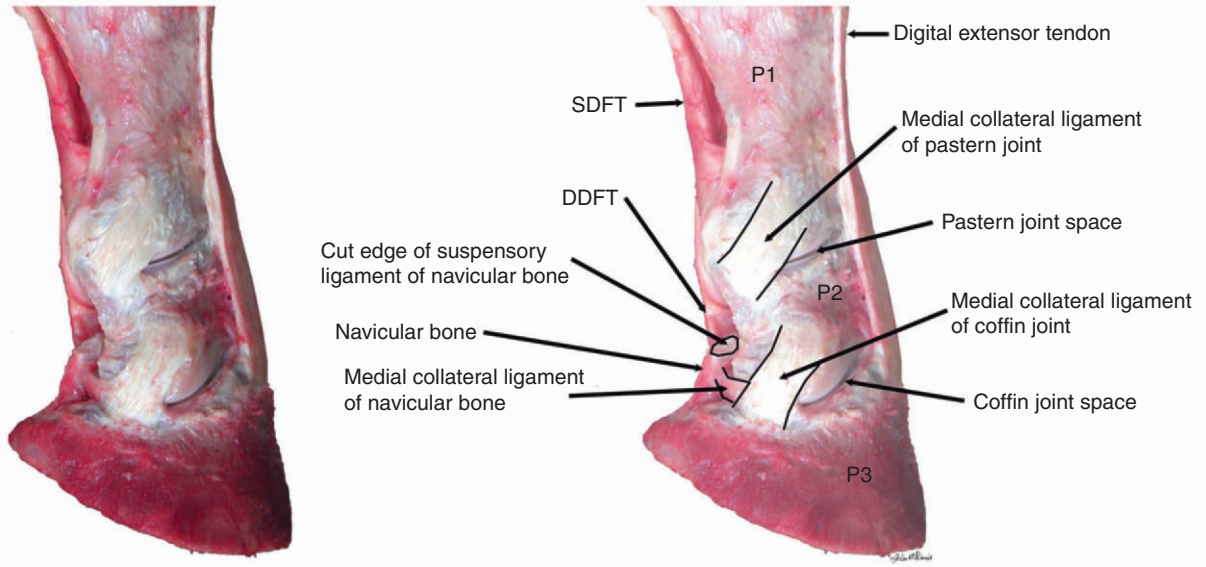

FIGURE 1-18 Collateral ligaments. Dorsomedial oblique view of digit with skin, hoof capsule, lateral cartilages, digital cushion, medial part of digital extensor tendon, fascia, and vessels removed. *DDFT,* Deep digital flexor tendon; *SDFT,* superficial digital flexor tendon.

part of the collateral ligaments of the coffin joint or as part of the suspensory ligament of the navicular bone. If viewed from inside the coffin joint, each collateral ligament of the coffin joint has an additional part that is about half the width of the main ligament. These additional parts connect P2 to the outer edges of the navicular bone at a slightly greater angle than the main parts of the coffin joint collateral ligaments; they also merge into the suspensory ligament of the navicular bone.

Fourth and Fifth Components

The fourth and fifth components of the navicular suspensory complex consist of short ligamentous attachments between P3 (including the hoof cartilages) and the medial and lateral extremities of the navicular bone. These short ligaments are called the chondronavicular ligaments in many texts.

The chondronavicular ligaments are described as attaching directly between the navicular bone and the hoof cartilages on each side of the foot. However, the collagenous material that connects across these regions in the specimens we have examined show clear continuations *past* the strong cartilage connection and onto P3 or P2. These ligaments can therefore be considered to be part of the complex of the coffin joint collateral ligaments or as medial and lateral extensions of the impar ligament that runs through the hoof cartilages.

Clarifying the Nomenclature

It would be clearer to confine the term *collateral ligaments of the navicular bone* to the parts of the collateral ligaments of the coffin joint that connect to the navicular bone, that is, the connection between P2 and the navicular bone, which takes in at least some of the attachments to the hoof cartilages and includes components two and three and parts of components four and five described above. The term *suspensory ligament of the navicular bone* should be used to indicate the part of the ligament that suspends the navicular bone from P1 and is intimately associated with the collateral ligaments of the pastern joint (i.e., the first of the five components described above).

Chondrocoronal Ligaments

The chondrocoronal ligaments attach the dorsal extensions of the hoof cartilages to the dorsal part of the collateral ligaments of the coffin joint. In the specimens we have examined, these attachments have been much thinner and more sheetlike than the clearly aligned collagen of the coffin joint collateral ligaments that join P2 to the navicular bone and to P3 and attach strongly to the cartilages along the way (Figure 1-23).

The chondrocoronal ligaments are located above, but are *continuous with,* the fascial attachments of the cartilage to the navicular bone. It appears that only a small proportion of the collagen strands attach the cartilage directly to P2 and could thus be accurately described as chondrocoronal ligaments. It would seem that the inconsistent descriptions and naming of these ligaments in the anatomical literature may be due to the different routes of dissection used to investigate them, or possibly to anatomical differences between different types or breeds of horse.

Other Ligaments Associated with the Navicular Bone

The other ligaments associated with the navicular bone (specifically, the T ligament and the impar ligament) are discussed, in the section on the Navicular Bursa.

Sesamoidean Ligaments

There are three ligaments that originate on the distal border of the proximal sesamoid bones at the back of the fetlock joint and are of interest in this discussion of the digit. They are the straight sesamoidean ligament and the pair of oblique sesamoidean ligaments (medial and lateral) (Figure 1-24; see also Figure 1-19).

Straight Sesamoidean Ligament

The straight sesamoidean ligament is a thick, wide ligament that runs from the distal border of the proximal sesamoid

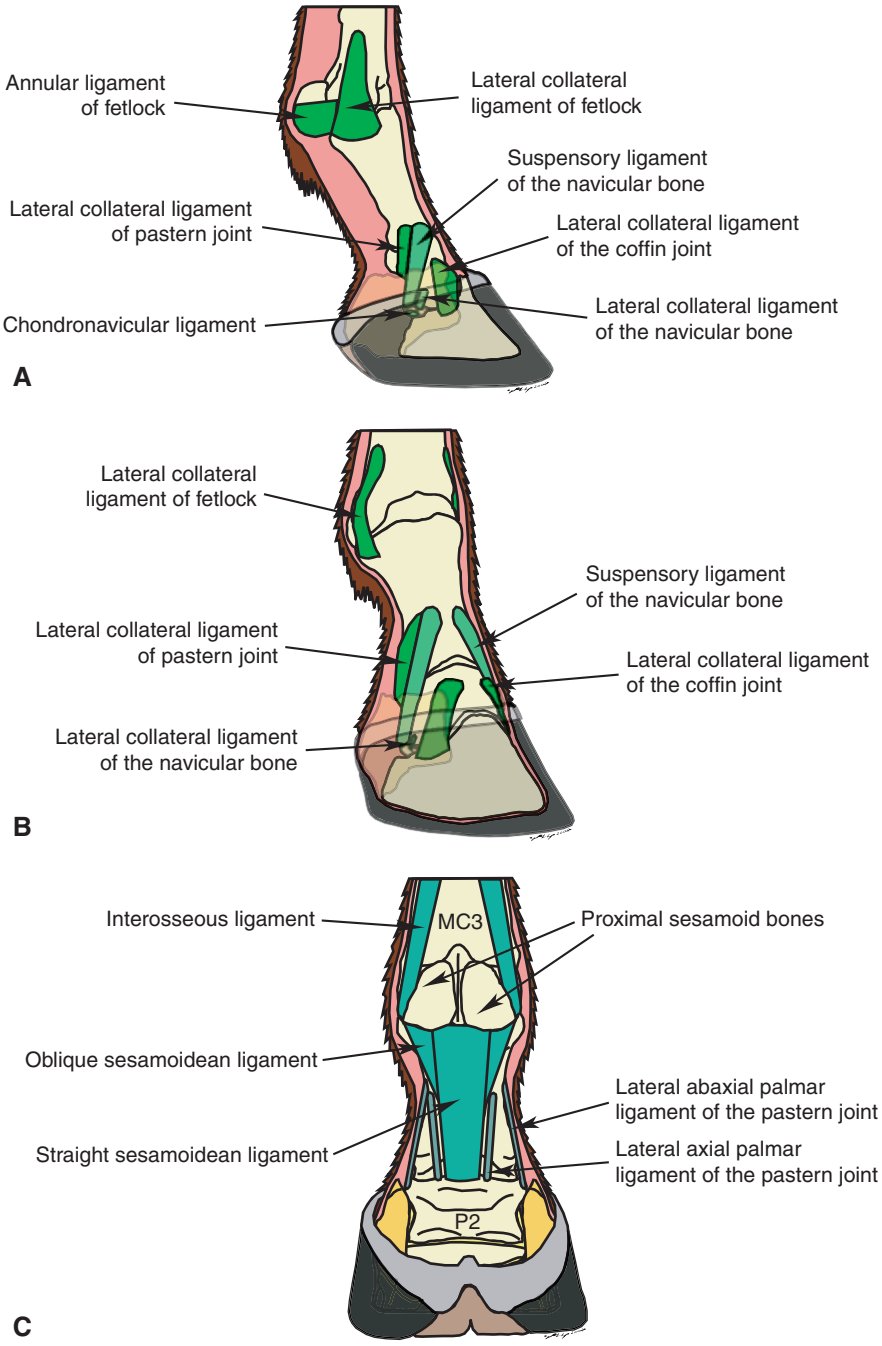

FIGURE 1-19 **A,** Right forefoot, standing, lateral view. **B,** Right forefoot, standing, craniolateral view. **C,** Right forefoot, standing, caudal view.

bones down to the middle scutum, where it joins with the axial palmar ligaments of the pastern at their insertion onto P2 (see Figure 1-19).

Oblique Sesamoidean Ligaments

The oblique sesamoidean ligaments are a pair of short straps that run distally from the distal border of the medial and lateral proximal sesamoid bones (see Figure 1-19). They converge as they attach onto the oblique bony ridges that form a V on the palmar surface of P1. These ligaments run along each side of the straight sesamoidean ligament and insert deep to it (the straight sesamoidean ligament inserting more distally).

TENDONS

Digital Extensor Tendon

The common (forelimb) or long (hindlimb) digital extensor tendon runs down the dorsal surface of the digit, ultimately inserting onto the extensor process of P3 (Figures 1-25 to 1-27).

Forelimb

In the forelimb, the common digital extensor is the tendon of a compound muscle (also called the common digital extensor) that lies against the radius on the lateral side of the

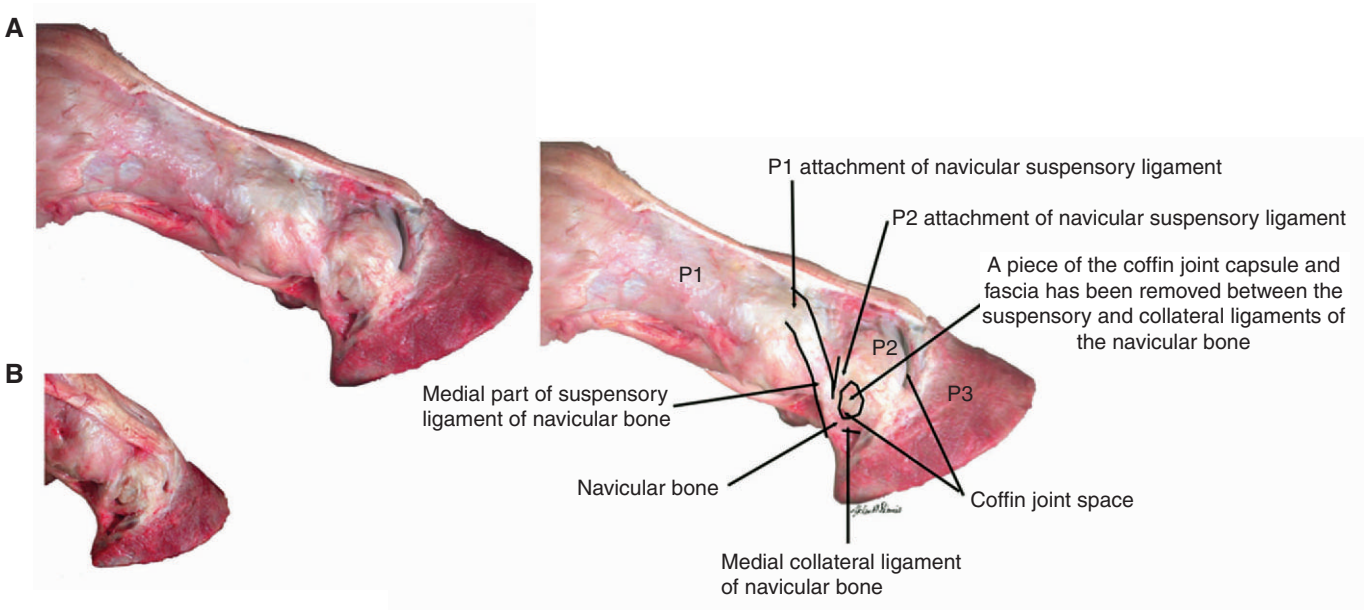

FIGURE 1-20 **A,** Palmaromedial oblique view of the digit with skin, hoof capsule, hoof cartilages, digital cushion, part of the digital extensor tendon, fascia, and vessels removed. **B,** With digital extensor intact.

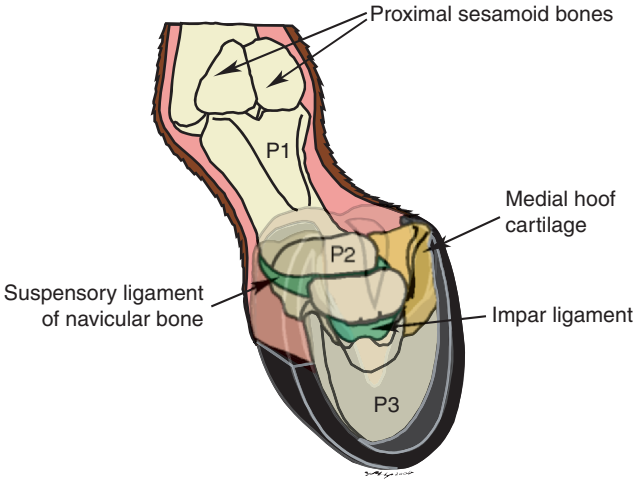

FIGURE 1-21 Left forefoot, elevated, caudolateral view.

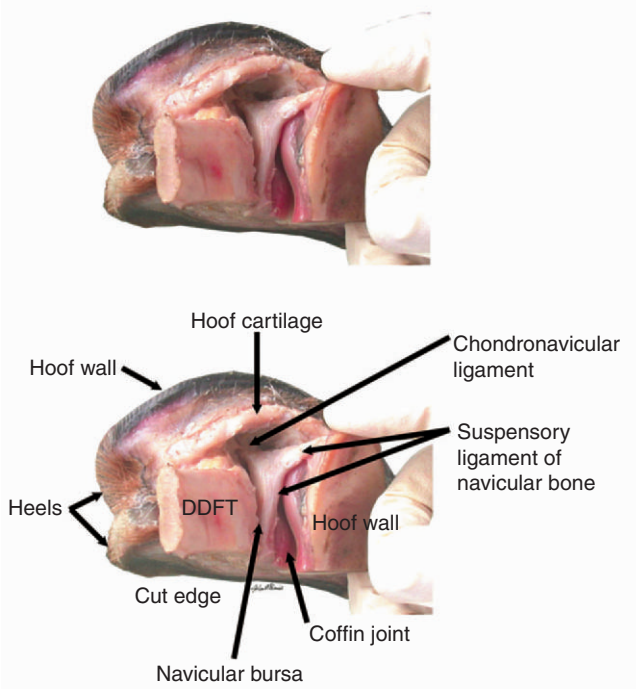

FIGURE 1-22 Chondronavicular ligament. Looking down onto a sagittally sectioned hoof with skin, fascia and T ligament removed. P2 has been cut transversely and vertically. *DDFT,* Deep digital flexor tendon.

forearm, between the extensor carpi radialis muscle and the lateral digital extensor muscle. It has a large humeral belly that arises from the lateral epicondyle of the humerus and smaller radial and ulnar bellies that arise from the lateral tuberosity and border of the radius and the body of the ulna, respectively. The common tendon arises from the muscle bellies just proximal to the carpus (knee) and continues over the dorsal surface of the carpus, metacarpus (cannon), and fetlock.

Hindlimb

In the hindlimb, the long digital extensor muscle originates from the extensor fossa of the femur, along with the peroneus tertius tendon. The single muscle belly lies immediately under the skin and fascia on the craniolateral aspect of the tibia, where it covers the cranial tibial muscle and peroneus tertius tendon in the gaskin. The extensor tendon arises from the muscle belly just proximal to the tarsus (hock) and continues over the dorsal surface of the tarsus, metatarsus (cannon), and fetlock.

Digital Attachments

Within the digit, the digital extensor tendon attaches to P2 and the pastern joint capsule at the level of the pastern joint

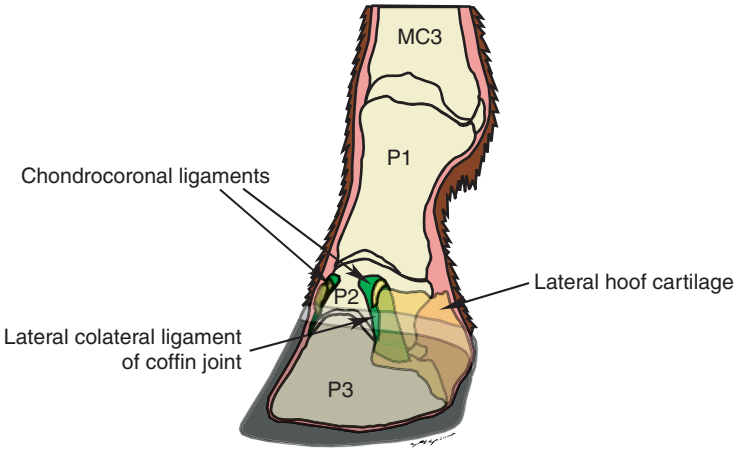

FIGURE 1-23 Left forefoot, standing, craniolateral view.

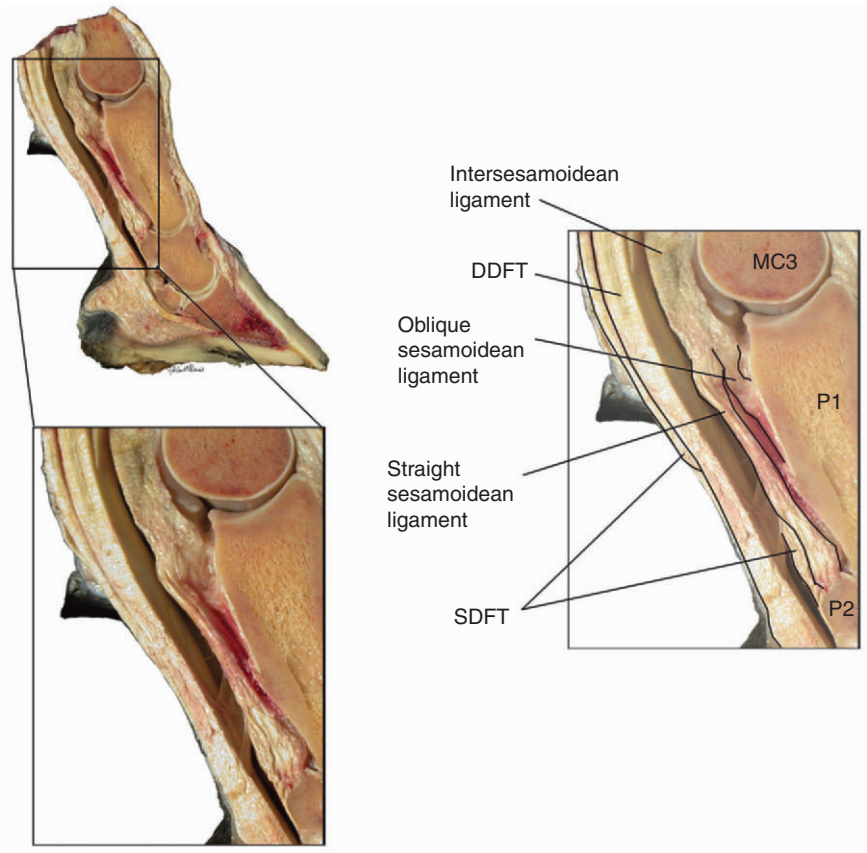

FIGURE 1-24 Sagittal section showing flexor tendons and sesamoiden ligaments. *DDFT,* Deep digital flexor tendon; *SDFT,* superficial digital flexor tendon.

and then continues to the extensor process of P3. At its termination on the extensor process, it is intimately related to the dorsal edges of the hoof cartilages and to the medial and lateral collateral ligaments of the coffin joint. At the level of the pastern, the edges of the digital extensor tendon merge with the tough, elastic fascia that lies just beneath the skin and envelops the deeper structures.

Extensor Branches of the Suspensory Ligament

The extensor branches (slips) of the suspensory, or interosseous, ligament are thin continuations of this ligament that extend past the main attachments on the proximal sesamoid bones at the back of the fetlock. The extensor branches continue obliquely across the sides of P1 and merge into the edges of the digital extensor tendon; thus, they ultimately insert onto the extensor process of P3 (Figure 1-28).

Superficial Digital Flexor Tendon

In the digit, the superficial digital flexor tendon (SDFT) is located under the skin and the proximal digital annular ligament (see Figure 1-28) on the palmar surface of the limb. Its final points of attachment are the proximal margins of P2.

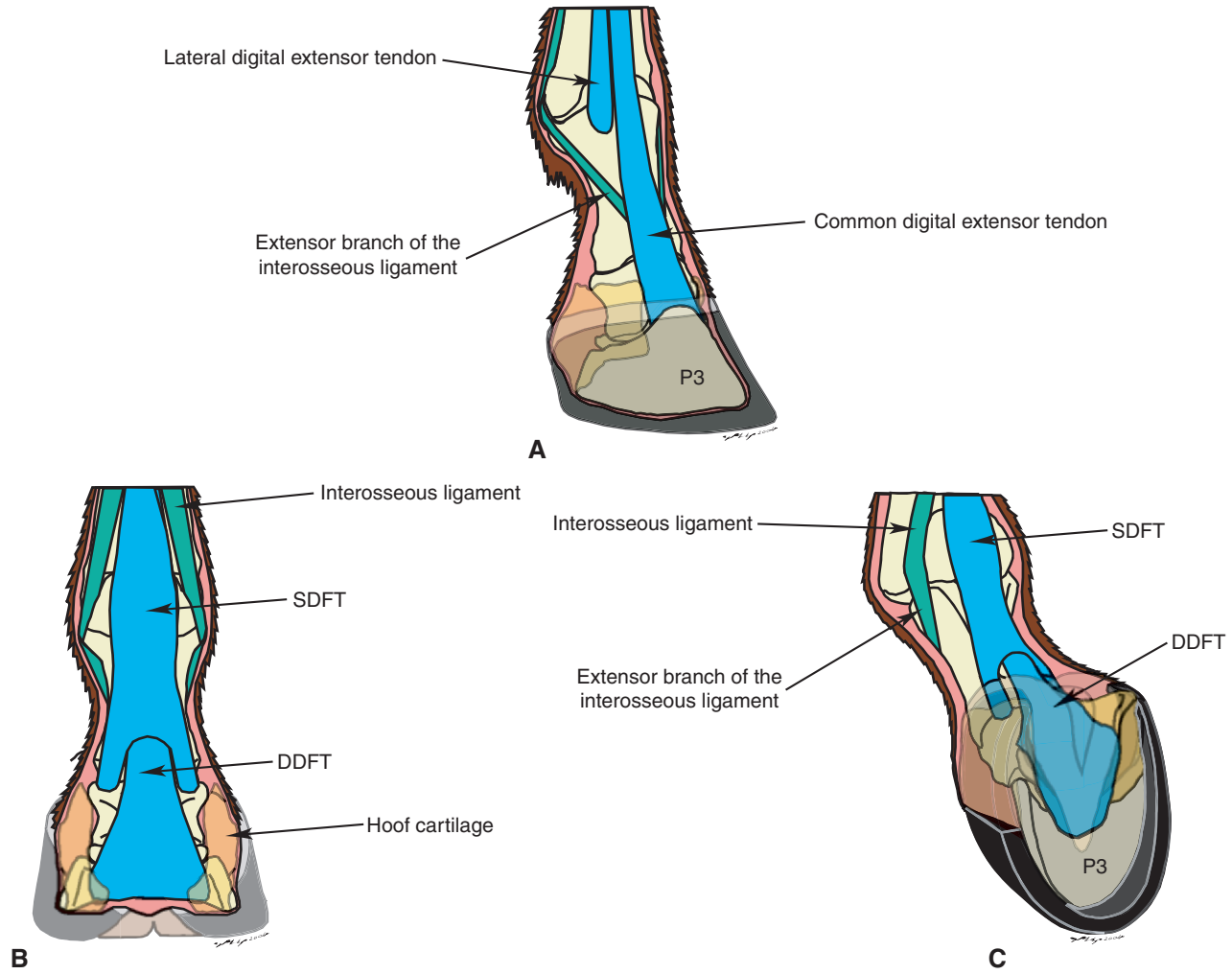

FIGURE 1-25 **A,** Right forefoot, standing, craniolateral view. **B,** Forefoot, standing, caudal view. **C,** Left forefoot, elevated, caudolateral view. *DDFT,* Deep digital flexor tendon; *SDFT,* superficial digital flexor tendon.

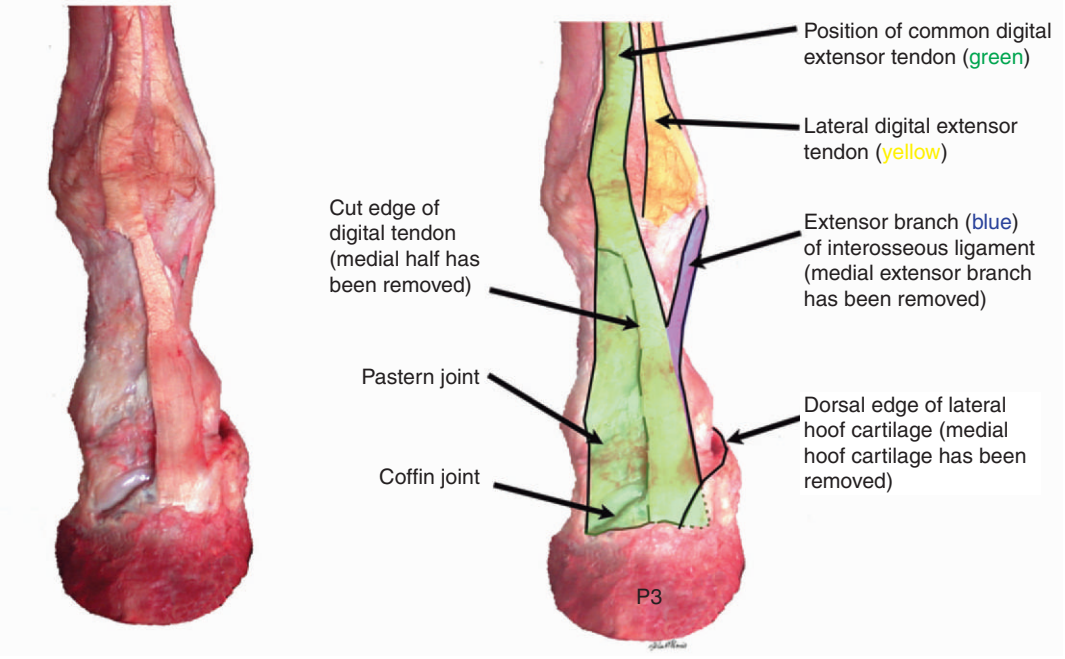

FIGURE 1-26 Digital extensor. Dorsal aspect of the left front digit with skin, dermis, and all the hoof capsule removed as well as one hoof cartilage, one extensor branch of the interosseous ligament, and half the distal portion of the digital extensor tendon (all on the medial side).

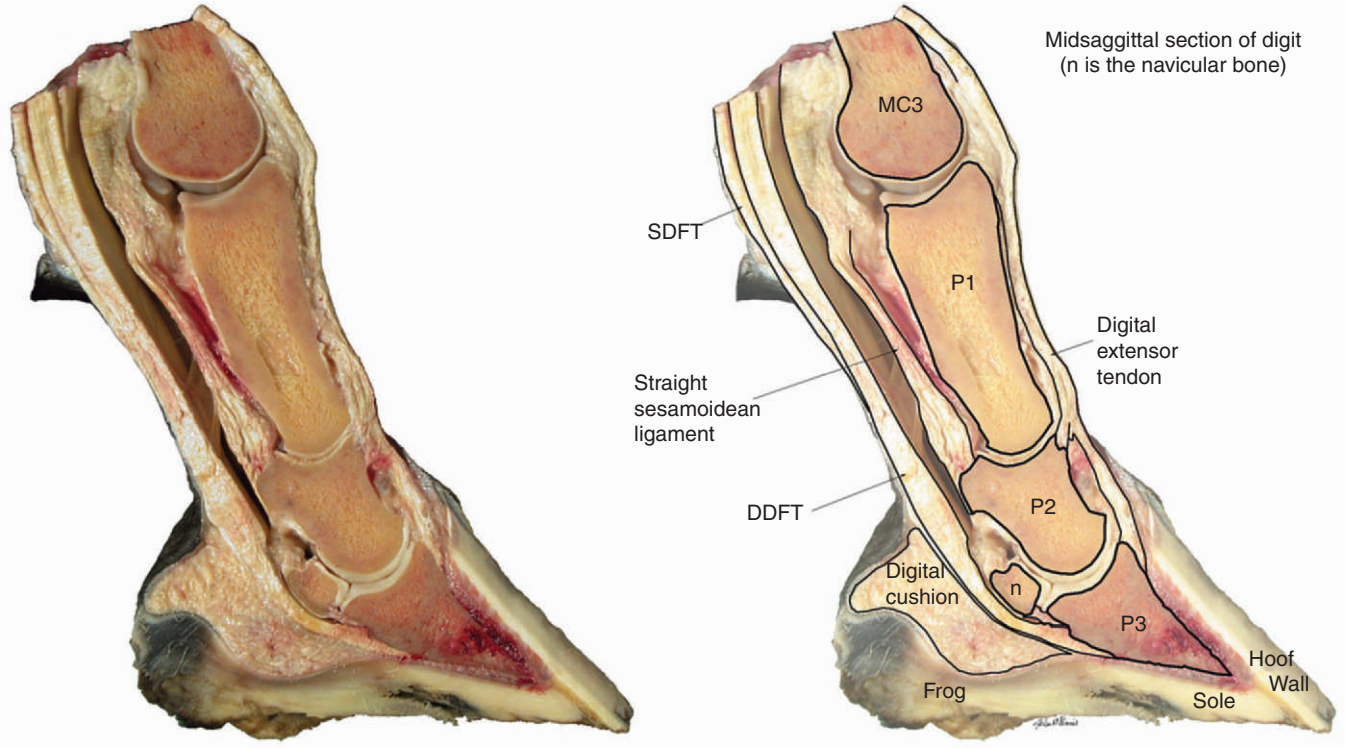

FIGURE 1-27 Midsaggittal section of the digit.

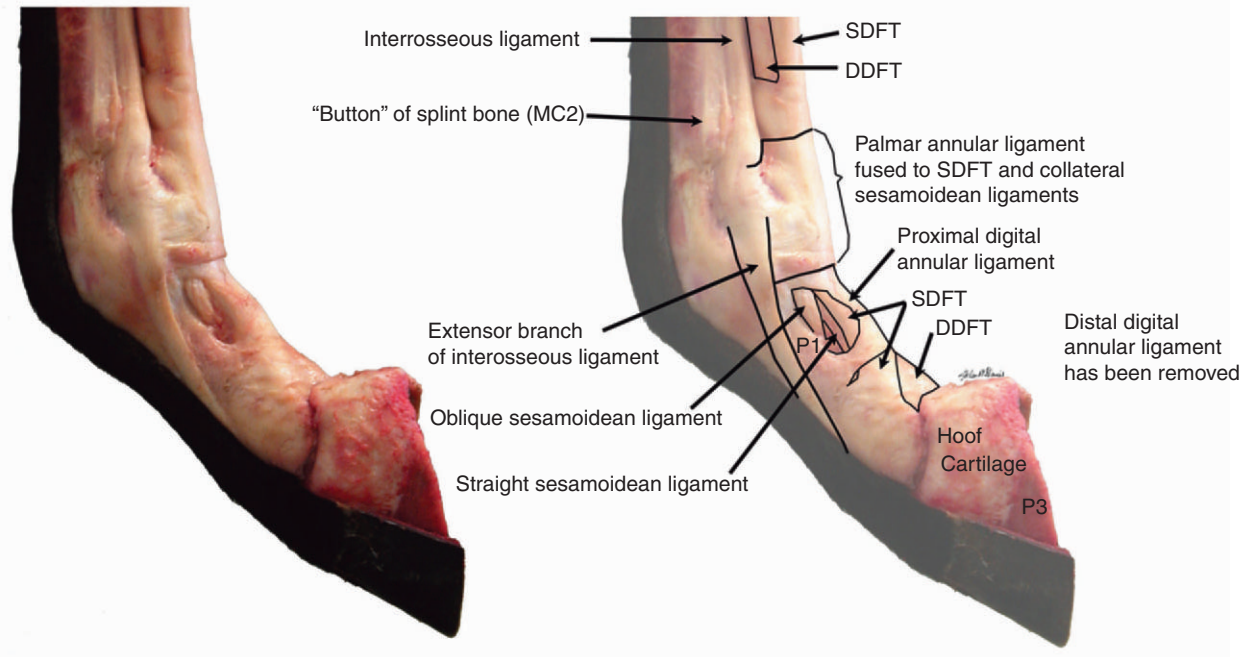

FIGURE 1-28 Flexed lateral aspect of the digit with skin, hoof capsule, dermis, vessels, and superficial fascia removed from the palmar and lateral surfaces. *DDFT,* Deep digital flexor tendon; *SDFT,* superficial digital flexor tendon.

Forelimb

In the forelimb, the SDFT is the tendon of a muscle (the superficial digital flexor) that originates on the medial epicondyle of the humerus and lies in the middle of the flexor group of muscles on the caudal aspect of the forearm. It has an accessory ligament (also called the superior check ligament or radial head of this muscle) that attaches to the caudal surface of the radius at about the level of the chestnut. At this point, the muscle has become entirely tendinous. The SDFT continues distally on the palmar aspect of the carpus, metacarpus, and fetlock.

Hindlimb

In the hindlimb, the superficial digital flexor muscle is almost entirely tendinous and originates from a deep fossa

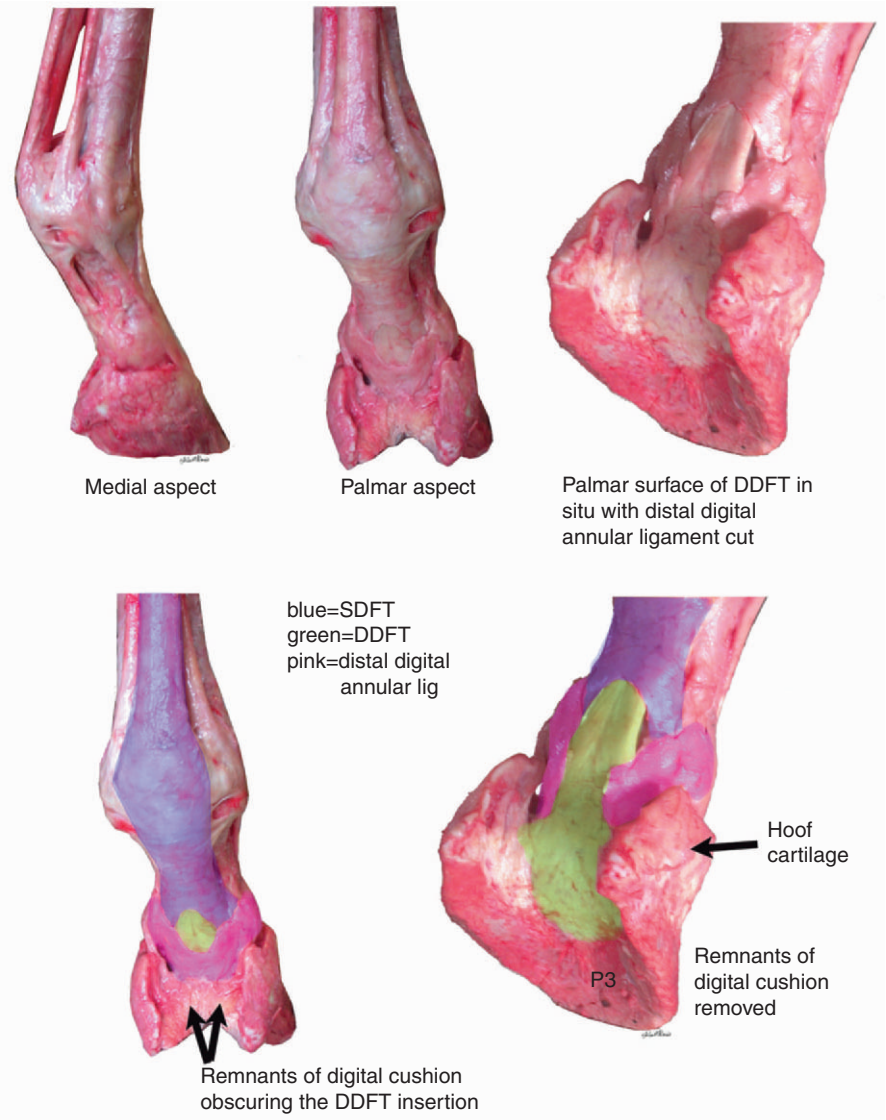

FIGURE 1-29 Lateral and caudal views of the equine digit with hoof capsule, skin, dermis, and most of the digital cushion, superficial fascia, and vessels removed. *DDFT,* Deep digital flexor tendon; *SDFT,* superficial digital flexor tendon.

(supracondyloid fossa) on the caudal aspect of the femur, beneath the heads of the gastrocnemius muscle. The SDFT forms part of the calcanean tendon, along with the tendons of the gastrocnemius muscle, and it attaches medially and laterally to the calcaneus (the point of the hock). Along with the peroneus tertius tendon, the SDFT forms the reciprocal apparatus that links stifle and hock flexion and extension. Distal to the hock, the SDFT courses down the plantar aspect of the metatarsus and fetlock to the digit.

Digital Attachments

Just proximal to the pastern joint, the SDFT divides into two limbs, which pass each side of the DDFT and attach to the proximal aspect of P2, just dorsal to the axial palmar ligaments of the pastern (see Figure 1-25). In many texts, these distal attachments of the SDFT are described as including minor attachments to P1, in addition to the attachments to P2. However, we consider that these minor attachments to P1 are actually part of the proximal digital annular ligament (see Figure 1-28), which is indivisible from the SDFT at this point.

Deep Digital Flexor Tendon

The deep digital flexor tendon (DDFT) lies deep to the SDFT on the palmar aspect of the digit until the SDFT divides to attach onto the sides of P2, as described above. The DDFT then continues distally until its insertion onto the solar surface of P3 (Figure 1-29).

The DDFT is the tendon of a large muscle (the deep digital flexor) that lies on the caudal surface of the radius (forelimb) or tibia (hindlimb) and is considered to have three muscle bellies in both fore- and hindlimbs. It has an accessory ligament that is a direct continuation of the palmar carpal (or tarsal) ligament and that joins the DDFT at midmetacarpus (metatarsus), where, in the forelimb, it forms up to half the bulk of the tendon. In the hindlimb, the accessory ligament is much smaller and may even be absent (Figure 1-30).

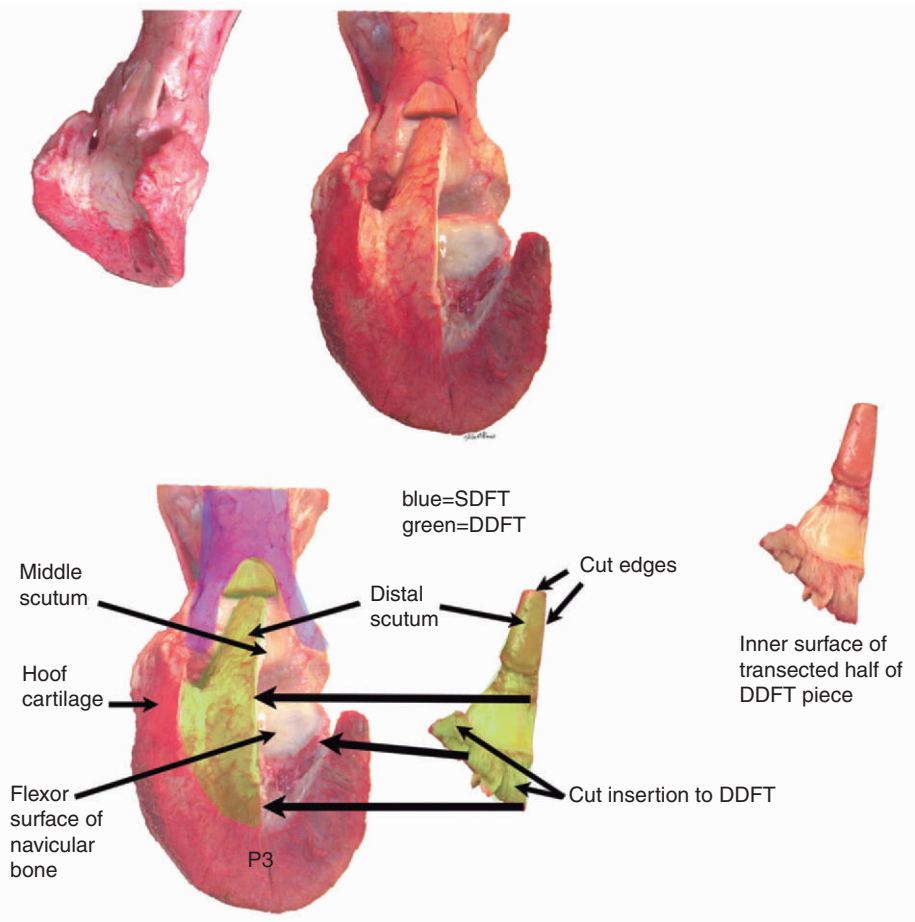

FIGURE 1-30 Deep digital flexor tendon (DDFT) insertion. Palmar aspect of digit with hoof capsule, skin, dermis, digital cushion, fascia, and vessels removed. DDFT has been cut at the proximal edge of the middle scutum and then half of the distal portion removed. One hoof cartilage and half the navicular suspensory ligament are removed from the same side.

Forelimb

In the forelimb, the humeral belly originates from the medial epicondyle of the humerus and lies against the caudal surface of the radius, where it forms the major bulk of the muscle. The humeral belly has large tendinous intersections and can be further divided into three parts (deep, middle, and lateral). The much smaller ulnar belly originates from the medial surface of the ulna and lies superficially between the ulnaris lateralis and flexor carpi ulnaris muscles. When the third belly (the radial belly) is present, it originates from the middle of the caudal surface of the radius and forms a small slip of muscle deep to the humeral belly on the caudal surface of the radius. Tendons from the ulna and radial bellies join with the large tendon from the humeral belly at the level of the carpus.

Hindlimb

In the hindlimb, the three bellies are the deep (long digital flexor muscle of digit 1 or flexor hallucis longus muscle), the superficial (or caudal tibial muscle), and the medial (or long digital flexor muscle). These muscles originate from the lateral surfaces of the proximal tibia distal to the lateral condyle, the caudal surface of the tibia, the interosseous ligament, and caudal regions of the fibula. The deep belly is much larger than the other two, is heavily permeated by tendinous tissue, and lies against the caudal surface of the tibia. The superficial belly is partially fused to the deep belly and lies lateral and distal to it. The smaller medial belly lies obliquely between the deep belly and the popliteus muscle, and its tendon joins with the main DDFT about halfway down the metatarsus.

Digital Attachments

The conjoined tendon courses distally on the palmar aspect of the metacarpus (or plantar metatarsus) and fetlock, deep to the SDFT. Just proximal to the pastern joint, the DDFT emerges from beneath the SDFT when the SDFT divides as described above. At this point, the DDFT lies immediately under the skin for a short length, before passing deep to the distal digital annular ligament (see Figure 1-25). It then dives deep to the digital cushion as it enters the hoof capsule. The DDFT attaches to the semilunar line on the palmar surface of P3 and adjacent regions on the axial (inner-facing) surfaces of the hoof cartilages (see Figure 1-25).

Just below the pastern joint, the DDFT has a thickening that is reinforced with fibrocartilage to form a plaque (the distal scutum; see Figure 1-16). Below this area, the DDFT widens and becomes thinner and sheetlike. Also at this level is a broad, thin sheet of collagen that attaches the DDFT to the navicular bone (thus separating the navicular bursa from the tendon sheath) and an additional attachment to the palmar surface of P2 (pastern branch of the DDFT; see Figure 1-31).

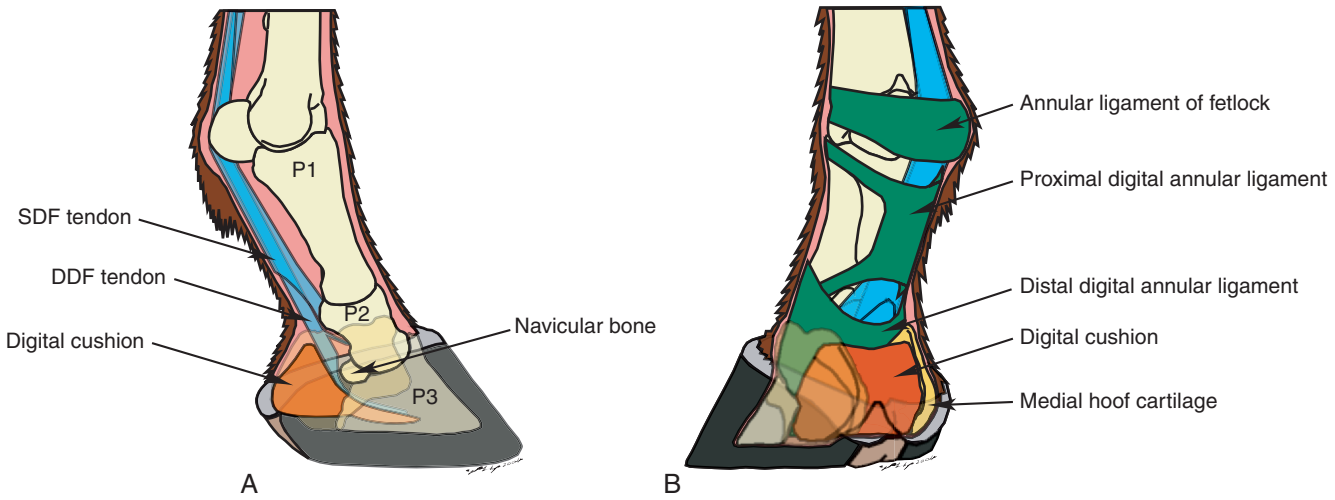

FIGURE 1-31 **A,** Right forefoot, standing, lateral view. **B,** Left forefoot, standing, caudolateral view.

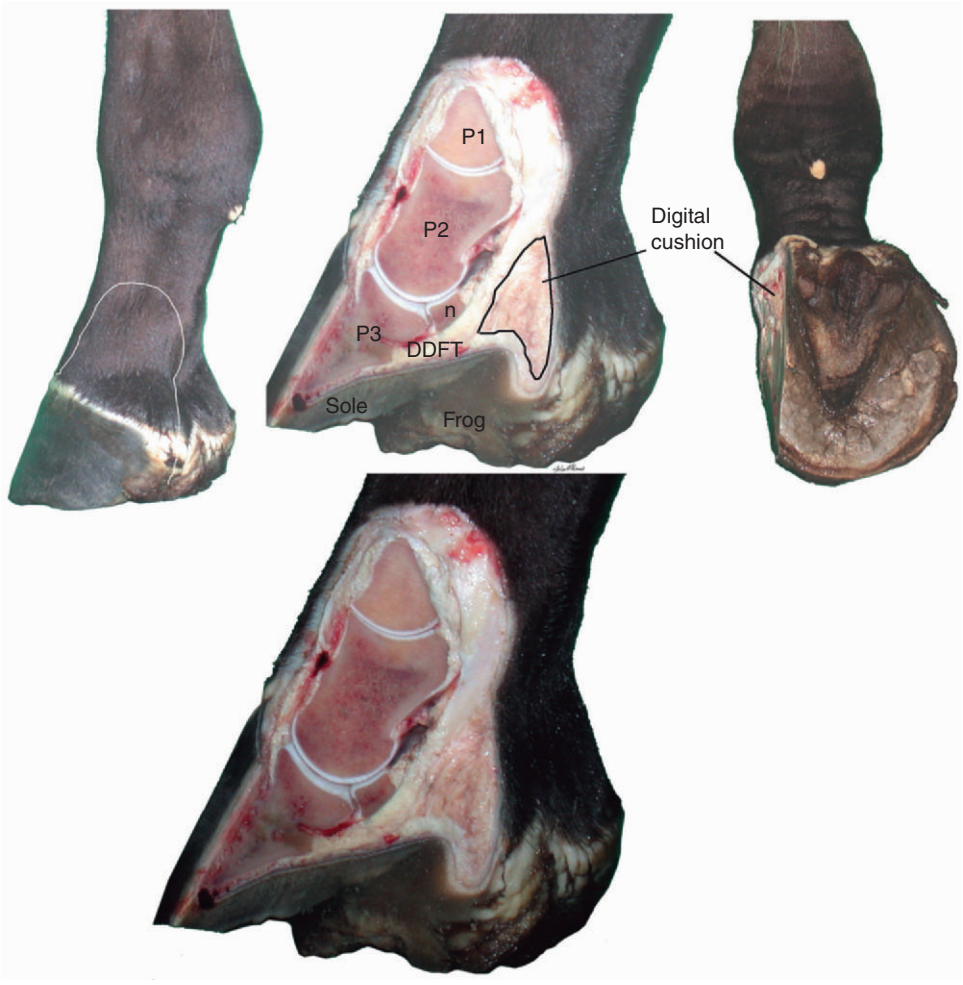

FIGURE 1-32 Hoof sectioned to show the relative position of the digital cushion. *DDFT,* Deep digital flexor tendon.

DIGITAL CUSHION

The digital cushion is a mass of collagen and elastic and fatty tissue that lies just proximal to the frog on the palmar aspect of the digit. The digital cushion has two parts: a large toric part and a smaller cunean part (Figures 1-31 to 1-33).

Toric Part

The major (toric) part is a thick, bilobed pillow of fat contained within an elastic meshwork of collagen. It fills in the region between the hoof cartilages, the frog (below), and the DDFT (above). It bulges caudally to fill out the bulbs of

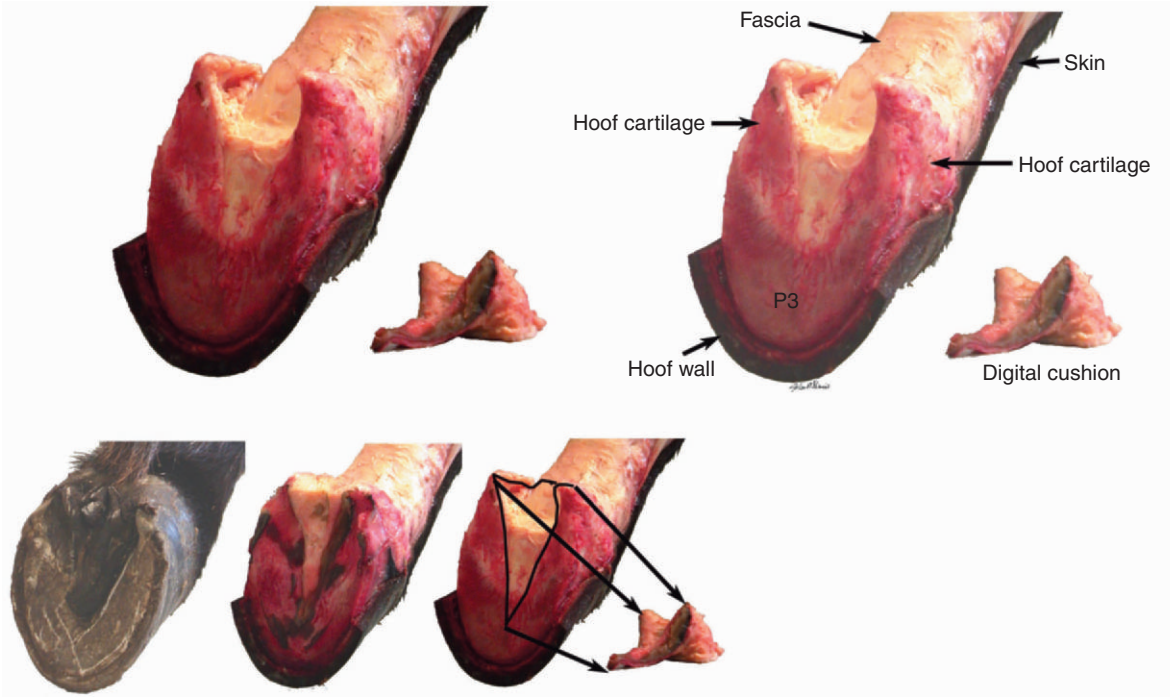

FIGURE 1-33 The digital cushion. Digit with all of the sole and frog and most of the hoof wall and skin removed. The digital cushion has been cut away from the hoof cartilage and the fascia covering the deep digital flexor tendon (DDFT) and placed to the side.

the heels, where it can easily be palpated as the soft material under the skin in this region.

Cunean Part

The cunean part is a much smaller, V-shaped collagenous extension of the toric part. It forms a central, finger-like projection that lies deep to the apex of the frog, filling the internal central cleft of the frog and bulging up internally and forward, under the central (midsagittal) part of the coffin joint. The cunean part of the digital cushion is much tougher and stiffer than the toric part, and it is very strongly attached to the dermis of the sole.

ANNULAR LIGAMENTS

Annular ligaments are thin sheets of collagen that partially or completely encircle the limb (i.e., form an annulus, or ring) and help to hold tendons and other structures down against the bone. Three annular ligaments surround the structures that lie along the palmar surface of the digit: the palmar annular ligament and a pair of digital annular ligaments (proximal and distal) (Figure 1-34).

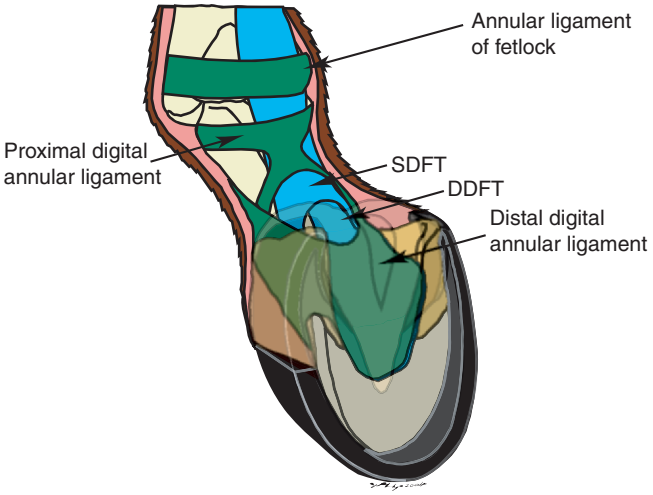

FIGURE 1-34 Left forefoot, elevated, caudolateral view. *DDFT,* Deep digital flexor tendon; *SDFT,* superficial digital flexor tendon.

Palmar Annular Ligament

The palmar annular ligament is a thin but tough sheet of fibrous tissue that encircles, and fuses with, the SDFT as it passes over the fetlock joint. It helps to tether the flexor tendons in place (Figure 1-35).

Digital Annular Ligaments

The two digital annular ligaments run from the medial and lateral sides of P1 around the palmar surface of the flexor tendons, thus holding the tendons down against the palmar surface of P1 (proximal digital annular ligament) and the navicular bone (distal digital annular ligament).

Proximal Digital Annular Ligament

This ligament is roughly X-shaped. The upper arms of the cross originate on each side of the proximal-palmar surface of P1 and come together to form a wide sheet that fuses with the SDFT. The distal arms of the cross appear to be part of the SDFT; they form a pair of distinct straps that provide the SDFT with attachments to P1 (see Figure 1-35). These attachments lie on each side and just proximal to the insertion of the SDFT onto P2 at the pastern joint.

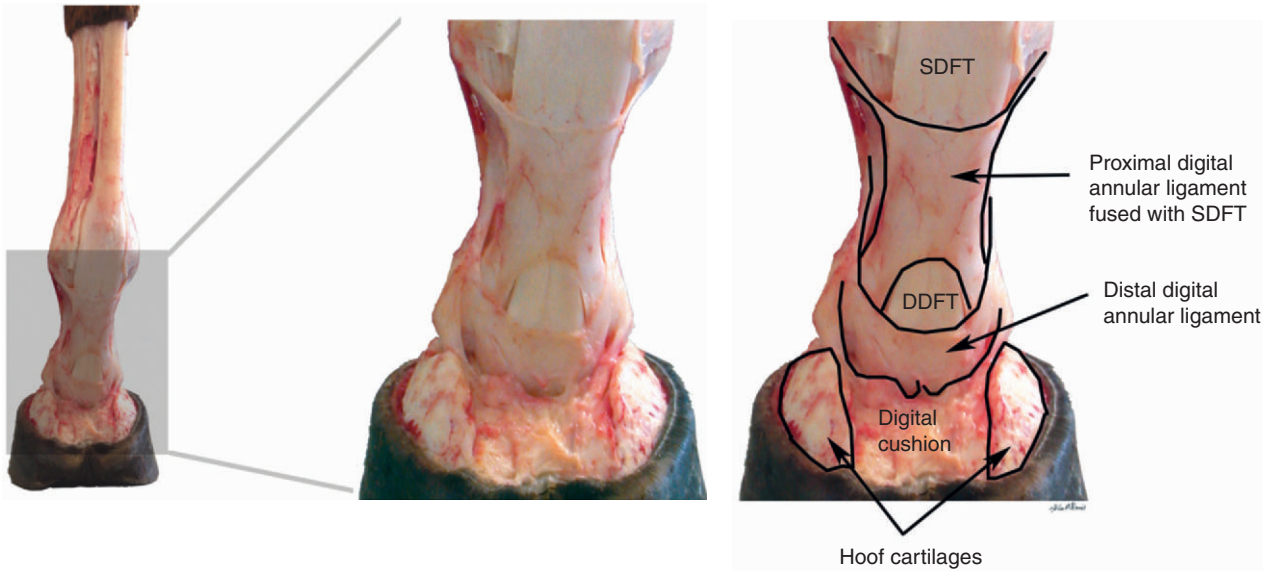

FIGURE 1-35 Annular ligaments. *DDFT,* Deep digital flexor tendon; *SDFT,* superficial digital flexor tendon.

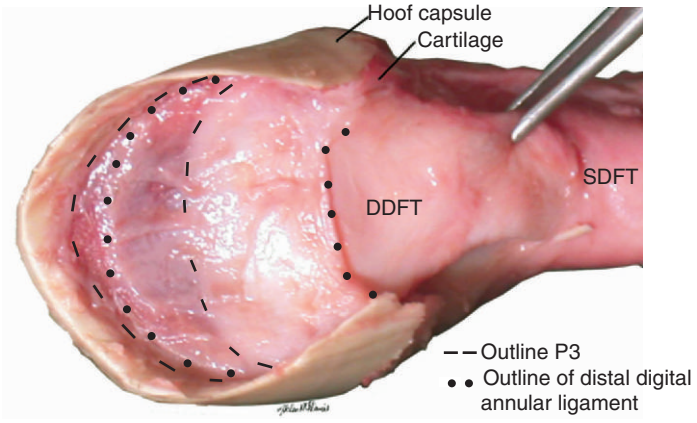

FIGURE 1-36 Fetal hoof with developing sole, frog, dermis, and digital cushion removed. *DDFT,* Deep digital flexor tendon; *SDFT,* superficial digital flexor tendon.

Distal Digital Annular Ligament

This ligament originates from the medial and lateral surfaces at the distal end of P1. It forms a sheet of fascia that envelops the distal portion of the palmar aspect of the digit. It lies immediately under the skin and fuses to the DDFT just before the tendon enters the hoof. The semicircular proximal border occasionally is palpable on each side of its midline fusion with the DDFT.

Within the foot, the distal digital annular ligament merges into the fibrous coverings of the hoof cartilages and digital cushion, as well as accompanying the DDFT down to its insertion onto P3. In the fetal digit (Figure 1-36), the distal portion of this annular ligament forms a distinct, tough sheet of collagen that separates the DDFT from the digital cushion and attaches across the entire underside of P3, from one side of the bone and cartilage to the other. In adult horses, this sheet generally is impossible to demonstrate as a discrete entity.

2

FUNCTIONAL ANATOMY OF THE EQUINE DIGIT: DETERMINING FUNCTION FROM STRUCTURE

HELEN M.S. DAVIES, CHRISTOPHER J. PHILIP, and

JONATHAN S. MERRITT

The function of the digit can be determined by several different methods. The approach used in this chapter is to consider what functions can be deduced from the form, composition, and relationships of the structures in the digit. This method involves dissections and observations; findings are then related to the basic mechanics of the component tissues, and hypotheses on the likely function of specific structures are developed.

Other approaches are discussed in the chapter on biomechanics of the hoof (Chapter 3). One is to measure the relationships between two parameters and deduce the likely functions from these relationships. This approach involves trial-and-error and observation or measurement, or both, of the effects of differences in measured parameters (e.g., the effect of differences in toe length on stride length). This approach is the one taken for much of the experimental work that has been done to date on hoof biomechanics.

Another method is to develop a set of hypotheses about the biomechanics of the system and use these hypotheses to build a theoretical model. Experiments are then designed to test that model; the model and its underlying hypotheses are adapted accordingly and then tested again. This approach is currently being employed in hoof biomechanical research.

This chapter discusses the function of each of the structures in the digit and then the function of the digit as an integrated unit. The reader is referred to Chapter 1 for detailed descriptions and illustrations of these structures.

SKIN AND HOOF CAPSULE

The structure of the skin suggests that its primary function is protection of the underlying structures. The skin and skin derivatives, including hair and the hoof capsule, protect the underlying tissues from damage by external organisms, water loss and other chemically induced changes, mechanical abrasion, and potentially harmful temperature changes. Because it is such a specialized structure and central to this discussion of the foot, the hoof capsule is discussed as a separate entity, even though it is essentially a skin derivative.

Skin

The skin consists of two layers. The inner layer, or dermis (corium within the hoof), supports the outer layer, or epidermis. The dermis may be strongly connected to the underlying tissues (e.g., the third phalanx [P3] in the case of the laminar dermis of the hoof wall) or quite loosely attached (e.g., the skin on the palmar aspect of the pastern, which slides easily over the flexor tendons).

Dermis

The dermis of the skin is a feltwork of collagen that is meshed with varying amounts of elastic fibers so that it forms a fibroelastic sheet that covers and contains the structures beneath it. This feltwork sleeve also encompasses blood vessels and nerves, as well as other structures such as hair follicles.

The skin is anchored to the underlying structures by a thin layer of fascia (subcutaneous tissue, or subcutis), which often contains fat. The fat provides some insulation and cushioning. Hair on the skin surface also acts as a variable (and seasonally adjustable) insulating and protecting layer. The hair coat reduces air movement across the skin surface; it is also water repellant by virtue of its covering of oils, which are secreted onto the hairs by specialized glands within the dermis.

Epidermis

The epidermis is a multilayered sheet of cells, the outermost layer of which is mostly composed of keratin (a structural protein). Hair and the horn of the hoof capsule also are composed of keratin; however, in the case of the hoof capsule, the keratin is specially arranged in rods and sheets (laminae).

The epidermis provides an insensitive barrier that can resist a certain amount of abrasion to protect the sensitive structures beneath from dehydration or invasion by foreign matter. To retain this protective function, the outer layers of skin and the hoof capsule have to be continuously replaced at no less than the same rate at which they are worn away. This activity requires a continuous supply of nutrients, delivered via the bloodstream, and is dependent on an intact and functioning dermis.

Hoof Capsule

The hoof capsule comprises the hoof wall, the sole, and the frog. These structures are continuous with one another, so together they form a coherent, resilient boot that encloses all of the living structures within the foot. In effect, the horse grows its own boots. The mechanical behavior of the hoof capsule depends on the mechanical properties of the materials

that make up the capsule, on its shape, and on its connections to the underlying structures. It also depends on the interaction between the hoof capsule and its immediate external environment and on the loads distributed through the bones into the hoof.

Material Properties

The materials that constitute the hoof capsule are somewhat elastic. The degree of elasticity is dependent both on the form of the material and on its state of hydration. The health and nutrition of the horse while the hoof capsule is growing is another important factor.

The frog is much more elastic than the hoof wall, and the hoof wall itself is stiffer toward the toe, where the material generally is thickest. These differences in material properties and thickness suggest that the hoof capsule is designed to flex and move in such a way that the frog is stretched and the heels expand while the toe region remains relatively static. Because the flexibility of the material is so dependent on its state of hydration, the hoof becomes much harder when the ground is hard and dry; conversely, it becomes much more elastic when the ground is soft and wet. Thus, the hoof capsule appears to function somewhat differently in direct response to the immediate external environment.

Shape and Connections

The complex shape and connections of the hoof capsule suggest that exactly how it deforms during movement depends on how loads are distributed through the column of bones into the hoof, which depends on how the horse is moving and loading the whole limb. A horse may choose to position the limb in such a way that the hoof lands or loads either to one side or the other, or more toward the toe or the heel. This variability in use of the hoof thus affects the way the hoof capsule distorts under load.

In general, the hoof capsule appears to be designed to be flexible and adaptable to a wide range of loading conditions. But regardless of the type or direction of load, the hoof capsule is relatively stable in the region of the toe and somewhat more moveable through the heels. The hoof wall component has been described as an incomplete and truncated cone. This shape suggests that any change in hoof wall shape that involves flattening of the toe would cause the heels to expand. Thus, the shape and the increase in wall thickness from heel to toe indicate that the toe is designed to be the most rigid part, whereas the heels are the most deformable part of the hoof wall. In other words, the hoof capsule is designed for stability when landing more toward the toe and for damping of impact forces when landing more toward the heels, where the material is more elastic and (in the normal foot) there is the thick pad of the frog on which to land.

The hoof capsule is strongly attached to the underlying dermis. In the case of the hoof wall and the bars of the heels, the connection comprises the 600 or so sensitive laminae of the dermis that interdigitate with the insensitive laminae of the hoof wall and bars. The most extensive laminar connections are found at the toe; the least extensive are those of the thinnest part of the bars, where they become continuous with the sole.

Deducing Its Function

The anatomy thus suggests that the connections of the hoof wall to the underlying dermis are most likely to be under the greatest tension in the region of the toe and under the least tension at the bars. It may simply be that, because of the shape of the hoof capsule, any tensile force on the hoof wall at the toe pulls directly against the relatively inflexible bone of P3. In contrast, tensile forces on one heel can be redirected and distributed through the underlying soft tissues (digital cushion, hoof cartilages) to the other heel, which means that the connection between hoof capsule and underlying dermis need not be so extensive in this area. In addition, the soft tissues in this area deform a great deal and hence tend to dampen the effects of any direct tensile forces on the hoof capsule connections at the heel.

Thus, although the connections of the hoof capsule might indicate that the greatest tension is on the toe region, the relationships of the parts and the overall shape of the hoof capsule suggest that movements are greatest at the heels and that the forces are redistributed from this region across the whole capsule, especially into the region of the toe. The toe, then, provides a stable point around which the whole hoof capsule tilts and twists and generally changes shape with different loading conditions.

Sole

The sole is generally concave in contour, and the wedge of the more elastic frog projects into the sole from behind so that any contraction of the frog material would tend to pull up on the center of the sole and make it more concave. This contraction of the frog might occur when a hoof starts to dry out because the frog material contains more water and can contract much more than the hoof wall or sole. It would be interesting to determine whether excessive concavity in the central part of the sole might cause excessive pressure on the overlying portion of the solar surface of P3. This sort of distortion might help to explain indeterminate foot soreness in horses that have recently changed to a drier environment.

Frog

The wedge shape of the frog is a useful shape to act as a brake if the hoof starts to slip on soft ground, especially in a forward direction. Tags of frog material that are partially torn off from the tip of the frog are commonly found in horses with untrimmed hooves, which supports the idea that this braking action is an important part of the frog's function.

With the cunean part of the digital cushion packing the center of the V, the frog is well designed to dig into soft ground or stick onto slightly slippery surfaces when the horse adjusts the angle of the hoof by contracting the deep digital flexor tendon (DDFT). The same movement would also tend to make the tip of the toe dig into the ground, so in a shod horse the sharp edge of the shoe probably serves instead of the frog to brake any forward slipping.

The frog's shape and structure are such that it may also play a role in stabilizing the hoof at the commencement of weight bearing. Along with the bars of the heels, the frog may seal the caudal edge of the concave sole so that any up-lift on the sole would cause it to act like a suction cup, sucking the hoof down onto the ground surface. Assuming that this suction cup effect does occur, this up-lift on the sole would result from tension in the DDFT. In the initial weight-bearing part of the stride, hyperextension (i.e., dorsiflexion) of the fetlock

causes the DDFT to pull against its semiconical attachment on the underside of P3, thus pulling up on the center of the sole via the attachment between the solar dermis and P3. However, although this effect makes theoretical sense, it remains to be proven.

The insertion of the DDFT includes fascial connections to the dermis of the bars of the heels and to the digital cushion, which suggests that increased tension in the DDFT is doing more than just flexing the coffin joint. Even if the DDFT does not exert this up-lifting effect on the sole, the concave shape of the sole, along with the thick wedge of elastic frog at its caudal boundary, indicate that the central part of the sole most likely is sucked down onto the ground as the heels rebound from their initial spread position after hoof impact and the body weight starts to load the hoof capsule. This action is the same as that required to affix a suction cup to a smooth surface and provides another possible explanation for the fact that horses living in regions where working surfaces are wet and muddy have flatter soles than horses in drier areas. This observation suggests that the sole might cause too great a suction force if it is too concave.

Apparently, it is quite normal for horses to occasionally shed a large part of the frog. Why this occurs is not clear, although it may be a response to a toxic insult or disturbance in the blood supply during formation that leads to a band of weakness through the frog tissue, which is then shed as a large piece, instead of the more normal slivers from the ground surface. There are no reported detrimental effects from such shedding, but if the hypothesis concerning the suction effect is true, then the presence of the frog may be important for hoof stability on certain surfaces and at the high speeds, when fetlock hyperextension (dorsiflexion) is extreme enough for the postulated suction effect to occur.

Dermis of the Hoof

The dermis, or corium, of the hoof is the blood-filled collagenous tissue that underlies the hoof capsule. It forms a thin pad that lines the entire inner surface of the hoof capsule—hoof wall, sole, and frog. The dermis connects the hoof capsule and the underlying structures in such a way that a small amount of "slip" is possible between these structures. This "bit of give" allows slight movement that may contribute to the absorption of concussion and may give the horse a little extra time to react with postural reflexes further up the limb before there is any chance of losing traction whenever the load through the limb is at an angle that would push the hoof capsule off balance. The dermis also acts as a cushioning pad for the bones so that their sensitive surfaces are not pinched directly against the hoof capsule, especially along the solar margin and over the sole.

Relationship to the Third Phalanx and the Hoof Cartilages

Bone surfaces that are not forming joints with adjacent bones (where they are covered by hyaline cartilage) normally are covered by a collagenous meshwork of fibers that forms a sheet (the periosteum). Unlike other bones, P3 is not covered by any clearly distinguishable collagenous sheet, except in a thin strip on the flexor surface, between the insertion of the DDFT and the attachment of the impar ligament. Instead, P3 and part of its cartilage extensions (the hoof cartilages) are covered directly by dermis.

The collagen network within the dermis that covers P3 and the hoof cartilages is continuous with the collagen that makes up the matrix of the bone and the cartilages. Thus, this dermis is strongly attached to P3 and the hoof cartilages. It is not possible to remove this dermis without cutting or tearing these collagen strands, many of which accompany blood vessels that penetrate the bone surface. These numerous blood vessels that penetrate P3 supply the laminar dermis with the nutrients needed to maintain a strong bond with the growing hoof wall.

Dermal Regions

The dermis of the hoof capsule supports the epidermis that grows hoof, sole, or frog material from its surface, the rate and direction of which depend on its position and perhaps on the way it is loaded. The dermis of the coronary band is a thickened band of dermis that encircles the top of P3 and continues around the hoof cartilages to form a ring of dermis from which the hoof wall grows down in tubules. The alignment of these tubules can be seen in the "grain" of the hoof wall and the bars.

As mentioned earlier, the dermis underlying the hoof wall and bars forms 600 or so thin, more-or-less vertical folds, or laminae, thus providing a large surface area for attachment to the insensitive laminae of the hoof wall. The remaining dermis of the hoof capsule consists of long papillae that likewise increase the surface area for attachment and hoof capsule growth in the sole, frog, and heel bulbs. The laminar attachment and growth of the hoof wall are discussed in Chapter 6, as is the histology (microstructure) of the hoof wall and dermis.

CONNECTIVE TISSUES: OVERVIEW

The connective tissue structures discussed in this section provide basic support and allow movements that enable the digit to perform its functions of body support and locomotion. These structures include the bones (P3, second phalanx [P2], first phalanx [P1], and navicular bone); the cartilaginous structures (hoof cartilages, middle and distal scutum, and articular cartilage); the tendons (DDFT, superficial digital flexor tendon, and common/long digital extensor tendon); the ligaments (collateral ligaments of the pastern and coffin joints, navicular ligaments, straight and oblique sesamoidean ligaments, axial and abaxial palmar ligaments of the pastern joint, and the pastern branch of the DDFT); and the joint sheaths, fascia, annular ligaments, and digital cushion.

In general, the connective tissues are composed primarily of collagen, which is a fibrous material that resists tension quite well. Basically, it acts like rope when it is formed into bundles, like cloth when it is formed into a meshwork of fibers, and like composite materials such as fiberglass when it is packed with crystals of hydroxyapatite (as in bone) and plastic when it is packed with glycoproteins (as in cartilage).

Collagenous structures appear white and are variously tinted yellow when they contain the protein elastin. Elastin fibers are about 300 times as elastic as collagen fibers, so when elastin is present, it increases the amount of initial "give" in a bundle of aligned collagen if the collagen has a coarse "crimp."

Collagen alone stretches to varying degrees, depending on the amount of "crimp" in the fibers, the orientation and length of the component fibers, their degree of cross-linking, their attachments to other structures, and their association with other materials such as the hydroxyapatite crystals in bone or the glycoproteins in cartilage.

BONES

When a collagen matrix is packed with hydroxyapatite crystals to make bone, it creates a material that can retain its form under considerable load, although it tends to crack quite easily when twisted or bent or put under a tensile load. Together, the bones of the limb form a solid supporting strut for the body and provide leverage during locomotion.

The individual elements that comprise the bony column of the digit (P1, P2, and P3) move freely against one another at their joint surfaces, yet they are tightly bound together and held in such a way that the compressive forces involved in body support and in deceleration of the limb at hoof impact are distributed through the bone column without loading any part beyond its strength. Thus, by the time any loads reach the cannon bone (third metacarpal [MC3] or metatarsal [MT3] bone), the effects of any tilts or twists occurring at the level of the ground surface of the hoof capsule have been removed, and the horse is able to bear weight on the limb without risking catastrophic fracture of the cannon bone.

Attachment of Soft Tissues to Bone

In regions where there are tensioning forces on the bones, the collagen matrix of the bone is continuous with the collagen of the tendons and ligaments that attach to it, or, in the case of P3, to the collagenous framework of the dermis that holds the hoof capsule in place. (The distinctly roughened regions on the bone surface when a bone is boiled or otherwise cleaned of collagenous tissues reflects these attachment points. Elsewhere, the bones in the digit are covered with a sheet of collagen [the periosteum] that is only lightly attached to the underlying bone and can be peeled off quite readily to leave a smooth surface.)

If the collagenous structures' attachments to the bone are aligned parallel to the bone surface, then traction on the collagenous structure is translated into compression of the bone. This arrangement optimizes the bone's ability to resist those loads and the ability of the ligament or tendon to apply those loads while minimizing the risk of damage to either structure. The majority of ligaments are attached to bones in this manner.

There are some specific instances in which the main purpose of a particular ligament appears to be in strengthening the bone to resist tensioning loads rather than holding different structures together. The oblique sesamoidean ligaments are one such example. The long and angled insertions of these ligaments strengthen the palmar surface of P1 and assist it in resisting the tensioning loads experienced during fetlock hyperextension (dorsiflexion). The middle part of the suspensory ligament of the navicular bone is another example. It strengthens the proximal edge of the navicular bone and assists it in resisting the tensioning loads that can be experienced when the heels spread during loading of the foot.

Bone Development and Form

All of the bones in the equine digit begin as hyaline cartilage models in the developing fetus. The cartilage is replaced by bone before birth so that these bones are strong enough to resist the forces involved in standing and moving immediately after birth. During subsequent growth, the bones develop into a shape that is basically dictated by genetics. However, the form adapts throughout life in response to the dynamic forces the individual bone experiences, to create a structure that resists these forces in the optimal way for that individual horse.

Thus, there is always an interrelationship between how the limb is used and the exact shape of the individual bones in a particular horse. The shape, size, and relationships of a bone may restrict the range of potential movements, but the habits of locomotion and posture will dictate how the bones develop and grow or remodel throughout the horse's life.

Compact Bone

Bone material consists of compact (lamellar or haversian) bone and trabecular (cancellous or spongy) bone. The compact bone is creamy white, dense, and hard; it is formed by sheets or rods of a crystalline solid that is deposited onto a collagen matrix. Compact bone provides the most effective support against compression for a given volume of bone, but it is about twice the weight of a similar volume of water or collagen. Thus compact bone is found only where loads are sufficiently great to stimulate its formation and preservation.

Trabecular Bone

Where compressive loads are less predictable or vary across a wider range of directions (as occurs near joint surfaces), trabecular bone forms. Trabecular bone is composed of the same material as compact bone, but it is laid down in thin spicules that align themselves like the support struts of a bridge to distribute compressive forces across joint surfaces and direct these forces into the areas of compact bone (e.g., along the bone shafts).

The position, shape, and distribution of compact bone and the orientation of the spicules of trabecular bone reveal how forces are normally distributed through each individual bone. The spaces between the trabeculae are filled with tissue fluid and soft tissues such as fat and bone marrow, so trabecular bone weighs less for a given volume than compact bone, especially when the spaces are filled with fat.

Individual Phalanges

The long pastern bone (P1) is so named because of its elongated shape, which allows it to act like a supporting rod. The more cuboidal short pastern bone (P2) clearly is more able to resist a wider range of movements, such as sideways twisting and bending. In contrast, the coffin bone (P3) has a much more complex shape. It looks on first examination to be highly prone to many ways of breaking, especially at its margins. However, in the living animal P3 is tightly woven into the collagenous meshwork of dermis that holds the hoof capsule in place; thus, P3 is likely to be significantly reinforced by the collagen of the dermis.

The coffin bone potentially is further strengthened by the hydrostatic pressure of the complex network of blood vessels that permeate it. These blood vessels are large and numerous, unlike in any other bone, and they give P3 the appearance of a dense sponge when it is cleaned of overlying soft tissues. Its highly perforated structure also makes P3 lighter for its size than the other bones in the limb.

Deducing Their Function

The shapes of the individual phalanges suggest that the column of body support might be expected to commence in the region of P2, which allows a certain amount of flexibility at the base of the strut formed with P1. It is probable that P3 is mainly a support and stabilizer for the hoof capsule and is involved only indirectly in body support through this action. This perspective on its function might help to explain why the solar margin of P3 is normally held at a slight angle to the ground in a standing horse.

Shape of the Articular Surfaces

The form of the articular surfaces of the three phalanges and the distal end of the cannon bone indicates how the major forces are directed through these joints. In each case, there is a concave surface and a facing convex surface. There is also a median ridge in the articular surface on one side of each joint and a matching groove on the facing surface. These median ridges and grooves appear to be placed to increase the stability of the joints and assist in restricting potential movements to the sagittal plane (i.e., flexion and extension).

In each case, the concave surfaces are located on the lower (distal) side of the joint, so they might be thought of as cupping or helping to retain the convex surfaces on the distal ends of the cannon bone, P1, and P2. The median ridges and grooves are not so consistently placed. Nevertheless, the ridges may serve to concentrate and direct forces coming through the bone into the joint in such a way that they are directed toward the center of the facing bone and hence directed centrally through the column of bone. The central ridge in the lower end of the cannon bone may be concentrating forces associated with weight bearing down into the digit, whereas the central ridge in the articular surface of P3 and the upper articular surface of P2 might be directing forces associated with ground impact up into the limb.

Deducing Their Function

The anatomy of this region suggests that the problem of directing ground reaction forces predominates in the area below the middle of P1, and the problem of directing forces associated with the horse's weight predominates in the fetlock joint and above. The forces associated with deceleration of the limb at initial ground impact would be mostly redirected up into the limb by dorsiflexion of the fetlock following impact, whereas the central ridge in the distal end of the cannon bone directs the force associated with support of body weight centrally down into the digit.

Ground reaction forces are directed up into P1 through the shallow central articular ridges/grooves of P3 and P2 and via the ligamentous connections of the navicular bone to P1, so it would appear that P1 absorbs any residual twisting or bending from the hoof only after it has been already dampened by the more distal bones and joints. The majority of twisting and bending forces on the pastern joint would be occurring through the palmar aspect; the middle scutum (see Chapter 1) is well placed to dampen much of these residual movements and allow the slight "give" that prevents catastrophic fracture of the phalanges.

The number and orientation of the other ligamentous connections around the pastern joint further suggest that motion through this joint is complex and is designed to prevent movements associated with tilts or twists originating in the coffin joint from adversely affecting structures higher in the limb. In clinical practice, it is P1 that sometimes catastrophically fractures (disintegrates) in horses that are asked to make sudden turns and changes of direction. It may be that these fractures occur when the pastern joint is not able to sufficiently reduce the twisting or bending movement from the coffin joint before it reaches P1.

Navicular Bone

The navicular bone is considered to be the distal palmar sesamoid bone, but it acts more like a palmar extension of the articular surface of P3 at the coffin joint. Its connections to the DDFT are minor in comparison to its attachment to P3 via the impar ligament (distal sesamoidean impar ligament). Its other strong connections are at the medial and lateral ends of the bone, where it attaches into P2 and the collateral ligaments of the coffin joint, connecting strongly to the hoof cartilages on the way past.

Regions of Greatest Load

Normally, there is a distinct layer of compact bone along the edge of the navicular bone that abuts P3, which suggests that compressive forces are experienced across this joint in that direction, possibly during deceleration of the limb following impact. It would be interesting to note whether this layer is thicker in horses that work on sticky surfaces than in horses that work on more resilient surfaces.

The other region of compact bone that is found in a normal navicular bone is a narrow shelf on the proximal surface that forms a continuation of the coffin joint surface. This region of the navicular bone seems to be adapted to take some of the compressive forces that occur during weight bearing, as a direct continuation of the joint surface of P3.

Suspensory Apparatus of the Navicular Bone

Where the navicular bone might be regularly exposed to greater tensioning forces than bone material can withstand—along its outer (caudal/palmar) edge—it is reinforced by a thick band of collagen. This ligamentous material completes a "sling" for the navicular bone by continuing as the suspensory ligament of the navicular bone on each side, which becomes intimately associated with the collateral ligaments of the pastern joint.

In this way, the navicular bone is suspended in a ligamentous cradle behind the articular surface of P3, to which it is strongly and broadly attached by the impar ligament. The navicular bone might therefore be considered to be a slightly moveable caudopalmar extension of the articular surface of P3. In this position, it is ideally suited to assist with joint stability while increasing the range of possible movements and providing more "give" during impact and stance than solid bone could absorb without risking fracture.

The suspensory ligament of the navicular bone would protect the navicular bone from being pulled apart when the heel lands and the heels are forced apart, as well as allowing a sideways slip when the landing has been skewed to one side or the other. Thus, movement of the navicular bone would dampen the effects of tilted positions of the hoof capsule or slight slippages and help maintain the direction of load up into the column of bones, starting with P2.

This system for absorbing tilts or twists in the coffin joint must be effective, because fractures of the navicular bone are not common in clinical practice, despite the bone's elongated shape. The most commonly recognized pathologic condition involving the navicular bone is erosive lesions on the flexor surface.

Pain Associated with the Navicular Structures

When a horse has a painful condition involving the navicular structures, any movements of the navicular bone within its sling are likely to increase the pain. The only way that a horse can directly reduce movement of the navicular bone is by contracting the muscle bellies associated with the DDFT just before impact or during stance. That action would press the flexor tendon against the navicular bone, which in turn is pressed against P2.

The horse has no other muscle that it can use to directly reduce movements of the navicular bone within the hoof capsule. It seems likely, however, that adapting the swing of the whole limb so that the hoof lands closer to the toe than to the heel would also reduce potential movements in the navicular region (because of the shape of the hoof capsule, as discussed earlier).

Where the DDFT runs over the flexor surface of the navicular bone, the bone is covered by fibrocartilage, which means that it is tougher and more able to distort without damage than the hyaline cartilage of the coffin joint surface. However, it also means that excessive, continuous, or otherwise abnormal compressive forces are likely to create roughened areas on this surface and hence add to the discomfort experienced by the horse. A vicious cycle can then be created, in which discomfort leads to increased tension in the DDFT and thus increased damage to the flexor surface of the navicular bone, which causes further discomfort.

CARTILAGENOUS STRUCTURES

When the collagen matrix is packed with glycoproteins, it forms cartilage, which has more of the properties of plastic than of rope or cloth. In general, cartilage is much more resilient than bone and tends to rebound to its original shape very easily. The extra springiness of cartilage means that it is useful for lining joint surfaces.

Cartilage cannot support the amount of load that bone can withstand, but it is much more resilient (as well as being about half the weight of bone), so cartilage is found in regions where the maintenance of shape is important but compressive forces are minimal. Examples include the cartilage rings that hold the trachea (windpipe) open, and the sheet of cartilage that underlies the skin of the external ear. Unlike bone, cartilage is insensitive, so it is especially useful in regions such as joint surfaces where the impact of normal movement might otherwise cause pain.

The particular type of cartilage that lines joint surfaces (hyaline cartilage) contains much shorter strands of collagen and has a much smoother surface than the tougher fibrocartilage that lines the flexor surface of the navicular bone and is found in the middle and distal scutum (see Chapter 1), where the higher density of fibrous collagen gives fibrocartilage its name. The hoof cartilages also contain a type of hyaline cartilage.

Hoof Cartilages

The hoof cartilages can be considered to be cartilagenous extensions of P3. They are composed of hyaline cartilage (just like the cartilaginous precursor of the coffin bone itself), although they may become a lot more fibrous over time in some horses. Cartilage is about half the weight of compact bone, so these cartilagenous extensions of P3 apparently provide some sort of energetic advantage during the stride, as it takes less energy to move a lighter digit than a heavier one. In addition, cartilage requires much less metabolic energy than bone to maintain. The primary function of the hoof cartilages appears to be to provide a flexible support for the more flexible portions of the hoof capsule.

Ossification of the Hoof Cartilages

In many horses, the hoof cartilages ossify (become bone) to varying degrees during life without causing any apparent problems, although their ossification does increase the weight of the digit, and ossified cartilages may be prone to fracture (which would cause pain). Ossification of the hoof cartilages would also reduce the flexibility in the overlying hoof capsule. This variable degree of ossification of the hoof cartilages means that the shape of P3 as seen on radiographs or in preserved specimens can be highly variable. (Cartilage does not show clearly on radiographs and generally is lost during the preparation of bone specimens.)

There cannot be too great a requirement for resilience in these cartilages, because there is often little apparent effect of some quite extreme levels of ossification of the hoof cartilages on the horse's soundness. However, by increasing the weight of the digit, this ossification must reduce the efficiency of movement, as well as increasing the potential for problems associated with increased inertial forces in the distal end of the limb pendulum.

Connections to Other Structures

Thin but distinct ligamentous bands connect the hoof cartilages to P2 across the coffin joint, but these connections are relatively minor. Most significant is the fact that the hoof cartilages are surrounded by a thick, fibrous connective tissue sheath that is continuous with the dermis covering P3 and the collateral ligaments of the coffin joint.

Interspersed with the connective tissue are large veins that penetrate the cartilage and form a large venous plexus associated with the hoof cartilage. These veins are tethered via more connective tissue along their length, which makes it difficult to separate individual ligaments (between the cartilages and other structures in this region) from the blood vessels.

Furthermore, on the inner (axial) surface of the cartilages, the collagenous bands are continuous with the collagen of the DDFT so that the arc of the tendon's attachment onto P3 extends around the inside of, and even into, the hoof carti-

lages. The connective tissue sheath also extends across to the hoof cartilage on the other side of the foot, beneath the DDFT, to form a sheet of collagen that separates the DDFT from the digital cushion at the back of the hoof. This semilunar sheet of collagen extends forward to attach to P3 in intimate association with the DDFT throughout the rim of the semilunar line on the flexor surface of P3. The free palmar edge of this collagen sheet is visible in some specimens, but it is often indistinct because of its attachment to the distal digital annular ligament (see Chapter 1).

Deducing Their Function

These various attachments suggest that when the hoof lands and the cartilages move apart as the heels spread, the cartilages are distorted in a complex way by their collagenous attachments. Exactly how they distort depends on how the hoof capsule is placed and moved, how the bones are loaded by the body weight, and the timing of any pull on the DDFT.

It could be that spreading of the heels and outward movements of the hoof cartilages assist in the circulation of blood through the digit by sucking blood into the region between the cartilages as they spread apart. The pull on the DDFT as the fetlock sinks into hyperextension may also assist in reducing the pressure in this region and help to hold the hoof down onto the ground during weight bearing (see p. 33), as well as perhaps sucking blood into this region to equalize the pressures. Exactly how these mechanisms function is not yet clear.

Articular Cartilage

Where bone ends meet to form a joint, they are covered by hyaline cartilage. This cartilage appears bluish white and glistening in a normal healthy joint. Its primary function is to provide some cushioning and minimize friction between the bone ends. Normal, healthy cartilage has a smooth surface and is made even more slippery by the synovial fluid (joint oil) that fills the space between the bone ends. In a healthy joint, the synovial fluid is clear, yellowish, slightly sticky, and oily.

Characteristics of Hyaline Cartilage

Hyaline cartilage consists of a collagen matrix that is packed with glycoproteins, so it can hold a large volume of water. The water content makes the material resilient and springy when the cartilage is loaded because the water cushions the impact and then diffuses away from regions of high pressure at a relatively slow rate. Hyaline cartilage is not designed to withstand sustained high pressures; such conditions cause loss of water and result in the cartilage becoming stiffer and more brittle.

The joint needs to move to restore the water content of the cartilage, as the water has to diffuse back into the cartilage from the synovial fluid or from the blood supply in the underlying bone. (Articular cartilage is one of the few tissues in the body that has no blood vessels or sensory nerves within it.) Thus, to remain effective and retain or regain its resilience, the joint needs to be moved, especially after it has been held under a static load for more than a few minutes.

Coffin and Pastern Joints

In general, articular cartilage is thicker in joints that move a great deal. In most specimens, the coffin joint has a thicker layer of cartilage than the pastern joint, which supports the idea that the coffin joint moves more than the pastern joint in a normal horse.

Fibrocartilage Pads (Middle and Distal Scutum)

The middle scutum is the region on the proximopalmar edge of P2 where the insertion of the straight sesamoidean ligament and the superficial digital flexor tendon (SDFT) is strengthened and thickened by fibrocartilage. Immediately below the middle scutum (when the digit is at a normal angle to the ground) is the distal scutum, a fibrocartilagenous thickening of the DDFT. (These fibrocartilagenous pads are described and illustrated in more detail in Chapter 1.)

Deducing Their Function

These pads appear to be designed to sit one on top of the other so that there is a continuous sequence of relatively elastic structures running upward from the frog to P2—frog, digital cushion, distal scutum, middle scutum—that can cushion the pastern joint and, specifically, the distal end of P1. This arrangement suggests that the system is designed to allow the ground reaction forces to be concentrated through into P1 without necessarily being directed through P3 or P2 first. At very fast speeds, it is known that horses tend to land more toward the back of the hoof; perhaps it is then that this sequence of structures becomes important. The location and sequence of fibrocartilagenous pads suggests that forces can be concentrated through into P1 with minimal risk to the bone, as the fibrocartilage would be able to absorb a considerable proportion of any twisting forces that were occurring.

This sequence of pads is not a system that would have a high degree of inherent stability or predictability, however. That might help to explain why the pastern joint is so strongly bound by additional ligaments (see Chapter 1). A joint that only moves a small amount and is bound by enough different ligaments for movements in any direction to be quickly discernable by the nervous system may solve the problem of the indeterminate amount and direction of "give" in the insensitive fibrocartilagenous pads during landing.

Presumably, the horse would slow down and place the hoof more centrally if it were moving over unstable or uneven ground, or it started to turn. In these situations, the twist- and tilt-damping mechanisms of the navicular bone in its sling (see p. 38) and the basic shape of the hoof capsule (see p. 37) might be more important in reducing the risk to P1 of these torsional forces.

TENDONS AND LIGAMENTS

When bundled into straps or cords, collagen forms the ligaments that tie different structures together and the tendons that tie muscles to bones. When formed into sheets, it makes the annular ligaments and fascial sheaths that hold the tendons and ligaments down against the bone surfaces and act like support bandages around the joints. The orientation of the collagen strands and the points and areas of attachment show where, and in what direction, the tensile loads occur. The size of the collagen bundles suggests how much load the particular structure is subjected to during normal movements.

When collagen forms a mesh, it becomes a composite material somewhat like fiberglass, and the material properties depend on what other materials are packed around the collagen. When a collagen matrix is packed with fat, it may form a springy cushion (as in the digital cushion) or a stretchy buffer that can retain its integrity while allowing for the movements of underlying structures (as in the subcutis of the skin).

The various tendons, ligaments, and fascial sheets contain differing amounts of elastin fibers, which, in addition to providing elasticity, make the structures appear more yellow. (This coloration has to be differentiated from color changes due to fat deposits, as yellow carotenoid pigments are present in fat.) Careful observation of the color of the fibrous bundles and sheets gives some insight into how elastic the structure is; but because of the large variability in cross-linkage and fiber alignment among different structures, it is important to investigate normal ranges of movement by manipulating the specimen and cutting or removing various structures in different sequences to see what effects that may have on the range and direction of movements.

Tendons

Flexion and extension of the joints in the digit are enacted by the flexor and extensor tendons that attach to P3. These are long tendons that extend from muscle bellies above the knee (carpus) or hock (tarsus). There are no muscle bellies below the horse's carpus or hock, so all of the movements of the distal limb are controlled by muscles higher in the limbs and by the posture and movements of the head, neck, and body.

Direct locomotive forces and individual movements of the forelimb are mostly created by the muscles of the shoulder and the extrinsic muscles of the limb (i.e., those that attach the forelimb to the body). In the hindlimb, the muscles of the thigh and rump are the drivers and controllers of the distal limb. The effects of these muscle contractions are continued into the limb through reciprocal movements of the limb joints, because the bones are tied together with ligaments and fascia in such a way that flexing of the shoulder or stifle joints causes folding of the entire limb, and extension of these joints causes the whole limb to extend.

Control of Foot Placement and Loading

Elastic ligamentous connections between the limbs and the body, and the muscles in the upper limb, create chains of connections that mean that any movements of any part of the horse can be reflected in changes in the tensioning of the ligaments and tendons in the foot. This interrelationship should not be surprising, as the horse has to balance precariously on long, slender limbs that contain several long bones that cannot be loaded to any significant degree of torsion or bending without breaking.

Thus, the hoof is designed to be controlled through movements of the main mass of the body and not through any consciously controlled local adjustments within the digit. It has to be able to land on uneven and slippery surfaces and "self-level" quickly enough to allow the horse to gallop and jump without falling.

Tendons differ from ligaments in that they are continuous with muscle tissue; when the muscle contracts, it pulls on the tendon and hence on the bones to which the tendon is connected. The tendons in the digit connect muscles further up the limb with the bones of the digit. These tendons are very long, and the associated muscle bellies are relatively small. In addition, the tendons have connections to other bones in the limb (proximal to the digit), so the effect of their muscles is to control the tension in the tendon and brace the limb against impact rather than to provide direct locomotory forces to the limb. Thus, all three tendons in the digit are functioning mainly as ligaments. The flexor tendons also function as major elastic energy stores to reduce the energy cost of locomotion (see p. 36).

Common/Long Digital Extensor Tendon

The common (forelimb) or long (hindlimb) digital extensor tendon runs down the dorsal (front) surface of the digit. It attaches to P2 and the joint sheath of the pastern joint on the way past and inserts into the extensor process of P3 at the coffin joint. At this final attachment site, the extensor tendon also is intimately related to the dorsal (foremost) edge of the hoof cartilages and to the medial and lateral collateral ligaments of the coffin joint. Its position, structure, and relationships mean that it acts to slightly lift the toe during limb extension, thus reducing the likelihood of the toe catching as the limb is drawn forward.

Effect of Severing the Tendon. This action is clearly apparent when the extensor tendon in a forelimb is severed; the horse must learn to flick the digit forward into extension indirectly, using extensor carpi radialis (which extends the carpus), to avoid catching the toe during the stride. Some horses appear to find this action difficult to learn, especially when they have a long-toe conformation, and they can remain prone to tripping for some time after the original injury. These horses tend to be the ones that contract the extrinsic musculature of the limb to avoid catching the toe, rather than adapting the timing of the intrinsic muscle contractions.

A severed extensor tendon in a hindlimb appears to be easier for the horse to accommodate, possibly because there is no requirement to contract body muscles to avoid losing balance when catching the toe in the hindlimb. Also, there are several potential ways in which the horse can increase joint flexion in the upper hind limb sufficiently to lift the toe clear off the ground.

Damage to a hindlimb extensor tendon rarely appears to affect performance in the long term. However, the attachments of the digital extensor tendon to the joint capsules of the fetlock and pastern joints, and to the collateral ligaments of the coffin joint, mean that any movements of the tendon are going to affect the whole digit. The digital extensor tendon appears to act as the front part of a circumferential fascial sheath that forms a strong elastic sleeve around the digit, together with the digital annular ligaments on the palmar side. This sheath, and its component parts (including the extensor tendon), appears to function mainly as part of the digital support bandage. Thus, any damage to it will increase the likelihood of damage to other structures in the digit.

Superficial Digital Flexor Tendon

The superficial digital flexor tendon (SDFT) runs superficially down the palmar/plantar (back) surface of the limb to the proximal portion of P1, where it divides and the two branches run either side of the DDFT. The SDFT is understood to be a major elastic energy store, but clearly it also acts

as a ligament to assist in stabilizing the limb. It tends to be flatter and partially surrounds the DDFT for much of its length, so it is well positioned to strengthen and protect the underlying DDFT.

Contraction of the superficial digital flexor muscle has the potential to flex the pastern joint, but because it inserts at an angle that is close to parallel with the long axis of the pastern bones, it mostly acts to stabilize the pastern joint and resist pastern flexion when the joint is straight. The SDFT attaches partly to the distal part of P1 but mainly to the proximal part of P2, crossing the pastern joint in the process.

The attachment of the SDFT to P1 is via its connection to the proximal digital annular ligament, which covers the SDFT in this region (see Chapter 1). Generally, it is not possible to separate the fibers of these two structures with any degree of certainty. In addition, some of the collagen fibers of the SDFT appear to continue alongside the distal digital annular ligament down at least as far as the digital cushion. Presumably, these connections assist in damping, spreading, and redirecting any tensile forces to reduce the risk of any of the structures breaking during movement. Tears in the SDFT do occur in the digit, but they are nowhere near as common as SDFT tears above the fetlock.

Deep Digital Flexor Tendon

The DDFT lies deep to the SDFT until the middle of the pastern, where it emerges from between the two branches of the SDFT to lie immediately under the skin for a short distance before diving deep into the hoof capsule, toward its insertion into P3 and the hoof cartilages.

Along most of its length the DDFT is approximately circular in cross section, and it is much larger than would be required to transmit any muscular forces and move P3. Thus, its function must be to assist in withstanding the large forces that occur during movement. Its anatomy suggests that a major subsidiary function is storage of elastic energy (see p. 36).

Check Ligament. The presence of the accessory (the distal or inferior check) ligament would increase the effective storage of elastic energy in the DDFT. It would also protect the muscle-tendon junction from overstretching by redirecting tensioning forces in the portion of the DDFT distal to the carpus into compression of the palmar surface of the cannon bone. The accessory ligament is especially large in the forelimb; it is smaller, and may even be absent, in the hindlimb, which suggests that withstanding tensioning forces and providing for elastic energy storage in the DDFT is especially important in the forelimb.

Effect on the Third Phalanx. The potential effect of contraction in the deep digital flexor unit (muscle bellies and tendon) is to flex the coffin joint. It may also be used to pull the toe of the hoof down into the ground and help the horse to grip on slippery surfaces, but it is likely that the main action of the muscle bellies is to give the horse the possibility to control movements of structures within the hoof and especially to stabilize the navicular bone at particular points in the stride (see p. 38). The deep digital flexor unit is the only structure that the horse has retained that can directly change the movement of structures within the hoof.

Distal Scutum. The distal scutum is essentially a thickening within the DDFT just before the tendon passes over the navicular bone. At the lower edge of the distal scutum, there is a sudden change in the tendon's consistency, which might be expected to make the tendon very prone to damage at this point. The fact that damage in this region is not commonly described may be because of its hard-to-access position above the digital cushion, but more likely it is because this area is not commonly damaged.

Evidently, this portion of the DDFT is protected from sudden tensions during fast exercise, possibly because the distal scutum is held firmly between the middle scutum and the digital cushion during initial hoof impact. If that is so, then it further suggests that movements of P3 beyond this point are not sufficient to impact this portion of the tendon either.

Any movement of P3 would necessarily pull on the impar ligament and hence on the navicular bone itself. The attachment between the navicular bone and the DDFT is around the lower edge of the distal scutum, via a thin sheet of collagen that is lined with synovial membrane, so clearly it is not designed to take any significant amount of tension. This observation suggests that large movements between the distal scutum and P3 are not normal, so there must be other mechanisms stabilizing P3 (and probably the hoof capsule as well) when the horse lands on the heels during fast exercise. An alternate explanation is that P3 and the hoof capsule are not loaded to any significant degree under these specific conditions, so tensile forces do not reach a level at which they could cause damage. Or, this thin part of the coffin joint capsule may simply be very elastic.

Insertion into the Third Phalanx. The insertion of the DDFT into P3 may be similarly protected from tearing and other damage by the presence of the distal scutum and its position during hoof impact. Dissections of the DDFT down to its insertion onto P3 and the hoof cartilages are tedious and difficult, so are rarely done in routine postmortem examinations. Thus, it is not possible to be certain whether damage in this region of the DDFT is common or a significant cause of lameness. With the continuing advancement in diagnostic imaging techniques, it will become possible to investigate the insertions of the DDFT for damage and hopefully answer this question.

Overall Function. Overall, it seems likely that the DDFT is primarily an elastic energy store, with secondary roles in flexion of the coffin joint, stabilization of the navicular bone, and attenuation of ground impact forces during fast exercise via its fibroelastic pad (the distal scutum). Another important function of the distal scutum is to protect the more distal parts of the DDFT from damage when the tendon is storing elastic energy in its more proximal regions.

Ligaments

Ligaments tie bones together in such a way that any stretching of the sheets or bands of ligament in one part tends to be distributed throughout the column of bones, compressing the bones between sheets of tightening ligament and avoiding concentration of tension at any particular point. Thus, ligaments act a little like a support bandage for the bones.

These collagen sheets and bands provide so much structural stability when loaded that they allow the bony column to act like a rigid strut, and yet they allow sufficient movement across the joints for the limb to fold right up. The sequences of connections between the bones and the ligaments limit most of the movements of the limb to either flexion (where the limb is

folded up and the ligaments are loose) and extension (where the limb is in a standing position and the ligaments are taut).

Forelimbs versus Hindlimbs

This observation is especially true for the forelimbs, and it means that the limb can act like a strut to support the body weight when it is under load and yet can be folded up and swung forward in each stride with minimum effort. In the hindlimbs, there is not the same requirement for body support during fast movement; instead, the hindlimbs are used to propel the body forward, so they need to be strongest when the load is being applied by the muscles high in the limb.

The ligament and tendon connections through the hindlimb cause any contraction of the muscles high up in the limb to have the potential to directly affect the ligaments in the digit by tightening them while the limb is extending. This tightening effect probably is highly dependent on the exact posture and timing of muscle contractions. However, it is clear that a skilled rider can influence the muscle chains in such a way that the horse can be induced to allow more or less elastic spring, which shows as cadence in the stride. Thus, in the hindlimb the bones in the digit are held together most tightly when the power is being applied, whereas in the forelimb they appear to be designed to be tightest in midstance, when the body weight is being supported by the limb.

Joint Capsules

Each joint is surrounded by a thick, fibrous (collagenous) capsule that encloses the space between the component bones. This joint capsule is lined on the inside by a synovial membrane, which secretes synovial fluid into the joint space, thus lubricating the cartilage surfaces of the bones. The joint capsule has various recesses, or pouches, that assist in allowing the full range of motion normal to that joint and which may be associated with dissipating concussive forces that might otherwise tear the joint capsule. These joint pouches can be useful in accessing the joint space or joint fluid but can also provide routes for infection to track into the joint space.

Collateral Ligaments

The bones are connected across each side of the joint (medial and lateral) by strong bands of collagen that form ligaments over the joint capsule. These collateral ligaments tie the bones together, restricting and directing the range of movement that occurs through the joint. They act to restrict medial and lateral bending (adduction and abduction) and rotational movements so that the joints are mainly limited to flexion and extension within a sagittal plane.

Both the pastern and the coffin joints have clear collateral ligaments that lie at an angle to the long axis of the pastern, being more perpendicular to the ground when the limb is standing normally. This orientation suggests that the main tensile force on these collateral ligaments is the direct effect of the interaction between the ground reaction force and body weight, because it acts in parallel with the effect of gravity.

Other Ligaments

Sequential cutting of the ligaments in the digit shows that the majority of the movement restriction at the pastern joint comes from the attachment of the straight sesamoidean ligament to P2 and from the tight bands of the axial and abaxial palmar ligaments that lie on each side of the attachment of the straight sesamoidean ligament. The other major ligaments in the digit are those associated with the navicular bone; their function is described with the function of the navicular bone (see p. 38).

FASCIAL SHEETS AND ANNULAR LIGAMENTS

The various sheets of fascia and the annular ligaments in the digit are described and illustrated in Chapter 1. These fascial structures do not have to be bulky to have a significant effect on distribution of load in the digit. It takes only a small force at an angle to the major forces to restrict the range of movement of the primary structures so that they can be optimally loaded. However, if a misstep does exceed the tolerances of this system to maintain the loading directions within a small range, then breakdown of a major structure is likely to occur, typically in an area that is distant from the region of influence of the fascial sheets. This breakdown occurs because the fascial sheets generally act to redistribute loads from any direction, so they tend to protect all of the structures within their range of influence.

DIGITAL CUSHION

The wedge-shaped digital cushion is a complex network of fat-filled, collagen-bounded spaces that create a tough yet elastic cushion that underlies the bones in the region of the pastern joint. It fills the whole space above (proximal to) the frog, behind (palmar to) the DDFT and navicular bone, and medial to the hoof cartilages. Along its abaxial (sides) and cranial boundaries, the digital cushion is continuous with the solar dermis.

Proximal to the hoof capsule, the digital cushion lies superficially, just beneath the skin at the heel bulbs, filling the space between the skin and the DDFT in this region. The name "digital cushion" is an old one and suggests that its function has long been understood to be that of cushioning this region of the digit.

Toric Part

The digital cushion has two parts. The major (toric) part is a thick, twin-lobed pillow that consists of fat within a fibroelastic meshwork that fills the region between the hoof cartilages, the frog, and the skin at the back of the foot. It bulges caudally to give form to the bulbs of the heels, where it can easily be palpated as the soft material under the skin in this region.

Deducing Its Function

In this position, it is well placed to cushion the pastern joint against impact when the heels strike the ground during the gallop. The lack of any tight ligamentous material retaining its shape in the region of the heel bulbs suggests that the purpose of the toric part of the digital cushion is to help dissipate impact forces and reduce the weight of the digit (because fat

weighs less than any other tissue). It probably is not significantly involved in either storage of elastic energy or spreading of the heels.

Its presence, structure, and relationships further suggest that this part of the digital cushion is placed in such a way that its deformation toward the heel bulbs is likely to reduce the effect of rotation in the pastern joint on the spreading of the heels. It is acting as a cushion to the pastern joint, specifically the middle scutum and related structures, during impact and during flexion of the coffin joint.

Cunean Part

The much smaller cunean part of the digital cushion is a V-shaped, collagenous extension of the toric part that fills the central cleft of the frog internally and bulges up (proximally) to form a finger-like projection under the central (midsagittal) part of the coffin joint. Its material is much tougher and stiffer than the toric part, and it is very strongly connected to the dermis of the sole, forming a continuous sheet with the solar dermis across the entire solar surface of the foot.

Deducing Its Function

It is unlikely that this part of the digital cushion is involved in cushioning the navicular bone against ground reaction forces, as has been suggested by some authors. Rather, its position suggests that any significant compression between it and the navicular bone would be likely to fracture the navicular bone by focusing the force in the center of the bone. It seems more likely that the cunean part of the digital cushion is primarily involved in strengthening the central part of the frog during sliding stops. And, by virtue of its intimate relationship with the insertion of the DDFT, it may also help to widen the wedge-shaped apex of the frog when the DDFT is pulling strongly on its insertion.

The presence of the thin, collagenous sheet of fascia between the digital cushion and the DDFT suggests that any spreading of the heels would tend to tighten this sheet and squeeze the digital cushion against the frog. The shape and connections of this sheet through the arc of the semilunar line of P3 and adjacent hoof cartilages suggest that it may also act as a ligament that resists tensioning loads in this region and perhaps protects the central part of P3 from fracturing when the heels spread.

BLOOD VESSELS

The blood supply to and from the foot is discussed in Chapter 17. Only a brief overview of structure and function is provided here. The blood vessels provide nutrients and remove wastes as well as providing a route by which blood-borne chemicals (including hormones and toxins) affect the hoof tissues.

Hydrostatic Effects

The presence of a large volume of blood within the hoof provides the opportunity for fluid pressure to dampen the impact of initial ground contact. It could be argued that the porous structure of P3 is specifically designed to hold a large volume of blood to provide this shock-absorbing effect, and that the large volume of blood contained therein is an effective way of providing impact resistance while minimizing foot mass.

As the hoof capsule is pressed against P3 during hoof loading, the blood-filled dermis is positioned to cushion the underlying bone surface. The highly vascular dermis thus acts a bit like a sock inside the boot of the hoof capsule. In return, the relative movements of the hoof capsule and P3 probably help to ensure optimal blood flow through the dermis.

Digital Pump

Movements between the hoof capsule and P3 with each step must affect the digital blood flow directly. The large changes in pressure, especially during faster gaits, clearly are involved in pumping blood around the digit. Blood can be squeezed away from the soft dermis and into the less deformable bone and hoof cartilages, and sucked into the spaces around the digital cushion as the heels expand.

The different movements between the hoof capsule, P3, the hoof cartilages, the digital cushion, and the dermis thus assist in the return of venous blood up the limb with each step. Exactly how their relative movements affect digital circulation would depend on exactly how the hoof lands as well as the proportions of the different tissues and their positions and relationships within the hoof capsule. It is likely that changes in the habits of use as well as changes in shoeing and the working environment all have the potential to change the blood flow patterns and distribution throughout the horse's life.

NERVES

Innervation of the foot is discussed in detail in Chapter 5. Only a brief overview of structure and function is provided here. As discussed in Chapter 5, the sensory nerves allow proprioceptive feedback for the maintenance of balance and the control of posture; the sympathetic nerves control the smooth muscle in the blood vessel walls and so, along with direct chemical effects of blood-borne factors, they control blood flow through the tissues.

Paths of the Major Nerves and Vessels

The blood vessels and nerves in the digit have to traverse regions of movement (e.g., across joint surfaces) in such a way that stretching or twisting of the vessels and nerves is minimized. That may be why the major vessels and nerves cross joints that have a large range of movement (e.g., the fetlock) in a position that is close to the center of rotation of that joint. Further, the nerves are placed so that they are never compressed during normal use. The blood vessels are placed where they will be exposed to compression for only short periods of time within each stride.

An Intrinsic, Self-Compensating System

The time required for proprioceptive messages to affect the local spinal reflexes is too slow to affect positioning of the hoof during impact. The basic functioning of the digit thus

must be intrinsic to its structure. In other words, the digit must function as an inherently self-compensating system within a wide range of potential positions.

During each step, the digit is placed in a way that is a composite effect of the horse's learned experience and the in-built patterns that are triggered by the speed of movement and the patterns and positions of the other limbs, as well as by the posture of the head, neck, and body. (The posture and movements of the head, neck, and body determine the basic pattern of muscle tone and movement within the limb through the vestibular reflexes and the local spinal reflexes.) Feedback from the sensory nerves within the digit allow the horse to adapt movements of other limbs to compensate for a digit that is moving in an unexpected way, so these nerves are important in maintaining balance and coordinating movements between the limbs.

Most of the intrinsic movements of the digit, however, are a direct mechanical effect of the anatomical connections between different parts of the limb. For example, in an intact cadaver, the digit can be made to move directly and predictably by changing the position of any of the upper limb joints (whether forelimb or hindlimb). In other words, the horse controls the digit indirectly by controlling the upper limb movements.

The only possible way that a horse is able to change the relative movements of the structures within the hoof directly is by contracting the muscles associated with the DDFT (i.e., by increasing tension in the DDFT at specific points within the stride). That means the horse's response to a wide range of problems within the hoof is likely to be an increase in the tension in the DDFT, which causes excess or abnormal patterns of pressure on the flexor surface of the navicular bone.

INTEGRATED FUNCTION OF THE DIGIT

The preceding discussion of the likely functions of individual structures within the digit required a somewhat arbitrary division between the different elements that comprise the digit. In reality, the digit fulfills a range of functions as a whole, with the different structures interacting in a complex way to perform these functions. The following discussion focuses on the contribution that the different structures make to various functions of the digit as a whole.

The digit is designed to perform the basic functions of body support, movement, and protection of the tissues against the ground surface. These functions have both static and dynamic aspects. The static aspects include protection and resistance to wear (discussed on p. 26), maintenance of tissue blood supply and innervation (discussed in the preceding section), and support of the body. Dynamic components include impact dissipation and attenuation, stability (i.e., balance) on different surfaces, elastic energy storage, and transmission of leverage.

Support of the Body

This function of the digit requires rigidity and an ability to resist compression, so it probably is mainly achieved by the bony column comprising P1, P2, and P3. These bones are tied together by collateral ligaments so that movement is largely restricted to flexion and extension. Flexion and extension are themselves restricted in the digit by the strong fascial sheaths and the connections of the various tendons and ligaments.

Contributions of the Connective Tissues

The connective tissue elements (which are especially thick and strong on the palmar aspect of the digit) allow the digit to rest at an angle to the ground, against the strong pull of the suspensory apparatus (suspensory ligament and sesamoidean ligaments). The strength of these connective tissue associations can be felt by trying to move the pastern joint when the limb is lifted. Likewise, in a normal horse it requires enormous effort to move the fetlock joint past 180 degrees (i.e., dorsiflex it), even though dorsiflexion is the fetlock's normal position when the horse is loading the limb normally.

The more the fetlock sinks (dorsiflexes), the greater the tension is in the oblique sesamoidean ligaments, so the greater the compression is of the palmar surface of P1 and the greater the resistance is to further sinking of the fetlock. A similar increase in tension in the straight sesamoidean ligament tends to resist hyperextension (dorsiflexion) of the pastern joint, as do the attachments of the SDFT. Flexion of the pastern joint would likewise be resisted by the extensor slips of the suspensory ligament and the strong attachments of the digital extensor tendon to the proximal edge of P2. Thus, these connective tissues resist movement of the fetlock and pastern joints when weight is on the limb, but they freely allow pastern joint flexion once the limb is lifted.

In contrast, the connections to P3 are nowhere near as stiff. When the limb is lifted, the coffin joint can be moved slightly from side to side and it can be flexed and extended, although these movements are restricted in their extent by the hoof capsule and hoof cartilages and by the ligamentous and fascial attachments between the bones, regardless of the tension in the DDFT. Also, it should be noted that the entire hoof capsule moves with P3 against P2.

Function as a Whole

The anatomy thus suggests that body support occurs through the column of bones, with P1 and P2 forming a relatively straight and rigid rod that is supported at an angle to the ground by the suspensory ligament and distal sesamoidean ligaments. In contrast, the hoof capsule and the structures it contains form a cushioned, slightly flexible ground support for the rod of bone that ends at P2. This column of support is designed to allow the hoof to meet the ground surface at a range of different angles, while still providing the relatively rigid column through the pastern that is needed to support the body weight.

Dynamic Functions and Intrinsic Control

In general, the limbs act like struts and either springs and pendulums (forelimb) or levers, springs, and pendulums (hindlimb). They are swung from the top of the limb by the large muscles in those regions. Thus, movements of the digits are tied to movements of the upper limb in such a way that flexion and extension occur in sequence whenever there is a change in the joint angles in the upper limb. Any extra weight, especially near the extremity of the limb, will have a large effect on the energy required to move that limb. Minimizing mass therefore appears to have a strong influence on digital anatomy.

Further, the movements of a jointed pendulum are impossible to predict accurately, so a large amount of intrinsic control is needed to prevent potentially catastrophic movements occurring randomly. The majority of this intrinsic control appears to be built into the connections and relationships among the connective tissues of the digit. That is probably why connective tissue envelops the digit in a strong sheath that binds all of the structures together and restricts all but a small range of potential movements, especially when the digit is under load.

Impact Dissipation and Attenuation

The elastic structures that can absorb impact are concentrated in the region below the distal end of P1. Without this impact attenuation, P1 would be prone to shattering because it is a long rod of bone, compared with the more cuboidal P2. Adding to its vulnerability in adult horses, P1 has a fat-filled medullary cavity within a thick ring of compact bone in the distal part of the midshaft.

The shape of the pastern bones at the pastern joint highlight this requirement for impact attenuation quite well. There is a large bulge of trabecular bone at the proximal end of P2, but no corresponding enlargement in the distal end of P1. The form thus suggests that the direction and type of load (ground reaction force) has become much more consistent and predictable by the time it reaches the distal end of P1.

Structures Involved

The structures that appear to be designed to absorb impact are concentrated around the central and palmar regions of the hoof: the frog, the digital cushion, and the associated cartilaginous structures. Horses tend to land further toward the heels at the faster gaits, which is likely to concentrate the impact load onto the frog and the digital cushion, and upwards onto the distal scutum and then the middle scutum, and probably also sideways into the heels and the hoof cartilages.

Influence of Hoof Conformation and Composition

Smaller hooves tend to be lighter and thus provide less resistance to movement at high speed than larger hooves; but in the smaller hoof, the load must be distributed over a smaller area, so the materials that absorb the impact need to be more concentrated. The higher frequencies of resonance caused by hard ground (rock and compacted soil) probably are more easily absorbed by cartilage than by fat. On the other hand, fat weighs less than cartilage for a given volume, so the total energy cost of movement is reduced if fat is used as the shock absorber.

Horses from semi-desert and arid regions tend to have smaller, more upright hooves with more extensive hoof cartilages. In contrast, horses raised in soft, lowland pastures or peat bogs tend to have larger, flatter feet and a fat-filled digital cushion. So, there seems to be a direct effect of environmental stimulation on the development of hoof shape and the extent of the hoof cartilages. Genetics clearly plays a part, too, as certain breeds are known for having "good" or "bad" feet.

It remains to be shown, however, exactly how much and what type of stimulation, applied at what time in the horse's life, is needed to optimize hoof structure and conformation for an individual—and further, what is needed to maintain the best hoof structure and conformation throughout the individual's working life. Various observations and conclusions have been made over the centuries. One of the earliest in recorded history is Xenophon's suggestion that a surface made of smooth, flat river stones the size of the palm of the hand is the best stimulus for maintaining sound hooves.

Fluid Damping of Impact Oscillations

Blood vessels in the digit are concentrated around the coronet, where (in addition to their nutritive functions) they may be involved in damping impact oscillations from the relatively hard and inflexible hoof capsule against the bones in this region. Similarly, P3 is riddled with vascular channels, particularly through the lower half of the parietal surface, where the bone might be expected to experience high levels of impact, especially when the toe of the hoof capsule digs into the ground and P3 is pushed up against the blood-filled elastic cushion of the laminar dermis and the relatively inflexible hoof capsule in this region.

The hoof cartilages and adjacent connective tissue also contain many blood vessels that are likely to be involved in the dissipation of impact forces. The particular distribution of blood vessels within the foot suggests that fluid damping of the shock of impact occurs throughout the toe region, around the coronet, and especially within and between the hoof cartilages. That may be another reason why horses tend to land further toward the heel as they increase their speed.

Stability (Balance) on Different Surfaces

In general, balance is probably more of a priority to the horse in the forelimb, whereas transmission of leverage is likely to be more of a priority in the hindlimb. The shape of the underside of the hoof capsule reflects this difference: it is more round in the forelimb and more oval in the hindlimb. A round shape is the most efficient way to support a weight, while the oval shape of the hind hoof might be more effective in providing leverage. From their appearance, it seems that the hind hooves are designed to slide and push, whereas the fore hooves are designed to bounce and hold.

Structural Components

Stability on different surfaces is achieved by damping and redirecting forces through the coffin and pastern joints so that P1 is loaded in a predictable and consistent way despite a wide range of potential landing postures. The collateral ligaments of the coffin and pastern joints restrict movement to the sagittal plane, and the pastern joint is further restricted to only very minor movements in other directions by strong ligamentous connections. Thus, the digit can be thought of as a relatively stiff and stable rod of bone (formed by P1 and P2) that balances on P3, which, in turn, is tightly held within the hoof capsule in the following way:

1. P3 fits snugly into the hoof capsule and attaches very strongly to it via the intervening dermis.
2. Laterally, the hoof cartilages are connected strongly to P3 and P2 and contribute to the elastic sheath that encloses the hoof contents around the proximal edge of the hoof capsule and includes the extensor tendon.

3. The extensor tendon fans out across the extensor process of P3, thickening as it inserts into P3; it also connects strongly to the edges of the hoof cartilages on each side.
4. The dermis of the coronary band attaches strongly to the external surfaces of the extensor tendon and the hoof cartilages to form a ring that supports the epidermis from which the hoof wall grows.
5. The hoof cartilages also are attached to the digital cushion via strong collagenous bands.

Thus, the hoof capsule, P3, the hoof cartilages, and the digital cushion combine to form a buttressed and padded slipper that is held tightly into the fascial sheaths of the rest of the digit, primarily through the extensor tendon connection.

Functional Integration

The shape of the hoof capsule means that it can maintain stability through a wide range of landing positions and angles and on many different surfaces. The strong, stabilizing hold of the extensor tendon, along with the collateral ligaments of the coffin joint, tend to maintain the hoof capsule's relationship to the long axis of the pastern bones.

The majority of potential movements between the hoof and the pastern bones are thus concentrated in the navicular region. The navicular bone, in its ligamentous sling, can dampen and redirect any twisting or tilting movements from the hoof capsule, assisted by the damping effect of the digital cushion and the elastic "give" of the hoof cartilages. In this way, the hoof capsule can be inherently stable under a wide variety of conditions.

In general, the hoof capsule acts as an integrated whole so that movements in any one part of the capsule are reflected by adjustments in the positioning of the rest of the capsule. Because of this integrated function, only specific movements and forces are experienced by the upper limb bones, almost regardless of how the hoof lands or on what type of surface.

Suction Cup Effect

Another mechanism that may play a role in stability is the proposed suction cup effect of solar concavity, perhaps with input from the DDFT insertions. As discussed in the earlier section on the frog (p. 34), the DDFT inserts into the flexor surface of P3 and into the dermis that underlies the bars of the heels. Thus, when the fetlock is hyperextended (dorsiflexed) immediately after placement of the foot, tension in the DDFT would tend to draw up on the interior of the sole and create a "suction cup" effect that pulls the bearing surface of the hoof wall firmly onto the ground.

This suction effect would be released as the body weight swings forward ahead of the limb and the fetlock straightens prior to the limb being lifted off the ground again. Because of its rounded shape, concave underside, and elastic wedge of the frog at the back, the shape of the hoof capsule on its own is likely to create a suction cup effect on at least some types of surface when the hoof capsule is loaded.

Elastic Energy Storage

All of the structures in the digit are elastic to some extent. The digital cushion and hoof cartilages are positioned and formed in a way that suggests they are designed to dampen impact loads, with a relatively small proportion of such energy being converted into useful movement. In contrast, the flexor tendons and sesamoidean ligaments seem to be formed and positioned to function as elastic energy stores—to stretch and recoil and thus minimize energy loss during movement. Exactly how much elastic energy is stored in any of these structures depends on many variables such as conformation, gait, and timing in the stride.

Hoof Capsule

The tissues of the hoof capsule are somewhat elastic. They "give" a little when loaded and spring back into shape when unloaded, thus cushioning the movement and providing some elastic energy storage. As long as the capsule remains free of major cracks, cuts, and tears, it has an intrinsic form that resists, absorbs, and dampens movement and distributes the loads of locomotion and of the body weight across its surface and through into the structures inside.

However, the hoof capsule will adapt to persistent deformations and gradually change shape towards any repetitive load, so its behavior is somewhat plastic in the long term. Also, the hoof capsule has to grow continuously to replace the bearing surface, which is being worn away through use. Its growth is affected by the way in which the hoof is loaded. Any change in the habitual pattern or frequency of loading or the type of working surface will therefore cause changes in the shape of the hoof capsule over time and affect its elastic responses.

Transmission of Leverage

In general, transmission of leverage is more of a priority in the hindlimb, whereas balance is probably more of a priority to the horse in the forelimb. In the hindlimb, the tendons and ligaments of the digit continue the movements of the reciprocal apparatus which ties flexion and extension of the hock joint to those same movements in the stifle joint. In this way, the digit flexes when the hock and stifle flex, and it extends when the hock and stifle extend.

This arrangement means that forces from the muscles in the thigh, rump, and back are transmitted to the hoof in such a way that the direction, timing, positioning, and amount of force through the digit is dictated by those large muscles and the general posture of the horse.

Influence of the Rider

The multiple and complex connections between the fascia of the digit and the fascia and muscles of the upper limb and back mean that a skilled and sensitive rider can learn to influence the movements of the hindlimb directly and hence control the whole horse. The saddle is balanced on the longissimus dorsi muscle, which is directly continued over the ilium as the middle gluteal muscle, which in turn is connected via complex fascial sheaths into all the other muscles of the rump and thigh. Thus, very slight movements of the rider's seat and weight can have a large effect on hindlimb movement, especially when the rider also encourages the horse to find "self carriage" (a posture that provides a maximum range of movements within balance) so that the reflex tension in the extensor muscles associated with maintenance of balance is at a minimum.

POSTURE AND MOVEMENT

This discussion of the functional anatomy of the digit would not be complete without mention of the potential influences of the habits of posture and movement. Loading of the hoof is directly influenced by where the limb is held in relation to the rest of the body. In a standing horse, the positions of the limbs are controlled by the tone and positioning of the muscles at the top of the limb as well as the overall posture of the horse's body.

Once the horse is moving, the muscles of the body and the overall posture of the horse determine how the limb moves in relation to the body. However, the tone and timing of contractions within the muscles of the limb, especially the digital flexors, can affect how the hoof interacts with the ground.

Influence of Habitual Patterns

These patterns of muscle use and postural control are built up through experience and practice, so the movements that are repeated the most are those that are the most likely to be used in any situation. The movements that are repeated the most thus have the greatest effect on the hoof, in that the hoof will change shape in response to these movements.

Influence of Pain

The most likely cause of a change in movement habits is a source of pain somewhere. In the equine athlete, the distal forelimb and the back are common sites for conditions causing pain. With pain in the distal forelimb, the horse stiffens and contracts the muscles to hold against the movements or postures that exacerbate the pain; these holding patterns usually include the muscles of the back, so these horses often become backsore.

When the back is the primary site of pain, the horse will also stiffen its muscles in response to the pain, so movement of the limbs, especially the forelimbs, becomes stiffer and less coordinated. As a consequence, these horses are more likely to develop pain in the distal limb (especially in the joints) because of inappropriate movement and loading.

Responsibility of the Rider/Trainer

The trainer and rider thus have a great responsibility to find and practice the movements and postures that suit each particular horse for each type of use, and to educate the horse and develop its training in a stepwise manner that allows the tissues to adapt to the loading that they will encounter during normal use. Also important is to give the horse a chance to develop proprioceptive skills in the control of its own body in these different ways of working.

Over time, the trainer's or rider's skill is reflected in the growth and shape of the horse's hooves and the horse's general soundness. If the trainer is getting it right for a particular horse, and there are no accidental causes of pain to the horse or major unilateral conformational defects, then the hooves will become more uniform in shape and become more of a "matched set."

This result of course requires the input of a skilled farrier, too. The best trainer cannot entirely overcome the effects of consistently poor trimming and shoeing; but neither can the best farrier completely mask the effects of continuously poor training—although each can provide considerable assistance in maintaining soundness, despite the problems caused by the influence of the other.

A skilled trainer or rider has many ways in which hoof loading can be positively influenced through repetitive adaptations of the horse's posture and movements and the practice of different exercises. A discussion of these concepts is beyond the scope of this book, but an understanding of how to maintain a horse's soundness in a particular discipline of horsemanship can be gained from consulting with a trainer who consistently competes the same group of sound horses with matched and even hooves.

Hoof Shape and Size: A Window to Movement Habits

The greatest effect on hoof wall growth in a healthy horse given a sufficient supply of nutrients and no extremes of conformation is the amount and type of exercise. It is *movement* that maintains the stimulus to grow a good-quality, well-shaped hoof capsule. Exactly what sort and amount of movement, and exactly how this occurs, remain to be shown.

Absolute Size

With small hooves, the load is distributed over a relatively small area, so these feet need to be tougher and more resilient than larger feet. But, while it would seem that large feet are better than small feet, there is a downside to having large feet. During movement, the horse's feet have to travel further and move faster than any other part of the body; and the longer the limb is, the greater the effect is of any extra mass at the end of the limb pendulum. Thus, it is a disadvantage to have feet that are larger than absolutely necessary. That may be why it is unusual for a horse to have very large feet unless the horse has been specifically bred for slow-speed work on soft surfaces (e.g., draft breeds).

Shape of the Solar Surface

It is a common belief among horsemen that horses with flat soles are more prone to sole bruising than are horses with what is considered to be the optimal sole shape: slightly concave. This may be a gross oversimplification. The flatter shape tends to be found in heavy draft breeds and horses living in softer environments, where the weight of the horse would cause the edge of the hoof wall to cut into the ground and extra concavity of the sole might provide too tight a seal for easy release in the later parts of the weight-bearing phase of the stride. Thus, flat soles may be an optimal feature in these horses.

In general, horses raised in the desert (i.e., very dry environments) or in the mountains have a more concave sole. This extra concavity may give extra stability at fast speeds, especially on harder surfaces, because of the suction cup effect discussed earlier. In addition, the increased proportion of cartilage compared with fat in the region of the digital cushion in horses bred in regions with harder ground and lower rainfall may mean that these hooves are much less prone to sole bruising because of their decreased deformability, not just because of the concave shape of the sole.

Relative Size: Matching of the Pairs

The size of the left and right hooves in comparison with each other is determined by the shape and size of the bone within

each hoof and the proportion of the horse's body weight that is placed on each hoof during normal movement and at rest. Over time, if the horse uses one hoof less for whatever reason (pain, dysfunction, or habit), then that hoof is likely to reduce in size compared to its lateral pair. If a horse develops with one coffin bone smaller or abnormally shaped in comparison to its pair, then the hoof that grows around that bone will necessarily be a different size or shape.

If a horse has different sized hooves, then the forces within those hooves must be different, and the hooves are prone to different problems, which in turn will tend to exaggerate any differences. For this reason, early care of the hooves and attention to detail in the young foal is extremely important. If the hooves are tending to grow differently, then the earlier any remedial trimming is instituted, the more likely it is to be successful.

Interpreting Differences. There are enormous variations in hoof size between and within horses, and over time and with changes in use within particular hooves, so an individual horse may have all four hooves very similar or very different in size. When there are large differences in hoof size between forehooves and hindhooves in a particular horse, it may reflect peculiarities of gait. It does not necessarily reflect a problem in that horse; it does, however, indicate that loads are not being evenly distributed between the limbs.

Large differences between left and right hooves are likely to be associated with major differences in the horse's movement between left and right sides (i.e., laterality). This difference may reflect ongoing discomfort within the limbs, with the smallest hooves (generally the least loaded) being the most likely source of ongoing problems. However, having mismatched feet does not always indicate a problem with the horse's soundness. The horse may simply have one small hoof, or one large hoof. In this case, the hooves may be seen to be of a consistent shape within themselves and the horse may be seen to move comfortably on them, dropping them onto the ground and loading the body weight onto them at all gaits with no apparent expectation of pain.

Changing the Hoof Shape

Trimming and shoeing, as well as changes in normal use, can change the shape of the hoof capsule. Hooves are often trimmed for cosmetic reasons; these changes in shape sometimes have a beneficial effect on the hoof, especially when the horse is less than 6 months of age or when a limb has been inappropriately used for some reason (e.g., previous lameness). However, any change in the shape of the hoof capsule tends to be dampened by the effects of the horse's habits of locomotion, which are practiced in the range of familiar movements.

Once the nervous system has learned to balance on a particular hoof shape and size, then any *sudden change* in that positioning will cause problems in the horse's general balance and coordination. That is because even slight differences in the resting length of muscles and in the positioning of all the locomotive structures at ground impact and take-off will change the sensory input and therefore change the accuracy of the normal automatic controls of balance, speed, and muscle patterning. (If you put on a pair of unfamiliar shoes and then go out and dance in them or climb an unstable slope, you might get some idea of these effects.)

A *large and sudden change* to hoof shape can be compared with a person wearing an unfamiliar style of shoe, but magnified many times because the horse can adapt movements of the lower limb only by changing how it uses the muscles in the upper limb. The effect is more like a person trying to walk in high heels for the first time—you have to learn to balance all over again, as does the horse when a large and sudden change in hoof shape is made. In general, too large and sudden a change in hoof shape is likely to increase the risk of damage to other structures in the limb.

Influence of the Farrier and the Rider

Obviously, the farrier can have a large effect on how a horse loads its hooves, and hence an indirect effect on hoof shape, as well as the direct effect of hoof trimming. However, the rider can have as great an influence. If a rider is sufficiently experienced and competent, she can adjust the way in which a horse loads its limbs and thus improve the load distribution through individual limbs so that the horse can work more comfortably and effectively once the limb structures have adapted to the new loading pattern. Over time, the hooves will show this change in use through a change in their shape, as will the underlying bones. Similarly, the farrier can balance the hoof so that the pattern of loading is gradually improved.

Unfortunately the opposite is also true. With a consistently harmful input from the horse's own bad habits (and/or previous injuries), or from the rider or the farrier, even the soundest horse is likely to become unsound. The problem is to determine when the loading is harmful for a particular hoof shape, limb conformation, and pattern of movement. At the moment, it all depends on the experience of the farrier, the trainer, and the rider, who have to develop their own feel for what will work for an individual horse.

Trimming and shoeing is likely to remain an art, although an increased understanding of the science of digital mechanics should assist the farrier in finding the optimum approach for any particular set of circumstances and help him at least avoid making things worse. The resilient, self-correcting mechanism of the hoof capsule, along with the underlying digital structures, allows for a large margin of error—and provides the farrier and trainer with the opportunity to learn how to avoid damaging it, and perhaps even to influence it positively.

PUTTING IT ALL TOGETHER

This chapter has discussed how the different structures of the digit may interact to perform their various roles in protection, support, and propulsion. In general, during each stride the digit absorbs enough of the impact shock to cushion the bones against fracture, while providing sufficient stability for the bones to be able to support the body weight as it moves over the limb. The digit is also designed to minimize the effort involved in its movement by minimizing mass and maximizing elastic energy storage.

Mass is minimized through the use of fat as packing material in the digital cushion and the maintenance of hyaline cartilage extensions to P3 (the hoof cartilages), but probably also by a high blood volume and pressure within P3 and the hoof cartilages at the time of impact. (This latter feature would reduce the requirement for compact bone by supporting the

bone at the time when compressive loads are greatest. However, such localized pressure changes within the bone remain to be demonstrated.) The pressure changes within the hoof also presumably pump blood around the digit and back up the limb.

All of the structures in the digit are at least partially elastic, and this elasticity assists in redistributing and/or dampening any forces on the digit. Elastic energy storage is employed through the use of the complex attachments of fascial sheaths and ligaments that control and redirect tensioning loads, focusing these loads into the large elastic tendons and ligaments on the palmar aspect of the digit. The relative proportions of elastic recoil versus dampening may be regulated both by the positioning of the hoof and limb at impact, and by the timing and degree of muscle contraction within the deep digital flexor muscle bellies, and possibly also the superficial digital flexor muscle as well.

At impact, the hoof tilts and slips, depending on the ground surface; it is then sucked down onto the ground as the heels recoil and the fetlock sinks, so that by the time it is loaded with the body weight, the hoof is held relatively stable. At the same time, proprioceptive influences react by adjusting tone in the muscles higher in the limb and body, including the deep and superficial digital flexor muscles, which potentially assist in maintaining stability within that digit by increasing the tension in their respective tendons. However, the complex fascial and ligamentous attachments create and maintain this stability within a range of hoof positions while the digit is under load, without any extra muscle contraction.

Once the body weight has moved beyond the support of that limb, then the change in posture within the limb causes the ligamentous holds against elastic recoil to be suddenly released. This sudden decrease in tension has a catapult-like effect, such that the fetlock straightens and the limb starts to lift with little extra effort.

Overall, the digit can be thought of as a suction cup on the end of a short rod, which is spring-loaded by the changes in joint (especially fetlock) angle and the attachments and relationships of the ligaments and tendons, so that it bounces along, landing, holding, and then recoiling in sequence within each stride.

3

BIOMECHANICS OF THE EQUINE FOOT

HELEN M.S. DAVIES, JONATHAN S. MERRITT, and JEFFREY J. THOMASON

INTRODUCTION

Helen M.S. Davies

The preceding chapter discussed how investigation of the digital anatomy gives some insight into how the structures might function. However, experimental evidence in this area is limited because of the complexity of interactions of even simple structures at the distal end of a jointed pendulum. Further to this, there is the added complexity of the high degree of feedback control of that system imposed by the nervous system. Thus the hypotheses developed through the careful dissection and observation of the functional anatomy are limited in their scope by the difficulty of identifying all the interrelationships of the different tissues.

To have any certainty about the way these structures function under different conditions, it is necessary to find ways of testing or measuring the effects of specific changes in these structures. In this chapter, we discuss the recent literature in this area of investigation and some of the methods that have been employed to attempt to elucidate the mechanics of the hoof. For a discussion on the history of this area of research, refer to the work by van Weeren.[1]

A description of the basic mechanics of the digit can be simplified, but these simplifications may be misleading when applied to an individual horse, especially when that individual starts to move. On the other hand, although not supported by the current experimental evidence, these simplifications can provide a starting point from which more resilient hypotheses can be developed.

Normal ranges of hoof shapes have been described for specific types of horses. There is no evidence that these shapes were optimum for those horses, however, only that those horses with those conformations, habits of movement, and lifestyle, in that environment, at that particular time, apparently managed well enough to survive with hooves of those shapes.

There is also the problem of determining what environmental conditions were associated with any such measurements. A decrease in the hoof wall angle at the toe has been shown to be associated with time in training in young racehorses, and with wet pasture conditions in free-ranging horses.[103] This change in angle in the young racehorses may have been a direct effect of the increasing proportion of their exercise, which was gallop exercise, as the hoof is known to land further toward the heel with increasing exercise speed, and hence might be expected to be distorted in that direction when exposed to an increased amount of gallop exercise. The effect of wet pasture conditions may have been a direct effect of increases in elasticity of the hoof wall material in wet conditions. Although this experiment clearly showed that the hoof angle decreased in individual horses under these conditions, it is not at all clear what effect this decreased angle might have had on the soundness of the horses, their performance, nor on any other measurable parameter of hoof capsule shape. The results show that the environmental conditions directly affect hoof shape in individual horses, and any measurements need to be related to the individual horse's immediate environment and recent exercise.

Several basic difficulties arise in attempts to investigate changes in any measurable parameters of hoof biomechanics. The first is deciding how a beneficial effect on the biomechanics might be identified. It is not clear whether lengthening a horse's stride, for example, will improve the overall speed of the horse over a given distance. A simple way of avoiding this problem is to use the horse's performance in a standard exercise test as the measure of the effect of any parameter that is changed.

The next problem is the identification of a measurable parameter that might have an association with hoof function. Then, a way of measuring that parameter in a reliable and repeatable way has to be developed and tested. In some cases, a mean of a set of measurements is taken as a reliable measure of a particular parameter; but without a careful investigation into possible sources of error associated with the measurements, they should not be relied on.

Once a reliable, repeatable, and pertinent measurement system has been developed, there is the problem of how to choose the horses to measure. Horses come in many shapes and sizes, and so do their hooves. Further, individual horses' hooves vary with time according to many different environmental and individual inputs, few of which have been described and even fewer of which have been tested experimentally. If horses are given more than one treatment (to use the horse as its own control), then it may be difficult to separate local short-term effects due to changes in the proprioceptive input from the true effects of changes in that parameter on the mechanics of the system. This means that the experiment will probably have to be done over a period of weeks if not months or years. Further, the variable that is being measured is only one part of a complex system and it is likely to be associated with changes to other parameters that may have opposite effects on performance.

This experimental approach attempts to change one specific parameter in a measured way, give the horse some weeks to adapt to that change, and then look at the effect on the mechanical efficiency of the whole horse. A small amount of work has been done in this way to investigate the effect of specific changes to hoof balance on the response to a standard exercise test,[2,3] but these experiments still give no assurance that the changes that produced "improved" performance in that exercise test would not be detrimental to the long-term

soundness of the horse. However, this approach at least allows a test of hypotheses concerning the potential effects of specific changes to the hoof balance. They are one way to investigate this complex system and improve our understanding of how changes to hoof balance really do affect the mechanics of the horse as a whole.

It is clear that the hoof continuously adapts and changes shape according to many different inputs, so a model that includes many different parameters might be expected to have a greater chance of successfully describing the mechanics of the system. This is the approach taken by Jeff Thomason's group, who have begun the long journey of exploration into hoof biomechanics by developing a model of the hoof capsule and then using direct measurement of the effects of various treatments to test the model. A discussion of some of Thomason's findings is given later in this chapter. If it can be developed far enough, this model should eventually allow a complete description of the hoof mechanics and hence a way of determining what the effects of specific treatments should be in a specific set of circumstances.

Considering the complexity of the system, it is remarkable that catastrophic breakdown of the system is so infrequent and generally occurs only when human interference has changed the mechanics of the system beyond its capacity to adapt, such as in shoeing with toe grabs,[4] or when there are other interfering factors (e.g., kicks from other horses, preexisting damage, sudden changes in an otherwise even working surface, major metabolic changes, and so on). Until such time as we have a better understanding of the underlying mechanisms, we will continue to be dependent on the skill and observation of the farrier and horse trainer working together to identify the optimal hoof balance for a particular horse doing a particular type of work.

PRACTICAL APPLICATIONS IN HOOF BIOMECHANICS RESEARCH

Jonathan S. Merritt and Helen M.S. Davies

Current scientific understanding of the hoof is inadequate to properly explain many of the practices that are common when dealing with the hoof and shoeing. Far from driving new innovation and understanding, the science of the hoof often struggles to explain even those things that are well accepted by many people. This situation is the result of the historical knowledge and experience regarding hooves that has been built over time, generally outside of any formal scientific context. Consequently, in many areas, scientific understanding lags behind conventional understanding.

There are many reasons why it is important to investigate the hoof scientifically. Most importantly, science must ask questions in such a way that they can be answered. Many people can scrutinize those answers and, if necessary, they can be tested further in a repeatable way. Ideally, this process minimizes the fallible elements of human experience and opinion. Practically, it means that the results of good scientific research can be trusted and understood more fully than results based on individual experience or opinion. Science also provides a framework in which theories dealing with the overall function of the hoof can be constructed and tested. This approach may eventually result in a more holistic understanding of the hoof, in which modifications can be understood in terms of a basic underlying theory, rather than requiring a detailed experiment for each scenario investigated.

The general field of biomechanics is concerned with the application of mechanical theory to biological systems. Because the hoof is the main point of mechanical interaction between the horse and its environment, it has very important mechanical functions of support and cushioning. Hoof biomechanics concerns the mechanical analysis of the hoof and the mechanical effects of modifications made to it.

Initially, it is interesting to consider why modifications may need to be made to the hoof in the first place. Natural selection has clearly made equine hooves very effective at what they do, so why would we wish to modify them? Although not a full list, some reasons include the unnatural surroundings in which horses are sometimes housed, the unnatural activities in which horses are often engaged, and the genetics of an individual horse that may predispose it to particular problems. Because it is generally accepted that these situations all represent deviations from "normal," it should be clear that understanding the normal function of the hoof is a primary objective in hoof biomechanics. Many investigations into the normal function of the hoof do not yield practical results, but they still constitute basic work that is critical for a full understanding.

When reading the literature on hoof biomechanics, it is also important to bear in mind the special limitations, assumptions, and simplifications that are inherent in some of the investigations reported. An example is the in vitro limb loading experiments that are sometimes used when very invasive measurements must be made. These experiments often call for the close replication of in vivo loads for their results to be valid (e.g., studies by Brommer et al.[5] and Colahan et al.[6]). However, the simulated loads that are applied to the limb are often chosen without detailed analysis or validation, and hence may justifiably be questioned. The problems caused by these special mechanical issues must be weighed in addition to issues that are more familiar to biologists, such as statistical considerations concerning interindividual variation, when attempting to apply research to practical situations.

PHASES OF THE STRIDE

Much of the biomechanics research on the hoof and distal limb deals with only one part of the stride of the horse. Knowing this fact can help in filtering through the available literature when investigating a particular topic. Typically, the stride is broadly separated into two parts: the stance phase and the flight phase. During the stance phase, the hoof is in contact with the ground. In contrast, during the flight, or airborne, phase the limb is not in contact with the ground. Mechanically, an important distinction between the two phases is the presence of a ground reaction force (discussed later) during the stance phase.

The stance phase and flight phase are separated from each other by the two transitional events called impact and breakover. *Impact* starts when the hoof first makes contact with the ground and finishes a short time later when the effects of impact, such as shock waves in the hoof, have dissipated to negligible levels. *Breakover* begins at the end of the stance phase when the heels first begin to leave the ground and continues

until the toe has left the ground. Both impact and breakover occur over a much shorter period of time than the rest of the stance phase or the flight phase. Because they occupy less time, they are correspondingly more difficult to investigate experimentally. Although few practical results are currently available from the research into impact and breakover, it is likely that unique phenomena occurring during these periods are important in the overall function of the hoof.

Ground Reaction Force

During the stance phase of the stride, the hoof is in contact with the ground. This contact allows the hoof to support the weight of the horse and any dynamic loads incurred during locomotion, such as those responsible for accelerating the horse forward. In many mechanical analyses, all of these effects can be summarized in a single quantity called the *ground reaction force* (or GRF), which has been a topic of extensive research.[7-33]

The GRF is a vector quantity, meaning that it has both a size, or magnitude, and a direction in space. The GRF also has a particular line of action along which it acts on the hoof. The line of action of the GRF is the same as its direction, but it is further constrained to pass through a particular point on the sole, which is known as the center of pressure.[18,34,35]

Impact

Impact begins at the instant any part of the hoof first makes contact with the ground and continues until the phenomena associated with impact have dissipated. In general, the toe makes first contact with the ground at slower speeds of locomotion, but at faster speeds, the heels are more likely to make contact first. The most well-documented phenomenon associated with impact is the existence of high-frequency oscillations in the ground reaction force.* It is unknown what effect these oscillations have on the hoof, but they are generally thought to be harmful.

The energy content of the oscillations must be absorbed somehow by components of the horse, the shoe, or the ground. Practically, it has been shown that different track surfaces and shoe types can alter the characteristics of the oscillations.[28,29] Shoes made of light polymer materials with a lower stiffness than traditional steel shoes were shown to attenuate high-frequency oscillations better than the traditional steel shoes.[29] It has been suggested that the ability of the shoe to absorb the energy of oscillation may be beneficial because it prevents this energy from being absorbed by the hoof and the distal limb.[29] However, the effect of shoeing on impact is still an area that requires a great deal of research before definite conclusions can be drawn.

Stance Phase

The stance phase continues from the start of impact, when some part of the hoof first makes contact with the ground, to the end of breakover, when the toe finally leaves the ground. During the stance phase, the horse interacts with the ground, supporting its own weight, propelling itself forward, decelerating itself, turning corners, and so on. The GRF enables all of these activities to take place and is a dominating feature of the stance phase. Due to the presence of the large GRF, the stance phase is the part of the stride in which the largest loads are applied to the hoof.

Components of the Ground Reaction Force

Since it is a vector, the GRF is often separated into two components: horizontal and vertical. Practically, these two components have different meanings. The vertical GRF is mainly responsible for support of the body weight, but it also has an oscillatory "bouncing" component connected with the vertical motion of the body during the stride. The horizontal GRF, on the other hand, is caused mainly by traction between the hoof and the ground. It is responsible for slowing down the limb in the first half of the stance phase and then accelerating the limb during the second half of the stance phase.[8,10,11,13] It has been shown that the forelimbs are responsible for slightly more of the deceleration part of the horizontal GRF component, whereas the hindlimbs are responsible for slightly more of the acceleration component.[11]

Many practical modifications to the hoof can be expected to alter the ground reaction force. For example, inadequate traction between the hoof and the ground can cause slipping of the hoof, which will particularly alter the horizontal GRF and reduce the horse's ability to control the position of its feet. Mechanical models of the limb can be used to guide research into the relationship between different modifications to the hoof and the ground reaction force. This remains a topic for future research, however, and currently no well-accepted descriptions of these relationships exist.

Mechanical Loads on the Third Phalanx

Ultimately, almost all of the body weight and dynamic loads of the horse must be supported by the third phalanx (P3, coffin bone). These loads are then transmitted to the hoof capsule and finally to the ground. Thus, the ground, hoof capsule, and P3 together form an important mechanical system whose function is one of the most critical aspects of hoof biomechanics.

It is instructive to consider the mechanical loads that act on P3. Unlike the more proximal limb, in which loading in any element is relatively simple, the complex anatomy of the hoof allows it to function in many different loading scenarios (see Chapter 2). However, the loading is proscribed to a certain extent by the functional requirement that pressure acting over the region of the hoof in contact with the ground must support the horse. During the stance phase, the complex loading on P3 can be separated into four major components. These loads are distributed over the region in which they act:

- The downward force exerted by the second phalanx (P2) on P3 over the articular surface.
- The proximopalmar pull of the deep digital flexor tendon on the solar surface of P3 (including the upward push on the navicular bone).
- The upward pull by the laminae of the hoof on the rounded, dorsal surface of P3. (And P3, in turn, pulls downward on the laminae, according to Newton's third law.)
- The upward push by the material of the sole, frog, and heels on the solar surface of P3.

*References 9, 13, 20, 21, 23, 26, 28-33.

The last two components are due to external loading on the hoof, whereas the first two are internal loads exerted by the rest of the body. The downward pull on the laminae is transferred to the ground via the hoof wall and is controlled by the contact between the hoof wall and the ground. Conversely, the upward push on the solar surface of P3 is controlled by contact between the ground and the sole, frog, and heels of the hoof.

To a certain extent, the vertical component of the pull by the laminae and the push on the solar P3 can offset each other. If one component is increased, then the other is likely to decrease. Practically, this is an important principle in many of the suggested treatments for laminitis.[36] Packing the sole or using heart-bar shoes increases the upward push on the solar surface of P3, supporting it from below, and hence is likely to decrease the pull on the laminae. However, it is important to note that the force exerted by the laminae can also balance the pull of the deep digital flexor tendon. This multiplicity of functions and the consequences of loading is common in the hoof.

Observations on Hoof Loading

The balance between loading of sole elements (sole, frog, heels, and bars) and the hoof wall can also be changed in a practical fashion by trimming and shoeing. The correct, or ideal, balance between these two types of load is still unknown scientifically. Some initial observations of wild horses placed onto an artificially hard surface demonstrated that the major points of contact occurred at three or four points around the hoof wall.[35] This finding is commonly known as the three- or four-point contact pattern, and some people have advocated trimming hooves to simulate this pattern.

Further work on the topic, however, has shown that the three- or four-point contact pattern may be an artificial finding that is caused by taking horses that are accustomed to soft ground and placing them on an unusually hard surface.[35] This more recent work has shown that when horses with the natural three- or four-point pattern are placed on a softer, natural surface, then a much more uniform pressure distribution exists over the entire sole. This observation suggests that the more uniform distribution may actually be more natural and perhaps more beneficial. Unfortunately, too little work has been done on this topic so far to draw any firm conclusions.

Hoof Capsule Deformation during Stance

During the stance phase, the hoof deforms under the weight of the horse and the dynamic loads of locomotion. The two main deformations observed are a horizontal spreading (or "expansion") of the heels away from each other[37] and a dorsoconcavity, or bowing-in, of the dorsal hoof wall.[38] More complicated deformations, such as the vertical compression of the hoof wall, and the effects of nail holes on the local strain environment have also been investigated.[39-44]

The mechanism of heel expansion is still unknown scientifically and is a continuing topic of research. The two main theories of heel expansion are that (1) pressure on the frog and other solar structures causes the material within the hoof to force the hoof wall outward, and (2) rotation of the phalanges forces the wall outward.[31,38,45,46] Another possible explanation for heel expansion, although one that has been less thoroughly investigated, is to be found in the shape of the hoof wall. Simulated loading of donkey hoof wall geometry by finite element analysis (FEA) showed expansion of the heels without the incorporation of any loading due to frog pressure or the more proximal phalanges.[38]

Regardless of the mechanism of heel expansion, it is known practically that heel expansion should be allowed for when shoeing a horse. This consideration leads to farriers refraining from nailing around the more palmar quarters of the hoof to allow the heels to move more freely. The repeated back-and-forth motion of the heels and quarters of the hoof often polishes the corresponding regions of shoes that make contact with them, leaving a shiny surface in these regions when shoes are removed.

Effect on Digital Blood Flow

The mass of the horse acting downward on P3 and the hoof affects the blood flow through the digit. It has been shown that the first half of the stance phase is associated with a large increase in pressure in the lateral digital vein.[47] Furthermore, static weight bearing on the hoof has been shown to result in a decreased blood flow in comparison with the non–weight-bearing state.[48] Although not properly established scientifically, it seems likely that the sequence of events during locomotion acts as a pump for blood in the hoof.

In this hypothesis, the first half of the stance phase would act to force blood out of the distal limb via forced venous drainage. This event would be followed by a period of relatively little blood flow during the second half of the stance phase. Finally, the flight phase of the hoof would draw blood back into the distal limb via recovery of previously compressed tissues, normal arterial flow, and the centrifugal effect of fast limb rotation. The presence of valves in the veins and overall cardiac pressure would ensure that the blood is pumped in the correct direction.

It is not known how practical modifications to the hoof affect blood flow, but ensuring adequate contact of the solar structures with the ground and allowing for the natural deformations of the hoof (such as heel expansion) can be expected to play a role in maintaining normal blood flow.

Damping Mechanism. Viscous friction in fluids increases with increased rate of fluid flow. The rate of flow, in turn, increases with an increased local pressure gradient. Hence, a large pressure gradient such as that generated at impact has the potential to cause viscous friction between the blood and the walls of the blood vessels within the hoof. This effect is further enhanced by the fact that the blood is forced to flow through vessels with a relatively small diameter. The energy dissipated by viscous friction may therefore be an important damping mechanism for the effects of impact.

Breakover

Breakover begins when the heels first begin to leave the ground and continues until the toe leaves the ground. It is preceded by a quick movement of the center of pressure of the GRF from its normal position within the contact area of the sole to a position at the tip of the toe.[49] Relatively little is known scientifically about breakover, and investigation is limited by the short period of time during which it occurs.[16] Recent work

has shown that the fast release of energy stored in the more proximal limb tendons during the stance phase, particularly the tendon of the biceps brachii muscle, is triggered at breakover. The fast recoil of the tendon (in comparison with the relatively slow stretching of the tendon as it is extended during the stance phase) leads to what has been described as a "catapult action" that allows for rapid limb protraction.[50] It seems likely that the geometry of the hoof affects breakover by altering the precise timing of this energy release,[51,52] although this theory has not yet been explored.

Flight Phase

The flight phase begins as the toe leaves the ground at the end of breakover and continues until some part of the hoof makes contact with the ground again at impact. The toe follows a curved path through the air during the flight phase. Unlike diagrams shown in many traditional textbooks on farriery, which typically show only one peak in the path of the toe, kinematic investigations have shown that the path has two peaks, forming what is described as a biphasic pattern.[51] However, beyond demonstrating its existence, this research has not yet demonstrated any practical consequences of the biphasic pattern.

The main scientific investigations into modification of the flight phase have centered on changing the hoof angle, toe length, and mass of the hoof. Although these are very practical modifications that can be made to the hoof, the existing studies that have investigated these changes generally are inadequate. Some of these inadequacies include the small numbers of horses used and lack of a specified adjustment period to allow the horses to become accustomed to each new shoeing scenario.

In one study of only two horses, decreased hoof angle was found to increase the frequency of toe-first landings and lengthen the duration of breakover, but it did not change the stride length.[49] A longer toe has been found to extend breakover time at the walk[49] and trot,[52] and to increase the maximum height of the toe and maximum flexion of the fetlock joint at the trot.[51] Increasing the mass of the hoof has also been found to increase the height of the toe at the trot and increase fetlock joint flexion.[52] Importantly, however, no mechanism has been proposed to account for these observed changes in the flight of the limb.

MECHANICAL MODELS OF THE DISTAL LIMB

A primary objective of much of the research into the equine distal limb has been the development of models that describe the mechanics of the distal limb and hoof. The two main types of models that have been developed are the FEA models of the hoof,[38,53-57] which are based on the principles of solid mechanics, and the mechanical models of the distal limb bones and tendons,[50,58-68] which are based on the principles of statics and dynamics. The application of these models to real-world scenarios relies, in part, on knowledge of the properties of the materials, geometry, and other relevant parameters of the hoof and distal limb.[69-86]

The importance of these models is that they allow aspects of the function of the hoof and distal limb to be investigated theoretically, rather than requiring all parts of a particular scenario to be investigated practically. For example, mechanical models of the distal limb bones and tendons have allowed the tension in the different tendons to be calculated from mechanical principles.[58,59,62,63,65] Without such models, tendon tension must be measured directly, which is difficult because it is often an invasive procedure. Pain from the presence of the measurement apparatus, or the analgesia required to prevent such pain, may affect the results. More recently, the noninvasive measurement of tendon tension using ultrasound has been reported,[87,88] but this technique is still in its infancy.

The use of mechanical models has enabled the effects of heel wedges on the tension in the superficial digital flexor tendon to be assessed.[58] It was found that the application of heel wedges increased, rather than decreased, the load in this tendon, and may therefore be detrimental in the treatment of conditions in which decreased tendon tension is desired.[58]

All mechanical models of parts of the limb take certain parameters as inputs and produce certain outputs. However, the current models deal only with isolated portions of the entire locomotive system. This incompleteness means that there are always extra potential dependencies among the inputs and outputs of the model. For example, suppose that the GRF can be changed in some way by modifying the hoof. A model may then correctly predict how the tension in the limb tendons changes when this change occurs in the GRF. However, in response to the change in the tendon tensions, the horse may adjust its movement, thereby changing the GRF from its presumed value once again. This external control is an additional loop in the system that falls outside of the scope of the original model.

To overcome the problem of outside dependencies, more complete models of the limb are always sought. The development and validation of such models is the main area in which biomechanics research can contribute to investigation of practical changes that are made to the hoof. In the meantime, all input parameters to any given model must be measured from real horses, thus allowing all external dependencies to be accounted for by the real system.

THE HOOF AS A SMART STRUCTURE: IS IT SMARTER THAN US?

Jeffrey J. Thomason

SMART STRUCTURES

Animals and plants have been using "smart" structures and materials since their origins far back in geologic time. Humans have taken a little longer to catch on, and the concept of "smartness" has become entrenched in the world of engineering only in the past few decades. It is from the engineers that we borrow this definition: Smart structures and materials are those that can adaptively respond to changes in their function with time.

An adaptive response is one that is appropriate for the change in function. Muscles provide a good example. If you take up weight lifting, you change the function of some

muscles. "Function" in the case of muscles is "producing force," and the change in function is an increase in the total amount of force you expect the muscles to produce on a daily basis. They respond within a few weeks by becoming bulkier and stronger. In contrast, if you spend an inordinate amount of time watching television, your muscles are used much less and they atrophy (decrease in size and strength) quickly. The skeleton is similarly smart, and can increase and decrease in bone mass and strength according to the forces acting on it over time. Smartness of this kind is found almost everywhere in biology.

Is the Hoof Smart?

It is fairly certain that the horse's hoof is another example of a smart structure. It is known that the hoof responds to changes in its function, but not enough is known about the normal workings of the hoof to be sure that the response is adaptive, that is, appropriate for the change.

It is easy to perturb the normal mechanical functioning of the hoof in many ways: a different trim or shoe, a new daily exercise or riding pattern, a change in feed, pregnancy, or in cold climates moving the horse from barn to pasture in the spring and vice versa in the winter. In the wild, seasonal changes in ground consistency and migration across varying terrains would achieve similar effects.

Dealing with Perturbations: Tolerating Them

The hoof has two ways of dealing with these perturbations. One way is simply to tolerate them. The hoof is a structure that is constructed to take a lot of mechanical abuse and to allow the animal to travel on footings that can be quite unpredictable. Because of the unpredictability of its contact with the ground, the hoof has a high tolerance for variability in the forces and shocks it has to withstand and can therefore tolerate the effects of many perturbations that may increase or decrease those forces and shocks.

Tests on the strength and stiffness of hoof wall material have shown exactly how far it is from failing under the loads applied during normal locomotion. To perform such a test, a bar is cut from the wall at the toe that runs from the coronet to the ground surface, measuring perhaps 6 ×6 × 900 mm. The ends of the bar are clamped in a machine that applies tensile force to the part of the bar exposed between the clamps and stretches it. As more force is applied, the bar continues to stretch (measurably but not always visibly) until it breaks. At the moment of breaking, the *stress* in the bar is the force divided by its cross-sectional area (in this example, 36 mm^2), and the *strain* is the amount of stretch divided by its original length.

Breaking stresses (or ultimate stresses) for hoof material at the toe are in the order of 17 to 30 megapascals (1 MPa = one million Newtons per square meter), compared with mild steel at 210 MPa. Breaking strains are in the order of 45% to 50% for fully hydrated specimens (i.e., hoof soaked in water),[89] which means the specimens elongate by up to half their original length before breaking. On the living animal, the average relative humidity of much of the wall is approximately 75%, and at this level of hydration breaking strains fall to about 15%.[90]

That seems to be a dramatic difference, until it is compared with strains that occur in the wall during normal loading. If small strain sensors or gauges are glued to the outer surface of the hoof wall of a live horse and recorded from while the horse is trotting quite fast, the normal operating strains are found to average about 0.25% and to peak at about 1%. Peak strains would have to increase by a factor of 15 to be a serious threat to the hoof. That is quite a wide margin of safety, and we can conclude that the hoof is very resistant to any perturbation in applied forces.

Dealing with Perturbations: Respond to Them over Time

The second way the hoof deals with perturbations is to respond to them over time, in a similar fashion to the changes seen in muscle and the skeleton. The most obvious responses to observers are those in growth rate and shape of the hoof. Use of heel studs on a racing hindshoe are often accompanied by lengthening of the hoof; some shoe types induce frog growth; and unshod horses on stony ground often develop low-heeled, long-toed hooves.

The list of examples is long, but in cases such as these, we have no easy way of telling whether the response is really adaptive or just a response. In other cases, it is easier to say that the response is adaptive, for example when a chronically lame individual is returned to soundness after the application of a therapeutic shoe. In a case such as this, the perturbation (applying the new shoe) is a deliberate attempt to resolve an existing problem. When successful, the treatment has either brought the forces and shocks on the hoof into a tolerable range, or it has induced a response that has done that. If a response was involved, we can be sure that it was appropriate and, therefore, was adaptive. But cases in which we are sure the response was adaptive are in the minority. For the moment, the question of whether the hoof is smart remains open.

Problems with Smart Structures in Biology

One of the main problems with smart structures in living creatures is that they are an integral part of their parent organism. They have evolved along with all the other parts of that organism. Because of this, the function of the smart structure is usually intimately and inextricably entwined with the function of other parts of the body. The skeleton is an example.

The minerals that give bones much of their strength are salts of calcium. Calcium salts are removed from the blood and added to bones in times of heavy exercise. They are replaced in the blood largely by calcium in the diet, and their presence in the blood is vitally important for the normal working of the heart, among other things. During more sedentary periods, some calcium salts are removed from bone and are used to restore levels in the blood. This is a normal adaptive, smart process. But calcium intimately links skeletal function with heart function, and this can cause trouble. The process starts to go awry when dietary calcium levels drop, or in some disease states such as osteoporosis, when blood levels of calcium start to fall. At this point, the heart takes precedence over the skeleton, and calcium is taken from the bones, whether or not the skeleton has a high demand for calcium at that time. Weakened bones are the result.

In the hoof, the laminar junction has a similarly intertwined function. The primary purpose of the laminar junc-

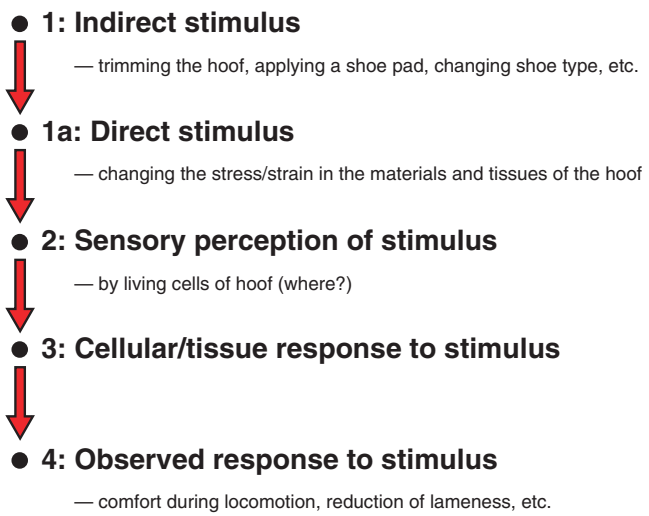

FIGURE 3-1 Flow chart illustrating the probable sequence of events from the application of an indirect stimulus to the observed response.

tion is to support the coffin bone inside the hoof wall and to resist forces tending to separate the bone and wall. The laminae also appear to act as shock absorbers. Every time the hoof hits the ground—like a sledge hammer being slammed onto a block of concrete—the shock waves in the hoof itself appear to be mostly absorbed within the laminar junction. It is a structure that is well constructed for a couple of different mechanical functions.

The laminar junction is very susceptible to events elsewhere in the body that have nothing to do with resisting force and shock in the feet, however. Grain overload, drinking large quantities of cold water after exercise, and colic are among the many causes of laminitis—the debilitating breakdown of the laminar junction—but none have any direct connection with the mechanics of the hoof. Each of them changes something in the blood, such as the concentration of carbohydrates or byproducts of inflammation, the temperature, or the acidity, and these changes trigger reactions in the living (sensitive) laminae that rapidly lead to the clinical signs of laminitis.

Understanding Hoof Smartness

Assuming the hoof is smart, it is possible to plot a likely chain of events between perturbation and response (Figure 3-1). The perturbation itself can be called an *indirect stimulus,* because the hoof probably does not detect it directly. For example, trimming the hoof is an indirect stimulus. It is unlikely that a horse can feel the difference in its hoof before and after trimming based on the sensations in the hoof while it is held in the air. Once it puts the hoof on the ground, however, the sensations are different. That is because the nerve endings in the hoof can detect subtle changes in how the forces of weight bearing compress or stretch the capsule and enclosed living tissues.

Many, if not all, of the other cells in the hoof, in additions to the nerves, will be able to detect these changes too, so the changes in compression and stretching of the capsule are probably *direct stimuli.* They are capable of being directly detected by cells of various types. Exactly what the direct stimuli might be is discussed in a later section.

Sensing Direct Stimuli

Sensing the direct stimulus or stimuli is the first stage in producing a response by the living cells of the hoof. A stimulus may exert pressure on the cells, or exert tension on the cellular membrane, or change the rate of flow of fluids carrying ions past the cell. All of these changes are potentially detectable by cells, many of which may also have a *cellular mechanism of response* involving a chemical signal being transmitted from cell membrane to nucleus, where they turn on specific genes or turn them off. This process increases or decreases the concentration of the products of those genes (usually enzymes) in the cell, which in turn affects the rate of biochemical reactions outside the nucleus. Alternately, the response may happen entirely outside the nucleus, involving the activation or deactivation of proteins already present.

In either case, the result may be a change in the rate of replication of the cell or in the amount of extracellular substances made by it, and these are the end responses of the cell to the initial stimulus. Such responses are entirely invisible to the observer and are detectable only by laboratory experiments on gene and enzyme activities.

Observable Response

If enough cells respond to the initial stimulus, they may produce a change in the hoof that is observable, for example, increased or decreased growth rates of the wall, enlargement of the frog, or changes in the shape of the whole hoof capsule. These are a first level of *observable response to the stimulus.* A second level may be the observer's perception of reduction in lameness or a general increase in the comfort of the animal while moving.

To be pragmatic, we can obtain a lot of useful information about hooves simply by applying an indirect stimulus and observing the visible response, that is, jumping directly from step 1 to step 4 in Figure 3-1, without worrying about the intervening steps. These kinds of empirical tests are made routinely by farriers and horse owners.

To be more scientific, we could measure the stimulus and response with a number of animals under controlled conditions and with suitable controls, although this kind of experiment is rare. Experiments on hooves are not rare, but experiments looking specifically at how hooves respond to mechanical stimuli over time are. This avenue of investigation has largely been bypassed in favor of studies of specific ways to reduce lameness or of basic biomechanics.

There are no studies of direct mechanical stimulus, sensory perception by hoof cells, and cellular responses (steps 1a to 3 in Figure 3-1). What these steps describe is the mechanism by which an indirect stimulus causes an observable response. Mechanisms by which laminitis occurs at the cellular and molecular level are beginning to be examined in detail with elegant laboratory experiments.[91] But responses to the everyday mechanical stimuli acting on the hoof have evaded direct study.

Figure 3-1 lists the types of information needed to fully understand how hooves, and the tissues inside them, respond to the perturbations that the environment, the animal's own activities, and human interventions inflict upon them. The advantage of obtaining such information in a scientific manner

is that it provides considerably better predictive ability about the kind of response expected for a given perturbation. Of course, the number of possible perturbations, variations in their intensities, and combinations of them make this degree of understanding a distant reality. But it should be the goal, perhaps the primary goal, of equine hoof biomechanicists.

Interpreting the Response

One more category of information is also needed—perhaps a step 5 in Figure 3-1—and that is the answer to the question: Is a given response mechanically appropriate for the stimulus that caused it? If the answer is "yes," then there is evidence that the hoof is indeed smart.

BIOMECHANICS OF THE HOOF

Up to this point, I have tried to establish a conceptual framework for understanding biological responses of the hoof to its mechanical functions. In this section, I will describe specific details of those mechanical functions. The description represents my interpretation of current studies of hoof biomechanics, mostly based on work in my own laboratory (with significant contributions from several students and technicians), but readers should be aware that other interpretations exist for some of this information. Indeed, I will mention some, and you can draw your own conclusions.

Most of the events described occur between impact of the hoof with the ground and midstance.

Loading of the Hoof

At racing speeds, each hoof hits the ground 150 times per minute, or more. At the moment of impact, the hoof decelerates extremely rapidly, like the head of a hammer hitting a block of wood. This action causes very large forces to be applied to the hoof for a very short period of time and sets up shock waves in the horn of the hoof wall (Figure 3-2), some of which travel into the deeper tissues of the hoof. So, the first way the hoof is loaded is by the forces of impact, which last for only the first 15 to 25 ms after initial contact (unless the hoof slips).

Once the hoof is firmly planted, the weight and the momentum of the body start acting on it in the form of forces transmitted down through the bones of the leg, through the pastern bones to the coffin bone within the hoof capsule. Total, or resultant, forces acting on the hoof have been measured using force plates embedded in a test trackway (e.g., by Merkens et al.[92] among others). After impact, the magnitude of the resultant force rises smoothly to peak at midstance.

At a medium trot, the peak force is approximately 1.2 times the animal's body weight and may exceed twice that at a fast gallop. It is this force acting on the leg that causes the fetlock to lower toward the ground, stretching the digital flexor tendons and suspensory ligament like large elastic bands. By stretching the bands, the fetlock acts as a shock absorber; otherwise, the weight of the animal would be applied to the leg in a brief instant that would set up even larger shock waves than hoof impact.

Hoof impact and the subsequent weight-bearing and locomotive forces are the two primary ways in which the hoof is loaded during each stance phase or step. Both types of loading can be altered or modified by a number of factors: the

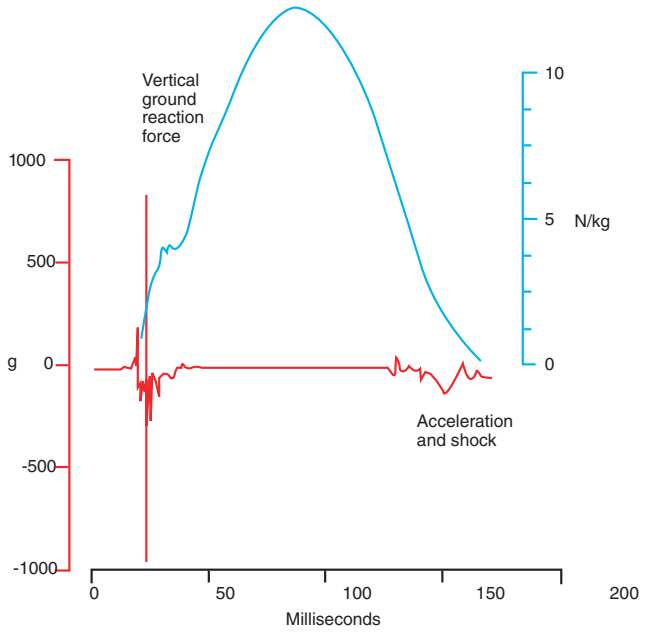

FIGURE 3-2 Accelerations of impact and breakover, and the forces of weight bearing acting on a forehoof during a single footfall at a trot. A force of 10 N/kg approximates to the animal's body weight. *(Force data courtesy Dr. Hilary Clayton; acceleration data courtesy Dr. J. Dickey.)*

speed of locomotion, the gait, the mass of the horse, and the firmness of the surface, for example. Also, whether the hoof is shod, the type of shoe, whether it has shoe pads, whether it has recently been trimmed, or whether it is being ridden or lunged all affect impact and force magnitudes.

If any of these modifying factors is changed, an indirect stimulus on the hoof is altered, in the terminology of Figure 3-1. Force and impact are themselves another level of indirect stimulus, because they are not directly sensed by cells of the foot. What then are the direct stimuli? In answering this question, the immediate effects of impact and force on the horn and living tissues of the hoof need to be discussed.

Mechanical Behavior of the Hoof

Impact sets up shock waves in the materials of the hoof, and forces induce stresses, strains, and wholesale changes in hoof shape (called *deformations*). The magnitudes of the shock, stress, strain, and deformation caused by known values of impact and force describe the mechanical behavior of the hoof (Figure 3-3).

What Are Shock Waves?

If a metal bar suspended from a string is tapped, it rings. The tap puts energy into the bar, which causes the molecules to oscillate back and forth in the bar without changing their relative position. At the surface of the bar, the oscillations vibrate the air, which we hear as sound. The molecules have a natural frequency that depends on the size of the bar and gives the pitch of the sound; this is how wind chimes work. Holding the bar, rather than suspending it, impedes, or damps, the oscillation and the ringing stops quickly.

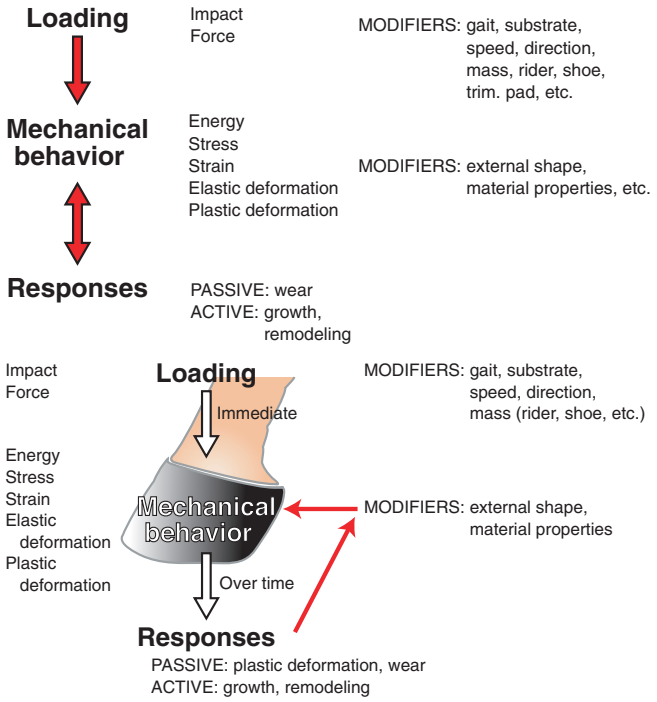

FIGURE 3-3 Flow chart illustrating the immediate induction of mechanical behavior in the hoof by external loading, and the biological response that occurs over time. Types of loading and mechanical behavior are listed **(A)** with the factors modifying them **(B)**. The triangle linking mechanical behavior, response, and modifiers of behavior is a classic feedback loop.

Vibrations at natural frequency are not shock waves, but tapping the bar causes one or two immediate oscillations at a much higher frequency and at a greater amplitude than the subsequent ones. The energy of the tap drives these initial oscillations, which spread from the place where the bar was tapped from molecule to adjacent molecule through the bar. These spreading oscillations are shock waves.

The potential danger of shock waves lies in their energy, which can disrupt the bonds between molecules and fracture the material. Each solid material has a characteristic energy at which its molecular bonds fail. Colloquially, this property is referred to as toughness. Glass and porcelain have a relatively low fracture energies (though both are quite strong) and they shatter easily under a light tap. The process of laminating and prestressing car windshields is designed to increase their toughness, so the impact of small stones does not break them. Steel is a tough material that tends to dent rather than shatter when hit.

Hoof Toughness. Hoof material is tough; it has to be, given the energy of repeated impacts it has to absorb. The characteristic energy required to fracture hoof material has been measured in the laboratory.[93,94] To determine how close the energy of impact shock waves comes to this threshold, we need to know the energy in the waves, and this has not yet been determined. What has been measured is the accelerations associated with them, which give an indication of how violently molecules in the material oscillate back and forth as the waves pass by.

At the University of Guelph, data were recently recorded from small accelerometers glued to the forehoof walls of five Standardbred trotters moving on a track at about three-quarters racing speed (J. Dickey et al., unpublished data). Impact accelerations averaged around 400 times the acceleration due to gravity (g) and peaked at more than 1000 g for some steps (at which point, we lost them because the accelerometer gauges only went to 1000 g). The waves lasted for only 1.5 to 2 ms but were clearly of high magnitude.

Hooves are naturally damped, which means they do not have an extended ring like a steel bar, and the waves die down soon after contact. At the end of the step, the renewed activity of acceleration shown in Figure 3-3 is due to the motion of the hoof at breakover.

We are working on calculating the energy associated with the impact shock waves to compare it with the energy the material can withstand. Clearly, hoof material can withstand the energy of the waves because hooves do not shatter on impact. In fact, hoof horn is laid down—specifically to increase its toughness—in a way that is very different from the construction of human fingernails (which are made of the same protein, keratin). Hoof material does fracture routinely at the bearing surface, but its structure forces the cracks to change direction repeatedly, so there is less danger of them running straight up the wall, or from outside to inside through the wall.[93] This feature may be the main reason that there are tubules in the hoof wall, and human fingernails (which would be useless for running on) do not have them. We exploit the toughness of hoof wall by driving nails through it to hold shoes.

Quarter Cracks. This discussion raises the question of why quarter cracks occur. The exact answer is different for every horse, but a number of factors are probably involved: the hardness of the surface the horse is worked on; the dryness of its hoof horn; whether the hooves are trimmed short or long; the angle of the toe, height of the heel, or thickness of frog and sole; whether the feed has the optimal nutrients for building keratin; and whether the horse itself is genetically capable of building horn of requisite toughness. Hoof pigmentation may be a factor, but all of the tests I have seen or done on the effects of horn stiffness, strength, and toughness speak against that. White horn does not seem inferior to dark horn.

Dissipation of Energy. There is also the question of where the energy in the shock waves goes. If all of it reached the skeleton, there would be a very real danger of fracturing bones. Some of the energy is absorbed in the wall itself. In addition, the soft tissues between the capsule and coffin bone (e.g., the laminar junction and solar dermis) probably are extremely important in making sure the high shock waves in the wall are somewhat attenuated by the time they reach the coffin bone. It is difficult to measure absorption of energy by these tissues; if the experiment has been done, I have not seen it.

Shock energy is probably of more concern in the front half of the hoof, where the wall has to be rigid for its weight-bearing function (see later in this section). Rigidity makes it less energy absorbent and more likely to transmit the wave. In the back half of the hoof, all of the soft-tissue structures are probably extremely important in absorbing the energy of the hoof's own impact with the ground. These structures include the frog, lateral cartilages underlying the heel bulbs,

and the digital pad or cushion between the cartilages and above the frog.

From a straightforward mechanical perspective, a large frog and prominent heels are likely to have a significant role in cushioning hoof impact. It makes intuitive sense that these relatively spongy structures (compared with the front half of the hoof) are good shock absorbers, but exactly how much energy they absorb has yet to be studied.

Some aspects of energy absorption in horse legs have been studied. By putting accelerometers on the long pastern bone (P1) and cannon bone, the attenuation (reduction) in shock by the joint cartilages separating these bones has been shown to be close to a factor of 10.[95] The attenuating capabilities of the laminar junction and frog are likely to be much higher.

Hoof Deformation

After the shock waves subside, the forces that begin to be applied to the hoof cause very different kinds of mechanical behavior to that of impact. From impact to midstance, force is progressively applied, causing the whole hoof to change shape or deform. Deformation is great enough to be visible but occurs too quickly to be easily seen. (Note that I am using *deformation* to mean the almost instantaneous shape change during one stance phase, not the long-term deformation in unkept or unbalanced hooves, which is a response to undesirable loading over time.)

Weight of the body pushing down on the coffin bone causes the bone to pull down on the hoof wall at the toe via the laminar junction. Under this force, the wall is dragged down and also moves backward because it is inclined to the vertical to start with, and the lower part of the wall may dish a little. At the coronet, the amount of motion may be 1 to 3 mm. This motion tends to push the quarters into the ground, but the ground pushes back, which forces the quarters to move outward rather than downward, causing them to flare. The heels are carried along with the quarters to some extent, but perhaps not as far. There is some evidence that, although the heels move outward, they actually move inward relative to the quarters; their exact motion has yet to be verified. The heels are also compressed in a vertical direction as they are pressed into the ground.

Heel Motion. It has long been thought that the heel motion is either due to compression of the frog pushing out on the bars or to the lowering of the short pastern bone (P2) pushing out on the lateral cartilages that form the heel bulbs. It now seems that the main contributor to heel motion is that the heels are carried along with the quarters as they flare. In other words, the motions of all of the parts of the wall—toe back and down, quarter and heel flaring—are caused by the *cone-like shape of the wall* itself, in response to the downward pull from the coffin bone suspended within it by the laminar junction. The heels are not pushed by a rise in internal pressure within the capsule (from frog or pastern movement) because the pressure in the capsule actually decreases as the quarters flare.[96]

Sole Motion. By midstance, the sole has also flattened (moved down) by a millimeter or so near its center, close to the point of the frog. It is easy to assume that the downward motion of the coffin bone simply pushes down on the sole, causing it to move, but the situation appears to be a little more complicated. The sole is a low dome, arching between the quarters on either side (and interrupted by the bars, lateral grooves, and frog in the rear half of the hoof). As the quarters flare, they pull outward on the edges of the sole, tending to flatten its domed shape.

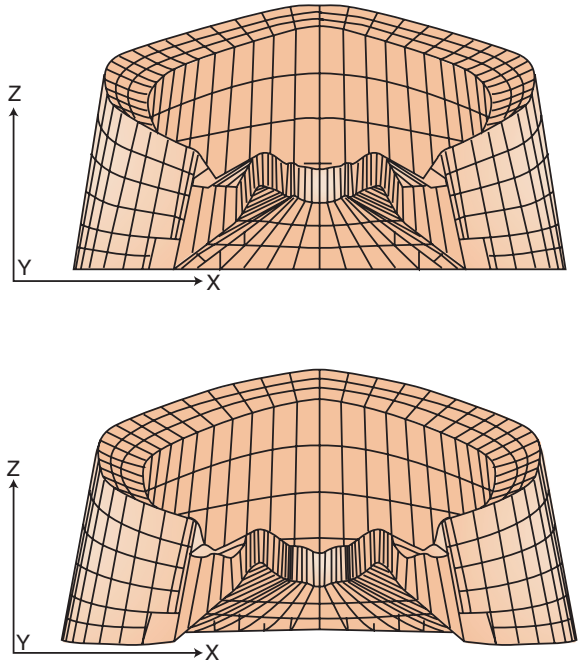

FIGURE 3-4 Rear view of a finite element model of the hoof capsule (minus the frog and internal structures). The left image shows the hoof capsule in an unloaded state and the right in a loaded state, with a force distributed over the area of contact of the laminar junction (mimicking the pull from the coffin bone). Even in the absence of the coffin bone, the sole moves downward when the model is loaded (compare the distance separating the white bars). *(Images courtesy Heather McClinchey.)*

At the toe, the coffin bone may press down on the sole to some extent (it tends to tilt down at the toe because of the pull of the deep digital flexor tendon on it), but the motion of the sole at the toe is minimal. Tilting of the coffin bone seems to actually lift its rear half away from the frog and sole, so it is not pressing down but pulling up on the sole for most of its length. The small amount of motion of the sole is not because it is being pushed down from above, but because it is being pulled out and down from the sides.

Evidence for this effect comes from computer modeling of the hoof by Heather McClinchey in my laboratory (using models similar to those described in Thomason et al.[97]). The most convincing model is one in which the coffin bone is not included within the capsule, yet the sole still flattens (Figure 3-4). Models that include the laminar junction and coffin bone show the tilting motion of the coffin bone quite clearly. Of course, there is a need to be cautious in accepting results of unverified computer models, so we have compared the output from them with data from experiments, such as measured deformations of the capsule and strains in the hoof wall.[97] The comparisons are sufficiently close for me to be confident in the descriptions above.

If we are prepared to take the descriptions of sole motion at face value, then we can speculate what happens on soft ground that pushes up past the bearing surface of the wall and exerts upward pressure directly on the sole and frog. Does this pressure prevent sole flattening and push the sole and frog up

against the coffin bone (and the navicular bone)? It appears not. In a model that was loaded evenly on the bearing border, sole, and frog, the sole flattened while there was still tension on the tissues between coffin bone and sole.

My interpretation is that the sole is like a drum skin that is attached to the wall at the white line. When you push on a taut drum skin, it resists your push—not because of the air pressure on the far side of it, but because it is stretched across the barrel of the drum and transfers the push to the edges of the barrel. In a similar manner, upward pressure on the sole is resisted at the white line primarily, and not generally by the sole being pushed up toward the coffin bone. This means that the white line has the important mechanical function of bracing the sole against the wall, in addition to providing a seal to prevent infection from entering the foot. A specialized tissue at the lower border of the coffin bone produces the horn of the white line, which effectively glues the wall and sole together as they meet.

It makes sense for the coffin bone not to press down on the sole over most of its area. For one reason, a blood vessel that is important in supplying the cells that make the white line runs around the lower edge of the coffin bone. Continuous pressure on it might obstruct the blood flow in it. For another, the sole grows down from a layer of cells on its inner surface. If those cells were continually under pressure, growth abnormalities of the sole would be likely.

A very different interpretation is given by Hood et al.[98] of how forces are transferred from the sole. They stood horses on hard surfaces and on soft sand, with an array of sensors under the hoof or the sand that measured pressure distribution. On sand, they found that pressure was distributed across most of the hoof's underside. They argued that the large pressures on the sole had to be transmitted upwards to the coffin bone.

My argument is that it is safer for the forces to go out to the wall, via the white line, and to the coffin bone via the laminar junction. McClinchey's computer models support this argument; but as with many questions in hoof biomechanics, the deciding experiments have not been done. Such experiments are difficult to perform on live horses because of their necessary invasiveness, and this is a limiting factor in answering many questions about hooves. Computer modeling and laboratory experiments on cadaver limbs help but are not always conclusive.

Resuming Unloaded Shape. By midstance, the hoof is maximally deformed for that step. The degree of deformation is dependent on the peak force at midstance, which varies from step to step and with all the factors that affect loading (see Figure 3-3). From midstance to breakover, deformation relaxes as the force subsides and the hoof begins to resume its unloaded shape. Some residual deformation as the hoof leaves the ground may cause momentary vibration in the soft tissues that are soon damped.

Elastic versus Plastic Deformations. Fully reversible deformations such as those described above are said to be *elastic*. Over time, the hoof may undergo irreversible changes in shape, such as flaring and dishing, which are related to its growth, but also to permanent deformation of the capsule. These are called *plastic* deformations and occur in most solid materials that are loaded heavily (past what is called the yield point for that material) or are loaded repeatedly until they fatigue and do not return to their original shape.

Stress and Strain

Accompanying the elastic deformations of the whole hoof are stresses and strains in all of the materials and tissues making up the hoof (see Figure 3-3). Stresses and strains occur naturally when a force is applied to any solid object.

Stress. *Stress* can be thought of as force dispersed through the material of the object. The bigger the cross-sectional area of the object is, the more material there is in the cross-section and the lower the stress is. Stress cannot be measured, only calculated from measurements of force and of the area over which it acts. Calculating stresses in the various materials of a complex shape like the hoof clearly is a challenge, but computer modeling of the kind described under Deformation is designed to do those calculations.

Strain. Strain is a measure of local deformation in small volumes of the material (think of the bars of hoof material described earlier being cut out and stretched in a machine). Strain can be measured because it is a change in the length of each side of the bar. The easiest way to measure strain on the hooves of a living horse is to glue on small, removable electric gauges that stretch and shorten with the material of the wall.

I have spent much of the past 10 years recording hoof wall strains under different conditions of movement, with the aid of my laboratory crew and colleagues.[99-102] They are enjoyable experiments, because you work with live horses but cause them no damage or distress, apart from a few minutes of boredom as they have to stand still while the glue cures. At the end of the experiment, a couple of strokes of a rasp removes the gauges.

As may be expected, all of the factors that modify hoof loading (see Figure 3-3) also affect strains on the surface of the hoof wall. Wall strains increase with speed and the addition of a rider. Somewhat surprisingly, they do not appear to vary significantly when the same hooves are shod or unshod (without any trimming in between). The largest change comes with a change in direction: on a turn, the quarter on the inside of the turn sees strains 40% higher, and the opposite one 40% lower, than when on a straight line. This difference reflects more of a redistribution of load from one quarter to another than a change in total load. Small but measurable redistributions in strain occur when a rider changes from posting to sitting at a trot. Trimming causes small changes in overall strain, more noticeably if the toe angle is changed during the trim.

Hoof Shape and Mechanical Behavior. These findings demonstrate that the shape of the hoof influences its own mechanical behavior (see Figure 3-3). That is, changes in hoof shape cause changes in the distribution of stress and strain within its tissues, even if the external load does not change. How the hoof responds appears to be controlled by a feedback loop, illustrated by the triangle of interaction among mechanical behavior, response, and factors modifying mechanical behavior. At the heart of this triangle are the steps in Figure 3-1 we know little about: the direct stimulus, how it is sensed, and the cellular response to it.

Stimulus for Response

The next question is which of the variables describing mechanical behavior are candidates be a direct mechanical stimulus to cells in the foot. The most likely candidates are the shock

waves and strains, because both involve movement within materials and both vary with location through the hoof and with time.

SCIENCE AND EMPIRICISM

It should be clear from the discussion so far that there is a great deal left to discover about the mechanics of the hoof and the mechanisms of its ability to respond to loading. Much of what we know about the hoof has been discovered empirically, by many generations of horse experts using their eyes. The process is simply to make observations (step 4 in Figure 3-1), or to apply an indirect stimulus (step 1) to an individual animal and observe the response (step 4).

This approach has been valuable, and for many years it was the only one available, but it has its drawbacks. The result may vary with the animal, and the interpretation with the observer. (A farrier once joked to me, half seriously, that if you want six opinions on the best way to shoe a problem horse, just ask five farriers!) Valuable information often is lost from generation to generation, and there is little or no understanding of the mechanisms by which the response occurs, or whether the response is adaptive, neutral, or counterproductive.

Scientific Approach: Value and Limitations

The scientific method is another means of information gathering that should eventually help increase our understanding of hoof biomechanics and responses. By performing the same experiment on several animals under the same conditions (with suitable controls), the results are less open to subjective interpretation, and variation among individuals can be measured. Laboratory experiments can be devised to examine the mechanism of response (steps 1a to 3 in Figure 3-1), which will be a considerable advance.

Hoof science has its own drawback, however. The problem is so complicated (because so many factors are involved) and so few experiments have been done relative to the number needed that it will be many years yet before major advances are made in our understanding of the concepts outlined in Figures 3-1 and 3-2. Basic biomechanics has received some attention, and the effects of varying loads on the biomechanical behavior of the hoof are slowly beginning to be understood. But issues of interest to owners of afflicted horses, such as the effect of hoof loading in the etiology of navicular syndrome, are still incompletely understood. And the area of real interest to me—the triangle of interaction among mechanical behavior, modifying factors, and biological response (see Figure 3-3)—is only just being brought into focus under the experimental microscope. It will be some time before real advances are made in that area.

Putting Cure-All Solutions in Context

As a sidebar to the general discussion, we now have a framework in which to understand the position of cure-all products that appear on the market regularly. I am talking specifically about products that affect the mechanics of the hoof, primarily shoes, wedges, pads, and so on. Other products, such as topical solutions that soften or harden hoof horn and thereby modify its mechanical behavior, are included at the periphery of the discussion. Let me be very clear that I am not going to make any kind of statement that all of these products, or individual specific products, have great value, or none at all. I just want to provide background information that will explain why it is difficult to critically and scientifically evaluate the worth of many of those products just yet.

Testimonial versus Evidence

The application of most products is an indirect stimulus, and the observed result is just that. Some products do produce remarkable observed results on individual horses, such as recovery of hoof balance and reduction in lameness. But putting the product on some horses and reporting testimonials of remarkable instances of cure/enhancement and so on is empiricism, not science.

To be scientific, a number of horses (say, 10) with the same initial lameness problem would have to receive exactly the same treatment, while all other conditions (e.g., stabling, feed) were kept constant. For controls, another 10 lame horses would remain untreated, and an initially healthy group of 10 horses would also receive the treatment. Observed results would be compared among groups. If all of the 10 treated horses recovered, with no change in either control group, you would have pretty unequivocal evidence in favor of the product's benefits.

But what if, as is more likely to happen, 6 horses recovered in the treated lame group, 4 horses recovered in the untreated lame group, and 3 horses in the healthy group went lame? This result is not quite so clear and is harder to interpret, but it certainly does not give overwhelming support for use of the product. In empirical applications of such products, you rarely hear about the horses that do not respond beneficially, and usually there are no proper controls. To be fair to the makers of horse shoes and related items, analyzing the efficacy of their products scientifically is hampered because of the relatively little scientific data we have on the normal workings of the hoof.

Where Next?

If we continue to do basic science with the aim of understanding lines 1a, 2, and 3 in Figure 3-1 and the triangle in Figure 3-3, then we may be able to get to a point where we can design products on the basis of being able to predict the response to the direct stimuli they will cause. I suspect that we are many years from that point, however. Until that time, we cannot be sure that the hoof really is a smart structure, but it will certainly remain smarter than us.

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4

FUNCTIONAL ANATOMY OF THE FOOT

JAMES R. ROONEY

This chapter introduces the reader to the mechanics of the foot, with and without shoes, together with consideration of some features and lesions that have mechanical components in their pathogenesis. The basic mechanics are not difficult to understand, and the explanation here is as nonmathematical as possible, although a few mathematical terms are introduced, such as *vector* and *moment*.

STATIC AND DYNAMIC EQUILIBRIUM

The statics of the digit involve how the digit retains or stays in its position when the horse is standing still. In fact, no horse is ever absolutely standing still, but for the purposes of discussion, the horse is considered to be standing still.

Linear Forces

It is necessary to have a good understanding of linear forces before discussing turning, twisting, and rotating forces. The load exerted on one front leg (either one) or a hindleg by a standing horse amounts to about 30% of the total body weight for each foreleg and 20% for each hindleg. The load, in this case, is mass multiplied by the acceleration of gravity and thus is a vertical force pulling downward on the upper end of the scapula, or thereabouts. For the hindleg, the downward pull is exerted in the general vicinity of the hip joint.

Force and *weight* are synonymous terms and have two parts, mass and the acceleration acting on that mass. The term *weight* is usually used when the acceleration is that of gravity acting downward on the mass. The term *force* is used for situations in which the acceleration is produced by an act on a given mass. This force follows the leg all the way down to the ground (Figure 4-1). If the foot is placed on a slippery surface and the force comes down the leg, the foot will slide forward on the surface. While that is quite clear and quite simple, we learn very little. To learn more, the surface should be made a bit rough (so that the foot does not slip) and then some calculations with vectors can be made, which are explained later.

A basic principle of mechanics is that every force, to be in equilibrium, must have an equal and opposite force. When a person leans against a wall with one hand, he exerts a force on the wall (Figure 4-2). The wall pushes against the person's hand with an equal and opposite force. The person and the wall are in *equilibrium*. In the same way, the force coming down through the horse's foot into the ground is in equilibrium with an equal and opposite force being exerted on the foot by the ground. If the ground is mud or soft sand, the horse may sink in until the surface material compacts enough or a deeper, firm layer is reached for equilibrium to be achieved (Figure 4-3).

In other words, the force coming down the leg is trying to push the foot forward and push the fetlock down to the ground. The force coming down the leg is named R', and the equal and opposite force of the surface pressing upward to resist R' is R. In Figure 4-2, for example, two arrows could be added to the person leaning against the wall, one on his arm and hand pressing against the wall and the other on the wall pressing against the hand.

This is where *vectors* come in. Another arrow can be added to the figure that will show the direction in which the force is acting. The length of this arrow indicates the amount, or quantity, of the force. However, for this discussion, the amount of force is not discussed.

The two forces, R and R', are resultant forces, the "result" of adding together two other forces: H and H' and F and F'. The force H' is that part of R' that slides the foot forward or tries to, whereas H is that part of the resisting force, R, of the ground that stops the foot from sliding forward. H of the ground is friction, which is absent, or nearly so, when the surface is very slippery. H' is present, however, and that is why the foot slides forward. F' is that part of R' that tries to push the fetlock down to the ground, and F is the ground reaction force pushing up against F'.

Another way to explain the mechanics of the horse standing still is illustrated in Figure 4-4. In this figure, I am trying to pull the weight, W, along the floor with a rope in the direction of the straight, larger arrow, but it is too heavy for me to move. (The length of the large arrow is the measure of the weight, W.) I enlist someone else to help me, who pulls a rope along the direction of the arrow to the right while I pull on another rope in the direction of the arrow to the left. The result of my pulling one way and the other person pulling another is the large arrow in the middle, which is all there is to vector addition. Vector addition is what I did above with the Rs, Hs, and Fs.

Moments

Moment is a turning force such as that exerted to twist the lid off a jar, turn a crank, or throw a ball. A moment acting around any given joint will always be the product of a linear force acting over a distance—a *moment arm*—from the center of rotation of that joint (Figure 4-5).

The mass (W) at one end of the seesaw is acted on by gravity in a straight line (linear) to the ground. It is attached to the center of the triangle at a distance (s), which is the moment arm of W acting around the center of rotation at the tip of the triangle. The amount of the moment (turning force) is then W multiplied by the distance s, or Ws.

At the other end of the seesaw is the weight (P), and it is a distance (l) from the center of rotation. If W is equal to P and s

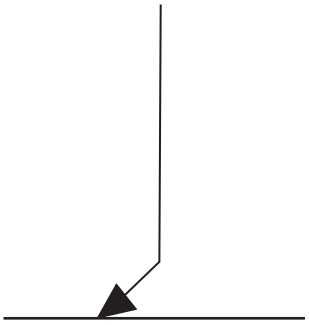

FIGURE 4-1 Illustration of force. The heavy line with the arrowhead indicates the force coming down the leg.

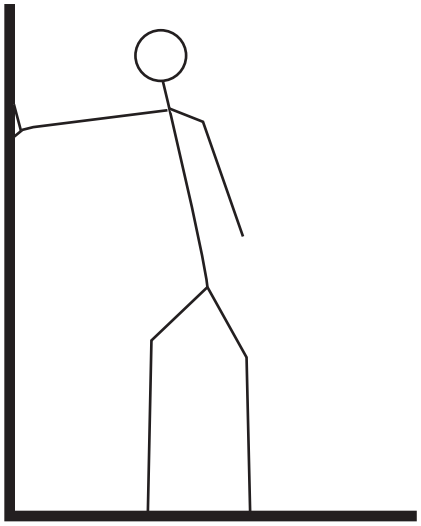

FIGURE 4-2 Illustration of equilibrium. When a person leans against a wall with one hand, they exert a force on the wall. The wall pushes against the person's hand with an equal and opposite force.

is equal to *l*, there is equilibrium, specifically *equilibrium of moments*. The moment *Ws* is trying to produce a counterclockwise rotation of the seesaw, whereas *Pl* wants to rotate it in a clockwise direction.

What if *P* is greater than *W*? How can there be equilibrium? What is needed is for *Pl* to equal *Ws*? If *P* is greater than *W*, for equilibrium *s* must be longer, so that the two products are equal.

This explanation is a vital, indispensable tool in understanding how the digit works, whether barefoot, shod, with bar shoes, or with angles. The reader should understand equilibrium of moments before continuing the rest of this chapter.

Moments of the Digit

Any displacement, or movement, of the digit always involves moments; similarly, the resisting of movement, stopping displacements, always involves moments (Figure 4-6).

This discussion of moments of the digits is concerned only with those linear forces that are vertical, or perpendicular, to the ground (*F*). *H*, or frictional force, and *R*, resultant force,

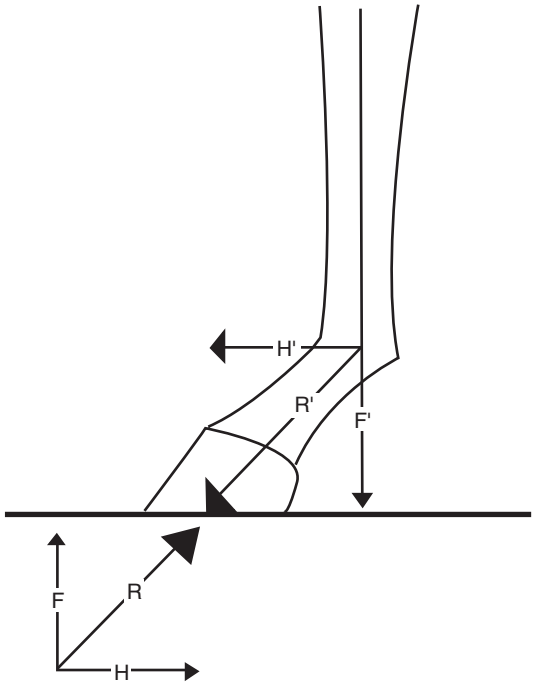

FIGURE 4-3 Illustration of the mechanics of the horse standing still.

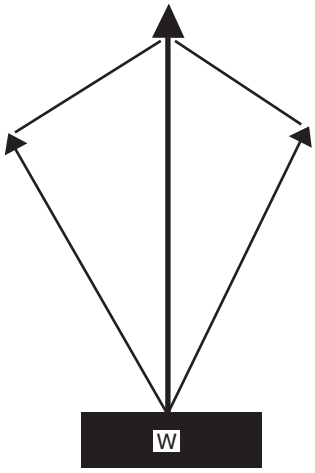

FIGURE 4-4 The mechanics of the horse standing still. *W*, Weight.

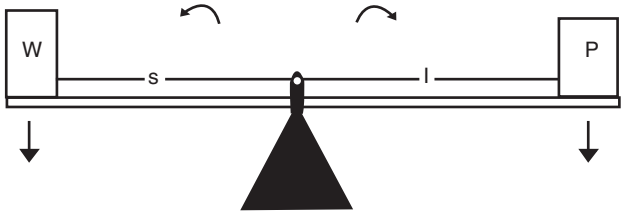

FIGURE 4-5 Moment arm is the product of a linear force acting over a distance. *W*, mass of one object; *P*, weight of second object; *s*, distance; *l*, distance.

are important, but here we shall look at the moments of the digit from the standpoint of one linear force only, *F*, the ground reaction force. The effect of *F* is to push the hoof upward, palmar-flexing (foreleg) or plantar-flexing (hindleg) the coffin joint and dorsiflexing the fetlock joint (Figure 4-7).

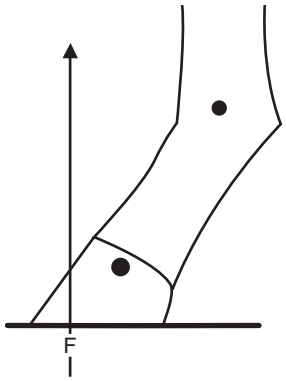

FIGURE 4-6 Any displacement, or movement, of the digit always involves moments; similarly, the resisting of movement, stopping displacements, always involves moments. *F,* Linear force.

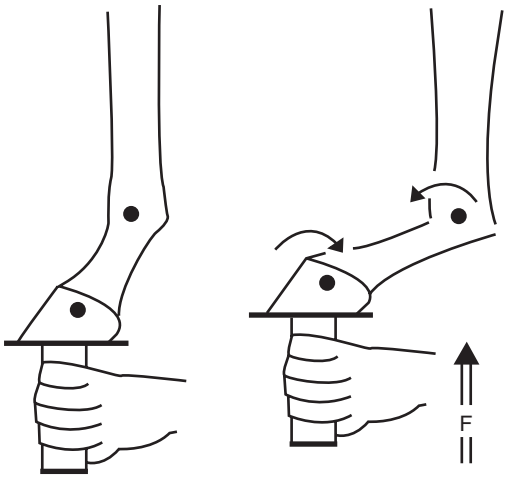

FIGURE 4-7 The effect of *F* is to push the hoof upward, palmar-flexing (foreleg) or plantar-flexing (hindleg) the coffin joint and dorsiflexing the fetlock joint. *F,* Linear force.

The use of the arrow, *F,* to represent the force causing joint movements is quite reasonable and simplifies talking about those joint movements.

Caveats

The pastern joint has been left out of this discussion because in the standing position and whenever the leg is experiencing load, the pastern joint is close-packed and stationary. Things can and do go wrong with and around the pastern joint, but that is not germane to this discussion of normal loads and movements.

The terms *extension* and *flexion* are not used here to describe joint movement. Dorsiflexion of the fetlock is what some call *extension*, whereas palmar (or plantar) flexion is what some call *flexion*.

A center of rotation is also used for each joint. There really is no such center, since joint surfaces roll and slide on each other, so that there are a number of instantaneous centers of rotation, forming a *locus* of joint movement rather than a single axis or axle. For our purposes, it is easier and nearly as accurate to use a single, compromise center of rotation.

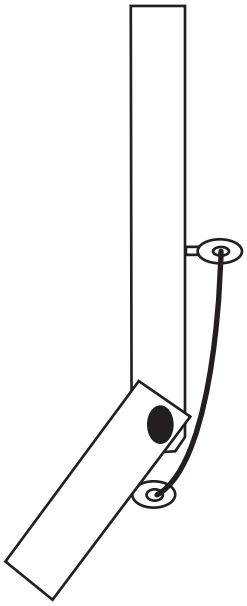

FIGURE 4-8 The fetlock joint is moving counterclockwise (dorsiflexing) and a force, or moment, is needed that acts in the clockwise direction (palmar-flexing) in opposition to that dorsiflexion. The anatomy of the fetlock area accomplishes that through the suspensory tendon, the deep flexor tendon, and the superficial flexor tendon.

Note, too, that there can only be one center of rotation for any given system, that is, only one center in the distal end of the metacarpal (metatarsal) bone for the fetlock system and only one center in the distal end of the middle (second) phalanx for the hoof system.

Fetlock Joint Moments

The coffin and fetlock joints move because of the ground reaction force. Something is needed to stop those movements—something like the brakes on a car or a bike—and it needs to operate smoothly without sudden stops and jerkiness (which could be destructive).

The fetlock joint is moving counterclockwise (dorsiflexing) and a force moment is needed that acts in the clockwise direction (palmar-flexing) in opposition to that dorsiflexion. The anatomy of the fetlock area accomplishes that through the suspensory tendon, the deep flexor tendon, and the superficial flexor tendon (Figure 4-8).

You may have caught a mistake in this description. For the digit, I said the fetlock was moving counterclockwise, and so the resistance at the fetlock by the tendons had to be clockwise. However, the legend for this wooden model in Figure 4-8 says just the opposite. This was done on purpose to help you always maintain a constant point of view when carrying out these kinds of studies. If the instructions said to move the upper piece of wood in Figure 4-8 to dorsiflex the joint, then there would have been counterclockwise movement at the joint, and the elastic cord/tendon resistance would act in a clockwise direction. The direction of movement of a joint (clockwise versus counterclockwise) is determined by looking at the movement of the upper of the two bones (or sticks of wood). Whatever method is used to determine direction of movement, consistency is the key. This same rule of consis-

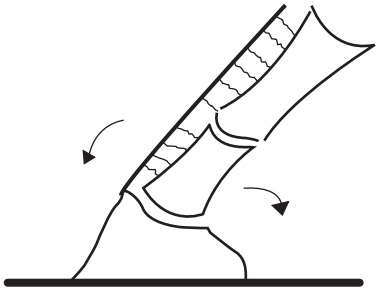

FIGURE 4-9 The coffin joint moves in a clockwise direction (second phalanx moving on third phalanx); therefore, there must be a counterclockwise resisting elastic band, which is the common extensor tendon.

tency applies using numbers for force or acceleration or any other parameters. An illustration of this rule would be mixing kilograms and pounds in the same equation and coming up with nonsense.

Coffin Joint Moments

The coffin joint moves in a clockwise direction (second phalanx moving on third phalanx); therefore, there must be a counterclockwise resisting elastic band. The common extensor tendon and extensor branches of the suspensory tendon could act at the coffin joint like the elastic band in Figure 4-4. But the common extensor tendon is firmly attached to the periosteum on the dorsal surfaces of the proximal and middle phalanges. Because of this attachment, the tendon can and does act as a strong, passive, counterclockwise ligament resisting the clockwise rotation of the coffin joint (Figure 4-9).

Resistance has been used to indicate the moment acting opposite to the direction of joint movement. Since we are talking about statics, it might be better to use the term *equilibrium.* The tendons and ligaments certainly do resist the rotation of the joints, but in the static situation, the two moments equal each other, and the joint is in equilibrium.

When the horse is moving, equilibrium is still essential. At each given instant of rotation, there is a balance between the force causing rotation and the force resisting that rotation. To move to the next instant, the rotatory moment exceeds the resisting moment for an instant; then the latter catches up and so on. This is *dynamic equilibrium* as opposed to the *static equilibrium* of the quietly standing horse. Note, however, that the horse is never standing absolutely still but is always swaying a little bit, just as humans do when standing. Static equilibrium is really an abstraction used to help in understanding the dynamic situation.

The model in Figure 4-10 can help in understanding the basic mechanics of the foot and can also be of considerable help when changes of hoof angle and the effects on tendons are discussed. Wood, screw eyes, elastic strips, and some rods for the joint axles are used to build the model in Figure 4-10, and Figure 4-11 shows how the joints can be made.

Third-Dimension View

Up to this point, we have been considering the digit in two dimensions from the side or lateral view. No joint, however, moves in two dimensions. All joints move in three spatial dimensions and the fourth dimension: time. Now, we will

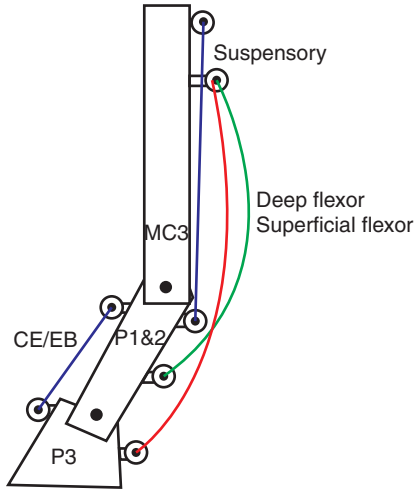

FIGURE 4-10 Model of the basic mechanics of the foot**.** *Mc3,* Metacarpal 3; *P1,* first phalanx; *P2,* second phalanx; *P3,* third phalanx; *CE,* force in common extensor tendon; *EB,* force in extensor branches of the suspensory ligament.

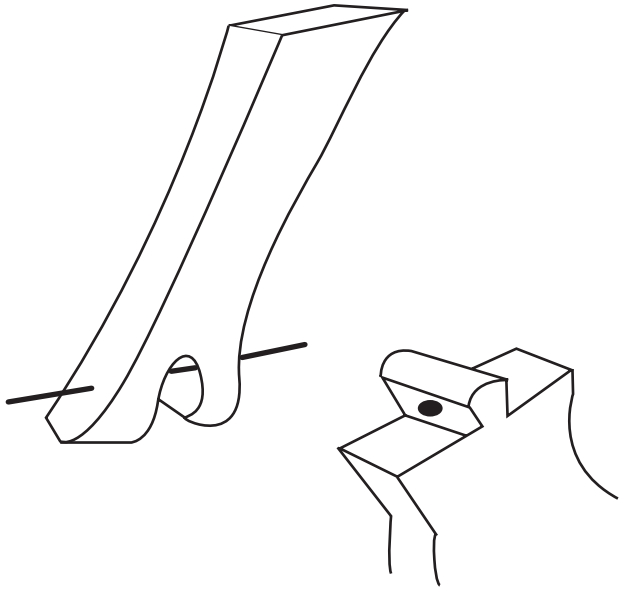

FIGURE 4-11 Illustration of how the joints can be made for the model in Figure 4-10.

look at the digit from the front (head-on, frontal) view, or the third dimension.

There is little to note from the static standpoint; the collateral ligaments hold the joints together (among other things). The dynamics are more interesting. As a joint *rotates* in the lateral view, it *spins* in the frontal view. Immediately after the foot impacts the ground, the fetlock begins to dorsiflex as the coffin joint palmar-flexes. Both of these movements are designated as rotations. As the fetlock rotates, the proximal phalanx also spins around its own long axis, from medial to lateral (Figure 4-12).

The amount of spin is only about 7 to 8 degrees and thus not readily seen except in high-speed film or video. This small amount of spin, however, is very important in the patho-

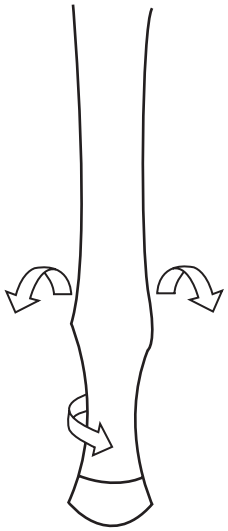

FIGURE 4-12 Illustration of rotations. As the fetlock rotates, the proximal phalanx also spins around its own long axis, from medial to lateral.

genesis of arthrosis of the fetlock joint and fractures of metacarpal 3, metatarsal 3, or the proximal (first) phalanx. At the end of support (still using the frontal view), the fetlock joint begins opening, and the proximal phalanx is now spinning from lateral back to medial, so that the entire digit is spinning in the same sense. This motion provides the normal, slight swinging out (paddling) of the foot as it leaves the ground, which is quite evident to the naked eye.

As the foot swings forward through the air and straightens out in protraction, the digit seems, in slow motion, to be doing a hula dance. The proximal phalanx is spinning once more from medial to lateral, the middle phalanx is making a similar but smaller spin around its long axis, and finally, the distal phalanx is making a very small spin around its long axis—all from medial to lateral.

These spins occur because of the three-dimensional geometry of the joint surfaces. Although the geometry of the joint surfaces is beyond the scope of this chapter, a demonstration is in order because this type of rotation plus spin is routine in joints of all species, including human.

To illustrate this geometry, lay a strip of fabric on the floor or draw a straight chalk line. Place your right foot along the line near the middle. Now shift your weight to the left foot, close your eyes, and swing the right foot forward and hold. Open your eyes and see that the right foot is outside the line. Now swing it back to the ground and continue backward until it leaves the ground. Note that the foot is once more outside the line. Clearly, what has happened is that your leg has swung in an arc from medial to lateral (inside to outside) as you moved the foot forward and swung back on that arc from to lateral to medial and finally, lateral again as your foot leaves the surface behind your body. This "swing" is the result of spinning of the femur around its long axis (mediated by the coxofemoral joint). This demonstration can be biased consciously, but if you do it in a relaxed manner with your eyes closed, the spin will be induced by the geometry of your hip joint. Most horse joints do not have the freedom of our hip joint and cannot bias the spinning of the digital joints.

THE SHAPE OF THE HOOF

The hoof of the horse evolved over about 50 million years, giving a selective advantage to equids in the environment in which they lived. It is apparent that the hooves of modern equids conform to a basic pattern, which is an optimal, or nearly so, pattern. There are variations of that basic pattern, however, and those variations can be understood as a function of the forces exerted on the hoof and the surfaces upon which the animals are habitually moving and standing. The shape of the hoof has been considered by several authors.[1]

Surfaces

The feral horse of the American West, the feral horses (brumbies) of Australia, and the zebra experience a variety of surfaces: hard and inelastic with variably loose, gritty surfaces, sandy desert, and packed sod. The hard, inelastic surface is not unlike a harness racetrack.

Barefoot domestic horses can experience a variety of surfaces as well, including sod of varying degrees of compaction and elasticity. The feral horses (so-called ponies) of the barrier islands of the East coast of the United States generally experience soft, marshy footing, but those conditions change during droughts (see later).

To study the interaction of the hoof and the surface, the different types of soil and surface conditions have been broadly categorized. The basic material is soil, which is disintegrated rock. The physical character of the soil and surface is a complex function of soil, organic matter (including plant roots), water, and grass cover. A more complete discussion of the soil and surface can be found on the website under *Optimum Athletic Surfaces for Horses*.[1]

Wear and Friction

The wearing of the bearing edge of the hoof wall occurs because of friction between the bearing edge and the ground surface.

There are three kinds of friction: static, sliding, and rolling. The equation for static and sliding friction is

$$H = \mu F$$

where H is friction and F is the vertical ground reaction force, which is the weight of the horse being borne by a given foot, static or dynamic. The coefficient of friction, μ, is a measure of the roughness or stickiness of the ground surface and can only be determined by experiment. Sliding friction is somewhat less than static friction. Rolling friction is the least of all, with a slightly different equation incorporating the radius of the rolling.

Wear is a function of friction, and friction (H) is a function of the weight of the horse (F) on the foot and the roughness of the surface (μ). Obviously, frictional wearing is increased as the weight of the horse plus rider, or a draft force, increases, as the roughness or abrasiveness of the surface increases, or all of these.

At the slow walk, the hoof usually contacts the surface over the whole bearing edge of the hoof wall—flat-footed. The entire bearing edge experiences primarily static friction with a variable amount of sliding, depending on the surface. A hard, dry surface allows some sliding, whereas a moist, elastic surface does not. In any case, the amount of

sliding friction is small compared to the amount of static friction.

As the walk speeds up or the horse shifts to a faster gait, sliding friction becomes more important. As the animal moves faster, the heel of the hoof normally impacts first and is subjected to a brief period of rolling friction as the quarters and finally the toe portions of the bearing edge come successively, or serially, onto the surface (Figure 4-13).

The frictional wearing, sliding and static, increases as each succeeding segment of the hoof reaches the surface because the vertical force of the body on the foot increases from the instant of impact until midsupport and decreases again until lift-off (Figure 4-14).

An analogy is sanding a board with a sanding block. It is well-known to carpenters that without due care, the sanding process can result in an unwanted beveling of the surface, as shown in Figure 4-15. More downward (vertical) pressure (force) is exerted during the middle of the sanding stroke than at the beginning and end. If the sanded board is inverted, it illustrates the situation of the hoof on the surface. The stationary surface does the "sanding" of the moving hoof rather than the moving block doing the sanding on the stationary board. This is an imperfect analogy but serves to make the point that the wearing of the hoof wall bearing surface is primarily a function of the varying loading of the foot during movement.

As the vertical loading of the hoof increases during the first half of support, the hoof wall itself will be compressed by the load, contributing to this caterpillar tread-like contact of the bearing edge of the wall with the surface.

The result of this wearing is well-known and shown in Figures 4-16 and 4-17. It is important to note, at this point, that the concavity seen in the lateral view is only seen when the foot is not bearing dynamic weight, which is about 1.8 times greater than static weight. When the foot is on the ground and experiencing dynamic loading, the entire bearing edge of the hoof wall is in contact with the surface. If this

FIGURE 4-13 Effects of sliding friction on the hoof.

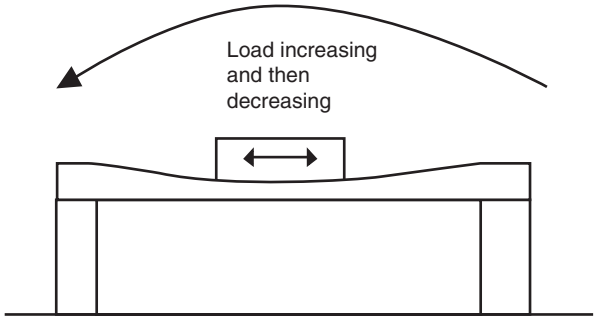

FIGURE 4-15 Beveling of the surface of a board with sanding.

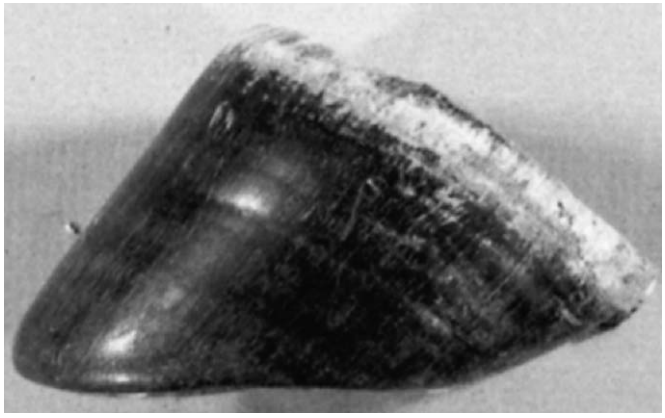

FIGURE 4-16 The result of wearing by vertical loading of the hoof.

FIGURE 4-17 The result of wearing by vertical loading of the hoof.

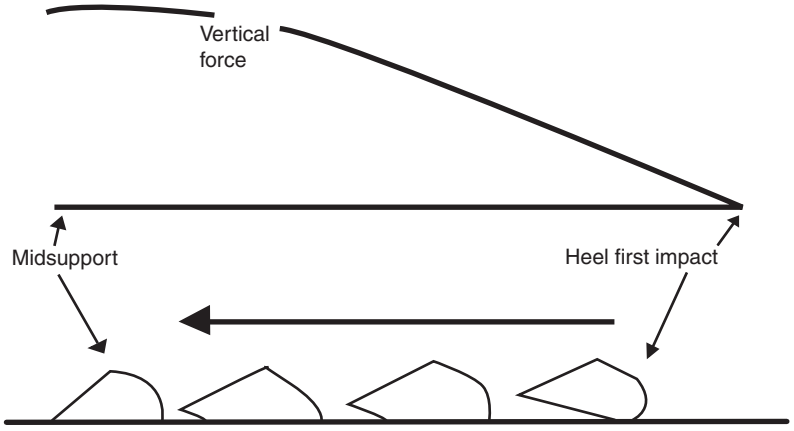

FIGURE 4-14 Frictional wearing of the hoof.

FIGURE 4-18 Imprint of a bare foot on a firm surface with a loose, sandy layer.

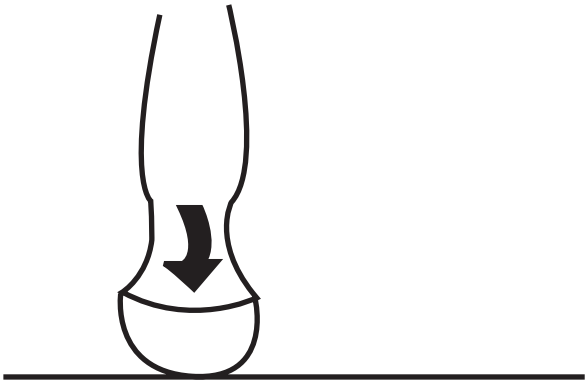

FIGURE 4-19 Wearing of the toe by rolling on the surface at lift-off. As the hoof rolls over the toe during lift-off (breakover), the bearing edge comes off the surface last in the center of the toe, so that the central part of the toe is rolling and sliding longer than the more abaxial part of the toe.

were not true, the wall would not wear the way it does. The same is generally true for the concavity at the toe, but the rounding off there is usually still evident, even when the foot is loaded, because the cause of the wear at the toe is somewhat different.

Figure 4-18 shows the imprint of a bare foot on a firm surface with a loose, sandy layer, such as that found on a harness horse training track. The heels have cut down into the loose surface material, whereas that material is compacted beneath the bearing edge of the quarters. The rolling over at the toe during lift-off has scooped out the loose dirt and so obliterated the compaction of the surface, which had continued from the quarters around to the toe before lift-off occurred.

It is clear that there is greater wearing of the quarters and the toe than of the heels. The wearing of the toe is easily explained by the rolling on the surface at lift-off. As the hoof rolls over the toe during lift-off (breakover, see later), the bearing edge comes off the surface last in the center of the toe, so that the central part of the toe is rolling and sliding longer than the more abaxial part of the toe (Figure 4-19). Also, as the

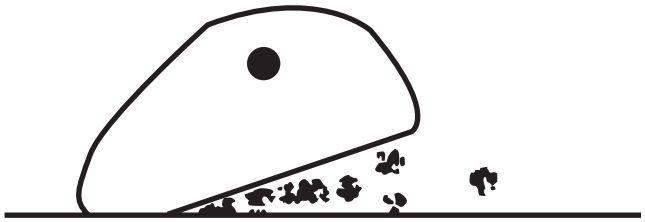

FIGURE 4-20 Wearing of the toe by rolling on the surface at lift-off. As the toe rolls, it pushes loose material backward into a mound and rolls through and over this mound.

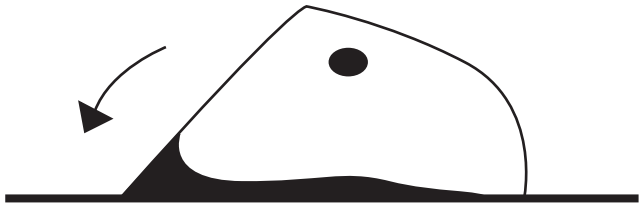

FIGURE 4-21 Wearing of the toe automatically allows the toe to "drop down."

toe rolls, it pushes loose material backward into a mound and rolls through and over this mound (Figure 4-20). This mounding of material occurs with the shod foot as well as the bare foot. It is this material that is responsible for the ridge of thickening on the sole just in front of the point of the frog. The thickening is essentially callus, which is increased stratum corneum of the sole epithelium in response to the wear induced by the mound of grit.

The inner and outer edges of the hoof wall are rounded off because of the gritty surface material through which the wall moves as it impacts and slides into the surface. The wearing of the toe automatically allows the toe to "drop down" as shown in Figure 4-21, the black representing the hoof material worn away, giving the larger hoof angle characteristic of the feral hoof on rough, abrasive surfaces. Ovnicek[2] and Jackson[3] found the hoof angles, fore and hind, to be about 50 to 60 degrees for feral horses, and my measurements of bare foot domestic horses, zebras, and one onager were the same.

These wearing effects on an inelastic, gritty surface are qualitatively the same but quantitatively greater on a predominantly sandy or loose sandy loam (such as the cushion of a dirt racetrack).

Barefoot horses on less abrasive surface conditions, such as pastures in the eastern part of the United States, develop the concave wearing of the quarters, though to a lesser extent than on a gritty, abrasive surface, and there may be little or no wearing of the toe. There are several reasons:

1. On a hard, dry pasture, the grass cover provides a smaller coefficient of friction.
2. There is less tendency to mound up gritty material behind the rolling toe.
3. The toe can rotate into softer moist surfaces.

Hood et al.[4,5] observed that the highest pressure contact (force) was at both heels and either side of the toe when barefoot horses were first taken from pasture to stand on a rubber-covered, pressure-sensitive mat. This is not inconsistent with

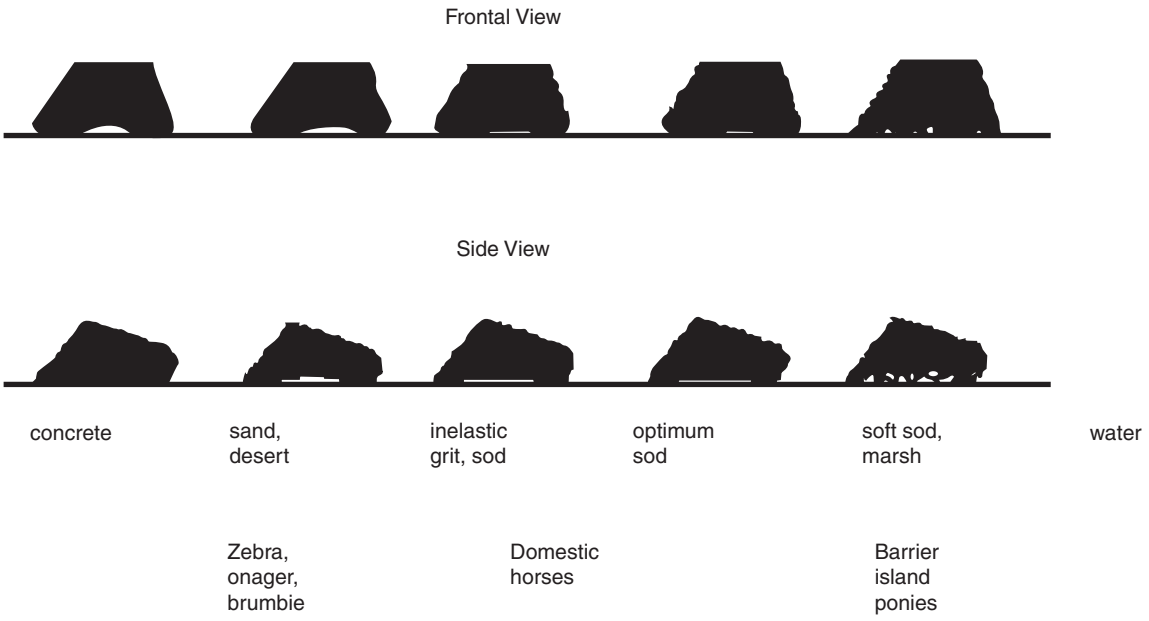

FIGURE 4-22 Summary of the shape of the hoof with specific surface conditions.

what I have described thus far, since the horses were standing still, not moving, and experiencing dynamic loading of the foot (as already noted, the dynamic load is about 1.8 times the static load).

Hood et al.[4,5] also noted that the concavity of the quarters disappeared if barefoot horses were moved to a concrete surface for seven days and that there was more contact of the sole with the surface after those seven days. This is similar to findings by Ovnicek et al.[2] in the foot, which had adapted to shale and granite surfaces. Ovnicek's figure shows the concavity of the quarters to be expected with the loose surface material on natural shale and granite as opposed to smooth concrete. The horses on concrete were no doubt standing or walking and would, therefore, not be experiencing the heel-to-toe impact necessary for wearing of the quarter. These horses did, however, wear the toe as would expected even at walking speed with the toe rolling on the concrete. The increased area of sole contact with the surface is readily explained by the rapid wearing away of the hoof wall, bringing the sole down to the concrete surface.

With the generally marshy, boggy surfaces experienced by barrier island horses and other horses during prolonged wet periods, the hoof is softer, is more flexible, and tends to flatten out. The bearing edge of the hoof wall does not wear at an appreciable rate and breaks up irregularly once it has extended sufficiently beyond the level of the sole. As such an animal moves onto a harder surface, or the surface becomes harder, the wall becomes drier, and the heel/toe wearing appears to the degree appropriate for the roughness of the surface. This wearing has been observed in Assateague Island horses over a number of years: the broken-up feet of wet years are replaced by quarter/toe wearing in drier years. Figure 4-22 summarizes the shape of the hoof with specific surface conditions, with the understanding that there is a continuum of change among the different conditions.

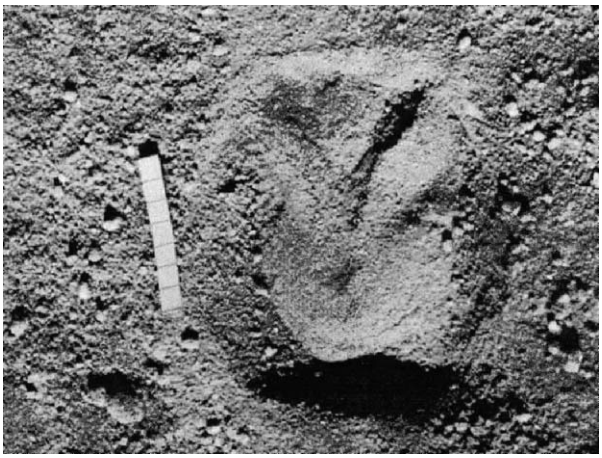

FIGURE 4-23 The imprint of frog and sole on the surface occurs after impact as the vertical force on the foot approaches maximum at midsupport.

Sole Pressure and Weight Bearing

It is generally recognized that the initial impact of the bare foot with the surface and the major weight bearing of the foot is on the bearing edge of the hoof wall, including the bars. The imprint of frog and sole on the surface (given that there is sufficient loose material on the surface to permit such an imprint) occurs after impact as the vertical force on the foot approaches maximum at midsupport (Figure 4-23).

No doubt, the frog and sole can share the weight bearing once the bearing edge of the hoof wall is fully loaded, particularly in marshy, sandy, or sandy loam conditions. Loose material on the surface tends to pile up under the hoof as the bearing edge plows down through it. There certainly can and will be some frictional wearing of the sole and frog under these conditions, with compensatory thickening

of the cornified epidermis (callus) as a result. Such thickening does not imply, however, that the sole and frog are primary or major weight-bearing structures, as is sometimes claimed.

Digital Cushion

The role of the digital cushion has been long debated, often in connection with so-called "frog pressure."[6] Dyhre-Poulsen et al.[7] showed that the pressure in the cushion dropped during weight bearing. In this experiment, horses were shod so that their frogs and soles did not make contact with the ground, and these researchers suggested this was caused by expansion of the hoof without pressure being exerted on the cushion itself. They showed that the pressure in the cushion builds up relatively slowly with the foot off the ground and is suddenly released as the foot is loaded. This is characteristic of a *relaxation oscillation*.[8] Dyhre-Poulsen saw an almost immediate (within 30 msec) drop of pressure in the cushion when the foot impacted the surface.[7] My interpretation of these data is that this pressure drop occurred because of squeezing of the extensive venous plexuses in the corium of the hoof.[9] This squeezing, in turn, forces blood out of the cushion to refill those emptied veins, and that explains the drop of pressure in the cushion itself.

The pressure drop was largest dorsally and was less toward the palmar aspect of the cushion, indicating less squeezing out of blood from the frog area of the solar surface and more under the distal phalanx during the first 30 msec after the impact of the hoof with the surface. This supports the contention that the frog and cushion are not significantly loaded during the initial impact of the foot with the surface.

The value of a relaxation oscillation is that it is "ideally suited to control systems in which an input stimulus should produce a response of fixed amplitude but adaptable frequency or repetition rate."[8] This is exactly what is needed in the case of the energy absorption action of venous blood movement in the foot.

Dyhre-Poulsen's measurements were done at the walk, at the trot, and in a standing pony.[7] At higher velocities, larger pressure drops might be expected in the cushion as the pastern joint pressed down, forcing more blood from the cushion. The drop in pressure in the digital cushion, then, is not because the cushion is not being compressed but because compression is forcing blood out of the cushion into the efferent veins. At slower gaits, the cushion is compressed by outward expansion of the hoof wall, whereas at faster gaits both outward expansion and downward pressure of the pastern are causing the compression.

Other Factors

There is an inherent genetic contribution of hoof wall strength and friction resistance; however, little information is available on these factors. The role of nutrition in the quality of hoof horn in relation to strength and friction resistance also cannot be overlooked. Again, however, little is currently known. The fact remains that the qualitative wearing pattern of the bare foot is primarily related to friction, the effects of which can be modified by the genetically and nutritionally determined quality of the horn of the hoof.

THE HORSESHOE

The experienced veterinarian and farrier may say that all this "mechanics stuff" is unnecessary. An experienced *rack of eye* is what is needed to properly shoe a horse. However, rack of eye is often no more than guesswork. For farriery to become a truly mature profession there must be science as well as art. The working farrier has developed the art to a remarkable extent; the science, however, lags behind.

This discussion will not enter into the polemics on the several horseshoeing "systems" (or lack of systems) said to be based on the study of feral horse feet or otherwise. Consideration of the section of this chapter on the wear of the bare foot shows how incorrect much of the interpretation of the shape and wearing of the bare foot has been. This misinterpretation has led to some bizarre shoeing systems that demonstrate how wonderfully adaptable the horse is to even the most misguided human interference.

Hoof Angles and Tendons

There is only one center of rotation (locus) in the foot, in the distal end of the middle phalanx. When a shoe is nailed or glued to the hoof, the shoe becomes part of the foot mechanically and does not introduce any new centers of rotation. It has been known at least since the late 1800s[10,11] that the angle of the pastern with the surface becomes more upright if the angle of the hoof, as measured at the toe, is decreased, whereas the angle becomes more sloping if the angle at the toe is increased. Such angle changes can occur by trimming, wear, or wedging.

A recurring question has been the use of changes of hoof angle in the treatment of the several types of tendon damage. The immediate response to decreasing hoof angle is an increase of the tension in the deep flexor tendon (DF). This increase "pushes" the fetlock up and forward, making the pastern more upright and tending to decrease the tension in the superficial flexor tendon (SF) and the suspensory tendon (SLT). The model made in Figures 4-10 and 4-11 can help illustrate this. If the third phalanx (P3) is turned as if the heels were cut down and the hoof angle decreased, the tightening of the deep flexor tendon is obvious. As discussed later, however, there is little or no change in SF and only a small decrease of the suspensory tendon. Once the pastern moves up, the fetlock joint opens, its dorsal angle increases, and tension of the DF tends to decrease. The end result is a modest decrease of tension in all three palmar (foreleg) or plantar (hindleg) tendons.

The superficial and deep flexor tendons are tightly bound together and to the related bones by strong deep fascia.[12] For example, the superficial flexor tendon can be severed either at the check ligament or below the carpus/tarsus and produce no obvious loss of tension in the superficial flexor tendon distal to the site of transection. The deep flexor tendon can be severed below the fetlock, and the tendon proximal to the cut will remain tense. This means that angular changes of the hoof that cause tensile change in one flexor tendon will cause tensile change in the other flexor tendon. In in vitro foreleg experiments, when all of the tendons are removed and the leg is loaded in a testing machine, the load the leg can sustain is 50% of the load sustained in the same leg in the same machine before the tendons were cut away. This means that the deep

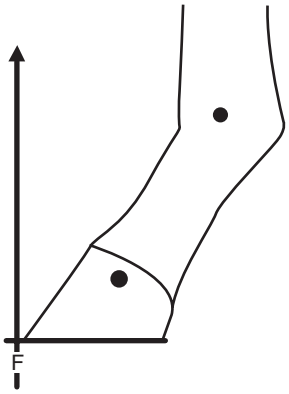

FIGURE 4-24 Extending the toe of the shoe moves F forward.

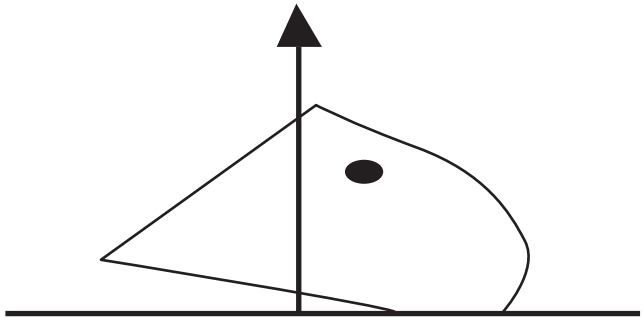

FIGURE 4-25 Mechanical characteristics of straight bar and egg bar shoes.

fascia and the collateral ligaments of the several joints can bear 50% of the total load experienced by the leg. Of course, this finding is in the dead leg and not in the live horse, but it is reasonable to think that the effect would be the same or very similar in the live leg. This can be illustrated with the model of Figures 4-10 and 4-11 by clipping the superficial and deep flexor tendons together with some pins or a paper clip to represent the deep fascia and then changing the hoof angles.

Similarly, with an increase of hoof angle, the DF decreases and SL and SF should increase. Again, the superficial flexor tendon might be thought to tighten with the decrease of the dorsal angle of the fetlock, but it loosens instead because of the deep fascial ties to the loosening deep flexor tendon. Again, the net result is no change in SF and a small increase of the superficial ligament tension.

Tendon Damage Treatment

Is it desirable, then, to change hoof angles, and so tension, in the treatment of a tear of the superficial flexor tendon (bowed tendon), the suspensory ligament, or the check ligament of the deep flexor tendon? From what has been presented, it seems clear that there is little or nothing to be gained by changing hoof angle in the treatment of bowed tendon or suspensory tendon damage. The decrease of tension in the deep flexor tendon with a larger hoof angle might be of value in the treatment of tearing of the check ligament of the deep flexor. There have been no measurements of the tension in that particular ligament, but the deep fascial "cancellation" of changes in the superficial flexor suggests that the identical or very similar situation would pertain with the check ligament.

Extended Toe Shoes

Can different types of shoe assist tendon healing apart from changes of hoof angle? Extended toe shoes are used for animals with so-called contracted tendon or tendons. The subject is discussed elsewhere[1] under the more correct rubric of "shortening tendons."

Extending the toe of the shoe moves *F* forward to the dotted-line position (Figure 4-24). (Review the earlier discussion of moments, if necessary.) This means there is a larger moment produced by *F* acting around the center of rotation in the coffin joint. The larger clockwise moment can resist the counterclockwise moments exerted by the shortening tendons, represented in Figure 4-24 by the deep flexor tendon only. Obviously, no amount of toe extension can stretch the shortened tendons to normal length unless the extended toe is raised from the surface. Although the extended toe shoe may be of some help in the young animal with shortening tendons, there are other, more useful strategies, as described elsewhere.[1] Recent work has also shown the advantage of toe extension when dealing with stump foot (i.e., clubfoot).[1]

The extended toe shoe can also be helpful during the healing of transected long extensor tendons in the hind leg, a frequent site of traumatic transection. The extended toe moves the line of action of *F* forward, as shown in the figure. The increase in the moment generated by *F* makes up for the moment lost when the long extensor tendon is cut. As is well known, the severed tendon will eventually adhere to the periosteum on the dorsal face of the cannon bone and act as a check ligament with virtually full restoration of normal function of the long extensor tendon at the fetlock.

It is important to recognize that the effect of the extended toe shoe and the egg bar shoe (see later) occurs *only when there is movement of the foot*. The extended toe shoe behaves as described only when the hoof moves, so that the angle of the hoof increases. If the foot is absolutely stationary on the surface, neither extended toe nor egg bar shoes have any effect whatsoever.

Bar Shoes

Straight bar and egg bar shoes have similar mechanical characteristics, but the egg bar shoe extends farther back and thus exerts more moment. If the deep flexor tendon were to be transected, the toe of the hoof would come off the ground (Figure 4-25). There is no deep flexor counterclockwise moment to counterbalance (equilibrate) the clockwise moment exerted by the ground reaction force, *F,* and the clockwise moment produced by the common (lateral) extensor tendon plus the extensor branches of the suspensory tendon. Obviously, *F* cannot push the toe up off the surface; that must be done by the extensor tendon system.

Analogous is the foal born with so-called flaccid flexor tendons of the hindlegs. The cause of this condition is not known. The tendons appear to lack tensile strength, and the situation corrects itself with time in many foals as the tendons strengthen. To deal with this before natural correction occurs, an egg bar shoe is applied, so that when the hoof rocks back

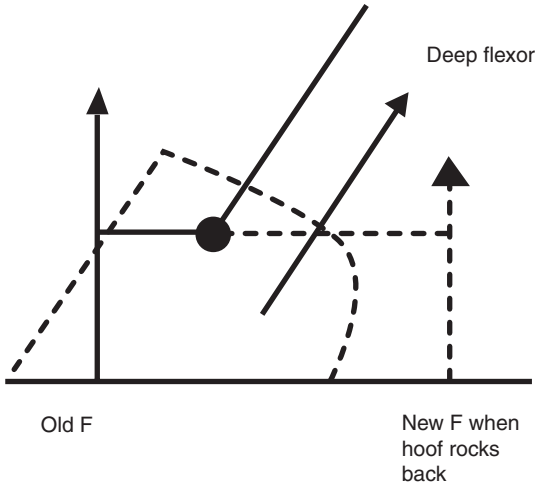

FIGURE 4-26 Flaccid flexor tendons of the hindlegs corrected with an egg bar shoe.

clockwise unto the heels, the egg bar shoe exerts a counterclockwise moment, and the toe is pressed back to the surface (Figure 4-26). The loose or flaccid deep flexor tendon is mechanically equivalent to (but not as severe as) a transected deep flexor tendon and the toe will be off the ground with the fetlock often resting on the ground. The egg bar shoe acts in the same manner as with a transected tendon and helps the foal with flaccid tendons achieve a more normal conformation until the tendons mature appropriately.

Suspensory Desmitis

Suspensory desmitis is a degenerative condition afflicting the hindlegs of, particularly but not solely, Peruvian Paso Fino horses. It can be at least temporarily ameliorated by the use of the egg bar shoe. In this case, the suspensory tendon is "degenerating," losing tensile strength and allowing the fetlock to sink toward the ground. This is similar to the situation with older multiparous mares in late pregnancy. When the foot tips back, so that the egg bar shoe exerts a counterclockwise moment around the coffin joint, tension in the deep flexor tendon is reduced and the deep flexor tendon shortens. The beneficial effect of the egg bar shoe is to allow shortening of the deep flexor tendon, so that it is supporting additional load, some of the load that can no longer be borne by the damaged suspensory tendon.

Additional Bar Shoes

It is apparent that the egg bar shoe moves the line of action of *F* toward the heels if the toe is tipped up and off the surface. If, however, *F* moves *behind* the center of rotation, the moment caused by *F* reverses, becoming positive, or counterclockwise. When the horse is standing still, moments are always present and in equilibrium. The slightest movement, or pitch, of the foot throws the system out of equilibrium. (*Pitch* means the backward and forward rocking movement of the hoof on the surface as seen from the lateral view.) Toe extensions or egg bar shoes simply exaggerate such responses to loss of equilibrium by moving the line of action of *F* when the pitching occurs.

Neither the extended toe shoe nor the bar shoe have any effect unless the hoof is rocking forward or backward, or changing pitch. First, we examine what a horse does without shoes or with its usual shoes when there is damage to one of the tendons. There is pain and several possible responses to that pain. The immediate response, of course, is to decrease the load on the damaged leg/foot, that is, to decrease *F*. Doing so, the leg straightens at the coffin and fetlock, thereby reducing tension in all the tendons. The moment generated by *F* decreases, thus the equilibrating tendon forces will decrease. After the acute pain subsides, no matter its origin, the horse may either stand normally (but with reduced *F*) or in the so-called standing-back position. When the horse stands back, the pastern becomes more upright, and the line of action of *F* moves back and the moment exerted by *F* decreases, and the tension in all three tendons decreases.

How can the egg bar shoe affect either of these situations? First, there is simple reduction of *F,* decreasing the body weight on the affected leg, and the bar shoe does nothing. The second case, standing back, seems to be more commonly associated with low-grade, persisting pain. The line of action of *F* moves back as the pastern becomes more upright with reduction of tension in the deep flexor tendon. In this situation, the egg bar shoe can have an effect only if the hoof angle decreases (toe moves up, the foot pitches back).

Dynamics

Egg bar shoes and the closely related trailer shoes have an effect when the hoof is moving or the whole horse is moving. The trailer, egg bar, or to a lesser extent the straight bar shoe contacts the surface first with the heel-quarter-toe contact sequence of the faster gaits. This will cause bending of the bar or trailer with consequent absorption and dissipation of energy (see later). With a trailer shoe, the foot tends to yaw, that is, to spin on the surface. Although it might appear that the trailer shoe in contact with the surface is acting as a center of rotation, the center of rotation is always at the coffin joint in the distal end of the middle phalanx. At the point of contact of the trailer shoe with the surface, the surface exerts an upwardly directed linear force (in effect *F*), which exerts a moment just as *F* almost always does.

The bending of the trailer or egg bar shoe absorbs and dissipates energy, as previously mentioned, which helps ameliorate the pain of impact of the foot with the surface. The trailer shoe is usually on the outside (lateral) branch of the hindshoe as an aid in preventing or minimizing cross-firing by pacers. Pacers are predisposed to cross-firing by the toed-in or toe narrow conformation. The trailer induces yaw, a turning out of the hoof at impact that tends to counteract the inwardly directed toe conformation.

BREAKOVER/LIFT-OFF

Breakover is the rolling of the hoof off the surface at the end of the support phase of the step. I usually refer to this as *lift-off*, which is an equally valid and useful term. Most farriers and veterinarians seem to prefer the term *breakover*, however, so both terms are used here interchangeably. Lift-off was previously discussed in connection with the rolling of the bare hoof at the end of support.

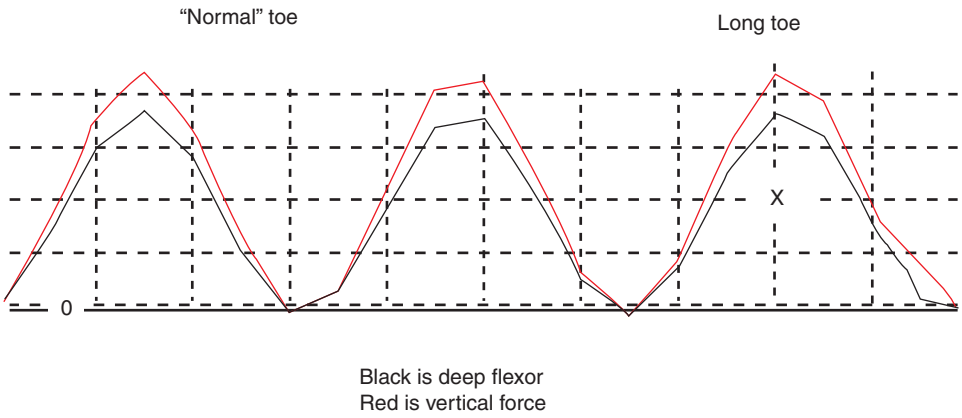

FIGURE 4-27 The vertical force on the foot and the tension in the deep flexor tendon increase and then decrease during the step (the support period).

Some people seem to believe that breakover begins much earlier, during the second half of support, than is actually the case. Careful examination of horses in motion, in Muybridge,[13] for example, convincingly shows that the frank lifting of the heels, quarters, and toe from the ground occurs in, perhaps, the last hundredth of a second of the step. The vertical force on the foot and the tension in the deep flexor tendon increase and then decrease during the step (the support period) (Figure 4-27). The deep flexor tendon is in tension before impact of the foot with the ground (although that does not show clearly in the illustration). The tension in the deep flexor tendon is always less than the vertical reaction force, which is not readily understood.

Important are not only the forces involved but also their moment arms about the respective joints. The ground reaction force (GRF) moment arm about the coffin joint is small, and the deep digital flexor (DDF) tendon has the benefit of passing around the navicular bone, which makes its moment arm quite large. The combined effect of all these factors produces a small DDF force. If the GRF moment arm about the coffin joint is large or the DDF moment arm is small, then it could indeed have a magnitude much greater than the GRF. The tendon tensions cannot be compared directly to the GRF, however (contrary to what this statement seems to suggest).

The length of the toe has not been included in the simulation, but we can include it in the discussion. The relationship between the vertical force and the tendon tensile force is not only linear but also a matter of the moments exerted by each force around the center of rotation in the distal end of the second phalanx (P2). Thinking of the curves shown in Figure 4-27 as moment curves, which is legitimate, the longer the toe is, the slower the decline of the moment exerted by the vertical force will be, and the vertical force moment will be shifted to the right. The tensile force moment is resisted more than with a shorter toe, and breakover (lift-off) will be delayed, as indicated in Figure 4-27 by the right-hand curves marked with an *X*.

RINGS OF THE HOOF WALL

The surface of the hoof is characterized by striations running from the coronary band to the bearing edge of the hoof wall and by rings and grooves at right angles to these striations, parallel to the coronary band. The striations are the superficial horn tubules. The rings and grooves are mentioned in many texts,[10,11,13] but there are only a few papers discussing their nature and cause. When there are rings, there must be grooves between the rings.

Gutenäcker[13] believed that the rings and grooves were related to nutrition. With poorer nutrition, there was less material for horn formation and less blood flow to the coronary band, all of which led to the formation of the grooves. With increased or intensive nutrition, the opposite conditions pertained and rings formed. He noted that the grooves also develop in animals suffering from systemic disease with consequent impaired nutrition.

Fambach[14,15] discussed what he considered to be physiologic and pathologic ring formation. With physiologic "ringbildung," only the more superficial portion of the hoof wall was involved, and the interstitial horn and horn tubules were markedly irregular. The normal, linear course of the tubules was bent out into the rings and inward into the grooves. He did not believe that feeds and feeding were factors in the ring and groove formation. They appeared when a horse had a marked change of diet, when grain supplementation was started, during the last part of pregnancy, and when foals were weaned. Fambach also suggested that ring formation could be related to new hair growth, particularly in the spring of the year.

Fambach[15] continued his studies to include more extreme ring formation such as that found with chronic laminitis and otherwise badly misshapen hooves. He illustrated, as did Lungwitz,[10,11] the enhanced ring formation with laminitis and the marked "dishing" of the dorsal and lateral or medial hoof walls.

Sagittal sections of the toe portion of the hoof wall show outward and inward bending of the superficial horn tubules corresponding to the grossly evident rings and grooves. Indeed, this waviness of the superficial horn tubules is apparent with low magnification even when rings and grooves are not apparent to the naked eye.

Histiogenesis

The wavy distortion of the superficial horn tubules is the result of bending of the tubules caused by compression. This distortion is actually buckling—the bending of a column

FIGURE 4-28 Buckling: the bending of a column under compressive loading.

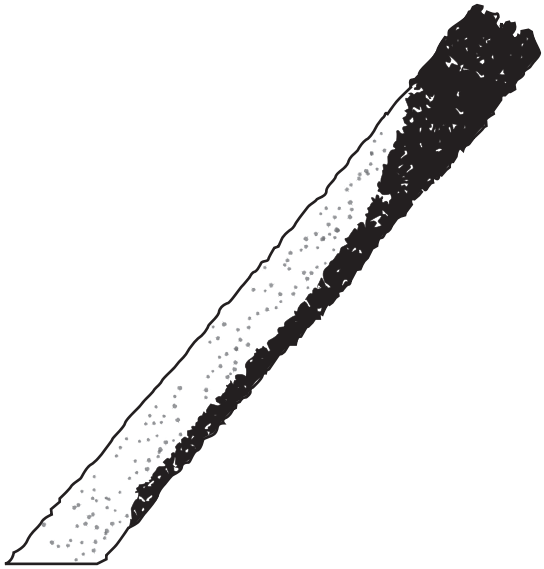

FIGURE 4-29 The proximal part of the hoof wall (closest to the coronary band) is the most hydrated part of the wall, with the amount of water decreasing steadily toward the bearing edge of the wall.

under compressive loading (Figure 4-28). It has been established that the hoof wall is normally in compression when loaded.[15] The waviness, rings and grooves, always appears first in the proximal part of the wall just beneath the coronary band and moves distally as the wall grows.

Bertram and Gosline[17] showed that the proximal part of the hoof wall (closest to the coronary band) was the most hydrated part of the wall, the amount of water decreasing steadily toward the bearing edge of the wall (Figure 4-29). These findings are clearly consistent with the proposed histiogenesis, since moist materials are more liable to buckling in compression than drier materials. This is easily demonstrated with small wet and dry sticks of wood.

Rings and grooves may not be apparent in some hooves, particularly those of feral horses and wild zebras. They are not present in the hooves of newborns or for the first weeks of life. Close examination may show the waviness of the outer horn tubules but not the more obvious rings seen frequently in domesticated equids. It can be suggested that the weight of animals on marginal diets and in thin condition does not compress the hoof wall sufficiently for obvious rings to form but does compress it sufficiently to produce minimal buckling and waviness.

CRACKS IN THE HOOF WALL

Toe and quarter cracks starting at the coronary band—the more common and clinically important form of cracks—are a direct result of disruption or damage of the coronary band. With such damage, of course, the process of tubular horn formation is interrupted, and a defect appears in the hoof wall as the adjacent, normal coronary band continues to produce new horn tubules. Usually such interruptions of growth are temporary, with the crack eventually disappearing as the coronary band is repaired and resumes growing. The term *crack,* then, is a misnomer; *defect* is a better term.

Direct trauma to the coronary band is a clear example. Such trauma occurs for a variety of reasons: interference by another foot, wire cuts, treading by the same or another horse, and so on. The purpose here is to consider the role of the horseshoe in the pathogenesis of quarter and toe cracks not attributable to such direct trauma.

As the bare foot is loaded, the quarter moves outward in a smooth curve, as indicated in Figure 4-30. When the horse is shod, the quarter is constrained by the nails and will bend at or near the last nail. This bending can cause tearing and bruising of the laminae along the bend line and at the coronary band. There is failure of production of new horn tubules in such a damaged area of the coronary band, and this appears as the defect in the wall.

A similar mechanism holds for toe cracks. Because of the restraint imposed by the nails, the toe bulges out and forward (Figure 4-31). In this case, the bending is in the opposite sense but still causes damage.

An interesting feature of many toe cracks is that, if not too large, they tend to close when the foot is loaded and open when unloaded. This clinical observation has been confirmed by Thomason et al.,[18] whose study showed with strain gauges that there is biaxial compression (two compressive loadings at right angles to each other) of the toe portion of the hoof wall. The major compression is vertical, and the compression at right angles is caused by the circumferential bending of the hoof wall. It is this circumferential bending that tends to close the gap when the foot is loaded.

Older literature indicates that toe cracks were more common in the hindfeet of draft horses. The damage could be done when the foot is lifting off from the surface with compressive force concentrated on a decreasing toe surface area. Even though the

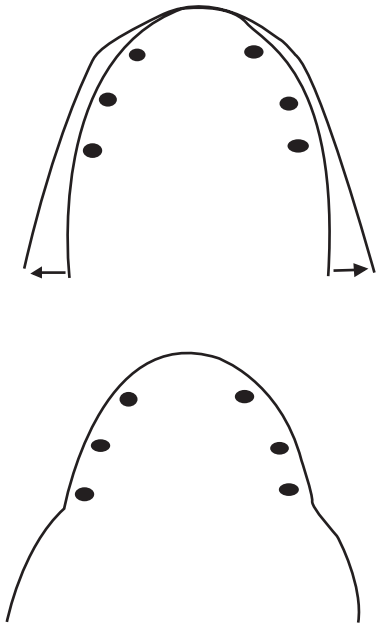

FIGURE 4-30 Mechanism for quarter defects: as the bare foot is loaded, the quarter moves outward in a smooth curve.

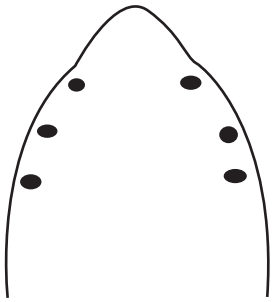

FIGURE 4-31 Mechanism for toe cracks: because of the restraint imposed by the nails, the toe bulges out and forward.

total force would be decreasing as the foot lifts off the surface, the force per unit area (the stress) at the toe would be increasing.

Assuming that this hypothesis is correct or nearly so, the question is why all shod horses do not crack their hoof walls. Since this cannot be determined for horses in general, a variety of factors must be considered for the individual horse. A few of those factors are genetic (or otherwise) quality of hoof horn, frequency of trimming or shoeing, surfaces on which horse habitually moves, body weight (more weight equals more load equals more movement of hoof wall), misplacement of nails, state of hydration of the hoof wall, contracted hoof, and so on. The important point is that we can provide a reasonable general pathogenesis applicable in all cases but we cannot provide general statements about auxiliary, contributing factors. Those can only come with evaluation of the individual horse.

WHITE LINE DISEASES

White line disease, seedy toe, classical laminitis, and road founder type laminitis have a great deal in common.

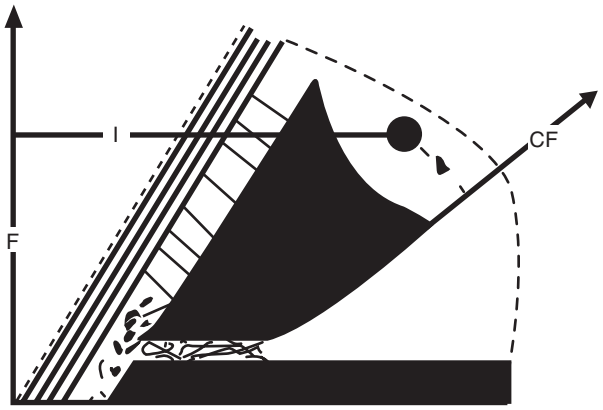

FIGURE 4-32 Road founder is tearing of the laminae near the distal border of the coffin bone.

Road Founder

Of the white line diseases diseases, only road founder needs to be defined. It was well known in past times, occurring particularly in the heavy carriage horses used on the roadways and turnpikes of England and Europe. These horses were characteristically trimmed with long toes and shod; the long toes provided a flashy, high-stepping gait.

Road founder is tearing of the laminae near the distal border of the coffin bone (Figure 4-32). If severe enough, the horse would be lame with the clinical signs of any case of laminitis. Both forefeet could be and often were affected at virtually the same time, thus closely resembling the classical "over-eating" type of laminitis.

In less severe cases, horses might show little or no lameness. The presence of the condition in such cases is indicated by blood spots in the white line area of the toe when the farrier trims the foot. Henry Heymering was the first to concentrate my attention on these bloody spots. It is now clear that they are hemorrhages migrating down into the white line area because of tearing of the laminae just above that area as shown schematically in Figure 4-32.

Torn tissue means hemorrhage and dead tissue, and those two factors predispose the area to infection with whatever bacteria or fungi happen to be prevalent in the horse's environment, that is, white line disease/seedy toe.

Pathogenesis

The tearing of the laminae occurs because of the increased resistance of the long-toed hoof to breaking over at the end of the support phase of the stride, as the foot is leaving the ground, which can be shown easily by the following equation:

$$DFb - Fl = 0$$

The moment exerted by the deep flexor tendon is equal to the opposite moment exerted by the body weight on that leg *(F)*. If the length *(l)* of the toe increases *(Fl)*, *DF* must pull harder and causes the tearing of the laminae. The laminae, clearly, are the connecting elements between those two forces.

Classic Founder (Laminitis)

Chronic laminitis predisposes to white line disease/seedy toe. As is well known, the laminae are destroyed or, at least, severely damaged. Even a little *F* and *DF* are sufficient to tear

what remains and to tear young repair tissue. Again, there is continuing hemorrhage and death of tissue predisposing the foundered foot to infection: white line disease/seedy toe. This type of case represents a severe example of the same type of process (though of different ultimate cause) as with road founder.

Shod and Barefoot

White line disease is more frequent in shod than barefoot horses because the toe continues to grow and is not worn down when the hoof is shod. The longer the shoe is in place before being replaced or reset, the longer the toe becomes, thereby increasing the risk of laminar tearing, or road founder.

Even barefoot horses and ponies, however, can develop road founder and white line disease if the surface on which they move does not wear down the bearing edge of the hoof wall sufficiently or the feet are not trimmed regularly. An excellent example is the Shetland pony with iron-hard hooves not adapted to lush, bluegrass pastures.

Wet and Dry

Wet conditions are said to predispose to white line disease. Certainly, wet standings will contribute to the softening of the already damaged white line area. It seems improbable that wet conditions would be a cause, although they can be the source for several bacteria and fungi that invade the damaged white line tissue.

NAVICULAR DISEASE

Navicular disease remains a subject of confusion and error despite thousands of words being written on the subject, defining what the condition actually is pathologically and how it may develop. These opinions are based on years of observation, primarily in the postmortem room[19] (Dr. Neil Williams, unpublished findings) together with in vitro experimental studies.[20] An extensive critical review of the literature is beyond this chapter, but additional references can be found elsewhere.[1]

Based on in vitro studies, increased friction because of increased normal force between the dorsal face of the deep flexor tendon and the palmar surface of the navicular articular fibrocartilage suggested itself as the immediate cause of damage to both tendon and fibrocartilage.

When a tendon (or a string or strap) is pulled straight, there is tension produced only in the tendon (Figure 4-33, *A*). If the tendon passes around a curve, as in Figure 4-33, *B*, tension again occurs along the length of the tendon, but there is also a force at right angles pressing against the surface. The more highly curved the surface is, the greater this perpendicular force, or *normal force,* will be. In Figure 4-34, the damage was done first to the more highly curved parts of the flexor surface of the navicular bone.

Figure 4-35 is a photograph of the carly stages of a spontaneous case of navicular disease. The brownish, translucent discoloration of the distal ridge of the navicular bone is apparent. No damage is yet apparent on the less curved proximal ridge. Once the damage has been done to these two ridges, it gradually spreads to involve more and more of the

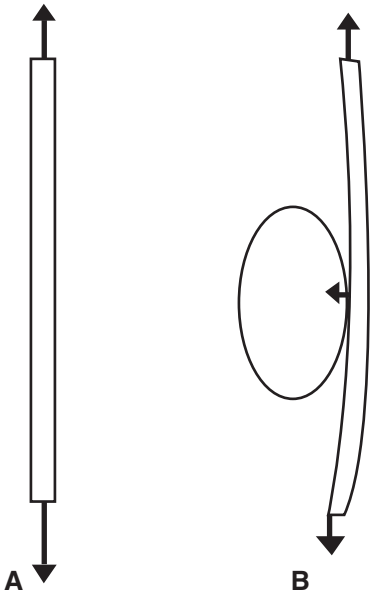

FIGURE 4-33 Tension in a tendon. **A,** when the tendon is pulled straight, there is tension only in the tendon. **B,** when the tendon curves around a surface, tension again occurs along the length of the tendon, but there is also a force at right angles pressing against the surface.

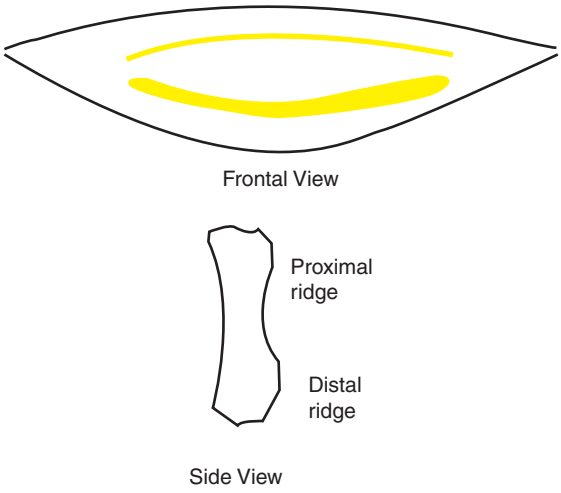

FIGURE 4-34 The earliest evidence of damage is a yellowish or brownish, often translucent discoloration of the fibrocartilage of the navicular bone and the corresponding fibrocartilage of the deep flexor tendon with superficial fraying of the cartilage.

articulating surfaces of the navicular bone and the deep flexor tendon.

Routine examination of the navicular area at postmortem in thousands of horses made it clear that the earliest evidence of damage is a yellowish or brownish, often translucent discoloration of the fibrocartilage of the navicular bone and the corresponding fibrocartilage of the deep flexor tendon with superficial fraying of the cartilage in the two areas indicated schematically in Figure 4-34. These are proximal and distal ridges on the palmar surface of the navicular bone. The distal ridge is more pronounced, that is, has greater curvature, thus there is greater normal force exerted on it. The lesions appear

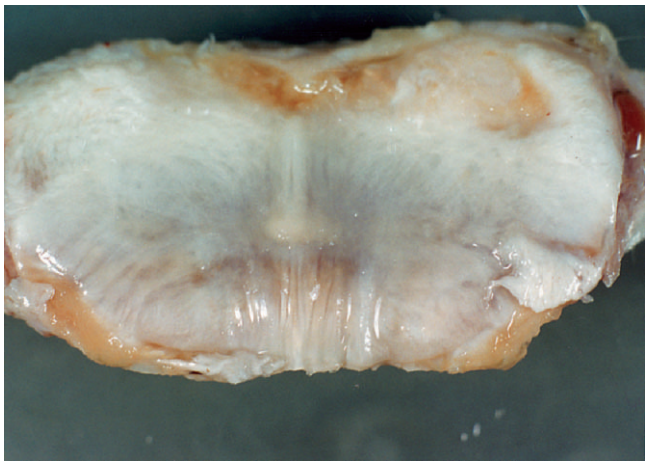

FIGURE 4-35 The early stages of a spontaneous case of navicular disease.

on the distal ridge first and more extensively than on the less curved proximal ridge (side view in Figure 4-34).

The discoloration of the fibrocartilage is, in fact, the earliest naked eye evidence of damage to any equine articular cartilage whether it be hyaline or fibrocartilage (see Figure 4-35). The yellow/brown color is not caused by pigment but rather by structural change in the cartilage. The precise nature of that change is not known and would be a fine subject for study. What is immediately evident histologically is loss of chondrocytes in the discolored area together with fraying of the superficial layer of fibrocartilage and increased eosinophilia of the cartilage matrix.

Pathogenesis

The hypothesis developed first with the testing machine trials was that toe-first impact of the foot with the ground caused damage to the navicular fibrocartilage and the deep flexor tendon. The toe-first impact itself does not do the damage. Normally, the foot impacts flat-footed on the ground at slower speeds and in heel-quarter-toe sequence at faster speeds, as discussed earlier. With flat-foot or heel-first impact, the coffin joint rotates, so that its rotation does not increase tension in the deep flexor tendon but rather decreases it. In contrast, with toe-first impact the coffin joint rotates in the opposite direction as the quarters and heel come down onto the surface. Such rotation causes an increase in the tension in the deep flexor tendon, thus an increase of the normal force between the tendon and the fibrocartilage of the navicular bone. This explains the well-known propensity of jumping horses for navicular disease, since the forefeet routinely impact toe-first from a jump.

Second, the results pointed to the hypothesis that vibration, high-frequency rocking back and forth of the hoof, would cause repeated peaks of normal force each time the hoof rocked back from toe to heel (Figure 4-36), as in the toe-first impact situation. Such vibration is to be expected on hard surfaces and/or in horses with small feet and large bodies, the latter delivering more energy than a small foot can properly store and dissipate. Examples are police horses working under heavy loads on hard pavement for hours, the type of quarter horse with draft horse bodies and Thoroughbred feet, and carriage horses used for long hours on hard pavement.

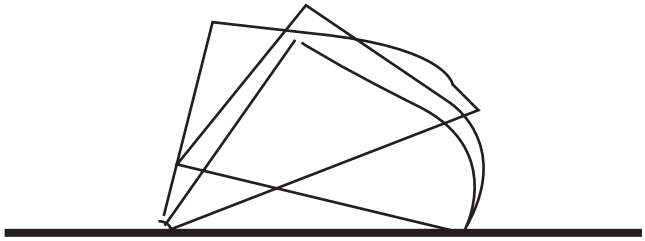

FIGURE 4-36 Vibration, high-frequency rocking back and forth of the hoof, is thought to cause repeated peaks of normal force each time the hoof rocked back from toe to heel, as in the toe-first impact situation.

Given the hypothesis that vibration is the proximate cause of navicular cartilage and deep flexor tendon damage, such vibration causes the fraying and yellow discoloration. Since heat is the major product of vibration, a small experiment was done. Fresh articular cartilage was exposed to the heat of a photoflood lamp until the synovial fluid just began to bubble and the cartilage turned yellow. This took only a few moments' exposure. Histologically, the chondrocytes had disappeared in the superficial part of the cartilage, and there was increased eosinophilia of the cartilage matrix, the same picture as in the naturally occurring early stages of fibrocartilage damage. Obviously, such an in vitro experiment is not decisive, but it does show that heat can produce damage similar to that seen in the naturally occurring lesion and is consistent with vibration causing damage to the articulating surfaces.

Vibration as a basic cause of navicular disease is certainly supported by the type of animal typically afflicted (large body/small foot) and type of work (jumping, work on hard roads). Central to this hypothesis is that the initial damage is to the surface of navicular fibrocartilage surface of the fibrocartilage of the deep flexor tendon (kissing lesions). A variety of competing hypotheses about sclerosis of subchondral bone and several different varieties of vascular disease have been discarded.

The so-called vascular channels are radiolucent loculi in the distal margin of the navicular bone. These are not vascular channels but sacculations of the coffin joint of variable size and shape. Despite the general and uncritical acceptance of these loculi as *prima facie* evidence of navicular disease, it is simply not true. These structures have nothing to do with navicular disease, as has been clearly apparent in many hundreds of postmortem examinations. These innocent structures have taken on mythic diagnostic proportions and that is shamefully incorrect.

The question occasionally arises as to why the forefeet are subject to navicular damage while the hindfeet are much less so. There are two answers: the first is that the hindfeet do not impact toe-first when the horse lands from a jump. A more important answer is that the interaction of the coffin and fetlock joints is somewhat different in the hindfeet. Upon impact of the hindfoot, the coffin joint rotates almost completely to its midsupport position before the fetlock joint begins to rotate. Thus, despite the toe-first impact, only the coffin joint is moving and increasing tension in the deep flexor tendon. The increasing normal force exerted on the navicular bone, from impact to midsupport, is occasioned by the movement of the coffin joint only.

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5

INNERVATION OF THE EQUINE FOOT: ITS IMPORTANCE TO THE HORSE AND TO THE CLINICIAN

ROBERT M. BOWKER

The equine foot represents a unique structure. It is designed to interact with the environment to detect and perceive various signals informing the horse about the ground surface that it is standing on. However, it is also enclosed within a hardened keratinized shell that protects the internal foot structures during movement.

The foot's detecting and perceiving various signals about the environment is crucial to the well-being of the horse. The sensory receptors in the foot must elicit the appropriate neuromuscular reflex response during movement, as well as protect the horse from potentially harmful events. In other domestic and wild animals, many and various sensory receptors are known to be present in feet.[1-3] However, many of these feet have only roughened or thickened cutaneous surfaces, such as pads, similar to those present in the superficial skin and subcutaneous tissues, whereas the horse has a hardened encasement around the distal limb. This hard keratinized hoof may serve as a "buffering agent" that decreases the sensations the horse can detect.

Presumably, the different types of sensory receptors within the equine foot are specially suited for detecting the unique and beneficial features of stimuli in the environment that a horse will encounter. Whether the hoof wall and sole mask these potential environmental stimuli or whether there are specific or specialized sensory receptors suited for detecting stimuli unique to equidae remains to be seen. Most identified sensory receptors have been divided into several broad categories based on the physiologic responses to the environmental stimulus required to activate them and to the response properties of the receptor during the application of the stimulus. These broad categories include the mechanoreceptors, the nociceptors, and the thermoreceptors. The mechanoreceptors respond to the rather subtle but innocuous physical deformations of the skin surface induced by various degrees of tactile or applied pressures. Other receptors located deeper in the tissues respond only to more strongly applied mechanical deformations of the tissues. The nociceptors respond to painful stimuli during damage to the tissues. The thermoreceptors are specifically adapted for detecting temperature changes in the surfaces of the cutaneous tissues.

The assumption that lightly applied stimuli to the surfaces of the foot are detectable by the horse can be made from observing the movements of the limbs when the foot is placed on the ground surface as well as when flying insects make contact with the hoof wall or superficial hairs around the coronet. Probably mechanoreceptors and nociceptors are common and plentiful within the foot, as the horse appears to respond purposefully when the foot is touched, its position is altered, or some pressure or painful stimulus is applied during movement and stance. On the other hand, the foot with sufficient sole thickness does not seem to appreciate temperature variations, at least not as evidenced by any noticeable or meaningful response of foot withdrawal when very hot metal shoes are placed on the trimmed solar and hoof wall surfaces (hot shoeing). These behavioral observations suggest that thermoreceptors within the foot are less common or at least more difficult to activate than other tactile receptors or thin nerves conveying pain to the central nervous system.

Within the environment, each type of sensory receptor has a specific type of stimulus to which it responds. Receptors that respond to the physical deformation of the hoof wall or sole are classified as mechanoreceptors.[3-5] Most of these mechanoreceptors have a physical structure at the nerve terminal that encapsulates the nerve fiber and becomes activated during the application of the sensory stimulus (Figure 5-1). Usually the stimulus of pressure, or other physical force, changes the ionic concentrations across the receptor surface, thereby generating an electrical potential (generator potential) that then proceeds along the afferent nerve fiber toward the spinal cord for the sensation to begin to be perceived. In many animals, such receptors are typically low threshold and may either have a rapid response time to a stimulus or be more slowly activated by a similar or other type of physical stimulus.

The first type of mechanoreceptor, the low-threshold, rapidly adapting type, responds by generating a rapid burst of nervous activity to a slight deformation of the hoof wall or sole but will quickly adapt and cease to respond as the electrical activity is transmitted along the thickly myelinated nerve fiber toward the spinal cord (see Figure 5-1). When the stimulus ceases to deform the hoof wall or sole, or the stimulus stops moving, the mechanoreceptor becomes inactive. One behavior that produces rapid activity responses followed by rapid adaptation of the mechanoreceptor is locomotion, such as during a walk or trot, when the horse's foot is moving across the ground surface and makes initial contact with the bulbs of the heel.

Activation of other receptors, low-threshold, slowly adapting, respond more slowly and tonically when pressure within the foot tissues increases (or perhaps decreases), such as when the horse is standing and grazing on the pasture or at the hayrack (see Figure 5-1). Such slowly adapting

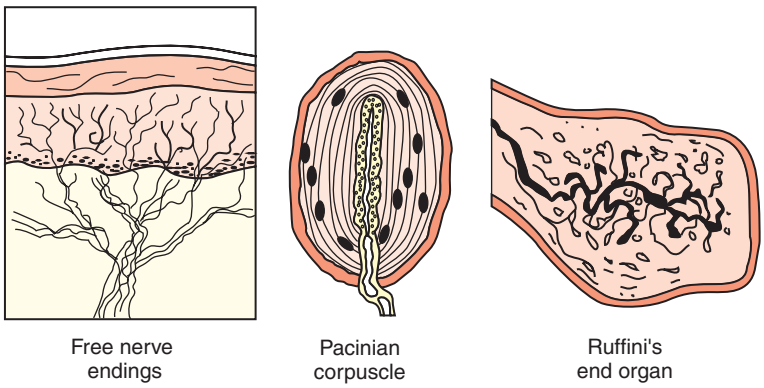

FIGURE 5-1 Schematic drawings of three types of sensory receptors present in the horse's foot. The free nerve endings respond to painful stimuli, whereas pacinian corpuscles respond to rapidly applied stimuli such as when the heel hits the ground. The Ruffini end organ responds slowly and for long time periods to applied stimuli, such as during stance.

mechanoreceptors will continue to generate receptor potentials for long periods of time within the nerve fibers during stance, as opposed to the more quickly adapting mechanoreceptors that are only transiently activated and have long ceased their nervous activity. Generally, when the horse is quietly standing and grazing, the transiently activated receptors are less likely to become activated during this behavior, unless the horse lifts its leg and quickly stamps its foot to the ground.

While the slowly reacting pressure receptors may become active during certain types of movement, the generated neural activity produced at these times will, in all likelihood, differ from the more tonic activity produced during stance. The physical characteristics of the stimulus being presented to the foot during movement at ground impact will differ from those stimulus characteristics produced during stance. Some of the characteristics affecting the mechanoreceptor include stimulus intensity, speed of onset, duration and speed of unloading receptor, and the relative numbers of receptors activated. These characteristics are all very important in describing the physical features of the ground stimulus. As a result, the nervous activity created by these stimuli may be different in the same mechanoreceptors during two types of limb-loading behaviors. Furthermore, the sensations being perceived by the horse will provide important information about the ground surface, but the information generated by each activated receptor will provide slightly different data that vary in both quality and quantity.

Other receptors within the foot that respond to rapidly applied stimuli may not respond to other physical stimuli applied more slowly and for longer periods of time. These examples of the rapidly and slowly adapting receptors indicate that mechanoreceptors are differentially sensitive to the many stimuli being applied to the foot, meaning that one mechanoreceptor may be highly sensitive to and preferentially activated by one type of stimulus applied to the foot but relatively unresponsive to other types of applied sensory stimuli. Because of this, the horse can perceive different sensations within the foot when the foot touches the ground during a walk or trot versus when the horse is quietly standing while grazing. This difference in sensitivities of receptors during applied stimuli enables the receptors to be discriminating as they detect and gather meaningful information about the local environment. Such information may be important and helpful to the horse in negotiating ground surfaces during movement, whereas other sensory information may inform the horse to lift its foot when standing on a small rock, by making the position of the foot seem out of balance or uncomfortable. These response characteristics of the sensory receptors provide for selectivity, enabling the horse to appreciate and interact within its environment more easily.

In the horse, few of these sensory mechanoreceptors have been described, mainly owing to the veterinarian's interest in lameness and its association with pain. However, what types of sensory receptors are present and where they are located are very important to the horse, as the receptors aid in its ability to negotiate the ground terrain as well as to remain aware of its environment during stance. Equally important, however, is the fact that the activation of certain receptors and their sequence of activation are critical for the locomotor reflexes needed to maintain a smooth, coordinated, and effortless gait during movement.

Another broad category of sensory receptors is nociceptors, or those receptors that are activated by painful stimuli.[1,2,4,5] In humans, the sensation of pain is subjective and has been defined as an unpleasant sensory and emotional experience associated with possible tissue damage.[6] Obviously, this complex response may be more difficult to assess in the horse, but most practitioners would agree that a horse with a detectable lameness due to a foot problem is experiencing sufficient discomfort or pain to alter its movement pattern. This behavioral response can be called physiologic pain (rather than emotional pain), because the stimulus is likely activating the high-threshold sensory pain fibers, informing the horse that tissue damage may be occurring. Pain receptors are generally understood to be composed of fine, free nerve endings in the subcutaneous tissues, including the laminar dermis of the hoof wall and of the foot (see Figure 5-1). They seem to lack an organized physical structure surrounding the nerve terminal for detecting the painfully applied stimulus.

As determined experimentally, the sensations of pain are mediated in part by the activation of thin nerve fibers. A stimulus with a low intensity of electrical activity applied to the tissues will activate only the large-diameter nerve fibers initially, providing a touch-like, or tactile, sensation. If the strength of the electrical activity is increased, the smaller diameter nerve fibers are activated. These are covered only by a thin layer of myelin or by very little myelin. Such activation results in the appreciation of a more complicated and intense

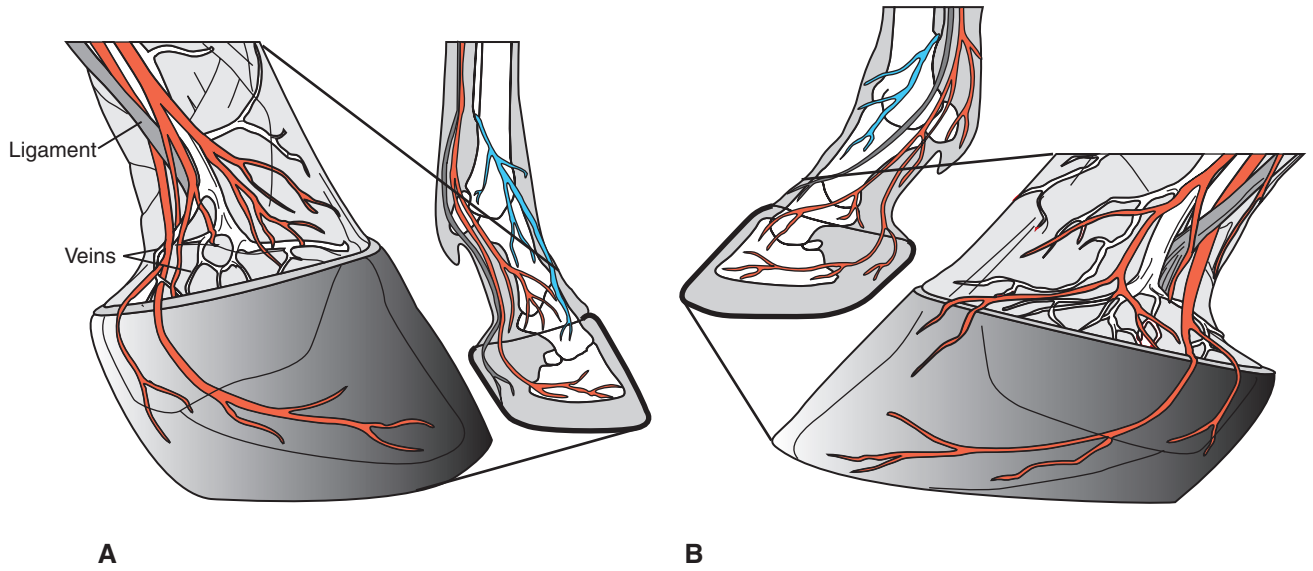

FIGURE 5-2 **A**, Schematic drawing of the medial palmar digital nerve showing the course of the nerves with the dorsal branch (*blue*) and variations (*gray*). **B**, Schematic drawing of lateral palmar digital nerve showing its course with the dorsal branch (*blue*) and variation (*gray*). *(Adapted from Kainer R:* The locomotor system. *In Stashak TS:* Adams' Lameness in Horses, *ed 5, Philadelphia, 2002, Lippincott Williams & Wilkins.)*

sensation recognized as pain. The application of local anesthetics around these same nerve fibers provides complementary information, as it enables us to differentially determine which nerve fibers are being desensitized. Local anesthetics will desensitize the unmyelinated nerve fibers conveying the pain sensations before it will desensitize the nerve fibers conveying touch and proprioception, which are heavily layered with myelin.

To complicate matters further on the issue of pain and the complex physiologic sensations that it engenders, the tissues surrounding the offending stimulus are often damaged, releasing other compounds and tissue products at the site of the injury. These released chemicals can then exacerbate the effects of the pain-inducing stimulus and the response characteristics of the nerves and hence the perception of pain. Such released compounds include inflammatory products, hydrogen and potassium ions, neuropeptides, enzymes (e.g., proteases, cyclooxygenase), and chemical mediators (e.g., histamine, bradykinin, cytokines, prostaglandins).[4,5,7-9] Such sensitization makes the lameness condition appear more painful under these conditions.

The actual make-up of the receptors and nerve fibers within the horse's foot structure and what the effects of local anesthetics are on these nerve fibers are the topics of this chapter. It is hoped that with a description of the distribution of the nerve fibers and the types of nerve fibers innervating the foot, as well as several of the receptors and sensory nerves conveying touch, proprioception, and other senses within the foot, we as "equine foot caretakers" can better appreciate how the foot interacts with the ground surface to convey information important to the horse for negotiating in its environment, as well as information that may be helpful to the veterinarian for diagnosing the site of the potential lameness.

These two systems of mechanoreceptors and nociceptors protect the foot and horse from potential injury and maintain proper functioning of the foot under all conditions. Often it is difficult to separate them during locomotive behavior of the horse because both contribute to the typical smooth gait of the horse. Although an alteration in the gait is usually attributed to the presence of a noxious stimulus within the foot tissues, on occasion the "lack of fluidness" in the movements with full extension or shortened strides of the limb is due in part to the incomplete activation of the sensory receptors in different regions of the foot. Once these sensory receptors are activated during ground contact, the entire forelimb can then become fully engaged in the locomotive reflex mechanisms to reach full extension and movement of all joints of the forelimb.

MACROSCOPIC DISTRIBUTION OF THE SENSORY NERVES TO THE FOOT

A detailed description of the sensory foot nerves is imperative given the clinical importance of innervation of the foot for diagnostic purposes. The detailed work of Sack[10] is one of the most informative and useful studies dealing with the distribution of these sensory nerves to the foot of the horse, and in this section many of Sack's important findings are summarized and interpreted.

The distal limb, specifically the foot, is innervated by a continuation of the medial and lateral palmar nerves that become the medial and lateral palmar digital nerves (PDN) distal to the metacarpophalangeal joint (Figure 5-2). Midway down the pastern, the medial PDN and the lateral PDN form dorsal branches that course superficially along the palmar digital vein, while the primary portion of the nerve continues along the deep digital flexor tendon, but palmar (caudal) to the palmar digital artery. In about one third of the cases, an intermediate branch arises from the dorsal aspect of the PDN to innervate the more superficial areas on the dorsum of the digit (see Figure 5-2).

The PDN and the accompanying artery pass under the ligament of the ergot as the ligament passes obliquely along the abaxial surfaces of the pastern. On occasion, a small

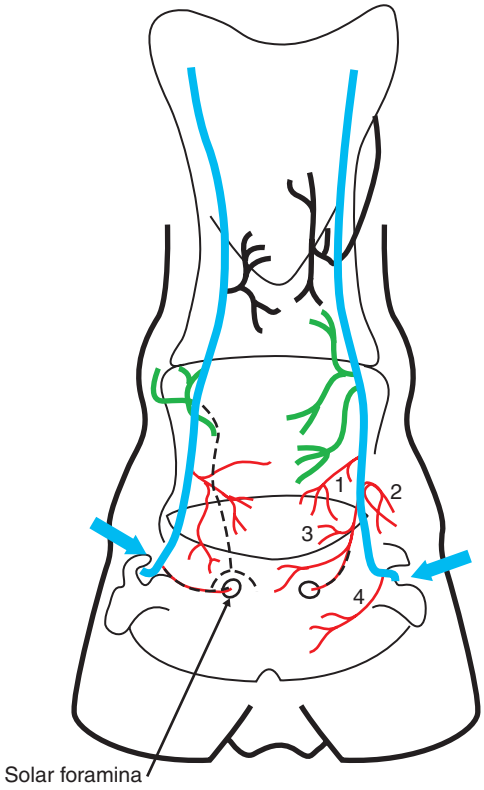

FIGURE 5-3 Palmar view of foot showing the palmar digital nerve to supply various structures of the distal limb. The green nerves supply the digital sheath, distal sesamoidean ligaments, and palmar pouch of coffin joint. The red nerves 1 through 3 innervate the palmar coffin joint, impar ligament, and proximal ligament to navicular bone, with nerve 4 supplying the navicular bursa. The dashed line represents the nerve that accompanies the artery of the digital cushion as it passes through the cushion to the frog and underlying sole. At the blue arrows, the parietal groove marks the distal continuation of the palmar digital nerve as they pass along side the coffin to supply the laminae at the quarters, dermis, and the coffin bone.

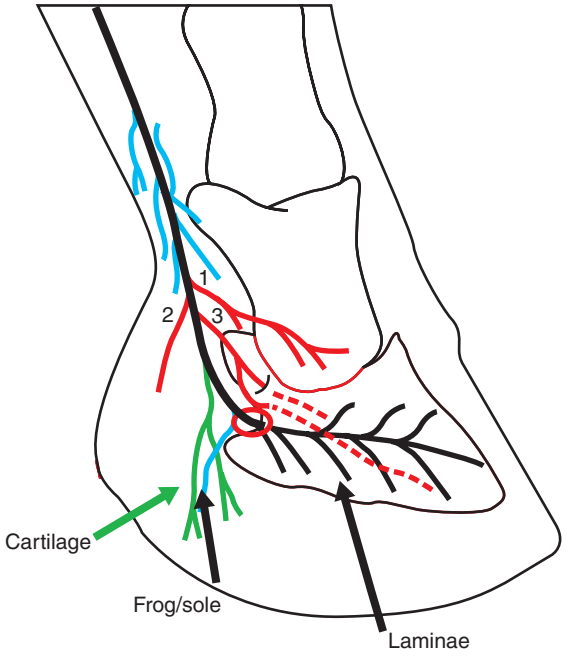

FIGURE 5-4 The medial palmar digital nerves course along the flexor tendon and digital artery to supply these and other structures associated with palmar foot (*blue*). The nerve courses through the parietal groove (*red circle*) to pass alongside the coffin bone. Nerve 1 supplies the coffin joint, nerve 2 passes to the flexor tendon, and nerve 3 supplies internal bone and sole under the coffin bone. The colored nerves supply the hoof cartilage (*green*), frog and underlying sole (*blue*), and the wall laminae at the quarters (*black*).

branch of the lateral PDN may pass through the ligament of the ergot. The distal continuation of the medial PDN courses to the distal phalanx and passes through the parietal groove on the coffin bone (Figure 5-3, *blue arrows;* Figure 5-4, *black nerve*) before branching and continuing along the external surface of the bone to supply the corium, epidermal laminae at the quarters, and most likely the dorsal regions of the toe overlying much of the distal phalanx. Also, small branches from this nerve pass around the border of the coffin bone to supply the sole corium.

Between this termination site of the medial PDN within the foot and the formation of its dorsal branch to the dorsal surface of the digit, several superficial branches are directed caudally to the skin over the mediopalmar areas and to the coria of the caudal hoof wall and the sole and frog. One of these branches exits at the midpastern level to pass along with the artery to the digital cushion to accompany the artery through the digital cushion to the corium of the frog (see Figure 5-3, *dashed line*). Continuing distally at the level of the lateral cartilage, another short branch leaves the medial PDN to supply the skin over the palmar parts of the lateral cartilage (see Figure 5-4, *green*), while a larger branch passes into the laminar corium of the hoof wall at the heel and to the caudal parts of the quarter (see Figure 5-4, *black nerve*). Just before the medial PDN enters the parietal groove, small branches are also given off to the corium of the sole and to the frog (see Figure 5-4, *blue*).

Regarding many of the deeper branches of the medial PDN between the metacarpophalangeal joint and the lateral cartilage, several branches are given off to innervate the palmar regions of the pastern, including the palmar synovial pouches of each of the joints and the distal sesamoidean ligaments (straight and oblique sesamoidean ligaments) and distal suspensory ligaments associated with the support of the distal sesamoid bone (navicular bone) (see Figures 5-3 and 5-4). Three to four small but important nerves leave the medial PDN to supply the structures on the deeper and palmar surfaces of the foot, including the distal interphalangeal (DIP) joint and the midline of the distal sesamoidean impar ligament (DSIL) (see Figure 5-3, *red nerves*). A small branch accompanies the digital artery into the solar foramen of the distal phalanx.

The lateral PDN is not a mirror image of the medial PDN (see Figures 5-2 and 5-3). There are four branches distal to the pastern joint, with the first branch innervating the skin on the lateral side of the hoof cartilage and passing through the digital cushion to terminate in the corium of the frog. Another branch supplies and innervates the corium of the frog, sole, and bar, with one branch passing into the digital cushion (see Figure 5-3, *dashed line*). The remaining two branches enter the laminar corium on the quarter and into the corium of the sole before the lateral PDN ramifies in the sole corium. There appear to be fewer branches of the lateral PDN than of

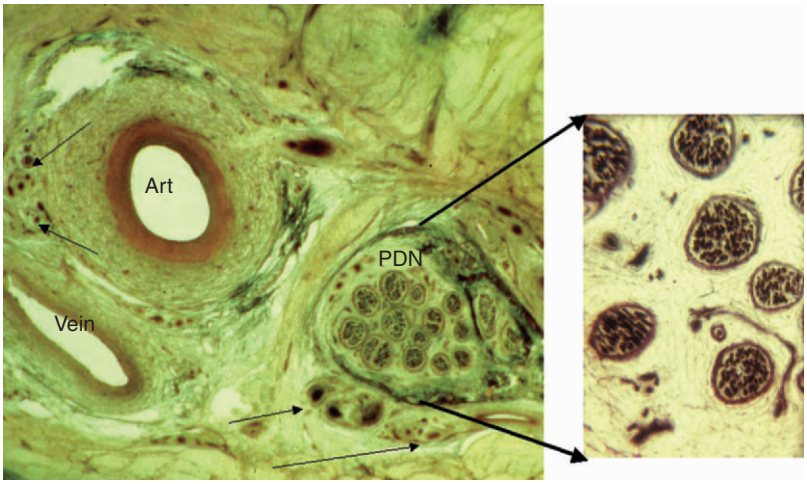

FIGURE 5-5 Tissue section stained to reveal blackened nerve fibers of the medial palmar digital nerve with both thick nerves for fast nerve conduction and thin nerves that convey sensory information slowly. *Art,* medial palmar digital artery and accompanying vein. Small arrows point to other nerves. At right, a high-power view of nerves of the medial palmar digital nerve (PDN).

the medial PDN to the foot innervating the deeper tissues along the mediopalmar surface of the foot. Just proximal to the navicular bone, a branch is given off to the palmar pouch of the DIP joint as well as the digital sheath (see Figure 5-3, *red*). A smaller nerve courses along the artery that enters the solar foramina to innervate the distal phalanx.

Regarding the innervation of the deep and more internal structures of the foot, several branches of the medial PDN and the lateral PDN provide the sensory innervation to the DIP joint, including the DSIL and the navicular bone.[10-12] From the medial PDN at the level of the pastern, a branch exits to innervate the palmar surfaces of the synovium and capsule of the DIP joint (see Figure 5-3, *nerve 2,* and Figure 5-4, *nerve 1*), while at the level of the lateral cartilage, nerves enter the DSIL to supply the navicular bone, parts of the DIP joint and navicular bone, and the palmar portions of the distal phalanx (see Figure 5-3, *nerve 3,* and Figure 5-4, *red nerves*). Another branch passes external to the navicular bursa before a small nerve bundle passes into the solar foramina.

Within the lateral PDN, branches passing superficially and palmarly leave the pastern level with the artery to the digital cushion as they pass through the digital cushion to innervate the corium of the frog. A second branch also innervates the corium of the sole, frog, and bars. There are fewer sensory nerves fibers in the lateral PDN than in the medial PDN. At the level of the pastern, a branch innervates the palmar pouch of the DIP joint as well as the navicular bursa. Farther distally, a small branch courses along with the small artery entering the solar foramina laterally. Importantly, from a clinical perspective, the sensory nerves innervating the navicular zone—the navicular bone, its suspensory ligaments, and insertions of the DSIL and the deep digital flexor tendon onto the coffin bone—form continuations that pass through the solar foramina to supply the majority of the distal phalanx with its bone structure and vessels.

The dorsal branches of the PDN arise a short distance proximal to the metacarpophalangeal joint, and it usually receives one or more branches from the digital nerve, which appear to form a variable plexus as they become part of the dorsal branches. These branches for the most part are cutaneous, supplying the dorsal surfaces and abaxial sides of the pastern and the middle phalanx. These superficial branches also send deeper branches to the dorsal pouch of the pastern and fetlock joints, with branches also extending to the lateral cartilage and into the laminar corium of the heels and quarters. The coronary corium is innervated by the dorsal branches of the PDN. Interestingly, the remainder of the hoof wall laminar corium at the toe is innervated primarily by the medial and lateral PDN.

TYPES OF NERVES WITHIN THE FOOT

The structural features of the medial and lateral PDNs to the equine foot are very important sensorily to the horse, because they aid in the horse's ability to negotiate the varied terrains of its environment. Structurally, they carry fibers similar to the peripheral nerves found in other species, with the PDNs containing many more thinly myelinated and unmyelinated nerves than the thicker myelinated nerve fibers (Figure 5-5).[12] Thickly myelinated fibers convey sensations rapidly to the spinal cord at 6 to 30 meters per second, which is critically important during the faster gaits because the sensory information must become integrated into the locomotive reflexes as the forelimbs are extended and the muscles contract to support weight. In contrast, thinly myelinated fibers convey sensation at a rate of less than 2 meters per second.

In the medial PDN, there is a ratio of four unmyelinated fibers to every one myelinated fiber (Figure 5-6). The unmyelinated nerves consist of both sensory nerve fibers and sympathetic postganglionic nerve fibers, with the latter making up approximately 25% of the unmyelinated nerve fibers within a peripheral nerve. Thus, three fourths of the unmyelinated nerve fibers to the equine foot are sensory in nature and, for the most part, contain peptidergic neurotransmitters. These sensory unmyelinated nerves convey a variety of sensations to the spinal cord, with nociceptors (stimuli that create tissue damage) and high-threshold mechanoreceptors accounting for most of these nerves.

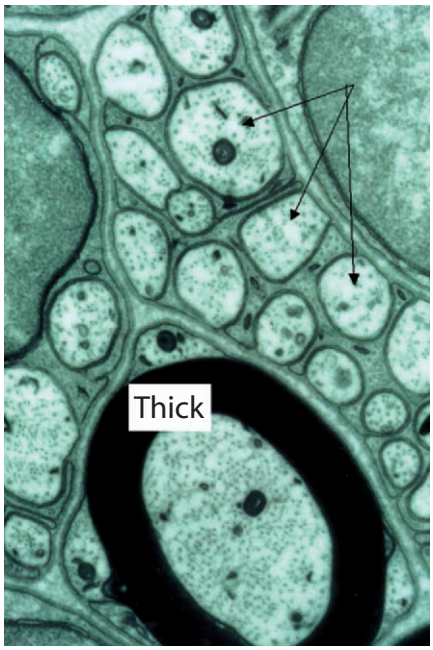

FIGURE 5-6 Electron micrograph showing many unmyelinated fibers (*thin arrows*) for every thick nerve fiber (*thick*). The thick nerves are important for conveying information about the ground during running, whereas the other, thin nerves are needed for pain information.

The sensations conveyed via these unmyelinated nerve fibers and the low-threshold tactile and proprioceptive sensations conveyed via the thicker myelinated nerve fibers are perhaps most important from the perspective of the horse and the veterinarian. It is these types of myelinated nerves that contribute to and influence the locomotive pattern during the movement of the horse when it is galloping over the uneven terrain of the countryside, whereas the nonmyelinated nerves convey the pain sensations that are so important during a lameness examination. Both types of nerve fibers definitely contribute to movement patterns during locomotion of the ground surfaces.

Although some of the myelinated nerve fibers convey painful sensations, such as those that may be important for certain protective or withdrawal reflexes, most of the larger diameter fibers are responsible for conveying information regarding the mechanical deformation of foot tissues during ground impact during locomotion as well as during stance. Other potential mechanoreceptors become active during the slow changes within the foot at this time.

Regarding temperature sensation, the horse foot seems to be less responsive to this sensation unless the foot is subjected to extreme temperatures for long periods of time.

Chemically Identified Nerves within the Equine Foot

Within the last 40 years or so, our understanding of neurotransmitters has grown immensely in terms of how they affect the cell membrane, intracellular organelles, and cytosolic transport and other intracellular processes. Early studies focused on the small molecular agents dealing with rapidly acting processes that usually produced the more acute responses in the nervous system, such as the transmission of sensory signals from one neuron to the next within the spinal cord or other part of the brain. Examples of the small-molecule types of transmitters include acetylcholine, norepinephrine, purines, and other biogenic amines. Historically, one of the most studied areas has been the neural control of the vasculature by noradrenaline (norepinephrine) and its release from the perivascular postganglionic sympathetic nerves. During the last half of the 20th century, however, evidence was beginning to accumulate indicating that there are other chemical agents acting like putative neurotransmitters that control the blood flow of many vascular beds but that are not adrenergic or cholinergic in nature. During this time period, many different putative neurotransmitters, neuromodulators, and chemical agents were discovered to innervate the vessels, ranging from proteins to the large peptides.[13-15]

The term *neuromodulator* has often been used to denote a chemical compound released from a nerve terminal that affects the adjacent neuron or nerve terminal only by increasing or decreasing its membrane excitability, that is, making it more or less excitable, respectively, rather than inducing an excitatory (or inhibitory) synaptic potential. Most of the neuropeptides fall into this category. The generation of a synaptic potential—a depolarization of the adjacent neuron—is one of several characteristics of a neurotransmitter.[4,5] In addition, during the middle and late 1970s, many nerve fibers were discovered to contain more than one neurotransmitter, thereby adding to the complexity of our understanding of neural control of the blood vessels. These findings suggested several possibilities, in that the transmitters may be released independently from each other or coreleased simultaneously from the same nerve fiber. Today, it is widely accepted that most nerve fibers have more than one neurotransmitter contained within them, rather than only one transmitter substance.

It probably would be more difficult to make a case that a nerve fiber had *only* a single putative neurotransmitter contained within its terminals rather than multiple neurotransmitters. Once these two or more transmitters are released from the same nerve fiber, they may act upon different targets within the local environment or act together synergistically to augment the effects of these two neuroactive agents. For example, the peptide vasoactive intestinal peptide has been shown to be a cotransmitter with acetylcholine in parasympathetic nerve fibers. When released together, vasoactive intestinal peptide is known to have a dilatory effect on the blood vessels supplying the salivary glands, while acetylcholine facilitates the secretory action on the glandular cells of this organ.[16] Similarly, in sympathetic nerves, the peptide neuropeptide Y has been shown to coexist and be coreleased with noradrenaline to promote constriction of a vascular bed.[17] To date, there are numerous examples of nerve fibers having multiple transmitters contained within them, and these findings represent a rapidly expanding field.

In the horse foot, several of these transmitters have been reported and are the focus of this discussion.

Acetylcholine, Biogenic Amines, and the Neuropeptides

Blood flow to the foot is regulated by different physiologic mechanisms, including neural and physical pressures, depending on the locomotive behavior that the foot is engaged in. At

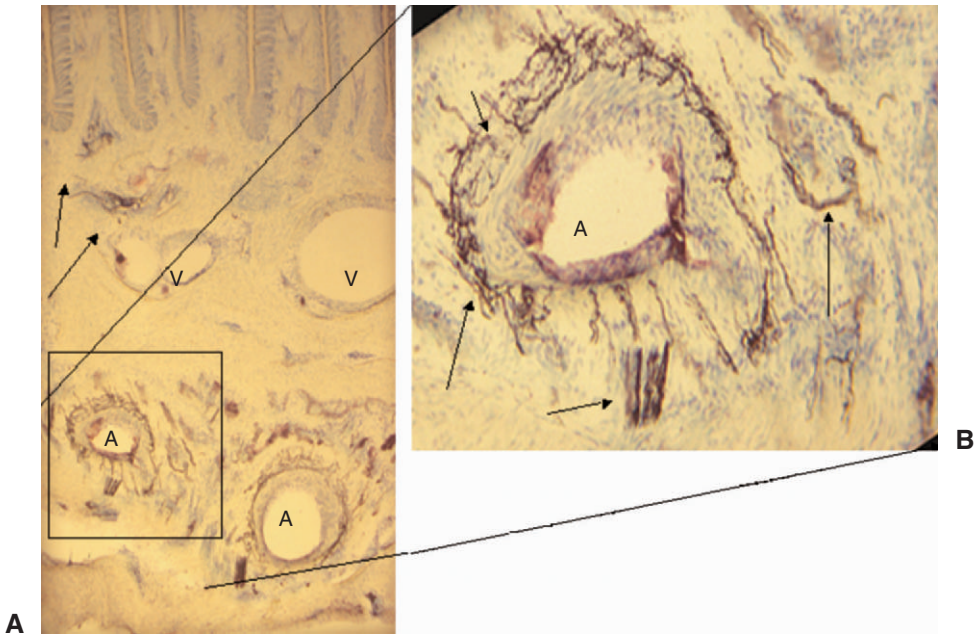

FIGURE 5-7 Immunocytochemical staining of corium in dorsal hoof wall showing distribution of noradrenaline being concentrated around arteries *(A)* with less around veins. Note the many black nerve fibers around vessels *(V)*.

certain times, blood flow through the foot encompasses virtually all areas of the foot, including the corium of the hoof wall and sole and the vasculature within the tissues of the palmar foot, such as during postural changes. Or, it can be redirected toward the palmar foot during limb movement, as in a rapidly locomoting animal. At other times, it may even be restricted to a small region of the foot, such as with activation of the arteriovenous anastomoses in the corium of the dorsal hoof during thermoregulation in extreme cold, or during any inflammatory process with activation of the axon reflex. The exact mechanisms of how blood supply to the foot can be regulated by physical pressures when the foot interacts with the ground during stance and movement or by changes in neuronal activity are not as yet fully appreciated and understood. This subject needs to be studied in greater detail.

Interestingly, although there appear to be many neurotransmitters within nerve fibers, as well as circulating vasoactive agents, that have both direct and indirect effects on the luminal diameter of the vasculature within the equine foot, these neuroactive agents all work in concert to produce the desired physiologic effects, rather than one putative neurotransmitter being the lone agent to control blood flow through the foot. Within the foot, vasoconstriction is usually mediated by noradrenaline when it is released from the sympathetic nerve terminals. Several studies have documented the locations of noradrenergic nerve fibers within the equine foot (unpublished observations).[18]

Noradrenaline originates from the sympathetic paravertebral ganglia to innervate the smooth muscles of the larger arterial and venous vessels within the foot. In Figure 5-7, *A*, the large arteriolar vessels within the corium of the dorsal hoof wall are densely supplied by the noradrenaline, with the veins appearing to have less adrenergic innervation. In Figure 5-7, *B*, the noradrenergic fibers can be seen to extend into the laminar dermis to innervate the smaller arteriolar vessels. The arteriovenous anastomoses are also innervated with this neurotransmitter.[18] Once released, noradrenaline acts through alpha adrenoceptors to produce vasoconstriction, which is directly related to the noradrenaline release.[19] Several studies have examined the effects of infusion of the catecholamines (noradrenaline and dopamine) through digital arterial and venous vasculature in the horse with similar vasoconstrictive results (unpublished observations).[20]

The peptide neuropeptide Y has been shown to be colocalized with noradrenaline and shares many of its vasoconstrictor properties.[17,18] The vasodilation of the vessels within the foot, on the other hand, may be more complicated because it is influenced by several processes—the direct physical forces acting on the foot, active neuronal control of luminal diameters of the large and smaller vascular beds, and more passive variations in the foot environment, such as temperature variations and changes in the local ionic and chemical tissue environment. Part of the vasodilatation occurs by the progressive inhibition of the vasoconstrictor alpha-adrenergic nerve fibers surrounding the arterial and venous vessels of the foot.

With an increase in temperature in other species, gradual dilation changes in vascular smooth muscle tone occur, especially in the hand and foot of humans and presumably in the foot of the horse. In the horse, the effects of temperature extremes in altering blood flow through the foot are evident clinically by the apparent constriction of the foot vasculature and the increased incidence of laminitis during severe winter cold spells in the Northern hemisphere. There is also an active noradrenergic vasodilating mechanism that appears to occur via the beta-adrenergic nerve cells and fibers.[14] Whether they occur in the horse remains unknown at this time.

Two other active mechanisms that likely exist in the equine foot, however, and promote or at least contribute to vasodilation are (1) acetylcholine and antidromic stimulation of nerve fibers and (2) nonneural vasoactive substances. Acetylcholine has long been known to produce a fall in peripheral

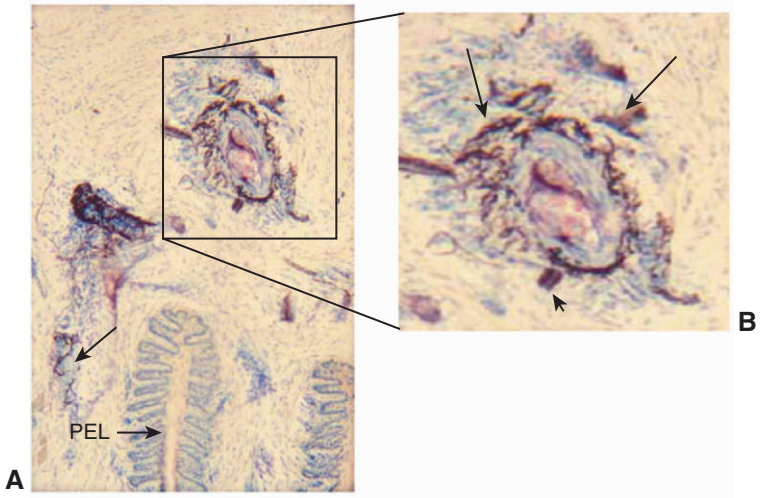

FIGURE 5-8 Photomicrographs showing section stained immunochemically with neuropeptide Y. **A,** The stained nerve fibers surround the arteries of the corium of the dorsal hoof and extend between the primary epidermal laminae (PEL). **B,** Higher power view shows stained nerves (*arrows*) surrounding a small artery.

resistance by dilating arteries when administered in vivo. This relaxation of the smooth muscle vasculature is thought to occur via the endothelial cells lining the blood vessels. The endothelium-derived relaxing factor (nitric oxide) in turn diffuses to the smooth muscles to cause relaxation. Similarly, one of several neuropeptides can be released from the peripheral sensory nerve terminals into the microvasculature to cause vascular bed dilatation, whereas the other non-neural tissue substances, including histamine released from mast cells, bradykinin, prostaglandins, and leukotrienes, contribute to a more localized vasodilatation process.[7,15]

The biogenic amine serotonin (5HT) has been localized to a number of different blood vessels within the body, primarily in the cerebral vasculature and smaller subpial vessels. These 5HT-containing fibers appear to arise from both the 5HT-rich cell groups of the brain (raphe nuclei) and the superior cervical ganglion.[21] Other vessels believed to have a 5HT innervation include the gastrointestinal tract and the vasa nervorum of certain large nerves. They have not been described to be present in the vessels of the horse foot. Activation of specific receptors for 5HT is a very potent vasoconstrictor, even more so than noradrenaline (unpublished observations).[14,20,21] Within the vasculature of the brain, activation of the 5HT receptors may be important in controlling vascular permeability and cerebral spinal fluid production, as well as detecting changes in tissue composition.

Within the equine foot, experimental infusion of 5HT produces a profound and long-lasting vasoconstriction.[20] Although the 5HT receptors may be present within equine foot vessels, or at least a receptor that is activated by 5HT, reasons for the intense vasoconstriction and their exact role in regulating noncerebral foot vessels are not known.

Dopamine is the immediate precursor of noradrenaline and adrenaline and also possesses pharmacologic properties. Commonly, its usefulness occurs by administering low intravenous doses to improve cardiac output and blood pressure during anesthesia or to reverse atrioventricular blocks that are nonresponsive to atropine.[22] In small animals and humans, dopamine is often used to improve perfusion of the kidneys during failure. In humans, however, dopamine is a potent constrictor of vasculature within the hand. The effects of dopamine on the vasculature are mediated through several types of receptors, including noradrenergic (beta-1 and alpha-1 adrenoreceptors) and dopamine (DA type 1 and DA type 2) receptors. In the horse foot, dopamine produces a profound digital vasoconstriction, but only at high molar concentrations, presumably through the alpha-1 adrenoceptors.[20] At lower molar concentrations, however, its vasoconstrictive properties are far less potent than those of noradrenaline, serotonin, and other agents.

In addition to the transmitters noradrenaline, dopamine, serotonin, acetylcholine, and the above-mentioned neuroactive compounds, the peptide neuropeptide Y is one of several vasoactive peptides that can have a profound effect on blood vessels in controlling blood flow to a tissue region, in this case the equine foot. Neuropeptide Y is present and widely distributed in the distal limb, being colocalized with noradrenergic nerve fibers and thereby paralleling the noradrenergic innervation of the vascular system (Figure 5-8). The peptide in the horse is located along the large palmar digital artery and vein to the foot as well as some of the smaller vessels (unpublished observations).[18,23] This peptide is a potent vasoconstrictor with a very long lasting action that potentiates the effects of noradrenaline. Its longer lasting vasoconstrictive effects are apparently produced by "uncovering" more of the alpha adrenoceptors, thereby making many more receptor sites available. Two other peptides present in the equine foot include peptide histidine isoleucine and vasoactive intestinal peptide. These two peptides are structurally similar, although their appearance varies somewhat among the animal species, and they often are associated with the parasympathetic ganglia. Although these peptides functionally produce vasodilatation in other animals, their potential role in the vasculature of the foot is not known.

With the correlations of the sensory modalities and the chemical identities of the specific fiber types, substance P, neurokinin A, and CGRP are the peptides that have been most extensively studied and are only beginning to be understood. Substance P appears to be the most common peptide present in the sensory nerve fibers in the foot, as it

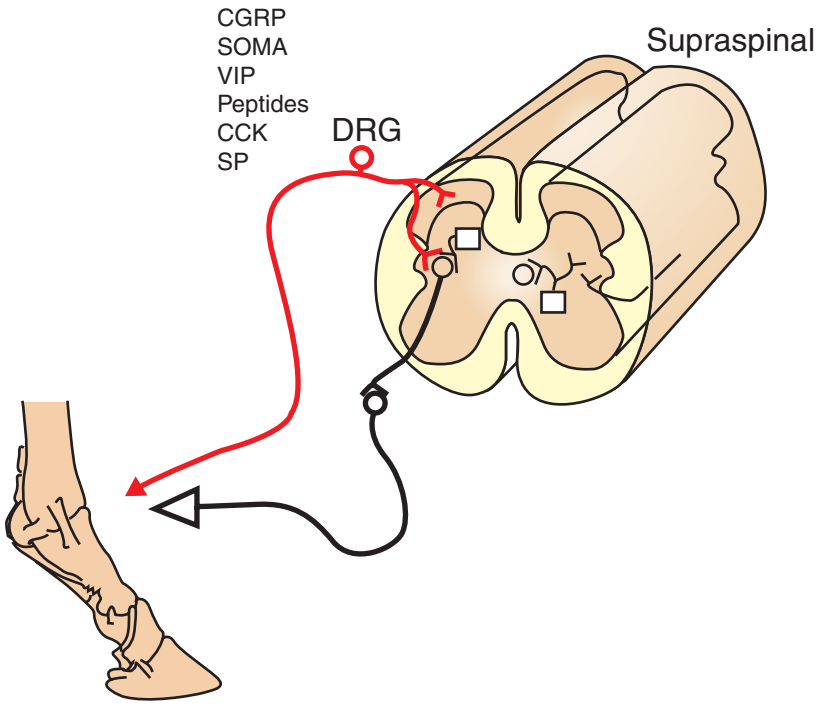

FIGURE 5-9 Schematic drawing showing sensory nerve from the horse limb to the spinal cord with many peptides (calcitonin gene–related peptide, somatostatin, vasoactive intestinal peptide, cholecystokinin, and substance P) present in the dorsal root ganglion. The other nerves represent sympathetic nerve blood vessels.

accounts for approximately 25% of the cells in the dorsal root ganglion (Figure 5-9). Whenever a noxious stimulus affects the foot tissues, the nerve activity is orthodromically transmitted toward the spinal cord and then via multisynaptic pathways to the brain for perception of the noxious stimulus. These afferents of the spinal nerve are classified as pain fibers and are composed of finely myelinated and unmyelinated nerve fibers, with the latter comprising the majority (40% to 90% in different animals) of the sensory afferents to the spinal cord.* The release of substance P from the afferent fibers into the spinal cord gray matter forms an important link in how the nervous system begins to consciously perceive pain.

During stimulation of these sensory nerve terminals, substance P is released from the peripheral nerve endings. The release of substance P is carried out in part by the "axon reflex," which is the impulse conducted in an orthograde manner from the periphery to the spinal cord (Figure 5-10). Along the afferent fiber, alterations in local ion concentrations occur at branches of collateral nerves that induce another action potential in the collateral nerve, which in turn is conducted toward the periphery antidromically or toward the terminal of the peripheral nerve.[9,26,27] At the nerve terminal, the SP is released into the tissues, causing inflammation, plasma extravasation, vasodilatation, and further release of histamine from mast cells within the tissues. These non-neurally released substances include potassium ions from damaged cells, histamine, bradykinin, and other inflammatory mediators that enhance the neurogenic effect of stimulating the sensory nerves. These secondary substances then have an added effect of inducing prostaglandin formation, which further exacerbates the effect on the sensory nerve terminals.

*References 2, 4, 6, 9, 24, 25.

This release of substance P from the peripheral sensory nerve terminals has direct effects on the microvasculature within the foot, as there are specific tachykinin receptors present on the microvasculature within various tissues.[28] Substance P acts on the tachykinin receptors—neurokinin 1, neurokinin 2, and neurokinin 3 receptors[29]—to produce a potent vasodilatation via an endothelium-derived relaxing factor. This endothelium-derived factor is now known to be nitric oxide.[30,31]

With the release of these peptides from the peripheral nerve fiber, several things are apparent: (1) the produced antidromic vasodilatation occurs mainly in cutaneous-related tissues and not in muscle (although infusions of muscle with substance P agonists will have a direct effect); (2) these dilatation effects are not transmitted by descending nerve fibers from the central nervous system of the spinal cord; (3) the effects are independent of the sympathetic nervous system; and (4) the vasodilatation effects last much longer than the nervous stimulation. Vasodilatation within the skin and presumably the equine foot occurs when the applied painful stimulus directly activates the peripheral endings of the sensory nerve fibers (unmyelinated nerve fibers) and indirectly cause the release of local substances.

In addition to vasodilatation, the release of substance P from the sensory nerve fibers produces an increased permeability of the microvasculature to plasma proteins. This inflammatory effect is neurogenic and is dependent on the interaction of the substance P contained within the sensory nerves and the local inflammatory mediators, including histamine within mast cells, bradykinin, and potassium ions, as mentioned earlier.[7,15] Once substance P is released from the nerve terminals, it acts on nearby mast cells to release histamine. This inflammatory process continues to spread as the released histamine further stimulates other sensory nerve

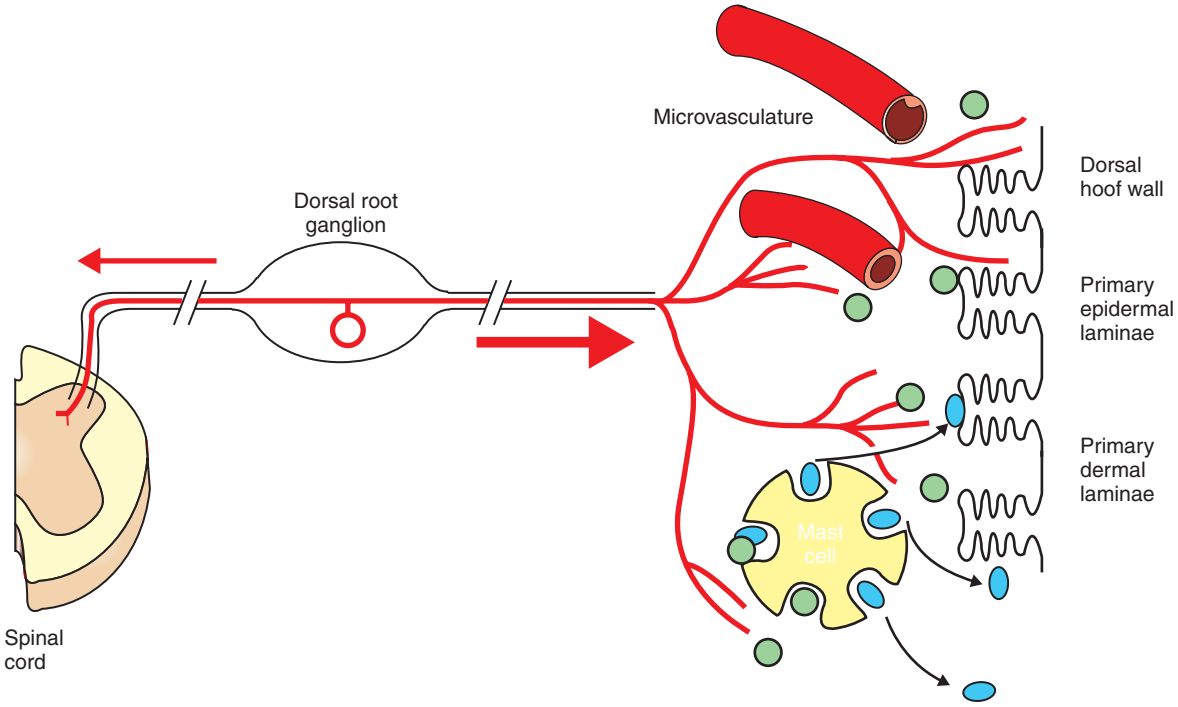

FIGURE 5-10 Avon reflex. Activation of substance P nerves results in the release of substance P (*green*) from the nerve, which then has an effect on blood vessels (*red*) and mast cells with the release of histamine (*blue*) to cause vasodilatation and the beginning of inflammatory process.

endings in adjacent tissues. This cascading effect of mutual nerve activation and histamine release prolongs the inflammation as the process spreads to the adjacent tissue sites.

Substance P may also contribute to the local migration and infiltration of leukocytes and macrophages into the inflamed tissue. Although not completely understood, each of the sensory peptides appears to play a unique role in mediating its effect within tissue, which ranges from control of blood flow through the microvasculature to plasma extravasation to interactions with the immune system to promote the inflammatory process. Two other colocalized transmitters, CGRP and neurokinin A, also produce a profound dilatation of the larger vessels and of the smaller microvasculature. The CGRP peptide can produce vascular dilatation even without an intact vascular endothelium, indicating a different vasodilatation process than that of substance P. Most interesting is the observation that CGRP is colocalized with substance P in most substance P–containing sensory nerve fibers and that CGRP is a potent inhibitor of substance P endopeptidase, an enzyme that inactivates substance P.[32] It is believed that when both substance P and CGRP are released from sensory nerve fibers, the CGRP potentiates the effects of substance P on the microvasculature by prolonging the half-life of substance P with the vascular dilation.

The neurokinins NK1 and NK2 are very similar pharmacologically to substance P, although there appear to be subtle differences in various tissues, with relatively little NK2 being present in the foot.[28] In the horse distal limb, substance P, CGRP, and neurokinin A are also plentiful, especially in the bones of the metacarpophalangeal joint, and the synovial linings and joint capsules of the carpus and distal sesamoid bone.* In naturally occurring arthritis of the equine carpal joint, the concentration of substance P is significantly increased, suggesting that it has a definite role in the inflammatory processes of horses. Neurokinin A and CGRP are colocalized with substance P in the sensory nerve fibers and are located in the medial and lateral palmar nerves and the medial and lateral palmar metacarpal nerves as these nerves course along the palmar surface of the canon bone. The medial PDN and the lateral PDN also contain all three of these sensory peptides.

Most of these peptides have been found to be associated with the tissues and microvasculature of the equine foot. The navicular bone and the distal phalanx have been shown to have these peptides course within nerve fibers to the peripheral zones bordering the articular cartilage on the surface of these joints,[11] through the DSIL and the deep digital flexor tendon, through the medullary cavities of each bone, and throughout the corium of the hoof wall.[18,23] Each of these peptides is mediated by local but specific tachykinin receptors located on the endothelium of the microvasculature of these many and various tissues. Such peptidergic receptors are present throughout the foot and are located in different tissues. For example, the peptide SP has extensive receptors in the joint capsule of the DIP and the navicular bursa as well as virtually all of the microvasculature in the corium of the hoof wall and the remainder of the foot. These substance P receptors provide a morphologic indicator that substance P present in the sensory nerve terminals will have an effect on the vasculature within the equine foot.

Substance P has a profound dilatory effect on the vessels within the equine foot when infused experimentally. Such vasodilatory effects of this peptide, along with the vasoconstrictive effects of the adrenergic nerves, could potentially

*References 11, 12, 18, 23, 33, 34.

provide an exquisite mechanism to regulate blood flow through different regions of the foot. In conjunction with the other peptides, these released substances bind to their specific receptors to promote inflammatory processes within the foot tissues.

PROPRIOCEPTION AND LOCOMOTION AND STANCE

As indicated earlier, the somatic sensation that seems important to the horse is the mechanoreceptive sense dealing with touch and position sensations of the foot that is activated by physical displacement of its tissues. Touch, or tactile, sense includes touch, pressure, vibration, and tickle senses, whereas position sense deals more with static positioning and rate of movement. Proprioceptive sensations include many of these sensations, as they have to do with the physical state of the foot, including its position in space, sensations from the tendinous and ligamentous insertions, and pressure sensations arising from the solar surface of the foot, including the frog, as well as those originating between the hoof wall and the bone of the distal phalanx.

Although touch, pressure, and vibration are often classified as different sensations, they are all detected by very similar types of sensory receptors. Some differences are apparent, however: (1) touch sensation usually results from the activation of receptors located superficially under the skin near the coronet or perhaps superficially within the corium of the softer keratin of the frog; (2) pressure sensation generally results from the greater physical deformation of tissues where these receptors are located; and (3) vibration sensation results from their activation by rapidly repetitive sensory stimuli. In the foot, these vibration detectors also adapt quickly and cease to fire with repetitive application of the stimuli.

In the horse foot, there are probably several different types of tactile receptors, most of which have not been described yet. However, different foot areas are currently being examined for such tactile receptors and it is likely that several of these will be discovered shortly. By realizing the environmental situations that the horse's foot encounters, it is possible to begin to search the tissues for these types of sensory receptors.

First, the many free nerve endings within the skin overlying the coronet are capable of detecting light tactile sensations. These tactile stimuli can be provided by insects lighting on or grasses brushing up against the foot or by other possible stimuli that the horse may sense and incorporate into the information needed to be transmitted to the central nervous system. Second, because the horse stands for long periods of time with approximately 25% to 30% of its load on the foot, there must be some type, or types, of pressure receptors located more deeply within the foot dermal tissues surrounding the distal phalanx. These receptors would be capable of detecting deep pressure changes, such as during walking once the foot is positioned on the ground surface. During this moment, the distal phalanx would move within the hoof capsule, activating the deep pressure receptors around the bone and the hoof capsule, which would transmit an initially strong signal to the spinal cord about the sudden change in position with placement of the foot, followed by a partially adapting response as the horse became stationary during stance. This information would provide some sensory signaling during weight shifting and limb movement as well as information on the steady state of the foot and its internal environment during stance.

At the same time, however, other sensory receptors would be activated during the strong tissue deformation when load is applied to the foot tissues during stance, but these receptors would provide continuous and steady-state signaling to the spinal cord about the high loads and would not adapt very rapidly. Together, these different types of mechanoreceptors would provide valuable information about potential changes and about the constant and steady-state environment of the internal foot tissues, which would then inform the spinal cord and engage its reflex mechanisms to adjust or adapt as needed.

The final type of sensory receptor is one present in the superficial and deep tissues that responds only by a very rapid movement of the tissues, such as during ground impact of the foot during locomotion, and ceases to respond quickly to continuously applied stimuli. Together, these sensory receptors enable the horse to engage and interact with its environment transiently and continuously.

The many and different types of sensory receptors present within the horse foot are not evenly distributed throughout the tissues. Some, such as the pain detectors, are distributed throughout much of the foot, but other sensory receptors are concentrated in specific regions of the foot, suggesting that the foot was constructed to have them preferentially activated.

The pacinian corpuscle is a rapidly adapting receptor that responds to transient mechanical changes in the environment when the foot impacts the ground (see Figures 5-1 and 5-11). The corpuscle is very sensitive to repetitive stimuli applied several times per second. With maintained compression of the foot, the pacinian corpuscle will generate a discharge during initial compression with ground contact and then cease to fire during stance, but will discharge again when the compression of the foot is lifted, creating an "on" and "off" response. Generally, the pacinian corpuscle is supplied by a myelinated nerve of larger diameter, which indicates a rapid transmission from the periphery to the spinal cord. The large pacinian corpuscles are located in the palmar areas of the foot: the bulbs of the heel and across the width of the frog palmarly (Figure 5-12). These proprioceptive receptors are up to 1 mm in diameter and are usually clustered in small groups of two to three corpuscles. These large types of mechanoreceptors are not evident in many other areas of the foot, including the sole toward the toe or along the hoof wall. These observations suggest that perhaps the foot is designed to have initial ground contact in this palmar region rather than toward the toe.

Another smaller group of these sensory receptors is also positioned internally (or axially) to the lateral cartilages at a more proximal level of the foot. This area next to the lateral cartilages and the one associated with the bulbs of the heels are two zones of greater movement of the foot. Both receptors are surrounded by loose connective tissue, which is consistent with the compression loading during ground contact initially and during the outward expansion of the lateral cartilages. The positions within the connective tissue of the palmar foot suggest that these two pacinian corpuscle types may respond to transient pressure changes, either positive or negative, within the foot. During heel contact and compression of the heel bulbs, the internal pressure at the heels will in

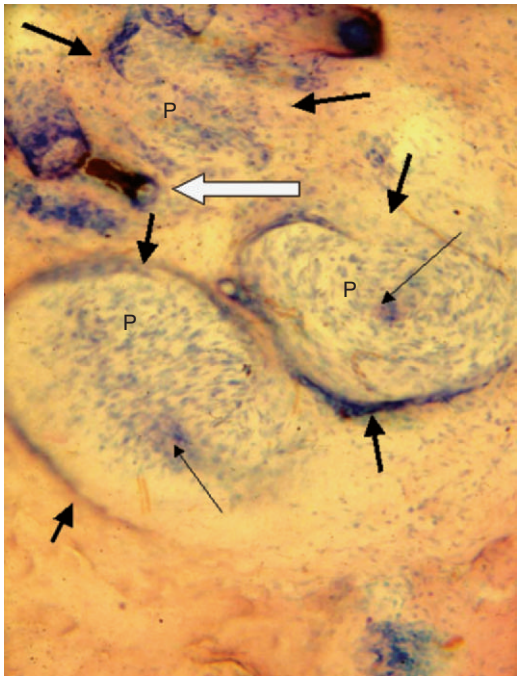

FIGURE 5-11 Section from palmar foot showing an example of a cluster of pacinian corpuscles (*P*), a type of mechanoreceptor that responds rapidly and transiently to stimuli at ground impact. This sensory receptor is up to 1 mm in diameter and is encapsulated (*thick arrows*) with a centrally located nerve (*small arrows*). Thick myelinated nerve fibers are also evident in section (*white arrow*). At ground contact, the sensory information is rapidly transmitted to spinal cord to become incorporated into locomotive reflex mechanisms.

all likelihood be positive. On the other hand, with the lateral expansion of the palmar foot, the pacinian receptors near the lateral cartilages may respond transiently to the negative pressure present within the digital cushion when the foot is on the ground.

Further proximally is another group of lamellated or pacinian sensory receptors associated with the secondary tendon of the deep digital flexor muscle that inserts onto the distal abaxial surface of the middle phalanx (Figure 5-13). During the initial phase of ground contact and the contraction of the deep digital flexor muscle and its tendons (the primary one inserting upon the flexor surface of the distal phalanx and each of the two secondary limbs attaching to the abaxial side of the middle phalanx), these tendons would tighten, producing a sudden pull of the surrounding loose connective tissues and hence activating these corpuscles. With their locations and their rapid but transient response characteristics, these sensory receptors may respond during the quick extension (dorsiflexion) of the digit immediately prior to the initial ground contact of the heel bulbs in a healthy footed horse. During stance, they may respond to the rapid movements of the fetlock joint when the horse shifts its weight or lifts its leg to drive insects away from its distal limb. These two sites suggest that the sensory receptors are involved in rapid changes in limb position during locomotion—extension of the digit prior to initial ground contact, flexion of the fetlock joint, and during the initial phases of ground contact and stance.

The presence of many clusters of lamellated corpuscles proximal to the ligament supporting the navicular bone suggests that this region may be a critical site for the percep-

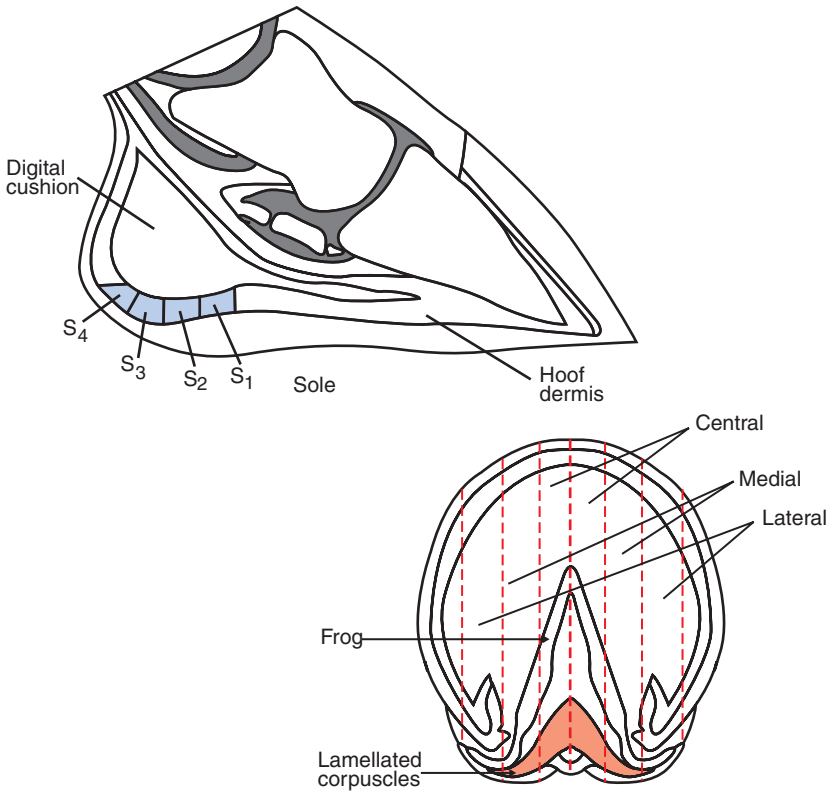

FIGURE 5-12 Schematic drawings showing location of pacinian corpuscle in palmar foot of horse. These receptors were concentrated in the dermis of the sole, especially a palmarmost site (S_4 *position*). In solar view, they were not evident cranial to the heel region in any of the presagittal planes (*central, medial,* or *lateral*). None were seen in the dorsal hoof wall.

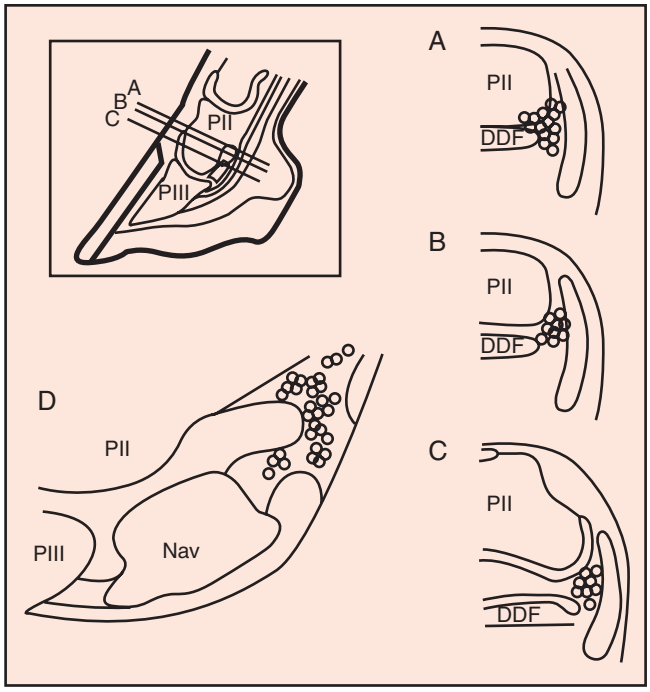

FIGURE 5-13 Schematic drawing showing the locations of the oblong lamellated mechanoreceptors (similar to the pacinian corpuscles) as determined from three serial 90-µm–thick sections. These mechanoreceptors are around the secondary tendon of the deep digital flexor muscle attaching onto the distal part of the middle phalanx (*PII*) and around the neurovascular bundle at three levels through the digit *A* (proximally) through *C* (distally). In *D*, the drawing shows a sagittal view. Each dot indicates one mechanoreceptor observed. These findings illustrate the numerous sensory receptors of this type at this proximal location in the foot.

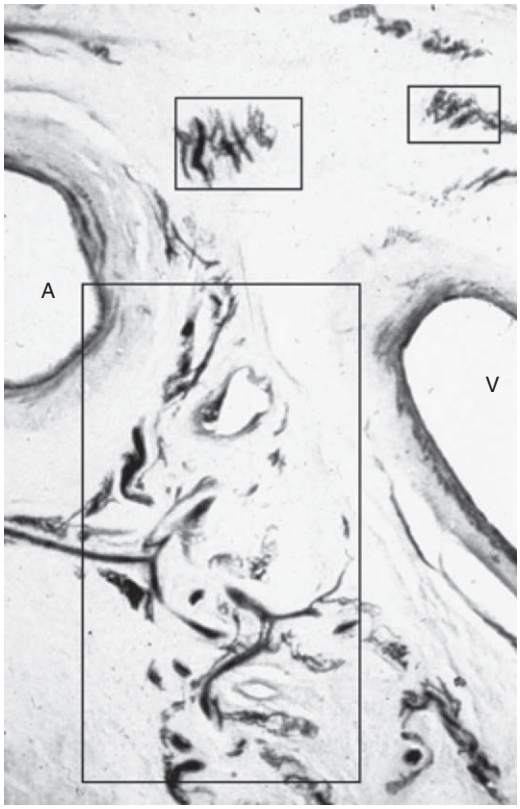

FIGURE 5-14 Histologically stained section showing the many impregnated nerve fibers and two mechanoreceptors (*boxed areas*) that resemble Ruffini endings. The mechanoreceptors respond to more slowly applied stimuli rather than transient stimuli and discharge for long time periods. Their locations are in foot areas with movement and potentially in areas where pressure changes during stance may be detected. The large enclosed area merely highlights the many neural structures that are associated with the neurovascular bundle within the foot.

tion of different proprioceptive stimuli by the horse during movement, as they are immediately proximal to many anticoncussive elements within the foot, such as the digital cushion, frog spine, and venous plexuses. In addition, the upward thrust of the frog stay into the digital cushion during ground impact would potentially direct the energy dorsally and distribute it to the region proximal to the navicular bone and its suspensory ligaments. Such ground impact of the foot and activation of the energy-absorbing mechanisms along with the deep digital flexor muscular contractions may be important in the activation of these corpuscular receptors proximal to the navicular bone. Since these lamellar corpuscles are proximal to many anticoncussive elements, the energy impacting upon these proximal receptors will be reduced, as the energy produced during ground impact is believed to be dissipated through the venous plexuses, the digital cushion, and the other connective tissues within the caudal parts of the foot.

Reduction of energy may provide a protective mechanism for the anatomical structures within the region proximal to the navicular bone and provide a means for a differential separation of forces impacting the foot, such as the receptors at the bulbs of the heel versus those lamellated receptors proximal to the navicular bone. Those lamellated corpuscles in the caudalmost regions of the foot would be impacted first, and forces may not dissipate to the extent of those energies impinging on corpuscles associated with the navicular bone, as they are more superficial to the anticoncussive structures and the mechanisms for energy dissipation. The resultant forces impinging on the CSL-associated corpuscles would be potentially reduced, or at least altered, by the anticoncussive structures and therefore provide a means for differential forces impinging on two groups of sensory detectors within the foot.

This morphologic observation suggests another function of the navicular apparatus in addition to the proposed directional change of constant angle forces of the deep digital flexor tendon by this bone. The energy and directional forces focused on the region proximal to the navicular bone during the initial phases of ground contact and prior to weight bearing and stance phases of the limb cycle may be a crucial site for the neurophysiologic assessment of sensory stimuli by the digit. Once assessed, this information will be conveyed to the spinal cord and the spinal cord reflex mechanisms during subsequent stages of the limb cycle.

Another mechanoreceptor present in the equine foot is the Ruffini corpuscle (see Figure 5-1 and Figure 5-14). These types of sensory receptors are present within the tissues of the neurovascular nerve bundle and on the solar surface of the foot. These receptors are beginning to be studied in more detail, because they respond differently to stimuli than the

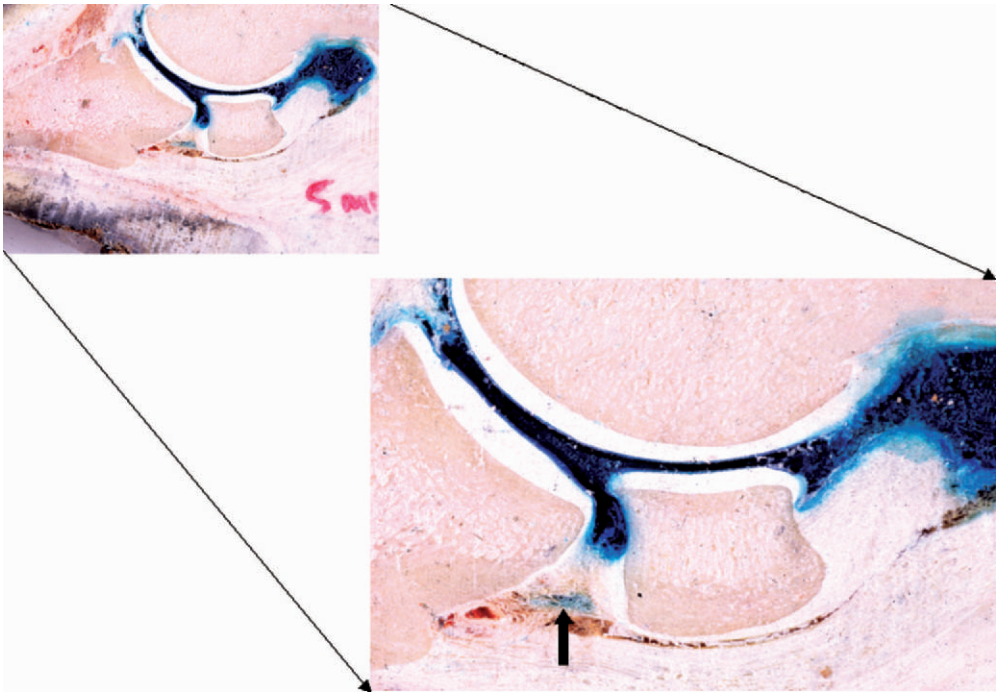

FIGURE 5-15 Joint injection with blue dye showing the dye passing through impar ligament (*black arrow*) within 5 minutes after injection into the coffin joint.

pacinian corpuscle. Ruffini corpuscles have a sustained and continuous response, such as during maintained pressures rather than abruptly applied pressures. These receptors may inform the horse about its environment during stance or similar foot positions.

The current morphologic findings indicate that the afferent component of the locomotive apparatus from the foot is complicated but well endowed with neural elements, with many different mechanoreceptors and nonmechanoreceptors, as well as chemically identified myelinated and unmyelinated nerve fibers. Furthermore, the many peptides in the unmyelinated sensory nerves serve not only the expected sensory function of conveying information about the environment to the spinal cord and brain but also an efferent function at the peripheral level in the control of blood flow (vasodilatation and vasoconstriction) and the interstitial fluid content (plasma extravasation), as well as interacting with the immune system, within the local tissue environment of the foot. These sensory peptidergic fibers function together with the peptides and the biogenic amines within the sympathetic autonomic nervous system to permit the tissues to respond and adapt to any new environmental situations that the foot tissues encounter. These two aspects of the sensory and motor nerves not only enable the horse to smoothly negotiate the ground surface during locomotion, but also provide a means for monitoring and controlling the physiologic, and possible pathologic, environment with the foot, as well as adapting to provide a protective cushioning effect within the foot during ground contact.

Perineural and Intraarticular/Bursal Analgesia of the Foot

Anesthesia of the PDN is the most common diagnostic procedure used during an examination of the foot and distal limb of the horse. There are several very good recent references dealing with the preparation and methodology of the PDN block.[23,35-37] Rather than reiterate the methodology of these routine procedures in this discussion, we will deal with the comparative effects of the local anesthetic injection sites and the structures potentially desensitized by these different avenues of administration of the local anesthetic. Interpretations of the effective zones of anesthesia and the structures desensitized are critical to the correct diagnosis of the potential lameness problem.

The medial and lateral PDNs course along the palmar digital vein and artery as they course toward the foot and are palpable proximal to the lateral cartilages. Once injected around the nerve bundles, the local anesthesia agent begins to infiltrate the nerve as well as diffuse from the injection site to affect those very small isolated nerves that have previously branched off from the major nerve bundle to course toward their intended innervated structure. Pharmacologically, the local anesthetic will affect the unmyelinated and thinly myelinated sensory nerves first, followed by the more thickly myelinated nerves fibers. Perineural anesthesia will vary somewhat depending upon where and how much of the local anesthetic solution is deposited in relationship to the nerves, because the anesthetic must diffuse through the connective tissues surrounding the nerves.

Intraarticular injections of the coffin joint and the navicular bursa have been discussed often within recent years as to their effectiveness and usefulness in diagnosing potential lameness problems associated with these two synovial cavities.[23,25,34,37,38] It is important to remember, however, that the local anesthetic solution, once injected into these cavities, diffuses through tissues in definite sequence rather than randomly, and that time following an injection is critically important in determining the tissues affected and the effect of the anesthetic. From the coffin joint, the local anesthetic

diffuses through the joint cavity and will desensitize the superficial nerves, probably within seconds to the first minutes after the injection. Within 5 minutes, the local anesthesia diffuses through the loose connective tissues comprising the DSIL, but not into the navicular bursa (Figure 5-15).

The important reminder for this observation is that this loose connective tissue septa contains the thin unmyelinated nerves between the dense fibers comprising the DSIL. These sensory nerves innervate the navicular bone and portions of the subchondral plate of the coffin bone. Our interpretation is that these areas will be desensitized within 5 minutes after the initial injection into the coffin joint. Thereafter, the local anesthetic will diffuse into the navicular bursa along the synovial linings, and begin to surround the sensory nerves passing through the solar foramina that provide much of the innervation to the coffin bone. The small nerve branches that arise from the medial and lateral PDNs will gradually be desensitized after this initial period because the local anesthetic must diffuse from the joint cavity through the loose connective tissues before it reaches these smaller nerve fibers. Our belief is that much of the navicular apparatus and the insertions region of the DSIL and the deep digital flexor tendon will be desensitized within 7 to 9 minutes after such a coffin joint injection. Several studies have indicated that this region will be desensitized much later.

Regarding local anesthetic to the navicular bursa, the initial effects of bathing of the bursa will be rapid, whereas diffusion from the bursa will take longer. The small nerves passing into the coffin bone, however, will be affected within several minutes. The flexor surface of the navicular bone will be affected rapidly, as will areas of the internal or trabecular structure, providing that there is an erosion on the flexor surface to permit the local anesthetic to enter the bone. Also, tissues ventral to the insertion areas of the deep digital flexor tendon will be affected by the local anesthetic injected into the navicular bursa but only minimally by an injection into the coffin joint. Otherwise, the effects on the navicular suspensory apparatus will be different from those produced by an injection into the coffin joint.

Clinically, during a lameness evaluation a thorough knowledge of the anatomy of the foot and an appreciation of the many problems and their effects on locomotion patterns are critical to the clinician in correctly diagnosing the site of the lameness problem. Such clinical skills provide a basis for interpretation of the effects of diagnostic nerve blocks when local anesthetic is applied perineurally around the sensory nerve fibers.

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6

MICROSCOPIC ANATOMY AND PHYSIOLOGY OF THE HOOF

CHRISTOPHER C. POLLITT

SECONDARY EPIDERMAL LAMELLAE

Projecting in parallel rows from the inner surface of the hoof wall and bars are 550 to 600 primary epidermal lamellae (PELs), the *stratum lamellatum* ("layer of leaves"). Examination of the hoof capsule, with its contents removed, shows that the lamellae of the dorsal hoof wall are shaped like long, thin rectangles approximately 7 mm wide and 50 mm long. One long edge of the rectangle is incorporated into the tough, heavily keratinized hoof wall proper *(stratum medium)* and the other long edge is free, facing the outer surface of the distal phalanx (Figure 6-1). The proximal short edge is curved and forms the inner shoulder of the coronary groove. The distal short edge merges with the sole and becomes part of the white zone visible at the ground surface of the hoof.

The role of the epidermal lamellae is suspensory, an anatomical specialization that increases the surface area for attachment of the dense connective tissue between the distal phalanx and the hoof. The specialization is the presence of 150 to 200 secondary epidermal lamellae (SELs) on the surface of each PEL. During the formation of an epidermal lamella, on the shoulders of the inner coronary groove, the epidermal basal cell layer proliferates,[1] causing folds (secondary lamellae) to form along the lamellar perimeter (Figure 6-2).

Normal SELs have a constant histologic appearance (Figure 6-3) that only laminitis alters. A description of hoof lamellar anatomy forms the basis of the histologic grading system of laminitis histopathology.[2]

The SEL tips are rounded (club-shaped). The basal cell nuclei are oval in shape and positioned away from the basement membrane (BM) at the apex of each cell. The long axis of each basal cell nucleus is at right angles to the long axis of the SEL. The secondary dermal lamellae are filled with connective tissue even at their very tips, between the SEL bases. Special stains such as periodic acid-Schiff are required to highlight the BM and locate the glycoprotein constituents of the basement membrane, for example laminin. When periodic acid-Schiff stain is applied to normal hoof lamellae, the BM of each SEL stains as a dark magenta line closely adherent to the SEL basal cells. Between the bases of each SEL, the BM penetrates deeply (see Figure 6-3, *arrowheads*) and is close to the anuclear, keratinized PEL. There are no polymorphonucleocytes (PMNs) in either the capillaries or the dermis. The BM of normal lamellae penetrates deeply into the crypt between pairs of SELs and clearly outlines the tapered tip of each secondary dermal lamella (SDL). The proximity of the SDL tip to the keratinized axis of the PEL is, therefore, readily appreciated; it is always a distance equivalent to the length of one or two epidermal basal cells. The club-shaped tips of the SELs, as outlined by the BM, have always been found to be rounded and never tapered or pointed. The tips of the lamellae (both primary and secondary) all orient toward the distal phalanx, thus indicating the lines of tension to which the lamellar suspensory apparatus is subjected.

The surface area of the equine inner hoof wall has been calculated to average 0.8 m^2, about the size of the surface area of the skin of a small adult human (a considerable increase over the inner surface area of bovine hooves, which lack secondary lamellae).[3] This large surface area for suspension of the distal phalanx and the great compliance of the interdigitating lamellar architecture helps reduce stress and ensures even energy transfer during peak loading of the equine foot. In life, the hoof distal phalangeal attachment apparatus is impressively strong; during peak loading, the hoof wall and the distal phalanx move in concert and separate only when laminitis interferes with lamellar anatomy.

THE BASEMENT MEMBRANE

At the interface of the epidermis and the dermis is a tough, unbroken sheet of extracellular matrix called the *basement membrane*. This key structure partitions lamellar epidermal cells from the lamellar dermis. On one side of the BM, epidermal basal cells are firmly attached; on the other (dermal) side, tendon-like connective tissue, emanating from the dorsal surface of the distal phalanx, is tightly woven into the matlike structure of the BM. The signature lesion of laminitis, failure of attachment between lamellar dermis and epidermis, occurs at this lamellar dermoepidermal junction and involves the lamellar BM. The ultrastructure of the equine hoof basement membrane is essentially the same as in other animals but with some important specializations. It is a sheetlike three-dimensional anastomosing latticework of fine interconnecting cords. The axial skeleton of the cord network consists of filaments of collagen IV. The collagen IV filaments are ensheathed with glycoproteins, in particular laminin-1, which together form the electron-dense *lamina densa* (Figure 6-4). Innumerable extensions of the *lamina densa* and banded anchoring fibrils (consisting of collagen VII) in the shape of recurved hooks intermesh with the type I collagen fibrils of the connective tissue of the lamellar corium, forming an important part of the attachment mechanism between dermis and epidermis. The equine lamellar basement membrane has a high density of *lamina densa* extensions and anchoring fibrils around the tips of the secondary epidermal lamellae, a feature perhaps not surprising in a large ungulate weight bearing on single digits.[4]

FIGURE 6-1 Dissected hoof with contents removed, showing the lamellae of the inner hoof wall.

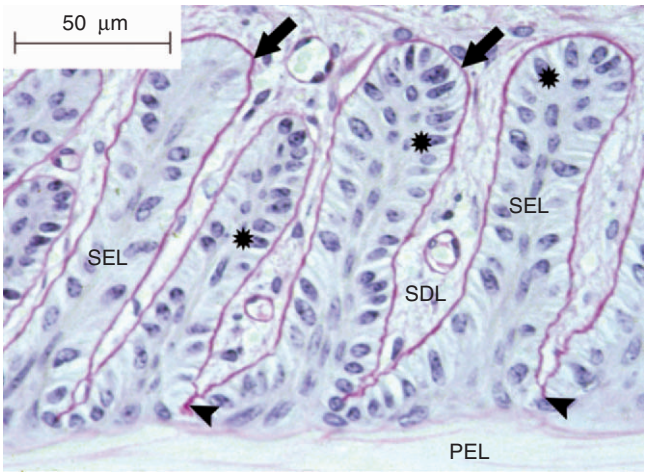

FIGURE 6-3 Micrograph of normal hoof lamellae stained to highlight the basement membrane. The basement membrane (*arrows*) of each secondary epidermal lamella (SEL) shows as a dark magenta line closely adherent to the SEL basal cells. Between the bases of each SEL, the basement membrane penetrates deeply (*arrowheads*) and is close to the anuclear, keratinized, primary epidermal lamella (PEL). The SEL tips are rounded (*club-shaped*). The basal cell nuclei are oval in shape (*stars*) and positioned away from the basement membrane at the apex of each cell. The long axis of each basal cell nucleus is at right angles to the long axis of the SEL. The secondary dermal lamellae (SDLs) are filled with connective tissue right to their tips, between the SEL bases. These parameters of hoof lamellar anatomy form the basis of the histologic grading system of laminitis histopathology. (Periodic acid-Schiff stain.)

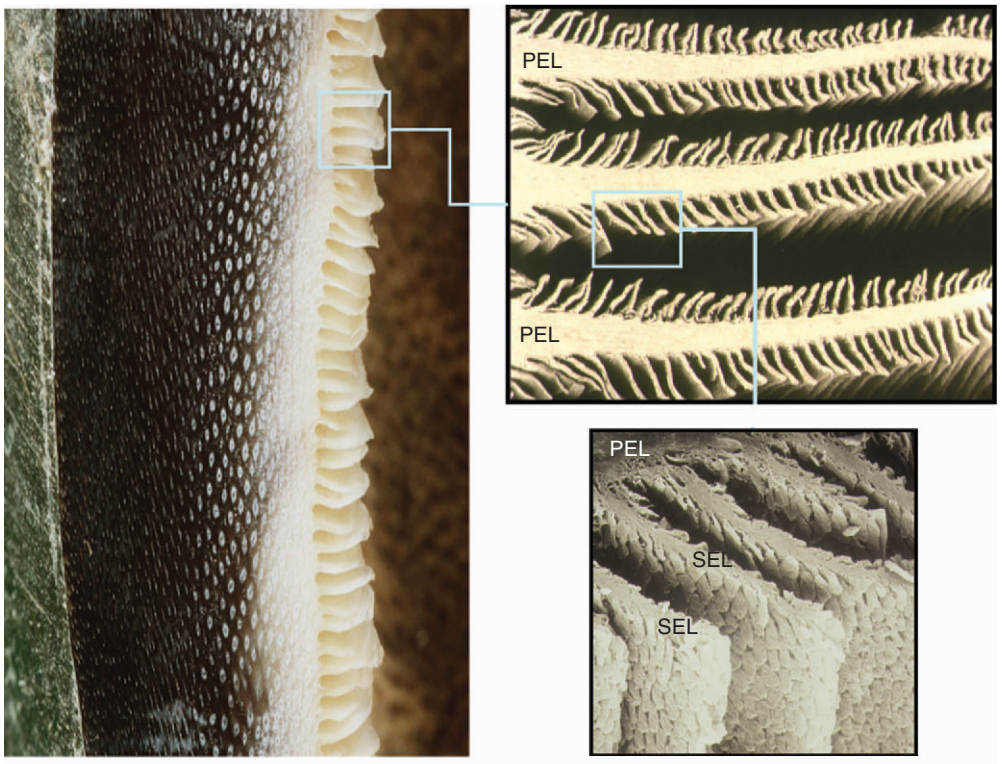

FIGURE 6-2 The equine hoof wall and its inner lamellar layer. Secondary epidermal lamellae (SELs) increase the surface area of each leaflike primary epidermal lamella (PEL). Epidermal basal cells cover the surface of each SEL.

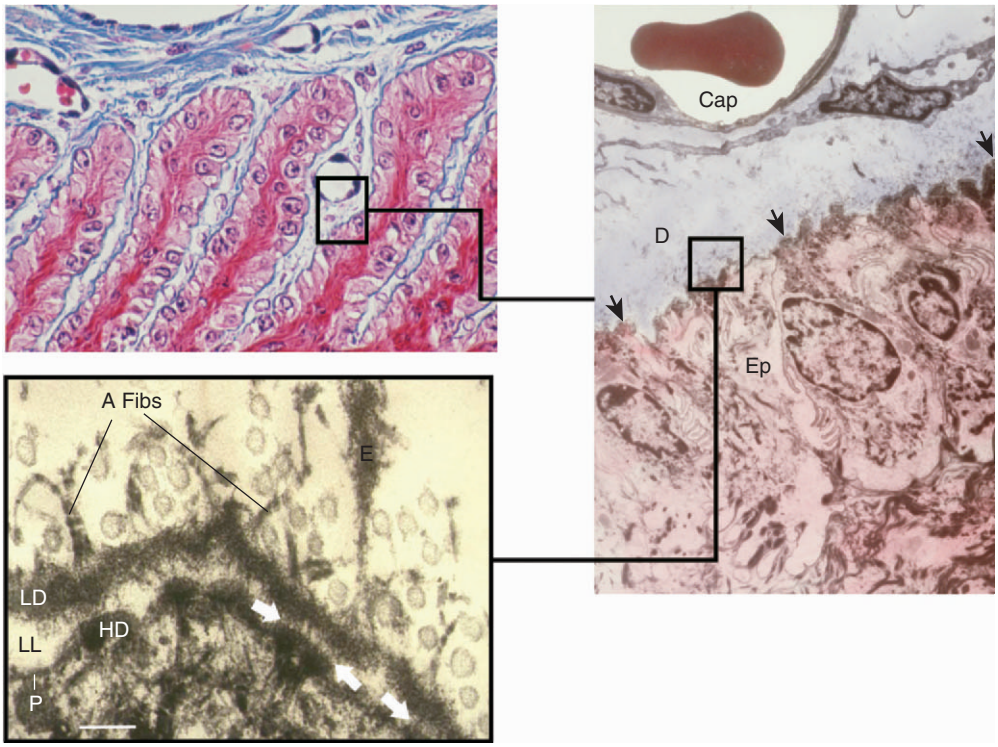

FIGURE 6-4 The basement membrane of each secondary epidermal lamella (SEL) stains as a dark blue line closely adherent to the SEL basal cells in light microscopic sections stained by Masson trichrome. In low-magnification transmission electron micrographs (TEMs), the basement membrane is just visible (*black arrows*) and follows the contours of the SEL basal cells (Ep). High-magnification TEMs show the lamina densa (LD) of the basement membrane separated from the plasmalemma (P) of the basal cell by a gap, the lamina lucida (LL). Inserted in the basal cell plasmalemma are numerous attachment plaques called hemidesmosomes (HDs). Extensions of the lamina densa (E) and banded anchoring fibrils (A Fibs) intermesh with collagen fibrils of the connective tissue of the lamellar dermis (D). Bridging the gap between HDs and the lamina densa are numerous fine anchoring filaments (*white arrows*). Cap, capillary. (Bar = 10 nm.)

Laminin-1, one of the key proteins of the BM, forms receptor sites and ligands for a complex array of growth factors, cytokines, adhesion molecules, and integrins. Without an intact, functional BM, the epidermis to which it is attached falls into disarray. Significantly, disintegration and separation of the lamellar basement membrane is a feature of acute laminitis. Laminin-1 and collagen IV disappear from the BM, which progressively loses its close attachment to the basal cells and strips away from the epidermal lamellae.[5]

HEMIDESMOSOMES

When viewed with the transmission electron microscope (TEM), the BM is dominated by the electron-dense *lamina densa,* which appears as a dark line following the contours of the epidermal basal cells. The plasma membrane (plasmalemma) at the base of each basal cell is attached to the BM by numerous electron-dense adhesion plaques, or hemidesmosomes (HDs). The various proteins of each HD occur on both sides of the basal cell plasmalemma, thus forming a bridge linking the interior of the basal cell to the exterior connective tissue (Figure 6-5).

The intracytoplasmic HD proteins that attach basal cells to the BM are named plectin, BP230, BP180, and integrin $\alpha_6\beta_4$. Importantly, HDs are maintained and assembled by glucose-consuming phosphorylation reactions. Integrin $\alpha_6\beta_4$ and BP180 have domains on both sides of the plasmalemma and form part of the extracytoplasmic, subbasal dense plaque of the HD. The protein that bridges the gap between the HD and the lamina densa is laminin-5.[6,7] Transmission electron microscopy resolves laminin-5 as innumerable fine anchoring filaments spanning the *lamina lucida*, the space between the basal cell and the *lamina densa* (Figure 6-6). The essential nature of hoof lamellar HDs and anchoring filaments is illustrated by horses that inherit mutations in the genes expressing HD proteins.

Within the Belgian horse population, an autosomal, recessive mutation within the gene encoding the gamma 2 chain of laminin-5 has been identified. The mutation causes a premature termination codon, and consequently the expression of the laminin-5 anchoring filament protein is absent, resulting in widespread dermoepidermal separation in skin and hoof lamellae. Belgian foals afflicted with this hereditary, junctional epidermolysis bullosa suffer hoof exunguilation and generalized skin lesions at pressure points and mucocutaneous junctions and die or are euthanized within a few days of birth.[8] Loss of plectin from the hemidesmosome adhesion complex of a Quarterhorse foal is a second example of congenital lamellar dysadhesion.[9] In one case, the foal had fragile skin and hoof pathologic lesions that resembled both acute and chronic laminitis. The distal phalanx had sunk into the hoof capsule and had perforated the sole. Interestingly, although the loss of plectin was ubiquitous in hooves and skin, only the forefeet showed lesions; the anatomy and histologic appearance of the hindfeet was normal, thus illustrating how closely the lesions of laminitis follow foot load distribution. If clinicians could do more to lessen the impact of weight bearing during the developmental stage, the destructiveness of laminitis in adult horses could be diminished.

A firm attachment between epidermal basal cells and the dermis is essential in weight-bearing hoof lamellae. This attachment is ensured by the ordered array of molecules linking the epidermal basal cell cytoskeleton to hemidesmo-

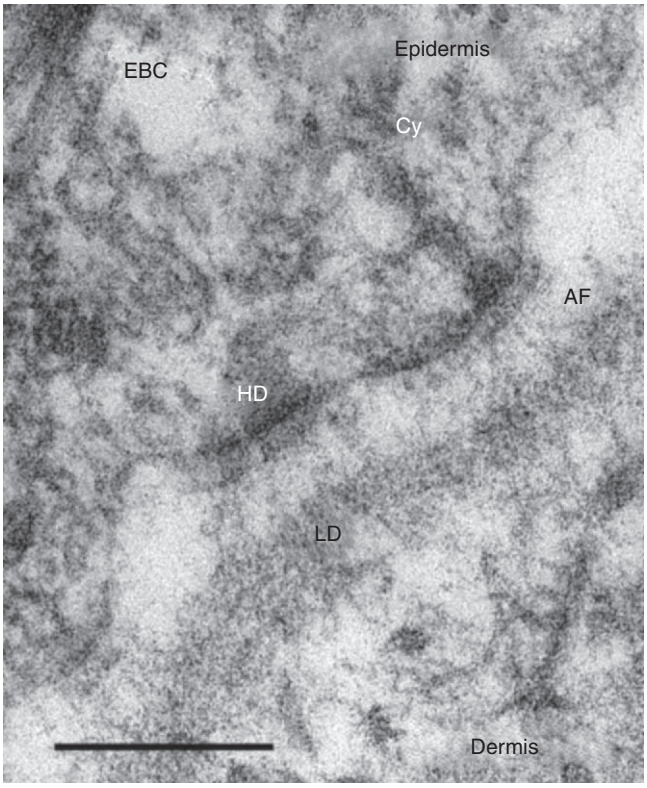

FIGURE 6-5 Hemidesmosomes at the dermoepidermal junction. The electron-dense lamina densa (LD) is the major structural component of the basement membrane. Hemidesmosomes are attachment plaques that serve to keep the lamina densa of the basement membrane firmly adherent to all lamellar epidermal basal cells (EBC). Each hemidesmosome is constructed of several proteins that stain dark when viewed by transmission electron microscopy. The internal skeleton or cytoskeleton (Cy) of the basal cell is constructed of fine keratin filaments that attach to the intracytoplasmic dense plaque of hemidesmosomes and interconnect to desmosomes and the nucleus. Bridging the gap between the dense plaque of the hemidesmosome (HD) and the lamina densa are numerous submicroscopic anchoring filaments (AF). (Bar = 10 nm.)

somes, anchoring filaments, and the BM. Any defect that weakens a link in this chain also weakens epidermal-dermal adhesion.

LAMELLAR REMODELING ENZYMES

Connective tissue and keratinocytes are now known to remodel and continually upgrade their spatial organization by the tightly controlled production of a specific class of enzymes known as matrix metalloproteinases (MMPs). MMPs are a group of zinc-dependent enzymes that, when activated, degrade extracellular matrix (ECM) and basement membrane components. The activity of MMPs—particularly the gelatinases MMP-2 and MMP-9—correlates strongly with BM destruction and the associated degree of malignancy and invasiveness of tumors.[10,11] Two members of the MMP family (MMP-2 and MMP-9) can be isolated from homogenized normal hoof wall lamellae and from normal lamellar explants cultured in tissue culture medium.[12] Secreted as inactive proenzymes and, when activated, promptly inhibited by locally produced inhibitors (tissue inhibitors of metalloproteinase [TIMPs]), it is MMP activity that is likely responsible for the remodeling of the various classes of epidermal cells between the basement membrane, the SELs, and the PELs (Figure 6-7).

The protein constituents of the basement membrane (type IV collagen, type VII collagen, and laminin-1) are known substrates of the matrix metalloproteinases MMP-2 and MMP-9.[13,14] In addition, laminin-5, the main component of the anchoring filaments bridging lamellar basal cell to the basement membrane is cleaved by MMP activity during laminitis development.[15] The disorganization of the epidermal cells of the SELs, the wholesale separation of basal cells from the basement membrane and lysis of basement membrane that occurs early in the pathologic development of laminitis[2] are now thought to be caused by the triggering of activation of uncontrolled, excessive MMP production.[16] MMPs, present in the cells of the SELs, presumably for normal remodeling purposes, appear to be important players in laminitis pathogenesis.

HOOF WALL GROWTH

The hoof wall grows throughout the life of the horse to replace hoof lost to wear and tear at the ground surface. Continual regeneration of the hoof wall occurs at the coronet, where germinal cells (epidermal basal cells) produce populations of daughter cells (keratinocytes or keratin-producing cells) that mature and keratinize, continually adding to the proximal hoof wall. Similarly, mitosis in the proximal hoof PELs also occurs.[17] Although mitotic figures among the basal cells of the proximal lamellar zone are easily observed, there is no convincing evidence that the more distal lamellae proliferate at all. The fundamental question is, how do the inner hoof wall lamellae remain attached to the connective tissue embedded on the surface of the stationary distal phalanx, while one moves over the other? Is it by continuous proliferation of the lamellar epidermis (laminar flow) or by some other remodeling process (that may somehow be involved in laminitis pathogenesis)? Cells in mitosis are rarely, if ever, found in normal lamellae below the proximal, proliferative zone. To determine precisely where in the hoof wall epidermal cell proliferation occurs, a proliferative index (PI) for basal cells of the coronet, lamellae, and toe of the dorsal hoof wall of ponies has been calculated.[1] The thymidine analogue 5-bromo-2′-deoxyuridine (BRdU), injected intravenously into living ponies, was incorporated into all cells undergoing mitosis during a 1-hour study period. Histologic sections of hoof tissue were stained immunohistochemically, using monoclonal antibodies against BRdU. As expected, the highest PI values (mean ± SE) were in the coronet (12.04 ± 1.59%) and proximal lamellae (7.13 ± 1.92%) (Figure 6-8).

These are the growth zones of the proximal hoof wall. Distal to this, the PI values of more distal lamellae were very much lower. They ranged from 0.11 ± 0.04% to 0.97 ± 0.29%, significantly lower ($P < 0.05$) than the proximal lamellar growth zone. Evidence for a constant supply of new cells in the lamellar region, generating a downward laminar flow, was not provided by this study. The few proliferating cells detected in the main lamellar region had a patchy distribution and were located at the PEL tips, not in cap-horn arcades. A twentyfold PI decrease between proximal and more distal lamellae suggests that the majority of the normal lamellae are non-

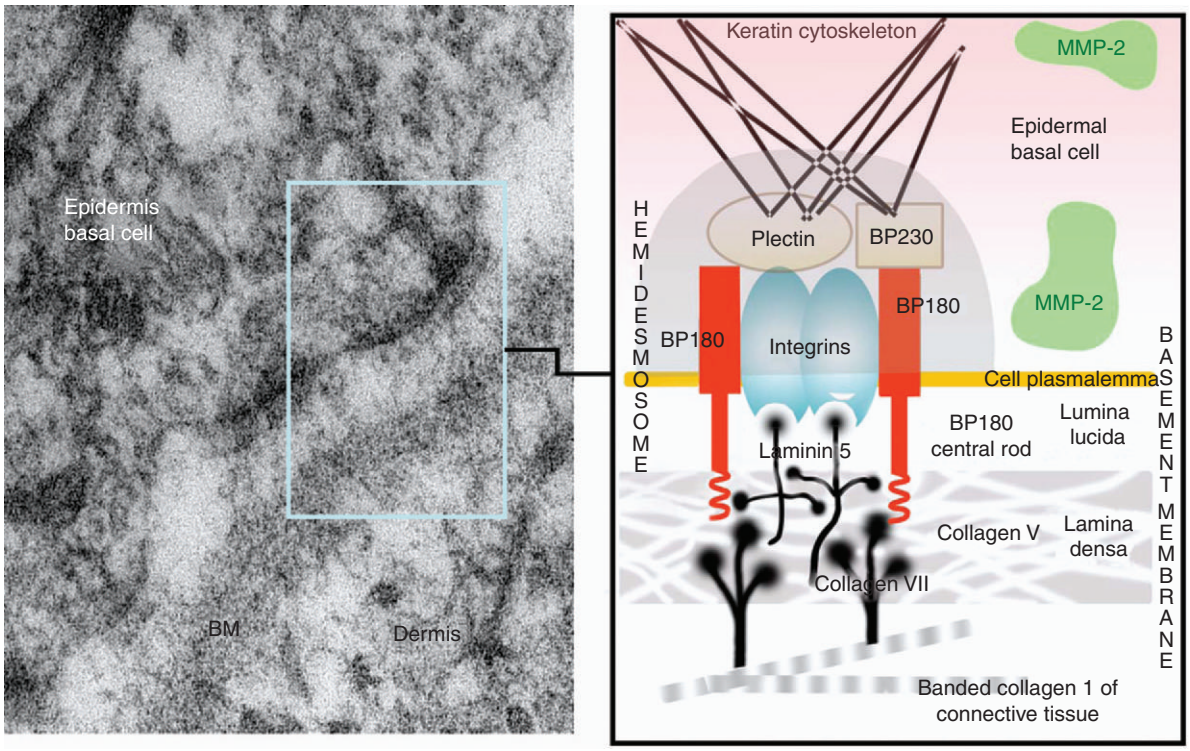

FIGURE 6-6 Diagram of hemidesmosome, the key structure anchoring the basal cells of the SEL to the basement membrane. The intracytoplasmic plaque consists of the proteins plectin, BP180, and integrin $\alpha_6\beta_4$. Keratin intermediate filaments of the cell cytoskeleton connect to plectin, which in turn communicates with laminin-5 anchoring filaments via integrin $\alpha_6\beta_4$. Integrin $\alpha_6\beta_4$ and BP180 have domains on both sides of the plasmalemma and form part of the extracytoplasmic, subbasal dense plaque of the hemidesmosome. The anchoring filaments are incorporated into the matrix of the basement membrane.

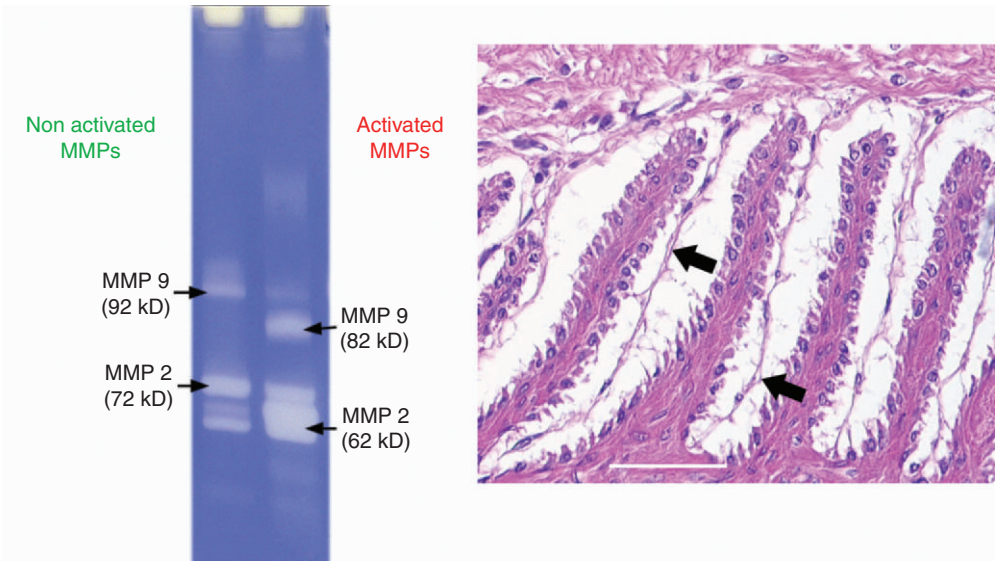

FIGURE 6-7 Zymograph of normal lamellar explants. The tissue culture fluid, in which explants were cultured, was applied to lanes in a polyacrylamide gel containing 0.1% gelatin. After electrophoresis and overnight incubation, the gel was stained for protein with Coomassie Blue G-250. Because the gel contains protein (the soluble collagen gelatin), the entire gel stains blue, except where gelatin has been digested by matrix metalloproteinase (MMP) activity. Thus the clear areas reveal the existence of MMP-9 and MMP-2 in lamellar hoof tissue. Proteins of known molecular weight (not shown) are subjected to electrophoresis at the same time to determine MMP molecular weights in kilodaltons (kD). Lane 1 shows the MMPs contained in a normal explant. There is pro-MMP-9 but no active MMP-9, a large band of pro-MMP-2, and some active MMP-2. Lane 2 shows the effect of MMP activation with APMA. The pro-MMP-9 has been converted to active MMP-9 and a similar conversion of pro-MMP-2 has occurred. Cleavage of a 10 kD fragment from pro-MMP 9 and 2 activates the enzyme. The micrograph shows a lamellar hoof explant after APMA activation of its constituent MMPs. The basement membrane (*arrows*) of the secondary epidermal lamellae is no longer attached to the basal cells. Activation of lamellar MMPs causes this in vitro lesion, which resembles natural laminitis. (Hematoxylin-eosin stain; bar = 10 μm.)

proliferative and their main function is to suspend the distal phalanx within the hoof capsule.

Remodeling within the hoof wall epidermal lamellae, which must occur as the hoof wall moves past the stationary distal phalanx, appears to be a process not requiring epidermal cell proliferation. Remodeling of epidermis and the extracellular matrix is now known to involve the controlled release of activated MMPs and their subsequent inhibition by TIMPs.[18] MMPs have been shown to exist in lamellar hoof[16] (Figure 6-9), and their uncontrolled activation has been proposed as a mechanism for the pathogenesis of laminitis.[12]

The molecular components of desmosomes, hemidesmosomes, and basement membranes are substrates for MMP activity,[18] so the mechanistic concept[19] of "formation and destruction of desmosomes in a staggered ratchet-like manner" now has a well-referenced, biological explanation. Lamellar epidermal cells and their adjacent basement membrane are constantly responding to the stresses and strains of growth and locomotion by releasing MMPs and TIMPS to accomplish whatever cellular reorganization is required. Because this involves enzymes capable of destroying key components of the attachment apparatus between distal phalanx and inner hoof wall, it is clear that triggering this "loaded gun" will have dire consequences for the future health of the foot. Inadvertent or uncontrolled lamellar MMP activation makes horses, with their generic reliance on a single digit per limb, uniquely susceptible to the destructive effects of laminitis.

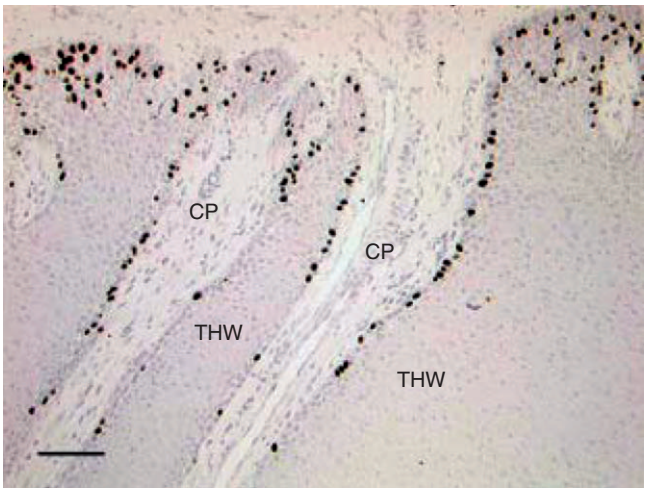

FIGURE 6-8 Basal cell proliferation in the coronary band. Longitudinal section of proximal hoof wall (coronary band) immunostained for BRdU that was injected intravenously into a normal pony 60 minutes previously. The positive, brown-staining cells are basal cells that incorporated BRdU as they underwent mitosis during the previous 60 minutes. Both the tubular and intertubular hoof show a high rate of basal cell mitosis. CP, coronary papilla; THW, tubular hoof wall. (Bar = 100 μm.)

THE CORIUM

The highly vascular corium, or dermis, underlies the hoof wall and consists of a dense matrix of tough connective tissue containing a network of arteries, veins, and capillaries and sensory and vasomotor nerves. All parts of the corium, except for the lamellar corium, have papillae that fit tightly into the holes in the adjacent hoof. The lamellar corium has dermal lamellae that interlock with the epidermal lamellae of the inner hoof wall and bars (Figure 6-10). The vascular system of the corium provides the hoof with nourishment. The dense matrix of corium connective tissue connects the BM of the dermal-epidermal junction to the periosteal surface of the distal phalanx and thus suspends the distal phalanx from the inner wall of the hoof capsule.

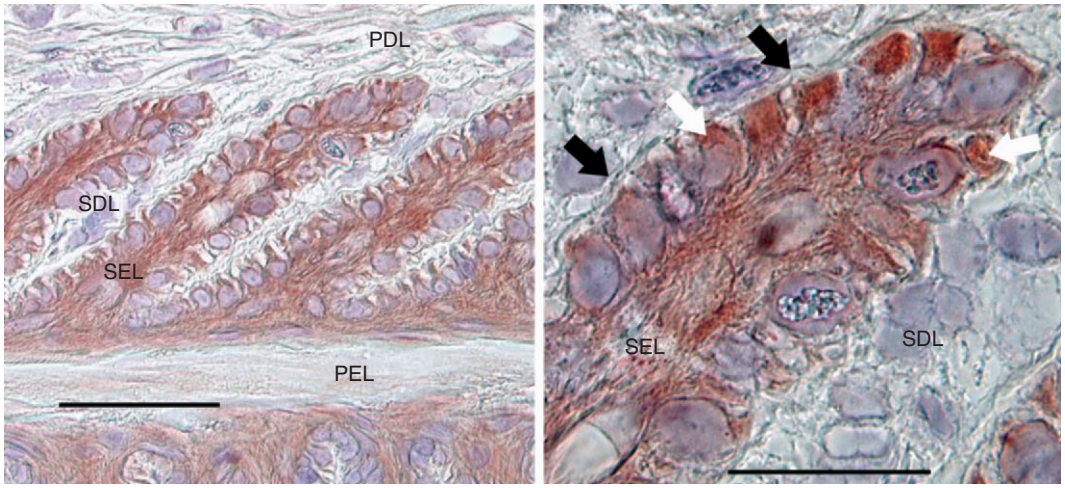

FIGURE 6-9 Immunostaining of normal hoof secondary epidermal lamella (SEL) with anti-matrix metalloproteinase (MMP)-2. Dark brown, positive cytoplasmic staining was located mainly in lamellar basal and parabasal cells (**A**). Primary epidermal lamellae (PELs) and primary dermal lamellae (PDLs) did not stain. Basal cell MMP-2 of SELs (**B**) was located in cytoplasm (*white arrows*) adjacent to the basement membrane (*black arrows*). Nuclei of epidermal cells and fibroblasts stained blue by the hematoxylin counterstain. (Bar in **A** = 50 μm. Bar in **B** = 20 μm.) *(From Kyaw-Tanner M, Pollitt CC: Equine laminitis: increased transcription of matrix metalloproteinase-z (MMP-2) occurs during the developmental phase,* Equine Vet J. *36:221-225, 2004.)*

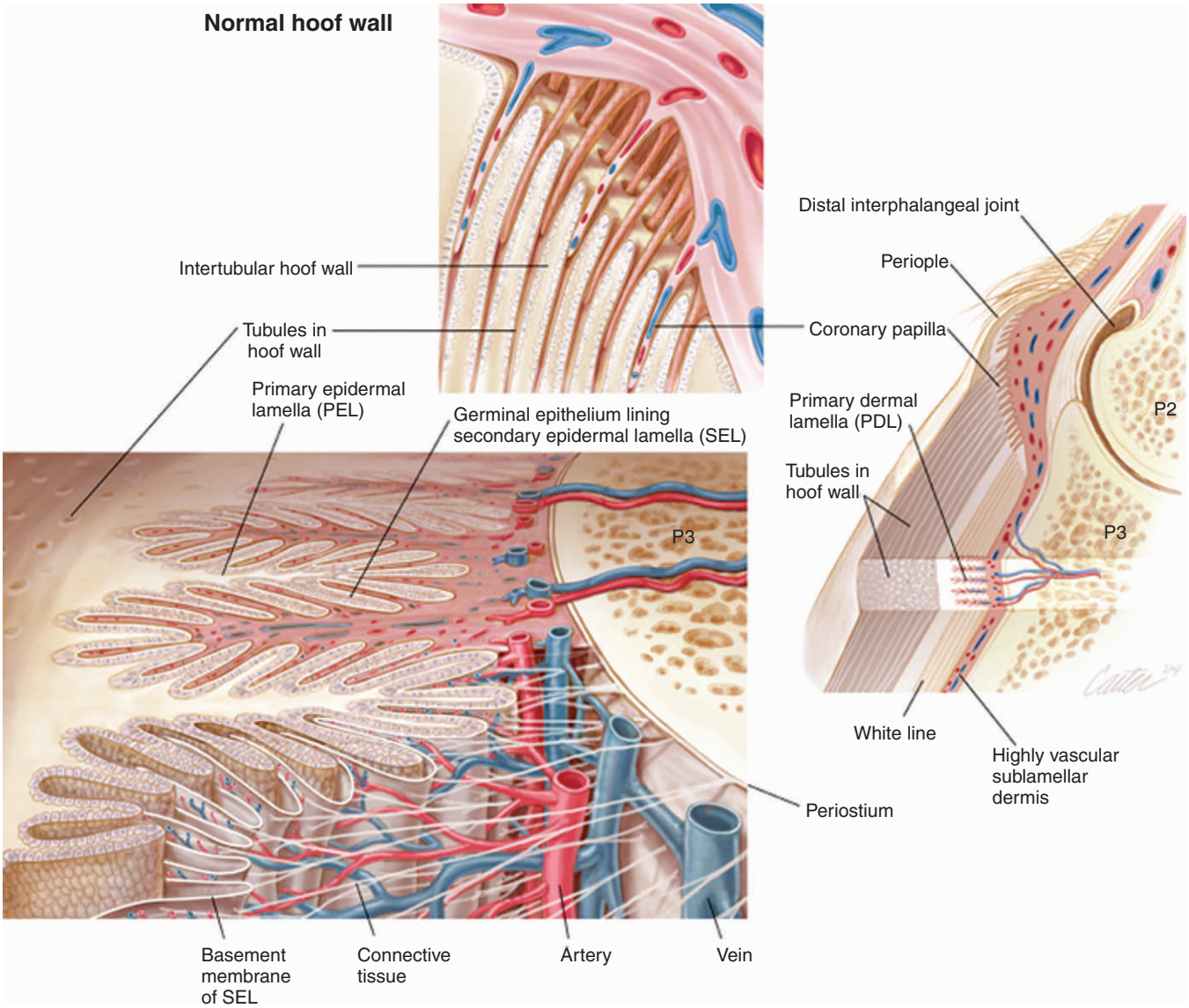

FIGURE 6-10 Diagram showing the dermal papillae of the coronet and interdigitating lamellae of the inner hoof wall. Connective tissue attaches the distal phalanx (P3) to the basement membrane of the lamellar dermal/epidermal junction. The basement membrane is shown artificially detached to reveal the basal cells of the secondary epidermal lamellae beneath. *(From Pollitt CC: Normal hoof wall,* Clin Tech Equine Prac *3:8, 2004.)*

The Coronet Corium

The coronet corium fills the coronary groove and blends distally with the lamellar corium. Its inner surface is attached to the extensor tendon and the ungual cartilages of the distal phalanx by the subcutaneous tissue of the coronary cushion. Collectively the coronary corium and the germinal epidermal cells that rest on its basement membrane are known as the *coronet* or *coronary band*. A feature of the coronet corium is the large number of hairlike papillae projecting from its surface. Each tapering papilla fits into one of the holes on the surface of the epidermal coronary groove and, in life, is responsible for nurturing an individual hoof wall tubule.

The BM surface of the hoof wall corium was examined with the scanning electron microscope after treatment of hoof tissue blocks with a detergent enzyme mixture.[4] A clean separation could be made between dermal and epidermal tissues, enabling the surface of the dermal basement membrane to be examined in detail. The BM of the coronary and terminal papillae was folded into numerous ridges parallel with the long axis of the papilla. These longitudinal ridges on the surface of the papillae are analogous to the secondary dermal lamellae and probably share the role of increasing the surface area of attachment between the epidermal hoof and the connective tissue on the parietal surface of the distal phalanx. They may also act as guides or channels directing columns of maturing keratinocytes in a correctly oriented proximodistal correction (Figure 6-11). The density of coronary papillae is greatest at the periphery and is least adjacent to the lamellae. This mirrors the arrangement of the hoof wall tubules of the *stratum medium* in zones based on tubule density.[20]

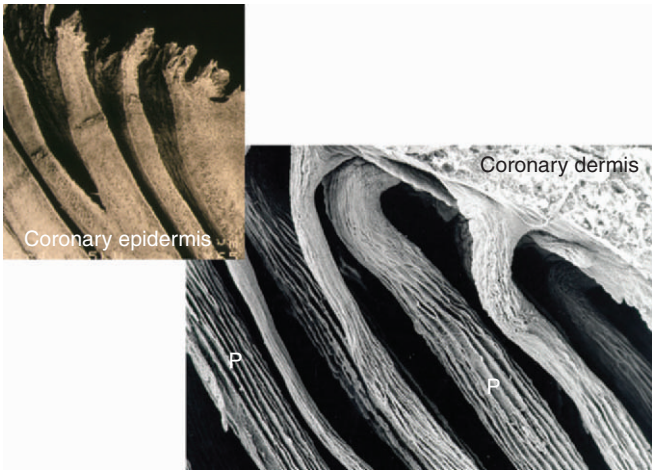

FIGURE 6-11 Epidermis and dermal papillae of the coronet. Each papilla (P) is responsible for the nutrition and organization of an individual hoof wall tubule. The papillae fit into complementary sockets in the coronary groove of the epidermal hoof wall (*upper inset*).

Normal Metabolism of the Hoof

The uptake of glucose and the production of lactate in the equine foot of normal horses was studied by comparing simultaneously taken blood samples from an artery, the jugular vein, and a digital vein at the level of the pastern of one of the front legs.[21] Differences between arterial and venous blood reflects the metabolic processes in the tissues drained by the two veins. The glucose levels in the digital veins showed that the feet in horses at rest have a glucose consumption that exceeds that of the head. The foot consists mainly of epidermis (hoof capsule), dermis (mainly connective tissue consisting of fibroblasts, blood vessels, and nerves), bone, and cartilage. Because the foot does not include tissue with the glycogen storage capacity of striated muscles, it is assumed that the glucose consumption reflects energy production via either the glycolytic pathway or the citric acid cycle. The levels of lactate were higher in blood from the digital veins, indicating that, at physiologic glucose concentrations at rest, a large part of the glucose that is taken up by the living cells in the foot is metabolized to lactate anaerobically via the glycolytic pathway.

Secondary epidermal lamellae are rich in the enzyme lactate dehydrogenase,[21] suggesting that the lamellar layer relies on anaerobically generated energy even under normal oxygenated conditions. Lactate dehydrogenase may also operate in reverse, and lactic acid may be an energy-generating substrate in the feet of horses. In immunostaining studies, SEL basal cells react strongly for the glucose transport protein GLUT1. SELs also react for small amounts of GLUT3 and GLUT4. The equine skin showed a similar expression of GLUT, thus resembling the skin of humans and rodents (Figure 6-12).[21]

The Sole Corium

As in the coronet, each dermal papilla of the sole corium fits into a socket in the epidermal (horn) sole. At the distal end of each dermal lamella is a set of papillae known as the *terminal papillae* (Figure 6-13). The epidermis surrounding the terminal papillae is nonpigmented and forms the inner part of the white zone (white line). The white zone is relatively soft and flexible and effectively seals the sole to the hoof wall. It is sometimes subject to degeneration and infection, usually described as "seedy toe" or "white line disease."

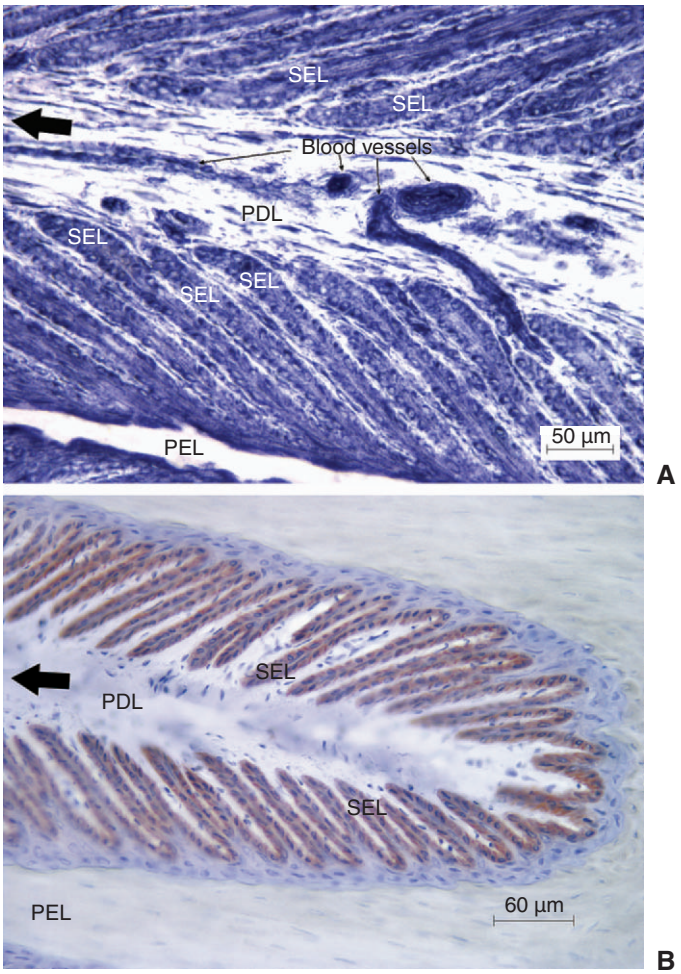

FIGURE 6-12 Transverse cryostat sections of the lamellar layer at the midlevel of the hoof wall. The section stained for lactate dehydrogenase (LDH) (**A**) shows dark blue staining, indicating the presence of the enzyme. A section stained with GLUT1 monoclonal antibody (**B**) shows brown staining, indicating the presence of GLUT1. *Large black arrow* points toward the distal phalanx of the foot.

Blood Supply of the Foot

Digital Arteries

The medial and lateral digital arteries arise by division of the medial palmar artery (common digital artery) between the suspensory ligament and the deep digital flexor tendon and enter the digit on the abaxial surfaces of the proximal sesamoid bones of the fetlock (Figure 6-14). Opposite the proximal phalanx, each digital artery gives rise to a branch that forms, with the artery of the opposite side, an arterial circle around the bone. At the level of the proximal interphalangeal joint, the digital arteries send major branches to the heels that supply the digital cushion, frog, lamellar corium of the heels and bars, and the palmar perioplic and coronary coria. Opposite the middle of the second phalanx, each digital artery again

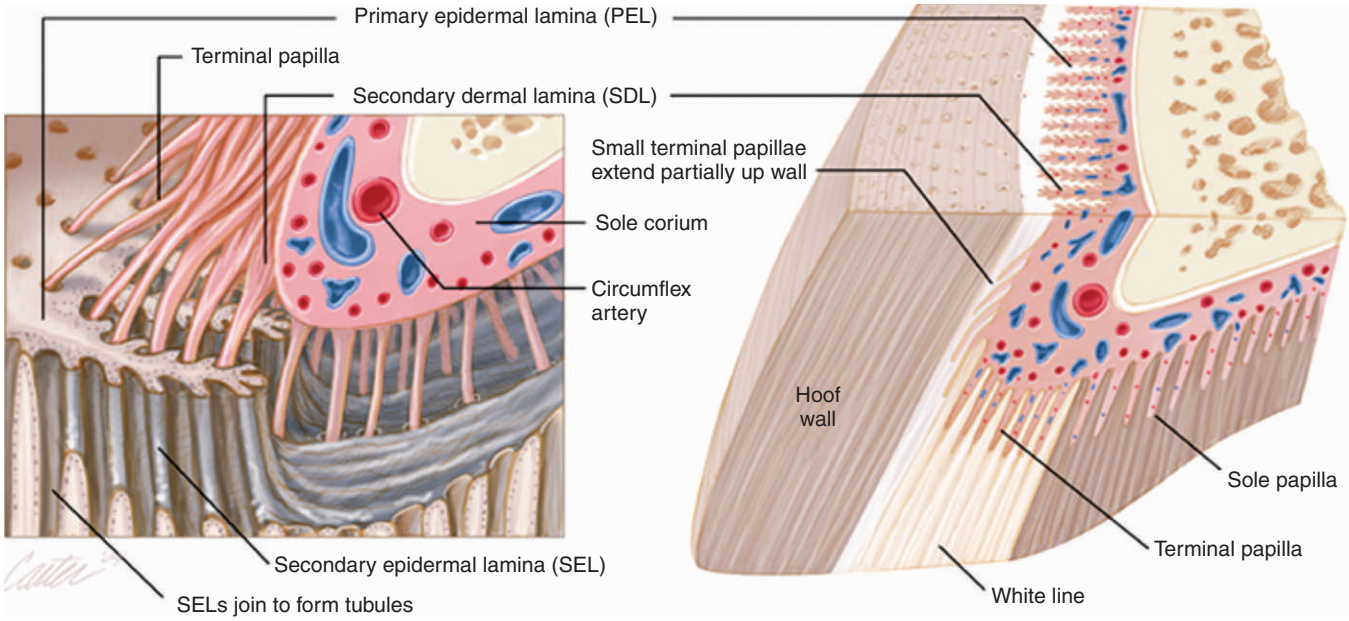

FIGURE 6-13 On the distal end of all dermal lamellae are numerous terminal papillae. Germinal epidermis lining the terminal papillae are responsible for generating keratinized epidermal cells, which fill the space between the primary epidermal lamellae as they grow toward the ground surface. *(From Pollitt CC: On the distal end of all dermal laminae are numerous terminal papillar,* Clin Tech Equine Prac *3:11, 2004.)*

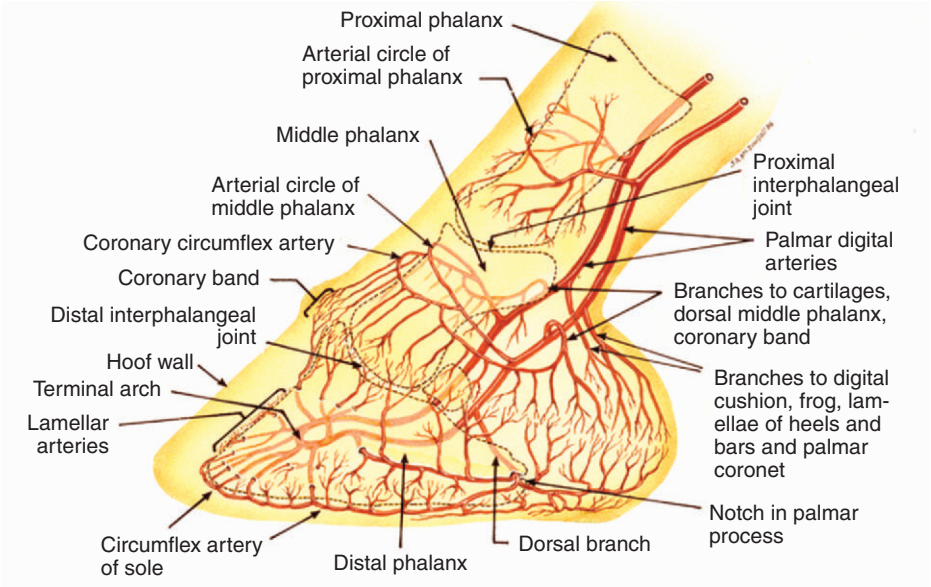

FIGURE 6-14 Diagram of the arteries of the equine foot. *(Design: Christopher C. Pollitt; artwork: John McDougall.)*

branches and forms an artery that runs deep to the cartilages and the extensor tendon and connects with the artery of the opposite side, to form an arterial circle around both the second phalanx and the coronary band. This coronary circumflex artery supplies the digital extensor tendon and distal interphalangeal joint and supplies numerous branches to the coronary corium and proximal lamellae of the toe and dorsal quarters.

Proximal to the navicular bone, each digital artery gives off a dorsal branch that passes through the notch or foramen in the palmar process of the distal phalanx and, running in the parietal groove on the dorsal surface of the distal phalanx, supplies the lamellar coria of the quarters and heels and anastomoses with the palmar part of the circumflex artery of the sole.

From the medial and lateral digital artery, branches arise to both the proximal and distal borders of the distal sesamoid (navicular) bone. The branches anastomose with each other and form direct cross-connections between medial and lateral digital arteries above and below the distal sesamoid bone. The proximal artery runs in the suspensory ligament of the distal sesamoid bone and its branches enter the proximal edge of the bone through fine vascular foramina. Along the distal border, a similar anastomotic arterial network runs in the distal interosseous (impar) ligament and its branches bifurcate and enter the distal sesamoid bone through fine

vascular foramina, adjacent to (but separate from) the three to five synovial fossae that characterize the distal border. The synovial fossae appear as small, radiolucent, inverted bottle- or flask-shaped areas along the distal border of the bone. Arterial injections of contrast medium and "fine focus" radiography has clearly shown that each synovial fossa is lined with synovial membrane and connects directly with the distal interphalangeal (coffin) joint.[22] Indeed, contrast medium injected into the coffin joint will outline not only the capsule of the joint, but also the synovial fossa of the distal sesamoid bone. Histologic sections through the region show branches of the distal artery running, through connective tissue, palmar to the synovial membrane of the fossa.[23] Several branches of the distal artery enter the bone independently of the synovial fossae.

The terminal part of the digital artery enters the solar canal of the distal phalanx via the paired sole foramina and unites with the artery of the opposite side to form the terminal arch deep within the bone. Branches of the terminal arch (4 to 5 middorsally and 8 to 10 distally, near the solar border) radiate outward through foramina in the dorsal surface of the distal phalanx and supply the lamellar corium and, after forming the circumflex artery, the corium of the sole.

In addition to the 12 to 15 main foramina, the dorsal surface of the distal third of the distal phalanx is perforated by numerous finer foramina (the bone in this region is very porous), and recent evidence (C.C. Pollitt and G.S. Molyneux, unpublished data) shows that many of the vessels within these foramina are arranged anatomically to perform countercurrent heat exchange, that is, a central artery surrounded by a sheath of capillaries and venules (similar to the pampiniform plexus of the mammalian testis). This evidence implies that the equine digit is an efficient thermoregulatory organ, which is not surprising when the range of equine habitats, from the subarctic to the equator, is taken into consideration. Horses tolerate having their feet subjected to low, near-freezing temperatures without discomfort or ill effect. The potential of long-term cryotherapy has been validated by keeping the distal end of one limb in a boot containing an ice-water slurry for 48 hours while the remaining feet are at room temperature.[24] In the study, the horses seemed oblivious to the wide disparity (30° versus 5° C) of foot temperatures they were experiencing. This evolutionary adaptation can be exploited therapeutically. Forty-eight–hour distal limb cryotherapy even prevents the onset of acute, experimentally induced laminitis.[25]

The lamellar corium derives most of its blood supply from the branches of the terminal arch that perforate the distal phalanx. Numerous anastomoses form an arterial lattice beneath and between the epidermal lamellae, and blood can flow proximally to the coronary circumflex artery and distally to the solar circumflex artery (Figure 6-15).

The circumflex artery of the sole is an anastomosis of all the distal branches of the terminal arch and the dorsal arteries of the distal phalanx and forms a complete arterial loop supplying the corium at the junction of the distal lamellae and peripheral sole close to the sharp solar margin of the bone. All of the arterial blood supply of the sole (except for the angle between the bars and the heels) comes from axially directed arteries branching inward from the circumflex artery (Figure 6-16).

There are no vascular foramina perforating the solar surface of the distal phalanx (except for the palmar processes). This means that almost the entire corium of the sole is dependent on a blood supply that arises first on the dorsal surface of the distal phalanx and then curls under the margin of the distal phalanx. The solar corium is sandwiched between the epidermal sole and the unyielding solar surface of the distal

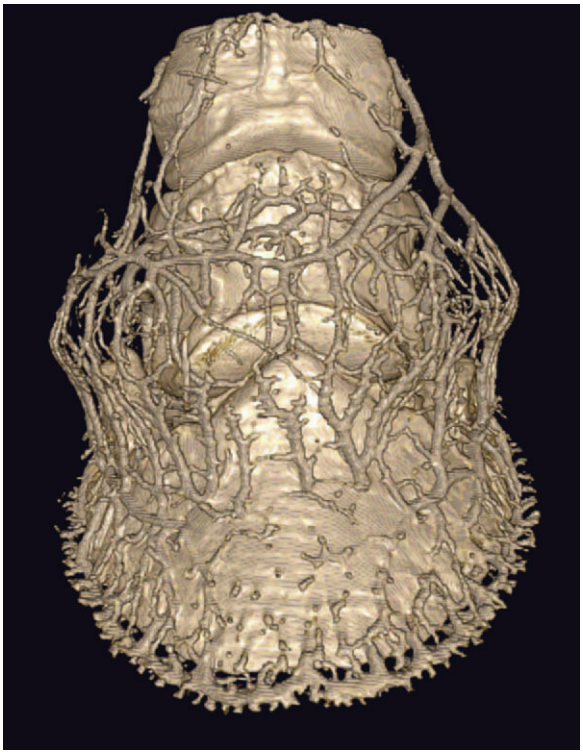

FIGURE 6-15 Three-dimensional reconstruction of a horse's foot scanned by computed tomography with the arterial circulation injected with contrast material. Note how arteries exit through foraminae in the dorsal surface of the distal phalanx and anastomose proximally with vessels of the coronet and distally to form the circumflex artery.

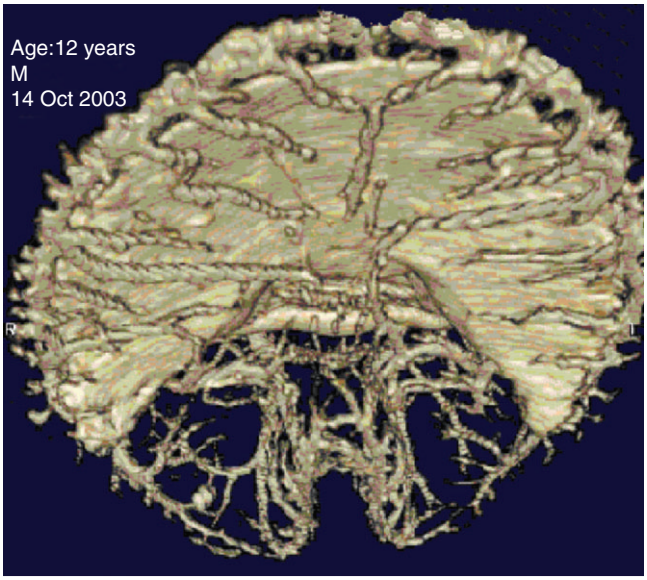

FIGURE 6-16 Three-dimensional reconstruction of a horse's foot scanned by computed tomography with the arterial circulation injected with contrast material. Note how axially directed branches of the circumflex artery supply the sole.

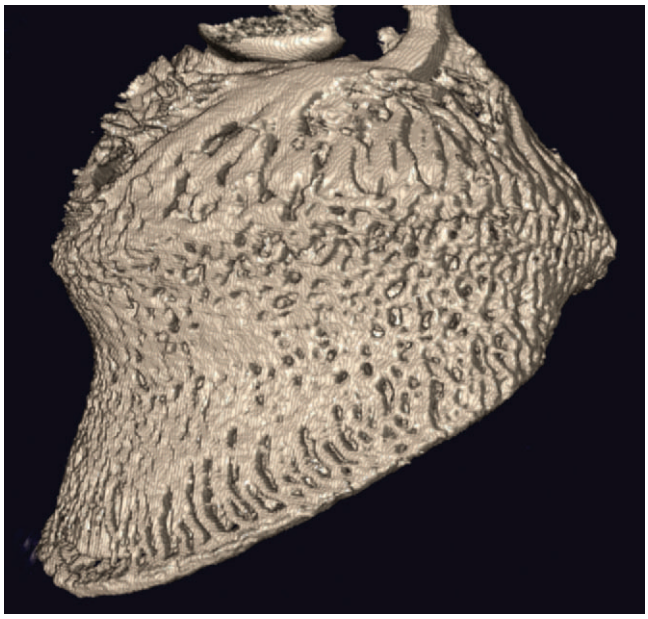

FIGURE 6-17 Three-dimensional reconstruction of a horse's foot scanned by computed tomography with the venous circulation injected with contrast material. Note the extensive degree of anastomosis among the valveless venous plexuses.

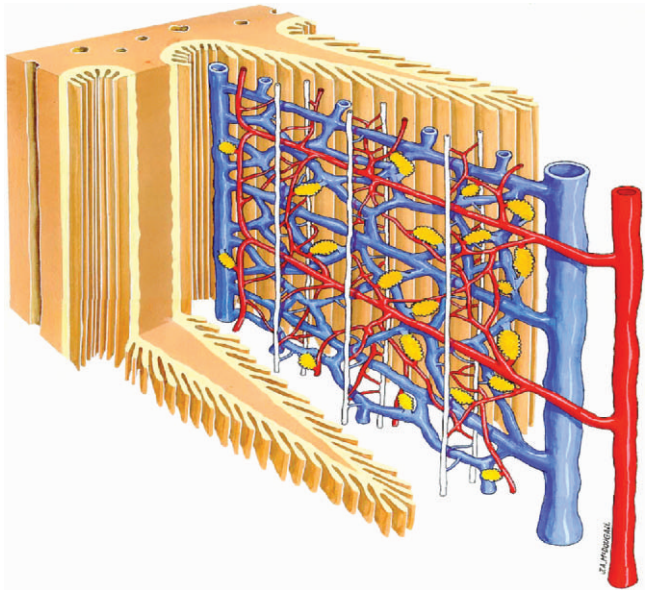

FIGURE 6-18 Diagram showing the dermal microcirculation. Arteriovenous anastomoses (*short yellow vessels*) are numerous and more concentrated near the bases of primary dermal lamellae. Secondary dermal lamella (SDL) capillaries (*white*) are shown reduced in number for diagrammatic reasons.

phalanx and is therefore prone to damage from compressive forces. If a horse is deliberately forced to stand or walk on the soles of its feet (by overzealous trimming of the ground surface wall), the sharp distal rim of the distal phalanx effectively cuts off the blood circulation to the central solar corium and results in severe lameness and, in some cases, necrosis of the sole. Laminitis-induced descent of the distal phalanx into the hoof capsule also causes dorsal sole necrosis by a similar mechanism.

Digital Veins

There are three interconnected valveless venous plexuses in the foot (Figure 6-17). The dorsal venous plexus lies in the deep part of the lamellar corium. The palmar/plantar venous plexus lies in the deep part of the sole corium and on the inner axial surfaces of the cartilages of the distal phalanx. The coronary venous plexus lies in the coronary cushion covering the digital extensor tendon and the outer abaxial surfaces of the cartilages of the distal phalanx. It anastomoses with the palmar/plantar venous plexus via foramina in the cartilages (note that both sides of the cartilages are covered by plexuses of veins). The three plexuses are drained by the medial and lateral digital veins. The deep veins within the foot are valveless, although valves occur in the more superficial coronary, subcoronary, and heel veins.

Reactions of the Venous Blood during Foot Loading

The hoof is subjected to a range of weight-bearing and locomotive forces. These forces are believed to cause expansion of the frog and to deform all the soft tissue of the hoof, including the digital cushion, the cartilages, and the vascular systems. Because the soft tissues of the hoof are encased by the hard keratinized wall, which cannot expand substantially,[26] the internal deformation of the hoof forces evacuation of the venous blood from the hoof quite quickly. The multiple routes of drainage of the wall and sole venous plexuses, the absence of valves in most veins of the hoof, the presence of valves in the proper digital veins and caudal hoof veins, and the presence of a double layer of venous plexuses on either side of the flexible cartilages are all mechanisms to evacuate the venous blood quickly and to distribute pressure evenly. The absence of valves would help evacuation by allowing venous blood to take any convenient path. The presence of the valves in the caudal hoof veins and proper digital veins prevents retrograde blood flow to the hoof and thereby ensures the efficient venous return of blood to the heart.[27,28]

The Inner Hoof Wall

In common with all epidermal structures, the lamellae of the inner hoof wall are avascular and depend on capillaries in the microcirculation of the adjacent dermis for gaseous exchange and nutrients. The epidermal cells closest to the dermis is the basal cell layer (germinal cell layer or *stratum germinativum*).

The Dermal Microcirculation

Numerous (500/cm^2) arteriovenous anastomoses (AVAs) connect the axial arteries and veins of the dermal lamellae.[29] AVAs are present throughout the dermal lamellae but are larger and more numerous around the axial vessels close to their bases (Figure 6-18). Studies with the transmission electron microscope show that AVAs are richly innervated by autonomic vasomotor nerves and their associated peptidergic nerves, have thick walls of smooth muscle, and have a specialized, characteristically tall, endothelium.[30] The normal role of AVAs is in relation to thermoregulation and pressure modulation. Dilated AVAs bring hot arterial blood to the inner

hoof wall and can cause rapid and large temperature fluctuations of the hoof wall. AVAs are equally numerous around the bases of the papillae of the coronary corium and the lamellar terminal papillae. In fact, the vascular architecture of a dermal papilla is basically the same whatever its source, and the blood vessels of the papillary dermis of the periople, coronary band, terminal papillae, sole, and frog regions have a common structural organization.

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7

EVOLUTION OF THE EQUINE DIGIT AND ITS RELEVANCE TO THE MODERN HORSE

ANDREA E. FLOYD

The prehistoric ancestors of the modern horse are a curious lot. In particular, the evolving digit and what the horse may have gained or lost during evolution, beginning with five toes and then culminating with a single digit, has intriguing implications for understanding of the modern equine digit. A highly recommended book on the subject is Bruce J. MacFadden's *Fossil Horses, Systemics, Paleobiology, and Evolution of the Family Equidae* (Cambridge University Press).*

This chapter takes the point of view of an equine podiatrist, not of a seasoned paleontologist, and is intended for reflection. The equine fossil collection at the Museum of Natural History in Gainesville, Florida, provided much of the basis for this discussion. The author's hope is that this discussion will raise interest in the subject in readers and will spark some thought and some discourse.

JOURNEY THROUGH TIME

Hyracotherium: Dawn Horse

In the distant past of the world, some 55 million years ago, in an epoch called the Eocene or "Dawn of the Recent," a pre-horse lived that was no larger than a modern house cat. The gargantuan dinosaurs had gradually given way to a new world of mammals. Steamy, lush forests of giant cypress, metasequoias, palms, and ferns dominated the landscape of the early and mid-Eocene epoch. This early world was the setting for a primordial mammalian stew pot that teemed with possibilities.

The little creature, first called *Eohippus* ("Dawn Horse") and then renamed *Hyracotherium* (Table 7-1), is the oldest pre-horse fossil identified by paleontologists. Some believe that this little ancestor may have been colored like a deer—tan, with spots to act as camouflage in the forests. It most likely had neither mane nor long hair in its tail. Its head would have been shorter than that of the modern horse, with a snout more like that of its relative the tapir.

On four toes in front and three toes behind, this little ancestor browsed on leaves, seeds, and fruits in the great, shadowy forests. Moving cautiously from shade cover to light, the little four-toed creature was well adapted to the terrain of the moist, subtropical world of the Eocene period, as having several toes made it easier to traverse wet ground. Each toe had a pad, much like that of a dog; but unlike the dog, it had hooves instead of claws (Figure 7-1).

The third metacarpal bone (equivalent to the modern cannon bone) and its phalanges were prominent and centrally weight bearing, whereas the second (medial) and fourth (lateral) metacarpal bones and their phalanges most likely acted as shock absorbers for the not yet fully weight-bearing primary toe. The second and fourth digits may also have been used for mediolateral balance; the fifth digit probably had little ground contact at this time (see Figure 7-1). The distal limb (metacarpal/metatarsal bones and phalanges) possessed rotational ability, much like the dog's foot. In other words, these creatures would have been able to rotate the leg and lick the pads of their feet. All of the major bones of the digit were present and unfused, unlike the second and fourth metacarpal bones (splint bones) of the modern horse.

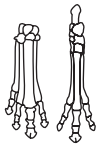
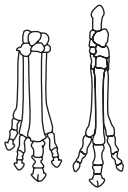
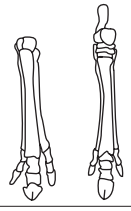
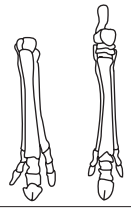
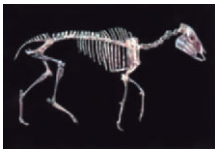
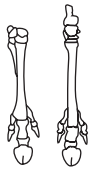
Orohippus: Pre-Horse of the Grasslands

As the continents drifted and great volcanic activity spread across the world, the Arctic and Antarctic land masses separated from their respective continents and were slowly drifting away. *Hyracotherium* slipped into the past and a slightly larger pre-horse, *Orohippus,* stepped out of the great forests and made its way into a new world—the emerging open patches of land. The forests were changing, too. Angiosperms (ancestors of the deciduous trees that exist today, such as walnut, oak, beech, and elm) were taking over the landscape from the previously dominant gymnosperms (the conifers or evergreens). This time was also the very beginning of a giant evolutionary leap: from ferns to branched plants to grasses. Grasses were the first plants that could effectively survive fire, as the leaves might be destroyed but the plant remained intact, allowing rapid regrowth. Although grasslands had not yet appeared as a dominant landscape, the appearance of grasses marked the beginning of a new way of life.

Still in the Eocene epoch, the time was about 50 million years ago, 5 million years after *Hyracotherium* first emerged. The climate was still subtropical and moist, and great herds of creatures—giant rhino and deer, camels, and piglike creatures—joined the little *Orohippus* in the emerging grassland patches. This small pre-horse was not much larger than *Hyracotherium.* It had the same number of toes but was starting to display predominance for weight bearing on the third digit (Figure 7-2). These little animals may have been hunted by ferocious giant cat-like and bear-like animals, strange bear-dogs, and primitive dog ancestors. The little pre-

*Special thanks to Dr. MacFadden for allowing access to the equine fossil collection at the Museum of Natural History in Gainesville, Florida.

Table 7-1 Evolutionary timeline of the equine digit

Horse	Epoch	Age, years (millions)	Body	Height, inches	Skeleton	Digit	Toes, front/back
Hyracotherium	Eocene	55		10			4/3
Orohippus	Eocene	50		12			4/3
Mesohippus	Oligocene	40		24			3/3
Miohippus	Oligocene	35		28			3/3
Parahippus	Miocene	20		36			3/3
Merychippus	Miocene	17		40			3/3
Pliohippus	Miocene	10		48			1/1
Dinohippus	Pliocene	8				First suspensory apparatus	1/1 or 3/3

continued

Table 7-1 Evolutionary timeline of the equine digit—cont'd

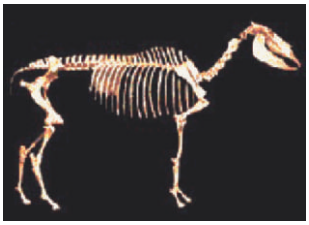
Horse	Epoch	Age, years (millions)	Body	Height, inches	Skeleton	Digit	Toes, front/back
Equus	Pleistocene to present age	5 and on		60			1/1

FIGURE 7-1 Distal limb of *Hyracotherium*.

FIGURE 7-2 Distal limb of *Orohippus*.

horse needed to be faster than its ancestor and always aware of its surroundings. One theory on this stage in evolution is that as the pre-horse moved from the great forests to the open grass areas, the need for speed started the adaptation of the upright central digit.

Mesohippus: Larger, Faster, Smarter?

In the new epoch, the Oligocene, a dramatic change occurred, affecting all species of animals on the planet. The earth cooled dramatically, starting glaciation in Antarctica. The capture of more of the world's water supply as glacial ice resulted in a drier climate. Dynamic tectonic (earth plate) movements isolated South America and Australia from the other continents. Tropical forests became restricted to the equatorial region, and more than 20% of the existing species of ancient mammals became extinct.

This was the time of a new pre-horse—a creature larger, faster, and perhaps smarter than the little dog-like *Hyracotherium* and *Orohippus*. This new horse, *Mesohippus*, was almost twice the size of *Orohippus*, standing 24 inches tall at the shoulders. It had a longer neck and face and was beginning to have other characteristics of the modern horse, including longer legs and a straighter back.

The grass plains were also changing. Ten million years after *Orohippus* walked the earth, the grasslands were expanding and becoming vast, open plains. As the climate cooled and many places became more arid, the edges of the great forests started to recede. Great conifer forests became dominant in the northern regions, and hardwood deciduous forests predominated

FIGURE 7-3 Distal limb of *Mesohippus.*

FIGURE 7-4 Distal limb of *Miohippus.*

in the middle regions of the earth. *Mesohippus* needed to have good grinding teeth for the tougher grass, as it contained silica (a hard, abrasive material), and longer legs to outrun the predators that were also evolving and increasing in size.

Mesohippus had three toes in the hind limb and three toes in the forelimb. The other toe of the forelimb had become quite small, or vestigial, and was barely visible (Figure 7-3). The swamps had given way to soft ground, so the side toes (digits two and four) were no longer needed as before. The medial and lateral supporting toes decreased in length and diameter, while the middle or third toe increased in size and strength. *Mesohippus* still had pads like its ancestors and still had a rotational lower limb, but its hooves started to have more contact with the ground. *Mesohippus* is considered the intermediate between the earlier dog-like pre-horses of the Eocene period and the more horse-like ancestors that were to come. Thus it was named *Mesohippus,* or "Middle Horse."

Miohippus: Further Adaptation and Diversity

During the Oligocene period, a new pre-horse evolved called *Miohippus.* This and other pre-horses actually coexisted for about 4 million years. In fact, in one part of what is now Wyoming, three species of late *Mesohippus* appear to have coexisted with two species of *Miohippus.*[1] By the middle of the Oligocene epoch, however, *Mesohippus* had disappeared. *Miohippus* was larger than *Mesohippus* and had a slightly longer skull. Still three-toed, *Miohippus* showed more increase in third metacarpal/metatarsal density and strength. The lateral and medial toes probably still contacted the ground, but only while the animal was running (Figure 7-4).

The first burst of diversity among the pre-horse species occurred during the Oligocene period, from which numerous and distinct species have been discovered. Commonly referred to as the Miohippus Radiation, two main lines of evolution and one side branch are recognized: the Anchitheres (or browsers), the Grazers, and *Archeohippus* (or "Pygmy Horse"). All were thought to coexist, but it was the Grazers that survived the coming changes and became the next pre-horse on the way up the evolutionary path to the modern horse.

Parahippus: On Tip-Toe

With the beginning of a new epoch 24 million years ago, the first horse of the Miocene period appeared and the earth assumed a more modern configuration. Twenty million years ago, Antarctica was covered by ice, not subtropical as it had been during the Eocene period, and the northern climate was cooling rapidly. Florida and parts of Asia were submerged under the ancient oceans.

As the climate cooled and became even drier, the great blanket forests of the past opened into woodlands interspersed with grasslands. Certain areas of the world saw dramatic increases in the size of their open plains; this was the time of formation of the African savannah, North American plains, and South American pampas. With the pressure of such vast, treeless grazing areas, a new species of horse, with speed and the potential for long-distance migration, evolved. Thus *Parahippus* was an early link between the old forest dwellers and the true plains grazers.

Parahippus still possessed three toes (Figure 7-5), but the lateral and medial toes were much smaller than those of earlier

FIGURE 7-5 Distal limb of *Parahippus.*

FIGURE 7-6 Distal limb of *Merychippus.*

species. The muscles that had supported the lower limb withdrew to the upper portion of the leg, creating longer and stronger tendons. Rotational movement, as well as adduction and abduction (medial and lateral movement) of the lower limb, were almost eliminated. The bones of the second and fourth digits began to fuse, and a specialized flexion and extension (forward and backward swinging movement) of the leg appeared, allowing for more efficient high-speed flight and long-distance migration.

Parahippus was the first horse to stand on tip-toe, no longer using its calloused pad for support. The pad started to recede inside the hoof, and the hoof changed in size and strength to assume a primary weight-bearing role. Of great interest is the recession of the pad of the foot into the distal interior of the hoof. This tough, pliable, blood-filled tissue became the bulbar tissue, frog, and digital cushion of the modern hoof—structures that are puzzling even today. It is possible that the distal and middle scutum of the deep digital flexor tendon (see Chapter 1) are also vestiges of the pad system of the early foot.

Parahippus possessed the first "spring foot," in which the weight of the body was supported by strong tendinous attachments in the lower limb. It stood about three feet tall and had specialized teeth to handle the tougher and drier grasses of the great plain regions. All of these changes occurred rapidly, geologically speaking. (One or two million years is considered a short period of time in evolutionary terms.)

Merychippus: The Hoof Adapts Further

During the late Miocene period, some 11 to 17 million years ago, the first true grazing horse appeared on the great plains. The vast plains of the late Miocene epoch were thick in drier, coarser grasses. This type of grass required that *Merychippus* become a specialized grazing animal. *Merychippus* stood about 35 to 40 inches tall and looked very much like the modern horse. It still retained three toes (Figure 7-6), but the second and fourth toes showed a great deal of variation in size as they became nonessential and lost their function. In some individuals, the side toes were still full-sized and touched the ground when the animal was running, whereas in others in the same herd, these digits were smaller and fused to the third digit.

Merychippus was fully spring-footed and stood on the tip of its distal, or third, phalanx (P3) in the new tip-toe stance of its predecessor, *Parahippus.* But now P3 of the third digit was expanding in circumference and becoming more concave as the digital cushion and frog withdrew further into the hoof capsule. The hoof capsule most likely was beginning to look more like that of the modern hoof.

The late Miocene plains—with its great savannahs and teeming, swirling herds of great herbivores—must have been equaled in species diversity only by the African plains of the modern epoch. The Merychippus Radiation, similar to the earlier Miohippus Radiation, saw 19 grazing species, divided into three groups, emerge about 15 million years ago.

The greatest diversity occurred about 10 million years ago and has not been equaled since. A great variety of browsers and grazers, large and small, lived together on the savannah. Some of these species may even have had scent glands in their faces. But the *Merychippus* dominated the landscape and represented the next evolutionary step.

FIGURE 7-7 Distal limb of *Pliohippus.*

Pliohippus and Dinohippus: Spanning the Epochs

Still in the late Miocene period, between 6 and 12 million years ago, the grandfather of the modern horse appeared. *Pliohippus,* larger and more horse-like in conformation than any prior, species consistently planted a single digit on the ground. The side toes were small and no longer of any use, giving way to new collateral ligaments that developed to stabilize the fetlock (metacarpophalangeal or metatarsophalangeal joint) at high speeds. The second and fourth metacarpal bones, now vestigial as splint bones, fused along the lengthened third metacarpal bone (Figure 7-7). *Pliohippus* spread throughout North America, South America, Asia, and Europe.

One more pre-horse has been discovered recently and is believed to be the closest relative to the modern horses, *Equus simplicedens* and finally *Equus caballus*. *Dinohippus* lived about 13 to 15 million years ago, and what little evidence has been found suggests that it was a single-toed species, with the vestigial side toes now no longer found. *Dinohippus* lived during two epochs, Miocene and Pliocene.

Emergence of Equus

In the Pliocene epoch, the climate was cooling and becoming sharply seasonal, very similar to the modern climate. Deciduous forests dominating the northern continents, and coniferous forests and tundra dominated the furthest northern regions. An arctic ice cap had formed, trapping more water as ice and drying the world climate even further; and Antarctica was covered in ice by the end of the Pliocene epoch. The tropical forests were limited to narrow bands around the equator, with dry savannah grasslands in Africa and Asia. Global temperatures were similar to those of today.

Many herbivores grew larger and developed huge hindguts to digest the increasingly coarse forage. The new hindgut fermenting vats meant these animals were able to survive longer periods of drought and food shortages, as they could retrieve more nutrients from the grass they were able to find. These great herds of herbivores included the horses of the Pliocene epoch. They were able to travel long distances in huge, mass-herd migrations in search of food and better climates for giving birth, sometimes traversing entire continents in their migrations. This was also the time of the emergence of an interesting species: early humans, another predator with which the early horse would have to contend.

Two million years ago there was a dramatic shift in world temperatures. The Amazon rainforest crept in from the coast, forming an impenetrable barrier between the two Americas. The thick forests stopped the movement of the plains animals, thus isolating the species from one another. Ice sheets encroached and retreated during four or five separate glacial periods that, combined, are known as the Great Ice Age. This period is the Pleistocene epoch, and at its peak 30% of the earth was covered in glaciers and frozen oceans. The ice in certain areas was as deep as 13,000 feet (4000 m), causing ocean levels to drop. It was cold and dry, with severe seasonal changes. So much water being locked in ice had devastating results for many species. At first they adapted, growing wooly coats and carrying more fat on their bodies as insulation, but owing to their lengthy gestational periods and human predation, many species became extinct.

With a brain larger than *Dinohippus* and a single toe supported by strong side ligaments to keep it from twisting and rotating, three early horse species called the *Equus simplicedens* group emerged (Figure 7-8). Stocky, shaggy, straight-shouldered, and thick-necked, with short, narrow, donkey-like skulls, these early *Equus* species must have looked much like their later descendants: the ass, the horse, the quagga, and the zebra. They may have had stiff, upright manes and ropey tails, medium-sized ears, and striped legs—perhaps quite similar to the animal shown in Figure 7-9, an interesting animal frequently seen in the Dominican Republic today. This group diversified further into 12 new species, in four different groups, all coexisting in separate herds for a time.

All of these species existed in Africa, Asia, Europe, and North and South America in enormous migrating herds until the late Pleistocene period, when extinctions killed off most of the larger mammals in North and South America, Europe, and Asia. For the first time in tens of millions of years, from 8000 years ago until the 15th century AD, the Western hemisphere was without any horses. They did not appear again until they were brought to the New World by the Spanish conquistadores.

Today's Horse

The current epoch is the Holocene. Every breed of horse alive today is of the species *Equus caballus* (Figure 7-10), yet there is great diversity among the breeds. Through less-than-thoughtful breeding programs, it might seem that the foot of the horse is in danger of being bred off altogether. However, change is a tremendous propellant for evolution, and evolution is still in progress. The genetically engineered horse of today is no less

FIGURE 7-8 Distal limb of *Equus simplicidens*.

FIGURE 7-9 Dominican donkey (*Equus caballus* species).

subject to the natural laws of the universe than were its predecessors. Left to its natural devices, for example, the feral horse of today has accommodated a handy little hoof that works perfectly in whatever environment it must adapt to.

MESSAGES FROM THE PAST

Vestigial Structures

There are several features of the modern equine digit that still reflect the origins of this majestic species. Those of clinical interest include the crena (a notch often found at the tip of

FIGURE 7-10 Distal limb of *Equus caballus*.

P3), the frog and underlying soft tissues, and the occasional appearance of a supernumerary (extra) digit.

Crena

The crena is a centrally located notch in the dorsal-distal margin of P3 that is often found in horses today. Its clinical significance is still debated by academics and practitioners. One of the most interesting findings in the research of fossil digits is that the distal phalanx of the third digit in the pre-horse species had a cleft or split in the center, from the toe to about one-third of the way up the dorsal surface of P3 (Figure 7-11). This cleft was found in all specimens observed up to the middle of the Pleistocene epoch, or about the time the immediate ancestor of the modern horse emerged. Although its function in the prehistoric horse remains to be determined, the common presence and consistent location of this notch in the modern horse indicates that the crena is vestigial in nature and should be considered nonpathologic.

Hoof Pad

Also of considerable interest is the evolutionary change that caused the pad of the third digit to become the soft bulbs, frog, and digital cushion of the modern hoof. The first known pre-horse *(Hyracotherium)* had a pad very much like that of a dog, with a hoof capsule that sat in front of the pad and was non–weight-bearing. Millions of years of evolutionary pressure brought the pad underneath the hoof as the hoof became a functional weight-bearing structure.

These observations raise the question of whether the frog should be in contact with the ground. It is this author's

FIGURE 7-11 **A,** Crena of the third phalanx (P3) of *Archeohippus* species. **B,** Radiograph of the crena of a Thoroughbred. **C,** Photograph of the crena of a Quarterhorse. *(**C,** Courtesy Lancaster and Bowker/MSU Foot Lab, 2003.)*

opinion, based on the evolutionary evidence, that the frog should not have ground contact, except at the moment of heel loading. The moment of heel impact is followed by a natural cupping or grabbing of the ground, which allows the soft tissues to move proximally within the hoof capsule, temporarily forcing blood out of the tissues of the digital cushion, bulbs, and frog (see Chapter 2). Constant pressure on the frog or bulbs causes compression of the bulbar circulation and weakens the heels and their soft tissue support system.

Supernumerary Digits

Extra digits, although rare, do occur in the modern horse (Figure 7-12) and are of great interest and novelty. It is possible that their sporadic appearance is simply a genetic anomaly left over from the ancestral code.

Balancing on a Single Toe: The Perfection of Asymmetry

Reducing the number of digits from five to one has required some unique adaptations to ensure balance and stability when standing and moving on the tip of that single digit. In humans, weight bearing is concentrated in the first, or primary, metatarsal and phalangeal bones, that is, the big toe and associated bones in the foot (Figure 7-13). The other four digits primarily provide lateral balance and stability to the limb; this function is accomplished by fanning out of these four digits. In carnivores, the primary digit (the dewclaw) has receded to a nonfunctional status, and weight bearing appears to take place primarily in the third and fourth digits; the second digit (and, to some extent, the fourth) assumes the role of providing mediolateral balance.

In horses, evolution has concentrated weight bearing and balance in each limb into one digit (the third digit) and its associated hoof capsule. The other four digits have long ago receded from ground contact, making the equine distal limb a highly specialized structure. It can be assumed that the equine digit has evolved to provide the same functions of support and balance provided by the other digits in multitoed mammalian species, through a sophisticated change in its anatomical construction.

Pattern of Asymmetry

There is a general symmetry to the equine distal limb, in that all four limbs are structurally similar, but within this general symmetry of form is a consistent *pattern of asymmetry* between medial and lateral sides. From the distal extremity of the third metacarpal/metatarsal bone (cannon bone) all the way down to P3, the articular surfaces of the cannon bone and each of the phalanges are slightly larger in area on the medial side of the limb than on the lateral side.

At the fetlock joint, the articular surface at the distal end of the cannon bone comprises two condyles, which are separated by a sagittal ridge. This ridge is not quite centrally located, so it divides the articular surface into a slightly larger medial condyle and a slightly smaller lateral condyle. This configuration is mirrored in the facing articular surface at the proximal end of the first phalanx (P1): the shallow articular cavity that lies medial to the sagittal groove is slightly larger than that on the lateral side of the groove (see Chapter 1).

This pattern continues down the digit, at the pastern joint (between the first and second phalanges) and at the coffin

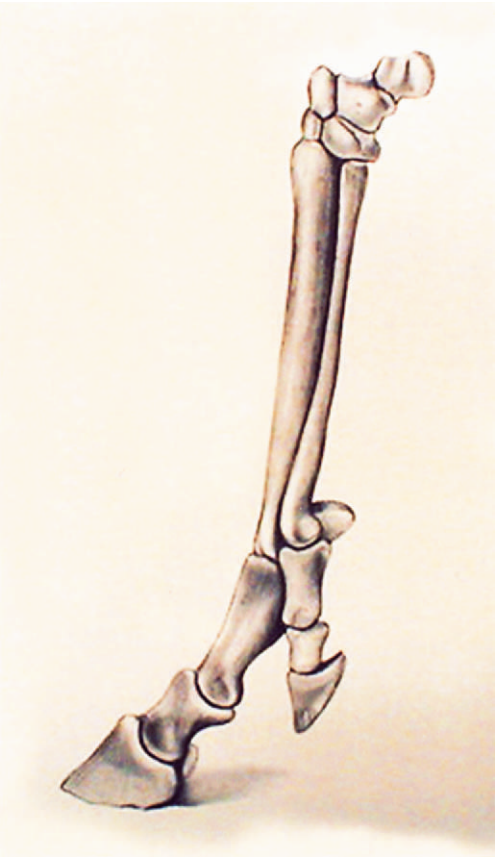


FIGURE 7-12 Example of supernumerary digits (polydactyly).

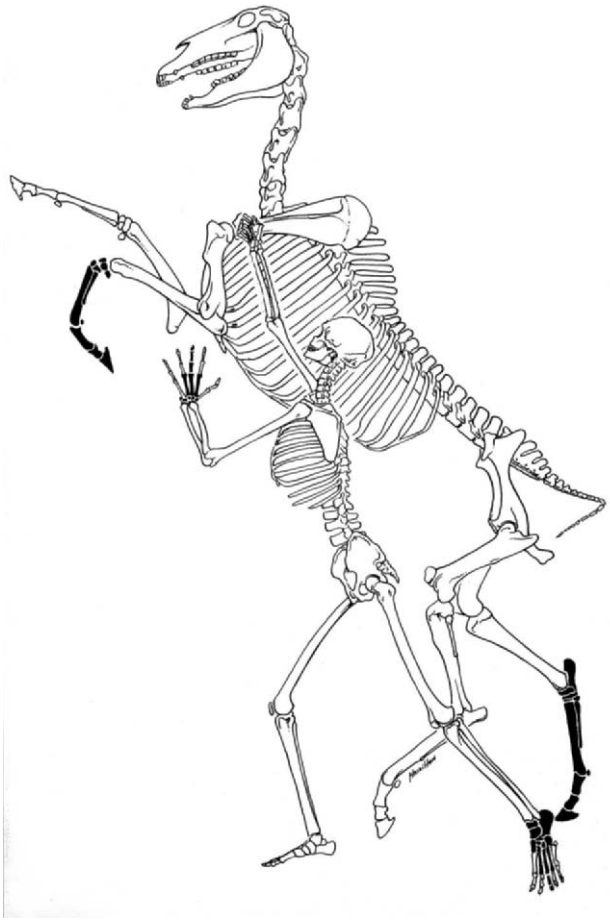


FIGURE 7-13 Comparative anatomy of horse and human. *(Drawing by Hamilton from exhibit at the Natural History Museum, New York, New York.)*

joint (between the second and third phalanges). It is also reflected in the shape of P3 itself. This remarkable bone is somewhat fan-shaped, in that it dramatically widens from proximal to distal (as does the overlying hoof wall). But while P3 retains a general symmetry between medial and lateral sides, the outer (parietal) surface of P3 on the medial side of the foot is a little more steeply sloped than that on the lateral side.

The overlying hoof wall likewise is slightly more upright on the medial side than on the lateral side. Looking at the solar surface of the foot, there is slightly less hoof mass medially than laterally. Prehistoric hoof capsules are not available for observation, but the phalanges of all of the spring-toe, or single-digit, pre-horses demonstrated the same medial and lateral condylar area size differences.

Implications

The horse appears to have adapted the distal limb so that the functions of weight bearing and mediolateral balance are both achieved in the one digit. Its structure suggests that the medial condyles and corresponding articular cavities are larger, to support proportionately more of the horse's body weight and its compressive forces. The fact that the parietal surface of P3 and the overlying hoof wall are slightly more upright medially than laterally also suggests that the hoof is adapted to support slightly more weight and impact on the medial side. Logically, a more upright parietal surface and hoof wall is better equipped to function in a load-bearing capacity than is a more sloped structure.

The slightly greater slope on the lateral side of P3 and the overlying hoof wall suggests that this side of the foot is adapted to play more of a role in stability and balance, similar to the role played by the lateral four digits in the human foot and the second and fourth digits in the canine foot. With a decrease in the number of digits providing balance and stability, it appears that the solitary digit of the horse has found a way to express functions of both weight bearing and balance simultaneously.

Studies

Balch et al.[2] studied the effects of altering mediolateral balance on the horse's gait by using wedges to elevate either the lateral or the medial side of the foot. Of particular interest was the finding that elevating the lateral side of the hoof caused no significant alteration in the horse's gait. In contrast, elevating the medial side of the hoof caused significant changes in the horse's gait. These observations suggest that the medial wall assumes more of a primary weight-bearing role than does the lateral wall.

Also of interest was the fact that one of the study subjects (a horse with normal limb conformation) preferentially loaded the medial hoof wall, regardless of hoof orientation or gait.

Another horse, this one with base-narrow and toe-out conformation, appeared to load its feet normally until the hoof was trimmed to correct the conformation, at which point the horse began landing on the lateral side of the hoof first. The authors concluded that, if preferential weight bearing on the medial side is characteristic of sound athletes, then the base-narrow, toe-out horse may benefit from being shod with a medial wedge to re-establish the pattern of optimal hoof kinetics.[2]

A recent study by Kane et al.[3] examined whether hoof imbalance was a significant cause of catastrophic injury in racehorses. The study involved data collected from Thoroughbred racehorses that died from any cause at racetracks in California. The authors proposed a biomechanical explanation for the relationship between hoof imbalance and the risk for suspensory apparatus failure and condylar fracture (a vertical fracture through the lower end of the cannon bone that separates one condyle from the main portion of the bone). Of interest was the hypothesis that lateral displacement of the weight-bearing surface (through hoof imbalance) could result in abnormal loading of the fetlock region, with proportionately more load being placed on the lateral condyle, thereby increasing the risk for a lateral condylar fracture.

Although the study evaluated many different indices,[4] the following findings are of particular interest:

- Catastrophic musculoskeletal injuries were more common in horses in which the ground surface of the foot was wider on the medial side than on the lateral side (i.e., the opposite of what is normally found).
- Specifically, in horses with condylar fractures, the surface area of the sole was larger on the medial side, compared with the control group (horses that did not sustain catastrophic injury).

The effects of greater ground surface width differences and solar area differences on the odds of injury were significant. The differences in lateral-medial angle, ground surface width, frog-to-wall distance, and sole area generally were greater in control horses than in injured horses, and increased sole area difference was significantly associated with lower odds of the above-mentioned injuries. In other words, the more asymmetrical the foot is (i.e., the more difference there is between medial and lateral sides), the lower the risk is for catastrophic injury during a race. Specifically, the authors concluded that:

1. The greater the ground surface area is on the lateral side of the foot, the lower the risk is for injury.
2. Perfect mediolateral symmetry may not equate with optimal mediolateral hoof balance.

Additional evidence for the importance of asymmetry is provided by data collected from feral horse hooves.[5] In these horses, there is an obvious difference between the medial and lateral hoof wall angles, with the medial hoof wall being steeper and straighter than the lateral hoof wall. Furthermore, the surface area of the sole on the medial side of the foot is smaller than that on the lateral side. Although these observations are not as apparent in the domestic hoof, the tendency toward this conformation is present.

SUMMARY

One of the most important lessons to be learned from the literature and from the feral hoof is that absolute symmetry of the bearing surface of the foot is not necessary and may in fact be detrimental. For the single digit to function in two different capacities simultaneously (i.e., load bearing and stability/balance), the two halves of the hoof need to be adapted accordingly: a steeper hoof capsule and smaller solar surface medially to provide better load-bearing capacity, and expansion of the hoof capsule and solar surface laterally to provide stability and balance.

It is clear that the horse has a highly specialized digit that functions as several digits in one. However, many questions are raised by study of the evolution, anatomy, and biomechanics of the equine digit, not the least of which are these:

1. Are there substantial and relevant differences between domestic and feral horses?
2. If so, are they genetically imposed by our breeding programs or are they directly imposed by our current farrier techniques?
3. Are we predisposing domestic horses to injury through our manipulations?

Hopefully some of these questions will be answered in the next few years as laboratory and clinical research into this remarkable structure continues.

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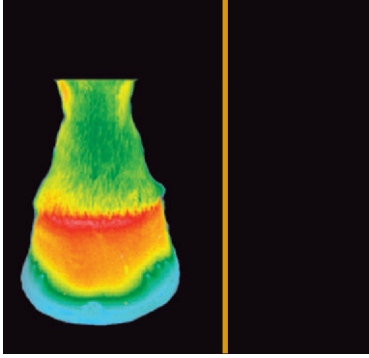

Section II DIAGNOSTIC EVALUATION OF THE EQUINE FOOT

8 EXAMINATION OF THE EQUINE FOOT

WILLIAM A. MOYER and G. KENT CARTER

This chapter is intended to deal with the most basic and important aspect of veterinary practice, conducting an examination. The physical examination is the beginning point; it is the one aspect of veterinary medical practice that is generally affordable to the consumer, and it remains the single most useful overall determinant in arriving at a diagnosis or diagnoses. How veterinarians examine a horse is based on how they were initially taught, their experience, and methodologies borrowed from myriad past and present equine practitioners. The procedure to be described is purposely long and detailed; however, the actual act of such an examination takes minutes to perform. The intent is to be complete and consistent, and ultimately the examination procedure will become habit.

An unknown percentage of cases occur in which an absolute diagnosis at the time of examination is not possible, as there is simply insufficient information available other than having proven that the pain and thus lameness originates in the foot or feet. Over the past few years, advances have been made in knowledge of anatomy,[1,2] imaging technology,[3,4] and the use of diagnostic anesthesia.[5] Such information has provided "new" diagnoses and questions concerning "older" diagnoses (navicular disease/syndrome being the prime example). A consistent and thorough physical examination in combination with an accurate history is often the most likely entity to direct a diagnosis in the absence of access to more advanced forms of imaging or to interpret findings using imaging technologies.

The success of any physical examination is dependent on a working knowledge of anatomy, the ability to control the animal, experience, and a willingness to be thorough and consistent. The past few decades have been blessed with wonderful advances in imaging and computer technology, but many cases do not require such interventions, at times they cannot be afforded by the client, and such advances are generally of limited value without an appreciation of history and clinical signs. The old platitude, "More diagnoses are missed from not looking rather than from not knowing," persists. This chapter is about the "looking" part.

Foot problems, considered as a group, are generally thought to be the most common cause of lameness in horses. The list of conceivable insults and problems is extensive. Foot and shoeing problems are also thought to influence or act as initiators of problems elsewhere on the musculoskeletal system, such as the use of toe grabs in Thoroughbred racing and the relationship to the incidence of suspensory ligament injuries. An individual horse's foot, regardless of age or breed of the horse, is a reasonably small object that is easily viewed, handled, and manipulated (assuming the examiner is physically able and the horse willing), yet as an anatomic unit accounts for myriad frequently occurring and recurring problems.

Unfortunately, interpreting what may be seen or discovered upon examining a horse's foot can be confusing. Any given foot will quite often demonstrate several coexisting abnormal findings or findings concurrent with problems elsewhere on the musculoskeletal system. Abnormal findings must be interpreted with the whole horse and in light of the clinical history. It is philosophically important to approach such an examination with the consistent goal of being complete and open-minded. A practiced and consistent examination procedure is likely to provide accurate results.

The examiner must develop a meticulous, "hands on" appreciation of foot anatomy. The construction of the majority of the foot is such that structures within the hoof capsule cannot be visualized or palpated; thus, the skilled examiner "sees and manipulates" the foot as if he or she had "x-ray vision" by trying to visualize the deep structures as if examining a radiograph of the structure. Such vision only comes with a deeper appreciation of internal anatomy, a consistent "practiced" approach, and constant correlation of radiographic findings to what is seen or palpated externally. It is very helpful for the examiner to have a thorough appreciation of the wide variety of possible disorders. The following list represents possibilities by their location:

- ***Coronet:*** direct trauma or bruising, foreign body penetration and infection, laceration, avulsion, displacement, dermatopathies (fungal, chemical, allergic, parasitic, neoplastic, and idiopathic) also termed *coronitis,* cracks, and scars
- ***Hoof wall:*** cracks at any location (to include the bars), wall separations, wall loss or avulsions, hoof wall growth abnormalities (focal absence of growth and shape changes such as flares, dishing, bulging, and ring formation), excessive or inadequate length, and poor hoof quality (flaky, brittle, excessively soft or weak)
- ***Sole:*** solar bruising, external solar penetration and infection, solar laceration or loss/avulsion, internal solar penetration via the third phalanx (severe laminitis), and excessive thinness, weakness, or flatness
- ***Laminar wall tissues:*** laminitis; keratoma; infection; hematoma and tearing; invasion via canker, sarcoid, or squamous cell carcinoma; abnormal cornification as the result of chronic wall separation; so-called white line disease; disorientation of laminar pattern from known and unknown sources; and toxic insults (e.g., selenium toxicity)
- ***Frog:*** thrush, canker, penetration and infection, loss via avulsion, bruising, and atrophy
- ***Heel bulbs:*** direct trauma and bruising, laceration, avulsion, dermatopathies (fungal, chemical [often iatrogenic from foot remedies], allergic, parasitic, neoplastic), abscessation and infection, and cracks
- ***Navicular bone:*** absence (agenesis), navicular disease and syndrome, fracture, ligamentous damage (impar and suspensory ligaments), infection (osteomyelitis), proximal displacement (impar ligament disruption), and incomplete ossification
- ***Navicular bursa:*** noninfectious and infectious (penetrating wounds) bursitis and partial or complete obliteration via adhesion formation
- ***Deep digital flexor tendon and sheath:*** tendinitis, severance or detachment at the third phalanx, degenerative and adhesive tendonitis (often the result of navicular disease or bursal infection), tenosynovitis (infectious [penetrating wounds] and noninfectious), and flexor tendon contracture
- ***Ligaments:*** desmitis, calcification, and disruption of the medial and lateral collateral ligaments of the coffin joint, impar and suspensory ligaments of the navicular bone
- ***Digital cushion:*** atrophy, absence, penetration and infection, extension of canker or thrush
- ***Third phalanx:*** fractures (types I-V); infection (septic osteitis); rotation or displacement within the hoof capsule; absence (agenesis) and incomplete ossification; pedal osteitis (which is a radiographic description of solar margin bony loss and widening of vascular channels and perhaps should not be considered a diagnosis); focal loss of bone as a result of space-occupying lesions (keratoma), bone infection, or avascular necrosis; and extensor process damage (fractures, common/long digital extensor tendons [can be mistaken for an extensor process fracture])
- ***Coffin joint:*** arthropathies (infectious, traumatic, iatrogenic, subluxation, luxation), ligamentous damage (collateral ligaments), osteochrondrosis, and intra-articular fractures of second or third phalanges), and extensor process damage (fractures, common/long digital extensor tendinitis at attachment, and focal calcification of common/long digital extensor tendons (can be mistaken for an extensor process fracture)
- ***Collateral cartilages (medial and lateral):*** ossification (sidebone), infection or aseptic necrosis (quittor), and fracture
- ***Palmar digital artery:*** occlusion (thrombus) and transection
- ***Palmar digital nerve:*** transection and neuroma

These abnormalities can also exist in various combinations. That is, an individual foot or horse may have multiple problems or findings. For example, the same foot may demonstrate third phalanx rotation, septic osteitis and bone loss, penetration of the sole, deep flexor tendon contracture, and subsolar infection. Horses with quarter cracks often show evidence of subsolar bruising, long toe/low heel conformation, and radiographic evidence of third phalanx (P3) marginal changes (so-called pedal osteitis). Failure to recognize the total situation could easily lead to inappropriate or incomplete therapy or inaccurate appreciation of the prognosis.

The clinical signs for each problem or combinations of problems of the horse's foot may point to a given diagnosis or diagnoses, but in most cases, foot problems often share clinical findings (lameness of varying degrees, similar responses to hoof testers, and response to diagnostic local anesthesia). For instance, a nonarticular fracture of the coffin bone, a mild case of laminitis, and focal subsolar bruising can all appear alike unless more detailed examination and radiography are undertaken.

HISTORY

Obtaining a useful and accurate history is critical to performing a good examination. Taking a history is a skill that should

improve with experience. A history may provide an instant diagnosis ("horse stepped on a sharp object") or a variety of clues that lead to the diagnosis. It is also very important to understand that the useful and critical information may not be forthcoming. That is, the examiner should not assume someone will automatically provide such; he or she has to ask for it and in a format that is both understandable and nonthreatening. Unfortunately, clear and accepted terminology for the various conditions and for trimming, balancing, and shoeing practices is still elusive in veterinary medicine; thus familiarity with common vernacular is important.

Hoof wall growth is a reasonably rapid process and is one that can respond to both internal and external factors, so historical information may lead to an explanation for why a given abnormal hoof wall shape or condition exists. The foot can change its shape and composition quickly. Thus the examiner should attempt to discover those factors that may have affected the individual patient.

Many horses are affected, for better or worse, by horseshoeing. The art and science of horseshoeing is an ancient practice but unfortunately clear and universally accepted terminology has not emerged. For example, the terminology (shoe types and trimming practices) can vary remarkably from one breed, sport type, or farrier to the next. The language and terms used to describe how a given Standardbred trotter is trimmed, balanced, and shod may be totally different from the language and terms used to describe how a Thoroughbred racehorse is handled. For example, a Standardbred racehorse farrier may indicate that a given horse is "balanced," which in their parlance means the horse, at racing speeds, is not hitting or interfering. In the event of confusion, it is best served to ask or be shown, as opposed to assuming a mutual understanding. It is also important to note that a given horse on the day of an examination may well have been shod differently before the examination. For example, a Thoroughbred racehorse with a hindleg lameness of 3 weeks' duration may have been shod with outside heel caulks to handle a muddy track 3 weeks ago and recently reshod with standard aluminum racing plates.

The questions asked in obtaining a good history vary with the patient. Many answers are often obvious and assumed, whereas others must be pursued and based on the individual. The following represents some salient questions and their rationale:

1. What is the presenting problem and how long has it existed?
2. Is there a pattern to the lameness and what does the owner or trainer suspect is the problem and the cause? It is prudent to never assume an owner or trainer will volunteer useful information; it is best *always* to ask. The breed, or more importantly the sport type, may provide information regarding the incidence (likelihood) of certain foot problems. There appears to be a predilection for certain problems based on sport type and breed, although foot abscesses, bruising, laminitis, and thrush tend to be ubiquitous. The following list contains specific problems to sport type and breeds:
   - *Thoroughbred and Quarter Horse racing:* foot bruising, pedal osteitis, P3 fractures, heel bulb damage (from overreaching), quarter cracks, nail problems, sheared heels, and long toe/low heel–related problems such as heel pain.
   - *Standardbred harness racing:* heel and quarter cracks, foot bruising, P3 fractures, pedal osteitis, and heel bulb damage from cross-firing. It should be noted that Standardbred racehorses are the most frequently shod of all sport types and they tend to train and race on less forgiving and more abrasive surfaces (which has significance when attempting to determine the presence or absence of abnormal shoe wear as well as interpretation of its meaning).
   - *Rodeo sports and polo:* pulled shoes and subsequent hoof wall loss, navicular disease and related problems, P3 fractures
   - *Show jumpers, 3-day event horses:* foot bruising, pulled shoes and hoof wall loss, navicular disease and heel pain, quarter cracks, and a high incidence of weak walls resulting from exposure to excessive bathing and detergents (a common practice in the show horse world)
   - *Gaited horses (Morgans, American Saddlebreds, Tennessee Walking horses, and Arabians):* hoof wall loss, hoof wall cracks at any location, and thrush. These are perhaps the most difficult horses to shoe properly as a result of the shoe-affected movement that the gaited show horse community desires.
   - *Endurance and trail horses:* foot bruising, hoof wall loss as the result of pulled shoes, heel bulb damage from overreaching, and fatigue. However, these horses often have some of the strongest feet in the equine athletic world (possibly a function of both horse selection to "do the job" and constant exercise).
   - *Turned-out horses (all breeds):* hoof wall loss and breakage, superficial hoof cracks, abscesses, and laminitis, as this group of horses is susceptible to environmental, weather, and pasture growth conditions and neglect.
   - *Morgan horses, ponies (of all breeds), older broodmares and stallions, heavily campaigned show horses:* high incidence of laminitis, which is perhaps related to body type (tendency toward obesity), lack of physical activity, and drug administration in show horses.
   - *Draft horses:* heel bulb damage from overreaching, hoof wall loss and cracks, canker, thrush, and lateral hind foot wall flares and wall separations
3. When was the horse last shod or trimmed? Foot problems (acute lameness) that follow shoeing within a few days may imply that a poorly placed nail or nails exist, that the nails were overclinched, or that the feet were excessively trimmed. It is also important to ascertain whether the farrier discovered any problems or has had to deal with persistent shoeing problems. This might include horses that are difficult to shoe because of behavioral problems, thin walls, and poor quality feet, continual evidence of bruising or thrush, and so on. Irregular trimming and shoeing patterns may well prove to be either a cause or contributor to the problems at hand.
4. In what kind of environment does the horse live, train, and compete, and to what kind of management scheme is the horse subjected? In the authors' experience, the environment plays a major role in the condition of the hoof capsule and the genesis of foot damage. The time of the year and the surface conditions can dramatically change the feet and the condition of the hoof walls, frog, and sole. For instance, a horse training on a stone dust track may show very rapid wear of the shoes, whereas the same horse

and shoes training on a deep, soft sand track may show very little evidence of shoe wear in 6 to 8 weeks. Foot bruising, pedal osteitis, and P3 fractures are more likely on hard, "fast" surfaces than on grass or deeper surfaces. Horses that are subjected to wet grass in the morning and hot dry conditions later in the day often develop weak hoof wall material, separated walls, and white line disease and are more prone to losing shoes. Knowledge of the environment and management can influence the treatment and prognosis. Some foot problems are more likely to decline or improve simply by changing the surface on which the horse has to live, train, or compete.

5. How has the horse been shod in the past (i.e., with what type of shoe and at what intervals)? A change in shoe types may be the problem as well as the solution. The use of traction devices on the shoes (e.g., toe grabs, block heels, screw-in calks, borium) can act as impact concentrators and can be, in the authors' experience, the cause of foot problems, especially foot bruising. The examiner must have an appreciation of shoe types and the nomenclature used by the farrier profession.
6. Information about the previous history of foot problems and other lameness problems is essential. The horse being examined may have previously experienced episodes of foot abscessation or laminitis. In the authors' experience, the likelihood of recurrence is high. Farms and ranches with a consistent pattern of foot problems realistically should be examined from a total management perspective (e.g., pasture, housing and environmental exposure, farrier regimen and expertise).
7. What previous treatments have been used for an ongoing problem? If the examiner is examining the same ongoing problem but for the first time, obtaining reasonable detail about the previous treatments will help prevent the use of a similar, but previously unsuccessful, treatments.
8. Discussing the horse and its problems with the horse's farrier can be rewarding, but it is very important to show common sense and discretion. For example, if it is believed that the problem is the fault of the farrier, it should be discussed with the farrier rather than with the client. The farrier may know or suspect what the problem is and will feel more ownership in solving the problem as opposed to being perceived as being the problem.

EQUIPMENT

Specific equipment is necessary to completely examine the foot. Some equipment requires expertise and experience to be used properly, or at least to prevent being used improperly. The following is a suggested list (Figure 8-1):

1. Hoof testers
2. Shoe pullers
3. Single nail ("crease") pullers
4. Hoof knives and sharpening tools (a dull knife can be useless as well as dangerous)
5. Rasp
6. Clinch cutters and a clinching tool
7. Hoof nippers (standard as well as half-round nippers, which are very useful for removing large sections of abnormal hoof wall material)

FIGURE 8-1 A rolling set-up for farrier equipment is useful in a hospital setting. The wheels allow the user to quickly move the equipment away from the horse in the event of a problem.

8. Shoeing apron
9. Shoeing hammer
10. Flexible probes (for exploring abscesses, tracts, and defects)
11. Portable motorized tools (variable speed)
12. Wire foot brushes (which significantly improve the ability to thoroughly examine the weight-bearing structures as well as enhance radiographic quality)
13. Hoof gauge (hoof protractor)

Most of this equipment has obvious application and requires very little explanation or discussion; however, others require careful selection by the individual examiner.

It is prudent to use a shoeing apron. A shoeing apron provides protection to the examiner and also helps to grip the limb being examined. The protection factor cannot be overemphasized, whether one is examining a young foal, a racehorse, or a large draft horse. It is also a means of keeping reasonably clean and professional appearing while working with dirty feet and legs (Figure 8-2).

Hoof testers come in a variety of shapes and designs. They are an absolutely necessary piece of equipment and should therefore be selected for the individual examiner and his or her practice. Hoof testers are more useful if they are light and small enough to be operated comfortably with one or both hands as well as strong enough to endure the years and circumstances of equine practice. The most commonly encountered failings of hoof testers are that they are too large to accommodate a small foot; they are too small and of insufficient strength to handle the large foot; they are poorly hinged (too loose, too tight, or bent); and there is insufficient grip at the contact points, allowing for slippage. It is also

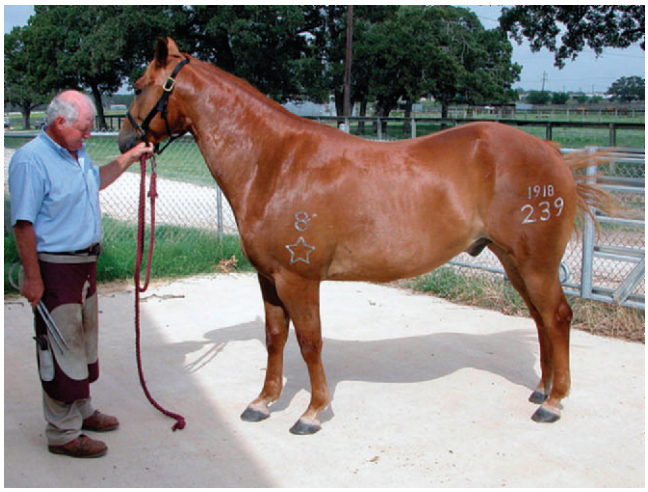

FIGURE 8-2 A shoeing apron provides protection and helps grip the lower limb. Combinations of leather and synthetic fabrics are lightweight and strong.

FIGURE 8-3 Hoof testers are essential and can be used in a variety of ways. In this instance, they are used to detect the presence or absence of focal pain.

worthwhile to have varying sizes to accommodate all variation in foot size, particularly if the practitioner works with a variety of horse sizes (Figure 8-3).

Hoof knives are also best individually selected by the examiner. This author prefers a handmade, looped, double-bladed type for most work (Figure 8-4).

It is imperative to have and carry spare knives (as they will break, wear out, get lost), as well as a sharpening stone or "stick" that is designed to also sharpen the curl at the end of the knife. Time should be taken to learn from a farrier how to properly put and keep an edge on hoof knives. Less conventional tools exist for all types of specific jobs, such as grooving the hoof wall or exploring abscesses. They are helpful and practical if the examiner does a great deal of foot work.

Flexible probes are cheap and very useful. The author prefers the plastic applicators used for culturing tissues. The flexibility is a safety factor not shared by rigid or breakable probes.

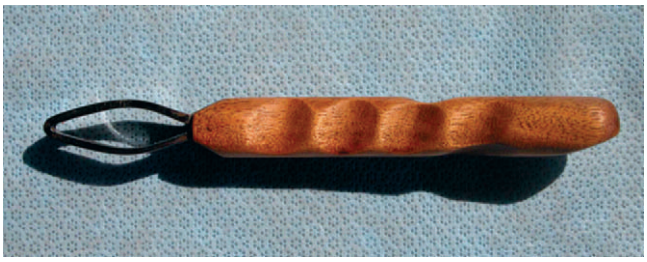

FIGURE 8-4 A double-bladed, looped hoof knife.

Shoe pullers are a necessity. They are used to carefully elevate and remove the shoe. The procedure should consist of straightening and rasping or cutting the nail clinches and then elevating the shoe. This will help avoid the unfortunate event of removing the hoof wall with the shoe. The individual nail puller (crease puller) is, by its design, able to elevate an individual nail head (after the clinch has been rasped and cut) so it can be removed without loosening the rest of the shoe and nails. This has very frequent application when dealing with a single nail problem or removing shoes from shelly-footed horses to prevent hoof wall breakage, which may occur using standard shoe pullers. It is a necessity when dealing with racehorses, as one often can help individual horses by removing a single nail (usually the medial heel nail) without having to have the shoe reset. It is also useful in the case of a horse that has a particularly painful foot problem because the shoe can be removed with considerably less pain.

Portable motorized tools are made by several manufacturers. They are primarily useful for removal of hoof wall material. The ideal, in the author's opinion, is a tool that is capable of at least 25,000 rpm, is small enough to be operated with one hand, is reasonably quiet, and can be fitted with several sizes of cutting burrs, drum sanders, and drill bits. The authors prefer equipment that features variable speeds. Examiners who do a great deal of foot work are well advised to have a "back up" set of tools because they will wear out, break, and malfunction. Those units with rechargeable batteries are, in the authors' opinion, currently incapable of sufficient speed and torque, and the batteries are often discharged when most inconvenient.

Hoof nippers are available from a wide variety of manufacturers and can be purchased in different sizes (lengths) and nipper head shapes (straight and half-round). It is likely that most practitioners prefer the standard 12- to 14-inch nipper. The half-round nipper provides the clinician with the ability to remove reasonably large and specific sections of the hoof wall and thus reduces the amount work compared with a hoof knife or rasp.

A shoeing hammer is useful diagnostically and occasionally helpful to reset a shoe or replace a loose nail. Focal percussion of the hoof wall, sole, and frog help in locating focal pain. Hammers can also facilitate testing of the foot. For example, the hammer head can also be used as a foot wedge (laid on its side with the foot bearing weight at the toe, heel, or individual quarter), or the handle can be used to place focal pressure on the middle of the frog with the horse bearing weight on it (the handle bottom is placed in the direction the horse is facing, the foot being examined is placed on the end of the handle so that the frog is bearing weight while the

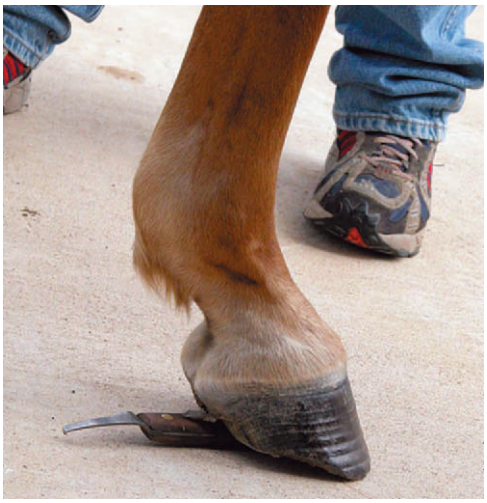

FIGURE 8-5 Demonstration of the use of a hoof knife handle to place focal pressure on the frog. The opposite limb is held up for 30 seconds.

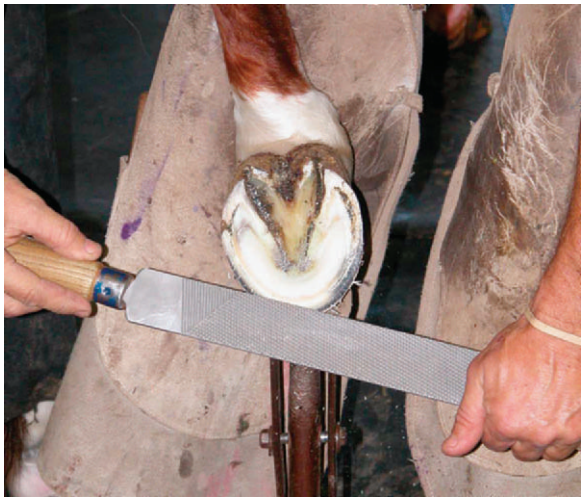

FIGURE 8-6 A shoeing rasp can be used for a variety of tasks; in this case, to better expose the bearing surface of a hindfoot. The picture also demonstrates the use of a hoof stand.

opposite limb is held up). A hoof knife handle can be used in the same manner (Figure 8-5).

The hoof gauge (protractor), in the authors' experience, has limited value unless it is used consistently by the same operator and thus can potentially provide repeatable information. It is very useful to point out to an owner or trainer obvious discrepancies in hoof angle. Its true value lies with the farrier. It has much greater use in harness racing than in other sports.

A shoeing rasp is a necessary tool to help remove clinches and thus shoes, improve visualization of the white line, and help remove excessive or separated hoof wall material, which if left in place is likely to break off or act as a trap for foreign material (Figure 8-6).

EXAMINATION PROCEDURE

The feet are best examined initially at a distance of approximately 20 feet on a flat surface. This allows for comparison of all four feet. One is assessing the size, shape, toe and heel length, approximate angles (toe, heels, quarters), and the position of each foot relative to each limb (limb conformation) and to each other. Subtle differences in foot shape and angle are best appreciated at a distance. Overall conformation should also be assessed, as it clearly affects foot shape and wear.

Examination in the Weight-Bearing Position

Each foot is examined with the leg in the weight-bearing position. The following areas should be palpated and carefully examined:

1. Palpation of the palmar digital vein, artery, and nerve bundle (Figure 8-7). The examiner should check for neurectomy scars (clipping the hair and/or wetting the skin may assist in their detection). The arterial pulse is lightly palpated to determine its character and to compare it with the pulse in other limbs if in doubt as to whether it is normal or abnormal. In many foot-related abnormalities, such as abscess, laminitis, or bruising, the character of the pulse in the affected foot is stronger than in the other feet, especially after exercise. A very useful test, especially when examining horses without clear evidence of where on the limb or limbs the lameness (or performance-related problem) is originating, is to subjectively evaluate the digital pulse intensity before exercise, followed by an immediate (as the horse pulls up) evaluation. It has been the authors' experience that pulse intensity will increase (often for a brief period of time) after exercise. Lack of such a finding does not preclude the presence of a foot problem, however.
2. Examination of the heel bulb area. This is a common site of various dermatopathic damage from trauma, as a point of exit for underlying focal and general infections. The position of one heel bulb to its mate provides information regarding foot conformation and foot balance (Figure 8-8).

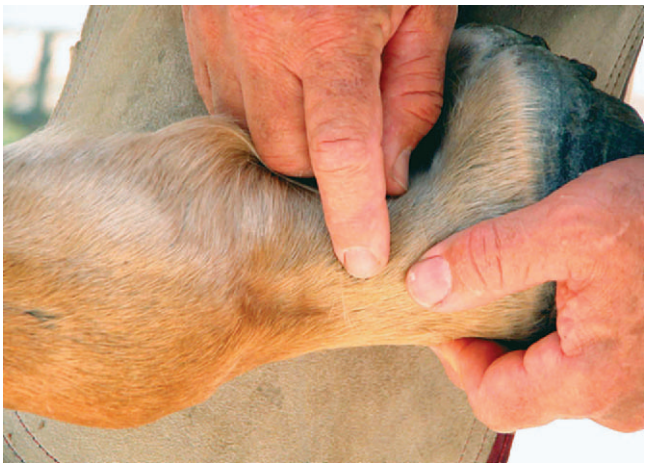

FIGURE 8-7 The digital vein, artery, and nerve complex are usually readily palpable. The examiner should check for scars and focal swelling and evaluate pulse pressure.

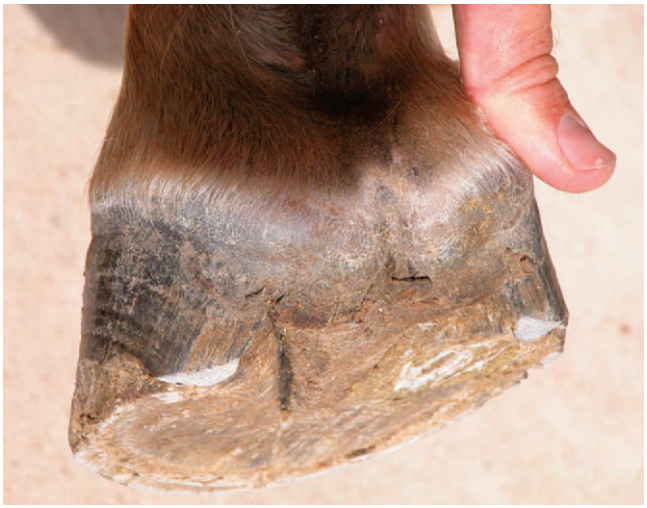

FIGURE 8-8 Examination of the horse in the non–weight-bearing position. The examiner is comparing the height and mass of the medial and lateral heel bulb region.

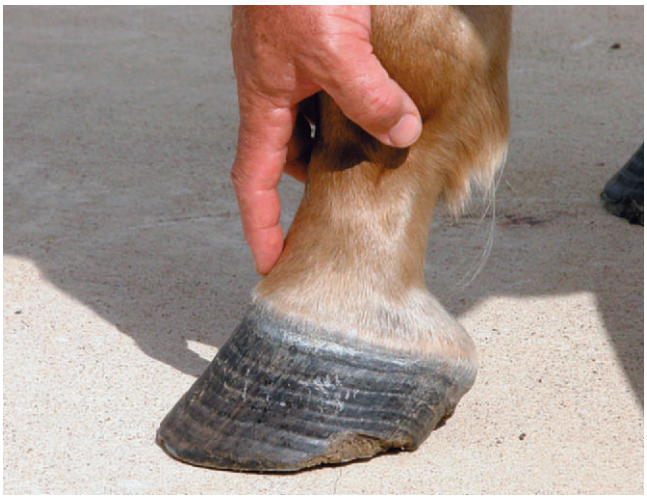

FIGURE 8-10 Palpation of the coronet and the underlying coronary cushion for the absence or presence of effusion of the distal interphalangeal joint. The position for palpation of the dorsal margins of the upward outpouching of the distal interphalangeal joint is depicted.

FIGURE 8-9 Palpation and digitally manipulation of the lateral and medial collateral cartilages.

3. Palpation of the deep digital flexor tendon and the digital sheath at the level of the pastern and continued palpation as the structures disappear into the heel bulb region. With the continued introduction and refinement of imaging modalities, the importance of these structures as they relate to the diagnosis of foot-related lameness will likely increase.
4. Palpation and manipulation of the collateral cartilages (medial and lateral). One should be able to define the palmar proximal edges of these cartilages. The structures should be assessed for their degree of pliability and the presence or absence of pain, swelling, or drainage (Figure 8-9).
5. Viewing and palpation of the coronet from the medial and lateral heel bulbs to the central toe region. On the normal foot, the coronet should sweep evenly toward the heel bulbs and one should appreciate a spongy feel at the margin of the hoof wall (the underlying coronary cushion). Any deviation from this normal sweep or contour (e.g., proximal displacement) or feel (e.g., swelling, discharge, focal pain or heat, absence of tissue, sinking or cleft formation) should be examined more closely and noted as being abnormal. The approximate location of the dorsal aspect of the coffin joint is just proximal to the palpable coronary cushion and thus should be palpated to appreciate the presence or absence of joint effusion (Figure 8-10). This is best appreciated by applying pressure in the area of the joint along the edge of the digital extensor tendon.[6]
6. Palpation and careful examination of the entire hoof wall for the presence of fissures, cracks, bulges, growth abnormalities, local heat, wall loss or breakage, or any similar defects. Often quarter and heel cracks begin as very fine fissure defects at the coronet that extend less than 1 inch distally. These fissures can be easily missed but may be a cause of foot pain and lameness. Further information can be attained on white-footed horses by applying water to the walls because water makes the horn material more translucent (in some instances, evidence of discoloration, usually hemorrhage, can be detected in underlying laminar tissues). The quality of the horn and the presence of variation away from the normal parallel horn tubular arrangement should be noted.
7. Examination of the exit position, exit hole, and clinch of all shoeing nails if the horse is shod. The higher the exit point is, the more likely it is that the individual nail is in or next to sensitive tissue. Overclinched nails and loose nails are worth noting.
8. Gentle tapping of the hoof wall with a closed pair of hoof testers or a shoeing hammer may detect evidence of pain or wall separation.
9. **Reminder:** Hindfeet deserve the same degree of consistent evaluation as do the front feet.

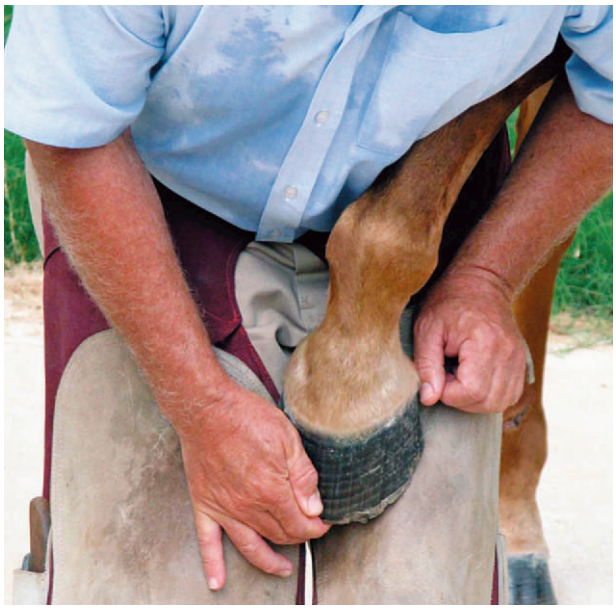

FIGURE 8-11 The foot-forward approach to facilitate visualization and palpation.

FIGURE 8-12 Assessment of strength, depth, and presence or absence of pain and other digitally determined parameters.

FIGURE 8-13 Viewing the bearing surface of the foot to appreciate the relative position and size of the components (heel, quarters, toe, and frog).

Examination with the Foot in the Non–Weight-Bearing Position

Each foot is then examined in the non–weight-bearing position. Again, it is good practice to wear a shoeing apron to protect oneself and to facilitate the examination. Examiners should learn how to assume the stance and posture that an experienced farrier uses, as this will provide greater comfort for both the examiner and the horse as well as place the examiner in a better mechanical position (Figure 8-11).

The suggested procedure is as follows with the unshod foot:

1. The examiner begins by cleaning the bottom of the hoof using the dull side of a hoof knife, steel brush, hoof pick, or the handle of the hoof testers. Greater visualization is often acquired with a wire brush and is highly recommended. The examiner should lightly pare away any debris that obscures an accurate visualization of the frog, sulci of the frog, sole, and white line if the horse is unshod. A hoof knife should *not* be used as an exploratory instrument without an accurate appreciation of sole depth, which can be estimated with digital pressure and hoof testers. The following structures should be examined:
   (a) The frog (size, shape, consistency, position relative to the foot [e.g., centered, directed medially or laterally], whether it is securely attached to the underlying tissue) and its sulci (medial, lateral, and central). The depth of the central sulci can be determined and explored atraumatically with the dull side of a hoof knife blade or a wooden tongue depressor.
   (b) Medial and lateral bars of the foot, which usually require light paring or brushing to appreciate problems such as bar cracks. Some horses have had the bars pared out. This has become a standard practice for some farriers and may represent a problem with regard to total support of the sole and deeper structures.
   (c) The entire sole of the foot, for fissures, punctures, consistency, discoloration (bruising), and degree of concavity. The consistency (relative degree of stiffness) is easily determined with digital pressure, as well as with hoof testers (Figure 8-12). Focal or general areas of sensitivity are usually significant.
   (d) The white line, to determine its width and character. The white line is usually wider at the toe and gradually tapers to a thinner structure as it approaches the heels. It is best visualized either with a wire brush, light paring with the hoof knife, or light rasping of the area (the key word being *light*).
   (e) The bearing surface of the foot, to determine symmetry or lack thereof (Figure 8-13).
2. The bulbs of the heels are examined to determine their relative position (height) to one another. The strength of the caudal foot is assessed manually by attempting to distract the two bulbs from one another in a vertical direction (Figure 8-14), as well as determining caudal foot mass.[7]

FIGURE 8-14 Digital assessment of the strength and mass of the heel bulbs.

FIGURE 8-16 Application of hoof testers. Note positioning of the arms of the testers at midfrog and mid–heel wall.

The examiner should digitally explore the heel bulbs for the presence of swelling, heat, pain, or separation at the coronet (Figure 8-15).

3. The examiner should lightly support the leg at the cannon bone and allow the foot to drop naturally, positioning the line of vision so as to appreciate foot balance and levelness of the walls (see Figure 8-8). Admittedly, determination of foot balance is difficult, as the so-called ideal foot balance has not yet been defined.
4. The entire bottom of the foot should be examined to determine the relative proportion that the divisions of the foot (toe, quarters, heels, and frog) occupy. Imagining a line drawn through the center of the axial skeleton of the limb, transecting the bottom of the foot will help in determining the relative proportion of the medial and lateral foot to this imaginary line. For example, a given foot may demonstrate a unilateral medial heel contraction in combination with a flared lateral quarter (see Figure 8-13).
5. The foot is next examined with hoof testers. It is prudent to always begin hoof tester application with light pressure to make a subjective evaluation regarding the individual horse's response. Some horses are more sensitive to manipulation than others, regardless of the presence or absence of pain, whereas others simply have thin walls and soles, in which case a response is likely. Abnormal responses should be compared to responses in the other feet and the tests should be repeated to be sure the responses are consistent. A suggested sequence is as follows:
   (a) The examination begins with the medial bar to the medial heel wall.
   (b) The testers are placed on the medial sole to medial heel wall and continue at approximately 1-inch intervals to the lateral wall and lateral bar. The examiner should be sure to include each exit point of the shoeing nails. At each interval, the examiner should look carefully at the junction of the wall and sole (white line) as pressure is applied, to detect separation, presence of fluid or

A B

FIGURE 8-15 **A,** Palpation of the deep digital flexor tendon to appreciate the presence or absence of flexor tendon sheath effusion, width of tendon, and pain and sensitivity. **B,** Application of digital pressure to assess presence or absence of pain in the deep digital flexor tendon and underlying distal sesamoidean ligaments.

exudates, or significant movement of the sole, suggesting underlying cavitation. One is not just attempting to determine the presence or absence of pain but also is characterizing the strength and character of the hoof capsule.

(c) The testers are then placed at the medial quarter wall, midway between the bearing surface of the foot and the coronet, to the lateral middle aspect of the frog (Figure 8-16). This procedure is repeated on the opposite side of the foot. The testers are placed in the middle bearing surface of the frog to midway between the coronet and the bearing surface at the toe. Finally, the testers are placed across the medial and lateral quarters in an attempt to isolate the length of the navicular bone. The examiner should keep in mind that hoof testers are essential but not foolproof. Hoof testers to do not apply pressure to the foot in the same direction and manner that the foot experiences with contact with the ground, and the depth and strength of wall and sole material have a protective influence. It is not unusual to ultimately discover a significant foot problem (e.g., third phalanx fracture, laminitis, navicular disease) in the absence of a meaningful response to hoof testers. In the event of a "positive" hoof tester finding (evidence of pain via reaction), it is prudent to complete the examination and retest the area of discovery, to be sure that the positive finding is repeatable.

(d) Finally, the examiner should gently tap the structures on the bearing surface of the sole and frog with the rounded end of either closed hoof testers or a shoeing hammer (see Figure 8-3).

6. The examiner should repeat the palpation of the collateral cartilages and the coronet. Palpation of the extensor process of the third phalanx region and the associated coffin joint can be facilitated by bringing the leg forward and flexing the toe caudally.
7. Palmar flexion of the lower limb is performed with the fetlock in somewhat of a fixed position (Figure 8-17). The examiner is manipulating both the pastern and coffin joints to determine range of motion and presence or absence of a painful reaction. The foot should be rotated (twisted) medially and laterally around the vertical axis of the pastern. This manipulative test, as with any joint manipulation, is quite subjective and is also subject to much interexaminer variation and individual horse response. In the event of a positive reaction, it is prudent to repeat this portion of the examination after the horse has been exercised at a jogging pace to determine if this either creates or enhances lameness.

The procedure and technique is the same if the horse is shod, but the following is added:

1. The examination begins by tapping of the shoe with closed hoof testers or a shoeing hammer at 1-inch intervals, as this greatly facilitates cleaning the bottom of the foot and also alerts the examiner to the presence of pain (often associated with a poorly placed nail or nails) or a loose shoe.
2. Note should be made of the shoe type and the presence or absence of additions such as toe grabs, block heels, or trailers.
3. The examiner should determine the security of the shoe to the foot.

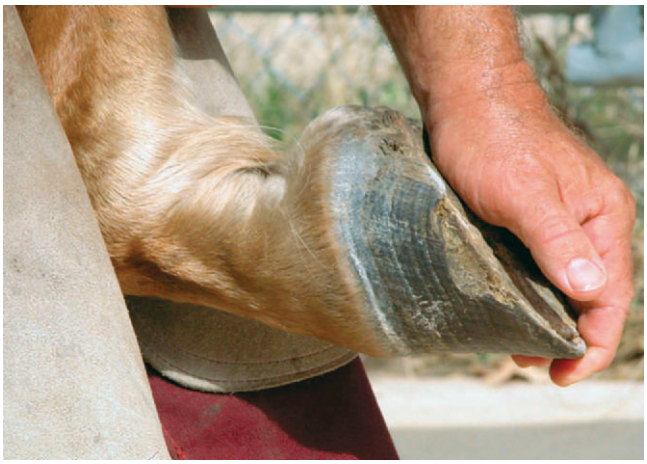

FIGURE 8-17 Palmar flexion of the distal joints. Total isolation of the interphalangeal joints is not possible with this manipulation.

4. The examiner should carefully examine the shoe for abnormal wear.
5. Finally, the examiner positions the hoof testers and uses the same procedure described for the unshod foot. The presence of full or partial pads may influence the findings.

Caution: It is best not to remove the shoes, or shoes and pads, until the horse has been examined in motion or subjected to diagnostic local anesthesia demonstrating convincing evidence of the presence of foot pain or problems. Removed shoes are not only costly to the client but often are protective for weak-footed horses. It is not unusual for horses both with or without foot problems to experience tenderness or pain because they may not be used to being without shoes. If a horse is to go without shoes for a period of time, it is useful and thoughtful to protect the foot with a few layers of duct tape, perhaps with padding, and to suggest that the horse remain confined until shod. The examiner should carefully record all findings, as it is easy to forget subtle discoveries that may ultimately determine how the horse is going to be treated or shod.

Examination with the Horse in Motion

This aspect of the examination of the foot is best accomplished with the horse being led, ridden or driven, or lunged on a flat, hard surface, if available. The question of what surface or surfaces are optimal (in a hospital or clinical setting) is best answered by considering the sport type that a given practice is most likely to work on. For example, the authors find the assessment of particularly subtle lameness or performance problems in Standardbred race horses is best determined at a suitable training or race track. Ideally for most horses (e.g., pleasure, hunter/jumper, Western performance) a well-drained asphalt or concrete surface that is of sufficient size to safely jog or lunge a horse works nicely. These cases, particularly the very subtle problems, are best served by viewing the horse under the circumstances in which they train and compete. This may include a training/race track, an arena suited for dressage and jumping, and a rodeo arena to watch roping events and barrel racing. Realistically, most lame horses are viewed where they live,

which at times can be less than ideal. The authors also prefer, when possible, to have the horse ridden if the lameness is subtle or intermittent simply because, assuming a reasonably experienced rider or driver, the individual on the horse can relate changes in motion after manipulative tests and diagnostic anesthesia. However, this is surely not recommended if one is dealing with a patient demonstrating significant lameness or if there is the possibility of further injury. The authors routinely use flexion tests, hoof testers, and other means of enhancing the information base with a mounted rider. Initially a horse under these circumstances is walked away from and back toward the visual plane.

The examination of the horse in motion begins by having the horse walked away from and back toward the examiner's visual plane. The visualization is enhanced if the examiner lowers his or her view by crouching. The foot strike for each foot should be examined and the examiner should try to determine if the foot (and thus feet) lands flat, heel or toe first, and medial or lateral hoof first. The landing position of the individual foot relative to the vertical axis of the respective limb should be appreciated (i.e., does it land under or medial or lateral to this axis). The examiner should evaluate the flight path the individual foot takes from foot break over to foot strike. Examination with the horse walking should always be included because it is the one gait that is sufficiently slow to detect fine movement and foot landing abnormalities. The same process should be repeated with the examiner viewing the horse from the left and right sides.

The horse is then trotted (or paced) and visualized in the same manner as at the walk. Circling the horse at a trot or pace will often intensify foot lameness (this is true of most types of lameness regardless of the source of pain), but the examiner should not assume that the painful foot (or limb) will always show greater lameness when it is on the inside of the circle. For example, if a horse is being lunged or ridden in a circle with the right front limb on the outside and is suffering from a medial heel or quarter crack on the right front, it may be more lame with the limb on the outside of the circle. In most instances, but not all, horses with high suspensory desmitis show increased lameness with the involved limb on the outside of a circle. The point here is that preconceived notions about the circles, limbs, and lameness can be wrong.

The examiner should also be aware that lameness, regardless of the source, will not always be attended with a discernible head nod (head "bob"), a good example being a horse with bilateral problems. Clues that a horse may have a sore foot that does not result in obvious lameness can be decreased performance or an inability to perform a particular task (e.g., occasional or consistent refusals to jump, poor performance on a hard race track, a loss of fluid motion in a dressage test, inability to "stop" properly). Palpation and appreciation of the pulse pressure of the digital arteries can be useful immediately after exercise. A horse that is clinically free from problems will experience no change in pulse pressures after exercise, whereas those with painful and inflammatory lesions often will. This feature, when present, can be very helpful in attempting to determine whether a horse with a subtle performance problem is experiencing foot-related disorders. Further examination of the horse in motion depends on the clinical signs seen, often requires a rider or driver, and may require that the horse work for a period of time or undertake specific exercises (e.g., side passes, sliding stops, jump safe obstacles) or with changes in terrain (e.g., up and down inclines, variation in ground quality and type of surface).

Localization of the problem at this point in the examination may be obvious, and very little further workup may be required. Some cases, however, can require more extensive examination to get an accurate appreciation of the location of the problem (or even to identify the affected limb). In those situations, localization of the problem is usually attained by either increasing the clinical signs (making the problem or lameness more apparent by performing specific tasks or examiner manipulations) or decreasing the clinical signs with local anesthesia. The method or methods of choice depend on the individual examiner, the problem at hand, the individual horse, and the wishes of the owner or trainer. There is no single technique that will always provide accurate results, including local anesthesia.

Further Examination

Each method or technique for further workup has advantages and disadvantages. Some are easier to accomplish than others and some depend on the horse and its environment as well as the wishes of the owner or trainer. The following additional examination can be performed:

1. Lower limb sustained flexion or rotation, or both, of the foot may help isolate joint pain. This is easily accomplished and often quite helpful. The authors generally hold the flexion for approximately 1 minute (unless significant pain exists) and the rotation for 15 to 20 seconds (as separate events). In the event of a positive reaction (increased lameness), it is prudent to perform the same manipulation on the opposing limb as well as repeating the manipulation to ensure consistency of reaction. One cannot isolate a given joint (coffin, pastern, or fetlock) with these manipulations. Positive reaction, particularly when dealing with "experienced" equine athletes, are not unusual. A marked response and increase in lameness, however, is a notable finding. **Caution:** If a horse is showing significant pain and reaction to the manipulations, the examiner should simply let the foot down and allow the horse to walk off rather than trot, to lessen the likelihood of an accident or a horrified owner or trainer.
2. Sustained or intermittent pressure applied to various parts of the foot with hoof testers followed by jogging is a very useful addition to the examination. The application consists of intermittent pressure for 15 to 20 seconds, assuming the horse tolerates it. If the horse does not tolerate it, that may also provide a clue to the origin of the lameness.
3. Having the horse ridden (or driven) at various gaits, patterns, and exercises may help identify the involved limb or limbs.
4. Moving the horse in hand or under tack over varying terrain (e.g., hard, soft, inclines), if possible, may help to localize and characterize the problem.
5. Foot wedges or items placed under the foot to put pressure on selected parts, such as the frog, are used by some examiners. Their use requires experience to provide an accurate assessment of the results, as well as requiring a manageable horse.
6. Local anesthesia is the most useful entity in identifying the approximate location of the source of pain.

LOCAL DIAGNOSTIC ANESTHESIA

Local anesthesia remains the most useful clinical tool in establishing the source of pain. It has known disadvantages, however, such as the following:

1. Local anesthesia will only improve lameness that results from pain; that is, it will not improve lameness that results from mechanical problems.
2. Normal variation of the location of nerves and branches can exist and thus confuse results and interpretation.
3. Injectable materials do not necessarily remain where they are deposited; that is, with time (after injection) and motion, their distribution could vary.
4. It is not clear whether adjoining synovial structures communicate with one another and how often. For example, does an injection into the coffin joint, digital sheath, or navicular bursa remain in the intended synovial structure on all horses? More recent information indicates that injection of a local anesthetic into the coffin joint, for instance, can desensitize the navicular region and subsolar pain and thus is not specific to the coffin joint. Recent work by Schumacher et al.[5] indicates that local anesthetics injected in the area of the palmar digital nerve, at the midpastern level, can negate pain in the pastern joint as well. Recent work also indicates that the timing (time of injection to time of improvement) in coffin joint anesthesia has no predictable relevance with regard to determining the source of pain.[8] In summary, there is considerable overlap among the commonly used techniques and thus an examiner can only guess as to the actual source of pain when dealing with foot problems requiring diagnostic anesthesia.
5. A recent paper[9] indicates that complete anesthesia requires at least 15 minutes, so it is important that time be allowed for such anesthesia to take effect.
6. The authors suspect that some horses who have experienced chronic pain and lameness (for months or years) may well have adopted a habitual way of moving even though the pain is negated with local anesthesia.

The examiner of a horse with a sore foot cannot be absolutely sure which or what portion of the structures have been successfully anesthetized—only the general region where the problem exists.

The sequence of diagnostic anesthetic injections preferred by the authors is as follows: medial and lateral palmar digital nerves blocks (below the level of the collateral cartilages to prevent possible pastern joint anesthesia), either one at a time or at the same time, followed by the abaxial sesamoid block. This will, in the authors' opinion, desensitize all conceivable painful foot problems if the injections are accurately placed and sufficient time is taken to allow the local anesthetics to work.

In the event of and following the use of imaging modalities, one can use the navicular bursal block (though usually not on the same day), because it has a more focal effect with regard to navicular bone and bursal pain. The usefulness of coffin joint anesthesia to differentiate specific sources of pain is of questionable value, based on the most recent research information.[5] If the practitioner chooses to use the navicular bursal block, the authors recommend practicing the technique on cadaver specimens as well as verifying the location of the needle radiographically or with fluoroscopy. The accuracy of a "blind stick" is such that one cannot be sure of where and thus what structures (tendon sheath or coffin joint) have been injected. The authors recommend allowing at least 15 minutes from the time of injection to the time of evaluation, regardless of the technique used. The authors further believe it is useful for a horse that has received an anesthetic injection to be walked for a few minutes before further evaluation is undertaken.

The order of this process and the selected time periods are arbitrary and based solely on experience. It is important to note and thus warn owners and trainers that some horses may be considerably lamer in the days following diagnostic anesthesia procedures. If the examiner suspects that will be the case, he or she may wish to provide antiinflammatory medications as well as the warning. If intrathecal anesthesia is used, the examining clinician should be alert to the possibility of postinjection reactions or infections and thus warn the owner, if the horse is not in the overnight care of the examiner. If multiple diagnostic or therapeutic injections are used, it is wise to consider lower limb bandaging and administration of nonsteroidal antiinflammatory medications.

THERAPY AS A DIAGNOSTIC TOOL

The presence or absence of response to chosen therapeutic regimens is, with careful evaluation, a diagnostic tool. For instance, in dealing with a lame horse that, in spite of the examiner's best efforts, has an inconclusive diagnosis, one can attempt a specific therapy and then gauge its success or failure. An example would be the use of intraarticular corticosteroids in the coffin joint or navicular bursa (done with owner consent). Assuming the horse improves or is sound, the examiner can then conclude that one of these two structures is likely involved without positive evidence via imaging. An additional example is the use of pads on horse with suspected, but unproven, subsolar bruising ("sore footed" horses). The use of more generalized, or so-called shotgun, approaches produces findings that are difficult to interpret.

INTERPRETATION OF FINDINGS

It is critical to use a record system that is not only retrievable but also, more importantly, organized so that it is useful months or years later when examining the same horse. Lameness and performance problems have a way of getting complicated, especially over a period of time, as most equine patients are athletes and acquire problems in training, in competing, and often with age. A readable and consistent record system is more than useful in attempting to solve problems through a given horse's career. The authors prefer a medical record that denotes the limbs (LF, RF, LH, RH) and the major anatomical sites (foot, pastern, fetlock, etc.). Furthermore, it is useful, if not critical, in the event of litigation, to be in the habit of using consistent language (including abbreviations) to describe findings and actions.

Lameness

A system of grading the degree of lameness was initially described by the American Association of Equine Practitioners and has become a standard in the literature. This system assigns a grade of 0 to 5, with 0 meaning no lameness and 5 denoting a state of non–weight bearing. The same gradations can be used to denote degrees of severity (e.g., "2/5 pain with hoof testers"). The following is an example of recording information about lame horses in a medical record:

LF foot—excessively flat sole, low/underslung heel, 2/5 pain with testers @ medial quarter/heel. 2/5 lame in straight line; 3/5 lame circling to the left; 3/5 with testers. Sound with PDN; and RADS—NSF. Treatment—¾ × ¼-inch flat Al shoes, concaved—solar surface, Bute 1 gram BID for 5 days, back to work on the 6th day following shoeing. Owner told to call on 7th or 8th day.

This notation in the record means that the horse had excessively flat soles and an underslung heel, showed moderate pain on the inside heel region, was moderately lame, and showed increased lameness with circling and focal hoof tester pressure. The horse was sound after a bilateral heel nerve block, radiographic examination was performed, and "no significant findings" were noted. The horse was shod with wide-web aluminum shoes that were deeply concaved on take weight bearing off the flat sole. The horse was given phenylbutazone (Bute) twice a day for 5 days and was to be put back into work on the sixth day, and the owner/trainer was instructed to provide a progress report. This information should be useful months or years later, if the horse returns to its original veterinarian or moves and a new veterinarian calls the original to find out what was discovered in the examination and what treatment was performed.

The most challenging aspect of foot and lameness work is the interpretation of findings. A portion of the cases are very straightforward and present little or no diagnostic challenge. Subtle, intermittent, and performance-related problems can be very challenging, however. Particularly frustrating are horses whose clinical signs have changed or disappeared since the owner or trainer first observed the problem; in such cases, it may be useful to have the horse worked before examination.

Hoof Tester

Interpretation of hoof tester findings is reasonably objective, but this examination can also provide confusing or conflicting information. Hoof testers do not necessarily direct pressure in the same direction as actual foot fall; each examiner applies pressure to greater or lesser extent, and for more or less time, than his or her colleagues; thin-soled and thin-walled feet are more likely to react to pressure than feet with a substantial hoof capsule; and some horses simply act as though a positive response has been elicited simply because they do not care to be tested. Interpretation of findings is often enhanced, however, by examining an opposing foot and repeating the manipulation. The use of hoof testers and interpretation of findings is in many ways a learned art form that requires experience and practice. In addition, very useful information about the integrity of the horny capsule can be gained with hoof testers (e.g., the depth and resiliency of the sole and wall, defects and motion at wall separation and hoof crack sites, presence of fluids [pus, blood, or serum], and evidence of underlying separation that may not be readily visible).

Shoe or Bearing Surface Wear

The interpretation of shoe or bearing surface wear can be very useful, but one should be careful to take in all the factors that influence shoe or foot wear. Abnormal wearing of these surfaces depends on the manner in which the foot contacts the ground surface as well as the character of the surface itself. Thus a horse can be landing in a less than ideal manner but that fact may not be evident when the shoes are examined if the horse is exposed to soft, forgiving, nonabrasive surfaces (grass, for instance). One way to avoid missing abnormal shoe wear as a sign of abnormal foot landing is to use a steel brush to clean or semipolish the shoe surface, followed by jogging the horse on asphalt or concrete, followed by re-examination.

Abnormal wear can occur from a less than ideal foot flight pattern resulting from conformational problems, lameness and the avoidance of pain, or improper application of either trimming or shoeing practices. An example of a foot flight pattern change that often affects shoe wear is an active degenerative joint disease of the lower joints of the hock (bone spavin). Afflicted horses tend to carry the affected limb or limbs toward the midline and make initial contact with the ground surface on the outside toe and quarter region of the shoe. This tendency is especially evident in Standardbred racehorses that are training and competing on abrasive surfaces. Interpretation of the findings and use of this portion of the examination is then best made by first examining the opposing foot for wear, an appreciation of the horse's conformation, and the presence or absence of lameness. Abnormal toe wear has often been assigned in the literature to horses experiencing navicular disease, but that has not been a consistent finding in the experience of the authors.

Hoof Wall Shape

Interpretation of abnormal hoof wall shape can be quite useful but, like abnormal wear, can vary with more than one factor (e.g., conformation, trimming and shoeing practices, environment, nutrition, and the presence or absence of problems within the hoof capsule). Generalized distortions can be the result of foot conformation, foot imbalance problems, generalized white line disease, or laminitis. Focal distortions of the hoof wall can occur as a result of a space-occupying lesion such as a keratoma or foreign body or underlying separation of the hoof wall (white line disease, laminitis, submural infections, or mechanical structural damage).

Hoof Balance

The evaluation and interpretation of foot balance (perhaps better thought of in terms of what can be most easily manipulated or changed as "hoof balance") is, in the authors' opinion, a difficult and subjective matter. Controversy continues to exist as to what constitutes normal or ideal balance. At present, hoof balance is described in two broad categories: morphologic hoof balance and dynamic hoof balance. It is logical to assume that one influences the other.

Visual examination of the foot is conducted to appreciate the respective proportion of the foot (heels, quarter, and toe)

to the total bearing surface. The ideal most frequently proposed is that of a symmetrical foot that consists of the frog dividing the foot into equal medial and lateral aspects and a level bearing surface when viewed from heel to toe. Absolute symmetry is virtually nonexistent in nature; horses are not symmetrical, nor should their feet be. The relative proportion of the bearing surface of a foot is determined by the position of the tissues responsible for hoof wall growth and their growth direction/shape, as well as the mechanics of the forces influencing both growth and the tissues (hoof wall material can be distorted), and wear.

The examiner must be careful in making a determination of "improper balance" without considering normal and abnormal forces that influence growth, distortion, and wear, lest erroneous interpretations be made. For instance, horses with less than ideal conformation are likely to have asymmetrical bearing surfaces. However, in some horses the weight-bearing surface may be in balance with a crooked or distorted limb, allowing the limb to more efficiently accept the forces of impact for a given conformation and swing pattern. Front to back balance is another aspect of overall foot balance and is generally described in terms of toe length and angle and heel depth and angle. The long toe/low heel foot conformation is undoubtedly a significant contributor to foot and limb disorders.

The basic factors in foot balance can be summarized as follows: toe length, hoof angle, mediolateral hoof orientation, wall contour and ground surface, and symmetry (or asymmetry) of pairs (i.e., left and right limbs).[10]

Diagnostic Local Anesthesia

Evaluation of the response to diagnostic local anesthesia is more objective, particularly if a horse's condition improves or the horse returns to a normal gait after injection, but it also has limitations. The major limitation of diagnostic procedures performed in the vicinity of the foot is the lack of specificity owing to the presence of anatomical variation among horses and the distribution of injected materials. Diagnostic anesthetic techniques used to determine pain within the foot are not anatomically specific. Positive results (improved or normal) should be interpreted by the examiner as indicating that the pain source is in an approximate region rather than specific anatomical parts.

ADDITIONAL EXAMINATION TECHNIQUES

Often physical examination procedures do not establish a firm diagnosis. In such cases, further examination of the horse's foot or feet is necessary to either diagnose or further characterize a given problem. The available techniques vary tremendously in cost, availability, usefulness, and the experience required to perform them. Techniques include synovial fluid analysis, imaging techniques (plain film radiography, contrast radiography, scintigraphy, ultrasonography, computed or digital radiography, thermography, computed tomography, and magnetic resonance imaging), high-speed photography analysis, force plate and instrumented shoe studies, and the use of high-speed treadmills to analyze motion.

Synovial fluid analysis (coffin joint or navicular bursa) may be the only premortem diagnostic test to assess early joint or bursal infections resulting from puncture wounds and similar injuries. Information gained from synovial fluid analysis early in the course of the infection can be life saving. Synovial fluid analysis will likely continue to evolve and provide accurate information with regard to early detection of joint dysfunction.

Ultrasonography is useful to examine soft tissues (deep digital flexor tendon and sheath) as they enter the palmar aspect of the foot. The tissues of the caudal heel can be accessed with ultrasonography through a carefully prepared frog.[11]

Plain film radiography remains the most commonly used imaging system for the equine foot. Discussion continues as to what constitutes a so-called foot series. The following views are considered by most practitioners as standard:

1. Lateral-medial view to assess the dorsal and palmar/plantar surfaces of the phalanges and interphalangeal joints, the hoof wall thickness, the position of the coffin bone within the hoof capsule, and the four surfaces of the navicular bone.
2. Two or possibly three views of the foot (two dorsal 65-degree palmarodistal or plantarodistal oblique views at different radiographic exposures, and possibly a 45-degree oblique view). A 65-degree lighter radiographic exposure is used to examine the solar margin and body of the third phalanx, whereas darker exposures at 65 degrees and possibly at 45 degrees are used to evaluate the navicular bone and the coffin joint. In the authors' experience, the lighter 65-degree exposure is often overlooked and is quite useful.
3. Dorsopalmar/plantar view to examine the medial and lateral surfaces of the third phalanx, the collateral cartilages, the borders and body of the navicular bone, and the interphalangeal joints.
4. Palmaroproximal/palmarodistal oblique view (skyline or flexor surface view) is necessary to isolate the medullary cavity and flexor cortex of the navicular bone and the palmar/plantar (wings) of the third phalanx.

Additional and creative views as variations of these are often very useful and may include oblique and other views intended to isolate a given portion of the bony and soft tissue structures of the foot.[7] More than one exposure may be necessary to capture specific regions; thus it is important to consider the exposure as well as the positioning of the horse, film, and machine to accommodate conformational differences.

Based on the results of initial plain film radiographic examinations, special techniques such as contrast radiography or the placement of radiopaque probes may also be required. Regardless of the techniques being employed, it is imperative to clean the surface of the body part being imaged, and, in the case of the hoof, to remove the shoe whenever possible. Before the radiographs are taken, the foot should be thoroughly cleaned and the frog crevices and sole packed with a radiolucent medium. It is important to note that foot radiographs taken for the purpose of prepurchase examinations should be taken with the shoes removed or a note should be made in the record indicating why they were not removed. Proper radiographic exposure, labeling, and processing are very important. Proper radiation safety practices must be used for all people who are around or are involved with the radiographic examination.

Scintigraphy requires the use of radiopharmaceuticals and a gamma camera to produce its image (often referred to as a

nuclear scan or *bone scan*). Scintigraphy tends to be more sensitive in detecting early bony reaction, but it is less specific than radiography for diagnosing bone abnormalities. The technique is expensive but is becoming increasing available.

Computed radiography and digital radiography are increasingly being used in place of traditional film-type radiography for radiographic examinations. Both computed and digital radiography use filmless image detectors, and both allow for digital manipulation of the image to provide better and more uniform image quality. Digital images can be transmitted over computer networks to distant sites.

Computed tomography (CT) is a form of x-ray imaging that has been used at some academic institutions to produce high-detail planar and three-dimensional reformatted images, in particular for evaluation of bone and joints. So-called interventional imaging (guided injections, implants, and other applications) using computed tomography and ultrasonography is also a very rapidly growing diagnostic and therapeutic tool.

Magnetic resonance imaging (MRI) uses the effects of relatively strong magnetic fields for planar evaluation of the equine foot, and in particular for its soft tissues. It is, however, very expensive, both for the equipment and for the regular maintenance that is required. Technically, MRI requires significant training. However, units are being placed throughout North America. Currently, CT and MRI equipment and examinations using these modalities are expensive, but they will become increasingly available and useful in the future. MRI provides information with regard to lesions in the foot that cannot currently be diagnosed by other means.[4]

Thermography is an imaging technique that measures infrared emissions and produces a graphic visualization of the surface temperature of the object being examined. It appears to have useful application in that it is capable of localizing subtle (as well as obvious) inflammatory lesions in both soft and bony tissues. A thermogram may be capable of detecting pathologic lesions before they are visible on a radiograph. The equipment costs are moderate to high, experience is necessary to both produce quality thermograms and interpret the results, and the conditions of the examination require some control. Handheld infrared thermometers are available and considerably cheaper, but their usefulness is limited.

High-speed cinematography for the purpose of gait analysis has been used for the most part as a research tool. The use of high-speed filming and videotape techniques allows the examiner to appreciate motion that occurs too rapidly to be perceived by the naked eye. The usefulness of this technique in horses with foot and shoeing problems is obvious, but the cost of equipment, film, and analysis is high and thus a factor. A less expensive option, but of a somewhat similar application, is the use of portable video equipment.

Force plate and other equine kinetic technologies offer, for the most part, unique research opportunities as well as clinical application. This includes individually designed horseshoes with strain gauges, floor-mounted force plates, computerized equine gait analysis systems, and angular motion detection equipment. Such systems are useful but expensive and not yet a practical situation for practices other than universities or similar large centers. The future may well produce more portable and affordable equipment.

High-speed treadmills have been used for many years to both condition horses and investigate physiologic parameters in the exercising horse. The use of such equipment to investigate limb motion and lameness problems appears to have limited value. The equipment is expensive, requires a reasonably large space (usually requiring sound proofing and air handling systems), and can be labor intensive, depending on the system employed. It is likely that the amount of information gained from high-speed treadmill technology will increase over the next several years, but this technique is still somewhat questionable with regards to its use in lameness and foot/shoeing research because of the fact that the surface is moving and quite forgiving, that is, a reasonably artificial means of evaluation.

DEVELOPING AN ACCURATE PROGNOSIS

The prognosis for any limb problem is an important consideration. The prognosis in most instances is an educated guess. However, statements made by the examiner often determine the decisions made by the owner or trainer. The prognosis of foot problems requires knowledge of a multitude of factors, some of which differ from those influencing problems elsewhere on the limb:

1. The diagnosis or diagnoses is of paramount importance.
2. Whether or not other limb problems coexist will influence the outcome.
3. The duration of the lameness is a very important aspect. The longer problems exist, generally the more difficult they are to solve.
4. The training and competing environment can be a limited factor. For example, foot bruising, hoof cracks, pedal osteitis, and similar problems are easily and negatively influenced by concussion, which is in turn influenced by the training and competing surfaces. The time of the year can also influence the surfaces.
5. The sport may dictate the types of available shoe and corrections possible. For example, Thoroughbred racing is limited in the variety of shoe designs, materials, and shoe weights that can be used. The Standardbred racehorse, on the other hand, can often race successfully with a number of shoe designs and types.
6. The expertise of the involved farrier may be the sole reason for the success or failure of a given correction. This means that a solid and trusting relationship must exist between the veterinarian and the farrier.
7. The expectations of the owner or trainer are very important. Chronic laminitis, as an example, is obviously a serious problem, but it may be manageable and of limited significance for a broodmare; a sole bruise, on the other hand, is usually a minor problem but takes on great importance if the horse is attempting to compete the next day.

The ability to provide an accurate prognosis is dependent on knowing and understanding as many of these variables as is possible.

SUMMARY

The foot is the most common source of limb pain. Problems vary from simple to very complex. An accurate diagnosis is

dependent on a thorough and detailed knowledge of anatomy, an appreciation of available examination techniques, and a significant dose of common sense.

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9

DIAGNOSTIC ANALGESIA OF THE EQUINE FOOT

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Diagnosis of some foot diseases was, for many years, based in part or totally on a positive response to regional, intraarticular, or intrabursal analgesia of the foot. Because there was little scientific basis for interpretation of these responses, some foot diseases (e.g., navicular disease) were often misdiagnosed. In recent years, clinical observations, anatomical studies, and results of clinical trials have helped to clarify interpretation of the results of regional, intrasynovial, and intrabursal analgesia of the forefoot of horses. This chapter presents a summary of current knowledge of the use of analgesia to localize sites of pain in the foot of lame horses.

CHOICE OF LOCAL ANESTHETIC AGENT

Mepivacaine (Carbocaine) is the local anesthetic agent most commonly used for regional or intrasynovial analgesia of the foot. Lidocaine is also used for regional anesthesia, but because this local agent is irritating to tissues, the authors do not use it for intrasynovial analgesia of the lower portion of the limb. Mepivacaine is less irritating to tissue than lidocaine. The onset of regional anesthesia is slightly quicker when using lidocaine than when using mepivacaine, but with either drug, the foot usually becomes anesthetized within 5 minutes after perineural administration.

The duration of action of lidocaine is short, and recurrence of lameness can be expected 30 to 45 minutes after perineural administration. Recurrence of lameness after perineural or intrasynovial administration of mepivacaine usually occurs within 90 to 120 minutes. In the examination of a horse with lameness caused by pain in multiple limbs, mepivacaine often is more useful than lidocaine. When lidocaine is used to resolve lameness in a horse with multiple limb lameness, lameness that has been resolved in one limb might recur before lameness associated with another limb can be evaluated. When examining the effects on gait of different analgesic techniques on the same region of the limb, using lidocaine rather than mepivacaine for regional anesthesia might shorten the lameness examination, because lidocaine allows lameness to recur more quickly so that other analgesic procedures can be examined.

PREPARATION OF SKIN FOR PERINEURAL OR INTRASYNOVIAL ADMINISTRATION OF LOCAL ANESTHETIC SOLUTION

Opinions vary concerning the amount of skin preparation necessary for analgesia of the lower portion of the limb. Simply cleaning the site of injection with 70% isopropyl alcohol is usually sufficient for perineural administration of local anesthetic solution. When the lower portion of the limb is particularly dirty, however, the region should be scrubbed with antiseptic soap. The consequences of a nonsterile subcutaneous injection are usually minimal, but inadvertent nonsterile injection of a tendon sheath or joint could have disastrous consequences.

After the site is scrubbed for 5 to 7 minutes with an antiseptic soap and rinsed with isopropyl alcohol, intrasynovial analgesia can be performed using sterile gloves, needles, and syringes and mepivacaine drawn from an unused bottle. Some clinicians have a strict policy of clipping or shaving the site of synoviocentesis, but one study showed that skin could be prepared adequately for aseptic arthrocentesis without removing the hair.[1] Some clinicians administer an antimicrobial drug, commonly amikacin (150 to 250 mg), along with the local anesthetic solution.

RESTRAINT FOR ADMINISTRATION OF LOCAL ANESTHETIC SOLUTION

For most horses, regional anesthesia of the lower portion of the limb can be accomplished with minimal restraint, but if the horse is fractious or if the solution is administered intrasynovially, using a lip twitch or lip chain may be necessary. Not all horses respond in the same way to different methods of restraint, but for most horses, application of a lip twitch provides adequate restraint. The twitch works best when applied immediately prior to needle placement. When a joint is injected with the horse's limb on the ground, the contralateral limb can be lifted off the ground to enhance the safety of the procedure for the clinician. Clinicians should be aware, however, that some horses might buckle in the knee of the weight-bearing limb when the needle is introduced and fall on both knees.

If application of a lip twitch or lip chain does not provide sufficient restraint, the horse can be sedated with xylazine (0.2 mg/kg IV) or detomidine (10 μg/kg IV), often without interfering significantly with assessment of gait.[2,3] The degree to which sedation interferes with gait assessment, however, might depend on the severity of lameness and the skill of the clinician performing the lameness examination. Because of the uncertainty of the effect of sedatives on gait, these drugs are best avoided, if possible.

Administration of an antagonist to the sedative diminishes most of the effects of sedation on gait.[3] Yohimbine (0.15 to 0.25 mg/kg), tolazoline (2 to 4 mg/kg), or atipamezole (0.15 mg/kg) can be administered intravenously to antagonize

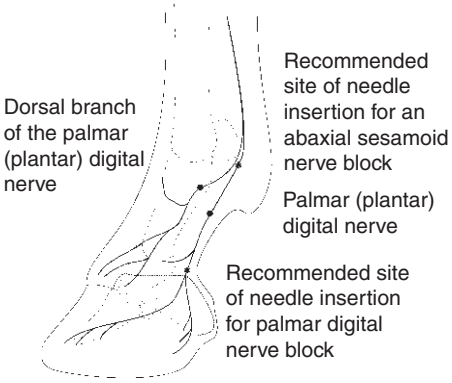

FIGURE 9-1 Sites of administration of local anesthetic solution for regional anesthesia of the forefoot.

FIGURE 9-2 When performing anesthesia of the foot of the forelimb, some clinicians prefer to hold the leg while facing the horse.

the effects of α_2-agonists such as xylazine and detomidine.[4] It is unwise to wait the 20 to 40 minutes after administration of xylazine for the drug's sedative and analgesic effects to dissipate before determining the effects on gait of mepivacaine in either the distal interphalangeal joint or the navicular bursa. After more than 10 minutes following administration of mepivacaine, it is difficult to tell which structures are desensitized by diffusion of the anesthetic solution.[5-7]

TECHNIQUE FOR REGIONAL ANESTHESIA OF THE FOREFOOT

The sites of administration of local anesthetic solution for regional anesthesia of the forefoot are shown in Figure 9-1. Anesthesia of the palmar digital nerves, an abaxial sesamoid nerve block, or a semi-ring block is usually performed with the horse's limb held. When performing anesthesia of the foot of the forelimb, it is preferable to hold the limb while facing toward the rear of the horse (Figure 9-2), but some clinicians prefer to perform these procedures facing toward the front of the horse (Figure 9-3). When facing the same direction as the horse, the foot can be held between the clinician's knees to

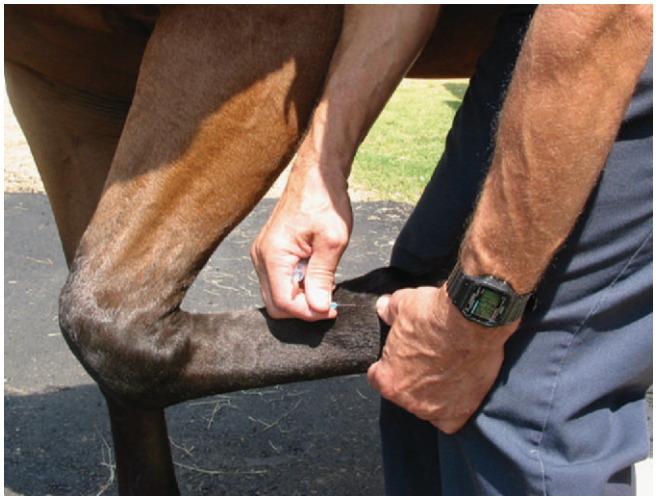

FIGURE 9-3 Some clinicians prefer to perform regional anesthesia of the lower portion of the limb while facing the same direction as the horse.

free both hands for the procedure. When facing the rear of the horse, the procedure is performed using a single hand because one hand must hold the limb. Local anesthetic solution is injected through a 25-gauge, 5/8-inch (0.5 × 16 mm) needle inserted subcutaneously in a distal direction. By inserting the needle subcutaneously directly over the nerve in the direction the nerve runs, local anesthetic solution is deposited over the nerve regardless of the depth of penetration. Injecting as the needle is withdrawn deposits the local anesthetic solution in different tissue planes, thereby increasing the likelihood of rapid contact of solution with the nerve and decreasing the likelihood of administering anesthetic solution into a digital artery or vein (Figure 9-4). This technique also results in a more proximal deposition of solution and possible anesthesia of unintended structures, however. The needle should not be inserted in a proximal direction because proximal diffusion of local anesthetic solution may cause anesthesia of more structures than intended (Figure 9-5).

When performing anesthesia of the palmar or plantar digital nerves, local anesthetic solution should be deposited near the junction of the nerve and the cartilage of the foot (Figure 9-6). More proximal deposition may cause partial analgesia of the proximal interphalangeal joint.[8] When performing an abaxial sesamoid nerve block, local anesthetic solution should be deposited at the base of the proximal sesamoid bones (Figure 9-7). More proximal deposition may cause analgesia of a portion of the metacarpophalangeal joint.[9]

TECHNIQUE FOR REGIONAL ANESTHESIA OF THE HINDFOOT

Techniques for regional anesthesia of the hindfoot are slightly different than techniques for the forelimb because branches of the deep peroneal (fibular) nerve supply additional innervation of the hindfoot. These branches, the medial and lateral dorsal metatarsal nerves, course dorsally along each side of the extensor tendon and continue distally to the laminar

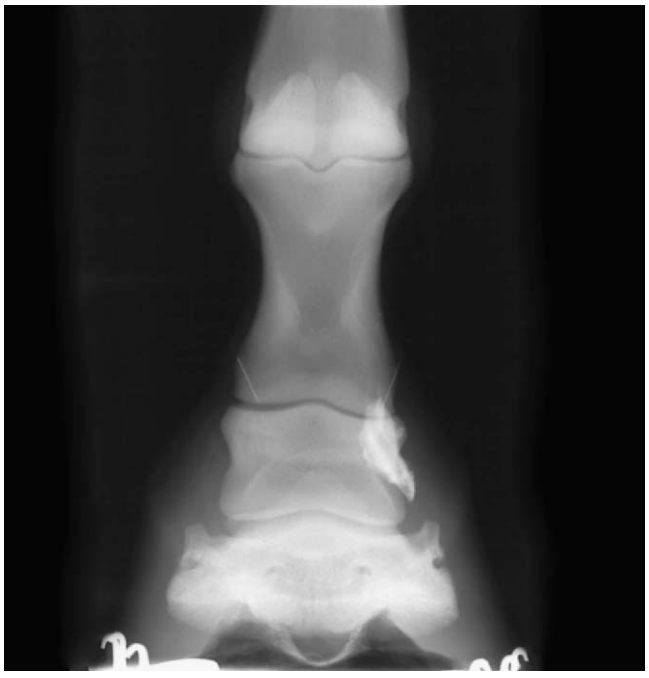

FIGURE 9-4 Radiograph of the region of deposition of local anesthetic solution when 1.5 mL of solution is deposited at the margin of the palmar digital nerves and the cartilage of the foot. Surprisingly, no radiocontrast solution is seen at one of the injection sites, probably because the injection was into the digital artery or vein. Withdrawing the needle during injection deposits the solution in different tissue planes and decreases the likelihood of administering anesthetic solution into a digital artery or vein.

FIGURE 9-5 When performing regional anesthesia of the lower portion of the limb, the needle should never be inserted in a proximal direction because proximal diffusion of local anesthetic solution may cause anesthesia of more structures than intended.

corium. After depositing local anesthetic solution for either a plantar digital or abaxial sesamoid nerve block, the needle is redirected 2 cm dorsally, and an additional 2 mL of local anesthetic solution is deposited subcutaneously as the needle is withdrawn (similar to a semi-ring block).

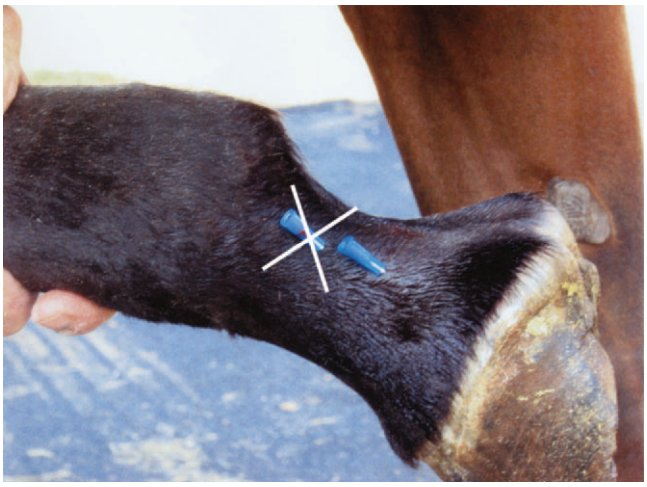

FIGURE 9-6 When performing anesthesia of the palmar or plantar digital nerves, local anesthetic solution should be deposited near the junction of the nerve and the cartilage of the foot because more proximal deposition may cause anesthesia of the proximal interphalangeal joint.

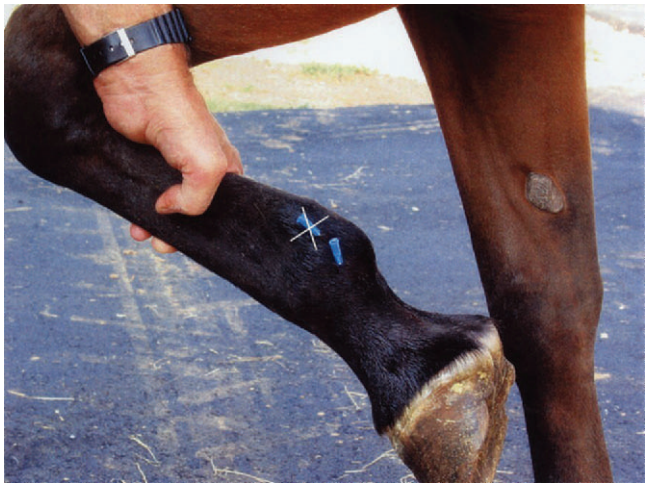

FIGURE 9-7 Performing an abaxial sesamoid nerve block at the base of the proximal sesamoid bones rather than more proximally, using a small volume of local anesthetic solution (2 mL), and directing the needle distally, rather than proximally, decreases the likelihood of the block causing partial analgesia of the metacarpophalangeal joint.

ASSESSMENT OF THE EFFECTS OF REGIONAL ANESTHESIA

Despite loss of sensation at the coronary band after anesthesia of a palmar digital nerve, analgesia of other structures in the foot may be incomplete. Conversely, some horses retain cutaneous sensation after lameness has been ameliorated. After administration of local anesthetic solution, the authors prefer to observe gait before checking for skin sensation at the coronary band. Resolution of lameness usually implies accurate administration of local anesthetic solution. When there is minimal improvement in gait after regional anesthesia, the horse's reaction to firm pressure applied with a ballpoint pen or key to the coronary band (superficial sensation) is tested. A palmar digital nerve block is generally considered successful

FIGURE 9-8 Because some horses react violently to stimulation of skin above the coronary band, it is safer to stimulate the coronary band with the horse's limb held (**A**) or by using a pointed stick several feet long (**B**).

if the horse shows no reaction to the noxious stimulus applied to the coronary band of the heel (elimination of superficial sensation), although complete elimination of deep sensation can only be tested with application of hoof testers to the foot. An abaxial sesamoid nerve block can be considered successful if the horse shows no reaction to a noxious stimulus applied to the coronary band at the toe and at the heel. Because some horses react violently to stimulation of the coronary band (especially if they previously have been examined using regional anesthesia of the foot), stimulating the coronary band with the horse's limb held may be safer than stimulating it while the horse is bearing weight on the limb. Using a long, pointed pole to apply the stimulus may also increase the safety of the procedure (Figure 9-8). Other horses show little reaction to stimulation of the coronary band even before regional anesthesia is performed. If there is a question as to whether the coronary band has been anesthetized or if the horse is merely stoical, the horse's reaction to stimulation of the coronary band of the contralateral foot should also be tested.

ANESTHESIA OF THE PALMAR DIGITAL NERVES

For many years, clinicians have believed that a positive response to anesthesia of the palmar digital nerves on the part of lame horses localizes pain to the palmar third of the foot.[10] A report describing the results of scintigraphic examination of 164 lame horses that had responded positively to anesthesia of the palmar digital nerves demonstrated the imprecision of this nerve block in localizing lameness within the foot.[11] Lame horses in that report that responded positively to anesthesia of the medial and lateral palmar digital nerves were found to be lame because of disease of the distal interphalangeal or proximal interphalangeal joints, fracture of the distal phalanx (including fracture of the extensor process), navicular disease, laminitis, or other diseases of the foot.

Many clinicians believe that anesthesia of the palmar digital nerves desensitizes only the palmar half of the distal interphalangeal joint and that anesthesia of the dorsal branches of this nerve is also necessary to achieve complete analgesia of the joint.[12] Sack,[13] however, demonstrated in an anatomical study that the dorsal branches of the palmar digital nerves do not innervate the distal interphalangeal joint. Easter et al.[14] found that anesthetizing the nerves just proximal to the bulbs of the heel alleviated lameness caused by endotoxin-induced pain in the joint, indicating that the palmar digital nerves alone innervate the distal interphalangeal joint.

Some clinicians consider the level of the pastern at which the palmar digital nerves are anesthetized to be important and recommend anesthetizing the nerves near the proximal margin of the cartilage of the foot.[15,16] Other clinicians, however, describe the proper site for this nerve block to be anywhere from the proximal margin of the cartilage of the foot to the midpastern region.[17] One theoretical advantage of anesthetizing the palmar digital nerves as far distally as possible is that anesthesia of the dorsal branches of the nerve is more likely to be avoided.[16] According to results of the studies by Sack[13] and Easter et al.,[14] however, the dorsal branches are unlikely to contribute much more than sensation to the dorsal aspect of the coronary band and dorsal laminae of the foot.

The palmar digital nerves should be anesthetized at or distal to the proximal margin of the cartilage of the foot because more proximal deposition of local anesthetic solution might result in analgesia of the proximal interphalangeal joint.[8] When a 25-gauge, 5/8-inch (0.5 ×16 mm) needle is inserted over the palmar digital nerve 1 cm above the proximal edge of the cartilage of the foot and directed distally, the needle tip is placed close to the level of the proximal edge of the lateral cartilage. Insertion of a needle at this site causes the solution to be deposited at or slightly below the level of the palmar border of the proximal interphalangeal joint because the height of the cartilage of the foot in relation to the level of the palmar border of the joint is probably similar for most horses.[8] In an anatomical study of innervation of the front digit of the horse, Sack found that deep branches of the lateral (third branch) and medial (fifth and sixth branches) palmar digital nerves supply sensory innervation to the palmar pouches of the proximal interphalangeal joint and that deep branches of the dorsal branch of the palmar digital nerves supply sensory innervation to the joint's dorsal pouch (see Figure 9-1).[13] The deep branches of the palmar digital nerve arise at the level of

the proximal interphalangeal joint, and therefore inadvertent anesthesia of these branches becomes more likely when local anesthetic solution is injected around the nerves farther proximally than the junction of the nerve and the proximal margin of the cartilage of the foot.

To avoid the possible complication of desensitizing part or all of the proximal interphalangeal joint, each palmar digital nerve should be anesthetized by depositing no more than 1.5 mL of local anesthetic solution at or distal to the proximal margin of the cartilage of the foot. Using this technique, branches of the nerve that supply the joint are unlikely to be anesthetized. For some horses, however, lameness caused by pain in the proximal interphalangeal joint may be obviously ameliorated when anesthesia of the palmar digital nerve is performed by inserting 5/8-inch needles over the nerves 1 cm above the proximal edge of the cartilage of the foot.[8] Depositing local anesthetic solution at sites more proximal to this distal site increases the likelihood of anesthetizing the joint.

Some clinicians use a large volume (i.e., 3 mL) of solution for local anesthesia of the palmar digital nerves.[14] Such a large volume of local anesthetic solution is more likely than a small volume (1.5 mL or less) to ameliorate lameness caused by pain in the proximal interphalangeal joint.

Because the proximal interphalangeal joint is innervated by both the palmar digital nerve and its dorsal branch,[13] complete analgesia of the joint should require anesthesia of both these nerves, which is accomplished by administering either an abaxial sesamoid nerve block or a so-called pastern semi-ring block. It is possible that the role of the dorsal branch of the palmar digital nerve in innervation of the proximal interphalangeal joint is not clinically significant, because, in a lameness study, one of six horses that were lame because of pain in the joint appeared sound after anesthesia of the nerves without apparent anesthesia of the dorsal branches (as determined by a reaction to pressure applied with a ballpoint pen to the dorsal aspect of the coronary band).[8]

ANESTHESIA OF THE RAMUS TORI DIGITALIS (RAMUS PULVINUS)

It has been proposed that anesthetizing a branch of the palmar digital nerves, the ramus tori digitalis (ramus pulvinus), results in selective desensitization of the navicular apparatus.[18] Although Sack never specified which branches of the palmar digital nerve innervate the navicular bone,[13] these authors believe that the ramus tori digitalis corresponds to the fourth deep branch of the lateral palmar digital nerve and the seventh or eighth deep branch of the medial palmar digital nerve. When performed as described by Langfeld and Hertsch,[18] anesthesia of the ramus tori digitalis may actually anesthetize the palmar digital nerve distal to the site where the palmar digital nerve gives off the superficial branches to the heel region of the foot, similar to the effect of analgesia of the distal interphalangeal joint (see discussion under Analgesia of the Distal Interphalangeal Joint).

To anesthetize the ramus tori digitalis, a needle is inserted immediately proximal and axial to the proximal margin of the cartilage of the foot, midway between the palmar border of the deep digital flexor tendon and the palmar extent of the cartilage of the foot, and is directed parallel to the slope of the dorsal hoof wall and the long axis of the digit (Figure 9-9). A dose of 2 to 2.5 mL of local anesthetic solution is injected on the medial side of each cartilage of the foot at a depth of 2 to 3.5 cm (for a pony to large horse, respectively) (H. Gerhards, personal communication, 2002).

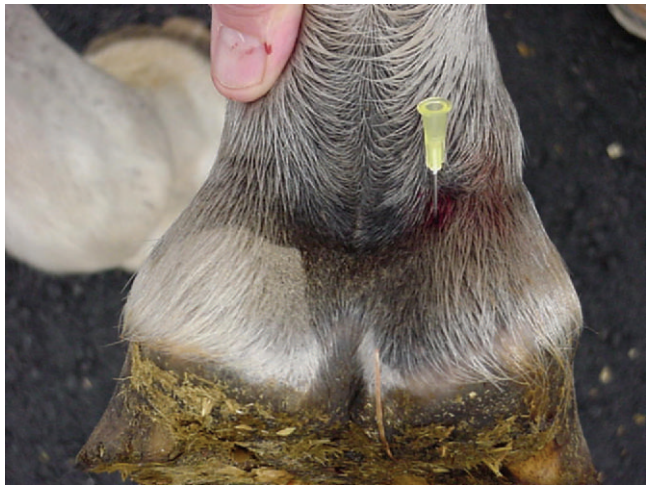

FIGURE 9-9 To anesthetize the ramus tori digitalis, a needle is inserted immediately proximal and axial to the proximal margin of the cartilage of the foot, midway between the palmar border of the deep digital flexor tendon and the palmar extent of the cartilage of the foot and is directed parallel to the slope of the dorsal hoof wall and the long axis of the digit.

PASTERN SEMI-RING BLOCK

When anesthesia of the palmar digital nerves fails to ameliorate lameness, a next step in the lameness examination can be desensitizing the entire foot using a semi-ring block performed at the level of the site of the palmar digital nerve block. A 20-gauge, 1-inch or 1.5-inch (0.90 × 25 or 38 mm) needle is inserted subcutaneously at the site of the previously performed palmar digital nerve block. The needle is directed dorsally, parallel with the bearing surface of the foot (Figure 9-10). A dose of 2 to 3 mL of local anesthetic solution is deposited as the needle is withdrawn. A semi-ring block performed after a negative response to a palmar digital nerve block is unlikely to result in a positive response, because the dorsal branches of the palmar digital nerves contribute little to sensation within the foot.[13] The palmar digital nerve block will already have anesthetized the entire foot, with the exception of the dorsal part of the coronary band and the dorsal laminae of the foot.

ABAXIAL, SESAMOID NERVE BLOCK

Anesthesia of the palmar digital nerves and their dorsal branches at the level of the proximal sesamoid bones (i.e., an abaxial sesamoid nerve block) desensitizes the foot, the middle phalanx and associated soft tissue, the proximal interphalangeal joint, the distal and palmar aspects of the proximal phalanx, and, occasionally, the palmar portion of the metacarpophalangeal joint.[9,19]

FIGURE 9-10 To perform a semi-ring block for anesthesia of the dorsal branches of the palmar digital nerves, a 20-gauge, 1-inch or 1.5-inch (0.90 × 25 or 38 mm) needle is inserted subcutaneously at the site of the previously applied palmar digital nerve block. The needle is directed dorsally on the lateral and medial aspect of the pastern, parallel with the bearing surface of the foot.

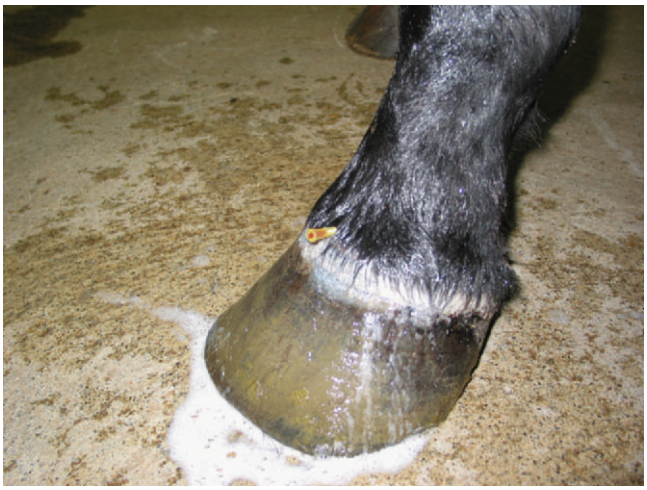

FIGURE 9-11 To administer local anesthetic solution into the distal interphalangeal joint, a 20-gauge, 1-inch (2.5 cm) needle is inserted through the coronary band parallel to the bearing surface of the foot, at the dorsal midline, with the horse bearing weight on the limb.

Performing the nerve block at the base of the proximal sesamoid bones decreases the likelihood of partially desensitizing the metacarpophalangeal joint (see Figures 9-1 and 9-7).[9,19] Using a small volume of local anesthetic solution (≤2 mL) and directing the needle distally, rather than proximally, also decreases the likelihood of an abaxial sesamoid nerve block causing partial analgesia of the metacarpophalangeal joint.

INTRASYNOVIAL ANALGESIA OF THE FOOT

Analgesia of the Distal Interphalangeal Joint

Lateral, palmar, and dorsal approaches for arthrocentesis of the distal interphalangeal joint have been described. Using the lateral or palmar approach, however, the navicular bursa or the digital flexor tendon sheath can be penetrated. A dorsal approach, in which the needle enters the dorsal pouch of the distal interphalangeal joint, is preferable. The clinician should be aware that using the dorsal approach might be more dangerous than using the lateral approach, for which the foot can be held off the ground.

To administer local anesthetic solution into the distal interphalangeal joint, a 20-gauge, 1-inch (1.4 × 25 mm) needle should be inserted through the coronary band parallel (rather than perpendicular, as is commonly described) to the bearing surface of the foot at the dorsal midline with the horse bearing weight on the limb (i.e., the parallel, dorsal approach) (Figure 9-11). Applying firm digital pressure at this site for several seconds before the needle is inserted may decrease pain to and reaction from the horse. Some horses react violently to insertion of the needle by thrusting the limb upward. To perform the procedure safely, the clinician and person holding the horse should anticipate this reaction and position themselves accordingly.

The lateral approach to inject the distal interphalangeal joint may be safer to perform than the dorsal approach because the foot can be held and because insertion of the needle at this location elicits a milder reaction from the horse. Using the lateral approach, however, the navicular bursa or the digital flexor tendon sheath can occasionally be penetrated.[20] The navicular bursa or digital flexor tendon sheath is less likely to be penetrated when the lateral approach is taken with the horse's foot bearing weight, with the limb directly under the horse, and by using a 1-inch (2.5 cm) needle, rather than a 1.5-inch (3.8 cm) needle (Figure 9-12).[21]

Administration of mepivacaine into the distal interphalangeal joint was shown experimentally to desensitize the distal interphalangeal joint,[14] the navicular bursa,[22] and the toe region of the sole.[5,23] When a large volume of mepivacaine (10 mL) was administered, the heel region of the sole was also desensitized.[5]

Bowker et al.[24] demonstrated, in an anatomical study, that the majority of the sensory fibers to the navicular bone and its collateral sesamoidean ligaments are located along the dorsal surface of the collateral sesamoidean ligaments and the impar ligament and are directly subsynovial to the distal interphalangeal joint. They theorized that local anesthetic solution administered into the distal interphalangeal joint might desensitize these nerves. Pleasant et al.[22] speculated that analgesia of the distal interphalangeal joint would likely anesthetize the palmar digital nerves, resulting in desensitization of the navicular bone and associated structures, because the palmar digital nerves lie in close proximity to the proximal, palmar pouch of the distal interphalangeal joint.

Gough et al.[25] found, in a study using cadavers, that dye of small molecular weight and local anesthetic solution diffused from the distal interphalangeal joint into the navicular bursa. In two separate studies, mepivacaine was found in the navicular bursa in sufficient concentration to produce analgesia of the bursa, after 5 or 8 mL of the solution was administered into the distal interphalangeal joint.[25,26] In one of those studies, the investigators also considered the concentration of

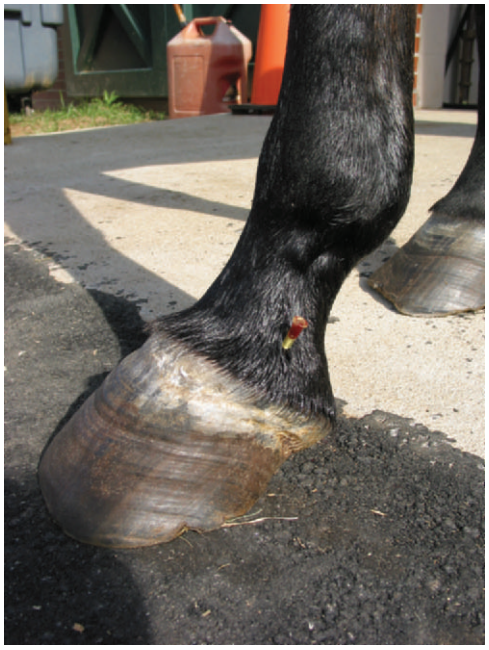

FIGURE 9-12 By performing the lateral approach to the distal interphalangeal joint with the horse's foot bearing weight, with the limb directly under the horse, and by using a 1-inch (2.5 cm) needle (rather than a 1.5-inch [3.8 cm] needle), penetration of the navicular bursa and digital flexor tendon sheath is unlikely.

mepivacaine in the medullary cavity of the navicular bone of some horses to be sufficient to cause analgesia of the navicular bone.[26]

Clinical experience indicates that a negative response to intraarticular analgesia of the distal interphalangeal joint may not eliminate the navicular bone and its related structures as the source of lameness. In a study of 102 horses with chronic foot pain, Dyson found that 21% of horses failed to respond to intraarticular analgesia of the distal interphalangeal joint but showed significant improvement after intrabursal analgesia of the navicular bursa.[27] A recent study showed that lesions of the deep digital flexor tendon at the level of the tendon's insertion to the distal phalanx were more effectively desensitized by administration of local anesthetic solution into the navicular bursa than by analgesia of the distal interphalangeal joint.[28] Some of the lame horses in Dyson's study that responded to analgesia of the bursa but were unresponsive to analgesia of the joint could have had painful lesions at the insertion of the deep digital flexor tendon rather than disease of the navicular bone and its related structures.

Administration of 6 mL mepivacaine into the distal interphalangeal joint desensitizes the toe region of the sole,[5,23] and if a large volume of mepivacaine (≥10 mL) is used, the heel portion of the sole may also become desensitized.[5] For lameness that can be ameliorated with a palmar digital nerve block, evaluation of gait after intraarticular analgesia of the distal interphalangeal joint with a low volume of mepivacaine (≤6 mL) may help in determining whether pain in the soft tissues of the heel region is the cause of lameness.[2,5] The pain is unlikely to originate from the soft tissues of the heel if the lameness is ameliorated by this analgesia.

Analgesia of the Navicular Bursa

A study comparing various techniques for inserting a needle into the navicular bursa showed that the distal, palmar approach to the "navicular position" is the most accurate approach.[29] To insert a needle into the navicular bursa, the skin between the bulbs of the heel, immediately above the coronary band, is desensitized with 1 mL of local anesthetic solution. A 20-gauge, 3.5-inch (1.4 × 89 mm), disposable spinal needle is inserted midway between the bulbs of the heel, immediately proximal to the coronary band, through the desensitized skin, with the limb positioned in a Hickman block.

The spinal needle is advanced along the sagittal plane of the foot, toward the bisecting point between the sagittal plane of the foot and the long axis of the navicular bone (Figure 9-13). The long axis of the navicular bone is assumed to be halfway between the most dorsal and the most palmar aspect of the coronary band and 1 cm distal to the coronary band.[30] The spinal needle is advanced until the tip of the needle contacts bone, and a mixture of local anesthetic solution (2 to 3 mL) and radiographic contrast medium (0.5 to 1 mL) is injected. Further flexing the lower portion of the limb may decrease resistance to injection (Figure 9-14). The tip of the needle is determined to be within the navicular bursa by low resistance to injection and the ability to aspirate the injected contents of the syringe. The foot is then examined radiographically immediately after injection of the bursa. Radiographic identification of the contrast medium within the bursa is interpreted as evidence of a successful bursal injection (Figure 9-15).

A positive response to administration of local anesthetic solution into the navicular bursa indicates disease of the navicular bursa, the navicular bone, or its supporting ligaments[2]; solar toe pain[6]; or disease of the deep digital flexor tendon.[28] Even though analgesia of the distal interphalangeal joint rapidly results in analgesia of the navicular bursa,[22] analgesia of the bursa does not rapidly result in analgesia of the joint.[2,7,27,32] Analgesia of the navicular bursa may help to differentiate pain associated with disease of the distal interphalangeal joint from pain associated with disease of the navicular bone and associated structures. Pain arising from the joint can reasonably be excluded as a cause of lameness when lameness is attenuated within 10 minutes by analgesia of the navicular bursa.[7]

The results of the studies of Pleasant et al.[22] and Schumacher et al.[7] suggest that the site of direct contact between the palmar pouch of the distal interphalangeal joint and the palmar digital nerves is located at a region proximal to the origin of the deep branches that innervate the distal interphalangeal joint and the navicular bursa, and that the site of direct contact between the navicular bursa and the palmar digital nerves is located distal to these branches.

Another possible explanation for the observation that analgesia of the navicular bursa does not cause analgesia of the distal interphalangeal joint is that local anesthetic solution appears to diffuse more slowly from the bursa to the joint than from the joint to the bursa.[24-26,31,33] Both Bowker et al.[24,33] and Gough et al.[25] found a significant difference between the extent of diffusion from the distal interphalangeal joint to the navicular bursa and the extent of diffusion from the bursa to the joint (Figure 9-16). In one study, four times more Luxol fast blue dye and mepivacaine diffused from the joint to the bursa (65%) than vice versa (12.5%).[24] In a cadaver

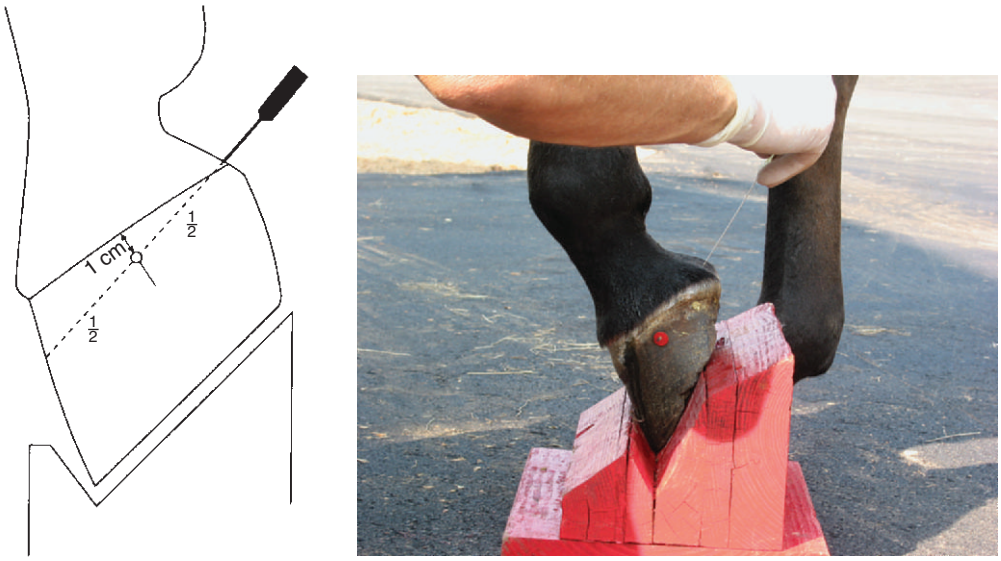

FIGURE 9-13 To administer local anesthetic solution into the navicular bursa, the limb is positioned in a Hickman block. The spinal needle is advanced along the sagittal plane of the foot, toward the bisecting point between the sagittal plane of the foot and the long axis of the navicular bone.

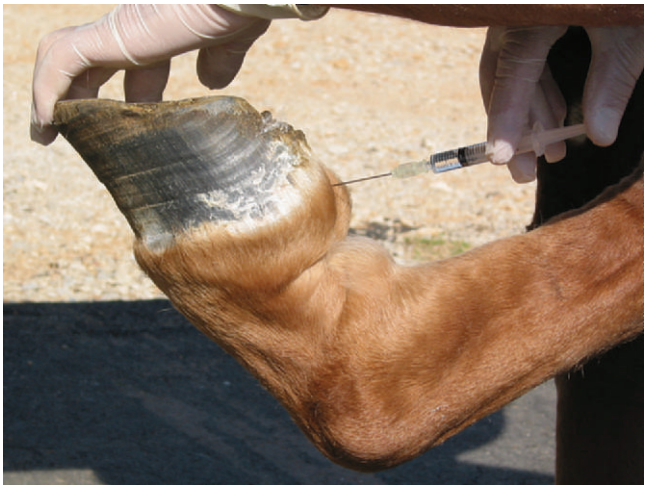

FIGURE 9-14 Further flexing the lower portion of the limb may decrease resistance to injection of the navicular bursa.

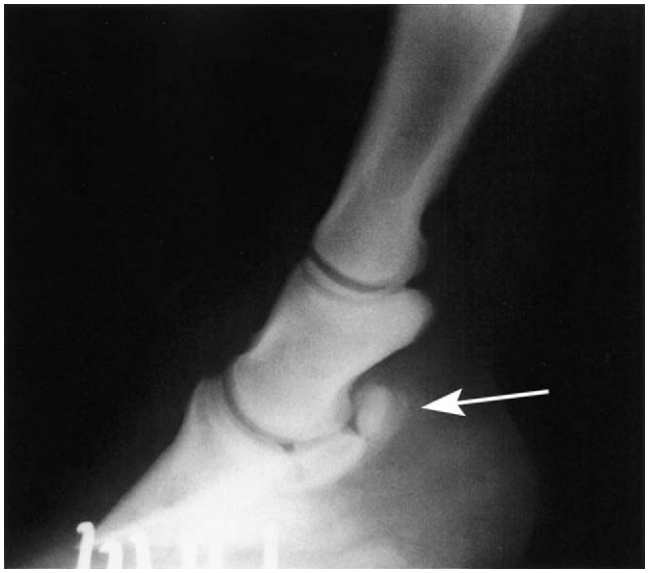

FIGURE 9-15 Radiographic identification of contrast medium within the bursa is interpreted as evidence of a successful bursal injection. The bursa in this slightly oblique lateral radiograph was injected with 3.5 mL mepivacaine mixed with 0.5 mL diatrizoate meglumine.

study of diffusion of mepivacaine, significantly more mepivacaine was found in the navicular bursa after injection of the distal interphalangeal joint than the converse.[25]

In addition to experimental findings concerning the effect of analgesia on the navicular bursa, clinical observations indicate that a positive response to intraarticular analgesia of the distal interphalangeal joint and a negative response to intrabursal analgesia of the navicular bursa point to pain within the joint as the cause of lameness.[32] This clinical observation is valid if solar pain can be eliminated as a cause of lameness.[5,6,23] The presence of solar pain can be determined, in many cases, by use of hoof testers (Figure 9-17).

The Effect of Time on Interpretation of Intraarticular or Intrabursal Analgesia of the Foot

The effect of intraarticular analgesia of the distal interphalangeal joint or intrabursal analgesia of the navicular bursa on lameness should be assessed soon after injection (i.e., within 10 minutes). After this period, the structures that are desensitized by diffusion of the anesthetic solution become uncertain.[5-7] Some clinicians have assumed that improvement in lameness observed within 10 minutes of injection of the joint with local anesthetic solution indicates that lameness is caused by joint pain alone, and that improvement observed more than 10 minutes after injection is caused by diffusion of local anesthetic solution into the navicular bursa or around the nerves providing sensory innervation to the navicular bone and its associated structures.[34,35] The authors dispute this time-lapse technique. In some reports, clinicians observed a

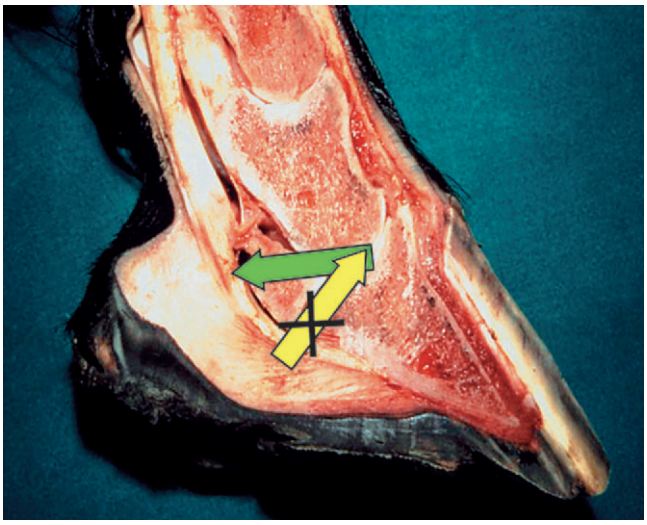

FIGURE 9-16 Significantly more local anesthetic solution diffuses from the distal interphalangeal joint to the navicular bursa than diffuses from the bursa to the joint.

FIGURE 9-17 Pain within the distal interphalangeal joint as a cause of lameness is likely after a positive response to intraarticular analgesia of the distal interphalangeal joint and a negative response to intrabursal analgesia of the navicular bursa, if solar pain was not detected using hoof testers.

positive response to intraarticular analgesia of the distal interphalangeal joint within 5 to 8 minutes of injection in 52% to 61% of horses with navicular disease or experimentally induced navicular bursal pain.[22,27,36]

Technique for Synoviocentesis of the Digital Flexor Tendon Sheath

Local anesthetic solution can be administered into the digital flexor tendon sheath to identify intrathecal pain. Synoviocentesis of the digital flexor tendon sheath can be performed in one of several areas where the sheath may bulge when it is distended with fluid (Figure 9-18). Access to these pouches is not difficult when the sheath is distended (Figure 9-19) but is difficult when it is not. Synoviocentesis can be achieved by introducing a 20-gauge, 1-inch (2.5 cm) needle along the dorsal

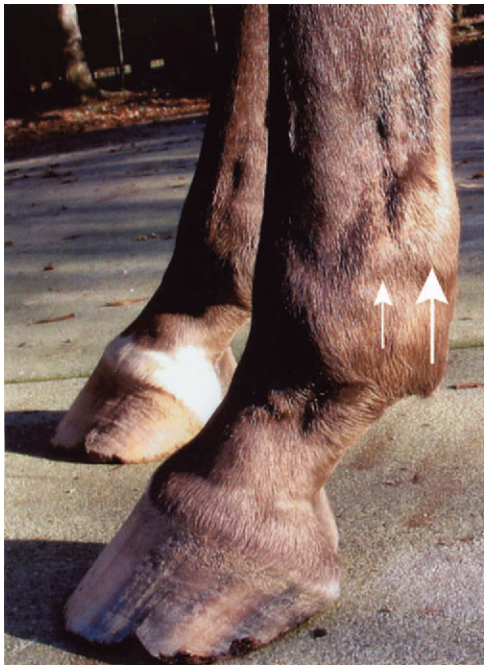

FIGURE 9-18 Synoviocentesis of the digital flexor tendon sheath can be performed in one of several areas where the sheath may bulge when distended with fluid.

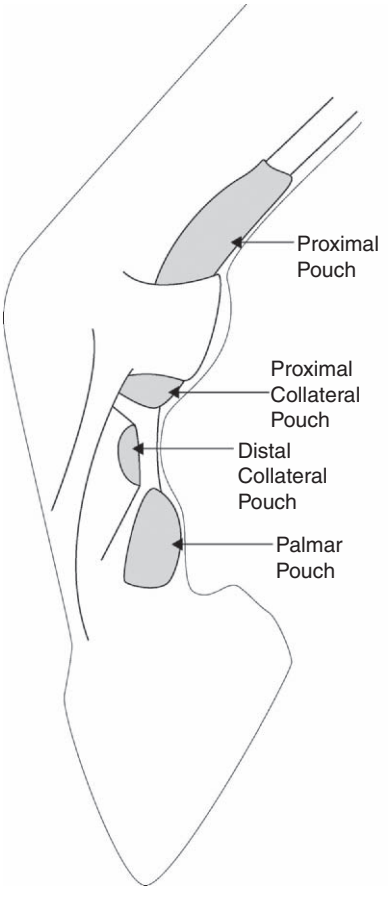

FIGURE 9-19 Synoviocentesis of the digital flexor tendon sheath is not difficult if the sheath is distended with synovial fluid. The lateral proximal pouch of the sheath (*large arrow*) is distended in this horse. The palmar pouch of the fetlock is also distended with fluid (*small arrow*).

aspect of the deep digital flexor tendon (DDFT), 1 cm proximal to the proximal border of the palmar annular ligament and 1 cm palmar to the lateral branch of the suspensory ligament. Easier access can be gained via the sheath's distal palmar pouch, which extends between the two branches of the superficial digital flexor tendon (SDFT) and between the two digital annular ligaments, along the palmar surface of the DDFT (Figure 9-20). This pouch is divided sagittally by the mesotenon of the DDFT in its distal part; therefore the needle should be aimed to access this pouch between the lateral or medial border of the DDFT and the ipsilateral distal branch of the SDFT to minimize the risk of inadvertent penetration of the DDFT.

FIGURE 9-20 The digital flexor sheath can be accessed at its distal palmar pouch. To minimize the risk of inadvertent needle penetration of the deep digital flexor tendon (DDFT), the needle should be aimed to access this pouch between the lateral or medial border of the DDFT and the ipsilateral distal branch of the superficial digital flexor tendon (SDFT).

The digital flexor tendon sheath can also be accessed via its proximal or distal collateral pouches. The proximal collateral pouch is situated in the triangular space between the base of the proximal sesamoid bone, the proximal insertion of the proximal digital annular ligament, and the dorsal border of the DDFT. It can be entered 1 cm distal to the base of the proximal sesamoid bone and 1 cm palmar to the neurovascular bundle. The distal collateral pouch is located on the lateral (or medial) aspect of the pastern, between the flexor tendons and the distal sesamoidean ligaments and between the proximal and distal insertions of the proximal digital annular ligament.

A cadaver study showed that synoviocentesis of the digital flexor tendon sheath is most consistently successful when performed at the level of the proximal lateral pouch on the non–weight-bearing limb.[37] More recently, an approach through the palmar annular ligament of the fetlock (palmar axial sesamoidean approach) was found to be even more reliable for consistent synoviocentesis of the digital flexor tendon sheath (Figure 9-21).[38]

To perform the palmar axial sesamoidean approach to the digital flexor tendon sheath, the fetlock joint is flexed to a dorsal angle of 225 degrees, and the needle is placed through the skin at the level of the midbody of the lateral proximal sesamoid bone, through the palmar annular ligament, 3 mm axial to the palpable palmar border of the lateral proximal sesamoid bone and immediately palmar to the palmar digital neurovascular bundle. The needle is inserted in a transverse plane and advanced at an angle of 45 degrees to the sagittal plane, aimed toward the central intersesamoidean region to a depth of 1.5 to 2.0 cm. This technique results in fewer attempts required for successful entry when compared with the proximolateral approach (Figure 9-22). Because the synovium in this location is less villous, cellular, and mobile than in the proximal pouch, synoviocentesis using the palmar axial sesamoidean approach is less likely to result in synovial hemorrhage. A dose of 10 mL of local anesthetic solution is injected for adequate desensitization of the digital flexor tendon sheath.

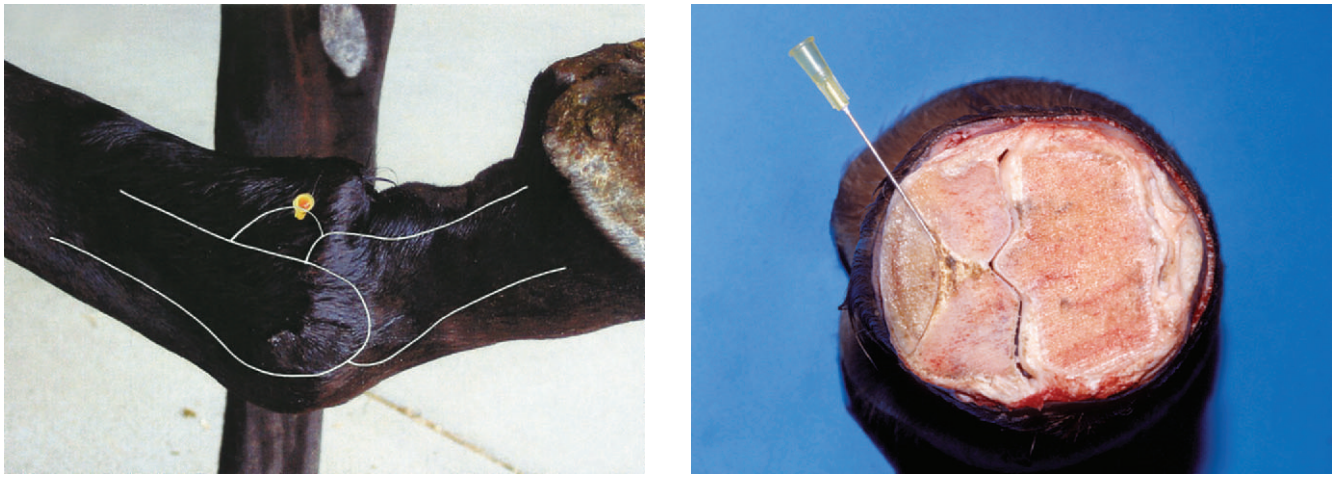

FIGURE 9-21 The palmar axial sesamoidean approach to the digital flexor tendon sheath was found to be reliable for consistent synoviocentesis. The fetlock joint is flexed, and the needle is placed through the skin at the level of the midbody of the lateral proximal sesamoid bone, immediately palmar to the neurovascular bundle. The needle is inserted and advanced at an angle of 45 degrees to the sagittal plane, aimed toward the central intersesamoidean region to a depth of 1.5 to 2.0 cm.

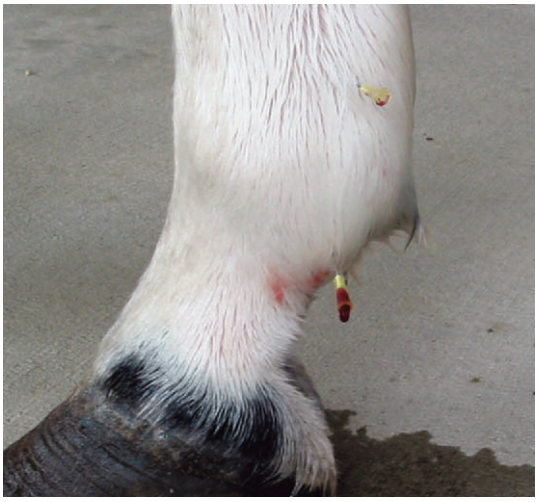

FIGURE 9-22 Synoviocentesis of the digital flexor tendon sheath has been performed at the proximolateral pouch (*upper needle*) and at the proximal collateral pouch (*lower needle*). Synovial hemorrhage is more likely to occur when synoviocentesis is performed at these sites than when the palmar axial sesamoidean approach is used.

Diagnostic Analgesia of the Digital Portion of the Deep Digital Flexor Tendon

In a study of 72 horses with foot pain, significant abnormalities of the digital portion of the DDFT were identified in 30 of the horses, using magnetic resonance imaging.[28] Based on the appearance of the abnormal signal in the DDFT and necropsy studies of the DDFT, lesions were subdivided into core lesions, parasagittal splits, insertional lesions, fibrillations and erosions of the dorsal surface, and multifocal tendonitis.

Desensitizing the palmar nerves at the level of the proximal sesamoid bones (i.e., an abaxial sesamoid nerve block) abolished or improved lameness in all 30 horses, but a palmar digital nerve block ameliorated lameness in only 60% of horses. Analgesia of the distal interphalangeal joint ameliorated lameness in 63% of horses, and analgesia of the navicular bursa ameliorated lameness in 67% of horses. A palmar digital nerve block was most frequently ineffective in ameliorating lameness caused by a core lesion in the DDFT, even though these core lesions rarely extended further proximally than the level of the proximal interphalangeal joint.

Because lameness caused by disease of the DDFT within the foot failed to improve significantly after anesthesia of the palmar digital nerves, the distal interphalangeal joint, or the navicular bursa in 30% to 40% of horses, a portion of the DDFT within the foot must receive its sensory supply from more proximal deep branches of the medial and lateral palmar digital nerves that enter the digital sheath. Improvement of lameness in horses with similar lesions of the DDFT after intrathecal analgesia of the digital synovial sheath has been described.[39]

Lesions at the level of the insertion of the DDFT to the distal phalanx were more effectively desensitized by administration of local anesthetic solution into the navicular bursa than by analgesia of the distal interphalangeal joint.[28] This finding was surprising in light of the reported diffusion of local anesthetic agents in therapeutic concentrations from the distal interphalangeal joint to the navicular bursa.

COMPLICATIONS OF ANESTHETIZING THE FOOT

Complications associated with anesthesia of the horse's foot, other than transient swelling of the lower limb, are rare. The risk of infecting a synovial cavity is minimal if the site of injection is prepared as previously described. Inadvertently administering a local anesthetic agent into a synovial cavity, rather than subcutaneously, could result in infection of that synovial cavity, particularly if the skin was minimally prepared for subcutaneous administration. This risk can be significantly reduced by taking care to insert the needle subcutaneously and by adequately preparing the skin for aseptic injection, in the unlikely event that the needle is inadvertently placed into a synovial cavity. Nevertheless, one of the authors has encountered iatrogenic infection of the proximal interphalangeal joint after a palmar digital nerve block for which the injection site was thoroughly prepared as described. A subcutaneous abscess developed at the injection site, leading to an infection of the proximal interphalangeal joint with fatal consequences.

Considering that lameness is a natural response to reduce concussion to the injured tissue, removing pain by use of regional or synovial analgesia can result in more tissue damage. Propagation of an unrecognized fracture of a phalanx, resulting in complete fracture, or additional tearing of a ligamentous or tendinous structure within the foot can occur after anesthesia of the foot. Unnecessary trauma to injured structures can be minimized during a lameness examination by forcing the horse to travel the minimum distance necessary to accurately evaluate its gait. Ruling out the presence of an incomplete fracture, using radiographic or scintigraphic examination of suspected sites of lameness, may be prudent before local anesthesia is used to determine the site of lameness in any horse in which a severe lameness of acute onset has been observed, even if only transiently. Caution is required even more when considering the use of regional anesthesia in racing Thoroughbreds because of the relatively increased risk of fracture in these horses.

SUMMARY

Structures in the foot anesthetized by regional, joint, and bursal analgesia are summarized in Figure 9-23. Based on clinical trials by Schumacher et al.,[5-8,23] Easter et al.,[14] and Pleasant et al.,[22] the conclusion can be drawn that anesthesia of the palmar digital nerves is the least specific analgesic technique used to localize pain in the foot. Anesthesia of these nerves resolves lameness caused by pain in both the toe and heel regions of the sole, the distal interphalangeal joint, and the navicular bursa. Partial analgesia of the proximal interphalangeal joint may result if the palmar digital nerves are anesthetized more proximally than at the level of the proximal margin of the cartilages of the foot.[8]

Analgesia of the distal interphalangeal joint is more specific than anesthesia of the palmar digital nerves in localizing pain within the foot, because lameness caused by solar pain in the heel region is not resolved, provided that 6 mL or less of local anesthetic solution, rather than 10 mL or more, is administered into the joint.[5] Analgesia of the navicular bursa, performed after localizing pain to the foot (using analgesia of the distal interphalangeal joint or anesthesia of the palmar digital

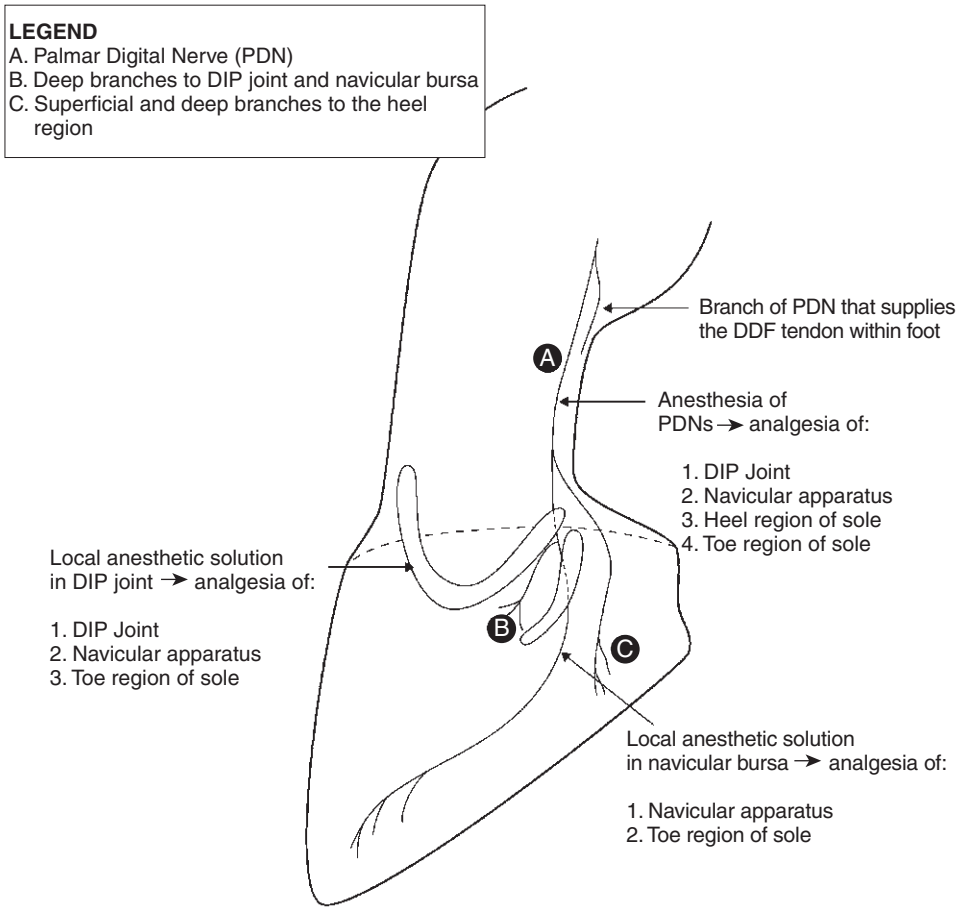

FIGURE 9-23 Structures anesthetized by regional, intraarticular, and bursal analgesia of the foot.

nerves), might help to localize pain within the foot, because lameness caused by pain either in the solar portion of the heel or within the distal interphalangeal joint is not likely to be ameliorated by analgesia of the navicular bursa. These experimental findings agree with the clinical observation that a positive response to intraarticular analgesia of the distal interphalangeal joint and a negative response to intrabursal analgesia of the navicular bursa indicate pain within the distal interphalangeal joint as a cause of lameness.[32] This clinical observation is valid if solar pain can be eliminated as a cause of lameness.[5,6,23] The presence of solar pain can be determined, in many cases, by use of hoof testers.

It may be useful to perform intrathecal analgesia of the digital flexor tendon sheath of horses with lameness that is unchanged after anesthesia of the palmar digital nerves but resolves after an abaxial sesamoid nerve block. Resolution of lameness after intrathecal analgesia of the flexor tendon sheath justifies suspicion of a lesion of the digital portion of the deep digital flexor tendon.

Clinicians should be aware that techniques of diagnostic analgesia of the horse's foot might provide misleading information concerning the site of pain causing lameness because of possible variations in digital neurologic anatomy or misdirection of a needle during administration of local anesthetic solution.[40] Results of articular, bursal, or regional analgesia of the foot should be interpreted with some degree of skepticism.

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10
DIAGNOSTIC IMAGING

DIANNE LITTLE and MICHAEL C. SCHRAMME, editors

RADIOGRAPHY AND RADIOLOGY OF THE FOOT

Michael C. Schramme

Comprehensive discussions of radiography of the foot are provided in many textbooks. A basic examination consists of a lateromedial, a dorsoproximal-palmarodistal oblique (upright pedal) projection with an appropriate exposure for the distal phalanx, a dorsoproximal-palmarodistal oblique projection with an appropriate exposure for the navicular bone, a palmaroproximal-palmarodistal oblique projection, and a standing zero-degree dorsopalmar projection. Additional views include the dorsal 45-degree lateral palmaromedial oblique (D45° LPMO) and dorsal 45-degree medial palmarolateral oblique (D45° MPLO) projections, as well as other lesion-oriented oblique views.

Diseases of the front feet are the most common cause of lameness in horses. A survey of 43,500 lame horses in general veterinary practice in the United Kingdom in the early 1960s found the foot to be the cause of the problem in more than 30% of horses. A more recent study estimated that the navicular bone alone was responsible for approximately one third of cases of chronic lameness in horses.[1] The diagnosis of lameness depends first and foremost on a detailed clinical evaluation, but it is then necessary to decide on the most appropriate imaging modality (or modalities) to use to make a final diagnosis. In spite of the ongoing development of sophisticated new imaging modalities and their increasing availability to the equine practitioner in the last decade, radiography still provides the practitioner with the baseline structural or anatomical information regarding the osseous tissues of the foot. Although the advent of digital radiography has made radiographic examination of the horse's foot much easier and has increased the diagnostic yield tremendously, the basic and time-honored steps of the radiographic examination still need to be followed in order to produce a standardized set of high-quality radiographs.

Frequently diagnosed causes of chronic foot lameness are palmar foot pain, DIP joint pain, and navicular pain. These diagnoses are commonly associated with radiologic abnormalities of the DIP joint, the distal phalanx, and the navicular bones. There is controversy about the interpretation of radiographs of these structures, and the arrival of more sophisticated imaging modalities such as scintigraphy, CT, and MRI have made it clear that radiography is not a particularly sensitive indicator of disease.

TECHNICAL AND SAFETY FACTORS

Radiography of the horse's limbs is performed with the aid of long-handled cassette holders. These can be mounted on wheels with a telescoping arm, to avoid any risk to assistants in the vicinity of the primary beam. Sedation is a useful aid in controlling the horse and minimizing the number of exposures. All standard rules of radiation safety are respected. The film focus distance is generally 1 meter and needs to be carefully measured each time to avoid exposure differences caused by film focus distance variations. Typical exposures for lower limb radiographs are in the region of 65 to 70 kV and 4 mAs with high-speed screen and film combinations. The use of a grid is advisable if the region under scrutiny is more than 11 cm in thickness. The exposure with a grid is multiplied by a grid factor, which generally results in exposures in the region of 70 kV and 12 to 15 mAs. Increasingly popular in equine practice is the use of high-definition film and screen combinations (mammography or medical imaging film). This requires a threefold to sixfold increase of the exposure factors (typically 70 kV and 15 to 25 mAs). High-frequency portable radiography systems allow exposure factors in this range with sufficiently short exposure times.

PREPARATION OF THE FOOT

The shoe should be removed and the foot trimmed and thoroughly cleaned. All loose and flaky horn should be removed and the walls cleaned with a hard brush or rasp. It is advisable to fill both the lateral and medial sulci and the central cleft of the frog with a material of similar radiopacity to horn, to avoid the disturbing superimposition of these air-filled spaces on the skeletal structures of the foot. Both Play-Doh (3M, St. Paul, Minn.) and soft soap can be used to this purpose. The radiopacity of different batches of Play-Doh may be somewhat inconsistent, but it remains attached to the foot better than soap. In the absence of a resident farrier, shoe removal is sometimes impractical because of owner reluctance or because the diagnostic work-up (e.g., further nerve blocks) will be continued afterward. In most cases with open shoes, it is possible to obtain diagnostic radiographs of the navicular bones with the shoes in place.

TECHNIQUE, NORMAL ANATOMY, AND RADIOLOGIC INTERPRETATION

Dorsoproximal-Palmarodistal Oblique (Upright Pedal) Projection

Technique

To obtain a dorsoproximal-palmarodistal oblique projection (Figures 10-1 and 10-2), the foot is placed on a navicular block (Figure 10-3), where it is held by an assistant. This block is essentially a stable wooden block with a trough-like arrangement at the top in which the toe of the foot can be rested. The oblique part of the top of the block that supports

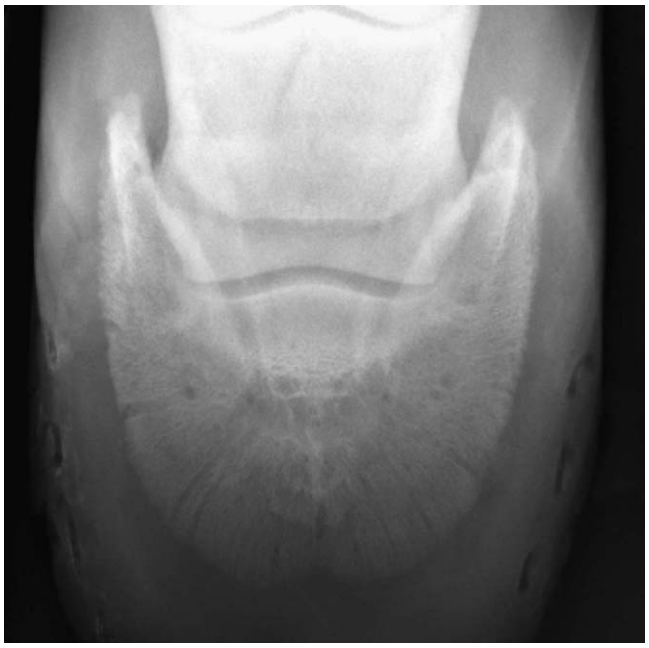

FIGURE 10-1 Normal dorsoproximal-palmarodistal oblique view (distal phalanx).

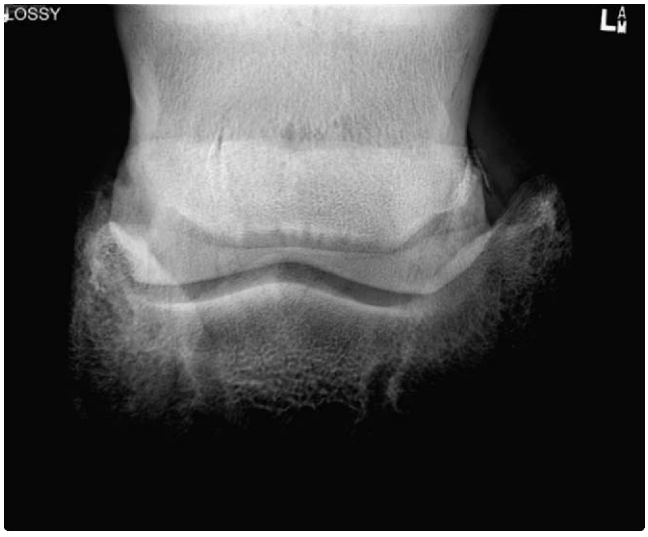

FIGURE 10-2 Normal dorsoproximal-palmarodistal oblique view (navicular bone).

the sole makes an angle of 55 degrees with the horizontal. The limb is held firmly by the cannon bone, and the foot is pushed back into the block to align the sole with the oblique surface of the top of the block. This alignment prevents forward knuckling of the pastern, which minimizes the amount of bony superimposition on the navicular bone by the middle phalanx. Low and high upright blocks are in use. The height of the block is determined by the lowest possible position in which the x-ray tube can produce a horizontal beam. Horses tend to stand quietly with the limb raised on a block 25 cm in height. With lower blocks, there is a continuous tendency on the part of the horse to try to stand on the limb. The beam is centered just proximal to the level of the coronary band in the midline of the foot. A cassette is positioned vertically behind the foot.

FIGURE 10-3 Foot positioned in a navicular block.

It is difficult to select exposure factors that will be satisfactory for both the distal phalanx and the navicular bone. Separate exposures are advisable, which also allows the examiner to collimate the beam for navicular views. Collimation is good radiographic practice and also improves the contrast and sharpness of the image by reducing the fogging effect of scatter radiation. All radiographs of the navicular bones should be obtained using a grid (12:1 ratio and 44 lines/cm). A perpendicular alignment between the central beam and the grid should be maintained at all times. Absence of a grid and lack of attention to grid alignment are some of the main causes for poor-quality radiographs in this projection.

This projection highlights the distal phalanx; the navicular bone, especially its distal border and the synovial invaginations (canales sesamoidales) originating from the synovial fossa; and the joint space of the DIP joint.

A second upright pedal view is taken with a radiolucent plastic wedge of 10 degrees inserted between the sole and the surface of the block. The angle of the sole with the horizontal is then 65 degrees. This view is optional, except in cases in which the distal border of the navicular bone is superimposed on the DIP joint space on the 55-degree projection, in which case it is mandatory in order to allow accurate assessment of the distal border of the navicular bone. This projection alters the image of the distal and proximal borders of the navicular bone. The distal border now shifts proximally relative to the DIP joint space. The joint space between the distal articular margin of the navicular bone and the palmar articular margin of the distal phalanx is now clearly visible. The distal border of the ridge from which the impar ligament originates is less clearly seen in this view.

Some clinicians prefer to use the high coronary projection (Figure 10-4), or dorsal 60-degree proximal palmarodistal oblique projection, to the upright pedal view with its horizontal beam. In this variant, the foot is placed in a normal weight-bearing position on a cassette tunnel, the cassette is placed horizontally in the tunnel, and the x-ray beam is centered on the coronary band pointing at a 60-degree downward angle. Positioning of the foot is considerably easier but alignment of a

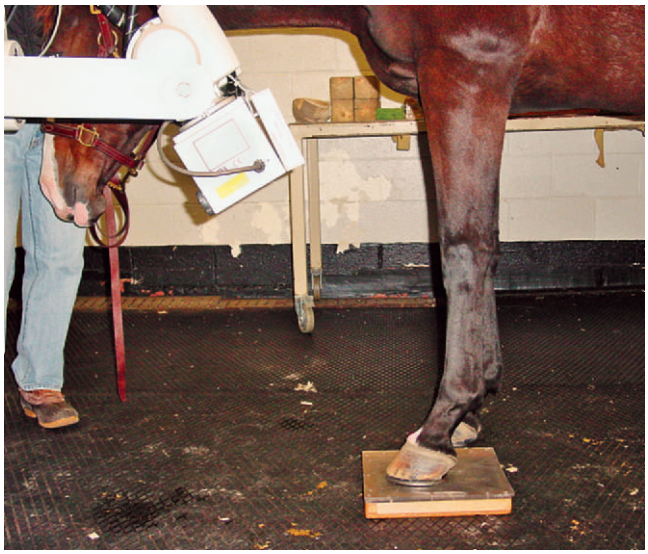

FIGURE 10-4 High coronary route for the dorsoproximal-palmarodistal oblique view of the foot.

FIGURE 10-5 Foot positioned in a 45-degree navicular block.

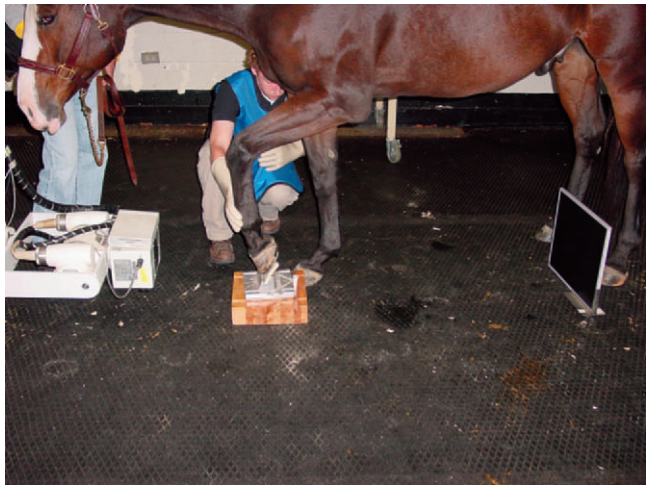

FIGURE 10-6 Air-gap technique for the dorsoproximal-palmarodistal oblique view of the foot.

grid in the cassette tunnel with the central beam is more difficult. The author prefers the upright pedal view over the high coronary view because the central beam is kept perpendicular to the cassette and therefore minimal distortion of the navicular bone occurs. The high coronary view causes an increase in the perceived proximodistal height of the navicular bone. The upright pedal route also makes it easier to use a grid.

The upright pedal view can also be performed with a 45-degree block (Figure 10-5), which is different from the classic navicular block. The 45-degree block is simply a square piece of plywood, supported at an angle of 45 degrees to the floor, with two wooden slats attached on its frontal face in a V-shape to support the toe of the non–weight-bearing foot. The central beam is angled at 15 degrees downward to the horizontal and is centered at the level of the coronary band in the midline of the foot, whereas the cassette is held behind the foot by two wooden slats in a tunnel arrangement. The author prefers to perform the upright pedal projection with the 45-degree block because this arrangement causes minimal distortion of the skeletal structures and minimal grid artifacts and is easy to perform.

Finally, this view can also be obtained using the air gap or magnification technique (Figure 10-6). The foot is positioned on a navicular block. The x-ray tube is positioned close to the dorsal aspect of the foot, centered, and collimated on the navicular bone at the level of the coronary band. The cassette is positioned approximately 80 cm behind the foot and a film focus distance of 100 cm is maintained. No grid is used in this projection, because the air gap effectively reduces scatter radiation. The improved contrast and sharpness of the technique, combined with the magnification provided, make this a useful view to assess the presence of distal border alterations in the navicular bone. This view can be obtained only with a very small focal spot and therefore requires a tube with an adjustable focal spot and a rotating anode.

Normal Variation

Artifacts in this projection are caused by either grid lines or "frog lines." Grid artifacts occur when the central beam is not orientated at 90 degrees to the grid. This causes grid cut-off, since too much of the primary beam is absorbed by the grid lamellae. Frog-lines are caused by poor preparation of the foot. In the absence of frog filler such as Play-Doh, broad radiolucent shadows originating from the air-filled spaces in the sulci of the frog are superimposed onto the radiographic image of the distal phalanx and navicular bone. Poorly compressed Play-Doh, which leaves air trapped in the deepest parts of the sulci, causes narrow radiolucent lines to be superimposed on the skeletal structures that are easily mistaken for fracture lines. Too much Play-Doh packed across the whole sole, rather than just in the sulci, increases the radiopacity of the foot too much, resulting in underexposure of the distal phalanx and disturbing superimposition of the edge of the Play-Doh packing over the solar margin of the distal phalanx. A poorly filled middle frog cleft causes the appearance of an often remarkably well-circumscribed radiolucency superimposed on the central part of the navicular bone. The best way to distinguish these frog lines from skeletal pathologic changes is to determine whether they can be seen to run across the skeletal outline of the bone concerned. A radiolucent line that continues outside the limits of a bone cannot be a fracture line.

The distal phalanx in this projection is characterized by the solar margin, the proximal and distal parts of the palmar process, the solar canal and its radiating vascular channels, the extensor process, the articular surface, and the DIP joint.

The solar margin is usually regular and smooth but can be slightly irregular in older horses. There may be a variably sized, V-shaped notch in the solar margin at the toe—the crena (crena margo solearis). This notch is usually present bilaterally and similar in size and shape in both front feet. The radiating vascular channels are variable in number and width and may widen close to the solar margin in normal horses. A small circumscribed osseous opacity palmar to the palmar processes is present in about 5% of normal horses. This may respresent a separate center of ossification or a fracture sustained early in life. If present, it is usually seen at both palmar processes in both feet. The diameter may vary from 1 to 7 mm. Clinically significant fractures of the palmar process tend to be larger. Some degree of ossification of the cartilages of the foot occurs commonly, especially in heavier types of horses. The normal depressions in the distal phalanx lateral and medial to the articular margins of the middle phalanx form the site of attachment of the collateral ligaments of the DIP joint. These depressions occasionally resemble a subchondral cystic lesion on high-contrast images.

The navicular bone derives its name from its appearance in this projection. Although it is normally boat-shaped, the presence of some asymmetry between the lateral and medial halves of normal navicular bones is not uncommon. The navicular bone acquires its adult shape around 18 months of age. The dorsopalmar outline of the navicular bone varies between animals, but contralateral symmetry should be present between both forelimbs.

The distal border of the navicular bone can only be assessed properly if it is projected proximally to the DIP joint. It is visualized as two horizontal lines, a proximal line reflecting the articular margin of the navicular bone with the distal phalanx and a more distal line representing the distal border of the ligamentous margin from which the impar ligament originates. A longitudinal groove runs in a lateromedial direction between both distal margins. This groove is the synovial fossa and it is lined with synovial membrane of the DIP joint. Several invaginations arise from the synovial fossa and course proximally into the navicular bone. Each invagination is lined by synovial membrane of the DIP joint and contains a nutrient artery entering the navicular bone in a subsynovial location. These invaginations can be seen as radiolucencies of variable shape and size along the distal border of the navicular bone and have variously been described as synovial channels or invaginations, canales sesamoidales, and nutrient foramina. Considerable debate exists about the normal appearance, size, and density of the synovial invaginations, but in reality these parameters vary so greatly among horses that some authors regard the appearance of these synovial channels as the normal fingerprint specific to each horse. The practice of grading each invagination according to its shape and size to decide whether a navicular bone is normal or abnormal is flawed, as so-called abnormal invaginations can be found in sound horses, whereas horses with chronic lameness associated with navicular pain may have only normal invaginations. Separate osseous fragments are frequently present at the angle between the horizontal and sloping parts of the distal border and are considered acceptable by some clinicians but not by this author. Their presence can be very hard to detect, even on good-quality radiographs. The presence of a small, crescent-shaped radiolucent line at the lateral or medial distal angle of the navicular bone is a strong indication that an osseous fragment is present. These fragments usually form as separate centers of ossification at the origin of the impar ligament. The crescent-shaped radiolucent line at the angle of the navicular bone is the base of the craterlike defect in the parent bone, adjacent to which the fragment resides.

The proximal border of the navicular bone should be sharply delineated and have a smooth contour. It is occasionally observed as two lines, one representing its dorsoproximal articular margin, the other its proximopalmar margin to which the navicular suspensory ligaments (ligamenta collateralia sesamoidale) attach. The proximal border of the navicular bone can be straight or have a concave or convex profile. Observations in Dutch Warmblood horses have suggested that navicular bones with a convex proximal border are at a higher risk of developing navicular disease.[2] The lateral and medial margins, or wings, of the navicular bone should have a smooth and regular contour. Although entheseophyte formation frequently disrupts the smooth contour of the proximal border and wings of the navicular bone in horses that do not show lameness, their presence cannot be considered normal.

Abnormal Findings

Radiolucencies in the laminar portion of the hoof wall can be seen around the contour of or distal to the solar margin in some cases of laminitis, white line disease, seedy toe, hoof wall separation, or subsolar abscessation. They are the result of air or fluid being trapped in this part of the foot as a consequence of infection or physical separation between the epidermal layers of the hoof wall. The solar margin itself may lose its sharply delineated, smooth contour on this projection for several reasons. Marginal chip fractures can result from abnormal loading of the distal phalanx after rotation associated with laminitis. For the same reason, laminitis or a flat-footed conformation with thin soles can also cause focal resorption of bone from the solar margin, resulting in an abnormally shaped distal phalanx. Other causes of focal bone lysis at the solar margin of the distal phalanx are the sustained pressure of a space-occupying mass such as a keratoma or the presence of localized infection. The outline of a pressure-induced area of bone resorption is usually characterized by its smooth, sharply delineated contour and the presence of a rim of reactive sclerosis. Resorption of bone caused by infection usually leaves an irregular, poorly defined edge without evidence of a sclerotic rim. Septic osteitis also often produces a sequestrum within the area of bone lysis, although the sequestrum itself may become resorbed with time. Another possible reason for the loss of smooth contour of the solar margin is peripheral widening and increase in number of the radiating vascular channels that arise from the solar canal. The diagnosis of pedal osteitis based on roughening of the solar margin and general loss of mineralization of the distal phalanx is controversial, but there is no doubt that the size and number of vascular channels can change with local bone inflammation (osteitis) in the foot. Many horses with flat, collapsed feet have abnormally shaped distal phalanges, but it remains unclear whether the abnormal shape of this bone is a cause of lameness in these horses. The presence of a focal radiolucency away from the solar margin in the distal

phalanx is usually evidence of a subchondral cystic lesion. Pseudocystic lesions in the distal phalanx can sometimes be seen on the upright projection in the region of insertion of the collateral ligaments. The normal collateral fossae in the proximal aspect of the distal phalanx adjacent to the lateral and medial margins of the DIP joint can become more radiolucent due to bone lysis associated with an insertional collateral desmitis. In these cases, reactive sclerosis is often present around the periphery of the affected fossa. Instead of resulting in a single localized radiolucency, collateral desmitis can also cause multiple small radiolucencies to appear in the area of transition between the central part and the palmar process of the distal phalanx. There are seven different types of fractures of the distal phalanx, most of which can be recognized on this projection. However, several other projections may be necessary to find a fracture line that is clinically suspected but cannot be visualized readily. In nondisplaced fractures, the fracture line may not be visible until 2 weeks after the fracture has occurred, when bone resorption along the fracture line results in sufficient demineralization to cause a change in radiopacity of the bone. Radiographic monitoring of fracture healing may be disappointing, as some fractures heal by a fibrous union that will ossify only very slowly or never completely. Navicular bone fractures almost invariably occur in the sagittal plane of the foot and are therefore best recognized on this projection. It is important to rule out the possibility of a frog line mimicking the presence of a navicular bone fracture by cross-referencing the fracture line on a flexor view and repacking the frog carefully. Changes in joint space width may be seen in advanced stages of osteoarthritis but may also be the result of uneven weight bearing.

The diagnosis of navicular bone disease has traditionally been based on the radiologic appearance of the synovial invaginations (nutrient foramina) along the distal border of the navicular bone in this projection. Navicular scoring systems based on the shape, size, and location of synovial invaginations

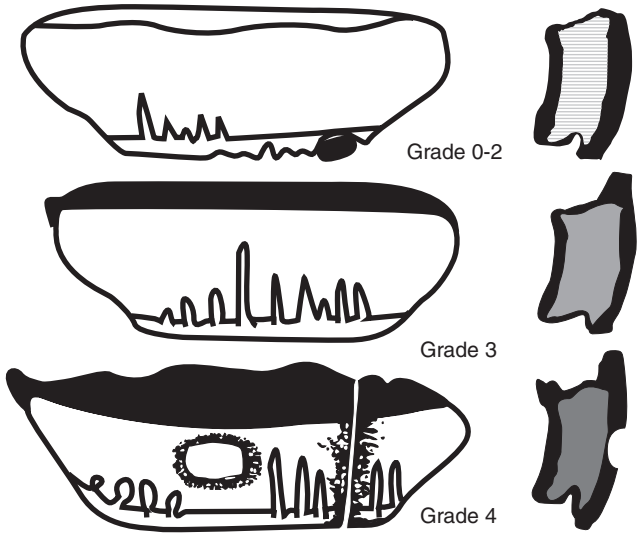

FIGURE 10-7 Schematic representation of the different grades based on the grading system.

are flawed because they focus on one (questionable) sign of disease only. Numerous studies have shown that "abnormal" synovial invaginations are poorly correlated with lameness, rarely progress in time, and are inconclusive for the diagnosis of navicular bone disease. Radiologic abnormalities more strongly associated with lameness in this projection are large medullary lucencies, medullary sclerosis, and proximal border remodeling. More successful navicular grading systems have been introduced that take a variety of radiologic signs into consideration. One popular standardized radiologic grading system used in the Netherlands assigns each navicular bone a grade from 0 to 4 (Figure 10-7 and Table 10-1).[2]

A low prevalence (15%) exists for navicular bones of grades 3 and 4 in normal horses, but a high prevalence (85%)

Table 10-1 Radiographic Classification of the Navicular Bone

Rights were not granted to include this table in electronic media.
Please refer to the printed publication.

From Dik KJ, Van den Broek J: Role of navicular bone shape in the pathogenesis of navicular disease: a radiological study, *Equine Vet J* 27:390-393, 1996.

Table 10-2 Summary of Importance of Radiologic Findings on the Dorsoproximal-Palmarodistal Oblique Projection of the Navicular Bone

Important Signs	Localized radiolucent area ("cyst") Extensive new bone formation proximal border, especially when unilateral Irregular trabecular structure Large distal border fragment(s)
Less Important Signs	Irregular distal border caused by increased or enlarged synovial invaginations (e.g., "cyst-shaped") Sharply delineated new bone at proximal border Entheseophytes at wings of navicular bone Asymmetric shape of navicular bone

From de Clercq T, Verschooten F, Ysebaert M: A comparison of the palmaroproximal-palmarodistal oblique view of the isolated navicular bone to other views, *Vet Radiol Ultrasound* 42:525-533, 2000.

for navicular bones of these same grades is present in lame horses. As a consequence of selecting out navicular bone grades 3 or 4 in breeding stallions the prevalence of these grades in the population of Dutch Warmblood horses has been significantly reduced in the last decade.

Other authors have used different assessment systems for evaluation of the navicular bone. One such system has been proposed by de Clercq et al.[3] and defines radiologic features of the navicular bone as being important, less important, or not important. To reach a diagnosis of navicular disease, the authors postulate that one important radiologic sign has to be present on any view, three less important signs on one view, or five less important signs on all three views. The importance of radiologic findings in the upright pedal projection of the navicular bone is summarized in Table 10-2.

Lateromedial Projection

Technique

The lateromedial projection (Figure 10-8) can be obtained in a lateromedial or mediolateral direction. The foot is elevated from ground level to include its bearing surface on the radiograph. This elevation can be achieved by positioning the foot in a weight-bearing stance on a flat block or in a non–weight-bearing stance on the navicular block. Assessment of foot balance is easier with weight-bearing radiographs.

The limb must be perpendicular to the ground and rotation of the foot on the block must be avoided. The central beam should be horizontal and directed tangential to both heel bulbs. It can either be centered on the navicular bone (i.e., 1 cm below the coronary band and midway between the most dorsal and most palmar aspects of the coronary band) or on the distal phalanx (i.e., on lateral aspect of the hoof, halfway between the dorsal and palmar aspects of the hoof wall and halfway between the coronary band and the solar margin). For accurate assessment of the foot in horses with laminitis, the central beam is best aimed at the level of the solar margin of the distal phalanx (i.e., 1 to 2 cm proximal to the solar margin of the hoof).

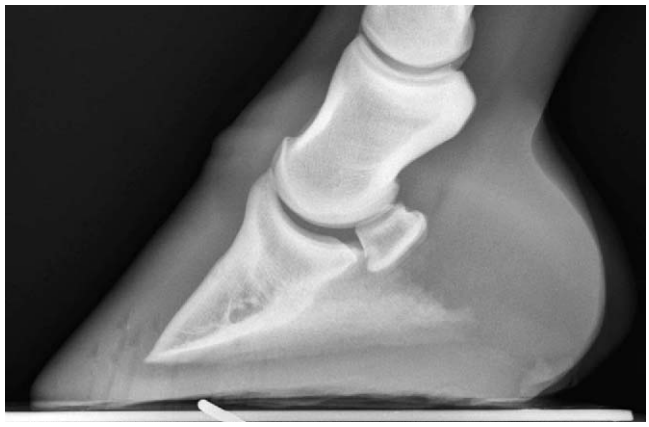

FIGURE 10-8 Normal lateromedial view.

To assess the relationship between the skeletal structures of the foot and the hoof capsule, it is useful to outline the hoof with radiopaque markers. A thin metal plate should be positioned between the sole and the upper surface of the block if the horse is unshod. A straight, stiff metal wire of known length should be attached to the dorsal hoof wall, such that the proximal end of the wire is level with the coronary band. Further metal markers are attached to the coronary band, in both the toe and heel regions. Finally, a thumbtack with a shortened point is placed in the frog, approximately 1 cm posterior to the point of the frog, to estimate where the DDFT inserts on the flexor surface of the distal phalanx.

Normal Variation

The distal phalanx has a smooth and opaque dorsal surface. The junction between the dorsal surface and the solar margin is sharply triangular, except in horses with a crena in which a radiolucent indentation can result in a double line. The solar margin of the distal phalanx should be more or less straight without pronounced concavity in its profile. It should make an angle of 5 to 10 degrees with the bearing surface of the foot, sloping down toward the toe. The terminal arch of the digital arteries runs through the solar canal of the distal phalanx. This is seen as a circular radiolucency of variable size midway between the distal interphalangeal joint and the solar surface of the bone. A small, circumscribed, osseous opacity palmar to the palmar processes of the distal phalanx is present in about 5% of normal horses. These fragments should not be confused with clinically significant fractures, which are usually larger and have a sharper outline. Some degree of ossification of the cartilages of the foot is common, especially in heavier types of horses. The cartilages may ossify from their origin on the palmar process of the distal phalanx proximally, or the ossification may originate within the center of the cartilage, progressing outward. This latter possibility may result in the presence of a permanent gap between the palmar process of the distal phalanx and the ossified cartilage, which should not be misinterpreted as a fractured sidebone. There are considerable variations in the shape of the extensor process, which can be round, pointed, or double pointed. The extensor process should be bilaterally symmetrical and a pointed or double pointed shape should not be confused with the presence of osteophytes or entheseophytes. A small circular radiopaque fragment is

sometimes present, proximal to the extensor process of the distal phalanx. The extensor process is the last part of the distal phalanx to ossify. Therefore these fragments can be separate centers of ossification or osteochondritis dissecans fragments, but they are rarely associated with lameness.

It is sometimes difficult to distinguish such fragments from true fracture fragments of the extensor process. If a fragment is round with a smooth outline, not associated with an obvious fracture defect and present in both forelimbs, it is more likely to be developmental in origin and not associated with lameness. Even so, some large separate centers of ossification at the extensor process can result in focal contact pressure against the dorsal surface of the middle phalanx during extension of the DIP joint and thus cause localized bone remodeling (exostosis and/or erosion).

Palmar to the solar canal is a sharply defined, smoothly delineated, relatively sclerotic band of bone, the facies flexoria (flexor surface) of the distal phalanx. This forms an area for insertion of the DDFT, which is usually straight or slightly concave.

The navicular bone can only be assessed on a perfect lateromedial projection without any evidence of obliquity. The bone should have a clean profile with a smooth outline. The proximal border of the bone should be straight without entheseophytes or osteophytes. Some proximodistal elongation of the bone has been considered a variation of normal.

The flexor surface can be seen as two separate proximodistal lines, the more palmar one representing the midsagittal palmar ridge of the bone, standing out slightly palmar to the main part of the flexor cortex. A normal, smoothly marginated depression or synovial fossa is seen in the middle third of the sagittal ridge as a small, smooth-edged defect in more than 50% of normal horses. A broad, radiopaque band of even thickness on the palmar aspect of the bone represents the dense layer of cortical bone in the flexor cortex. The flexor cortex tends to be thinner in horses with an upright foot conformation and in Thoroughbreds when compared to Warmbloods. Its thickness further seems to be determined by the age and the workload of the horse and should be symmetrical between contralateral limbs. Thinning of the flexor cortex may also be caused by focal fibrocartilage and subsequent bone loss. The medullary cavity is less radiopaque than the cortex and the trabeculae can be clearly seen, orientated in a linear fashion perpendicular to the flexor surface. The transition between the dense cortical bone and the cancellous bone in the medullary cavity should be sharply defined and similar in appearance to the corticomedullary transition in the proximopalmar aspect of the middle phalanx. If the corticomedullary transition in the navicular bone is not sharply outlined due to underexposure of the radiograph, it will be equally difficult to discern corticomedullary definition in the proximopalmar aspect of the middle phalanx.

The dorsal articular surface of the navicular bone is also visualized as two proximodistal lines, a more dorsal line outlining the convex part of the middle third of the dorsal articular surface of the navicular bone protruding in the concave intercondylar depression of the middle phalanx, and a more palmar line representing the flat dorsal surface of the lateral and medial aspects of the navicular bone, which articulate with the palmar surface of the lateral and medial condyles of the middle phalanx. The distal border of the navicular bone contains a groove (synovial fossa) that separates its dorsal half, which articulates with the palmar border of the distal phalanx, from its palmar half, which forms the ligamentous margin from which the impar ligament arises.

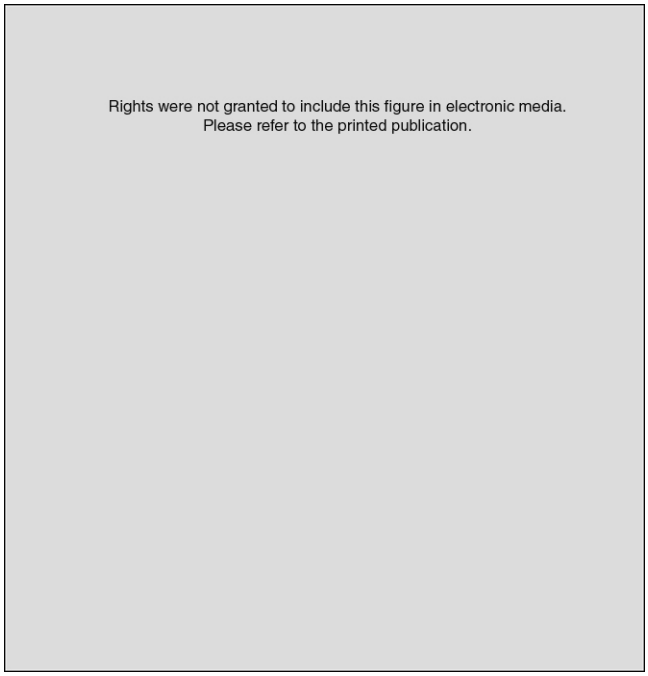

FIGURE 10-9 Schematic representation of the ideal alignment of the osseous and soft-tissue structures of the foot and the pastern in a well balanced foot. *(From Colles CM: Interpreting radiographs. I. The foot,* Equine Vet J *15:297-303, 1983.)*

The middle phalanx contains a small bony ridge on its distal dorsal aspect. This is usually smooth and regular in outline and forms the area of attachment for the collateral ligaments of the DIP joint.

The shape of the hoof capsule in the "ideal" foot is shown in Figure 10-9. There are three basic parameters to assess whether the foot is correctly balanced. The angle of the dorsal wall of the hoof with the ground should be the same as that of the dorsal aspect of the pastern when the horse is bearing weight on both forelimbs evenly. The dorsal aspect of the hoof wall should be parallel to the hoof wall at the heels. A perpendicular line taken from the center of a circle drawn through the DIP joint to the bearing surface should bisect the bearing surface of the foot (shoe). Although it is useful to examine each lateromedial foot radiograph with these parameters in mind, it would be unrealistic to expect every "normal" horse's foot conformation to comply with these rules.

Normal measurements have been identified for the distances between the various anatomical structures within the foot in horses and ponies.[4] Radiographic identification of the dorsal wall, coronary band, and solar surface of the foot is aided by the positioning of radiopaque markers, as described previously. The ratio between the width of the keratinized part of the dorsal hoof wall and the dermal laminae should be 1. The dorsal hoof wall thickness in normal Thoroughbreds should be 18 mm or less. The distance from the distal tip of the distal phalanx to the sole has been measured in normal Warmbloods as 21.0 ± 5.8 mm but is of course subject to the degree to which the foot has been trimmed. The normal

perpendicular distance from the coronary band to the extensor process in normal Warmbloods has been documented as being between 0 and 10 mm. The perpendicular distance from the coronary band to the proximal border of the navicular bone in normal Warmbloods also lies between 0 and 10 mm. The founder distance is defined as the vertical distance between the most proximal limit of the dorsal hoof wall and the proximal limit of the extensor process of the distal phalanx. It is measured by drawing two lines parallel to the ground surface on the radiograph, one through the top of the extensor process and one through the top of the dorsal hoof wall at the coronary band. The distance measured between these two lines is corrected for magnification by multiplying the measured distance by the ratio between the true length and the radiographic length of the dorsal wire. This distance has been shown not to exceed 10 mm in normal horses and ponies.[5] The degree of rotation of the distal phalanx is assessed by subtracting the angle between the dorsal hoof wall and the sole from the angle between the dorsal margin of the distal phalanx and the sole.

Abnormal Findings

Abnormalities of foot balance, of the relationship between the distal phalanx and the hoof capsule, of the DIP joint, and of the contour of the distal phalanx and navicular bones are best seen on this projection as long as a perfect lateromedial image is obtained. Dorsopalmar foot imbalance caused by a long toe/low heel conformation is the most commonly recognized imbalance. A perpendicular line dropped from the center of rotation of the DIP joint typically bisects the solar surface much closer to the heel than to the toe region of the foot in horses with this imbalance. In severe cases of dorsopalmar imbalance, the orientation of the solar surface of the distal phalanx is frequently reversed, with the heel region closer to the ground surface than the toe. This orientation is referred to as a *negative palmar angle* and is commonly associated with lameness. Displacement of the distal phalanx in a dorsopalmar plane relative to the hoof capsule is associated with severe laminitis and referred to as *rotation and sinking*. Rotation occurs when the parallel alignment between the dorsal hoof wall and the dorsal surface of the distal phalanx is lost due to disruption of the interlaminar bond between the dorsal hoof wall and the dorsal surface of the distal phalanx. If the interlaminar bond is lost around the entire circumference of the foot, the distal phalanx sinks distally within the hoof capsule. As rotation is not typically recognized in this instance, sinking may be hard to identify. Clues are gained from the presence of a palpable and visible depression between the skin and the proximal aspect of the hoof wall, mainly at the dorsal aspect of the foot, and from an increase in the measurable distance between the coronary band and the extensor process of the distal phalanx (founder distance). Because this distance may grow as the disease progresses, sequential radiographs are useful to monitor the effect of treatment on the stability of the distal phalanx. Occasionally radiolucent areas are seen between the sensitive and insensitive layers of the hoof wall as an indication of accumulation of air due to submural infection or physical separation of the hoof capsule.

Localized, poorly defined new bone production along the dorsal surface of the distal phalanx can be caused by laminar tearing or inflammation and is not always related to lameness. The shape of the sharply triangular distal tip of the distal phalanx is lost when sustained pressure from phalangeal rotation (laminitis) or from a space-occupying mass (keratoma) causes bone lysis. Focal bone lysis associated with keratoma typically extends proximally from the distal tip along the distal third to half of the dorsal surface of the distal phalanx. The resulting depression in the dorsal contour of the bone gives the distal phalanx a typical cloven-hoof appearance on the lateromedial projection. Phalangeal rotation can also result in marginal fractures of the tip of the distal phalanx that are visible on this projection. Localized bacterial osteitis from a penetrating wound or chronic subsolar abscess can also result in bone lysis in the region of the toe, but this is usually less sharply delineated with a more irregular margin to the defect.

Changes to the contour of the solar surface of the distal phalanx are seen as part of the "pedal osteitis complex" in flat-footed, thin-soled horses with recurring lameness caused by subsolar bruising. The solar profile of the distal phalanx may become excessively concave and its outline irregular rather than smooth, resulting in a misshapen distal phalanx. Focal contour changes may also be seen as small semicircular or concave depressions in the facies flexoria of the distal phalanx secondary to insertional enthesopathy of the DDFT or impar ligament.

Fractures of the palmar process of the distal phalanx are best seen on a lateromedial view but may take several weeks before becoming visible because of the delayed bone resorption of the fracture margins that occurs in the first few weeks after injury.

A diagnosis of osteoarthritis of the DIP joint is commonly based on the presence of osteophytes at the joint margins, the most prominent of which is the extensor process of the distal phalanx. However, the importance of radiologic changes of the extensor process is frequently hard to establish because of the inability of intra-articular anesthesia of the DIP joint to distinguish between navicular pain and pain arising from the joint itself. The presence of an isolated singular osteophyte on the extensor process can be of dubious importance. A certain diagnosis of osteoarthritis can only be made when accompanying osteophytes are also present at the other three sites where joint margins of the DIP joint can be assessed on this projection, the dorsodistal and palmarodistal margins of the middle phalanx and the dorsoproximal margin of the navicular bone. As always, it is important to distinguish entheseophytes from osteophytes in these locations. Periarticular spur formation away from the actual articular margin is not indicative of joint disease but of enthesopathy of selected soft tissue structures in the foot; entheseophytes can be seen at the level of the extensor process within the attachment of the common digital extensor tendon, at the proximal margin of the navicular bone within the attachment of the navicular suspensory ligaments, and along the dorsal surface of the middle phalanx within the joint capsule of the DIP joint. It is worth remembering that the attachment of the dorsal joint capsule of the DIP joint extends proximally along the dorsal surface of almost the entire middle phalanx to within a few millimeters of the proximal interphalangeal joint.

As long as no significant superimposition exists between the navicular bone and ossified ungular cartilage, the lateromedial projection is most sensitive for navicular bone abnormalities. Instead of focusing on the upright pedal projection, several authors have emphasized the greater importance of a good lateromedial projection of the navicular bone in the identification of navicular disease.[3,6] Table 10-3 lists radiologic

Table 10-3 Summary of Importance of Radiologic Findings on the Lateromedial Projection of the Navicular Bone

Important Signs	Localized, ill-defined radiolucent area in flexor cortex or spongiosa Medullary sclerosis Blurred flexor corticomedullary demarcation Irregular new bone at the proximal border Changes in shape resulting in enlarged proximodistal or dorsopalmar silhouette
Less Important Signs	Decreased opacity of the flexor cortex Thinning/thickening/uneven thickness cortex Sharply delineated new bone at proximal border
Pseudopathologic Signs (Anatomical Variant)	Sharply delineated synovial depression in middle third of the sagittal ridge

From de Clercq T, Verschooten F, Ysebaert M: A comparison of the palmaroproximal-palmarodistal oblique view of the isolated navicular bone to other views, *Vet Radiol Ultrasound* 42:525-533, 2000.

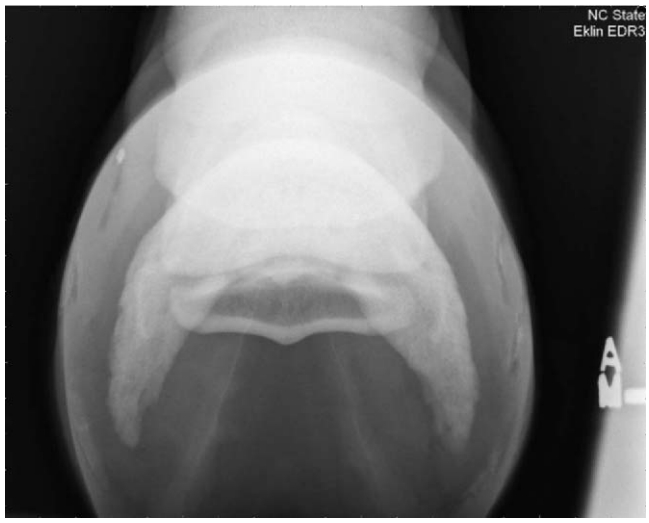

FIGURE 10-10 Normal palmaroproximal-palmarodistal oblique view.

features of the navicular bone on the lateromedial projection as important or less important for the diagnosis of navicular disease.

Palmar 45-Degree Proximal Palmarodistal Oblique Projection (Flexor or Skyline View)

Technique

The palmaroproximal-palmarodistal oblique view (Figure 10-10) is used to skyline the navicular bone and the palmar processes of the distal phalanx. The skyline view is obtained with the horse standing on a cassette-tunnel. The foot should be positioned as far caudally underneath the horse as possible while the horse is still fully bearing weight. The x-ray machine is positioned ventral to the patient's abdomen and the central beam is aimed between both heel bulbs at an angle of approximately 45 degrees. In horses with a low, sloping pastern, an angle of 35 to 40 degrees may be necessary to clear the region of the ergot, although this results in more distortion of the navicular image due to the lower angle between the central beam and the film. For horses with a low-heeled foot conformation and shallow angle of the navicular cortex, the use of a 15-degree angled cassette tunnel that elevates the heel of the foot by 15 degrees has been advocated to increase the angle of the navicular bone relative to the film. This helps to avoid angle-induced artifacts that result in loss of sharp distinction of the corticomedullary junction of the navicular bone.[7] Positive contrast bursography of the navicular bursa has been described using this projection.[8] The bursa is injected with 3 ml of a 1/1 mixture of a water-soluble contrast medium and mepivacaine hydrochloride and a radiograph is taken immediately after the injection. This technique highlights the flexor fibrocartilage of the navicular bone and the opposing dorsal surface of the DDFT.

Normal Variation

The skyline view projects each palmar process of the distal phalanx free of superimposing structures. The contour of these processes should be smooth and sharply delineated, but the abaxial border can be irregular in some horses without causing lameness. Focal irregular radiolucencies may be present in the palmar processes. A small, circumscribed, osseous opacity palmar to the palmar processes of the distal phalanx is present in about 5% of normal horses.

The navicular bone has separate radiodense palmar and dorsal cortices, separated by a radiolucent medullary cavity. The transition between each cortex and the medullary space is referred to as the *corticomedullary junction*. This junction is rather abrupt, with a sharp contrast between the dense cortical bone and the lucent cancellous bone. Both the dorsal (endosteal) and the palmar (cortical) outline of the flexor cortex should be sharply delineated, even in thickness throughout, and smooth without irregularities in the outline of the bone surface. Apparent loss of a sharp corticomedullary distinction can result from a poorly angled projection, especially in horses with low-heeled feet, or from superimposition of entheseophytes along the proximal border of the navicular bone onto the medullary cavity. The thickness of the flexor cortex can vary with the age and the workload of the horse and should be symmetrical between contralateral limbs. It should be noted that the cortical thickness can further vary significantly depending on the angle of the central beam relative to the proximodistal axis of the navicular bone. Imaging of isolated bones has indicated that slight changes in angulation of the central beam relative to the proximodistal axis of the navicular bone result in marked alterations in the thickness of the flexor cortex, loss of definition of medullary trabeculation, loss of sharpness of the corticomedullary delineation, and even perceived medullary sclerosis.

A small oval or crescent-shaped radiolucency is frequently seen within the triangular part of the sagittal ridge of the navicular bone. This radiolucency may be caused by either the presence of a small depression in the middle third of the sagittal ridge or, more likely, by incomplete mineralization of the flexor cortex in the proximal third of the sagittal ridge. In this location, the flexor cortex contains a central area of

reduced bone density, sandwiched between its palmar border and a more dorsal reinforcement line of compacted cancellous bone.[9] This pattern can be observed on MRI scans of the midsagittal part of the navicular bone in many horses. The synovial invaginations are visible as small circular radiolucencies, superimposed on the medullary space. With the use of positive contrast bursography, the flexor fibrocartilage of the navicular bone can be identified as a uniform radiolucent layer separating the contrast material from the palmar cortical part of the navicular bone.

Abnormal Findings

Abnormalities in the contour of the palmar processes of the distal phalanx are seen as part of the "pedal osteitis complex" and consist mainly of various degrees of irregularity in the abaxial outline of the bone. Palmar process fractures can occasionally be visualized most favorably with this view. For many clinicians, however, this view has become the favored projection for evaluation of the navicular bone. Dyson[10] reported that radiologic abnormalities of the navicular bone could only be detected on the flexor view in 78% of horses that were identified with a radiologic diagnosis of navicular bone disease. These radiologic abnormalities consisted of decreased corticomedullary definition; alteration of opacity, contour, or thickness of the flexor cortex; medullary sclerosis; disruption of the medullary trabecular pattern; and abnormal contour of the sagittal ridge. It must be remembered, however, that the flexor view is a very technique-sensitive projection, in which even a slight change in angulation of the central beam versus the navicular bone can mimic subtle radiologic abnormalities. The potential for overinterpretation of normal radiologic variation is real, and the clinical significance of many subtle abnormalities still needs to be substantiated. A comparative study of the navicular flexor cortex using radiography and computed tomography concluded that medullary sclerosis and poor corticomedullary junction were commonly overinterpreted radiologic features.[11] Therefore some radiologists maintain that the flexor view is rarely necessary to make an accurate diagnosis of navicular disease, because a combination of perfect lateromedial and dorsoproximal-palmarodistal oblique projections should deliver the same radiologic information.[3] In spite of this, the authors have attempted to define the importance of radiologic findings on the flexor view with relation to the diagnosis of navicular disease (Table 10-4).

Positive contrast bursography of the navicular bursa can identify four types of abnormalities on the flexor aspect of the navicular bone: thinning or erosions of the flexor fibrocartilage, focal loss of dye column as a result of DDFT adhesions to the bone, focal filling of the flexor cortical area due to the presence of cystic defects in the flexor subchondral bone, and filling defects along the bursal surface of the DDFT due to fibrillation of the tendon. Analysis of the results of this technique in 97 horses with palmar foot pain suggested that a flexor view after positive contrast bursography was able to identify radiologic abnormalities 60% more frequently than a flexor view with plain film radiography. Unfortunately, the technique does not have a high sensitivity or specificity for the presence of navicular pain, as normal fibrocartilage was more commonly found in horses with navicular pain.[8]

Table 10-4 Summary of Importance of Radiologic Findings on the Palmaroproximal-Palmarodistal Oblique Projection of the Navicular Bone

Important Signs	Localized radiolucent area in flexor cortex Medullary sclerosis Blurred flexor corticomedullary demarcation New bone formation at the articular surface
Less Important Signs	Decreased opacity of the flexor cortex Thinning/thickening/uneven thickness of cortex Interruption of smooth silhouette of flexor cortex
Pseudopathologic Signs (Anatomical Variant)	Sharp, crescentic lucency in sagittal ridge

From de Clercq T, Verschooten F, Ysebaert M: A comparison of the palmaroproximal-palmarodistal oblique view of the isolated navicular bone to other views, *Vet Radiol Ultrasound* 42:525-533, 2000.

Standing (Zero-degree) Dorsopalmar Projection

Technique

For the standing (zero-degree) dorsopalmar projection (Figure 10-11), the foot is placed in fully weight-bearing stance on a block elevating it from ground level. A vertical or sloping groove can be created in the block to hold the cassette in an upright position behind the foot. The cassette can be positioned vertically or aligned with the angle of the pastern, while the central beam is always kept perpendicular to the cassette. This allows assessment of joint space width in different parts of the DIP joint.

Normal Variation

Two distinct circular radiolucencies in the distal phalanx represent the end-on appearance of the solar canal. A small semicircular radiolucency in the lateral and medial surfaces of the distal phalanx is caused by the presence of the parietal groove, which accommodates the distal phalangeal artery and the distal continuation of the palmar digital nerve. The solar surface of the distal phalanx should be horizontal, indicating a similar distance between the ground surface and the lateral and medial solar margins. Consequently the DIP joint space should be oriented parallel with the ground and the joint space width should be symmetrical. This joint space is clearly wider than the joint spaces of the proximal interphalangeal and fetlock joints. The extensor process is enlarged and appears asymmetrical as it is superimposed on the distal aspect of the middle phalanx in this projection.

The navicular bone is mostly obscured in this projection, except for its proximolateral and proximomedial wings, which tend to be visible lateral and medial to the DIP joint space.

Abnormal Findings

Lateromedial instability of the distal phalanx within the hoof capsule can only be recognized on this projection. Typically the coronary band remains parallel to the ground surface but the solar margin of the distal phalanx and the DIP joint do not. In

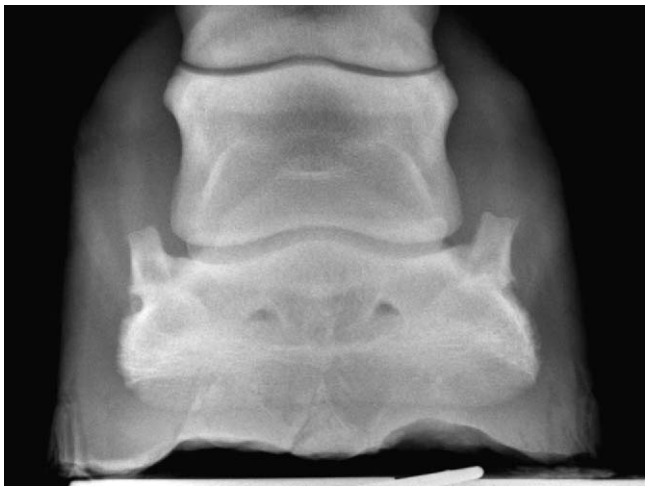

FIGURE 10-11 Normal standing dorsopalmar view.

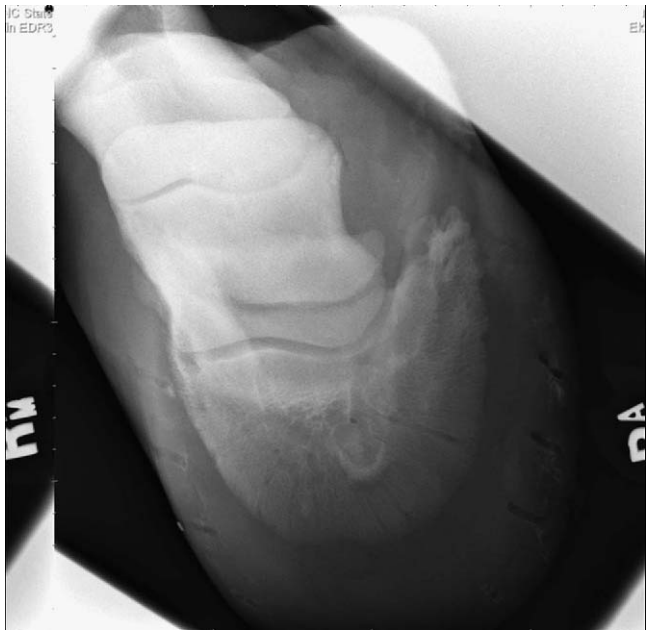

FIGURE 10-12 Normal dorsal, 60-degree proximal, 45-degree lateral palmarodistomedial oblique view.

extreme cases of white line disease or some cases of laminitis, the unsupported side of the distal phalanx moves distally so that the distance between the ground surface and the solar margin on the affected side is shorter than on the unaffected side. Sagittal or parasagittal fractures of the distal phalanx or navicular bone can be visualized satisfactorily on this projection without superimposition of radiolucent lines emanating from the frog or sole, which is useful to distinguish a real from an artifactual fracture line. Alterations or asymmetry in the width of the DIP joint space can also be seen, particularly in cases of advanced osteoarthritis with asymmetric loss of articular cartilage. Severe joint sprains with collateral ligament damage can result in asymmetric opening of the joint space on stress radiographs. For these radiographs, the joint should be alternately stressed laterally and medially by placing the opposite side of the foot on the block and leaving the side under scrutiny unsupported.

Comparison of the lateral and medial sides is necessary because stressing the joint in this fashion will always result in some unilateral widening of the joint space. In some cases of enthesopathy of the collateral ligaments of the DIP joint, remodeling changes (bone lysis, sclerosis, and/or cyst formation) occur at the level of the collateral fossa adjacent to the lateral and medial margins of the DIP joint, which is highlighted on this projection.

45-Degree Oblique Projections

Technique

Forty-five–degree oblique projections (Figure 10-12) can be obtained with the foot bearing weight on a cassette tunnel or with the foot supported in a navicular block. When the foot is supported in a navicular block, a horizontal beam is used, angled 45 degrees from dorsal (i.e., dorsal 45-degree lateral palmaromedial oblique projection) to highlight the palmar process of the distal phalanx, or angled 60 degrees from dorsal (i.e., dorsal 60-degree lateral palmaromedial oblique projection) to highlight the dorsolateral and dorsomedial margins of the DIP joint. When the foot is bearing weight on a protective cassette tunnel, the beam is moved 60 degrees proximally to the horizontal plane and angled 45 degrees from dorsal (dorsal 60-degree proximal 45-degree lateral palmarodistomedial oblique projection), to highlight the lateral palmar process of the distal phalanx. Both 45-degree oblique views are useful to project out a palmar process of the distal phalanx and the corresponding wing of the navicular bone palmar to the rest of the foot.

Normal Variation

The palmar process of the distal phalanx can be seen free of superimposing structures. The proximal and distal parts of the palmar process are separated by the parietal foramen that gives rise to parietal groove. This foramen can be completely surrounded by bone or can form an incomplete notch in the bone surface. Depending on whether the view is taken in flexion or extension, the extensor process is superimposed on the middle phalanx.

The lateral or medial wing of the navicular bone can be seen free of superimposition and appears rounded, smooth, and sharply delineated. The lateral or medial angle of the distal border with the corresponding sloping border should be just visible and should be continuous, rounded, and sharply delineated.

Abnormal Findings

These oblique projections are particularly useful for detection of palmar process fractures of the distal phalanx that may not be obvious in either the upright pedal or lateromedial projections. Other osseous abnormalities involving the palmar processes that are best seen on this projection include atypically located keratomas, septic osteitis after penetrating wounds, and osseous cyst–like lesions. Osseous fragments at the distal border of the navicular bone are easily missed on dorsoproximal palmarodistal oblique projections but should be consistently visible on the oblique projections as long as the abaxial part of the navicular bone is projected palmar to the middle phalanx. This also helps to identify entheseophytes arising from the attachment of the ipsilateral navicular

suspensory ligament to the abaxial part of the proximal border of the navicular bone. Although the presence of entheseophytes is definitely abnormal, their association with lameness has not been well established. Although most cases of osteoarthritis of the DIP joint can be diagnosed on lateromedial projections, occasionally the 60-degree oblique view of the flexed foot helps identify osteophytes at the dorsolateral and dorsomedial margins of the DIP joint in horses that have no obvious osteophyte formation at the level of the extensor process.

Equally, entheseophytes at the level of the insertion of the collateral ligaments of the DIP joint to the collateral fossa of the distal phalanx can be highlighted with the 60-degree oblique projection.

THE DIGITAL AGE AND RADIOLOGY OF THE FOOT

Dianne Little

Radiographic examination is an integral part of a complete evaluation of the foot. The advent of digital radiography and the improved versatility of computer technology currently available has led to an increase in the use of radiography for assessment of the relationship of the bony column to the soft tissue supporting structures, assessment of the response of the foot to manipulation of foot balance, and the ability to identify more subtle osseous lesions than previously possible. To maximize the benefits of digital radiography, to determine whether such a system has a role in an individual practice setting, and to minimize some of the pitfalls, it is essential to understand some of the advantages and disadvantages of the different systems available. Once a digital system is in place, a very rapid learning curve will occur both during use of the system and in interpretation of the increased amount of information visible within each image. An increased awareness of potential positioning artifacts and potential problems specific to digital radiography is essential to minimize some of the problems associated with integration of this system into an equine podiatry practice.

LIMITATIONS OF CONVENTIONAL RADIOGRAPHY

Use of conventional x-ray film requires access to facilities for correct storage, handling, and processing of film. Processing of film requires not only a processor but also facilities for handling and disposal of chemicals. X-ray film is bulky and occupies considerable space in storage and archiving systems. Considerable personnel time may be tied up in film archiving and attempted retrieval.

Conventional radiography is limited by a number of variables that can be manipulated to produce a diagnostic image of a region. For every image produced, a specific film-screen combination is chosen from the two or three possible combinations available within each clinical situation. The number of x-rays produced in a given time (mAs), the penetrating ability of the x-rays (kVp), and the distance from the focal spot of the x-ray tube to the beam (film focal distance) are then selected for the individual radiographic projection, tissue density, and region of interest. The film is then developed and fixed, usually according to a standardized automated protocol, and only then can the image be evaluated for diagnostic quality as well as for normalcy or pathology. Once the film is developed, there is no way of changing the image produced without repeating the entire process. Evaluation of an area of the radiograph that has poor contrast or that is underexposed or overexposed requires that a new image be produced, increasing radiation exposure to personnel. Subtle osseous lesions may be missed because, in the interests of economy and time, usually only one or two radiographic techniques are used for evaluation of each projection, unless subtle lesions are suspected at the time of the examination.

Comparison of progression of a radiographic abnormality may be difficult because of variation in radiographic technique or film-screen combinations between the two time points in conventional radiography. There is sometimes considerable delay between taking and viewing of the image, which may be a disadvantage in the field situation and intraoperatively. Sharing the images with other professionals and the owner requires that the original films or relatively expensive copied films be sent out from the practice, raising legal issues if original films are not returned, leaving the veterinarian with only the original radiographic report as evidence of findings and interpretation.

While these problems are not completely eliminated by digital radiography, they are much reduced. However, other variables that are frequently a source of frustration when trying to produce good-quality radiographs, such as limb positioning, preparation, and patient movement, remain the same whether digital or conventional radiography is used.

BASICS OF DIGITAL IMAGING

Digital imaging has become increasingly accessible in equine medicine with the advent and increased sophistication of a variety of modalities (ultrasonography, scintigraphy, magnetic resonance imaging, computed tomography). A digital image is composed of a precisely defined number of pixels arranged in rows and columns. Each pixel can only have one location in the image, because it has a predetermined value on the x-axis and y-axis of the image. Each pixel also has only one color or gray value, although this can be selected from a predefined number of gray-values or color levels. The number of possible colors or gray tones is referred to as the *image* or *bit depth*. Therefore, if the information about pixel location and bit depth can be transmitted, then the original image and original image quality can be reproduced anywhere. The number of pixels in a given row or column is the primary determinant of the special resolution of an image and is described in terms of pixels per centimeter or pixels per inch. These values must be as high as possible for very small details to be visible and to allow an image to be enlarged without each individual pixel becoming evident, known as *pixilation*. There comes a point, however, when increases in bit depth and numbers of pixels do not further enhance the diagnostic quality of the image but only increase the amount of storage space required for each file and the time required to transmit each file.

DIGITAL IMAGE CAPTURE

The most simple and cheapest means of digitizing a radiograph involves digital formatting of a standard radiographic film and is known as *secondary image capture*. This can be achieved in several ways. A digital camera can be used to take images of the x-ray film, and imaging of specific regions of interest within a radiograph is enhanced if optical rather than digital zoom lenses are used. Lighting is important, since ambient light creates reflections on the x-ray film that is evident on the digital image, and stray light from the x-ray viewer will interfere with exposure times. Photographs taken of radiographic film displayed on a fluorescent light viewer with standard color film or digital settings may appear to have a green/blue hue. This can be eliminated either at the time of taking the image, using a black/white film or program, or using camera software or filters to reduce this effect. It may be necessary to experiment with equipment available to produce the best image possible.

The second way of producing a digital image from a standard x-ray film is to use a flatbed scanner. Most films of the equine foot are of a size that would fit on any standard-sized scanner. Some form of back lighting is required, so if an overhead light source is not built into the scanner, then either a radiograph or transparency viewing box can be used.

The third way that a standard radiograph can be converted to digital format is through the use of dedicated radiograph scanners. The advantage of these over a flatbed scanner is that they can digitize radiographs of any size and into any of the three most common formats (JPEG, TIFF, or DICOM) used in digital radiography. Some are also able to scan multiple radiographs automatically. The quality of the scanner varies, with scanners having higher numbers of pixel image formats being the best quality and most expensive. Of the two scanner technologies, CCD (charged couple devices) are less expensive and now produce comparable image quality to the laser readers that were once the gold standard.

The primary disadvantage with any secondary image capture is that information is lost in the capture process. Therefore the quality of the digital image obtained is limited by the quality of the original radiograph and contains less information than the original radiograph. If information was not evident on the original radiograph because radiographic technique or processing was not appropriate for the region of interest, then no amount of manipulation of the digitized image will make this information available. In fact, because information is lost in digitization, less information will be available.

In *primary image capture,* once the x-ray beam has passed through the patient, the processes of image acquisition, processing, and display are isolated so that each process may be individually optimized to produce a variety of images, depending on the characteristics of the region of interest. This is in contrast to conventional radiography, in which, once the x-ray beam contacts the plate, the processes of image acquisition, processing, and display are linked inextricably by the combination of film, emulsion, screen, and processing used to produce a single image. In true digital radiography, once the x-ray beam has exited the patient, it is directly converted to a digital format using a combination of a photoconductor intensifying screen and active matrix array. The x-ray beam activates a photoconductor that then couples to the millions of semiconductors attached to a base substrate that makes up the active matrix array. The active matrix array is connected by a cable directly to a digital image processor and computer that controls operation of the array and is responsible for processing, display, and storage of images. The need for a cable can be a disadvantage in the field situation, where portability is important and potential for damage high, but these systems are becoming more robust. Improvements in wireless technology to allow transmission of large image files will eventually mean that the cable from these systems can be eliminated. The greatest advantage of digital radiography is the short image acquisition-to-preview time, which is somewhere between 3 and 10 seconds.

A second method of primary image capture is computed radiography. This technology is an indirect system, meaning that the image is recorded on reusable plates before being transferred to a computer. When the plates are exposed to x-ray photons, electrons on the storage phosphor plate become excited and store part of the energy to which they were exposed. The storage phosphor plate is read in a digital image reader, equivalent to the film processor, where the latent image is removed from the storage phosphor plate by scanning with laser light. Electrons in the phosphor plate return to their ground state by releasing visible light, which is detected and converted to an electrical signal in a photomultiplier tube. The stored image is then fully erased on the phosphor plate by exposing the plate to bright light. Computed radiography systems are much more robust for equine work than many of the direct digital radiography systems currently available, because the plates, although expensive to replace, are more robust than the digital radiography photoconductor and active matrix array. The time taken to view the image is slightly longer for computed radiography than for direct digital radiography because of the requirement for plate reading. Computed radiography systems are every bit as "field-friendly" as digital radiography systems, because compact mobile plate readers are available that are networked to a laptop computer and printer.

IMAGE MANAGEMENT AND POST-PROCESSING

In primary digital image capture systems, there is an almost linear association between the number of x-rays that arrive at the storage phosphor plate or the photoconductor/active matrix array and the electrical signal produced. This means that the signal produced from digital technology has a higher gray-scale resolution than film images and therefore allows visualization of very small differences in attenuation of the primary x-ray beam, which may not be visible with standard film-screen combinations. It is therefore possible to see both subtle osseous lesions or soft tissue and dense bone within the same image, albeit with some assistance from post-processing techniques to allow the human eye to perceive the information present. In addition, post-processing modification allows retrieval of an image obtained with suboptimal radiographic technique. Information captured digitally has less special resolution than that captured on most radiographic film, but the ability to magnify the image, apply edge enhance-

ment, and modify contrast and brightness allows maximal use of the information available and more than compensates for this reduction. There have been multiple studies in human radiography and several in veterinary radiography that demonstrate that images produced from computed or digital radiography are of comparable or better diagnostic image quality than conventional radiographs.

The amount of information stored on the active matrix array or on the storage phosphor plate is far greater than the amount of information than can be printed on film, displayed on monitors, or perceived by the human eye. Therefore a fraction of this information is extracted from the stored information for display during post-processing. The choice of what information to display is typically made through selection of an appropriate algorithm, based on the projection and anatomic area of interest. For example, display of data from a lateral radiograph of the foot of a horse with a suspected distal phalanx fracture requires that the gray levels available be distributed across bone, soft tissue, and gas, but for a lateral foot radiograph of a horse with laminitis, the distribution of gray levels across the dense bone component may be less important relative to the distribution through soft tissue and gas, so the distribution of the gray levels available can be changed in this instance to maximize the information available from the soft tissues. The information can be extracted from the same imaging plate, but two different algorithms can be applied, depending on the circumstance. This is analogous to taking two different conventional radiographs with two different radiographic techniques to evaluate different structures in the same anatomic region. Besides application of an appropriate algorithm prior to display, contrast enhancement, edge enhancement, unsharp masking, and combinations of these to optimize the image of the region of interest can also be applied, depending on an individual system.

The DICOM (Digital Imaging and Communications in Medicine) standard was developed to define protocols for storage, searching, retrieving, and printing digital images in human radiography. It was intended to ensure interoperability between equipment manufactured by different vendors, and it imposes strict requirements for the "header" information supplied with the image, such as patient name, case number and date of study, type of scan, and image dimensions. DICOM images are different in this respect than JPEG or TIFF images, in which this "header" information is not supplied. In addition, DICOM images have safeguards built in to prevent permanent alteration of an image, so that even though a DICOM image can be altered for viewing (rotation, magnification, change contrast and brightness), the original image cannot be altered. Changes made to the DICOM image (e.g., measurements) can be saved as JPEG or TIFF files, and therefore the only way of ensuring that unaltered original images of a specific study are being viewed is by using the DICOM image.

Currently, not every imaging device fully conforms to DICOM standards such that another device (such as a printer) can fully interoperate. To maximize the benefits of DICOM, it is important to maximize compliance of the different components of a system. Essentially, use of fully DICOM-compliant products should prevent a clinic from getting into a situation similar to having a half-Mac and half-PC formatted computer network and expecting the two networks to communicate with each other. The DICOM system itself is complicated, and expert technical advice should be sought prior to purchase of system components.

One important component for successful use of DICOM and the digital image is the ability to import and export images into a system and to other workstations and other Windows-based programs. eFilm (eFilm Medical, Toronto, Canada) is one such Windows-based product. This software allows the user to retrieve images from archives, magnify, change brightness and contrast of DICOM images, compare images from previous studies side-by-side, and manipulate brightness and contrast so that they are the same between two different sets of images for comparative purposes and it allows for distance and angle measurements. Manipulated images and measurements can be exported and saved as JPEG images for incorporation into discharge papers or reports to referring veterinarians and farriers. Measurements are simplified because the system automatically corrects for magnification, given a constant film focal distance.

Given that digital images will be viewed on computer monitors, the graphics card and monitor itself are obviously major potential limiting factors for viewing these high-resolution images. The most important parameters to consider for a monitor are the possible resolution, diagonal measurement, and brightness. The medical-grade high-resolution monitors (2K or 2000 × 2000 pixel matrix) are expensive but well worth the extra investment to optimize the advantages of digital radiography. Lower resolution (e.g., 1280 × 1024 pixels) monitors can be used, but for the full information in the image to be viewed, magnification functions should be used at sites of interest, to avoid missing subtle lesions.

IMAGE STORAGE

Medical digital images are large files, with high-resolution images up to 35 MB and standard images up to 4 to 6 MB. There are therefore advantages to compression of files to reduce the storage space required, and to reduce the time needed to retrieve and transmit such large files. Images can be subjected to lossless (reversible) or lossy (irreversible) compression for image transmission or storage. In lossless compression, using appropriate algorithms, there is no permanent alteration of original image information on retrieval, and the size of the file can be reduced by approximately half. On the other hand, in lossy compression there is some permanent alteration of digital image data and some loss of quality when the image is retrieved, but lossy compression can achieve data compression by the order of a power of 10. In all cases, whether lossy or lossless compression is used, there should be no loss of clinically significant data when the image is retrieved. The degree and type of compression used should be checked periodically to ensure that clinically significant information is retained. DICOM-compatible compression software is readily available to the veterinary profession. Back-up copies of all files should be made on a regular basis to provide both long-term archiving and disaster recovery. CDs can be used and will hold about 150 images each, but DVDs can each hold about 1600 images.

INTEGRATION OF DIGITAL IMAGING TECHNOLOGY WITHIN THE PRACTICE

Images from other modalities, such as gamma scintigraphy, sonography, computed tomography, magnetic resonance imaging, and thermography and digital pictures of a lesion can all be uploaded from a JPEG or TIFF format if necessary and converted to DICOM format, imported to a DICOM workstation, then linked to the digital or computed radiography system using a Picture Archive and Communication System (PACS). PACS allows efficient storage and retrieval of images and involves linking imaging devices, archiving systems, diagnostic workstations, and medical record software through a network capable of handling the anticipated level of "traffic." The choice of PACS is critical to the efficient functioning of the entire digital imaging capability within a clinic, and compatibility with all components of the digital imaging system is essential. Currently available systems for the veterinary market have limited capacity to integrate fully with medical record software but are capable of linking radiographic reports and stored images from all imaging modalities.

DISADVANTAGES OF DIGITAL RADIOGRAPHY

Although digital radiography offers many advantages over conventional radiography, as with any new technology there is a steep learning curve, and good technical support services are mandatory. The system itself is more costly than conventional systems, although it becomes readily affordable and economical when through-put is high. The cost-savings are generally maximized when the system is completely film-less, although in equine radiography, the benefits of convenience and reduced number of farm visits for retakes play a greater role in offsetting costs than in small animal radiography. A plan for implementing the system and integrating the system should be considered imperative to the success of the project, and each stage should be considered prior to starting the project. The systems are generally very attractive to clients and referring veterinarians, who can be provided copies of images at a fraction of the cost and inconvenience that occurs with conventional systems. The images produced display much more information than the practitioner may be accustomed to; therefore each individual reading films will have to re-establish his or her "normal" ranges for individual examinations and learn the new capabilities of the system. Accurate labeling and archiving are imperative, and good radiographic technique must still be maintained, as cost and time savings from reduced retakes of poorly exposed images will quickly be eliminated if patient positioning is poor. Indeed, because more subtle abnormalities can be identified and a greater variety of measurements made more accurately than with conventional radiography, patient positioning becomes even more critical.

COMPUTER SOFTWARE DESIGNED FOR EVALUATION OF EQUINE FEET

A recent development in radiography of the equine foot has been the Metron software (Eponatech, Calif.). This software package is designed for assessment and monitoring of change in balance of the equine foot over time using both selected radiographic and photographic projections. The software uses images (DICOM, JPEG, BMP, or TIFF format) of radiographs obtained by either primary or secondary image capture, with reference points marked on the foot at the time the original radiograph is taken. A marker of predetermined length is included in each radiograph or photograph to allow for correction of magnification and accurate measurement. A large number of measurements of the zero-degree dorsopalmar and lateral radiographic projections, and of the lateral, solar, and dorsal photographic projections of an individual foot can be archived with the complete radiographic series and photographic files for later reference and comparison. These measurements can also be printed as a report for a farrier, an owner, or the paper medical record. Once an image has been imported into Metron, a wavelet filter algorithm can be applied to optimize information available from radiographs. This algorithm increases perception of detail in regions of images that are relatively overexposed or underexposed. In addition, once an image has been imported into Metron, brightness and contrast can be manipulated to facilitate placement of measurement markers on the image and improve the accuracy of subsequent measurements. Each software upgrade of the Metron software has offered additional features such as integration of complete radiographic and sonographic studies into the archiving system, improved archiving and database features, and user-friendly movement around the program. Metron is also bundled with several digital and computed radiography systems specifically for the equine market.

Whether the Metron system or software similar to eFilm is used to generate measurements of the foot to guide farriers and veterinarians in treatment of foot imbalance and lameness, accurate and consistent positioning of the foot for radiography is critical. As the quality of radiographs has improved and more features are consistently evident on routine radiographs, the numbers of measurements that can be made routinely from radiographs have increased. Radiographic assessment of foot balance and the use of radiographs to determine the immediate effect of manipulation on foot balance has become more common and is a valuable aid to the traditional dynamic and static assessment of foot balance. However, as these measurements have become more sophisticated, it is important to remember that the radiograph represents a two-dimensional static picture of a three-dimensional dynamic object and that the relationship of structures within the foot to each other change with movement and loading, and hence with positioning of the foot for radiographs. This is particularly true when assessing measurements and angles associated with the joint spaces of the distal and proximal interphalangeal joint and navicular bone, where complex movement in three planes between the phalanges occurs during locomotion. A radiograph will capture these angles and measurements as a static measurement, but it is essential to know how the horse and foot was positioned for the radiograph before interpretation can be made. Three critical factors should be evaluated. First, the ground on which the horse is standing should be completely flat, as assessed with a level, or if unavoidable, the positioning of the horse on the slight slope should be such that the effects of a slight slope are minimized when

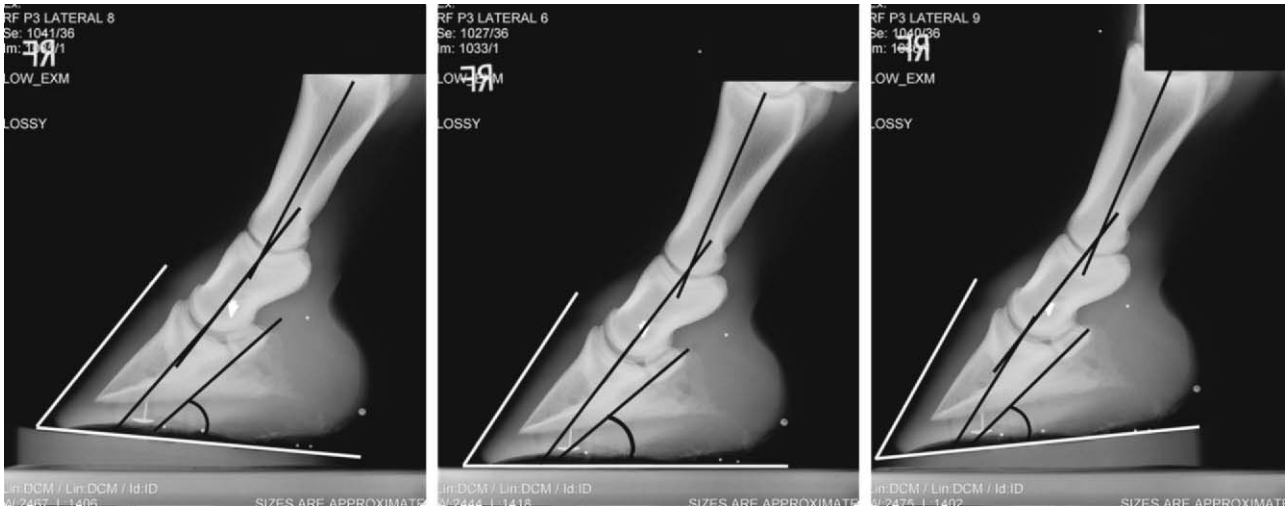

FIGURE 10-13 Lateromedial radiographs of the right front foot, demonstrating the apparent effect of a toe-up incline **(A),** level ground **(B),** and a heel-up incline **(C)** on hoof-pastern axis and angle of the navicular bone. The navicular angle is 42 degrees **(A),** 40 degrees **(B),** and 37 degrees **(C)** relative to the ground-line. The angle of the distal phalanx to the ground-line is 52 degrees in all three views. The angle of the middle phalanx to the distal phalanx is 8 degrees more upright in view **A,** 3 degrees more upright in view **B,** and 5 degrees less upright in view **C**. The angle of the proximal phalanx to the middle phalanx is 9 degrees more upright in view **A,** 9 degrees more upright in view **B,** and 12 degrees more upright in view **C**.

assessing the radiograph. If necessary, the horse should be moved between projections to minimize the effects of an incline on both zero-degree dorsopalmar and lateromedial projections if foot balance is being assessed. For example, an incline will affect the hoof-pastern axis and angle of the navicular bone in either direction on a lateromedial radiograph, depending on the direction of the slope (Figure 10-13).

Second, unlevel ground will also alter the relationship of the middle phalanx to the distal phalanx and may lead to apparent rotation or to artifactual narrowing of one side of the distal interphalangeal (DIP) joint in the zero-degree dorsopalmar view.

Third, the horse should stand completely square with each metatarsus or metacarpus perpendicular to the ground in both median and transverse planes, bearing equal weight on each limb of a pair (Figure 10-14). This will typically mean that all four feet need to be positioned on wooden blocks of equal height to allow the radiograph to be taken. It is also critical to evaluate the axis of the limb, digit, and foot before taking the zero-degree dorsopalmar radiograph, because aligning the vertical of the x-ray beam with the axis of the fetlock or pastern may result in an entirely different set of measurements to those of a radiograph in which the vertical of the x-ray machine is aligned with the axis of the hoof capsule. The x-ray beam should pass directly through the long axis of the foot, without being offset relative to the long axis or at an angle to the long axis, as this again will impose a degree of rotation on the radiographic image.

When assessing foot balance radiographically, it may be a useful illustration to take radiographs both with and without shoes on. The effect of the shoe on hoof-pastern axis, on base of support, and on breakover can be visualized and if necessary explained to the owner, veterinarian, or farrier (Figure 10-15).

MEDICAL AND LEGAL CONSIDERATIONS

Although not specific to digital imaging of the foot, at the time of writing, neither the American Veterinary Medical Association nor the Royal College of Veterinary Surgeons (United Kingdom) have any specific guidelines or regulations regarding digital radiography or other digital imaging modalities. The American College of Veterinary Radiologists (ACVR) is in the process of compiling guidelines for the use of digital imaging in veterinary medicine. The American College of Radiology (ACR), however, has published several technical standards that represent both policy statements and guidelines pertaining to several aspects of digital radiology in human medicine, and it is likely that the ACVR guidelines will be based on relevant aspects of these technical standards. Until such time as the veterinary governing bodies or specialty colleges produce similar guidelines, it is recommended that users of veterinary digital imaging systems be familiar with the parts of ACR technical standards that may relate to veterinary practice.[12,13] Several guidelines are outlined here that should be considered during data acquisition and image interpretation and when evaluating images that have been sent electronically for consultation or interpretation.

1. The image should contain sufficient information to allow interpretation to be made. This applies to both the original image and any copy images transmitted electronically to other parties. Compression of images (lossy and lossless) is frequently done to increase speed of transmission and decrease data storage space requirements. However, this compression should not result in loss of clinically significant image quality. Just as with interpretation of conventional radiographs, digital images should be

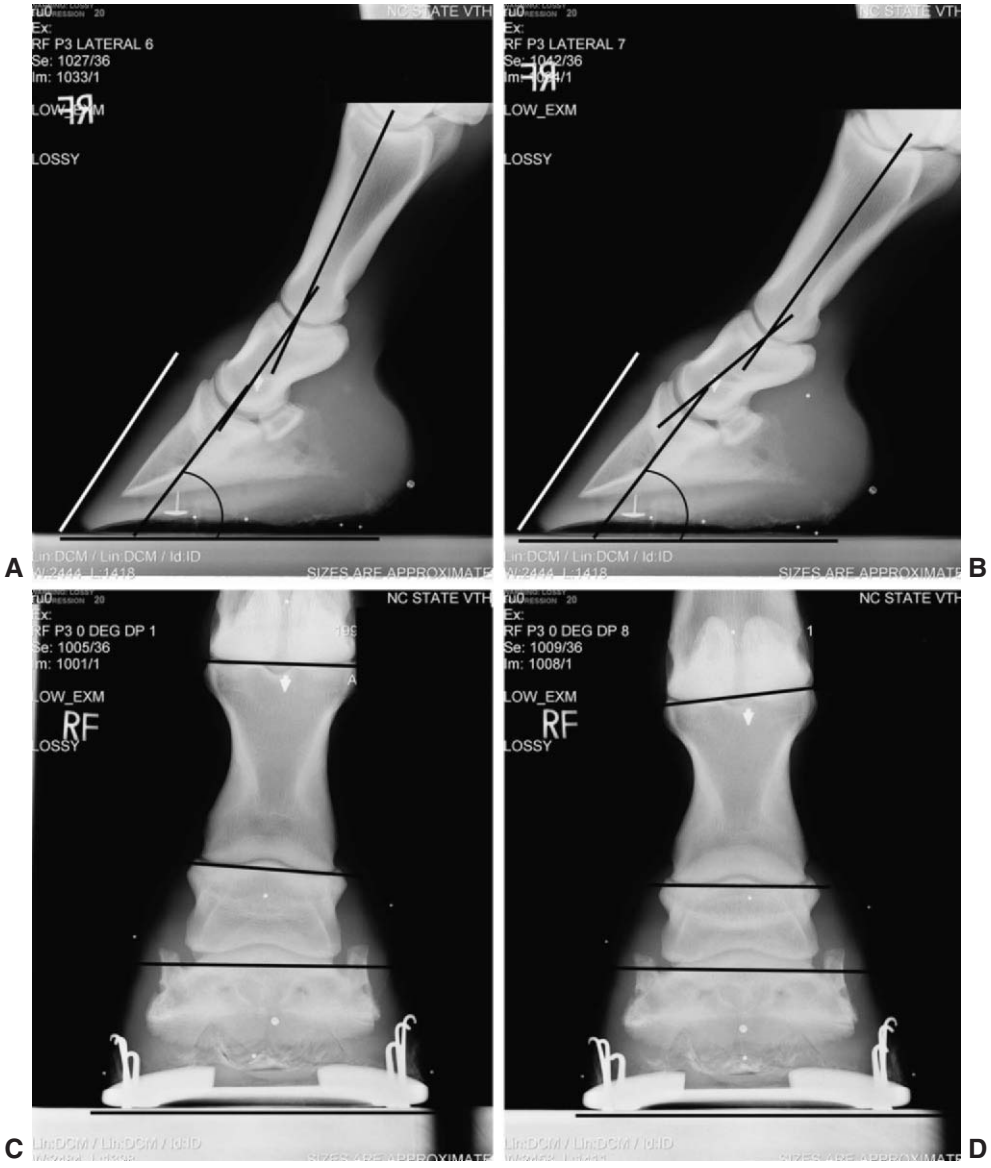

FIGURE 10-14 Lateromedial **(A, B)** and zero-degree dorsopalmar **(C, D)** radiographs of the right front foot with each front foot equally loaded **(A, C)** or with the left front foot held off the ground **(B, D),** demonstrating the effect of uneven limb loading on radiographic interpretation of alignment of the phalanges relative to each other. In the lateromedial views, the angle of the distal phalanx to the ground-line is 52 degrees **(A, B)**. The angle of the middle phalanx to the distal phalanx is 2 degrees more upright **(A)** in the equally loaded foot and becomes 10 degrees less upright when the foot is loaded more. The angle of the proximal phalanx to the middle phalanx is 12 degrees more upright **(A)** in the equally loaded foot and becomes 19 degrees more upright **(B)** when the foot is loaded more. In the dorsopalmar views, the medial aspect of the proximal interphalangeal (PIP) joint appears to be 3 degrees below the plane of the distal interphalangeal (DIP) joint **(C)** when both front feet are bearing equal weight. When the right front foot is loaded more **(D)**, the PIP joint appears to move into the same horizontal plane as the DIP joint. The medial aspect of the PIP joint appears to be 4 degrees below the plane of the fetlock joint during equal loading **(C),** but when the right front foot is loaded more **(D),** the medial aspect of the PIP joint appears to be 7 degrees below the plane of the fetlock joint. These changes in the joint angles on the dorsopalmar views are partially caused by rotation of the phalanges relative to each other during increased loading of a stationary foot and can be appreciated if the contours of the phalanges are compared to each other in views **C** and **D**.

assessed for diagnostic quality prior to interpretation. Temporary manipulation of images by altering contrast and brightness, similar to using a hot lamp can still be done to aid interpretation, but the original image should be retained in its original format (Figure 10-16).

2. At the time of image acquisition, the system must include annotation of patient and owner name, case number if available, date and time of examination, name of practice producing the image, and standard radiographic anatomic labeling (header information). Images are protected from changes to this information after the correct algorithm has been applied using the DICOM system. The amount and method of data compression will also be included.
3. Computer workstations used for image display should be of adequate quality to allow image interpretation and to allow display of previous image compression, processing, or cropping.

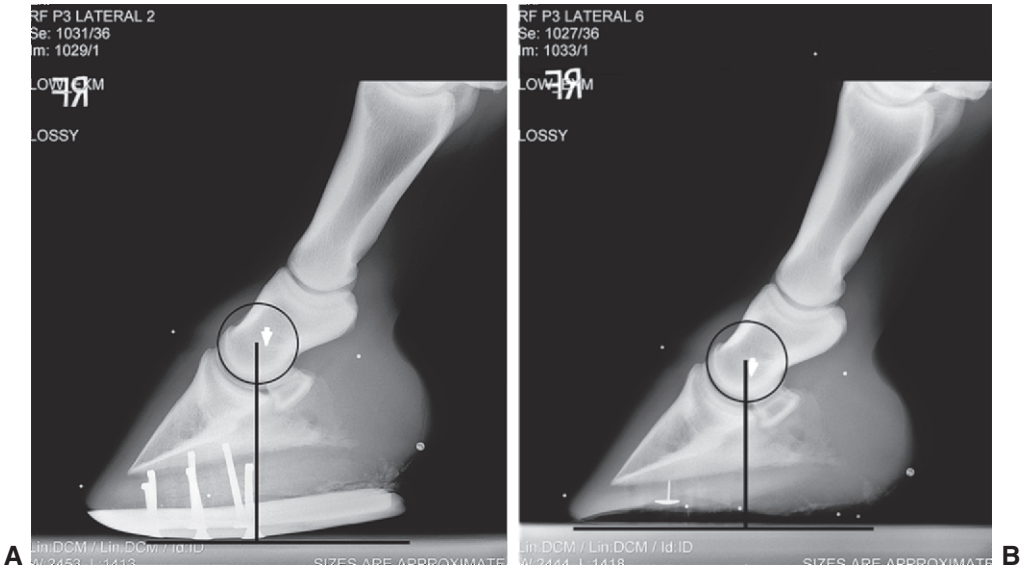

FIGURE 10-15 Lateromedial radiograph of the shod **(A)** and unshod **(B)** right front foot of the same horse demonstrating the effect of the shoe on assessment of foot balance. In the unshod foot **(B),** there is 7.4 cm of ground contact dorsal to a vertical line dropped from the center of rotation of the distal interphalangeal (DIP) joint to the ground surface and 4.6 cm palmar to the line. In the shod foot **(A),** the breakover is reduced to 4.5 cm dorsal to this line, and the base of heel support has increased to 5.8 cm palmar to this line.

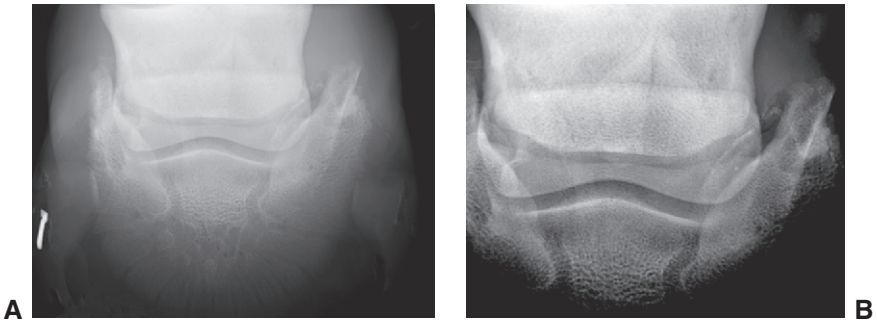

FIGURE 10-16 Dorsoproximal-palmarodistal oblique view of the navicular bone before **(A)** and after **(B)** temporary manipulation of the image contrast, size, and brightness to aid interpretation of the radiograph.

4. Images should be archived and stored securely to prevent accidental or deliberate alteration or access. Archiving should comply with medical record legal requirements of an individual state or country.
5. If teleradiology is used (electronic transmission of images to a distant site for interpretation or consultation), then the licensing requirements of the states or countries involved should be considered. The veterinarian performing the official interpretation is responsible for the quality of the images from which that interpretation is made. When a veterinarian receives images for consultation or interpretation, that individual should consider the source of the images, the annotation of the image, and the degree of compression and format of the image provided to determine whether fraudulent image manipulation may have occurred prior to electronic transmission. Although fraudulent radiographs may be obtained in conventional radiography, by deliberate misrepresentation of the subject or deliberate anatomic mislabeling, the possibilities for fraudulent manipulation of digital images are far greater (Figure 10-17). All the above criteria are readily met by complying with the DICOM standard adopted by the American College of Radiology.
6. DICOM (Digital Imaging and Communications in Medicine) is the standard medical image file format and transmission protocol for the electronic storage and transfer of medical images. This protocol allows the universal generation, storage, and transmission of medical images and is not modality or vendor specific. The protocol allows permanent identification of medical images and prevents the unauthorized electronic modification of such information. In addition, the protocol also prevents permanent modification of the original image file and allows for rapid retrieval and transmission of image data.
7. It is the author's opinion that DICOM should be the only medical image file format and transmission protocol endorsed by the veterinary profession as the *sole* image file storage and transmission protocol for veterinary images. While the author supports the dissemination of veterinary medical images for remote consultation, the use of proprietary (non-DICOM) medical image storage

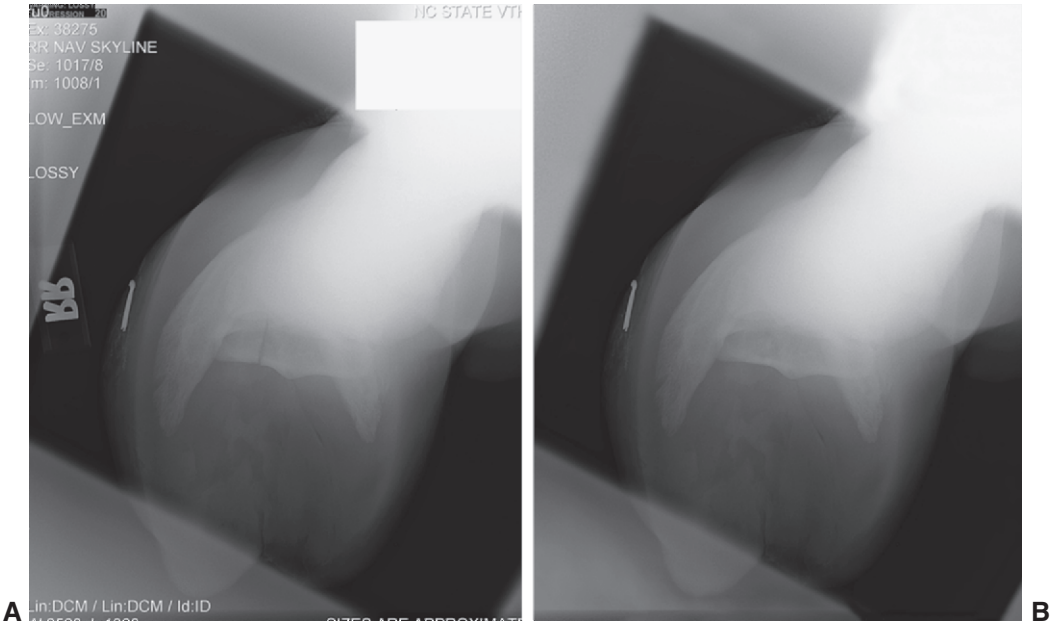

FIGURE 10-17 **A,** Palmaroproximal-palmarodistal oblique radiograph of a navicular bone fracture with appropriate DICOM annotation. Client information has been removed for publication to preserve confidentiality. Note the annotation "LOSSY" on the top left indicating lossy compression of the original image, without loss of clinically relevant information. **B,** The same radiograph after manipulation of the image as a JPEG file, demonstrating that the lesion can be substantially altered and the original annotation removed or altered for potentially fraudulent purposes.

and distribution software as the *only* format and transmission protocol available to a veterinary facility is discouraged.

8. Legal requirements for release of medical information apply to digital images the same way that they apply to conventional images, and if it is a requirement in an individual country, client consent should be obtained prior to electronic transmission of images. It is also important to remember that while images can be readily sent electronically all over the world in a matter of seconds for consultation, a bone fide veterinarian/client/patient relationship should always exist.
9. The original radiographic interpretation should be readily available with the original archived set of images. The report should be protected from alteration once it has been finalized or digitally signed and dated for two reasons: to protect the veterinarian making the interpretation from allegations of fraud and to protect the original document from alteration.
10. Even though digital radiography is still in its relative infancy in the field of equine medicine and surgery, anecdotal reports of fraudulent manipulation of digital radiographs during prepurchase examination already exist. Therefore it is recommended that, as with any prepurchase examination, particularly across international borders, potential purchasers and veterinarians performing the prepurchase examination establish a working relationship prior to the examination and, if possible, the purchaser or the purchaser's veterinarian be present at the time of the examination.

As the use of digital radiography becomes more common in equine practice around the world, differences may occur between countries as to what is and is not acceptable in a court of law. If readers are in any doubt as to the regulations in a given state or country, they should seek advice from their governing body or veterinary medical association.

NUCLEAR SCINTIGRAPHY OF THE EQUINE FOOT

Mark J. Martinelli

Nuclear scintigraphy is a metabolic imaging modality that has been used for more than 25 years in the diagnosis of lameness, particularly for lameness originating in the foot. Nuclear scintigraphy has been considered by researchers and clinicians to be a very sensitive, but not specific, imaging modality. This means that subtle alterations in bone remodeling can be effectively detected. This remodeling can be due to either direct bone change, such as bone resorption or deposition, or to alterations of bone associated with the attachment of soft tissue structures. Some of the early gamma cameras used for nuclear scintigraphy did not possess very good spatial resolution. In some of those cases, it was relatively easy to observe increased radiopharmaceutical uptake (IRU) in the foot but very difficult to determine whether a specific anatomical structure was involved. Once IRU was detected in the foot, radiography was used to determine whether any anatomical defects could be seen. In the mid-1990s, gamma camera technology and image acquisition software improved dramatically. The resolution of the image improved, providing more specificity within the bone scan and enabling clinicians to determine whether the IRU was related strictly to the navicular

bone, to the insertion of the deep digital flexor tendon (DDFT), or to the distal phalanx.[14] Once nuclear scintigraphy reached this level of technical competence, radiography became useful only as an adjunct imaging modality to look for obvious abnormalities, such as fractures, or for subtle changes in navicular bone density. The introduction of magnetic resonance imaging (MRI) of the equine foot has provided information about structures and disease processes that could only be speculated about previously. The information MRI provides has clarified, improved, and altered much of what equine veterinarians know or thought they knew about foot diseases, particularly how nuclear scintigraphy has aided equine clinicians in the diagnostic process. In spite of the additional information that MRI has provided, nuclear scintigraphy remains an important imaging modality for detecting and differentiating diseases and conditions of the equine foot.

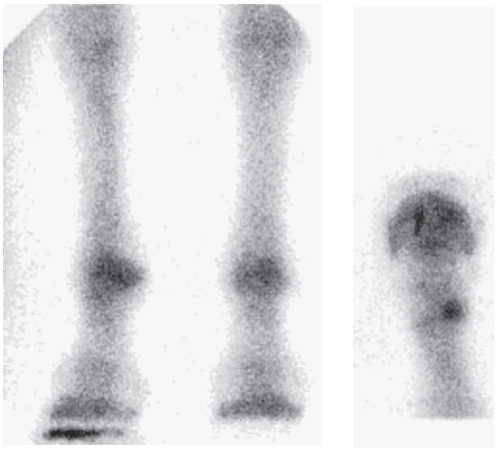

FIGURE 10-18 In these images, there is urine contamination of the right forefoot. On the dorsal view, the very dark line below the level of the sole of the distal phalanx is consistent with urine contamination of the sole. On the solar view, the linear region of increased radiopharmaceutical uptake could be confused with a lesion. Instead, the dorsal view suggested contamination, so the bottom of the foot was inspected. There was organic material in the lateral sulcus of the frog. Once this material was removed and the sole rinsed with RadiacWash, the "hotspot" on the solar view disappeared.

PROCEDURE

The imaging agent most commonly used for nuclear scintigraphy is a radiopharmaceutical made by labeling technetium-99m with a bone-seeking agent such as methylene diphosphonate. The dose published in the literature is 10 MBq/kg (0.3 mCi/kg), although no reference is made to whether the scan is of the entire skeletal system or just one part.[15] For a foot scan (partial body scan), the author's routine practice is to give a total dose of 150 mCi. This dose is slightly lower than the published dose for the average Warmblood breed but provides scans of excellent quality.

Vascular Phase Acquisition

The horse is tranquilized and positioned in front of the camera. It is important to make sure that both front feet are located within the field of view before injection. A lead shield is placed just behind the front feet to prevent any gamma rays coming from the hindlimbs from being detected. The software is set up for a dynamic acquisition, allowing for frame-by-frame analysis of the radioactivity. Most current software programs allow the user to determine the number of frames to be collected and the time duration of the acquisition frame. Typically for a dynamic study of the feet, each frame should be set for 3 seconds and there should be a minimum of 40 frames in the study. This will allow for an imaging period of 120 seconds, or 2 minutes. Because most software programs allow the user to stop the acquisition at any time, it is better to set the computer for more frames, such as 70, just in case an anomaly is detected or blood flow is impeded.

In most cases, the acquisition is started immediately upon injecting the horse because it will take 15 to 30 seconds (five to 10 frames) for the radioactivity to appear in the horse's limbs. If the injection is to be given by the same person responsible for starting the acquisition, then acquisition can be started before the injection so that no radioactivity flow is missed.

Soft-Tissue Phase Acquisition

At any time after the injection and lasting for 20 to 30 minutes, radioactivity is present in the soft tissues. For reasons relating to radiation safety, the soft-tissue phase acquisition should be limited to this very short window of time. Immediately after injection, the horse emits the highest levels of radioactivity, so exposure to personnel is also highest at that time. A short soft-tissue phase acquisition keeps that exposure to a minimum.

The typical views for a soft tissue phase study include a dorsal view of the front feet and at least one lateral view of each front foot. In cases in which lameness has been localized to one foot, the lame foot should be imaged first. That way, if there is any doubt about a "hot spot" being caused by time or a true lesion, a second lateral view of the lame foot can be acquired after the lateral view of the opposite limb has been acquired (i.e., lame foot, then opposite foot, and again lame foot). If a dynamic study has been performed using a computer that provides "motion correction" software, then the dynamic study can be registered or "corrected" to provide a soft tissue frame as well. Typically no solar view is acquired in the soft tissue phase, because the length of time required increases the exposure of personnel. In the soft tissue phase, each acquisition lasts for approximately 150,000 counts.

After the last acquisition of the soft tissue phase, the front feet must be covered with an impervious material to protect them from urine contamination before the bone phase is undertaken (Figure 10-18). If the horse urinates in the stall and then steps in the contaminated bedding, the resultant superficial radioactivity on the hoof can be very difficult to remove and can result in significant difficulty in interpreting the solar view. The impervious covering can be a commercially available product such as an Easyboot (Easycare, Inc., Tuscon, Ariz.) or an improvised foot cover such as an empty 3-L fluid bag. Once the feet have been covered, the horse is returned to the stall for approximately 2 hours to allow for soft tissue clearance of the radiopharmaceutical agent.

Bone Phase Acquisition

The horse is taken from the stall before the foot coverings are removed so that no radioactivity from the contaminated bedding adheres to the sole of the foot. The horse is tranquilized, and imaging commences. For the bone phase images, a static acquisition sequence is used. A routine study consists of five views of the front feet: a dorsal view with both front feet visible on the camera at the same time, a lateral view of each front foot, and a solar view of each foot. Although conditions for this are rarely available, if the camera can be placed completely below the ground surface, a solar view can be obtained of both front feet at the same time. This can be beneficial if there is a concern about whether one foot has a comparatively more active lesion ("hotter") than the other. Even if this view is attainable, however, a solar view taken of one foot at a time is recommended, so that the foot under scrutiny can be stretched out in front of the horse onto the face of the camera. Stretching the foot out as far as possible in front of the horse reduces the superimposition of skeletal structures further proximally up the limb over the foot within the field of view of the camera. This method aids in distinguishing IRU in the foot, particularly the navicular region, from that in the pastern or even the fetlock. To reduce the risk of superimposition further, the foot can be draped with a wrap-around piece of lead shielding, such as a thyroid shield.

The dorsal and lateral views are typically acquired for 200,000 to 250,000 counts, whereas the solar view is usually taken for 80,000 to 150,000 counts. The length of time allotted to the latter views is sometimes dependent on the temperament of the horse. In rare cases, the lameness present in one foot may prevent the horse from standing on that foot long enough for a diagnostic image of the contralateral foot to be acquired.

INTERPRETATION

In veterinary medicine, it is conventional to view the image as if the interpreter were looking at the horse. Therefore, the left front foot should be facing left and the right front foot should be facing right. When viewing the dorsal images, just as if the individual were facing the horse, the left front foot is to the right of the image and vice versa. With the solar views, it is often more difficult to remember how to determine the medial side from the lateral side of the foot. Because the image is acquired with the horse's foot on the camera face, the image is being viewed from the solar surface of the foot, as if the examiner were under the horse looking up at the sole of the foot. This means right and left are reversed again. One trick that can aid in remembering this concept is to view the solar image as if looking at the palm of the hand. When looking at the palm of the right hand, the thumb is medial and is to the right; likewise, medial on the solar view of the right forefoot is to the right of the image.

The terminology used for interpreting images describes two basic principles in the image, intensity and distribution. Distribution is usually expressed as either focal or diffuse. Intensity is often considered to be related to the severity of the lesion and so is more important. Because the entire scintigraphic image is based on the relative intensity of one pixel to another within any individual image, interpretation is also a subjective and relative exercise. Most of the newer acquisition software packages include analysis aids such as "region of interest," but their application is still somewhat subjective and is not covered in this chapter. Hence, most lesions are described as being associated with IRU that is mild, moderate, or intense. Finally, the structure or region involved with the IRU should be identified when possible, although this always remains speculative on nuclear medicine images. Therefore, a typical report may state that "there is focal and intense IRU associated with the region of the navicular bone" or "there is moderate, diffuse IRU in the region of the insertion of the DDFT."

To date, no studies have been conducted in the equine field that can substantiate with histopathologic findings what a region of IRU truly represents. This means that all interpretation in this area of veterinary imaging has been subject to speculation and relative confirmation over time and with the use of other imaging modalities. The recent introduction of MRI for the equine foot has provided more information and a better understanding of the pathologic conditions involved with IRU in the foot. Even so, new concepts and diagnoses continue to emerge.

Soft-Tissue Phase

The soft-tissue phase images can provide some very useful information to the equine clinician. More importantly, they can provide information that conveys a diagnosis for a case that has no IRU present in the bone phase.

When describing a soft-tissue scan, most imagers will label the hot spot as IRU, although it should be more accurately described as pooling of the radiopharmaceutical agent. The most common region with radiopharmaceutical pooling in the soft-tissue phase of distribution is the area of the distal phalanx. This radiopharmaceutical pooling is usually best observed on the lateral view, especially because the solar view is seldom obtained during the soft-tissue phase (Figure 10-19). There may be radiopharmaceutical pooling associated with the region of the DDFT palmar to the navicular bone (Figure 10-20) or early IRU associated with the navicular bone itself. On the dorsal view, there may be radiopharmaceutical pooling at the lateral or medial aspect of the coronary band, especially in comparison to the opposite side of the same foot or to the contralateral foot, seen as a vertical "stripe" of activity extending distally from one side of the coronary band (Figure 10-21).

Bone Phase

Once the radiopharmaceutical material has cleared from the soft tissues, imaging of the bone phase of radiopharmaceutical distribution takes place. In this phase it is accurate to describe the hot spot as IRU. There are several regions in which IRU is commonly observed.

Region of the Navicular Bone

Increased radiopharmaceutical uptake associated with the navicular bone can be seen on both the lateral and solar views. On the lateral view, it is observed as a round to elliptical spot of IRU in the middle of the foot, just palmar to the distal phalanx (Figure 10-22). On the solar view, the IRU can take on several different configurations but is most commonly seen as a round, more or less focal spot of IRU in

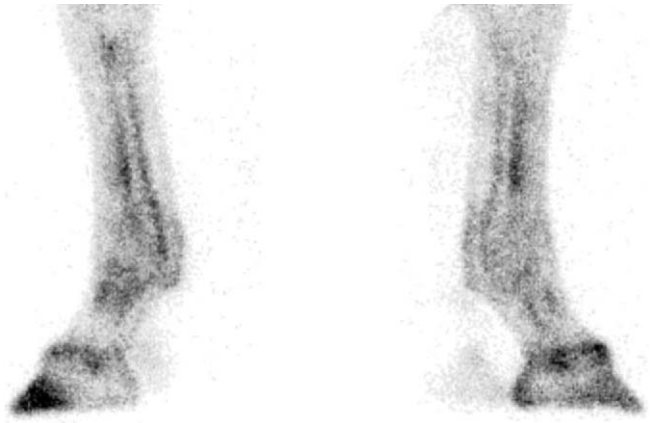

FIGURE 10-19 This is a soft-tissue phase of the front feet showing radiopharmaceutical pooling associated with the distal phalanx in the left forefoot compared to the normal right forefoot. If this area appears normal in the bone phase, this pattern of radiopharmaceutical pooling would be considered indicative of inflammation associated with the sole or lamina of the right forefoot.

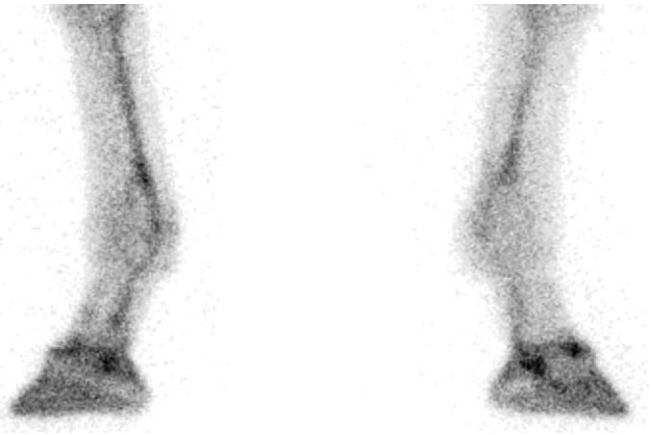

FIGURE 10-20 These two soft-tissue phase lateral images of the feet demonstrate a normal pattern *(left)* and radiopharmaceutical pooling associated with the deep digital flexor tendon (DDFT) *(right)*. Such radiopharmaceutical pooling often disappears in the bone phase, as this one did, resulting in a "normal" bone scan. In fact, if no soft tissue phase had been done in this case, the lesion would not have been detected at all. In this case, a subsequent magnetic resonance imaging scan demonstrated a lesion in the DDFT.

the sagittal midline region of the navicular bone. That is, a circular region of IRU toward the palmar aspect of the foot directly in the center as viewed from medial to lateral. In some cases, the IRU seems to extend all the way across from lateral to medial, over the entire the surface of the navicular bone, and is observed as a more linear region of IRU along the palmar aspect of the foot (Figure 10-23). In rare cases, the IRU appears to be associated with only the medial or lateral aspect of the navicular bone (Figure 10-24). The solar view is more sensitive at detecting IRU in the navicular region, probably because most of the IRU is located in the central part of the palmar aspect of the bone and this area is closer to the camera on the solar view than on the lateral view. For this reason, IRU associated with the navicular bone is considered most significant if it is observed on both the lateral and solar views.

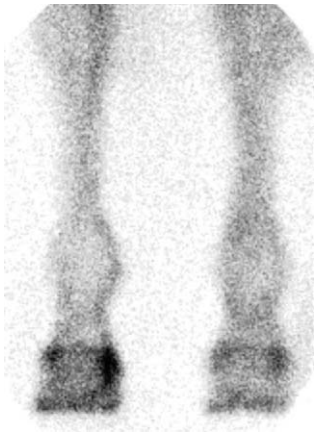

FIGURE 10-21 Dorsal view of the soft-tissue phase showing radiopharmaceutical pooling in a vertical pattern along the medial aspect of the right forelimb. Such a lesion in the soft-tissue phase is consistent with an acute collateral ligament injury.

FIGURE 10-22 In this case, there is focal and intense increased radiopharmaceutical uptake associated with the navicular bone on both the lateral and solar views. It should be noted that increased radiopharmaceutical uptake in the navicular bone is generally located mainly in the very center of the navicular bone, as fibrocartilage degeneration frequently starts near the center of the sagittal ridge.

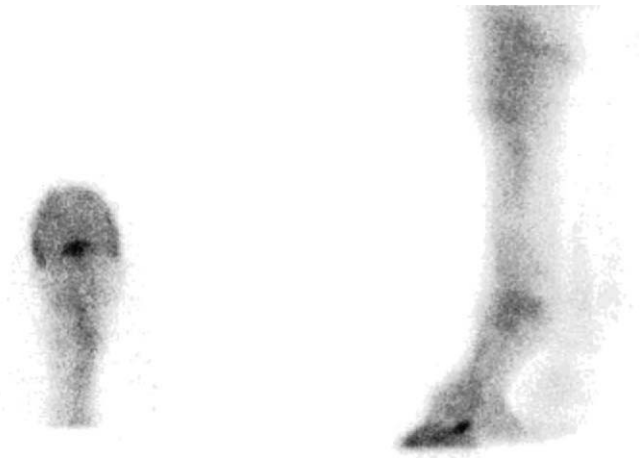

FIGURE 10-23 In this case, increased radiopharmaceutical uptake in the navicular bone is not limited to the center of the bone as seen on Figure 10-22. Such a generalized pattern of increased radiopharmaceutical uptake may be more likely associated with acute trauma of the bone.

Region of the Distal Phalanx

There are many different patterns of IRU associated with the distal phalanx and the soft tissue structures around it. On the lateral view, the IRU may involve the entire "triangular" outline of the distal phalanx, or it may be observed as a focal spot, usually in the center of the bone. Again, the solar view is usually most helpful in determining the true distribution of IRU. The pattern of IRU on the solar view usually falls into one of the following six categories: (1) it may outline the entire nonarticular margin of the distal phalanx; (2) it may outline only the lateral or medial palmar process of the distal phalanx (Figure 10-25); (3) it may run in a linear or semilunar pattern across the foot just dorsal to the midline (Figure 10-26); (4) it may form a focal spot at the site of the insertion of the lateral or medial collateral ligament (Figure 10-27); focal hot spots of this nature are found immediately adjacent to the lateral or medial articular margin of the distal interphalangeal joint, in the region of the ipsilateral quarter/palmar process of the distal phalanx; (5) it may form a focal spot at the site of the insertion of the DDFT, immediately dorsal to the navicular region. The pattern may be relatively focal or, more commonly, linear or curvilinear following the arc of insertion of the DDFT onto the palmar distal border of the distal phalanx (Figure 10-28); (6) other focal regions of IRU may be located anywhere in the distal phalanx (Figures 10-29 and 10-30).

CLINICAL IMPLICATIONS OF PATTERNS OF INCREASED RADIOPHARMACEUTICAL UPTAKE IN THE FOOT

It can not be sufficiently reiterated how important it is always to interpret the results of a nuclear scan in light of the results of regional anesthesia of the foot. IRU does not necessarily equate with pain and frequently reflects physiologic adaptation of bone to alterations in loading.

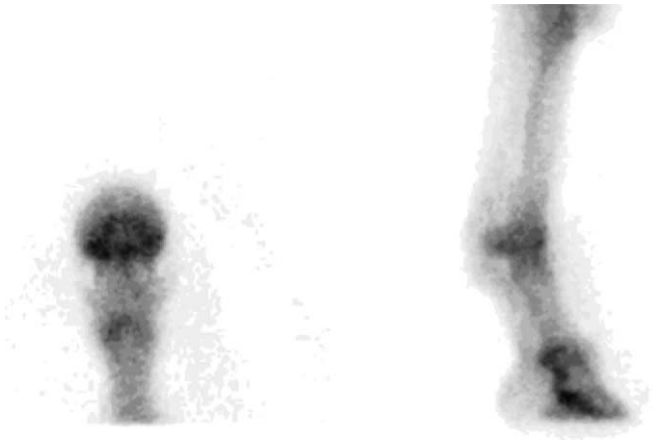

FIGURE 10-24 On the lateral view of this foot, the focal and intense increased radiopharmaceutical uptake seems to be associated with the navicular bone, in a distribution most typically seen in navicular disease. On the solar view, however, the focus of increased radiopharmaceutical uptake is located more on the lateral side of the navicular bone. Magnetic resonance imaging confirmed a lesion associated with the insertion of the lateral aspect of the impar ligament.

FIGURE 10-26 On the solar view, there is increased radiopharmaceutical uptake associated with both the navicular bone and the semilunar pattern outlining the deep digital flexor tendon insertion site on the distal phalanx. The lateral view highlights the soft tissue insertion much more than the navicular bone. There may be summation through the site of the soft tissue insertion on the lateral view, that is, a relatively greater area of increased radiopharmaceutical uptake when viewed from the side compared to the very focal region of increased radiopharmaceutical uptake in the center of the navicular bone. Alternatively, the region of increased radiopharmaceutical uptake in the soft tissues may be closer to the camera, that is, lateral on the foot, than the navicular bone, which is located more centrally.

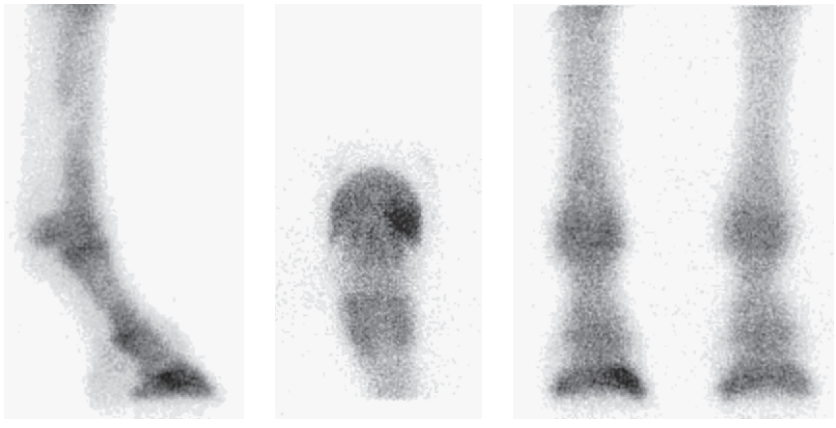

FIGURE 10-25 Three images of the right forefoot of a horse during the bone phase show increased radiopharmaceutical uptake associated with the entire medial wing of the right forefoot, consistent with an injury to the insertion of the medial collateral ligament.

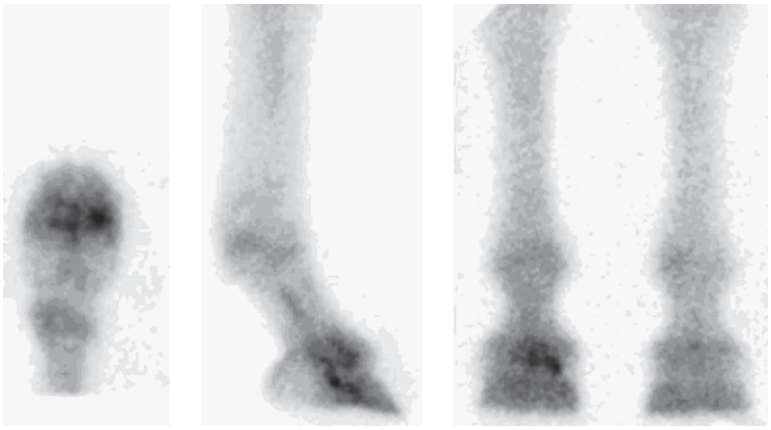

FIGURE 10-27 Bone phase images showing focal and intense increased radiopharmaceutical uptake associated with the insertion site of the medial collateral ligament in the right forefoot. Lesser increased radiopharmaceutical uptake is also present in the navicular bone.

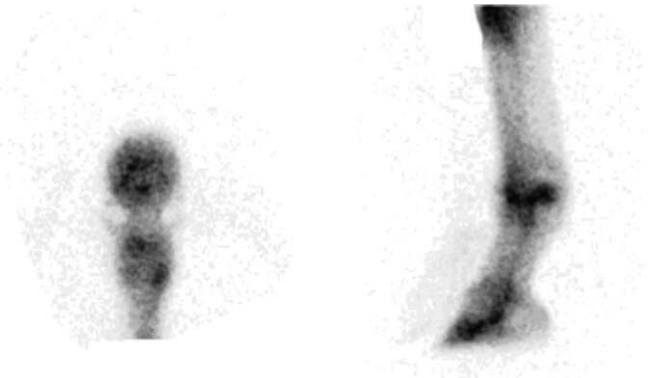

FIGURE 10-28 In this case, the lateral view suggests increased radiopharmaceutical uptake associated with the navicular bone and the insertion of the deep digital flexor tendon (DDFT). The solar view confirms the increased radiopharmaceutical uptake in the center of the navicular bone, but the increased radiopharmaceutical uptake associated with the soft tissue insertion site is only on the medial side of the foot.

FIGURE 10-30 Solar view showing focal and intense increased radiopharmaceutical uptake associated with the very tip of the wing of the distal phalanx. Such increased radiopharmaceutical uptake is considered to be too far palmar to be associated with the collateral ligament insertion. It is more likely related to the collateral cartilage or the palmar process of the distal phalanx. In most cases, especially in racehorses, such IRU is considered to be incidental and not associated with lameness.

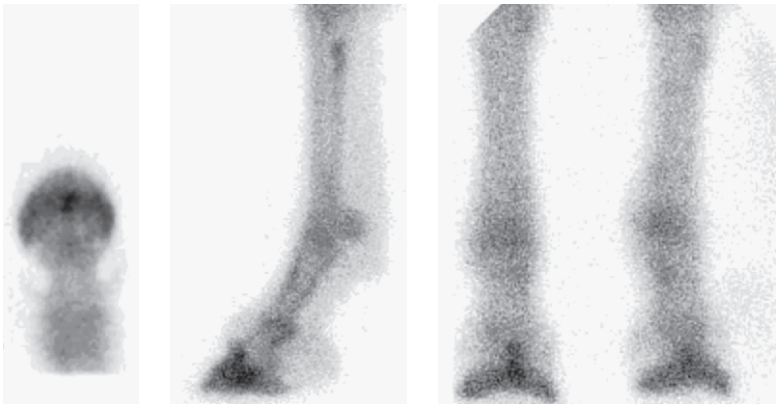

FIGURE 10-29 Three views of the right forefoot showing increased radiopharmaceutical uptake associated with the extensor process of the distal phalanx. Such focal and intense increased radiopharmaceutical uptake in that region should be investigated radiographically. This horse had a very large disunited extensor process fragment.

Region of the Navicular Bone

The solar view is the most sensitive at identifying IRU associated with the navicular bone. The most common pattern is a small, circular region of IRU in the middle of the foot or slightly toward the palmar aspect of the foot. This IRU is most frequently found directly on the sagittal midline. The intensity of the IRU is described as mild, moderate, or intense. In most cases, when the intensity increases, it becomes more likely that the IRU will be apparent on the lateral view as well. IRU associated with the navicular bone on both the solar and lateral views is considered to be consistent with navicular injury or disease (see Figures 10-22 and 10-23). When IRU is apparent only in the center of the foot on the solar view, this is more consistent with navicular bone remodeling, which can reflect either a physiologic adaptation process or a pathologic failure of adaptation (see Figure 10-26). The difference between a diagnosis of navicular bone remodeling and a diagnosis of navicular injury or disease is that navicular bone remodeling may be more likely to reverse itself or resolve when compared to the true disease process.

Dyson quantified the intensity of the IRU in this region relative to the radiopharmaceutical uptake in the distal phalanx.[16] Not surprisingly, she found a significant difference in radiopharmaceutical uptake in the region of the navicular bone between horses with foot lameness and horses without foot lameness. However, there was also a high incidence of IRU in the navicular region in horses without foot lameness that could be as high 30% to 40%. In another variation to the pattern of IRU associated with the navicular bone, although rarely observed, the IRU extends in a linear pattern along the length of the navicular bone as seen on the solar view. Most often, the IRU will also be seen on the lateral view. In this scenario, the IRU appears to be related to injury, possibly to the impar ligament origin at the distal margin of the navicular bone. Focal IRU associated with the more outlying lateral or medial aspects of the navicular bone (not situated on the sagittal midline), is very uncommon and likely to be caused by an enthesiopathy or injury to the attachments of the navicular suspensory ligament, the impar ligament, or the chondronavicular collateral ligament to the bone. Soft tissue phase abnormalities associated with the navicular bone are not common either but may be related to radiopharmaceutical pooling due to a soft tissue injury or to very early IRU most commonly associated with a severe case of navicular injury or disease.

The introduction of MRI into the imaging arsenal of the equine practitioner has shed new light on the significance of the classic pattern of IRU associated with the central region of the navicular bone. In the author's practice, three different patterns of magnetic resonance signal abnormality in the navicular bone have been identified, all of which appear to be indistinguishable from each other on scintigraphs. The first lesion is mainly observed on T1-weighted sequences and is characterized by structural osseous changes to the navicular bone that are not always evident radiographically. The second lesion consists of abnormal fluid accumulation only (physiologic change) in the medullary cavity of the navicular bone and is observed best on fat-suppressed sequences such as short-T1 inversion recovery (STIR). This fluid accumulation is thought to be associated with inflammation or edema and is unlikely to be visible radiographically. Finally, the pattern of central IRU in the navicular bone can be associated with apparent injury to the origin of the impar ligament. This injury is characterized by the presence of abnormal magnetic resonance signal in the navicular bone at the origin of the impar ligament on T1-weighted or fat-suppressed sequences and can rarely be confirmed with radiography unless an avulsion fracture is visible. Each of these magnetic resonance signal abnormalities seems to be associated with a different prognosis, but it is unlikely that nuclear scintigraphy will provide any more definitive information to allow the clinician to differentiate them from one another without MRI.

Although IRU associated with the navicular bone can be seen in horses of any discipline, some trends can be identified. Classic navicular disease, characterized by significant IRU noted on both the solar and lateral views, is most commonly observed in Quarterhorses, particularly those used for Western performance activities. When the IRU in the navicular region of sporthorses (i.e., Warmblood breeds) has been investigated with MRI, it is usually associated with inflammation in the medullary cavity or with injury to the impar ligament insertion. Racehorses, both Thoroughbred and Standardbred, have a relatively low incidence of IRU associated with the navicular bone unless there has been an injury or fracture. Fractures of the navicular bone are indistinguishable from severe navicular disease on the bone scan and must be confirmed radiographically. The clinical signs, however, are often suggestive of a more severe injury.

Region of the Deep Digital Flexor Tendon

Abnormalities associated with the DDFT are usually represented in one of two ways on a nuclear scan. In the soft tissue phase, there may be radiopharmaceutical pooling on the lateral view in a curvilinear pattern following the anatomical path of the DDFT as it crosses the navicular bone (see Figure 10-20). Although not very common, this pattern is considered to be indicative of an injury to that region of the DDFT. In very rare cases, there may be radiopharmaceutical pooling at that site in the soft tissue phase and no abnormalities in the bone phase at all. In the bone phase, the only abnormality associated with the DDFT appears to be IRU at its insertion onto the distal phalanx (see Figure 10-26). On the lateral view, the IRU can be seen as a relatively focal hot spot in the center of the distal phalanx. On the solar view, the IRU is dorsal to the navicular region but well palmar to the dorsal aspect of the distal phalanx. The pattern may be relatively focal or, more commonly, linear or curvilinear following the arc of insertion of the DDFT onto the palmar distal border of the distal phalanx. This pattern of IRU has been labeled as an enthesiopathy and is generally associated with a good prognosis for return to soundness. It is most often identified in sporthorses, such as jumpers, eventers, and dressage horses, although it can be seen in any discipline. According to Dyson,[16] IRU associated with the insertion of the DDFT is also frequently seen in horses without foot lameness.

Region of the Distal Phalanx

Several different patterns of IRU can be identified in the distal phalanx. These can be seen in either the soft tissue or the bone phase.

Diffuse Increased Radiopharmaceutical Uptake in the Distal Phalanx

The distribution for this abnormality on the lateral view involves radiopharmaceutical pooling or IRU outlining the entire silhouette of the distal phalanx, almost in the shape of a triangle (see Figure 10-19). In the solar view of the bone phase, the IRU forms a rim around the outer or distalmost border, the solar margin of the distal phalanx. Most often, it extends from one wing of the distal phalanx all the way around to the other. In some cases, the distribution of radiopharmaceutical material will be present in exactly the same pattern on the soft tissue and bone phases, whereas in others it will only be present in one phase and not the other.

If the radiopharmaceutical pooling outlines the distal phalanx in the soft tissue phase only with a normal distribution in the bone phase, then the condition is considered to be inflammatory and not likely related to any bone remodeling. The inflammation can be associated with different processes within the foot, and it is difficult to distinguish them from one another. The radiopharmaceutical pooling can be related to the sole, such as that seen in a thin-soled or poorly shod horse. It can be associated with the mural laminae, such as with an actual case of laminitis or in a horse that has laminar tearing due to trauma or overwork (i.e., road founder). Finally, the radiopharmaceutical pooling can be associated with the distal phalanx itself, in which case the term *pedal osteitis* may be appropriately applied. It is the opinion of the author, however, that radiopharmaceutical pooling associated with the distal phalanx is usually associated with one of the two former conditions and not with pedal osteitis. When there is diffuse IRU associated with the distal phalanx in the bone phase, the same three conditions are suspected, but a form of pedal osteitis is more likely. This is especially true if the IRU is noted in the nonlame foot, possibly because of overload compensation to spare the lame limb.

Region of the Distal Interphalangeal Collateral Ligament

Radiopharmaceutical pooling or IRU associated with the collateral ligaments of the distal interphalangeal (DIP) joint involves the distal phalanx but is a condition that should be discussed entirely on its own.

In the soft-tissue phase, radiopharmaceutical pooling may be noted on the dorsal view, most often medially, as a "hot stripe" running from the coronary band distally down the inside of the hoof wall (see Figure 10-21). Alternatively, the radiopharmaceutical pooling may be evident as a large hot spot just at the coronary band of the lame foot (medial or lateral). In some cases of collateral ligament damage that have been confirmed with MRI, there is radiopharmaceutical pooling in this region on the soft tissue phase but no IRU in the bone phase. When there is IRU associated with the collateral ligament in the bone phase, it is almost always at the insertion site on the distal phalanx and not at all associated with the origin on the middle phalanx. This is especially perplexing from an imaging standpoint, since many of these cases show signal changes in the ligament near its origin on the magnetic resonance images. More importantly, magnetic resonance signal increase is often seen on the T2-weighted and fat-suppressed images, indicating fluid accumulation and likely inflammation in that region. The pattern of IRU associated with the insertion of the collateral ligament on the distal phalanx appears as one of two distributions on the solar view. The IRU outlines the entire palmar process of the distal phalanx (see Figure 10-25) or just forms a focal, circular spot on the palmar process near the DIP joint. On the lateral view, the IRU is usually more difficult to distinguish from some other patterns because it appears as a circular region of IRU in the center of the distal phalanx. In these cases, in fact, the distribution may be similar to the pattern seen for IRU associated with the insertion of the DDFT, and these conditions must be differentiated with the solar view.

The pattern of IRU on the solar view that diffusely outlines the palmar process of the distal phalanx can be mild or intense. Recent cases confirmed with MRI have led to a more rigorous inspection of this region on the solar view of the bone phase for that reason. Diffuse and intense IRU associated with the entire palmar process emphatically points to that area of the foot but was frequently considered to be caused by an "occult" palmar process fracture. Investigation with MRI has confirmed that this pattern is most often associated with a collateral ligament insertional desmopathy. As with any region of intense IRU, however, a fracture must be ruled out. This is particularly true with racehorses of any breed.

Distal Phalangeal Fractures

As stated earlier, with any pattern of intense IRU seen on the bone phase of the distal phalanx, a fracture should be ruled out. In most cases of fracture of the distal phalanx, severe lameness would be present clinically, the fracture may be detected radiographically, and the case may never proceed to a bone scan. However, especially in the case of a hairline fracture that remains radiographically invisible, particularly in a racehorse, a bone scan may show intense IRU in the distal phalanx. In most cases, the pattern would be described as being somewhere between focal and diffuse but centering on the fracture line. If the fracture involves the palmar process, the IRU usually encompasses the entire process. If the fracture is anywhere within the body of the distal phalanx, the IRU is usually somewhat linear, extending from the solar margin of the distal phalanx proximally to the DIP joint. If these patterns are detected, particularly with a history or clinical signs consistent with a fracture, then a thorough radiographic examination focusing on the region of IRU is indicated.

Other Regions of the Distal Phalanx

Increased radiopharmaceutical uptake associated with the extensor process of the distal phalanx may be evident on the dorsal and lateral views (see Figure 10-29). It is most often associated with trauma, such as a chip fracture off the extensor process. Alternatively, it may be caused by a separate center of ossification at the extensor process. Because it is not very common, any IRU associated with the extensor process of the distal phalanx should be investigated radiographically. If there are clinical signs associated with this IRU, such as resolution of the lameness with a DIP joint block, then arthroscopic surgery is indicated.

Any focal IRU in the distal phalanx should be considered significant if lameness has been isolated to the foot with regional anesthesia. Such regions of IRU could be associated with the insertion of a soft tissue structure, the body of the distal phalanx, or the solar or articular margin of the distal phalanx. One such case had a focal and moderately intense

region of IRU near the DIP joint as seen on the solar view. MRI confirmed an articular defect in the distal phalanx that could not be seen radiographically.

CONTAMINATION

Once the radiopharmaceutical material has been injected intravenously, it begins to circulate throughout the body. Elimination is by decay and via the kidney into the urine. After the soft tissue phase, the feet should be cleaned of any organic debris and covered with an impervious material to limit exposure to any urine that the horse may excrete between the soft tissue and bone phases of the scan. Contamination of the foot can make interpretation of the solar view difficult to impossible, depending on where the radioactivity collects. If there is any doubt about whether the hot spot on the solar view is due to contamination, then the lateral view should be studied. Urine contamination will usually be visible as a line of radioactivity below the distal phalanx, but following the contour of the bottom of the hoof (see Figure 10-18). If urine contamination is present, the horse should be removed from the imaging area and decontaminated with a special solution (RadiacWash; Biodex Medical Systems, Shirley, N.Y.). The handler should wear latex gloves and liberally rinse the area with the solution while scrubbing gently. Any organic material still present in the foot will harbor the contaminated urine and should be carefully removed. Once the foot is clean and dry, acquisition of both the lateral and solar views should be repeated. If any contamination remains, the cleaning process should be repeated.

SUMMARY

Nuclear scintigraphy of the equine foot has come a long way since the first scans were performed in the 1970s. It has progressed even further since the first papers about scintigraphy of the foot were written in the mid-1990s. Nuclear scintigraphy presents information to the equine clinician that no other conventional imaging modality has been capable of providing. The equine industry has slowly come to realize over the last 25 years that there are many lesions and pathologic conditions causing lameness for which this metabolic imaging modality is required in order to convey a definitive diagnosis. The physical examination and gait analysis, including regional anesthesia, are always important aspects of the diagnostic process, but visual confirmation of the lesion, especially of one within the relatively inaccessible hoof wall, is invaluable to the lameness clinician. MRI will continue to refine the diagnostic process of the foot, providing further information about what each region of radiopharmaceutical pooling and IRU represents.

THERMOGRAPHY

Andrew P. Bathe

Infrared thermography pictorially represents the surface temperature of an object. It is a noninvasive method of detecting superficial inflammation and thus can have a role in lameness diagnosis. Although the technique has been used for a number of years, it is slowly gaining popularity as improvements in imaging quality yield images that are easier to interpret. It is a physiologic imaging modality, as is gamma scintigraphy, and thus has a lower reproducibility than anatomic imaging modalities such as radiography and ultrasonography. The foot and its blood flow represent a very dynamic system, so assessment of its physiology would be expected to yield considerable variability. This, along with the fact that thermography is prone to artifacts, has led some people to doubt its clinical applicability. With experience and care in interpretation, however, thermography can be a useful adjunct to the lameness evaluation of the foot, as part of an integrated clinical and imaging approach. Eddy et al.[17] reported on the use of thermography in 64 lame horses and found a 63% correlation between thermographic findings and those of ultrasonography, nuclear scintigraphy, and radiography.

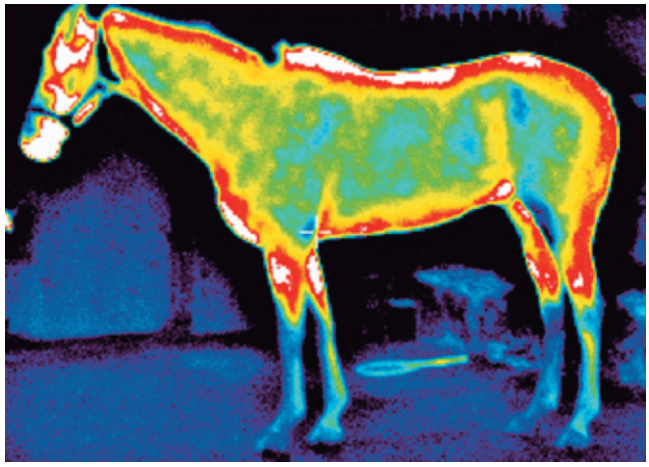

FIGURE 10-31 Lateral thermograph of a horse during an English summer showing thermal cut-off distal to the carpi and tarsi. This is a thermoregulatory response, in comparison to Figure 10-21, which shows the more normal pattern with hot coronary bands.

Heat is lost through the skin by radiation, convection, conduction, and evaporation. Thermal cameras assess infrared radiation from an object. This radiation is optically focused, collected, and transformed by detector arrays to an electronic signal that, in modern systems, then generates a real-time video image. The skin and hoof derive their heat from tissue metabolism and local circulation and as the former is generally constant, variation in superficial temperature normally relates to changes in local tissue perfusion. Radiation from the equine foot and limb is more significant than reflection, and ambient lighting levels are not a concern. Thermographic evaluation of the foot and distal limb is also complicated by the thermoregulatory role of the distal limb, whereby the blood supply can be dramatically reduced to conserve heat in cold conditions[18] (Figure 10-31). Thus an understanding of the physiology of the distal limb is essential for optimal image interpretation.

A number of different thermal imaging systems are available. Older systems were primarily developed for military or industrial applications, are relatively cumbersome, and have poor image quality. The top-end systems have a cooling mechanism to ensure temperature stability of the detector, which increases the fragility of the system and the maintenance

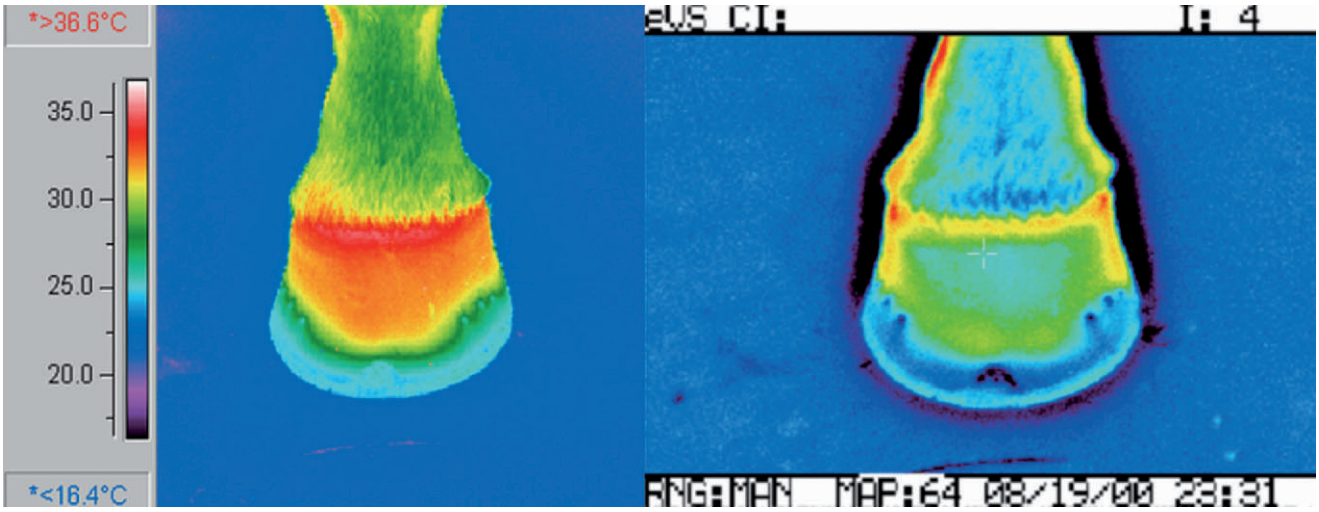

FIGURE 10-32 Comparison images of the same foot imaged at the same time with two different camera systems. The image on the left is obtained with a top-end Thermacam radiometer (Inframetrics, North Billerica, Mass.) and represents an accurate thermal image. In comparison, the image on the right was obtained with a DTS 500 (Emerge Vision Systems, Sebastian, Fla.). This is a relatively inexpensive and widely used system that generates a very different thermal image. Note the color scale to the left of the figure, demonstrating that the rainbow palette used in all these images ranges from showing hot as white and red to cool as blue, purple, and then black.

costs. Uncooled camera technology is preferable in a veterinary environment, as it is cheaper, lighter, and more robust. These uncooled systems have become more available recently and the system cost for diagnostic-quality thermographic imaging is no longer prohibitive. A wide variation still exists in the image quality between different systems. Figure 10-32 demonstrates such variation in an image of the same foot taken at the same time with two different systems. The image on the right from this particular uncooled system contains artifacts induced by post-processing image manipulation, which gives the impression of a temperature increase around the margin of the limb. In comparison, the reference image from a cooled system on the left demonstrates a more accurate representation of the true thermal pattern. It is very difficult to compare between these different systems, but the user becomes accustomed to the different thermal patterns produced by each machine. Some systems are also radiometers and will allow an accurate temperature measurement to be obtained, whereas other systems may not measure the absolute temperature but be able to determine the difference between two areas within the same image. Although a radiometer is not essential, it does assist in reproducibility between different examinations. Higher image quality leads to a greater ease of interpretation of the image. The quality of image processing software is also improving, but the majority of interpretation is carried out in real time. Images can be stored in a variety of digital formats for archiving and later comparison. Infrared thermographic instrumentation is far more sensitive than human hands in detecting temperature changes in an object. There may be variability of up to 1° C due to the camera in clinical imaging,[19] but differences in excess of 1° C are normally considered potentially significant in image interpretation.

IMAGE ACQUISITION

The feet should be clean for thermographic imaging of the digit and should be picked out and brushed to remove external contamination. If the soles are uneven or overgrown, then they may need to be lightly pared. Areas of thick, undermined sole will appear cooler and potentially hide the underlying heat pattern. Preferably the feet should be dry, but images can be obtained from wet feet, so long as the degree of moisture is uniform and images are given time to equilibrate. A long hair coat acts as an insulator, but this is less of a problem for the hoof than for the distal limb. Imaging should be performed in a relatively bare room without radiant heat sources. Some authors recommend allowing the horse to equilibrate thermally in the environment that it is to be imaged in. Equilibration can take up to 1 hour, but although the absolute temperature changes, there is little change in the relative thermal pattern.[19] Thus equilibration is not necessary for clinical imaging. The optimal ambient temperature for imaging is 20° to 25° C. At temperatures below this level, the distal limbs are more prone to thermal cut-off,[18,20] and above this range the contrast between the horse and the background is lost. Thus in many climates it can be advantageous to have a "hot box" to raise the ambient temperature if thermal cut-off prevents diagnostic imaging. It can also be advantageous to image the feet after the horse has been trotted and lunged, which increases the blood supply to the limbs and hence increases the inherent contrast of the digit relative to the background. Conditions of the front feet are more common, and imaging is normally concentrated here, although the hindfeet can provide a useful comparison in bilateral conditions. The distal limb should be imaged from dorsal, palmar/plantar, and left and right sides. The limbs are then lifted and a solar view is obtained of each foot (Figure 10-33). Close-up images of regions of interest can then be obtained if necessary.

It is normally most intuitive to image using a rainbow color palate with as great a range of color depth as the system will allow. Because of the variability in the absolute temperature of the distal limb, the thermal range should be adjusted for each individual horse, rather than a set figure being used. The coronary band is normally the hottest area within the

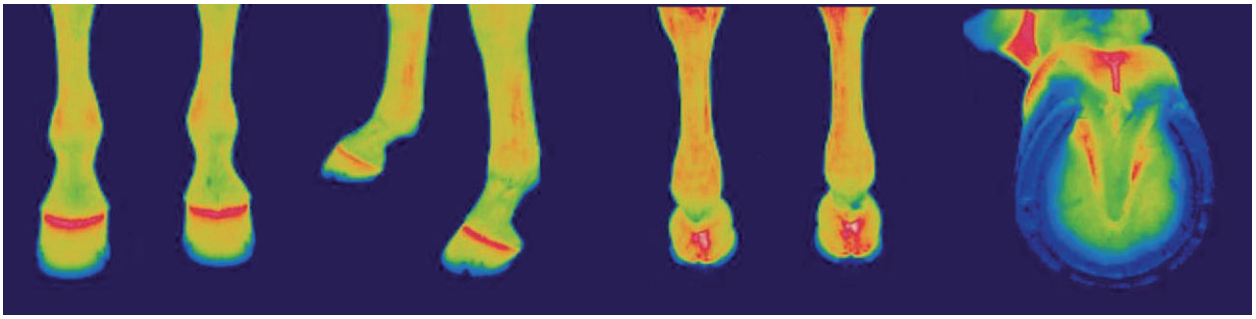

FIGURE 10-33 Set of normal images of the distal limbs of a horse showing dorsal, left lateral, palmar, and solar projections. These images show good symmetry between the limbs. Note that the coronary band is the warmest area and the rest of the hoof then gets colder in a regular pattern the nearer it is to the ground surface. The palmar view shows a normal increase in heat between the heel bulbs. On the solar view there is a V-shaped pattern of increased heat representing the sulci of the frog.

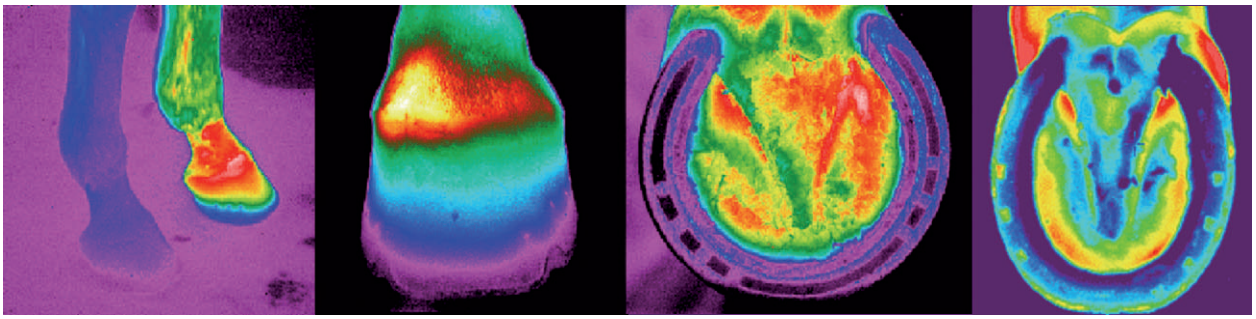

FIGURE 10-34 **A,** A subsolar abscess in the left forefoot has caused a massive increase in heat within the foot and distal limb, in comparison to the right forefoot, which has thermoregulatory cut-off. **B,** A superficial infection on the dorsal coronary band has led to a focal increase in heat without affecting the hoof itself. **C,** A medial corn (*right of image*) manifested as a marked increase in heat in the medial angle of the sole when compared to the lateral side. **D,** Solar bruising and inflammation evident thermographically as the temperature of the sole adjacent to the shoe has become equal to the temperature of the heel bulb.

image, reflecting its high vascularity. The temperature level and span of the camera are adjusted to use the whole color range for the image so that the coronary band would normally appear white and the coldest part of the image is blue or black, thus maximizing the visual contrast within the image. Some cameras have an automatic setting that constantly readjusts the image in this way, but this feature does not allow comparison of left to right symmetry and thus is not useful clinically.

The camera should be carefully focused and a series of still images obtained as described. The absolute temperature of points of interest can be determined using a radiometer so that absolute temperature differences can be calculated. Some of the systems allow more detailed analysis of the images on a computer, including calculation of mean temperature within regions of interest or graphical representation along lines of interest. The software tends to be expensive and there is a greater need for these features in research than in a clinical setting.

CLINICAL IMAGING

Figure 10-33 demonstrates a series of normal views of the distal limb and digit. The coronary band is the warmest part of the image and then the hoof becomes progressively cooler toward its ground surface. The bulbs of the heel are also warm, and within the distal limb heat generally follows the pattern of blood vessels such that the medial metacarpal region is normally warmer than the lateral. On the solar view, the sulci of the frog appear warmest because there is less tissue depth in this area. Ideally, the limbs appear symmetrical, but there can be variation in normal horses, especially at low ambient temperatures.[20,21] Thermoregulatory cut-off can be quite variable, and there will be intermittent periods of vasodilatation. This is not necessarily symmetrical between left and right,[20] nor between front limbs and hindlimbs. Re-evaluation of the horse some hours later may yield a different image, especially if the horse can be placed in a heated box. Exercising the horse for approximately 20 minutes, as well as administration of vasodilators such as acepromazine, can also be used to increase peripheral blood flow and temperature to allow improved diagnostic imaging. As demonstrated in Figure 10-31, thermoregulatory cut-off of the distal limb dramatically reduces the temperature and thus the contrast within the feet such that subtle lesions may be missed. Severe inflammation in the presence of thermoregulatory cut-off will, however, still cause a sufficient increase in blood flow to be detectable (Figure 10-34, *A*).

Thermography is very sensitive at detecting the presence of superficial inflammation within the foot, such as caused by subsolar abscess in the horse in Figure 10-34, *A*. Figure 10-34, *B*, demonstrates a horse with an infection in the coronary band following direct collision with a show-jumping fence. This horse was suffering from severe lameness and had previously

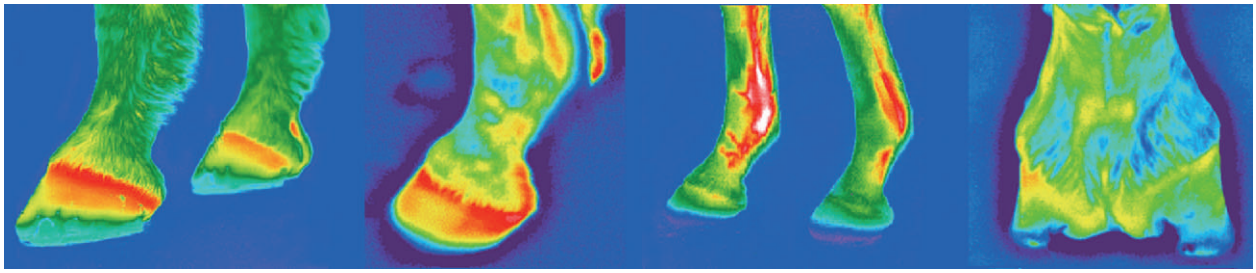

FIGURE 10-35 **A,** Foot-sore horse showing uniformly increased width of high temperature zone distal to the coronary band in both feet. **B,** Acute palmar foot pain associated with increased heat in the heel region. **C,** Decreased heat pattern within the foot relative to the distal limb in a horse with navicular disease. **D,** Mediolateral foot imbalance with a high lateral wall (*right of image*) has resulted in increased heat in the medial heel.

undergone scintigraphic examination to rule out the presence of a fracture of the distal phalanx. The nuclear scan had shown no increased uptake in the distal limb in bone phase images. Figure 10-34, *C,* shows a horse with a medial corn that has resulted in a focal temperature increase in the medial angle of the sole when compared to the lateral seat of corn. These images must be obtained before the foot is pared out, as removing tissue will make the region appear hotter. Figure 10-34, *D,* shows a horse with solar bruising. The temperature of the sole adjacent to the shoe, which is normally lower than the coronary band, has become equal to the temperature of the heel bulb.

Figure 10-35, *A,* shows a horse with generalized foot inflammation and tenderness after shoeing. The normal band of high temperature immediately distal to the coronary band has widened considerably in response to laminar inflammation. When re-evaluated 3 days later, this horse had a normal thermal pattern and was sound. Horses with acute palmar heel pain sometimes show increased heat in the heels (Figure 10-35, *B*). Chronic palmar foot pain and navicular syndrome do not show superficial inflammation and have either normal thermographic patterns or a colder pattern than normal, especially in the heel region (Figure 10-35, *C*). Contrary to normal horses, the temperature in the heels of horses with navicular disease does not increase after the horse is exercised,[22,23] probably due to a decrease in loading of this area rather than to an inherent ischemic disease. Superficial foot inflammation can obviously be diagnosed clinically in the majority of horses. Thermography is helpful in horses that have mild signs of inflammation and response to hoof testers but in which there is still the suspicion of an underlying pathologic lesion. Thus if a mildly lame horse has slight foot tenderness yet is thermographically cold, greater attention should be paid to ruling out the presence of pain from the deeper structures within the hoof. Conversely, if the foot appeared thermographically hot, then superficial foot pain would be more likely. These decisions can be helpful in optimizing the efficiency of a lameness evaluation, especially in a horse that is close to competition and in the extremely fractious horse, in which it is advantageous to minimize the amount of diagnostic local analgesia employed.

Thermography can also be used to assess foot balance. When a horse with metal shoes is imaged thermographically immediately after trotting on a hard surface, the branch of the shoe covering that side of the foot taking the greatest load will appear hottest. In horses with significant mediolateral foot imbalance that land lateral-wall-first and roll over to the medial side during weight bearing, a temperature increase can be observed at the level of the medial coronary band (Figure 10-35, *D*), rather than on the lateral side, which lands first.

Thermographic evaluation can also be useful in monitoring of laminitis. In cases of acute clinical laminitis, there is increased heat within the foot (Figure 10-36, *A*),[20] although some authors still claim that this is preceded by a prodromal drop in temperature.[24] The normal gradation of temperature from the coronary band to the solar margin is lost as the temperature of the dorsal wall approaches that of the coronary band. These changes are more subtle in cases of subacute laminitis (Figure 10-36, *B*). On the solar views, there may be an increased thermal pattern in the toe region of the distal phalanx (Figure 10-36, *C*). In cases of chronic laminitis, areas of decreased temperature may be identified at the dorsal aspect of the coronary band and the dorsal hoof wall (Figure 10-36, *D*), indicating decreased perfusion and laminar separation. This is a poor prognostic indicator.

CONCLUSION

Thermographic imaging can be a useful adjunct to the standard lameness evaluation of the foot. It does require a considerable investment of time to gain the necessary experience to differentiate between normal variation and patterns suggestive of pathology. Improvements in technology have brought high-quality imaging within the reach of the equine practitioner, which consequently makes image interpretation much easier. A standard protocol for imaging is also important for obtaining accurate results.

PRINCIPLES OF COMPUTED TOMOGRAPHY

William R. Widmer

Computed tomographic (CT) scanners consist of an x-ray tube, a gantry, a moveable table or couch, a digital storage system, a printer, and a workstation (Figure 10-37).[25] The workstation consists of a separate computer, software for image acquisition, and a monitor. The workstation is often integrated with a local

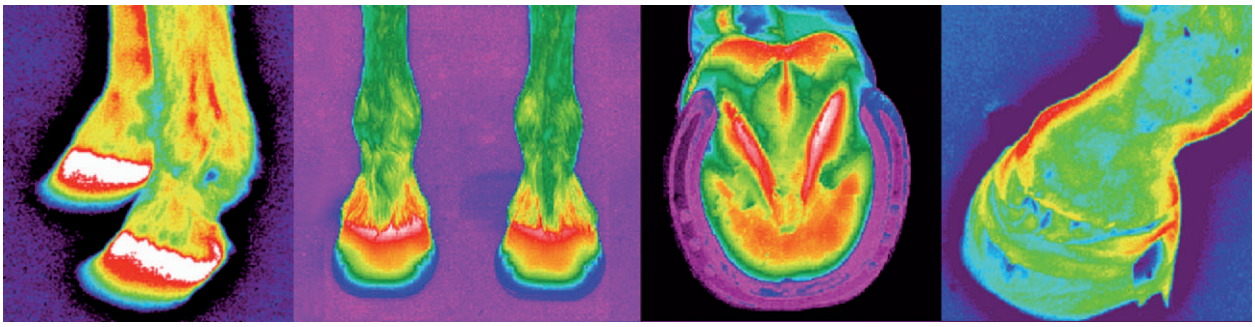

FIGURE 10-36 **A,** Acute laminitis with increased width of the coronary band heat zone. **B,** Subacute laminitis with a more subtle increase in heat of the dorsal hoof wall than observed in part *A*. **C,** Solar view of subacute laminitis, with increased heat pattern around the toe region of the distal phalanx dorsal to the point of the frog. Note differences between this image and Figure 10-34, *C* and *D*. **D,** Chronic laminitis with decreased perfusion of the dorsal aspect of the coronary band and the dorsal hoof wall, consistent with laminar separation.

FIGURE 10-37 Typical layout of a computed tomography imaging suite, with the workstation in the foreground, shielded from the x-ray tube, gantry, and moveable patient couch in the adjacent room.

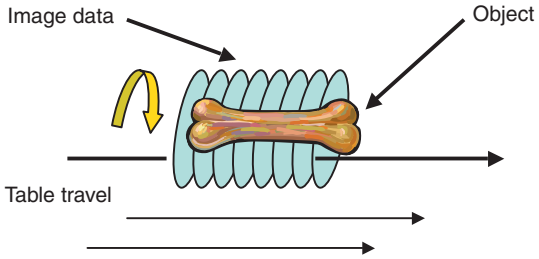

FIGURE 10-38 Schematic of image acquisition in a helical computed tomography unit. The patient moves through the CT scanner on a table as the x-ray tube within the scanner moves rapidly, generating very thin tissue slices of the region of interest. These slices can then be reconstructed and displayed in different planes or as a three-dimensional image.

network and called a *picture archiving and communication system* (PACS). A separate computer with imaging-analysis software and a gray scale monitor is used for imaging interpretation. The PACS allows display of all types of medical images in various formats. It also provides archiving capability for long term storage and rapid retrieval. Integration with a local network allows internal communications with other hospital departments. The PACS easily interfaces with external digital links allowing export and import of images.

CT images are acquired by rotating an x-ray tube around the long axis of a subject while multiple exposures are recorded on an array of x-ray detectors. The x-ray beam has a narrow fan shape, traversing a very small volume or "slice" of tissue as it moves through an arc of 360 degrees around the subject. Slice thickness in equine imaging varies from 1.5 to 5 mm, depending on the capability of the scanner and the study being performed.

With older scanners, a moveable couch is used to position the animal along the CT gantry and is held stationary while each slice is acquired. Newer scanners use sliding concentric rings or *slip ring* technology[25] for electronic circuitry that allows continuous rotation of the x-ray tube as the couch moves. These helical, or spiral, CT scanners rapidly and continuously acquire scan data as the subject moves within the gantry (Figure 10-38). Scan times are just a few seconds, minimizing problems with motion of the animal.

X-rays exiting the animal excite the detectors, producing an electrical signal that is proportional to x-ray intensity. A computer processes, digitizes, and stores the electrical signal. The ability of tissues within the image slice to attenuate incident x-rays determines the intensity of the x-rays reaching the detectors. Tissues that are thick or have a high physical density or atomic number attenuate more of the x-ray beam than tissues that are thin or have a low physical density or atomic number.

After each exposure, a computer calculates the attenuation for each narrow band of tissue that is interrogated by the x-ray beam.[25-27] Because each part of the tissue slice is "seen" many times from various directions, it is possible to divide the three-dimensional tissue slice into a grid of rows and columns analogous to a crossword puzzle. Each tiny block of the tissue grid is called a volume element, or *voxel*. Voxels have length, width, and depth. The end (length and width) of a voxel is square and represented as a picture element or a *pixel* on a two-dimensional image. The depth of a voxel is determined by the thickness of the tissue slice. The computer analyzes the mean attenuation of each voxel through numerous algebraic methods and assigns a *CT number*, again in a manner similar to solving a crossword puzzle. (CT numbers are also called *Hounsfield units* in honor of Godfrey N. Hounsfield, who developed the first CT scanner.) The CT

number indicates the relative attenuation of the tissue contained within each voxel. Water is the reference standard and is assigned a CT number of 0; cortical bone is +800 to 1000 and air is –1000. Fluids and soft tissues have CT numbers between 0 and about +45, whereas fat has a CT number of –40 to –50.

It would be impossible to make sense of a printout of an image slice displaying a small matrix of CT numbers; therefore, they are displayed as various shades of gray for image interpretation.[25,27] Sixty-four to 128 shades of gray are used to make the final image, and each shade of gray represents a group of several CT numbers. Each CT number cannot be assigned a specific shade of gray because the human eye can only distinguish between 60 to 80 shades of gray. Thus each shade of gray represents a group of several similar CT numbers. The complete gray scale range is only assigned to the CT numbers of interest, the so-called *window*. The window of CT numbers representing the gray scale can be adjusted to maximize the tissue of interest. The window *width* determines the range of CT numbers over which the available gray-scale is spread (the CT numbers of interest). The window *level* is the median CT number in the window. For instance, a soft tissue study might have a window width of 400 with a window level or median gray of 40. For bone imaging, the window width is set at 300 to 400 with a window level of 1000 to 1200.

Choice of window settings affects image contrast.[27] Narrow windows provide high contrast and better differentiation of soft tissues because gray scale is spread over a relative few CT numbers. Wide window settings reduce the contrast because available gray shades are spread over a wide range of CT numbers. Wide window settings are preferred for imaging bone because soft tissue structures lack contrast and are suppressed while bony structures are well visualized.

Data acquired in the transverse plane (perpendicular to the long axis of the scanner, also called *axial* slices) can be reconstructed and displayed in sagittal or dorsal planes. Magnetic resonance imaging differs, being truly multiplanar because original image data can be obtained in any plane and reconstruction is unnecessary.

ACQUIRING A COMPUTED TOMOGRAPHIC SCAN OF THE EQUINE DIGIT: TECHNICAL CONSIDERATIONS

Computed tomographic imaging of adult horses is limited to the extremities and the head and neck. General anesthesia is used to facilitate positioning and to reduce motion of the animal during the scan. A customized table capable of supporting a horse must be linked to the scanner's existing table because original equipment tables have a load limit of 180 kg (400 lb) or less (Figure 10-39). Custom tables are positioned in front of the bore of the scanner and the area being imaged is incrementally advanced into the scanner as slices are acquired.[28] The resistance of a customized table must be low, because the table motor must move the combined weight of the horse and the table through the gantry. An alternative method is to move the scanner along a stationary table as each slice is acquired (Phillips Tomoscan M; Phillips Medical Systems, Best, Netherlands). Overheating of the x-ray tube is a problem with low-output equipment. The large size and bone mass of the horse require high exposures and can overload the heating capacity of the x-ray generator. Fortunately, CT scanners have warning devices that limit damage to the x-ray tube, but the scan must be stopped to allow cooling.

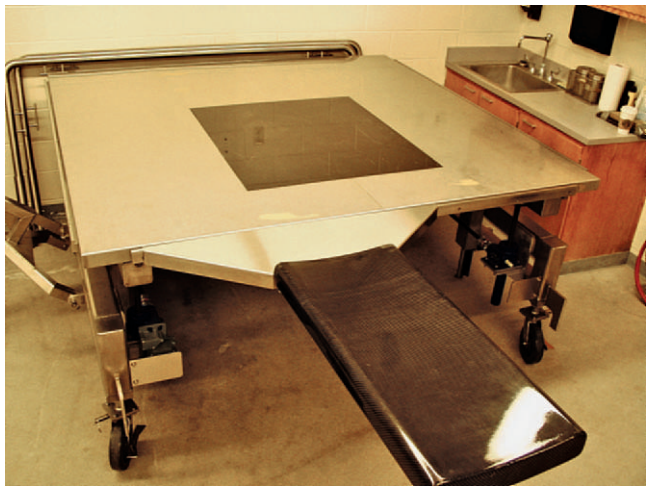

FIGURE 10-39 Example of a customized equine CT table that can both support a horse's weight and link to the scanner's existing table.

COMPUTED TOMOGRAPHIC APPLICATIONS FOR IMAGING THE EQUINE DIGIT

Navicular Syndrome

Compared to conventional radiography, CT is superior for osseous changes of navicular syndrome and also offers some advantages for identification of soft-tissue changes (Figures 10-40 to 10-44).[29] The complex shape of the navicular bone limits its radiographic evaluation. Even with meticulous technique, many lesions are not seen because of two inherent problems of radiography: superimposition and poor subject contrast. Because of its superior resolution, CT can provide accurate information on the number and extent of enlarged synovial fossae in the distal navicular border. Important pathologic changes with prognostic implications such as cystic changes or erosions of the flexor surface are easily documented with CT.[30] Chip fractures of the distal border of the navicular bone can be distinguished from other causes of osseous body formation because the donor site of the fracture can be verified (see Figures 10-42 and 10-43). Finally, CT can confirm the onset of medullary sclerosis and accurately locate cystic lesions. Reconstruction in dorsal and sagittal planes increases the yield of image information and can overcome problems associated with the complex shape of the navicular bone. For instance, dorsal plane images provide the best visualization of the synovial fossae, and flexor cortex changes are best seen on transverse and sagittal plane images (see Figure 10-44).

Fractures of the Digit

Fractures of the phalanges present a challenge to the clinician because they are easily underestimated on radiographic examination. Incomplete fractures extending from the solar

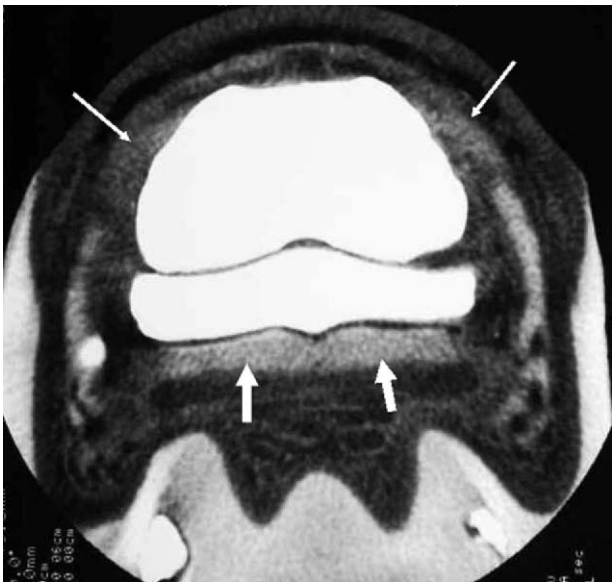

FIGURE 10-40 Transverse computed tomography image of a horse's foot at the level of the navicular bone. The image is windowed for soft-tissue detail. The *large arrows* show the deep digital flexor tendon and the *small arrows* indicate the position of the collateral ligaments of the distal interphalangeal joint. *(Courtesy Francis Verschooten.)*

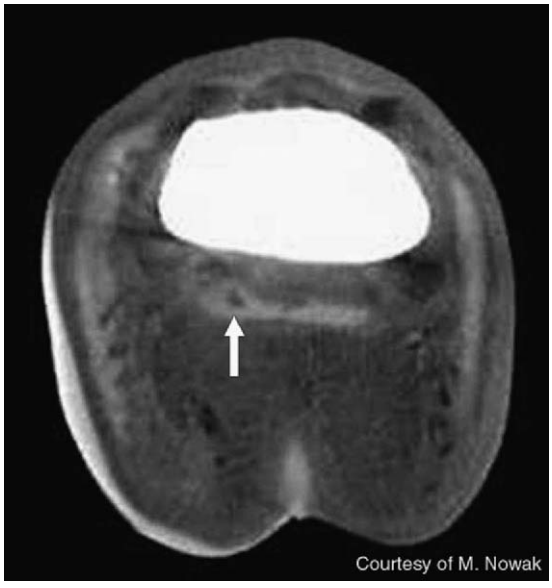

FIGURE 10-41 Transverse computed tomography image (soft tissue window) of the left front foot at the level of the middle phalanx of a 12-year-old Showjumper with acute foot lameness. The *arrow* indicates that the lateral lobe of the deep digital flexor tendon contains a central radiolucency and is enlarged in comparison with the medial lobe. This represent lateral lobe tendinitis with a core lesion. *(Courtesy Michael Nowak.)*

margin of the distal phalanx can be difficult to recognize, even with careful technique and multiple projections. Compared to standard radiographs, transverse CT images of the distal phalanx provide cross-sectional information that improves identification and characterization of most fractures (Figure 10-45). Computed tomography is also useful for determining the full extent of solar margin fractures and may identify those that

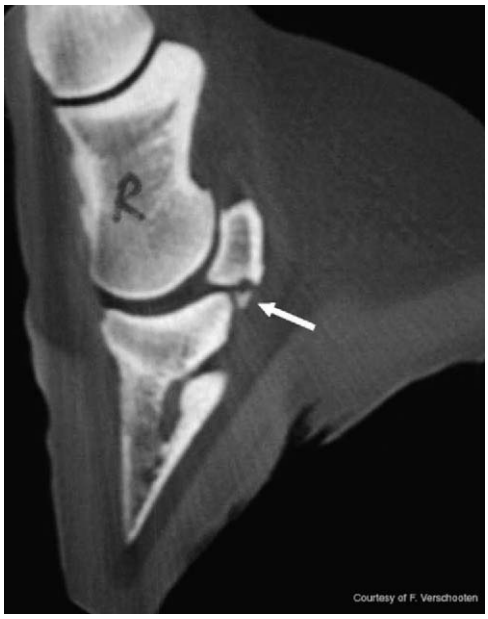

FIGURE 10-42 Sagittal computed tomography image (bone window) of the right front foot of a horse with chronic foot lameness. The *arrow* points to the presence of a small osseous fragment off the distal margin of the navicular bone. There is a radiolucent defect in the parent bone proximal to the fragment suggestive of local bone remodeling. *(Courtesy Francis Verschooten.)*

are nondisplaced but extend to the joint space.[31] Comminuted fractures of the proximal and middle phalanges are often complex with several components and multiple fracture planes.[32] Computed tomographic examination provides an exact knowledge of the extent of the fracture, number of fragments, and plane of involvement is necessary for surgical planning and prognosis. Preparation for surgical repair must be made in advance because these fractures should be immediately repaired after CT, while the horse is still under anesthesia.

Laminitis

Computed tomography is potentially useful for evaluating horses with laminitis. Although the degree of rotation of the distal phalanx can be critically assessed with CT, its best use may be the evaluation of the vascular integrity of the soft tissues of the digit. Iodinated contrast media can be administered intravenously, allowing visualization of the capillary beds of the sensitive laminae (dose is not to exceed 3 ml/kg body weight injected at a rate of 3 ml/second of a 50/50 iodinated contrast medium [370 mg I/ml diluted with normal saline]). Post-contrast CT studies show enhancement of the soft tissues where there is an adequate blood supply. Recently, CT angiography has been used to describe the vascular anatomy of the equine digit.[33] Arteriograms were used to detail the vascular supply of the distal limb, including the capillary beds of the digit. Arterial studies can be performed via injection of the median artery, have few complications, and likely give more information regarding the vascular supply than venograms. Either type of angiography may give insight into the integrity of the circulation of the foot, a critical issue in cases of severe laminitis.

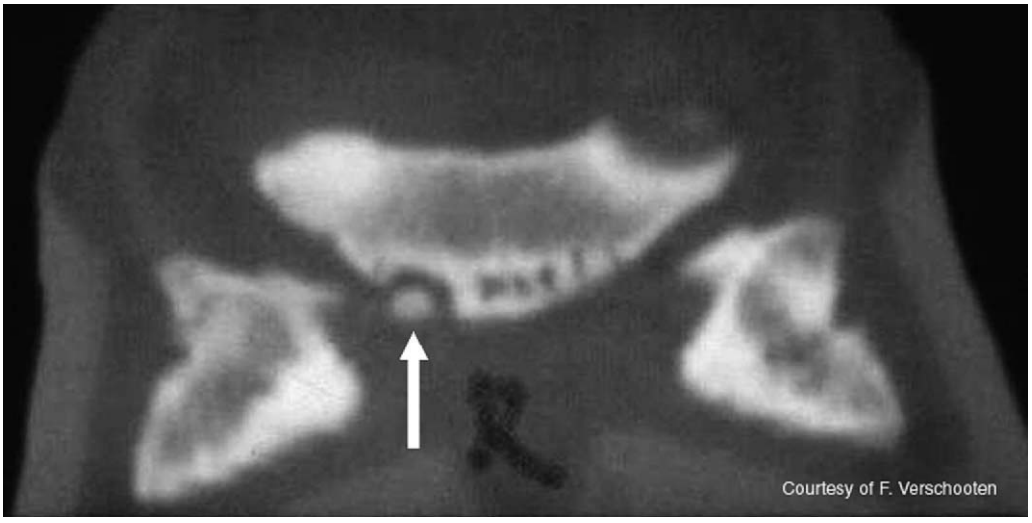

FIGURE 10-43 Coronal reconstruction of the sagittal computed tomography image of the same foot shown in Figure 10-42. As before, the arrow points to the distal border fragment, which is located at the angle of the distal and lateral sloping borders of the navicular bone. The large, crater-like defect in the parent bone adjacent to the fragment is suggestive of bone resorption secondary to movement of the fragment. Notice the sclerotic margin outlining the defect. *(Courtesy Francis Verschooten.)*

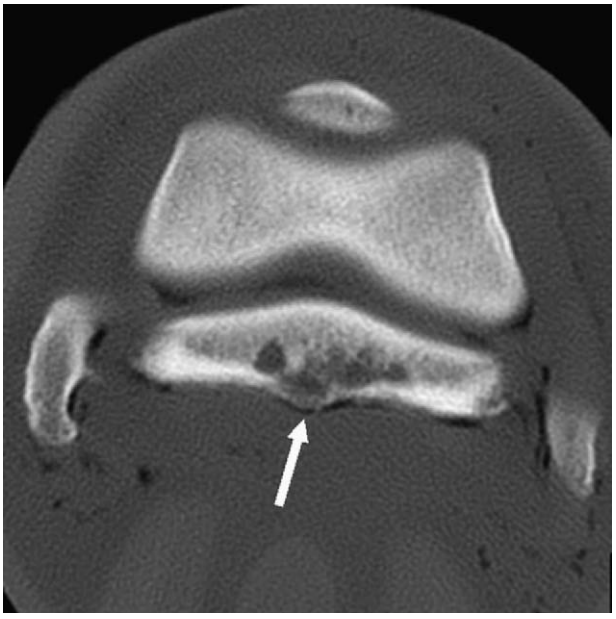

FIGURE 10-44 Transverse computed tomography image (bone window) at the level of the navicular bone of a 14-year-old Quarterhorse with acute severe lameness of the right front foot. There is a large radiolucent medullary defect continuous with a flexor cortex erosion on the palmar aspect of the navicular bone (*arrow*).

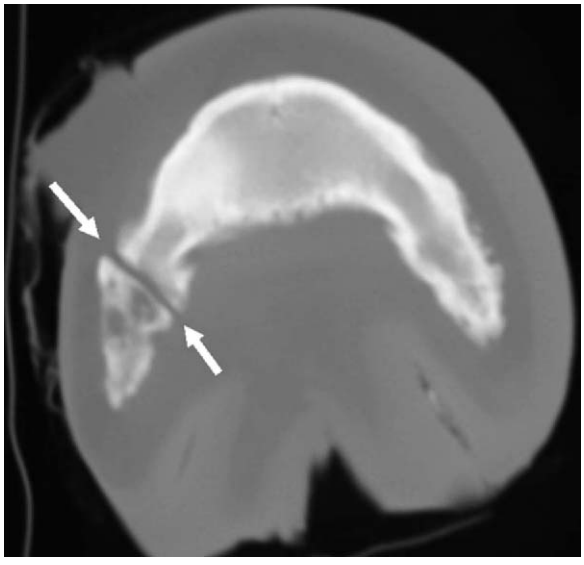

FIGURE 10-45 Transverse computed tomography image (bone window) of the left front distal phalanx of a 6-year-old Quarterhorse stallion with a healed deep heel bulb laceration, but with persistent lameness. There is a fracture of the lateral palmar process of the distal phalanx with evidence of new bone production at the fracture margins and osteolysis in the fracture gap.

Pedal Osteitis

Computed tomography provides excellent bony detail. It can give information regarding the degree of new bone formation, lysis, and remodeling of the solar margin of the distal phalanx. Identification of solar margin fractures (Type VI fractures), which is often problematic with radiography, is easier with CT. Perhaps angiography or contrast-enhanced studies will be useful with newer multislice scanners for evaluation of soft-tissue changes associated with active osteitis.

Subchondral Diseases

Osteoarthritis of the interphalangeal joints is commonly associated with subchondral sclerosis and other changes that can be recognized with conventional radiography. However, radiographic confirmation of subchondral sclerosis is problematic. Circumferential enthesophytes and osteophytes can mimic subchondral sclerosis because of their summation with subchondral bone. With CT, the effect of summation is avoided, and subchondral bone is clearly visualized on transverse slices, separate from osteophytes and entheso-

phytes. Thus, CT might be effective for identifying early osteoarthritis in the interphalangeal joints, where subchondral changes often precede other bony changes.

Subchondral erosions and cystic lesions are easily recognized with CT. These changes are associated with a variety of arthroses, including traumatic, developmental, and degenerative arthritis. Computed tomography was used to diagnose subchondral fractures and osteochondritis dissecans–like lesions of the proximal articular surface of the proximal phalanx in a 6-year-old horse.[34] In this case, lameness was localized to the metacarpophalangeal joint. Nuclear scintigraphy revealed increased uptake in the proximal aspect of the proximal phalanx, and radiographic examination depicted subtle regions of radiolucency and sclerosis in the same area. However, only CT elucidated subchondral changes typical of osteochondrosis dissecans. Subchondral cystic lesions of the distal interphalangeal joint are difficult to establish on conventional radiographs but are confirmed with CT examination. In addition, CT can identify communication of subchondral cystic lesions with the articular surface of the distal phalanx.[35]

Soft-Tissue Changes

Musculoskeletal soft-tissue changes have received little attention in the CT literature. Because of the widespread use of diagnostic ultrasound and no requirement for general anesthesia, CT has not been thoroughly utilized for soft tissue problems of the digit. However, with newer CT scanners and good quality software, some of the important ligaments, tendons and other retinacular structures of the equine digit can be visualized (see Figure 10-41). This may change further with the advent of multislice scanners (see next section) which have improved contrast resolution.

FUTURE USES OF COMPUTED TOMOGRAPHY IN THE EQUINE DIGIT

The advent of new multislice CT scanners has changed human musculoskeletal imaging. These scanners can acquire up to 40 slices per revolution of the x-ray tube. Multislice CT allows rapid acquisition of a "volume" of thin slices than can be reconstructed with little loss of detail in any plane. Therefore, patient motion and positioning are not critical. Scanning time is often 15 to 20 seconds, limiting motion artifact. If the patient cannot be positioned for a true axial scan, for example a foal with flexural deformity, image data can be reconstructed in a transverse plane. Soft tissue contrast, although not on a par with that of magnetic resonance imaging, is improved compared with previous single-slice CT scanners. In addition, use of intravenous contrast can facilitate evaluation of the soft tissues of the musculoskeletal system. These features, along with lower imaging cost and superior hard tissue resolution, have allowed CT to recoup part of the imaging market that had been lost to magnetic resonance imaging over the last decade. A new lightweight, mobile, multislice helical CT scanner is available for use with human patients (CereTom; NeuroLogica Corp., Danvers, Mass.), which offers a 25-cm field of view and has promise for imaging of the equine extremity. The system is primarily designed for the human head and neck and makes 8 slices per revolution.

It follows that new multislice CT scanners have a logical place in equine imaging. These units are less expensive to buy, lease, and maintain than magnetic resonance units. They do not require shielding for radiofrequency waves, and there is no problem with a magnetic field. Increased use of modern CT scanners can improve our ability to diagnose conditions of the digit and provide improved understanding of conditions such as navicular syndrome, laminitis, and subchondral diseases.

MAGNETIC RESONANCE IMAGING OF THE FOOT

Michael C. Schramme, Rachel C. Murray, Sue J. Dyson, and Jane Boswell

The true causes of pain in horses with foot lameness have remained elusive for a long time. This mystery has led to endless arguments about the relevance or lack of radiographic findings in the navicular bones, and to the use of diagnoses such as radiographic navicular disease, clinical navicular disease, radiographic and clinical navicular disease, and navicular syndrome. Navicular syndrome has been defined as a clinical manifestation of a number of different disease processes in the foot.[36] It has indeed become apparent that in addition to degeneration of the navicular bone and the DIP joint, soft tissue injuries make an important contribution to pain in the foot.[37,38] Magnetic resonance imaging is becoming increasingly prominent in the diagnosis of the cause of lameness in these patients because of its ability to show superior soft tissue contrast and detail. In fact, many of the known causes of foot lameness cannot be identified conclusively without the use of MRI.

Thanks to the introduction of low-field magnets, the availability of MRI is increasing rapidly for the purpose of diagnosing the cause of lameness. In spite of this, however, studies with high-field MRI have provided the first detailed clinical and pathologic studies to explain the range of foot injuries, and it therefore remains the standard for MRI of the equine foot.

HIGH-FIELD MAGNETIC RESONANCE IMAGING

Magnetic resonance imaging essentially uses a large magnet, a transmitter and receiver coil, magnetic field gradients, and a powerful computer to produce a gray scale image of the tissue hydrogen protons in the body part being examined. High signal is white and low signal is black. Among the many advantages for use in equine orthopedic diagnosis are the lack of ionizing radiation, tomographic image analysis, fine anatomical detail, and high contrast, especially for soft tissues. In addition, knowledge of the water content of tissues reflects not only their anatomical but also their physiologic status. Increased tissue fluid, mineralization, or tissue fibrosis can all be detected because of alterations in signal intensity

High-field MRI uses closed superconducting magnets in which it is easier to maintain a homogeneous magnetic field,

Table 10-5 Different Protocols for High-Field Magnetic Resonance Imaging of the Foot

Dual-Echo–Based Protocol (1.5 Tesla Siemens Symphony Magnet†)

Sequence	Matrix	NEX	FOV (mm)	SNR (%)	Scan Time (minutes)	Coil	Slices/ Slab	Slice Thickness (mm)
Three-plane localizer	256 × 128	1	300	100	0.36	Phase array	3	5.0
Sagittal STIR	256 × 192	1	120	100	3.19	Phase array	26	3.5
Sagittal PD TSE	320 × 202	2	120	100	5.47	Phase array	26	3.5
Sagittal T2 TSE	320 × 202	2	120	100	5.47	Phase array	26	3.5
TS PD TSE	320 × 224	2	150	100	6.22	Phase array	30	4.0
TS T2 TSE	320 × 224	2	150	100	6.22	Phase array	30	4.0
TS STIR	256 × 192	1	150	100	3.42	Phase array	30	4.0
TS T1 FLASH 3D FS	256 × 256	1	120	100	4.16	Phase array	40	2.0
Dorsal T1 FLASH 3D	256 × 256	2	120	100	3.45	Phase array	24	2.0
Oblique PD TSE FS	320 × 202	1	150	100	2.54	Phase array	24	3.0

Three-Dimensional Gradient Echo-Based Protocol (1.5 Tesla GE Signa Echospeed Magnet‡)

Sequence	Matrix	NEX	FOV (mm)	SNR (%)	Scan Time (minutes)	Coil	Locations/ Slab	Slice Thickness (mm)
Three-plane localizer	256 × 128	2	280	136	2.12	Extremity		5
Sagittal 3D SPGR	256 × 256	2	280	160	2.55	Extremity	44	3
Sagittal fast STIR	256 × 192	1	240	119	5.46	Extremity	30	4
Sagittal 3D T2*	256 × 256	2	280	160	2.04	Extremity	44	3
Dorsal 3D SPGR	256 × 256	2	280	163	3.03	Extremity	46	3
Dorsal 3D T2*	256 × 256	2	280	163	2.09	Extremity	46	3
TS 3D SPGR	256 × 256	2	240	135	5.27	Extremity	80	3
TS 3D T2*	256 × 192	2	240	143	3.1	Extremity	90	3
TS Fast STIR	256 × 192	1	220	100	5.46	Extremity	30	4
Dorsal fast STIR	256 × 192	1	240	119	2.51	Extremity	30	4

NEX, Number of excitations; *FOV*, field of view; *SNR*, signal-to-noise ratio; *FLASH*, fast low angle shot; *FS*, fat suppression; *PD*, proton density; *SPGR*, spoiled gradient; *STIR*, short T1-inversion recovery; *TS*, transverse; *TSE*, turbo spin echo; *3D*, three-dimensional.
† Siemens, Malvern, Penn.
‡ General Electric, Milwaukee, Wis.

uses fast scanning times, and produces good signal-to-noise ratio. All these factors contribute to a high image quality. Such scanners are designed for human use and necessitate general anesthesia of the horse for the duration of the scan, which generally takes about 40 minutes per foot.

HIGH-FIELD MAGNETIC RESONANCE IMAGING EXAMINATION PROTOCOL FOR THE FOOT

Standard MRI examinations include the use of several different acquisition sequences. The choice of sequence is determined by the type of scanner, the tissues examined, and the information sought. In addition, it is important to keep total anesthesia time and therefore imaging time under 90 minutes because of recumbency-related problems in horses. Alternatively, clinicians do not want a reduction in scanning time to result in a reduction in data and image quality. These considerations have led to two basic protocols for scanning horses' feet with high-field MRI systems.

The first protocol (Table 10-5) is based on the dual-echo scan (T2-weighted and proton density [PD]). Because T2-weighted scans can take a long time to acquire, T2-weighted and PD scans are conducted concurrently to maximize information and minimize scan times. The PD sequence demonstrates the differences in proton density between different tissues and provides excellent anatomical detail. T2-weighted sequences provide poorer anatomical detail but show fluid accumulation in tissues as hyperintense signal. Transverse and sagittal dual echoes are supplemented with sagittal and transverse fat-suppressed sequences in this protocol. Fat suppression can be most consistently achieved with a short T1-inversion recovery sequence (STIR). During this acquisition process, it may be necessary to correct the location of the digital fat suppression manually to exactly 220 MHz away from the water peak. Fat suppression can also be obtained by a high detail spectral presaturation technique, although results of this technique are less consistent. Fat suppression allows specifically for the differentiation between hyperintense signal arising from water and fat on T2-weighted images, because hyperintense signal arising

from fat (e.g., in bone marrow) is suppressed. This makes it possible to recognize the presence in bone of fluid associated with pathologic processes. Other sequences can supplement the basic dual echo sequence to improve the diagnostic yield, including the transverse and coronal T1-weighted gradient echoes and an oblique, fat-saturated PD echo. High-detail, thin-slice, T1-weighted three-dimensional gradient echo sequences are particularly useful for the detection of subtle abnormalities in the DDFT (transverse) or distal border of the navicular bone (coronal). The oblique, fat-saturated PD sequence is oriented perpendicular to the long axis of the collateral ligaments of the DIP joint and is most suitable for detection of signal change and size variation in these ligaments.

A different protocol (see Table 10-5) consists of fast, high-detail sequences such as three-dimensional gradient echoes (T2* GRE and T1-weighted SPGR) in all three slice planes (coronal, transverse, and sagittal). Three-dimensional data sets allow thinner slice thickness and improve the signal detected compared with the surrounding noise interference but are also more prone to field inhomogeneities and therefore imaging artifacts. This approach allows for integration of information from both T1- and T2-weighted imaging in the final analysis. This three-dimensional gradient echo based protocol is further supplemented with the sagittal and transverse fat suppressed sequences (STIR). Newer, sophisticated sequences with specific target tissues in mind (e.g., volumetric interpolated breath-holding examination [VIBE], dual-echo steady state [DESS], driven equilibrium fourier transformation [DEFT], and delayed gadolinium enhanced magnetic resonance imaging of cartilage [dGEMRIC]), especially cartilage, are constantly evolving and under investigation.

The choice of coils is dictated by manufacturer availability. Most foot studies are performed with human knee coils. These are quadrature coils (volume coils) that surround the entire body part and have good field homogeneity over a large area. The first author's practice uses a human torso array coil (Siemens, Malvern, Penn.), which is a surface or phased array coil that can be wrapped around the horse's foot and secured with Velcro straps. Surface array coils are "receive only" coils that have a good signal-to-noise ratio with an additional 20% signal gain. They allow faster scanning with finer detail and have greatest sensitivity nearest to the coil surface.

IMAGE INTERPRETATION

The foot is characterized by a complex anatomy, with a wide range of normal variation and tissue interactions. In addition, the guidelines for interpretation of regional analgesia can be confusing. Finally, MRI is susceptible to previously unknown imaging artifacts, and correct positioning of image slices is crucial to avoid image obliquity. Given these circumstances, the fine anatomical detail provided by MRI can easily lead both to overinterpreting and to missing subtle information. It is therefore essential to have a thorough knowledge of the regional anatomy, to understand the origin of MRI artifacts (Table 10-6), to be able to form a link between the clinical presentation and the lesion, and to have the experience of reading equine images. The differences in tissue contrast between T1-, T2-, and PD-weighted sequences are summarized in Table 10-7.

Tissue injury results in changes in biochemical and water content and therefore signal intensity. For a complete assessment of injury, images must be analyzed in all pulse sequences and planes. Cross-referencing of images should be performed on any suspected lesion to confirm its location.

The following tissue abnormalities result in magnetic resonance signal irregularities:

- Tendon and ligament injury: damage is seen as focal increase in signal intensity on both T1- and T2-weighted sequences, potentially accompanied by swelling in the acute stages of injury. In chronic stages of healing by fibrosis, signal intensity returns to normal in T2-weighted sequences but stays high much longer in T1-weighted sequences. In some cases of DDFT injury in the foot, focal hyperintensity remained present on T1-weighted sequences in spite of histologic evidence of mature scar tissue. It should be remembered that collagen fibers oriented at 55 degrees to the main magnetic field result in hyperintense signal that is unrelated to injury (the magic angle effect).
- Bone remodeling (cortical bone loss, medullary fluid, and mineralization): trabecular architectural changes are visible as a loss in signal homogeneity. Local increase in fluid content in bone is recognized as local increase in signal intensity on fat-suppressed images and T2-weighted images, whereas there is local decrease in signal intensity on T1-weighted images. This phenomenon has been referred to as bone edema, although it has been shown to reflect the possibility of bone necrosis, hemorrhage, trabecular microdamage, or medullary fibrosis, while edema itself has proved very difficult to identify histologically. Areas of sclerosis are characterized by increased bone density and produce low signal intensity on both T2- and T1-weighted images.
- Occult fractures: fractures are seen as defects in the bone outline and structure and may show as lines of increased signal intensity on T2-weighted images.
- Cartilage damage (hyaline cartilage and fibrocartilage): morphologic changes observed may consist of cartilage defects, decreased thickness, and volume changes. Cartilage defects result in signal hypointensities on T1-weighted sequences, whereas hyperintense areas are seen in degenerating cartilage on T2-weighted images, mostly because synovial fluid becomes trapped in the physical defect. The strength of the T2 signal is also affected by differences in collagen orientation. Although MRI has mostly been reported as a good imaging modality for cartilage, studies have reported continuing difficulties with accurate MRI evaluation of traumatic cartilage lesions. Focal cartilage defects are more easily identified than generalized degeneration (wear lines, thinning). Difficulties with imaging cartilage are manifold. Firstly, equine distal limb cartilage is frequently too thin for the spatial resolution of MRI. Furthermore, the radial orientation of collagen causes internal signal changes in cartilage. Very disturbing is the volume-averaging that results from imaging cartilage on oblique joint surfaces. Other artifacts that interfere with clear definition of cartilage are inherent to three-dimensional gradient echo imaging needed for its resolution and include zero-fill interpolation (ZIP)

Table 10-6 Commonly Occurring Signal Irregularities in the Foot, Probably Caused by Artifacts or Normal Anatomical Variation

Structure	Signal Irregularity	Sequence	Orientation	Explanation
DDFT	Repeated lines parallel to DDFT edge	All	All	Gibbs (Truncation) artifact
	Bright signal near insertion of DDFT	All Max SPGR	All	Magic angle artifact
	Indistinct dorsal outline to the DDFT at proximal extent of the navicular bursa	T2* GRE	All	Volume averaging
	Incomplete/complete oblique linear areas of high signal in the DDFT at the level of the navicular bone	SPGR, T2* GRE	Transverse	Blood vessels or fibrocartilaginous septa
	Focal punctate areas of high signal in the DDFT at the level of the navicular bursa	SPGR, T2* GRE	Transverse	Possible chondroid metaplasia
	Short linear areas of low signal between the dorsal surface of the DDFT and the flexor border of the navicular bone	T2* GRE	Transverse	Volume averaging, possibly combined with ZIP reconstruction artifact
	Longitudinal area of high signal including 20%-50% of the width of the DDFT at its insertion	STIR	Sagittal	Magic angle effect
DSIL	Focal areas of high signal in the body without enlargement of the DSIL	T2* GRE	All	Synovial invaginations
	Focal areas of high signal at insertion without enlargement of the DSIL	T2* GRE	All	Synovial invaginations and blood vessel near entrance to terminal arch
CSL	Bright signal at the insertion to the proximal border of the navicular bone without enlargement or entheseophyte	STIR	Sagittal	Possible chondroid metaplasia or magic angle artifact
	Bright signal within substance without enlargement of the CSL	STIR, T2* GRE	Sagittal	Magic angle effect
DIP joint	Indistinct joint space	T2* GRE, SPGR	Sagittal, dorsal	Volume averaging ZIP reconstruction artifact
DIP joint	Asymmetry between lateral and medial collateral ligaments	T2* GRE, SPGR	Transverse	Asymmetric joint compression Asymmetric slicing
Navicular bursa	Blurring of the interface DDFT-navicular bone, especially at the level of the sagittal midline	T2* GRE	Sagittal	Volume averaging
Digital cushion	Interspersed focal areas of high and low signal	T2* GRE, SPGR, STIR	Sagittal, dorsal, transverse	Vasculature
SDSL	Focal high signal at the insertion of the SSL to the palmar margin of the middle phalanx	T2* GRE, SPGR, STIR	Sagittal, transverse	Fibrocartilage
Laminae	Relatively poorly circumscribed areas of low signal	All	All	Magnetic susceptibility artifact (metal nail fragments)

CSL, Collateral sesamoidean ligament; *DDFT*, deep digital flexor tendon; *DIP*, distal interphalangeal; *DSIL*, distal sesamoidean impar ligament; *GRE*, gradient echo; *SDSL*, straight distal sesamoidean ligament; *SPGR*, spoiled gradient echo; *STIR*, short T1-inversion recovery.

Table 10-7 Appearance of Different Tissue Types in Different Pulse Sequences

Sequence	Cortical Bone	Medullary Bone	Fluid	Fat	Tendon/Ligament	Cartilage
T2	Black	Light gray	White	White	Black	Gray
T1	Black	Light gray	Dark gray	White	Black	Gray
PD	Black	Light gray	Light gray	White	Black	Gray
STIR	Black	Dark gray	White	Black	Black	Gray

PD, Proton density; *STIR*, short T1-inversion recovery.

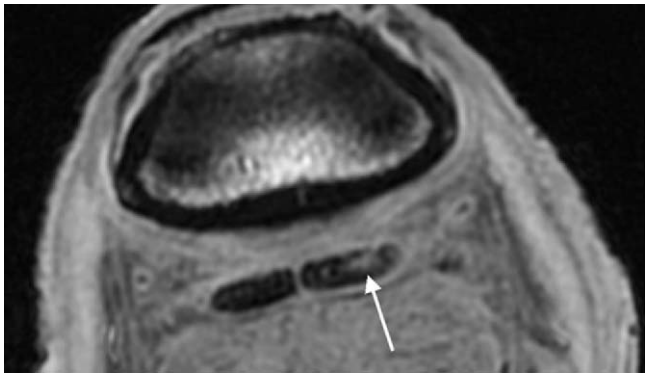

FIGURE 10-46 Transverse 3D T1-weighted SPGR image of the deep digital flexor tendon of a horse that underwent a palmar digital neurectomy 3 months previously. There is a focal area of high intensity signal in the core of the lateral lobe of the tendon (*white arrow*).

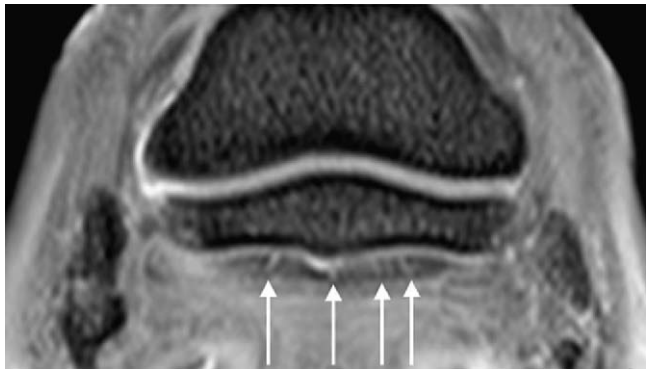

FIGURE 10-47 Detail of transverse 3D T2* GRE image with fat saturation at the level of the navicular bone of a lame foot. There are multiple, short linear areas of high signal intensity arising from the dorsal surface of the deep digital flexor tendon (DDFT) and coursing into the body of the DDFT in a palmar direction, reflecting dorsal fibrillation of the DDFT. The dorsal surface of the DDFT is irregular (*white arrows*).

reconstruction, truncation, and chemical shift artifacts. Finally, there is a normal depression in the palmar surface of the middle third of the sagittal ridge of the navicular bone in approximately 50% of normal horses. As synovial fluid becomes physically trapped in the depression, the difference between this normal depression and an early fibrocartilage lesion is very difficult to call.

DETECTABLE PATHOLOGIC LESIONS IN THE FOOT

Lesions of the Deep Digital Flexor Tendon

Lesions of the DDFT can best be described as core lesions, parasagittal splits, dorsal surface fibrillations, insertional lesions (including enthesopathy), and multifocal tendinitis. Core lesions are commonly identified in the region just proximal to the navicular bone (Figure 10-46) or near the insertion, sometimes extending the entire distance from the distal aspect of the digital sheath to the insertion onto the distal phalanx. Dorsal fibrillations (Figure 10-47) are most often observed at the level of the navicular bone and can be very hard to identify, especially in the early stages of degeneration. Parasagittal splits (Figure 10-48) appear to occur at any level and are not always associated with lameness. Insertional enthesopathy may be demonstrated by cortical irregularities, thickening of the tendon with or without clear tendon signal alterations, or small focal core lesions limited to the most distal portion of the DDFT (Figure 10-49). Swelling of the affected tendon lobe and high signal intensity on T2-weighted and fat-suppressed images seems common in the acute stages of all types of tendon lesions.

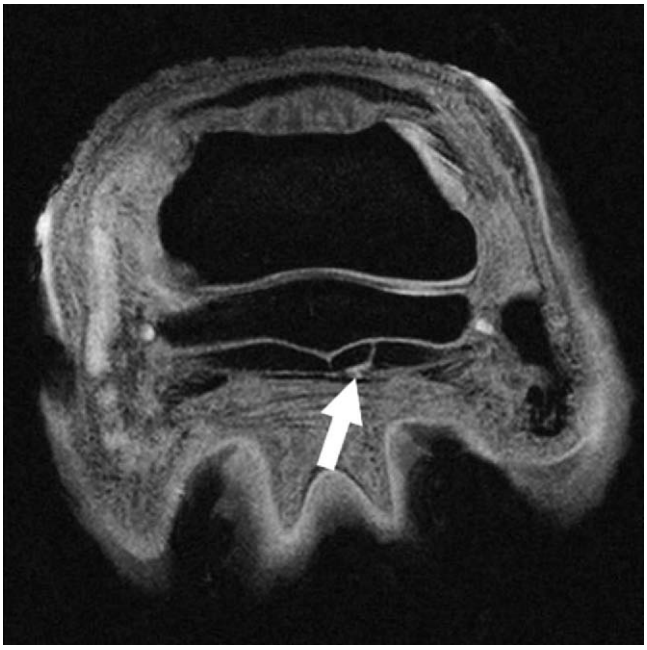

FIGURE 10-48 Transverse 3D FLASH GRE image with fat saturation at the level of the navicular bone of a lame right foot. There is a (para)sagittal linear area of high intensity signal in the lateral lobe of the deep digital flexor tendon, reflecting the presence of a full-thickness abaxial split in the lateral lobe (*white arrow*).

Lesions of the Impar Ligament

Due to the presence of penetrating synovial invaginations and small blood vessels, the structure of the distal sesamoidean impar ligament (DSIL) is quite variable between and within individuals. However, clear asymmetry and loss of fiber pattern seem to be associated with pathologic changes. Clear pathologic findings include marked thickening, extensive adhesion of the palmar surface of the impar ligament to the dorsal surface of the DDFT, cortical irregularity caused by bone proliferation, and lysis at the insertion of this ligament (Figure 10-50). Impar ligament lesions can also be recognized as a focal increase in signal intensity at its origin on the distal border of the navicular bone or by the presence of distal border fragments or mineralization within the proximal part of the ligament. Adhesions to the DDFT appear to occur with chronic pathologic conditions of the navicular bursa or DDFT.

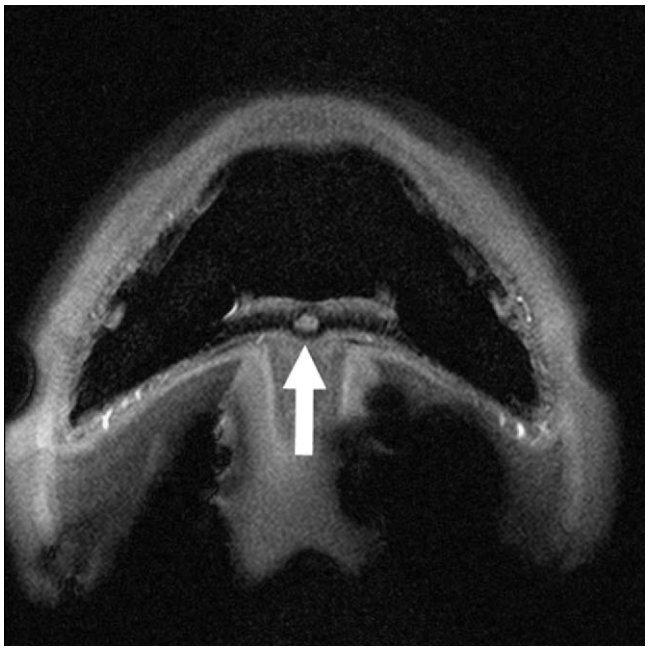

FIGURE 10-49 Transverse 3D FLASH GRE image with fat saturation at the level of the insertion of the deep digital flexor tendon (DDFT) to the flexor surface of the distal phalanx in a lame left foot. There is a circular focal area of high intensity signal in the insertion of the DDFT, exactly on the midline, reflecting the presence of a core lesion (*white arrow*).

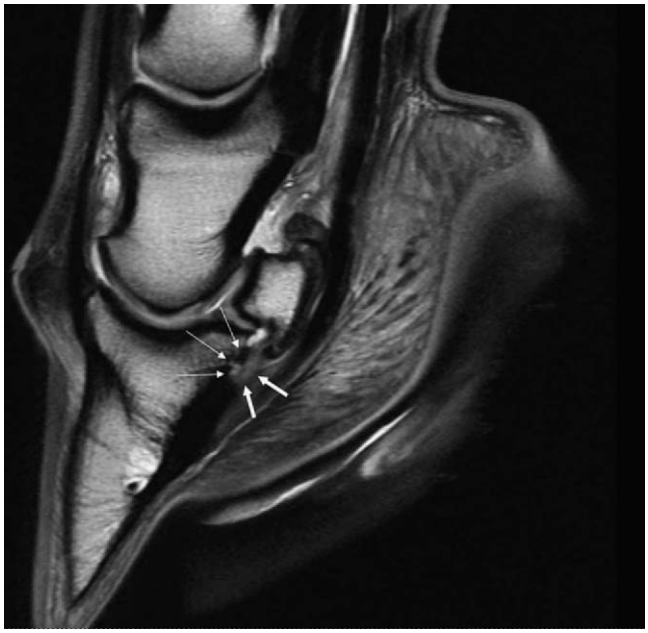

FIGURE 10-50 Sagittal 2D proton density image in the midsagittal region of a lame foot. There is thickening of the impar ligament. The palmar surface of the impar ligament and dorsal surface of the DDFT are adhered to each other (*large arrows*). There is entheseous new bone formation and focal osteolysis at the insertion of the impar ligament to the distal phalanx (*thin arrows*).

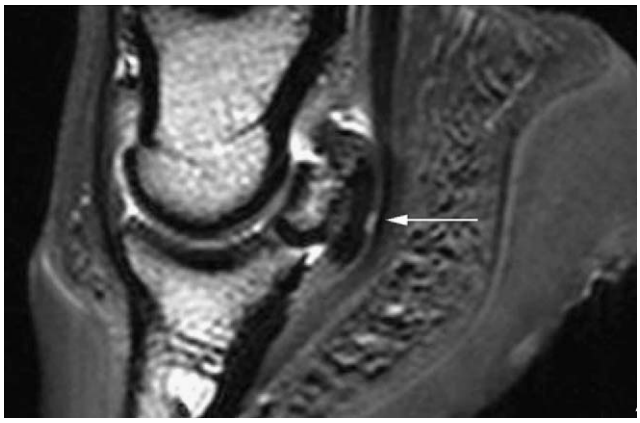

FIGURE 10-51 Sagittal 3D T2* GRE image of the navicular bone of a lame limb, with a focal accumulation of synovial fluid indicating a depression in the (fibrocartilage of the) flexor surface (*white arrow*).

Lesions of the Navicular Bone

Magnetic resonance images of the navicular bone in horses with foot pain show a range of abnormalities. The most common type of abnormality seen in cases of chronic foot lameness is increased signal intensity involving the palmar aspect of the navicular bone reflecting fluid accumulation (Figure 10-51). This can be a very subtle, focal increase at the site of early fibrocartilage loss and thinning, best seen on PD- or T2-weighted images. Increase in signal intensity can also be more generalized, extending deeper within the cortical bone of the flexor cortex, which is best seen on fat-suppressed images. In affected horses, there is often concurrent irregularity of the normally smooth endosteal surface of the flexor cortex, especially at the site of focal signal increase. Fibrocartilage degeneration is frequently accompanied by fibrillation of the dorsal aspect of the DDFT.

Horses with so-called navicular edema (Figure 10-52), more generalized high fluid signal throughout portions of the medulla of the navicular bone, may have primary navicular pathologic lesions without a DDFT lesion, although navicular medullary edema is in most horses associated with degenerative changes of the flexor border (fibrocartilage and/or cortex). In some cases, no radiographic abnormality is detectable in such horses, although most have increased radiopharmaceutical uptake in the navicular bone on scintigraphy. Advanced cystic lesions involving the flexor border are virtually always associated with adhesions to the adjacent DDFT (Figure 10-53) but may not be easily detected radiographically.

Another form of navicular bone disease is undoubtedly caused by the presence of distal border fragments (Figure 10-54). Especially when the fragments are large, relative motion between the fragments and parent bone appears to initiate a remodeling response that is painful. In cases in which distal border fragmentation is accompanied by bone edema, cortical irregularity, focal medullary sclerosis, and sometimes DSIL damage, pain does seem to be related to this site. Pain may be attributable to another cause when these magnetic resonance alterations are absent. In the case of navicular bone fracture, the fracture configuration can be demonstrated on

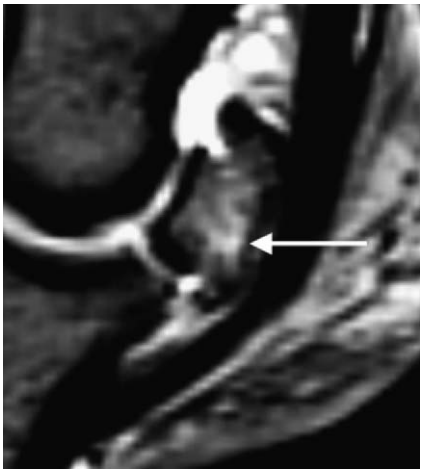

FIGURE 10-52 Sagittal short-T1 inversion recovery image of the navicular bone of a lame limb shows diffuse high-intensity signal in the medulla (*white arrow*) close to the subchondral bone plate of the flexor cortex, reflecting navicular bone edema.

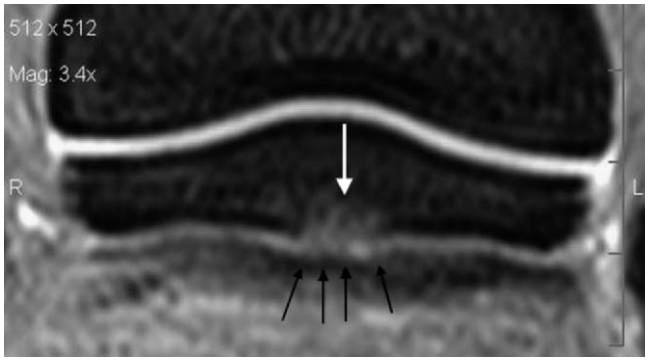

FIGURE 10-53 Transverse 3D T2* GRE image with fat saturation of a lame foot. There is discontinuity of the flexor cortex of the navicular bone with medium high intensity signal in the subchondral bone (*white arrow*). There are continuous strands of medium high signal adhesions between the dorsal surface of the deep digital flexor tendon and the palmar surface of the navicular bone (*black arrows*).

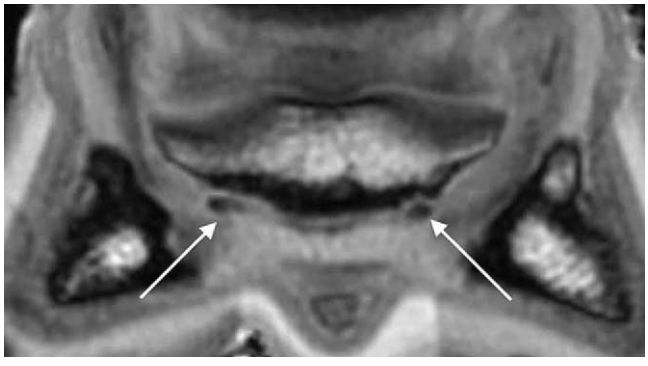

FIGURE 10-54 Frontal 3D T1-weighted SPGR image of the navicular bone of a lame foot. There are two focal areas of low signal intensity at the angles of the horizontal distal border with the sloping borders of the navicular bone, representing two osseous distal border fragments *(arrows)*.

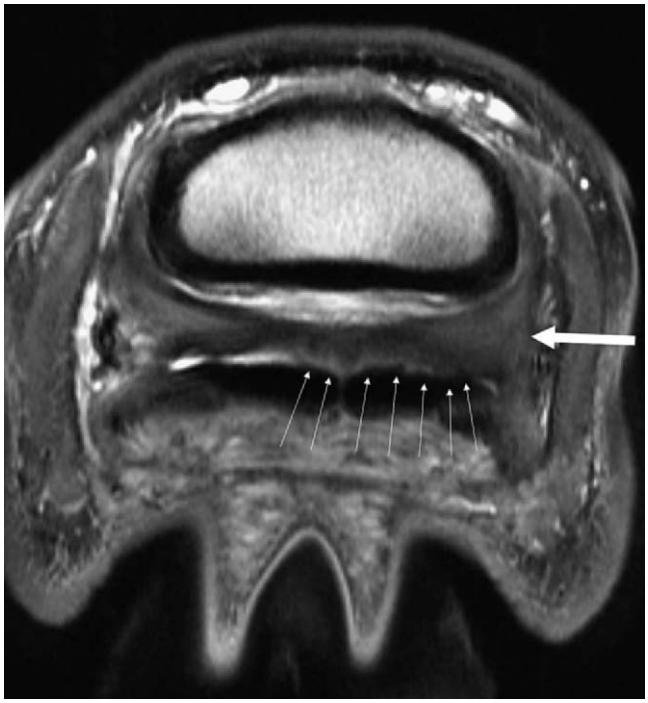

FIGURE 10-55 Transverse 2D proton density image of a lame right front foot. There is thickening of the lateral navicular suspensory ligament (*large arrow*). The dorsal surface of the lateral lobe of the deep digital flexor tendon is extensively adhered to the palmar surface of the lateral navicular suspensory ligament (*small arrows*).

three-dimensional images, and any associated damage to the DDFT can be observed.

Lesions of the Navicular Suspensory Ligament

Damage to the navicular suspensory ligaments (NSL; also collateral sesamoidean ligaments [CSL]) may be evident as shape alteration with signal heterogeneity in the body of the ligament. However, the examiner should keep in mind that signal heterogeneity is a common normal variation, presumably due to either differences in fiber orientation or fibrocartilaginous metaplasia within the ligament. In addition, it is worth remembering that most size and shape changes of the ligament are not due to alterations within the ligament per se, but to thickening of the navicular bursal lining covering the ligament or to adhesion formation with the fibrillated dorsal surface of the DDFT (Figure 10-55). Injury to the ligament itself in isolation is rare. More commonly, injury is seen in association with other pathology of the podotrochlear apparatus. Thickening of the lateral or medial NSL is most easily identified when the opposite NSL is not affected.

Lesions of the Collateral Ligaments of the Distal Interphalangeal Joint

The collateral ligaments (CLs) of the DIP joint may be difficult to image ultrasonographically except near the origin, but MRI has demonstrated that damage can occur at any part. Normal CLs can be seen clearly as well-delineated structures of homogeneous low signal on high-field MRI scans. Damage seems to be associated with increased cross-sectional area,

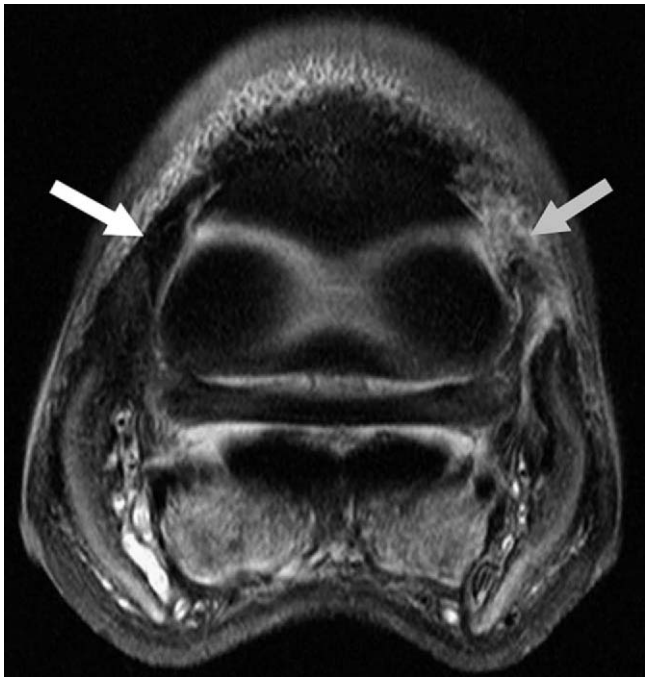

FIGURE 10-56 Transverse (horizontal) two-dimensional proton density image of a lame right front foot. The normal medial collateral ligament (*white arrow*) is characterized by its well-defined contour and homogeneous low signal strength. The injured lateral collateral ligament is uniformly hyperintense throughout its cross-sectional area and the contour is poorly defined (*gray arrow*).

irregular contour, and increased signal intensity. As there is often adaptive asymmetry in size, altered signal intensity and contour seem to be more selective criteria (Figure 10-56). Damage at the origin and insertion of these ligaments may also appear as signal alterations in the bone. Horses with injury of the body of a CL or at the origin or insertion may have focal increase in radiopharmaceutical uptake on scintigraphy. There is an increasing realization when using low-field systems that increase in signal intensity may be a frequent incidental finding in one collateral ligament when compared to the opposite collateral ligament, especially near the origin of the ligament on transverse T1-weighted and PD sequences. Especially when the slice plane is not perfectly perpendicular to the long axis of the ligaments and exactly symmetrical between the lateral and medial ligaments, signal alterations can occur as a consequence of difference in fiber direction relative to the main magnetic field. Even if these conditions are fulfilled, both collateral ligaments are not always oriented in the same plane, which again may give rise to asymmetry in signal intensity between lateral and medial collateral ligaments. Unless the changes are obvious and accompanied by both MRI and scintigraphic evidence of bone remodeling, it can be difficult to be certain about the clinical significance of the MRI finding of signal increase in a CL.

Lesions of the Distal Interphalangeal Joint

In the DIP joint, cartilage and subchondral bone damage can be detected by an altered surface contour and signal increases, as well as by synovial changes. Altered surface contour of cartilage can only be assessed accurately in areas where the articular surface is perfectly flat, as any obliquity of the surface will result in volume averaging across the width of the MRI slice. Signal changes in cartilage are not easily recognizable, and careful slice-per-slice comparison with the contralateral limb is necessary. Subchondral cyst–like lesions or osteochondral indentations can occur in sound limbs and may not necessarily be associated with pain. The thickness and delineation of the subchondral plate can be a useful reflection of what is occurring in the overlying cartilage. Chronic cartilage damage can result in thickening and irregular outline of the subchondral bone.

Lesions of the Distal and Middle Phalanges

Osseous trauma to the phalanges is most often seen on an MRI scan as a focal high medullary signal intensity on fat-suppressed images and low signal intensity on T1-weighted images, which reflects increased fluid content. Increased radiopharmaceutical uptake frequently occurs in the affected region. In the authors' experience, if there is no concurrent soft tissue damage, most of these injuries resolve with rest. Occasionally only sclerosis (low signal on T1-weighted and T2-weighted images) is seen in more chronic cases, without evidence of increased fluid. Fracture of the phalanges may be detected on MRI even if it has not been identified radiographically due to the orientation of the fracture plane or the incomplete or microscopic nature of the fracture. Likewise, changes in the distal phalanx and laminae related to chronic laminitis, which are not evident on radiographs, can be detected on MRI scans and allow for a more comprehensive evaluation of the patient.

Other Abnormalities

In the pastern region, distal sesamoidean desmitis is observed as increased signal intensity with swelling, although it pays to be aware that almost every horse has a central area of hyperintensity near the insertion of the straight sesamoidean ligament on the middle scutum. Proximal interphalangeal joint and collateral ligament pathologic lesions are detectable. Distal sesamoidean and collateral ligament abnormalities diagnosed with MRI are often undetectable using ultrasonography. In addition to the assessment of horses with confirmed foot pain of undiagnosed cause, MRI has proved helpful in the evaluation of horses with unresolved lameness after penetrating injuries to the foot.[39,40] Damage to soft tissues such as the DDFT, incurred at the time of injury, can result in long-term pain even after resolution of an initial infection and healing of the discharging tract. Isolated cases of desmitis (asymmetric thickening) of the distal annular ligament of the pastern and its proximal and distal attachments, the proximal ligament of the digital cushion, the chondrocompedal ligament, and the chondrosesamoidean ligament have also been observed.

Lesions that Are Poorly Detectable with Magnetic Resonance Imaging

Some tissue abnormalities do not show up well on MRI scans. Although some marked subsolar abscesses or bruises can result in laminar and osseous signal increase in the solar region of the foot, it has been the first author's observation that many horses with solar pain responsive to application of hoof testers have an unremarkable foot when viewed with

MRI. Similarly, horses with poor dorsopalmar foot balance resulting in palmar heel pain often have unremarkable MRI scans. Early hyaline or fibrocartilage degeneration in the DIP joint or navicular flexor surface as well as mild fibrillation of the dorsal surface of the DDFT in the navicular bursa can also be very difficult to identify. Finally, the resolution of low-field MRI systems is usually not sufficient to identify accurately the DSIL and signal irregularities associated with injury to this structure.

CASE SELECTION

Magnetic resonance imaging is not a screening tool. The interpretation of the significance of MRI signal irregularities can be difficult at best and is not helped by the absence of knowledge regarding the precise character of the lameness and localization of pain. Therefore MRI is not a substitute for a careful and complete clinical examination of the foot (with hoof testers), including a lameness examination evaluating the response to intraarticular anesthesia of the DIP joint, intrathecal anesthesia of the navicular bursa, a palmar digital nerve block, and an abaxial sesamoid nerve block. Magnetic resonance imaging is of greatest value for imaging tissues that cannot be evaluated effectively using other diagnostic imaging modalities. In conjunction with scintigraphic evaluation, MRI can provide further information on the stage and activity of a lesion and therefore its clinical significance.

CLINICAL PATIENT STUDY

Between January 2001 and December 2003, clinical MRI scans were performed of the feet of 199 horses (191 forelimbs and 8 hindlimbs) presented for investigation of foot lameness at the Animal Health Trust (Newmarket, United Kingdom).[41] Horses were included in the study if lameness was abolished by an abaxial sesamoid nerve block and the results of clinical, radiographic, and ultrasonographic examinations did not satisfactorily explain the degree of lameness. Feet were examined by MRI using a human extremity radiofrequency coil, in the isocenter of a short-bore, flared-end 1.5-T GE Signa Echospeed magnet. The clinical significance of lesions was based on their MRI appearance and the results of local analgesia, radiography, ultrasonography, and nuclear scintigraphy. Between one and six signal abnormalities were reported in each horse, with an average of 3.3 abnormalities per horse. Abnormalities were also detected in the contralateral limb in some horses. In the majority of horses it was possible to ascribe lameness to one or two major injuries, although in some of these, minor concurrent lesions were identified of questionable significance. Common nonspecific MRI findings in lame limbs included the following:

- Distension of the DIP joint, with or without intraarticular soft tissue proliferation
- Distension of the navicular bursa, which was often distended in lame limbs, with or without intrathecal soft tissue proliferation
- Mildly increased medullary signal intensity in the navicular bone in fat-suppressed images, especially toward the palmar half of the bone
- Fluid-filled outpouchings from the DIP joint of variable size, interdigitating between the fascicles of the DSIL
- Minor focal adhesions between the DDFT and the DSIL
- Endosteal irregularity in the distal phalanx at the sites of insertion of either the DDFT or the DSIL
- Focal, incomplete sagittal plane splits or minor irregularities in the dorsal contour of the DDFT
- Mineralization of a collateral cartilage of the distal phalanx
- Apparently inactive osseous cyst–like lesions in the distal aspect of the proximal phalanx or proximal aspect of the middle phalanx (five horses)
- Congenital defects in the center of the proximal articular surface of the distal phalanx, unassociated with any other bony reaction (three horses)
- Unilateral or bilateral laminitis in addition to another primary cause of lameness (three horses)

Table 10-8 Magnetic Resonance Imaging Findings in 199 Horses with Foot Pain

Lesion	Primary, %	Total, %
DDFT	33	60
DDFT and navicular bone	14	18
Collateral ligament DIP joint	15	31
Navicular bone	4	22
DSIL	5	10
DIP joint	2	3
Multiple injuries	17	17
Traumatic injury P2 or P3	7	7
DSL	0.5	0.5

DDFT, Deep digital flexor tendon; *DIP,* distal interphalangeal; *DSIL,* distal sesamoidean impar ligament; *DSL,* distal sesamoiden ligament.

Injuries that were considered significant primary causes of lameness are listed in Table 10-8.

Deep digital flexor tendonitis was by far the most common primary injury (33%). The total number of horses with abnormalities of the DDFT was even larger (60%), when the horses with combined injuries that included the DDFT were considered. The second most common injury was desmitis of a CL of the DIP joint (15%). The total number of horses with CL injury increased to 31% when horses with multiple injuries that included the DIP collateral ligament were added. Another common finding was the presence of a combination of different injuries, including the DDFT, the DIP collateral ligament, the navicular bone and its ligaments, the DIP and proximal interphalangeal joints, and the middle and distal phalanges. All other injury categories had an incidence of less that 10%. Primary injuries of the DSIL were suspected in 5% of patients. The normal regular fascicular structure of the ligament was disrupted, and sometimes enlarged, with increased signal in T1- and T2-weighted images and sometimes also in fat-suppressed images. The palmar border was irregular and in some horses the enlarged DSIL was in close apposition to the DDFT. Remarkably, a mere 4% of horses had only navicular bone abnormalities, although another 14% had combined lesions of the DDFT and the navicular bone. Navicular bone abnormalities in the

presence of DDFT lesions usually consisted of extensive hyperintense signal in the navicular bone in fat-suppressed images. DIP joint disease was the least common diagnosis, made in only 2% of patients. Lesions of the navicular suspensory ligaments (NSLs) were never encountered as primary injuries.

Follow-up MRI was performed after at least 6 months' rest in five horses with primary tendonitis of the DDFT. In all five horses, hyperintense signal was still present in the DDFT in both the T1- and T2-weighted sequences, even though none of them still showed evidence of lameness. It therefore appeared difficult to establish the age of a tendon lesion with MRI or to determine when a sound horse with a previous tendon injury can safely resume work.

PATHOLOGIC VALIDATION OF MAGNETIC RESONANCE IMAGING OF THE FOOT

To understand better the nature of the lesions responsible for foot pain and the ability of MRI to detect it, pathologic conditions of the horse's foot and their relationship with magnetic resonance signal characteristics must be investigated carefully and meticulously. Only then will it be possible to improve interpretation skills, avoid the pitfalls of misdiagnosis, and use MRI to its full potential.

Thirty-four limbs were collected from 18 horses (Group L) in which a diagnosis of navicular syndrome or palmar foot pain was based on lameness of at least 2 months' duration, elimination of lameness by a palmar digital nerve block, and absence of obvious other causes of foot pain.[42-45] Twenty-five limbs were also collected from 15 horses without evidence or history of front foot lameness (Group N). All feet were examined, following the same MRI protocol as in the clinical study. Dissection of each foot was performed immediately after MRI, and macroscopic and histopathologic findings were recorded for the DDFT, NSL, DSIL, DIP collateral ligament, fibrocartilage of the navicular bone, navicular bone, synovial membrane of the DIP joint, and synovial membrane of the navicular bursa. MRI findings were compared with pathologic findings for each structure. The pathologic findings that were statistically more frequently encountered in the lame group than the nonlame group are summarized (Table 10-9). The results of the pathologic examination produced a clear pattern of abnormalities. The significant changes associated with palmar foot pain were confined to the structures that outline the navicular bursa, a feature that has previously been described as classic navicular disease.[45] Abnormalities of the DDFT were most commonly present concurrently with lesions of the flexor surface of the navicular bone. The previously discussed clinical MRI study of horses with palmar foot pain,[41] however, proposed a much higher incidence of primary tendonitis of the DDFT and collateral desmitis of the DIP joint, independent from abnormalities of the navicular bone. This is best explained by the fact that horses undergoing clinical MRI rarely had any radiological changes of navicular disease, and therefore the advanced stage of classical navicular disease was clearly underrepresented in the clinical MRI study. In addition, an association has been established between the use of horses for jumping purposes and the incidence of injuries to the DDFT in the foot (S. Dyson, personal communication, 2004). Other clinical MRI studies have encountered a much higher incidence of primary navicular bone abnormalities and lower incidence of tendonitis of the DDFT.[47] It is likely that these discrepancies are related to population differences between veterinary hospitals in different parts of North America and Europe. A patient population with a high proportion of Warmbloods used for show jumping might be expected to result in a higher incidence of MRI abnormalities of the DDFT, whereas a population with a high proportion of Quarterhorses may result in a predominance of navicular bone abnormalities.

Table 10-9 Summary of Pathologic Findings in the Lame Group versus the Nonlame Group

	Normal Horses		Navicular Disease	
Gross Features	***N=25***	**%**	***N=34***	**%**
NAVICULAR BONE				
Fibrocartilage loss	4/25	16	14/34	41
Flexor cortex erosions	2/25	0	15/34	44
Distal border fragments	3/25	12	10/34	29
DDFT				
Dorsal fibrillations	0/25	0	16/34	47
Adhesions	0/25	0	10/34	29
Parasagittal split	0/25	0	10/34	29
Core lesion	0/25	0	7/34	20
Core/split insertion	0/25	0	7/34	20
Adhesion to impar	0/25	0	9/34	26
Abnormalities DDFT (total)	1/25	4	21/34	62
NAVICULAR BURSA				
Synovial proliferation	0/25	0	8/34	23
NAVICULAR SUSPENSORY LIGAMENT				
Adhesions to DDFT	0/25	0	10/34	29
IMPAR LIGAMENT				
Insertion entheseophytes	0/25	0	6/34	18
Osseous fragments	3/25	12	10/34	29

DDFT, Deep digital flexor tendon.

The sensitivity and specificity of MRI was investigated for abnormalities of the navicular bone, DDFT, impar ligament, navicular suspensory ligaments, and DIP joint encountered in the pathologic study.[43] In general, alterations in magnetic resonance signal intensity and tissue contour of the structures examined during this pathologic study reliably represented changes in tissue structure detectable using gross and histopathologic examination.

Magnetic resonance imaging assessment had a poor sensitivity (36%) but high specificity (100%) for gross evidence of partial or complete loss of fibrocartilage from the flexor surface of the navicular bone (Figure 10-57). The sensitivity (100%) and specificity (97%) for the presence of a midridge depression (synovial fossa) was high. The distinction between fibrocartilage loss and the presence of a normal depression was sometimes difficult on MRI scans because the main MRI characteristic of both consisted of an accumulation of high intensity signal bursal fluid by the

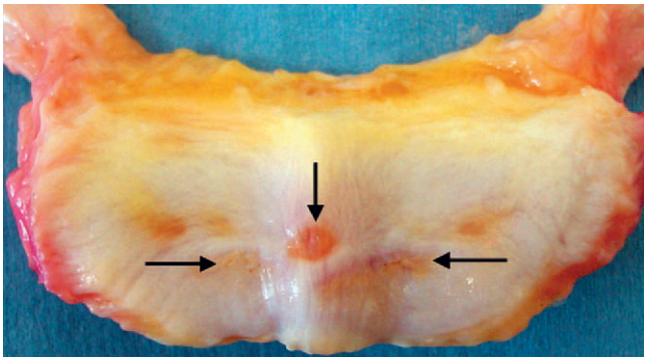

FIGURE 10-57 Palmar view of the same navicular bone as in Figure 10-51 with a mid-ridge depression and two areas of fibrocartilage loss on both sides of the sagittal ridge (*black arrows*).

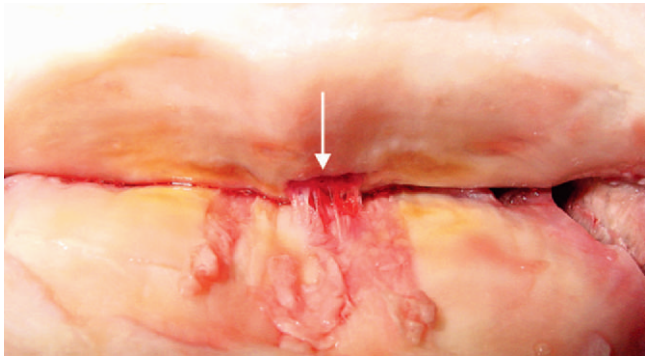

FIGURE 10-58 Proximodistal view of the navicular bursa of the same foot as Figure 10-53, with a full-thickness flexor cortex erosion and adhesions between the palmar surface of the navicular bone and the dorsal surface of the deep digital flexor tendon (*white arrow*).

indentation in the flexor surface of the navicular bone. The sensitivity and specificity for the presence of macroscopic partial or complete flexor cortex erosions were excellent.

When MRI findings were compared with histologic findings, all focal, saucer-like lesions of greater than 50% depth of the fibrocartilage on histologic examination were characterized by adjacent fluid accumulation, endosteal irregularity, and irregularity of the osteochondral junction on MRI. More superficial lesions were less consistently identified. Evidence of adhesion to the DDFT was observed as loss of navicular bursa fluid signal, cortical defects, and apparent continuity of tissue between the DDFT and navicular bone (Figure 10-58). The overall sensitivity of MRI for all histologic abnormalities of the flexor surface was good (83%), but the specificity was fair (65%). All navicular bones with severely high medullary signal intensity on fat-suppressed images and low signal intensity on spoiled gradient echo (SRGR) images had corresponding histologic evidence of focal or generalized medullary osteonecrosis and fibrosis (Figure 10-59). The sensitivity (92%) and specificity (93%) of MRI for macroscopically visible distal border fragments was high (Figure 10-60).

In regard to pathologic lesions of the DDFT, MRI examination had a fair sensitivity (74%), but an excellent specificity (93%) for fibrillations and erosions of the dorsal surface (Figure 10-61) of the DDFT in the area of the navicular bursa identified on necropsy. The sensitivity and specificity of

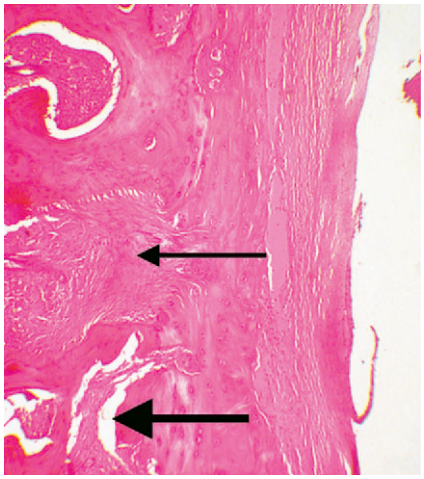

FIGURE 10-59 Stain of the flexor cortex and medulla of the same bone as Figure 10-52. There is evidence of focal osteonecrosis (*wide arrow*) and fibrosis in the intertrabecular spaces (*narrow arrow*) adjacent to a defect in the flexor surface, where fibrocartilage and cortical bone have been replaced by fibrous tissue. (Haematoxylin and eosin stain; original magnification ×100.)

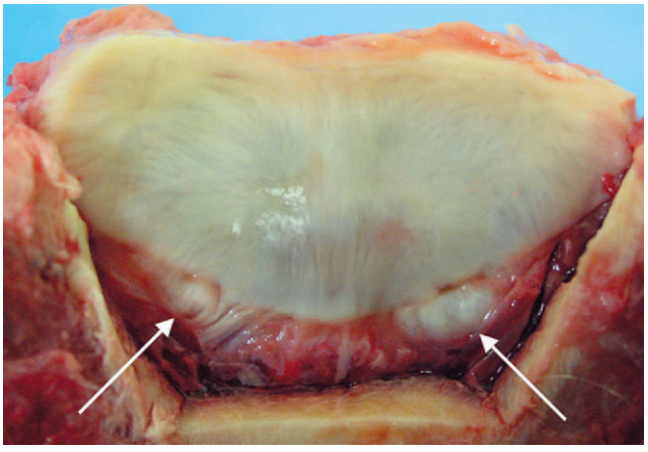

FIGURE 10-60 Palmar view of the navicular bone of the same foot as Figure 10-54, after removal of the deep digital flexor tendon. There is an osseous body (distal border fragment) within the insertion of the distal sesamoidean impar ligament (DSIL) at each angle of the distal border of the navicular bone with the sloping borders *(arrows)*.

MRI was excellent or good for all other lesions of the DDFT. Histologically, all feet with mild, moderate, or severe irregularity of the dorsal tendon surface on MRI were characterized by superficial dorsal fibrillation, crevicing, or splitting, thereby confirming the excellent specificity of MRI for recognition of dorsal lesions. Parasagittal splits (Figure 10-62) on MRI matched with histologic descriptions of dorsal crevices, dorsal ridges, and multiple crevices of the appropriate depth with respect to MRI. Small core lesions on MRI were seen histologically as focal areas of pale tendon fascicles, increased cellularity, pale septa, and increased blood vessels in the septa. Larger core lesions (Figure 10-63) on MRI scans were seen histologically as obliteration of normal tendon fascicle structure, markedly increased vascularization, highly cellular matrix, vacuolization, loss of normal septal divisions, and fibrocartilaginous metaplasia in all feet.

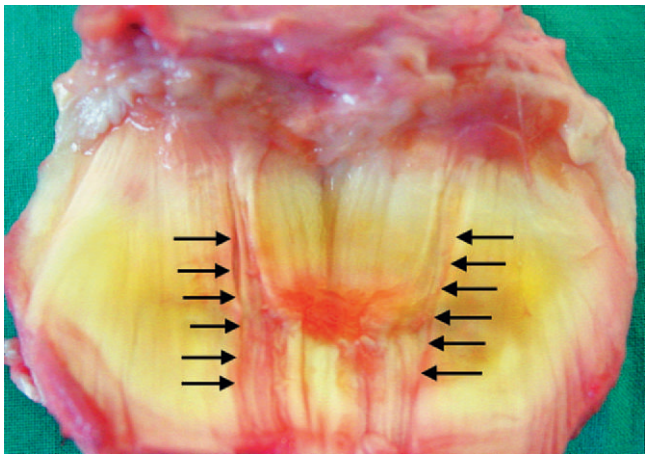

FIGURE 10-61 Frontal view of the deep digital flexor tendon (DDFT) at the level of the navicular bursa in the same foot as Figure 10-47. There are two vertical linear fibrillations/erosions on the dorsal surface of the DDFT with some loose tendon fibers on the exposed surface of the fibrillations (*black arrows*). Remnants of severed adhesions between the dorsal surface of the DDFT and the palmar surface of the navicular bone are visible in the center of the DDFT.

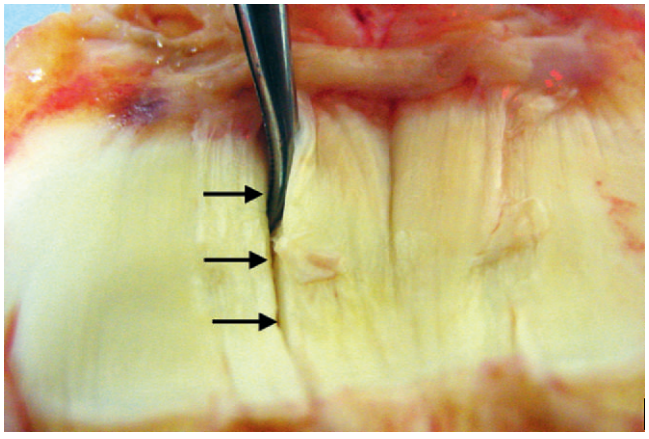

FIGURE 10-62 Frontal view of the deep digital flexor tendon at the level of the navicular bursa of a lame foot. There are fibrillations on the dorsal surface of both tendon lobes. There is a full-thickness parasagittal split in the lateral lobe, through which forceps can be passed to the palmar surface of the tendon (*black arrows*).

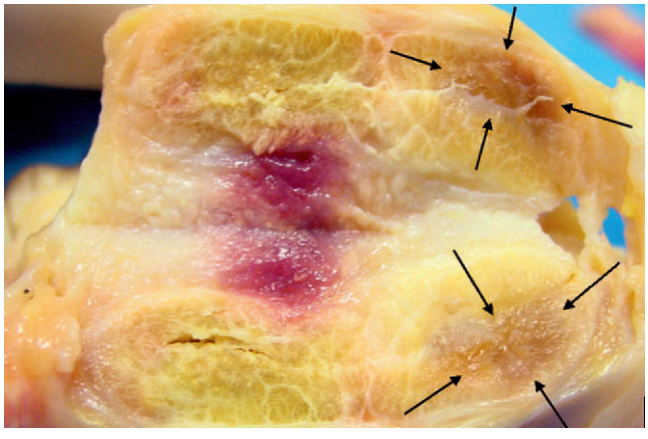

FIGURE 10-63 Transected surfaces of the deep digital flexor tendon (DDFT) of the same foot as in Figure 10-46, proximal to the level of the navicular bursa. There is evidence of a core lesion in the lateral lobe of the DDFT, characterized by dark discoloration (*black arrows*).

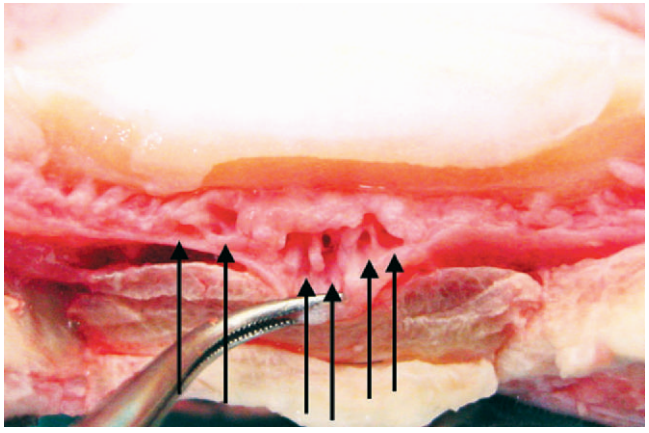

FIGURE 10-64 Palmar view of the transected surfaces of the impar ligament and deep digital flexor tendon within 1 cm of their insertions to the flexor surface of the distal phalanx. There are multiple empty spaces between the concentrated fiber bundles of the distal sesamoidean impar ligament into which synovial outpouchings of the distal interphalangeal joint and the navicular bursa protrude (*black arrows*).

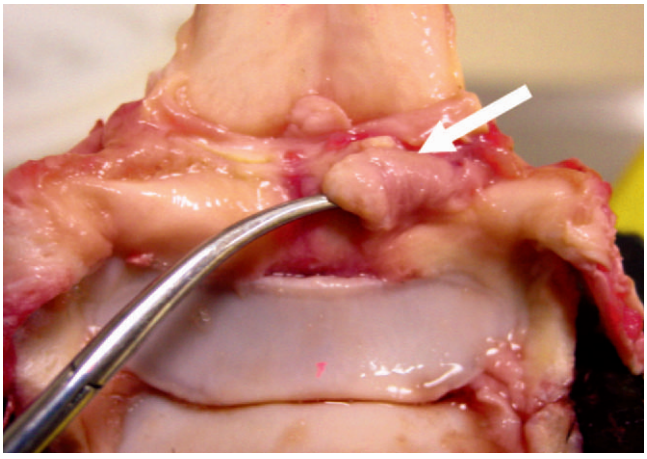

FIGURE 10-65 Dorsal view of the navicular bone, the distal phalanx, and navicular suspensory ligament. There is a protruding cystic structure on the proximal border of the navicular suspensory ligament (*white arrow*), that contained synovial fluid and a synovial membrane lining.

Histologically, limbs with moderate to severe signal irregularities of the DSIL on MRI were characterized by large intraligamentous synovial pockets, prominent vascularity, and fibrocartilaginous metaplasia. The overall sensitivity of MRI for histologic abnormalities of the impar ligament was good (80%), but the specificity was poor (50%). This was mainly due to the fact that intraligamentous synovial spaces are part of the normal anatomy of the impar ligament (Figure 10-64). MRI detected gross periligamentous tissue proliferation, adhesion formation between the DDFT and the NSL, and cysts in the NSL (Figure 10-65) with high levels of sensitivity. Histologically, moderate to severe signal heterogeneity was generally associated with fibrocartilaginous metaplasia and occasionally with blood vessel occlusion or increased vascularization within the NSL. Focal high-signal intensity in the NSL was associated with synovial in-pouchings and hyperplasia in three limbs. MRI had a poor sensitivity for

gross evidence of periarticular osteophytes and a fair sensitivity for articular cartilage abnormalities in the DIP joint.

COMPARISON OF HIGH-FIELD AND LOW-FIELD MAGNETIC RESONANCE IMAGING FOR THE FOOT

Both high-field and low-field MRI units are currently in clinical use for diagnosis of the causes of foot lameness in horses. High-field MRI uses a superconductory magnet, with a field strength of 1.0 to 1.5 T, that requires constant cooling with helium. Low-field MRI is performed in the horse with a permanent, open magnet with a field strength of 0.21 to 0.3 T. High-field units produce a high-resolution image with overwhelming anatomical detail. The cost of purchasing and maintaining a high-field MRI unit is high, and its availability is therefore limited in equine practice. In addition, high-field MRI requires the patient to be placed under general anesthesia, which increases the cost and risk of the procedure. Low-field MRI produces a lower signal-to-noise ratio, resulting in reduced resolution of the images, but is still capable of producing diagnostic-quality images of the distal limb. An open low-field MRI scanner designed specifically for imaging distal limbs of standing horses has been in clinical use since 2002 (Hallmarq Equine Limbscanner; Hallmarq Veterinary Imaging, Guildford, United Kingdom). Between January 2004 and May 2005, this apparatus was used to perform clinical low-field MRI scans of the feet of 170 horses that were examined for foot lameness at the Liphook Equine Hospital in the United Kingdom.[48] Horses were included in the study if lameness was abolished by an abaxial sesamoid nerve block and the results of clinical, radiographic, and ultrasonographic examinations did not satisfactorily explain the degree of lameness. The feet were examined by MRI using a purpose-designed equine foot radiofrequency coil, in an open, permanent 0.27-T magnet (Hallmarq Veterinary Imaging). Sagittal, dorsal, and transverse MRI scans were obtained using three-dimensional T1-weighted gradient echo, three-dimensional T2* gradient echo and short-T1 inversion recovery (STIR) sequences, with a slice thickness of 2.5 mm or 4 mm (STIR).

When the results were compared (Table 10-10) to the previously described diagnoses of 199 horses examined with a high-field magnet at the Animal Health Trust[41] (Table 10-11) during the same period, a striking similarity was observed between the diagnoses and their relative contribution to the overall number of horses in both studies.

Synovitis of the DIP joint was a more common finding using the low-field MRI unit. Primary injuries of the DSIL were more frequently suggested using high-field MRI. The lower resolution of the images obtained with low-field MRI made this area difficult to assess in low-field MR scans, and it is possible that some of the horses diagnosed with primary pathologic lesions of the DIP joint may actually have had DSIL injury that was not detected. In 6% of horses scanned using the low-field system, the results of MRI scans were classified as inconclusive. In these horses, either the lesions identified were considered to explain the severity of lameness inadequately, or the quality of the scans was compromised by movement artifact due to poor horse cooperation, which made diagnosis difficult. Of these 10 horses, 6-month follow-up was available for six. Four of these six horses were sound and back in full work within 3 months of the MRI scan. In the two other horses, severe foot imbalance was evident on clinical examination and both horses responded well to remedial farriery. Although none of the high-field MRI investigations was reportedly inconclusive, it was sometimes difficult to determine the significance of the frequently subtle and multiple signal alterations that were available on these high-resolution images. While clinical significance was ascribed to these subtle signal alterations on the basis of their MRI appearance as well as the results of local analgesia, radiography, ultrasonography, and nuclear scintigraphy, no postmortem data were available to confirm these conclusions.

Follow-up MRI was performed after 6 to 9 months of rest in five horses diagnosed using high-field MRI and 14 horses diagnosed using the low-field MRI with primary tendonitis of the DDFT. In all horses, hyperintense signal was still

Table 10-10 Low-Field Magnetic Resonance Imaging Findings in 170 Horses with Foot Pain

Lesion	Primary, %	Total, %
DDFT	34	52
DDFT and navicular bone	13.5	
Collateral ligament DIP joint	19	23
Navicular bone	8	12
DSIL	0.5	2
DIP joint	9	11
Multiple injuries	3.5	3.5
Traumatic injury to middle or distal phalanges	6	6
DSL	0.5	0.5
Inconclusive	6	6

DDFT, Deep digital flexor tendon; *DIP,* distal interphalangeal; *DSIL,* distal sesamoidean impar ligament; *P2,* middle phalanx; *P3,* distal phalanx; *DSL,* distal sesamoidean ligament.

Table 10-11 High-Field Magnetic Resonance Imaging Findings in 199 Horses with Foot Pain

Lesion	Primary, %	Total, %
DDFT	33	60
DDFT and navicular bone	14	18
Collateral ligament DIP joint	15	31
Navicular bone	4	22
DSIL	5	10
DIP joint	2	3
Multiple injuries	17	17
Traumatic injury to middle or distal phalanges	7	7
DSL	0.5	0.5
Inconclusive	0	0

DDFT, Deep digital flexor tendon; *DIP,* distal interphalangeal; *DSIL,* distal sesamoidean impar ligament; *P2,* middle phalanx; *P3,* distal phalanx; *DSL,* distal sesamoidean ligament.

present in the DDFT on the T1 and T2 weighted sequences, even if they showed no evidence of lameness. It therefore appeared difficult to establish the age of a tendon lesion with MRI to determine when a sound horse with a previous tendon injury can safely resume work.

The results of the low-field MRI scans described in this study showed that a similar range of lesions was encountered to those seen in high field MRI scans. These findings suggest that standing low field MRI is an effective technique for diagnosing pathologic lesions of the DDFT, the navicular bone, the ligaments of the navicular bone, and the collateral ligaments of the DIP joint. Although it is clear that low-field MRI is able to identify similar lesion types to the high-field systems, it remains unclear how the sensitivity of both systems compares. Experience of equine clinicians with MRI is limited, and image interpretation is generally extrapolated from knowledge of other imaging modalities or from human MRI. To improve our understanding of the nature of the lesions causing foot pain, the pathologic conditions of the horse's foot and their relationship with both low-field and high-field magnetic resonance signal characteristics must be investigated carefully and meticulously; only then will it be possible to improve interpretation skills, to avoid the pitfalls of misdiagnosis, and to use MRI to its full potential.

ULTRASONOGRAPHIC EVALUATION OF THE EQUINE FOOT

Jeevraj S. Grewal

Pain arising from the foot, particularly its palmar aspect, is a common cause of lameness in the horse. Navicular syndrome is a frequent cause of chronic forelimb lameness associated with pain arising from the navicular bone.[49-53] Diagnosis of navicular syndrome has traditionally been made on the basis of history, signalment, and the findings of lameness evaluation, including regional anesthesia of the limb.[49-53] Diagnostic procedures have historically been limited to radiographic evaluation of the bony structures within the foot; however, clinical experience has shown that lameness associated with navicular syndrome often precedes convincing radiographic abnormalities.[49-53] Navicular syndrome is not a single disease but is the product of a combination of conditions that result in pain arising from the foot. Soft-tissue abnormalities have been implicated in the development of navicular syndrome, yet the involvement of soft tissue may be difficult to confirm without specialized and expensive imaging modalities such as nuclear scintigraphy, computed tomography, and magnetic resonance imaging.[54-56] Contrast navicular bursography can provide valuable information, but this technique is invasive and introduces the risk of sepsis.[57]

In contrast, ultrasonography is an economically feasible and readily available method to assess some of the soft tissue and bony structures within the foot.[58,59] This method has not been widely used to diagnose the cause of foot lameness because of the perception that the hoof capsule does not allow penetration of an ultrasound beam. In addition, ultrasonographic evaluation of the foot can be complex, owing to the various soft tissues and bony structures that can contribute to a clinical lameness.

The purpose of this section is to describe a comprehensive and systematic approach to ultrasonographic evaluation of the foot. Following such an approach will make it easier for the veterinarian to accurately diagnose various combinations of pathologic conditions that can contribute to lameness isolated to the foot.

PATIENT PREPARATION

To prepare the foot for ultrasonographic evaluation, the frog is trimmed down to moist, pliable tissue. This step, which is the most important aspect of the foot preparation, must be performed before soaking the foot. The foot should be thoroughly cleaned using warm water and detergent. The foot is then incorporated in a wet foot bandage, and a plastic boot is placed over the bandage. For most horses, soaking the foot in warm water for 30 minutes is sufficient to adequately evaluate the foot using the transcuneal approach. Horses with exceptionally large, dry, and hard feet may require a warm-water soak with dish-washing detergent for up to 120 minutes.

The hair on the pastern should be clipped circumferentially from the midpastern level distally to the coronary band, using a No. 40 surgical clipper blade. The skin should then be thoroughly cleaned using warm water, followed by application of warm ultrasound coupling gel. The gel should be applied to the entire clipped area that is to be examined and worked in well, following the direction of the hair. Sterile gel should be used if an aseptic examination is necessary. Shaving the skin may be necessary, or preferable in some situations, to obtain optimal image quality.[60]

NORMAL EVALUATION

The comprehensive ultrasonographic evaluation of the foot includes examination of the foot in both weight-bearing and non–weight-bearing positions. The foot can be held in a non–weight-bearing position by either the veterinarian or an assistant. Having an assistant hold the foot is preferable, because this arrangement has the advantage of freeing up the veterinarian to operate the controls on the ultrasonography machine with the nonscanning hand. In the event the veterinarian is holding the foot in a non–weight-bearing position, a trained assistant is required to operate the controls on the ultrasonography machine.

Routine ultrasonographic evaluation of the foot should include an examination of the DIP joint, the collateral ligaments of the DIP joint, the navicular bone, the navicular bursa, the NSL of the navicular bone, the distal sesamoidean impar ligament (DSIL), the deep digital flexor tendon (DDFT), and the distal digital annular ligament (DDAL). Because the majority of pathologic foot changes occur in the forelimb, the terms *dorsal* and *palmar* are used in this chapter when referring to the anatomic location of structures.

The podotrochlear apparatus is made up of the navicular bone, the navicular bursa, the NSL, the DSIL, the DDFT, and the DDAL. The proximal aspect of the podotrochlear apparatus can be evaluated from the palmar aspect of the distal pastern at the level of the heel bulbs of the foot. A transcuneal approach for the ultrasonographic evaluation of the

podotrocheal apparatus has been described.[58,59,61] With this approach, the frog is used as an acoustic window to provide valuable information about the podotrochlear apparatus. A dorsal approach is used to evaluate the DIP joint and the collateral ligaments of the DIP joint. This approach uses the dorsal aspect of the coronary band and the periople as an acoustic window with the foot in a weight-bearing position.

WEIGHT-BEARING EXAMINATION

Distal Interphalangeal Joint

Anatomy

The DIP joint is a ginglymus formed by the junction of the middle and distal phalanges and the navicular bone. The surface of the distal end of the middle phalanx is convex in the sagittal direction and concave transversely. The articular surface of the middle phalanx slopes sharply proximally and dorsally; its central part is prominent and is flanked by two glenoid cavities. It is completed on the palmar aspect by the articular surface of the distal navicular bone.

The DIP joint capsule is attached around the margins of the articular surfaces. Dorsally and on the sides, the joint capsule is tight and combines with the extensor tendon and the collateral ligaments. The DIP joint capsule forms a substantial pouch palmary, which extends proximally to about the middle of the middle phalanx. At this level, a fibrous membrane separates the capsule from the digital synovial sheath. On each side, small pouches project against the collateral cartilages of the distal phalanx just palmar to the collateral ligaments. These pouches can be seen especially during palmar flexion.

Scanning Technique

The dorsal aspect of the DIP joint and the collateral ligaments of the DIP joint are evaluated through the periople of the foot, with the foot in a weight-bearing position at the level of the coronary band. A 7.5- to 10-MHz linear array transducer can be used to evaluate the dorsal compartment of the DIP joint just proximal to the coronary band. A standoff pad usually improves the quality of the image, but may not be necessary if the entire foot has been prepared adequately. The best diagnostic view is obtained in longitudinal section. The structures to be evaluated in this window are the attachments of the extensor tendon on the extensor process of the distal phalanx, the extensor process of the distal phalanx, the distal articular surface of the middle phalanx, and the synovial joint capsule (Figures 10-66 and 10-67). The acoustic character of the synovial fluid that is present should also be noted.

The palmar aspect of the DIP joint is evaluated with the foot in a non–weight-bearing position. A 5.0- to 7.5-MHz linear array microconvex transducer can be used with the distal palmar pastern as an acoustic window. A standoff pad is not recommended, since it decreases the contact between the transducer and the curved anatomy of the distal pastern and the heel bulbs. Instead, a diagnostic view can be obtained by soaking the foot and using ultrasound coupling gel, as was described previously. The joint capsule of the palmar pouch appears as a hyperechoic reflection within which there is anechoic fluid. In the ultrasonographic image, the deep boundaries of this proximal palmar reflection are marked by the curved palmar aspect of the middle phalanx proximally and the proximal aspect of the navicular bone distally. The navicular suspensory ligament marks the superficial boundary of the palmar pouch.

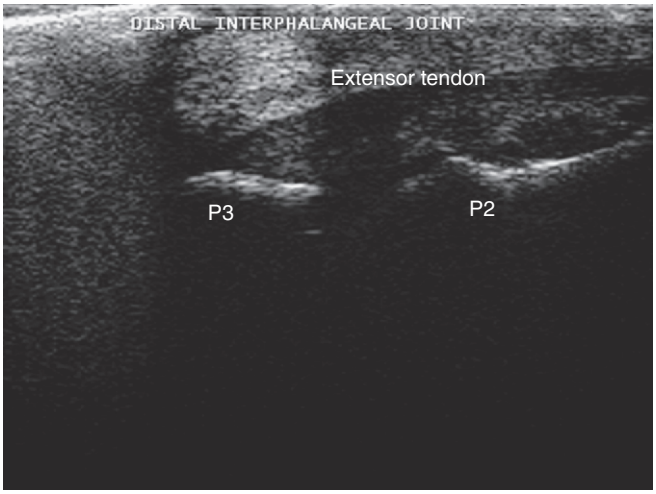

FIGURE 10-66 Normal sonogram of the dorsal aspect of the distal interphalangeal joint. This sagittal image was obtained with a 7.5- to 10-MHz linear array transducer using the coronary band as an acoustic window. The right side of the image is proximal and the left side distal. *P2,* Middle phalanx; *P3,* distal phalanx.

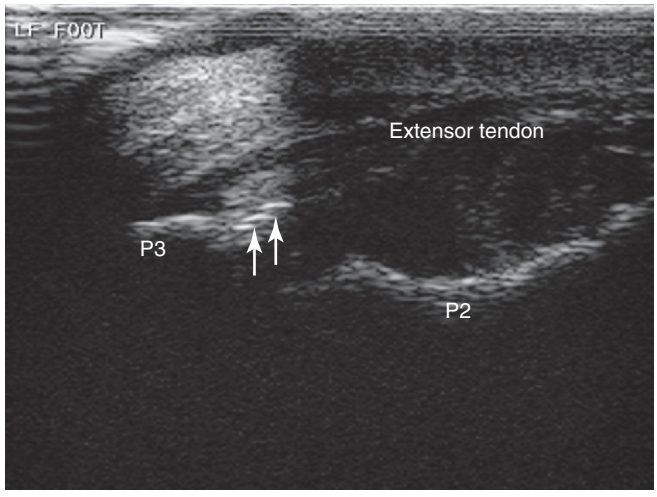

FIGURE 10-67 Sonogram of a fracture of the extensor process of the distal phalanx. The fragment can be visualized as a hyperechoic structure *(arrows)* located within the attachment of the extensor tendon and lifted away from the parent bone. This longitudinal image was obtained with a 7.5- to 10-MHz linear array transducer on the dorsal aspect of the distal interphalangeal joint. The right side of the image is proximal and the left side is distal. *P3,* Distal phalanx; *P2,* middle phalanx.

Collateral Ligaments of the Distal Interphalangeal Joint

Anatomy

The collateral ligaments are short bands that originate in the depressions on either side of the distal part of the middle phalanx, under the cover of the collateral cartilages of the distal phalanx. They widen distally and insert in the depres-

sions on either side of the extensor process and on the dorsal aspect of the collateral cartilages.

Scanning Technique

The collateral ligaments of the DIP joint should be evaluated with a 7.5- to 10-MHz linear array transducer with the foot in a weight-bearing position. The periople of the foot, just distal to the hairline of the coronary band, is used as an acoustic window. The transverse scan plane is generally of more diagnostic value for evaluation of the collateral ligaments of the DIP joint, but the collateral ligaments should also be evaluated in the longitudinal plane. The origin of the ligament can be visualized in the collateral fossae on the dorsal aspect of the middle phalanx in the 10 o'clock and 2 o'clock positions. The origin can also be evaluated in the longitudinal section. The insertion of these ligaments is often challenging to visualize, because of the horn of the hoof capsule. Because of the short nature of the ligament, however, any pathologic finding can usually be documented in this acoustic window. If insertional desmopathy is suggested based on findings of other diagnostic modalities (e.g., radiography or nuclear scintigraphy), the hoof wall can be thinned with a rasp and the foot soaked as described previously for evaluation of the insertion on the distal phalanx.

The operator of the ultrasonographic equipment must be mindful of the anatomic course of the collateral ligaments, especially when scanning in the longitudinal plane. The ligaments are oriented perpendicularly to the ground surface and are not parallel to the angle of the pastern. A comparison must always be made between the two collateral ligaments on the same foot, as well as to the corresponding ligament on the other foot. When making these comparisons, cross-sectional area, echogenicity, and fiber pattern should be evaluated. The normal appearance of the collateral ligaments of the DIP joint is an oval shape with uniform echogenicity and fiber pattern as they originate from the collateral fossae of the middle phalanx. The collateral ligaments widen distally at their insertion on the distal phalanx (Figures 10-68 to 10-70).

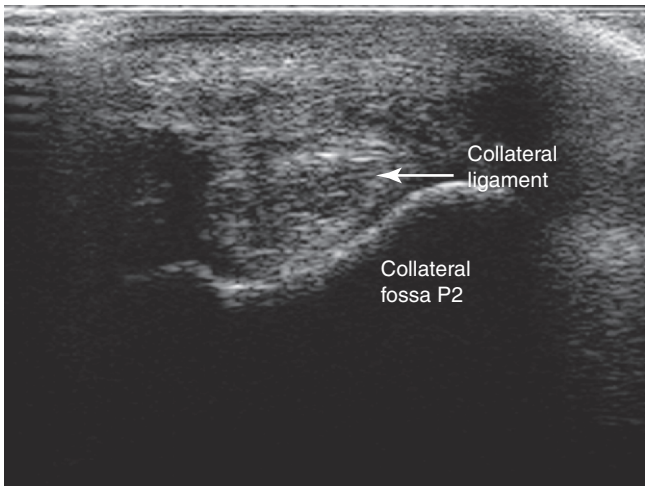

FIGURE 10-68 Sonogram of a normal collateral ligament of the distal interphalangeal joint as visualized in transverse section. This image was obtained with a 7.5- to 10-MHz linear array transducer. The right side of the image is lateral and the left side is medial. *P2,* Middle phalanx.

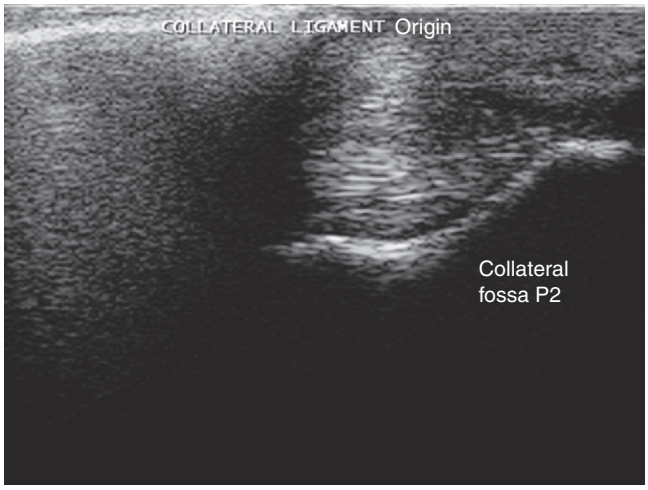

FIGURE 10-69 Sonogram of a normal origin of the collateral ligament of the distal interphlangeal joint, viewed in longitudinal section. This image was obtained with a 7.5- to 10-MHz linear array transducer using the dorsolateral aspect of the coronary band as an acoustic window. The right side of the image is proximal and the left side is distal.

NON–WEIGHT-BEARING EXAMINATION

Navicular Bone

Anatomy

The navicular bone is shuttle-shaped; it is located on the palmar aspect of the DIP joint. The long axis of the navicular bone is positioned in a transverse plane. The extremities of the navicular bone are smooth and round in shape. The navicular bone possesses two surfaces, two borders, and two extremities. The articular surface, which faces dorsoproximally, consists of a central eminence flanked by two concave areas, and articulates with the distal epiphysis of the middle phalanx. The flexor surface, which is directed palmarodistally, resembles the articular surface in form but is more extensive and not so smooth. Fibrocartilage covers the flexor surface, over which the DDFT runs. The proximal border is wide and grooved in the middle and narrower and rounded on either side. The distal border has a narrow facet dorsally for articulation with the distal phalanx. Palmar to this facet is a groove—the synovial fossa—that contains a number of relatively large invaginations and is bounded palmary by a prominent edge from which the DSIL originates.

Scanning Technique

The proximal flexor and distal surfaces of the navicular bone can be evaluated from the distal aspect of the palmar pastern and the transcuneal approach with the foot held in a non–weight-bearing position and the toe in extension.

The proximal aspect of the navicular bone is evaluated from the distal pastern window with the transducer wedged between the heel bulbs and angled distally. A 5.0- to 7.5-MHz linear array microconvex transducer is ideal for this purpose, because of its small footpad and the contour of the local anatomy. Diagnostic images are easier to obtain in a horse with low heels, but can be challenging to obtain in a high-heel foot conformation. The proximal aspect of the navicular bone

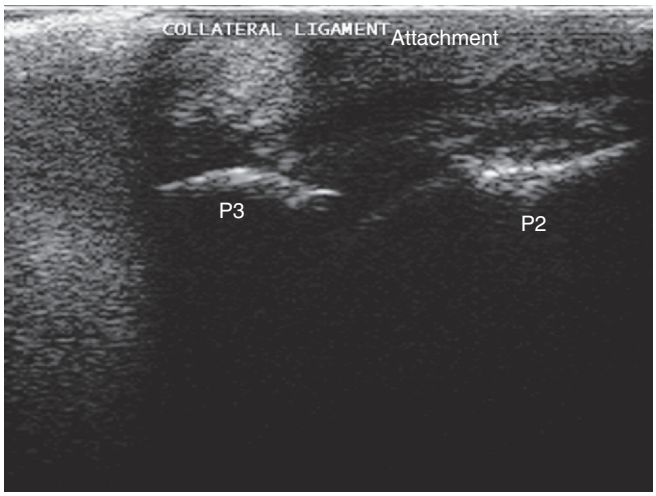

FIGURE 10-70 Sonogram of a normal insertion of the collateral ligament of the distal interphalangeal joint onto the distal phalanx in longitudinal section. This image was obtained with a 7.5- to 10-MHz linear array transducer using the dorsal coronary band and hoof wall as an acoustic window. The right side of the image is proximal and the left side is distal. *P2,* Middle phalanx; *P3,* distal phalanx.

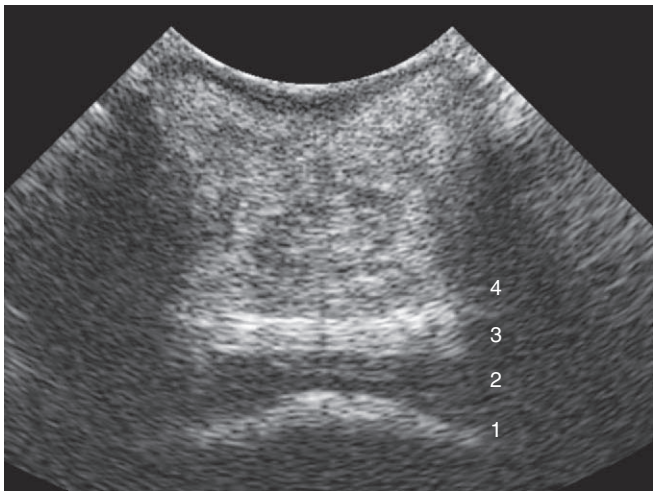

FIGURE 10-71 Normal sonogram of the podotrochlear apparatus obtained with a 5.0- to 7.0-MHz microconvex linear array transducer using the transcuneal acoustic window in a transverse plane. The right side of the image is lateral and the left side is medial. *1,* Navicular bone; *2,* combined fibrocartilage of the navicular bone, navicular bursa and fibrocartilaginous layer of the deep digital flexor tendon; *3,* combined tendinous portion of the deep digital flexor tendon and distal digital annular ligament; *4,* digital cushion.

should be imaged in sagittal and parasagittal planes and the attachment of the navicular suspensory ligaments evaluated. In a sagittal section, the compact bone of the proximal navicular bone appears as a smooth hyperechoic line that has a curved surface as it articulates with the palmarodistal aspect of the middle phalanx. These hyperechoic lines formed by the palmar contour of the distal middle phalanx and the proximal navicular bone form the deep limit of the image.

Although the sagittal view is easier to obtain, an attempt must always be made to obtain transverse sections of the proximal aspect of the navicular bone. This view may be particularly challenging to obtain in a horse with a high heel conformation. The proximal aspect of the navicular bone appears as a smooth horizontal hyperechoic line that is located just superficial to the contoured hyperechoic line formed by the palmarodistal aspect of the middle phalanx articulation. The palmar aspect of the distal phalanx therefore forms the deep limit of the image.

The flexor surface of the navicular bone is imaged with the foot held in a non–weight-bearing position using the transcuneal approach and a 5.0- to 7.5-MHz linear array convex transducer or a 7.5- to 10-MHz linear array microconvex transducer. In a sagittal plane, the compact bone of the navicular bone appears as a hyperechoic line that is covered by structures of mixed echogenicity corresponding to the navicular bone fibrocartilage, navicular bursa, and the dorsal fibrocartilagenous layer of the DDFT.[58] The flexor surface of the navicular bone should have a smooth convex curved contour, appearing as a clean hyperechoic line marking the deep limit of the image obtained. In addition, it is imperative to obtain parasagittal images, as previously described, because of the potential for pathologic changes to be located abaxially. Transverse images of the flexor surface should also be obtained at the level of the navicular bone.

A 0.3- to 0.5-mm–thick layer of fibrocartilage covers the flexor surface of the navicular bone.[58] In the absence of navicular bursa distention, it is difficult to differentiate the palmar limit of this fibrocartilage, as it combines with the navicular bursa and the fibrocartilagenous portion of the DDFT to appear as a hypoechoic to anechoic rim palmar to the hypoechoic line marking the flexor surface of the navicular bone.[58] The sonographic appearance of the contour of the flexor surface should be similar to the contour of the flexor surface when the palmaroproximal-palmarodistal oblique (skyline) projection of the navicular bone is obtained radiographically. The sagittal ridge is evident, although the lateral and medial edges may be difficult to visualize due to the width of the scan plane (Figures 10-71 and 10-72).

Navicular Bursa

Anatomy

The navicular bursa is found between the DDFT and the navicular bone. The navicular bursa extends about 1 to 1.5 cm proximal to the navicular bone and distally to the insertion of the tendon.

Scanning Technique

The navicular bursa should be evaluated with the foot in a non–weight-bearing position, held by the operator or an assistant. The bursa can be evaluated at two levels: (1) suprasesamoidean (i.e., proximal to the navicular bone) or (2) subsesamoidan (i.e., distal to the navicular bone).

The suprasesamoidean portion of the navicular bursa is best imaged from between the heel bulbs. A 5.0- to 7.5-MHz microconvex probe offers the best contact between the footpad and the local anatomy. Angling the probe distally in the sagittal plane provides an image of the proximal aspect of the navicular bone, with the bursa appearing as an anechoic structure located on the palmaroproximal aspect of the proximal surface of the navicular bone. The proximal aspect of the navicular bursa is easier to visualize in horses with low heels. Intuitively, the proximal aspect is also more prominent in horses with distention of the navicular bursa, as might occur with acute navicular bursitis.

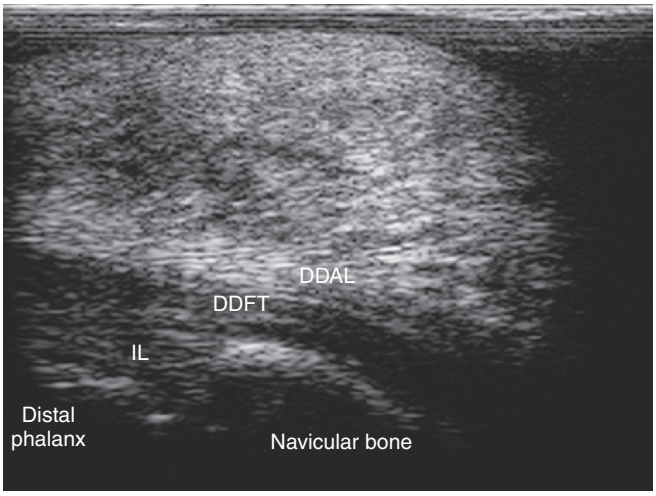

FIGURE 10-72 Sonogram of a normal sagittal section of the podotrochlear apparatus obtained with a 7.5- to 10-MHz linear array transducer using the transcuneal acoustic window. The right side of the image is proximal and the left side is distal. *DDFT,* Deep digital flexor tendon; *DDAL,* distal digital annular ligament; *IL,* impar ligament.

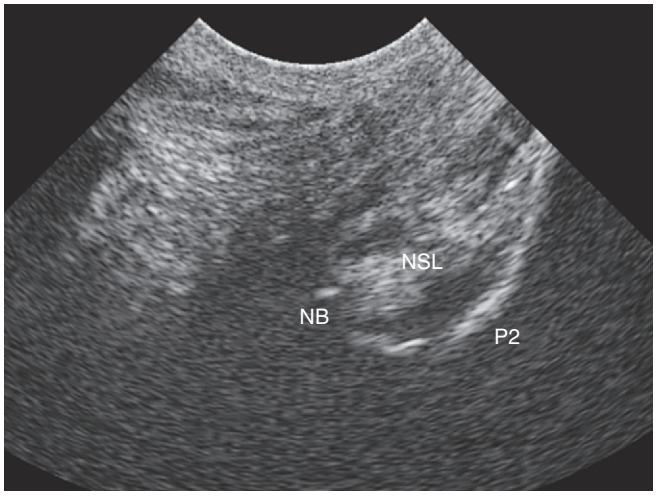

FIGURE 10-73 Sonogram of a normal navicular suspensory ligament (NSL) obtained in sagittal section with a 5.0- to 7.5-MHz microconvex linear array transducer using the heel bulbs as an acoustic window. The right side of the image is proximal and the left side is distal. *NB,* Navicular bone; *P2,* middle phalanx.

The subsamoidean aspect of the navicular bursa is best visualized using a 5.0- to 7.5-MHz linear array microconvex transducer, or a 7.5- to 10-MHz linear array microconvex transducer using the transcuneal approach. In the sagittal plane, the navicular bursa appears as an anechoic structure. Its proximal boundary is the distal aspect of the navicular bone, the dorsal boundary is the more echogenic distal sesamoidean impar ligament, and the palmar boundary is the DDFT. In normal horses, the distal extent of the navicular bursa extends just beyond the distal aspect of the navicular bone but can be challenging to differentiate from the navicular bone fibrocartilage and the fibrocartilaginous portion of the DDFT. When there is an increased volume of fluid in the navicular bursa, as in the case of acute navicular bursitis, this portion of the navicular bursa tends to distend in a palmarodistal direction, thus making it somewhat easier to visualize. Parasagittal images of the navicular bursa should also be obtained by orienting the probe laterally and medially from this location on the central sulcus of the frog. A transverse section of the distal aspect of the bursa can be obtained at this level. The location of the scan for the distal sesamoidean impar ligament is described in detail later. The distal rim of the podotrochlear bursa appears as a thin hypoechoic rim at this level.

The subsesamoidean portion of the navicular bursa is very difficult to image in the absence of moderate to severe distention of the bursa. As described earlier, with ultrasonography it is very difficult to differentiate the fibrocartilage covering the flexor surface of the navicular bone, the navicular bursa, and the fibrocartilagenous portion of the DDFT. These three structures collectively contribute to the hypoechoic-anechoic rim, seen ultrasonographically palmar to the hyperechoic line of the flexor surface of the navicular bone.

Navicular Suspensory (Collateral Sesamoidean) Ligaments

Anatomy

The navicular suspensory ligaments (NSLs) are strong, elastic bands that form a suspensory apparatus for the navicular bone. They are attached proximal to the medial and lateral epicondylar fossae of the distal proximal phalanx, where they are partially blended with the collateral ligaments of the proximal interphalangeal joint. The NSLs are directed obliquely distally and palmary and end chiefly on the proximal border of the navicular bone, but they detach a branch to the axial surface of each collateral cartilage and angle of the distal phalanx.

Scanning Technique

The NSLs are best imaged with the foot in a non–weight-bearing position, through the distal pastern window, using a 5.0- to 7.5-MHz linear array microconvex transducer. The attachment of the ligament on the proximal border of the navicular bone is easier to image in a foot with a low heel conformation. Sagittal and parasagittal images of the ligament should be obtained at this level, since they are of more diagnostic value than the transverse section, which can be difficult to image in all horses. The ligament appears as a hypoechoic structure with longitudinal fibers running distally to their attachment on the proximal aspect of the navicular bone. The deep limit of the image is formed by the palmar aspect of the middle phalanx proximally and the proximal aspect of the navicular bone distally. The DDFT is located superficially to the NSL and appears hypoechoic to anechoic due to the nonperpendicular scan plane. The distal digital annular ligament appears hyperechoic and forms the superficial border of the image (Figures 10-73 and 10-74).

Distal Sesamoidean Impar Ligament

Anatomy

The DSIL reinforces the joint capsule distally. It is a strong band of fibers that extends from the distal border of the navicular bone to the flexor surface of the distal phalanx.

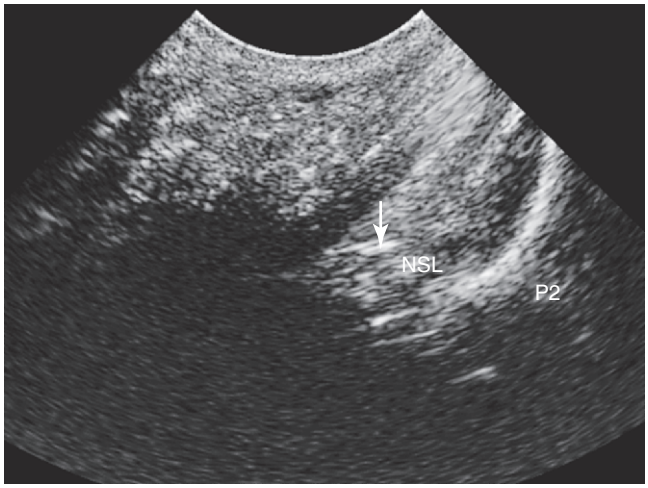

FIGURE 10-74 Sonogram of dystrophic mineralization and associated desmitis of the navicular suspensory ligament. The mineralization *(arrow)* is best visualized in the sagittal plane and is casting an acoustic shadow. The surrounding ligament lacks a normal fiber pattern. This image was obtained with a 5.0- to 7.5-MHz microconvex linear array transducer using the heel bulbs as an acoustic window. The right side of the sagittal image is proximal and the left side is distal. ↓, Mineralization within the distal sesamoidean collateral ligament; *NSL,* navicular suspensory ligament; *P2,* middle phalanx.

Scanning Technique

The DSIL can be visualized with the foot in a non–weight-bearing position using the transcuneal approach. A 3.5- to 5-MHz convex linear array transducer can be used on horses with a high-heel comformation and a deep central sulcus of the frog. Either a 5.0- to 7.5-MHz microconvex linear array transducer or a 7.5- to 10-MHz linear array transducer should be used in horses with low-heel conformation and a shallow central sulcus of the frog.

The DSIL occupies a triangular area the boundaries of which are marked by the distal aspect of the navicular bone proximally, the solar margin of the distal phalanx dorsally and distally, and the navicular bursa and DDFT on the palmar boundary. When navicular bursa distention is present, the hypoechoic bursa marks the palmar boundary of the DSIL proximally. The fibers of the DSIL are usually more echogenic than the DDFT, and in most cases it is relatively easy to orient the ultrasound beam perpendicular to these fibers. The examination is started in a sagittal plane, although parasagittal views should always be obtained prior to conclusion of the examination. The parasagittal views are obtained by orienting the probe laterally and medially from its position on the central sulcus.

In sagittal section, the DSIL originates from the distal aspect of the navicular bone and courses distally to its insertion on the flexor surface of the distal phalanx. It is more echogenic than the palmary located deep digital flexor tendon, and although it is a short ligament, a parallel fiber pattern can be easily appreciated ultrasonographically. Parallel dorsal and palmar edges can be documented in horses without pathologic lesions in both sagittal and parasagittal sections. The dorsal to palmar thickness of the DSIL was measured at 2 to 3 mm in one study.[58] In another study, the corresponding measurement was 3.19 ± 0.23 mm in clinically normal feet and 3.43 ± 0.10 mm in affected feet.[62]

Transverse images of the DSIL should also be obtained. The DSIL can be located in transverse section just distal to the distal aspect of the navicular bone. This location is recognized by two distinctly hyperechoic lines marking the deep limit of the image. The more superficial line is the distal aspect of the navicular bone, and the deeper line is the solar surface of the distal phalanx. Just distal to this location, the DSIL can be seen occupying the space between the distal phalanx and the DDFT.[58] The DSIL appears less echogenic when compared to the DDFT, and the thin hypoechoic rim representing the distal recess of the navicular bursa can be seen at its palmar aspect.[58]

Deep Digital Flexor Tendon

Anatomy

From the distal fourth of the metacarpus to the middle of the middle phalanx, the DDFT is enclosed in the digital synovial sheath, described in connection with the superficial digital flexor tendon. The insertion of the DDFT is on the semilunar line of the flexor surface of the distal phalanx and the adjacent surface of the collateral cartilages of the distal phalanx.

Scanning Technique

The DDFT is evaluated at three levels: suprasesamoidean, sesamoidean, and subsesamoidean. The suprasesamoidean part of the DDFT can be evaluated in the distal pastern with the foot in a non–weight-bearing position and the toe extended. The use of a 5.0- to 7.5-MHz linear array microconvex transducer is ideal, owing to the small footprint of the probe head and the nature of the local anatomy. A standoff pad is not recommended because it decreases contact between the footpad and contact surface of the palmar distal pastern and heel bulbs. The probe has to be wedged between the heel bulbs and oriented distally. Consequently, diagnostic images are easier to obtain on a horse with a low-heel conformation as opposed to an animal with high heels or a flexural deformity of the distal interphalangeal joint (e.g., clubfoot conformation).

At the suprasesamoidean level, the DDFT appears as a bilobed structure when viewed in transverse section.[60] The two lobes of the DDFT in this region should be symmetrical in size and shape. As a result, the DDFT in this area must be scanned in paramedian transverse sectional views, and both lobes of the DDFT must be compared. This is particularly important with horses that have clubfoot conformation, because a single transverse view obtained at the palmar midline often will not adequately illustrate the medial and lateral margins of the corresponding DDFT lobes.

Although the transverse view is of more diagnostic value in this region, the DDFT should also be evaluated in the sagittal plane, as well as medial and lateral parasagittal planes. A comparison between the the medial and lateral lobes should be made, with particular attention paid to the dorsal and palmar edges. As the DDFT courses over the proximal aspect of the navicular bone, its fibers are not perpendicular to the scan plane and consequently it appears hypoechoic. The dorsal-to-palmar thickness and lateral-to-medial width of the DDFT decrease somewhat distally in the middle pastern and then increase again in the distal pastern.[60] The DDFT has been measured as 7 to 12 mm thick in the distal pastern in a dorsal-to-palmar direction, and its width in

a lateral-to-medial direction ranges from 15 to 23 mm in the distal pastern.[60] With this in mind, any sudden convexity of a section of the DDFT, without a corresponding change in the contralateral lobe, can be interpreted as an abnormality.

At the sesamoidean level, the DDFT has a fibrocartilaginous part dorsally and a fibrous part palmary. Examination of this portion of the tendon is performed with the foot in a non–weight-bearing position using the transcuneal approach. The distal limb can be held by an assistant or placed on the operator's knee, with the distal limb in a neutral position. The frog is used as a window to image this portion of the DDFT. A 3.5- to 7.5-MHz linear array convex transducer is adequate to begin the examination and obtain diagnostic-quality images. In a horse with low heels and a shallow frog, a 7.5- to 10-MHz linear array transducer can be used to obtain better image resolution. A standoff pad is not necessary when the foot is prepared adequately.

The examination is started with the probe oriented in a sagittal plane at the central sulcus of the frog. At the level of the distal scutum, the dorsal aspect of the DDFT is fibrocartilagenous and appears hypoechoic ultrasonographically.[58] This anatomical feature makes it difficult to distinguish the dorsal aspect of the DDFT from the navicular bursa and the flexor surface of the navicular bone in this region. As stated previously, the DDFT should be examined in a sagittal and parasagittal plane by orienting the probe laterally and medially from its position on the central sulcus. The longitudinal view is of a higher diagnostic value, but the transverse view should also be obtained. The compact bone of the flexor surface of the navicular bone represents the hyperechoic deep limit of the image.[58] A 0.3- to 0.5-mm–thick layer of fibrocartilage covers the flexor surface of the navicular bone.[58] In the absence of any distention of the navicular bursa, however, this fibrocartilage becomes difficult to differentiate from the navicular bursa and the dorsal fibrocartilage of the DDFT. These structures collectively appear as a hypoechoic to anechoic rim along the flexor surface of the compact bone of the navicular bone. The fibrous portion of the DDFT appears as a 4- to 6-mm–thick structure.[58] The lateral and medial edges of these structures cannot be visualized from the central sulcus due to the limit of the scan plane, even when using convex and microconvex linear array transducers.

The subsesamoidean portion of the DDFT extends from the distal aspect of the navicular bone to its insertion on the flexor surface of the distal phalanx. Views of this portion of the tendon are best obtained with the foot in a non–weight-bearing position. Once again, an assistant can hold the foot or the operator can place the foot on their knee with the distal limb in a neutral position. A 3.5- to 7.5-MHz convex transducer is ordinarily sufficient to provide adequate diagnostic images. A 7.5- to 10-MHz microconvex linear array transducer should also be used to provide images with better resolution when the anatomy of the foot permits (i.e., low heel and shallow frog conformation).

Images should be obtained initially in a sagittal plane. The fibrous portion of the DDFT appears as a 4- to 6-mm–thick structure.[58] The dorsopalmar thickness of the DDFT increases gradually as it courses distally and inserts on the flexor surface of the distal phalanx. This fibrous portion of the deep digital flexor tendon is more echogenic than the fibrocartilaginous portion that is present proximally. The flexor surface of the distal phalanx has a regular hyperechoic appearance. The attachment of the DDFT onto this flexor surface of the distal phalanx must be evaluated in a sagittal and parasagittal plane by angling the transducer medially and laterally from the central sulcus. On feet with high heels, it may be challenging to orient the probe perpendicular to the fibers of the DDFT as they attach on the flexor surface of the distal phalanx. In these cases, the deep digital flexor tendon may appear hyperechoic at its attachment.

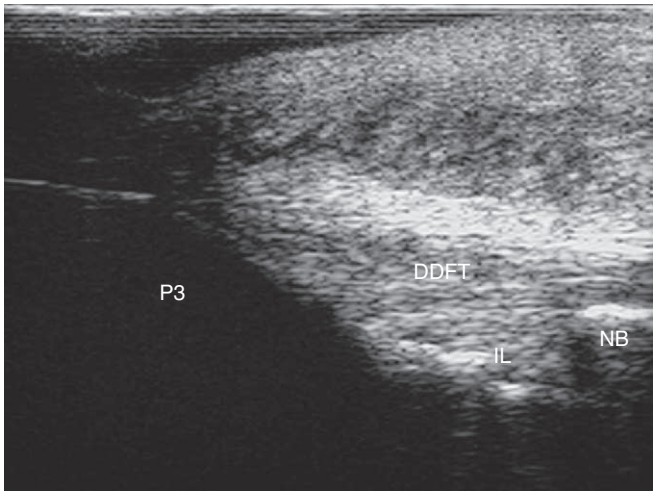

FIGURE 10-75 Sonogram of a normal attachment of the deep digital flexor tendon to the distal plalanx. This is best visualized in sagittal plane. The image was obtained with a 7.5- to 10-MHz linear array transducer using the transcuneal acoustic window. The right side of the image is proximal and the left side is distal. *P3,* Distal phalanx; *DDFT,* deep digital flexor tendon; *IL,* distal sesamoidean impar ligament; *NB,* navicular bone.

Evaluation of the subsesamoidean deep digital flexor tendon is continued in the transverse plane. When scanning in the transverse plane, two distinct hyperechoic lines mark the distal aspect of the image. The more superficial line represents the distal aspect of the flexor surface of the navicular bone. A hyperechoic line, representing the flexor surface of the distal phalanx, marks the deep limit of the image. An attempt should be made to follow this portion of the DDFT to its attachment on the flexor surface of the distal phalanx. As previously stated, this can be challenging in horses with a high heel conformation or a robust frog with a deep central sulcus. The fibrous portion of the DDFT was reported to measure between 4 and 6 mm in this region.[58] Another study reported the combined thickness of the DDFT and the distal digital annular ligament (DDAL) as 4.26 ± 0.19 mm in clinically normal feet and 4.33 ± 0.14 mm in feet affected with navicular syndrome (Figure 10-75).[62]

Distal Digital Annular Ligament

Anatomy

The terminal portion of the DDFT is bound down by the DDAL.

Scanning Technique

The DDAL is best visualized with the foot in a non–weight-bearing position using the transcuneal approach. A 3.5- to 7.5-

MHz linear array convex or a 7.5- to 10-MHz linear array microconvex transducer can be used to obtain diagnostic-quality images.

The DDAL can be considered an accessory ligament of the DDFT, with its dorsal boundary being the DDFT and its palmar boundary being the digital cushion. Differentiating the DDAL from the palmar aspect of the fibrous portion of the DDFT can be difficult, except when the ultrasound beam is perpendicular to the fibers of the DDFT.[58] When viewed in this orientation, the fibrous portion of the tendon appears echogenic and the interfaces between the digital cushion, DDAL, and DDFT become visible.[58]

The examination should commence with a sagittal image obtained at the sesamoidean and subsesamoidean levels. Parasagittal images should also be obtained. Transverse images should be obtained at the sesamoidean and subsesamoidean levels. When the ultrasound beam is oriented perpendicular to the DDFT fibers, the DDAL can be isolated as a hypoechoic structure on the palmar surface of the fibrous portion of the DDFT. The combined thickness of the DDFT and the DDAL was reported as 4.26 ± 0.19 mm in clinically normal feet and 4.33 ± 0.14 mm in feet affected with navicular syndrome.[62] Primary pathologic lesions of the DDAL are also possible in conjunction with damage to other soft-tissue structures, particularly the DDFT.

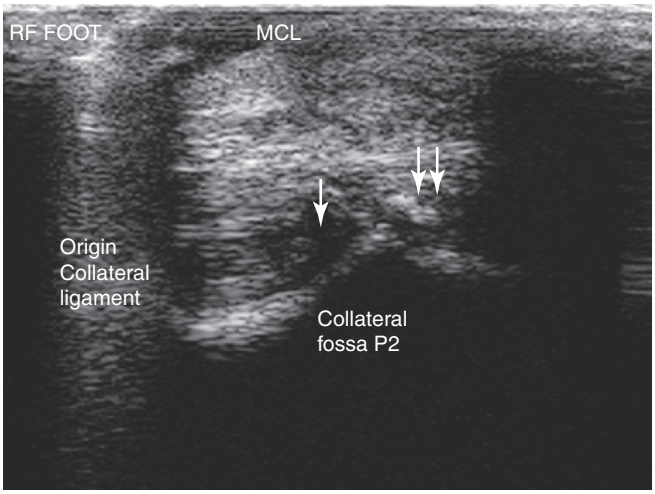

FIGURE 10-76 Sonogram of an avulsion involving the origin of the medial collateral ligament of the distal interphlangeal joint from the middle phalanx. There is a complete loss of the fiber pattern with fragments of bone casting acoustic shadows. This is best visualized in sagittal plane. This sagittal image is obtained with a 7.5- to 10-MHz linear array transducer using the dorsal coronary band as an acoustic window. The right side of the screen is proximal and the left side is distal. ↓↓, Avulsion fragments; ↓, loss of fiber pattern; *P2*, middle phalanx; *MCL*, medial collateral ligament.

ABNORMAL FINDINGS

Desmitis of the Collateral Ligaments of the Distal Interphalangeal Joint

Hypoechoic areas or a mottled appearance to the collateral ligament in conjunction with a compromised fiber pattern consistent with fiber tearing of the ligament are abnormal findings. An increased cross-sectional area, when compared with the contralateral ligament, should also be considered abnormal and consistent with desmitis of the ligament. In one study of 13 horses with palmar foot pain, five exhibited abnormalities of one of the collateral ligaments (three medial, two lateral) and one horse had both medial and lateral ligaments affected.[59] A hypoechoic lesion was present in five ligaments and a mottled echogenicity in two of the ligaments.[59] All ligaments with a core lesion had disruption of the normal parallel fiber alignment on a longitudinal section.[59]

Roughening of the dorsal aspect of the middle phalanx in the region of the collateral fossa can also be seen ultrasonographically in some cases (Figure 10-76). The origin of the collateral ligament in the region should be critically evaluated. Ultrasonography appears to be more sensitive than radiography when detecting changes on the surface of bone, although radiographic evidence of mild degenerative joint disease of the dorsal distal interphalangeal joint was noted in two cases described in the aforementioned study.[59]

Navicular Bone Abnormalities

Abnormal changes on the proximal border of the navicular bone include anechoic or hyperechoic areas identified as roughening of the bone consistent with insertional desmopathy with enthesiophyte formation in the region. Abnormal changes of the flexor surface include focal hypoechoic areas, thinning of the normal compact cortical bone echo, roughening of the surface, adhesion formation, lack of continuity of the hyperechoic bone surface as in a navicular bone fracture or bipartate or tripartate navicular bones.

In one study on ultrasonographic examination of 24 feet in 15 of 28 horses with navicular syndrome, the flexor surface of the navicular bone appeared roughened, resulting in a more hypoechoic image when compared to the smooth crisp hyperechoic bony surface of the flexor surface of the navicular bone in clinically normal horses.[62] Pathologic changes of the distal border of the navicular bone, evaluated using the subsesamoidean approach, consist of hypoechoic or hyperechoic areas consistent with remodeling of the distal aspect of the navicular bone. These changes can be associated with a concurrent desmopathy of the origin of the distal sesamoidean impar ligament (Figures 10-77 and 10-78).

Navicular Bursitis

Acute nonseptic bursitis can be characterized ultrasonographically as an increase in anechoic fluid within the bursa when viewed through the transcuneal approach/subsesamoidean window. The effusion associated with a septic process appears more echogenic on ultrasonograms.[60] Echogenic strands and loculations of fibrin can also be visualized in the synovial fluid in both septic and nonseptic bursitis. The fibrin can organize to form fibrous adhesions between the bursa and the DDFT or between the flexor surface of the navicular bone and the DDFT.

In a study of 28 horses with navicular syndrome, 22 had evidence of navicular bursitis on ultrasonographic examination.[62] With acute aseptic bursitis, the amount of anechoic

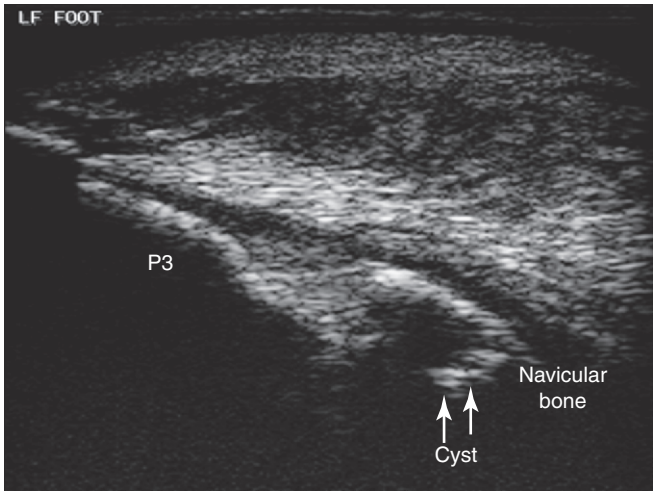

FIGURE 10-77 Sonogram of a navicular bone cyst. The deep extent of the cyst can be visualized in the sagittal plane as a hyperechoic area of the navicular bone. This sagittal image was obtained with a 7.5- to 10-MHz linear array transducer using the transcuneal acoustic window. The right side of the image is proximal and the left side is distal. *P3,* Distal phalanx.

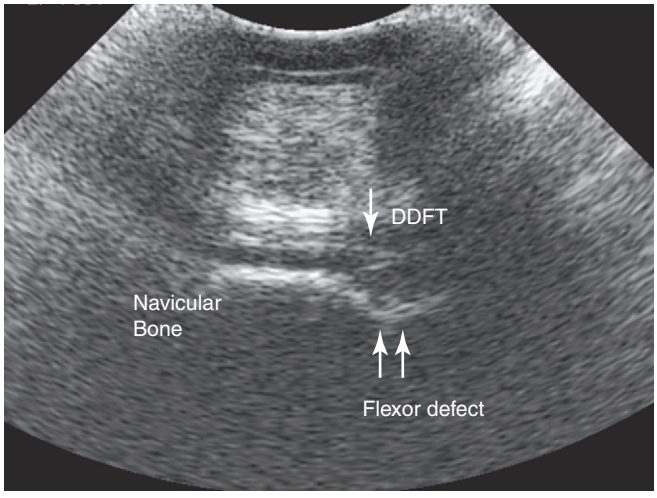

FIGURE 10-78 Sonogram of a large flexor surface defect on the lateral aspect of the flexor cortex of the navicular bone. This is best visualized in the transverse plane. This transverse image was obtained with a 5- to 7.5-MHz microconvex linear array transducer using the transcuneal acoustic window. The right side of the image is lateral and the left side is medial. *DDFT,* Deep digital flexor tendon; ↑↑, flexor defect of the navicular bone; ↓, corresponding hypoechoic area in the DDFT.

fluid within the navicular bursa was increased when compared to clinically normal horses, as evidenced by protrusion of the bursa in a palmarodistal direction ultrasonographically. In cases of chronic bursitis, a small amount of hypoechoic fluid was evident within the bursa but the synovial lining of the bursa appeared thickened.[62] Because of the inability to differentiate the synovial capsule from synovial villous thickening, these structures collectively contributed to the thickened appearance seen on ultrasonogram.

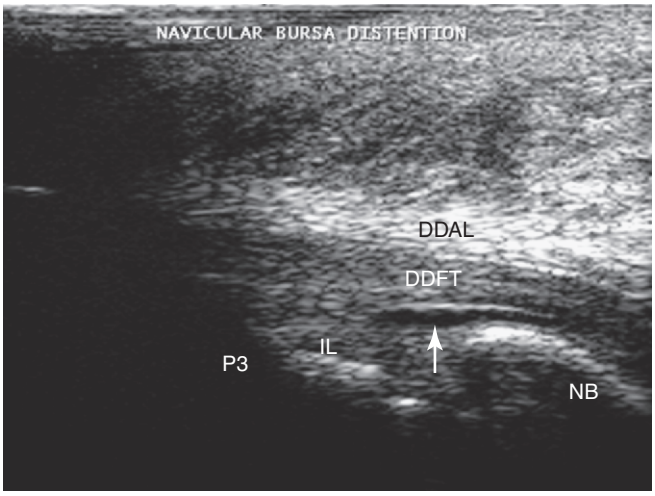

FIGURE 10-79 Sonogram of acute severe navicular bursitis visualized in the sagittal plane. There is a large amount of anechoic fluid present within the navicular bursa at its distal aspect *(arrow).* This sagittal image was obtained with a 7.5- to 10-MHz linear array transducer using the transcuneal acoustic window. The right side of the image is proximal and the left side distal. *DDAL,* Distal digital annular ligament; *DDFT,* deep digital flexor tendon; *P3,* distal phalanx; *NB,* navicular bone; *IL,* distal sesamoidean impar ligament.

These changes were not observed in clinically normal horses.[62]

With select cases of acute aseptic navicular bursitis in horses with a low heel conformation, it is possible to see the proximal aspect of the navicular bursa through the heel bulb approach/suprasesamoidean window. Care must be taken not to apply too much pressure with the footpad of the transducer, as the operator may collapse the proximal aspect of the bursa. In acute cases, the bursa has an increased amount of anechoic fluid and extends in a palmaroproximal direction from the proximal aspect of the navicular bone (Figures 10-79 and 10-80).

Adhesions

Adhesions between the DDFT and the flexor surface of the navicular bone can be seen ultrasonographically as a hyperechoic area just palmar to the flexor surface of the navicular bone. This is best visualized on longitudinal section through the sesamoidean window using the transcuneal approach. When an adhesion is suggested, a dynamic examination should be attempted by flexing and extending the toe, even though there is minimal motion of the DDFT in this region.

If there is mature fibrous adhesion, there will be no focal movement of the DDFT in the area of interest. One study of horses with navicular syndrome detected adhesions between the navicular bone and the DDFT in three feet of three horses.[62]

Deep Digital Flexor Tendinitis

Deep digital flexor tendonitis can be visualized through the suprasesamoidean window using the distal pastern/heel bulb

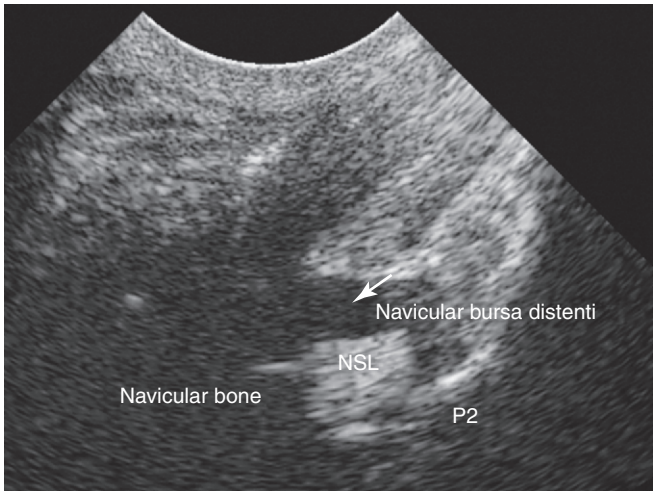

FIGURE 10-80 Sonogram of navicular bursitis evident at the proximal aspect of the navicular bursa. There is distention of the bursa with anechoic fluid *(arrow)*. This is best visualized in the longitudinal plane. The image was obtained with a 5.0- to 7.5-MHz microconvex linear array transducer, using the heel bulbs as an acoustic window. The right side of the image is proximal and the left is distal. *NSL,* Navicular suspensory ligament; *P2,* middle phalanx.

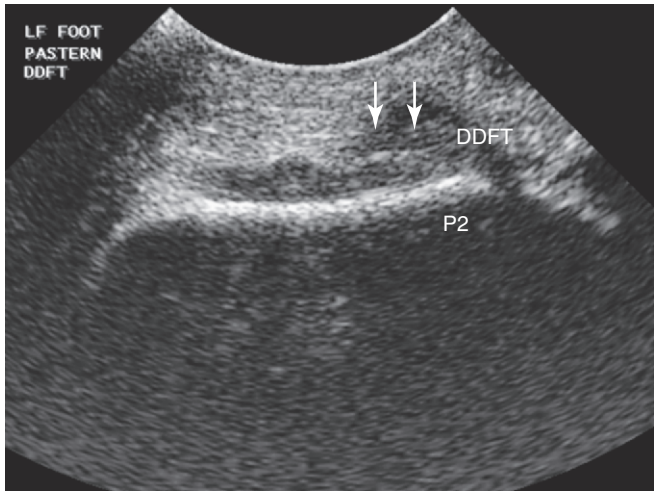

FIGURE 10-81 Sonogram of deep digital flexor tendonitis just proximal to the navicular bone. The lateral lobe of the deep digital flexor tendon is enlarged and hypoechoic *(arrows)*. This is seen best in transverse and parasagittal planes. The transverse image was obtained with a 5.0- to 7.5-MHz microconvex transducer using the heel bulbs as an acoustic window. The right side of the image is lateral and the left side is medial. *DDFT,* Deep digital flexor tendon; *P2,* middle phalanx.

approach. Asymmetry of the two lobes on transverse section, or convexity of nonparallel edges on longitudinal section, should be considered abnormal. Because of the nonperpendicular scan plane, hypoechoic areas should be interpreted with caution. Hyperechoic regions in an off-incidence scan plane, especially those casting acoustic shadows, are compatible with dystrophic mineralization of the deep digital flexor tendon and should be considered abnormal.

Deep digital flexor tendonitis can also be visualized through the sesamoidean window using the transcuneal approach. Adhesions to the tendon are usually present concurrently with deep digital flexor tendonitis in the region and should be considered abnormal. Hypoechoic to anechoic areas can also be identified in both longitudinal and transverse planes. One study identified such abnormalities in five feet, and the ultrasonographic findings were considered consistent with fiber tearing and tendonitis.[62]

Deep digital flexor tendonitis can also be identified at the subsesamoidean level using the transcuneal approach. The sagittal and parasagittal planes are the most important views to evaluate. Because of the nonperpendicular scan plane, the DDFT appears hypoechoic at its insertion on the flexor surface of the distal phalanx and therefore hypoechoic areas must be interpreted with caution. Hyperechoic areas in an off-incidence plane, or those casting acoustic shadows, are considered abnormal, consistent with dystrophic mineralization and insertional tenopathy (Figures 10-81 and 10-82).

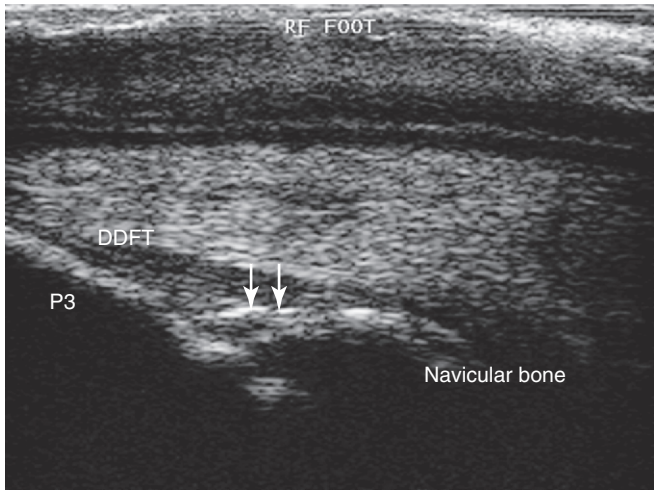

FIGURE 10-82 Sonogram of deep digital flexor tendonitis with dystrophic mineralization *(arrows)* just proximal to its insertion on the distal phalanx. The mineralization is best visualized in the sagittal plane as a hyperechoic structure especially when viewed at an "off incidence" angle. There is also a poor fiber pattern in the DDFT surrounding the area. The sagittal image was obtained with a 7.5- to 10-MHz linear array transducer using the transcuneal acoustic window. The right side of the image is proximal and the left side is distal. *DDFT,* Deep digital flexor tendon; *P3,* distal phalanx.

Distal Sesamoidean Impar Ligament Desmitis

A poor fiber pattern on longitudinal section is consistent with desmitis of the DSIL. The fiber disruption may also be accompanied by hyperechoic areas that cast acoustic shadows consistent with dystrophic mineralization of the DSIL. Swelling of the ligament is noted ultrasonographically as nonparallel dorsal and palmar edges with convexity of one or both edges. Fibers at the origin of the DSIL may also be disrupted. This change is often accompanied by a remodeling and roughening of the distal aspect of the navicular bone (Figures 10-83 and 10-84).

Penetrating Wounds to the Foot

Puncture wounds to the bottom of the foot are a cause of potentially serious and severe lameness, especially those in the area of the central frog and sulci.[63] In the past, determination of the depth and direction of the tracts was limited to radiography of the foot with the penetrating foreign body (e.g., a nail) in place or positive contrast radiography. There are severe limitations to this approach, however, since the foreign body is often removed prior to the initial examination. The soft horn of the frog collapses the tract quickly.[62] This makes determination of the full extent of the tract difficult. Injecting contrast media under pressure into the tract may potentially have the undesired affect of extending contamination into deeper tissues, including synovial structures. Distension of the bursa with contrast medium under aseptic conditions may allow determination of the tract only if the bursa is involved. This technique is invasive, and in chronic cases local cellulitis may preclude injection between the heel bulbs. Penetrating injuries of the navicular bursa and the DIP joint have a more unfavorable prognosis than injuries to other parts of the foot.[62] The DIP joint is usually penetrated from the solar surface through the distal sesamoidean impar ligament.

Fortunately, the central sulcus of the frog provides the perfect window to thoroughly evaluate all important structures using a transcuneal approach. A 3.5- to 5-MHz transducer is used for initial evaluation because it is extremely important to determine the deep extent of the tract. Once this has been determined, a 7.5- to 10-MHz transducer can be used to improve resolution for evaluation of the soft tissue structures. Noninvasive, ultrasonographic evaluation rapidly determines the direction and depth of the tract. In this region of the foot, the tracts have a hypoechoic appearance with hyperechoic areas inside. The hypoechoic to anechoic appearance is usually due to blood as a result of penetration of sensitive structures. The hyperechoic areas casting acoustic shadows are a result of air trapped within the tract. In more chronic cases, purulent material, particularly anaerobic in origin, may have a similar ultrasonographic appearance. Aggressive therapy can be based on the structures involved in the traumatic puncture wound. Foreign object penetration in the foot with material like wood is also visualized more easily with the use of ultrasonography than with radiography.

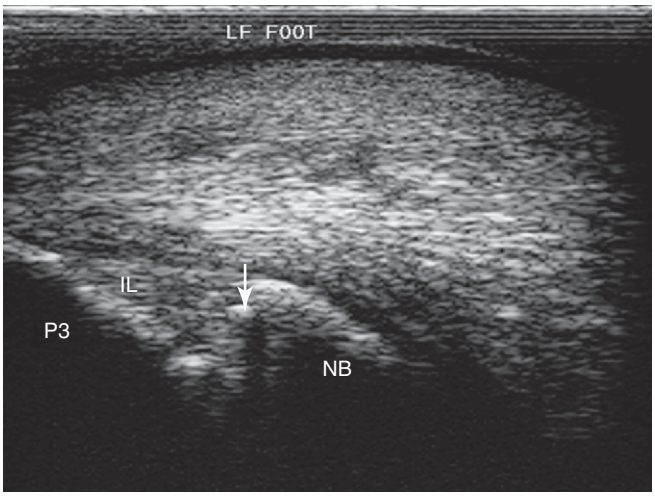

FIGURE 10-83 Sonogram of distal sesamoid impar ligament desmopathy. There is dystrophic mineralization evident at the origin of the impar ligament *(arrow)* casting a strong acoustic shadow. This is best visualized in the sagittal plane. The image was obtained with a 7.5- to 10-MHz linear array transducer using the transcuneal acoustic window. The right side of the image is proximal and the left side is distal. *IL,* Distal sesamoidean impar ligament; *NB,* navicular bone; *P3,* distal phalanx.

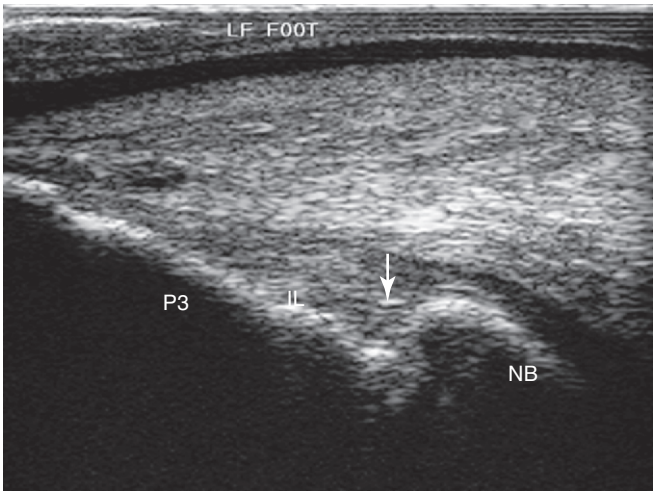

FIGURE 10-84 Sonogram of distal sesamoidean impar ligament desmitis with dystrophic mineralization *(arrow)* within the ligament at its origin from the distal aspect of the navicular bone. The pathologic change is best visualized in the sagittal plane as hyperechoic areas surrounded by a poor fiber pattern. This sagittal image was obtained with a 7.5- to 10-MHz linear array transducer using the transcuneal acoustic window. The right side of the image is proximal and the left side is distal. *IL,* Distal sesamoidean impar ligament; *NB,* navicular bone; *P3,* distal phalanx.

Ultrasound-Guided Injection of the Navicular Bursa

The transcuneal approach permits real-time imaging of synoviocentesis of the navicular bursa. After aseptic preparation of the caudal aspect of the foot, an assistant should hold the foot in a non–weight-bearing position. The elevated foot position is also better than leaving the foot on a potentially contaminated ground surface. The veterinarian operates the transducer with his or her nondominant hand. Once the sesamoidean window is visualized, the veterinarian can insert the long needle with the dominant hand, guided into the accurate position by real-time monitoring of the direction and depth of the needle. The needle is guided into the proximal aspect of the navicular bursa and the bursa is injected under sonographic guidance. This procedure is easy to perform and does not require expensive digital radiography equipment or processing delays to confirm accurate injection of the navicular bursa (Figure 10-85).

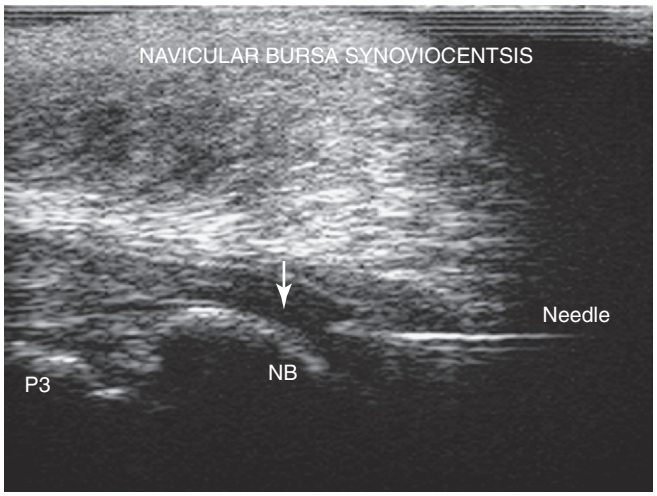

FIGURE 10-85 Sonogram of synoviocentesis of the navicular bursa *(arrow)*. The needle is visualized in the sagittal plane as a linear hyperechoic structure casting an acoustic shadow with reverberation artifact. This sagittal image was obtained with a 7.5- to 10-MHz linear array transducer using the transcuneal acoustic window. The right side of the image is proximal and the left side is distal. *P3,* Distal phalanx; *NB,* navicular bone.

RADIOGRAPHIC VIEWS OF MOST VALUE TO FARRIERS

Ric F. Redden

Farriers bear the responsibility of keeping the horse's foot healthy by using effective yet primitive means of controlling the ill effects of horn growth and of wear and tear on the hoof capsule. They must do so with little or no information about the state of the underlying soft tissues, vasculature, or bone, nor the effect of their interventions on these structures. Radiography can thus be an extremely valuable tool for farriers.

For the most part, farrier-interest radiographs are those that reveal relationships, in particular the relationship between the distal phalanx (P3) and the hoof capsule, and between P3 and the ground surface. Thus the two routine radiographic views that are most useful to farriers are the lateromedial (lateral) and the dorsopalmar (DP) views.

Although medical diagnosis is the province of the veterinarian, it can be of great value to the veterinarian-farrier team if the farrier also develops an eye for the radiographic characteristics of the soft-tissue zones within the hoof capsule. Of particular importance are the solar and laminar zones, which comprise both sensitive (corium) and insensitive (horn) tissues. These tissues are affected, either directly or indirectly, by every decision the farrier makes.

Before going any further, it is important to point out that the interests of the horse and its owner are best served when farriers and veterinarians work together, combining the knowledge and skills of the farrier with the medical and surgical training of the veterinarian. Whenever possible, both the veterinarian and the farrier involved in managing a particular case should arrange to review the radiographs together, and discuss treatment options while viewing the films, before any work on the foot begins.

EVALUATING THE SOFT TISSUES

The distal phalanx is supported within its protective shell by soft tissues whose health is crucial to the structural and functional integrity of the foot as a whole. Dysfunction is inevitable when any of the soft tissues are compromised or strained beyond their normal limits. Because pathologic soft-tissue findings are present to some degree in every footsore horse, evaluation of the soft tissues within the hoof capsule is an extremely important part of examination of the foot.

Careful observation of the hoof capsule allows the farrier to make certain inferences about the soft tissues within, but he or she can only guess as to their health and integrity. With appropriate technique, radiography greatly enhances the farrier's ability to evaluate the soft tissues within the hoof capsule. A lot of valuable information about these soft tissues can be gleaned from good-quality radiographs taken with soft-tissue detail in mind.

Measurements

The following indices should be measured on all routine lateral films: sole depth, width of the dorsal horn-lamellar zone, vertical distance between the coronary band and the top of the extensor process, and palmar angle. Positioning for the lateral view is described on p. 201.

Sole Depth

Sole depth is extremely important to farriers, as inadequate sole depth can affect the horse's soundness, reflect negatively on the farrier's competence, and limit the choice of shoe or shoeing method. Radiographically, sole depth is defined as the vertical distance between the solar margin of P3 and the outer surface of the sole. It is routinely measured at the apex of P3. A normal, healthy foot has a sole depth of at least 15 mm.

A sole depth of less than 15 mm should be considered clinically significant. Venograms in horses with a sole depth less than 15 mm show solar papillae that are bent, compressed, or even absent. This distortion or compression inhibits sole growth, creating a vicious cycle of thin, tender soles—the bane of any conscientious farrier's life.

Dorsal Horn-Lamellar Zone Width

Dorsal horn-lamellar (H-L) zone width is the distance between the dorsal surface of P3 and the outer surface of the dorsal hoof wall, measured with the ruler perpendicular to the dorsal surface of P3. (NOTE: This zone can be accurately measured only if the dorsal hoof wall is delineated using a radiopaque marker. Although a piece of wire or thin metal strip may suffice, a thin line of radiopaque paste is preferable, as it follows the contour of the hoof wall from proximal to distal more accurately than does the wire or metal strip.)

Dorsal H-L zone width can be measured anywhere along the dorsal face of P3, but it should be routinely measured at

two locations: just below the extensor process, and near the distal tip of P3. The measurements are recorded as proximal/distal (e.g., 15/15, meaning that the H-L zone is 15 mm at both locations). Normal H-L zone width is 15 to 16 mm in Quarterhorses, Thoroughbreds, and most other light horse breeds, and closer to 20 mm in Standardbreds. Normal H-L zone width for Warmbloods depends on the size of the foot; in many cases it is similar to that for light breeds. In a normal adult foot, the measurements should be the same proximally as distally. In the immature foot, the proximal value may be greater than the distal value.

On a good soft-tissue–detail film (see discussion under Exposure Settings), one can readily distinguish between the more radiopaque lamellar zone and the more radiolucent horn zone and even identify the junction between these two zones. In a normal foot, the lamellar corium and the horn of the dorsal hoof wall are the same thickness. So, in a foot with a dorsal H-L zone width of 15 mm, each zone measures 7.5 mm.

Dorsal H-L zone width is an important measurement, as this zone widens in conditions that affect the laminar corium, laminar attachment, or wall thickness. Laminitis and white line disease are two common and clinically important conditions in which the dorsal H-L zone widens. Widening as one moves down the wall from proximal to distal (i.e., H-L zone wider distally than proximally) can also be seen with either of these conditions.

This assessment, when used in conjunction with the palmar angle, provides a meaningful way to identify and describe displacement of P3. The conventional method of identifying and quantitating P3 rotation is inaccurate and misleading. The fact that the hoof capsule can be substantially altered by the farrier and is also affected by the disease process means that the whole basis of this measurement (P3–hoof wall angle) is seriously flawed.

When widening of the dorsal H-L zone is found, evaluation of the width of each component is important, as it can provide diagnostically and prognostically valuable information. For example, the lamellar zone widens in cases of laminitis, whereas it is the horn zone that widens in cases of white line disease. White line disease is readily apparent during thorough examination of the hoof itself; however, it can be difficult to distinguish from laminitis radiographically unless the widths of the horn and lamellar zones are compared.

Coronary Band–Extensor Process Distance

The coronary band–extensor process (C-E) distance is the vertical distance between the most proximal extent of the outer hoof wall and the top of the extensor process of P3. In most normal horses, the C-E distance is in the range of 0 to 15 mm. This measurement can be important in confirming displacement of P3, provided a baseline is established for that horse before, or at the onset of, the disease process. (NOTE: The C-E distance can be accurately measured only if the radiopaque marker on the dorsal hoof wall extends all the way to the proximal limit of the wall.)

Palmar Angle

Palmar angle refers to the angle of the solar margin of P3 relative to the ground surface. It can be measured relative to (1) the ground/bearing surface of the hoof capsule or (2) the ground itself. In the first instance, the angle is largely unrelated to the mechanical effect of the shoe or other device that may be attached to the foot. It provides information about the structural integrity of the supporting nonbony tissues in the heel area. When the palmar angle is measured relative to the ground, the angle also is indicative of the mechanical effect of any shoe/device that is attached to the foot.

In most healthy feet with strong heels and a robust digital cushion, the palmar angle is positive, meaning that the wings of P3 are higher than the apex. The range of normal for palmar angle is dependent, in part, on the horse's breed. Breeds that tend to have upright hooves typically have higher palmar angles than breeds with naturally lower hoof angles. (The shoeing package can also affect the palmar angle.)

A high palmar angle (relative to the range of normal for that breed) can be found in horses with clubfeet, laminitis, and certain other pathologic conditions. A negative palmar angle (wings of P3 lower than the apex) indicates substantial loss of structural integrity in the heel area, a situation that can usually be predicted simply by looking at the foot and estimating the depth of the digital cushion. (This simple assessment is made by placing the thumb in the shallow depression between the heel bulbs and a finger of the same hand in the center of the frog, squeezing gently, and estimating the distance between finger and thumb.)

Qualitative Assessments

In addition to these measurements, a high-quality radiograph taken at a soft exposure (see Exposure Settings) can reveal variations in radiodensity within these soft tissues. Evaluating the soft tissue zones around P3 is particularly important in the diseased foot, as congestion, edema, or accumulations of inflammatory exudate or gas can alter the radiodensity of the tissue, in addition to altering its thickness.

Thus a lot of useful information regarding the soft tissues of the hoof can be obtained, either directly or by inference, if one only looks for it. This approach is particularly useful in the lame, footsore horse that has no radiographic abnormalities on "standard" foot films (i.e., no obvious pathologic bone findings). Careful evaluation of the soft tissue zones surrounding P3 often reveals interesting details to the trained eye.

EXPOSURE SETTINGS

For any radiographic view, the ideal exposure setting depends on the equipment used, hoof mass, and the purpose of the examination (i.e., the structures of primary interest). The terms *soft*, *medium*, and *hard* describe the exposure settings selected for a particular view, depending on which tissue is being evaluated. (Specific values for kilovolts peak [kVp] and milliampere-seconds [mAs] depend on the equipment used and the size of the foot being examined, so it is not possible to provide even general guidelines here.)

Soft exposures are for nonbony tissues, such as the horn and corium of the hoof wall and sole, and for the solar margin

of P3. A good soft exposure reveals differences in radiodensity within the hoof wall, which allows differentiation between the laminar corium and the keratinized layers of the hoof wall. (When looking for abnormalities at the solar margin of P3 on the 65-degree DP view, a very soft exposure is needed. If the perimeter of P3 cannot be seen without the use of a hot light, the view should be retaken at an even lower mAs setting.)

Medium and hard exposures are used when the structure of interest is bone. Medium exposure is used for bony structures of moderate density or thickness, such as the body of P3, and for articular surfaces. Hard exposure is used for denser bone or superimposed structures, in particular the navicular bone. A 6:1 parallel grid is recommended when using a hard exposure setting.

As this approach illustrates, it is important to tailor the settings to the goal of the examination—to the structures one is most interested in evaluating. "Underexposed" is a relative term. What may seem grossly underexposed for examining bone may be the perfect exposure to show soft-tissue detail within the hoof wall or sole, or the palmar margin of P3.

Unless taking radiographs simply to guide farriery decisions, it is recommended to take at least two exposures for each view: one soft exposure and one bone detail (medium or hard) exposure. Soft-tissue detail is essential, as the nonbony structures surrounding P3 are an integral part of virtually every foot problem.

The coffin bone is surrounded by a dense, cornified shell whose thickness, density, and water content affect radiographic detail of the bones and soft tissues it encases and even of the hoof capsule itself. These characteristics of the hoof capsule must also be factored in to the radiographic technique. If a particular view does not show the amount of detail the examiner is looking for, the view should be taken again at a slightly different mAs setting.

POSITIONING FOR FARRIER-INTEREST VIEWS

The two routine views of most value to farriers are the lateromedial and the standing (zero-degree) dorsopalmar (DP). These views, taken with the horse standing squarely and bearing equal weight on both feet (if able), provide the farrier with an "inside look" of the foot as it appears during visual inspection from the side (lateromedial) and the front (DP). These films show the orientation of P3 relative to the hoof capsule and to the ground, the thickness of the sole and the hoof wall, the position of the shoe relative to the internal structures, and other view-specific information of value to the farrier.

It is a common misconception that the horse's shoe must be removed before taking radiographs of the foot. Whether or not to remove the shoe depends on the purpose of the examination. In fact, taking routine lateromedial and DP views with the shoe on can provide both veterinarian and farrier with valuable information regarding the current shoeing strategy, such as balance, breakover, and load zones.

Lateromedial View

When the principal item of interest is P3 in relation to the hoof capsule and the associated soft tissue zones, the beam should be centered 5/8 to 3/4 inch above the bearing surface (i.e., close to the solar margin of P3), midway between toe and heel. To accurately measure sole depth, dorsal H-L zone width, and palmar angle, the beam must be centered as close to the solar margin of P3 as possible. These indices cannot be accurately measured when the beam is centered at or near the coronary band. In most light horse breeds shod with a normal shoe, the solar margin of P3 is approximately 5/8 to 3/4 inch above the bearing surface of the wall. In Tennessee Walking Horses and other breeds shod with a raised package or with excess length of hoof wall, the beam must be raised accordingly.

Before taking the radiograph, the examiner must ensure that the beam is horizontal and perpendicular to the sagittal plane of the foot, the cassette is positioned so that the entire foot is included and is centered on the film, and the cassette contacts the foot and is perpendicular to the beam. (NOTE: Lining up the heel bulbs by eye as a way of orienting the beam will result in a slightly obliqued view if there is even a slight disparity in the heels, as the beam will not be perpendicular to the sagittal plane of the foot.)

Evaluating the Positioning

If the positioning block is an appropriate height and the x-ray beam is horizontal and centered between the shoe and the solar margin of P3, both branches of the shoe will be precisely superimposed (i.e., only one shoe branch is seen). If the foot is balanced lateromedially, both wings of P3 will also be precisely superimposed (i.e., only one wing is seen).

When the shoe branches are superimposed but the wings of P3 are not (i.e., one shoe branch but two wings are seen), it indicates lateral-medial imbalance, which can be confirmed on the DP view. (NOTE: If the solar margin of P3 is greater than 1 inch [25 mm] above the block, it may be necessary to raise the beam a little to accurately assess lateral-medial balance on the lateromedial view.) If the shoe branches are not superimposed, it indicates a positioning problem, such as the beam not being horizontal or being centered too high on the foot. Sole depth, palmar angle, and dorsal H-L zone width cannot be accurately measured on such a film.

Exposures Recommended

The two exposures recommended for this view are soft and medium. The soft exposure, in particular, is a valuable farrier-interest view. Provided the dorsal hoof wall is delineated along its entire length with a radiopaque marker, this view allows accurate assessment of sole thickness and cup depth, lateral-medial balance, digital breakover, dimensions, and radiodensity of the H-L zone, C-E distance, and palmar angle. In addition, it reveals the profile of P3, and even bone detail along the thin solar margin. In horses with white line disease, this view also shows the extent of cavitation in the deteriorated soft inner zone of the hoof wall. This view is a blueprint for all therapeutic trimming and shoeing strategies.

Standing (Zero-degree) Dorsopalmar View

When the principal item of interest is P3 in relation to the hoof capsule and to the ground, the beam should be centered at the toe, 5/8 to 3/4 inch above the bearing surface of the wall. The examiner must ensure that the beam is horizontal and parallel with the sagittal plane of the foot, the cassette is positioned so that the entire foot is included and is centered on the film, and the cassette is perpendicular to the beam.

For routine DP views, the cassette is placed behind the foot, as close to the heels as possible, while making sure the cassette remains perpendicular to the beam. This approach produces a somewhat magnified yet relatively undistorted image.

The exposure recommended for this view is soft to medium. This exposure allows evaluation of P3 in relation to the hoof capsule, the hoof capsule in relation to the ground, and thus lateral-medial balance. It also allows evaluation of sole depth, hoof wall thickness at the quarters, and the extent of cavitation in horses with white line disease.

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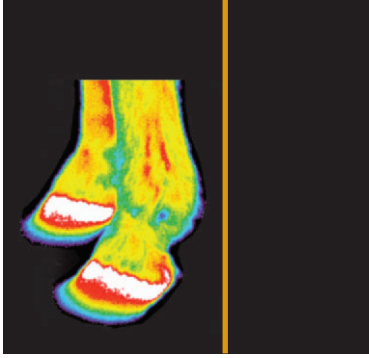

Section III PATHOLOGIC CONDITIONS OF THE EQUINE FOOT

11 DEFORMITIES OF THE LIMB AND THEIR RELEVANCE TO THE FOOT

ANDREA E. FLOYD

A variety of musculoskeletal (muscle and bony column) conditions can result in deformity of one or more limbs. Interest in these conditions for the equine podiatrist is twofold:

- The deformity can negatively affect the foot by altering the way the foot is loaded.
- In certain situations, it is possible to positively affect the limb in the growing animal, and improve biomechanics in the mature animal, by carefully manipulating the hoof capsule.

Deformity of a limb can develop at any age, from before birth to well into old age; however, it is much more likely to occur while the horse is growing. A constellation of musculoskeletal conditions exist that either are present at birth or develop as the foal grows. Collectively, they are known as developmental orthopedic disorders (DODs).

The most common DODs are:

- Angular limb deformities (ALDs)—deviation of the limb when viewed from the front or rear of the horse (crooked legs; e.g., knock knees, bow legs) (Figure 11-1)
- Flexural limb deformities (FLDs)—deviation of the limb when viewed from the side of the horse, divided into flexor laxity and flexor contracture (e.g., clubfoot, knuckled fetlocks) (Figure 11-2)
- Physitis/epiphysitis—abnormalities of growth cartilage
- Osteochondrosis—abnormal development involving a joint surface

Rotational limb deformities, in which the limb (or a portion of the limb) twists or spirals slightly along its long axis, also are common. For example, horses that toe in (pigeon-toed) or toe out (splay-footed) usually have some degree of rotational deformity of the lower limb. When the limb is examined carefully, it is apparent that the rotational deformity often has components of both ALD and FLD, although both defects may be very mild in functionally sound horses with a toe-in or toe-out conformation.

Uncommon conditions that could be characterized as DODs include osteochondroma (abnormal outgrowth of bone and cartilage from a growth plate), hypoplasia or aplasia (incomplete development or complete absence of a structure, respectively), and polydactyly (supernumerary digits).

In addition to being among the most common of these conditions, angular and flexural limb deformities also are the ones most likely to affect the foot or be affected by manipulation of the foot. Thus they are the focus of this chapter. It is important to note, however, that any condition that affects the way the limb is used or loaded inevitably affects the foot in some way if it is not promptly and appropriately addressed.

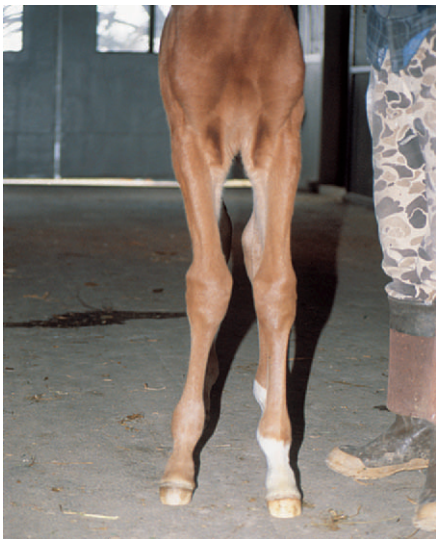

FIGURE 11-1 Angular limb deformity, in this case carpal valgus. *(Courtesy R.F. Redden.)*

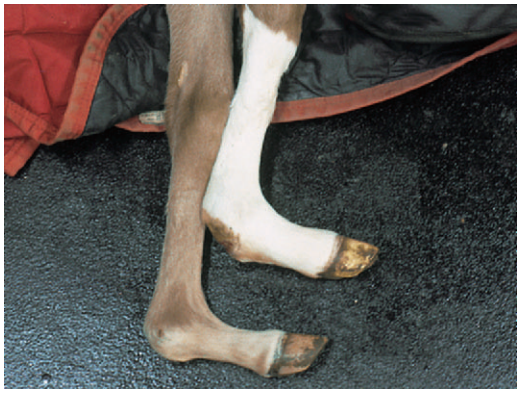

FIGURE 11-2 Flexural limb deformity, in this case flexor laxity. *(Courtesy R.F. Redden.)*

MECHANISMS OF ABNORMAL LIMB ALIGNMENT

To effectively manage or prevent limb deformities, it is important to understand how and why they develop. That discussion begins with a brief overview of how the bones and soft tissues of the limb normally develop.

Normal Bone Development

In the fetus, the skeleton is formed on a scaffolding of cartilage. This specialized cartilage is called *growth cartilage.* As the developing fetus, and later the newborn foal, grows, this growth cartilage proliferates in an organized manner and progressively transforms into bone in a beautifully orchestrated production that ultimately results in the fully formed bones of the adult horse. This process is called *endochondral ossification* (meaning "bone production from within cartilage"). It continues in the various growth centers of the bones until the horse reaches skeletal maturity, usually around 4 or 5 years of age (earlier or later, depending on the breed).

Growth Centers in the Long Bones

In the long bones of the limb (i.e., the bones that are longer than they are wide), there are two important growth centers at both ends of the bone: the physis and the epiphysis. The physis, or "growth plate," is a band of growth cartilage located near each end of the long bones. The physis is primarily responsible for lengthwise bone growth, as the cartilage cells in the physis proliferate in an organized manner along the long axis of the bone before becoming mineralized and transforming into bone.

Unless the growth cartilage is irreparably damaged, each physis "closes" or ceases lengthwise bone growth at a relatively predictable time in the young horse's life. Some of the physes in the limb are effectively closed before or soon after birth, whereas others may not be completely closed until the horse is at least 4 years of age (sometimes older, depending on the breed). For the most part, however, the majority of lengthwise limb growth occurs before the horse is 12 months of age.

The epiphysis is the other primary growth center for the long bones. As its name suggests (*epi*-physis: "*upon* the physis"), the epiphysis is the portion of the bone that lies beyond the physis, between the physis and the articular (i.e., joint) surface. There is an epiphysis at each end of the long bones. Initially, the epiphysis is composed of growth cartilage that gradually ossifies as the fetus develops. By the time the fetal foal reaches full term, most of the epiphyseal growth cartilage has been transformed into bone. Ossification continues as the newborn foal grows, until the entire epiphysis is composed of bone except for a thin layer of articular cartilage at the joint surface.

Growth of Other Bones

Other bones in the limb, such as the block-shaped "cuboidal" bones in the knee and hock, the sesamoid bones at the back of the fetlock, and the third phalanx (P3), also develop and mature by the process of endochondral ossification. However, these bones do not have physes and epiphyses. The cuboidal bones progressively ossify from their centers to their articular surfaces, and P3 ossifies from its body and from three distinct secondary ossification centers: one at the base of the extensor process and one at each wing (medial and lateral).

Bone Modeling throughout Life

Bone is living, dynamic tissue. Even after the horse reaches skeletal maturity, its bones continue to respond and adapt to the various and variable loads placed on them, which include compression, tension, rotation, shearing, and bending forces. Throughout the horse's entire life, each individual bone continually remodels in response to the loads it experiences. Thus, while the period of growth is certainly the time of greatest change in bone and the time when bone shape (and thus limb conformation) is most susceptible to alteration (for better or for worse), bone remains responsive to some degree throughout the horse's life.

Normal Soft Tissue Development

The soft tissues of the limb—muscles, tendons, ligaments, fascia, joint capsules, nerves, skin (and its specialized product, the hoof)—also develop in an organized, coordinated manner as the young horse grows. All of these soft tissues are fully formed

when the foal is born, although they are still in a relatively immature and largely "untried" state and must grow in size and strength (i.e., functional capacity) as the skeleton grows. As with bone, these tissues are responsive to the loads placed on them, especially during growth but also throughout adulthood.

Neuromuscular Development

Neuromuscular development is particularly relevant to this discussion of limb deformities. Muscle tone affects the degree of tension in the associated tendons and fascia and thus affects the bones and joints influenced by these tissues. Muscle tone is determined in large part by input from the nerves that supply that muscle, so ultimately joint angle and bone loading are dictated largely by neuromuscular activity.

Basic nerve–muscle pathways and circuits are laid down before birth, which is how a foal that is mere hours old is able to stand, walk, and even run with its mother. More specific pathways are created and reinforced after birth, based on how the individual foal uses its limbs and the rest of its body. Persistence or repetition of a particular stance or movement very quickly becomes habitual. So to some extent, postural and movement patterns are highly individualized, develop early, and can be very difficult (but not impossible) to change later in life.

Requirements for Normal Development

The normal body is programmed for normal development. However, for this potential to be realized—for the horse to develop and function normally—a number of conditions must be met, including the following:

- Good genetic foundation
- Optimal uterine environment during fetal development
- Optimal nutrient intake, from fetus to maturity
- Optimal stimulation in the form of movement

Genetic Foundation

The genetic blueprint, obtained from both parents, is one of the prime determinants of how the foal will develop. A blueprint for optimal conformation (however one defines it) is an essential starting point, although the effect of diet, activity, and all of the other external factors that have an impact on a body before or after birth have an equally important role in the outcome. (So, in the "Nature versus Nurture" debate with regard to horses, one must answer, "Both.")

Hereditary Conditions. Certain DODs—for example, osteochondrosis in the hock and some cases of mismatched feet and clubfoot—are known or suspected to have a genetic component. This area of equine science has not been extensively researched. In time, it will very likely be discovered that the expression of a genetic propensity for a particular condition is largely determined by external factors. In other words, even if a foal has a genetic predisposition for club foot, early recognition and appropriate treatment can minimize the expression and impact of this condition on the horse's health and function (see p. 218).

Rapid Growth and Large Size. Also speculated, but not well researched, as a factor in DODs is the simple genetic propensity for rapid growth or large body size, or both. Typically, it is the large, rapidly growing foals in a group that are most prone to DODs that may lead to limb deformity. However, rapid growth or large body size on its own may not be enough to precipitate these problems. Usually there are compounding factors, such as pain or dysfunction somewhere in the limb that alters the way the limbs are loaded or used, an inappropriate diet (especially an excess of dietary carbohydrates), or an inappropriate level of activity (e.g., forced exercise in a foal that is raised in confinement, or complete confinement that does not allow for natural movement and exercise).

Uterine Environment

In addition to her genetic contribution, the mare's part includes provision of a uterine environment that is conducive to optimal fetal development. It must be spacious enough to accommodate the growing fetus, provide sufficient nutrients and efficient removal of waste products, and protect the fetus from potentially detrimental substances and events.

Lack of Space. Lack of space in the uterus has long been proposed as one of the primary causes of ALDs and FLDs that are present at birth. It may be that insufficient space contributes to some of these problems; however, it is probably only part of the story, as the size of the uterus and the functional capacity of the placenta influence the size and growth rate of the fetus.

In a recent study examining the influence of maternal size on placental, fetal, and postnatal development, researchers compared four different breeding combinations: Thoroughbred (TB) stallions and Thoroughbred mares (TB-in-TB); pony (P) stallions and pony mares (P-in-P); Thoroughbred embryos placed in pony mares (TB-in-P); and pony embryos placed in Thoroughbred mares (P-in-TB). The presumption was that the P-in-TB combination represented a "luxurious" uterine environment, and the TB-in-P combination represented a restricted or "deprived" uterine environment, in terms of both space and placental nutrition.[1,2]

As might be expected, at birth the TB-in-TB foals were largest, the P-in-P foals were smallest, and other two combinations were intermediate in size. Interestingly, the pony foals that gestated in a Thoroughbred uterus were larger than the Thoroughbred foals that gestated in a pony uterus, even though the average gestation length was the same for both groups. The authors concluded that the size of the mare interacts with the foal's genotype (its genetic blueprint) to control the rate and extent of fetal growth, by influencing the size and functional capacity of the placenta.[1]

The authors did not report any ALDs or FLDs at birth in any of the foals, other than temporary hyperextension of the fetlock joints (flexor laxity; see p. 213) in some of the TB-in-P foals. In comparison with the other foals, the TB-in-P foals (i.e., those from a deprived uterine environment) appeared somewhat dysmature (developmentally immature for the gestational age) at birth. They required some assistance to stand and suck during the first 24 hours of life and showed various degrees of muscle underdevelopment on the body and upper limbs. However, those foals quickly made up ground after birth.[1] The researchers monitored all of the foals from birth to 3 years of age and reported that only one foal (a TB-in-P filly) developed a limb deformity.[2]

Lack of physical space in the uterus may therefore be getting too much credit for causing ALDs and FLDs in newborn foals. More likely, restricted placental area and thus fetal nourishment is the key factor, as is also seen when the foal is a twin. Twin fetuses compete for physical space in the uterus and for placental nourishment, as both commodities are in

limited supply, particularly in the latter half of pregnancy when fetal needs are greatly increased. In twin equine pregnancies that manage to remain viable until term, it is common for one or both foals to be dysmature. These foals commonly have significant ALDs or FLDs at birth that are related simply to joint and tendon laxity and incomplete ossification of the cuboidal bones of the knees and hocks—in other words, musculoskeletal immaturity.

Fescue toxicity, caused by ingestion of endophyte-infected tall fescue grass or hay by the pregnant mare, may indirectly affect fetal development through similar means. Typically in these cases, the placenta is abnormally thickened and gestation is abnormally prolonged because of hormonal interference by the ergot alkaloids in the infected fescue. By the time the foal is born, it may be very large (and thus quite cramped for space); yet despite the longer-than-normal gestation length, these foals often are somewhat dysmature, presumably because placental function is so impaired.

Other Maternal Factors. Several other maternal conditions can negatively affect fetal development, and thus cause or contribute to limb deformities in the newborn foal, by affecting the fetus directly or by compromising the health or function of the placenta. They include placentitis (inflammation of the placenta, usually caused by bacterial infection or rarely by fungal or viral infection); severe maternal illness that causes high fever or toxemia (e.g., influenza, severe colic); and ingestion of various toxic plants, including locoweed, Sudan grass, and goitrogenic plants (plants that interfere with thyroid function).

Nutrient Intake

Normal musculoskeletal development requires optimal nutrient intake. An adequate supply of nutrients and an appropriate balance of nutrients must be provided throughout the entire growth period, taking into account the foal's specific nutritional requirements at each stage of development (fetus, neonate, suckling, weanling, yearling, and so on).

A complete discussion of the nutritional requirements of the pregnant mare and the growing horse is beyond the scope of this chapter. The following books, published by the American Association of Equine Practitioners, are good starting points for interested readers: *Basic Equine Nutrition and Its Physiological Functions* and *The Veterinarian's Practical Reference to Equine Nutrition.*[*]

The dietary factors related to DOD in foals have recently been reviewed,[3-5] so only a brief overview is presented here. Of particular importance are these:

- *Carbohydrates*—particularly excessive intake of grain-based feeds
- *Minerals*—deficiency, excess, or imbalance of calcium, phosphorus, copper, or zinc

Carbohydrates. The dietary intake of carbohydrates must be adequate to fuel normal growth, otherwise the foal's development may be delayed or stunted. However, excessive carbohydrate intake, particularly grain-based feeds (grain in any form) and perhaps lush pasture, is a major contributing factor with various DODs, including osteochondrosis, physitis/epiphysitis, and FLDs. For example, osteochondrosis can be induced simply by feeding weanling foals as little as 30% more dietary carbohydrate than needed (i.e., an extra 0.3 lb for every 1 lb fed). In the nursing foal, excessive carbohydrate intake by the mare, and thus overabundant milk production, can lead to DOD in the foal.

The exact reason why an excess of dietary carbohydrate increases the risk for DOD is still being researched. Recent findings point to abnormalities in blood insulin and thyroid hormone levels associated with high-carbohydrate meals, as abnormal levels of these hormones can adversely affect growth cartilage.

The goal in devising a ration for the nursing mare or the weanling foal is to attain a moderate rate of growth, rather than rapid growth. Although it may seem appropriate to increase the amount of feed for a foal that is going through a growth spurt, it is a far better strategy to decrease the foal's intake of carbohydrates (grain-based feeds and time spent on lush pasture) until the foal's growth rate is more moderate. Careful attention to mineral intake and balance also is important in these foals.

(Note: Excessive protein intake is not a contributing factor in DOD, unless the high-protein diet also is high in carbohydrates, in which case it is the excessive carbohydrate intake that is the problem.)

Calcium and Phosphorus. These minerals are the major inert constituents of bone. They are not the only minerals stored in bone, but they also comprise the bulk of bone's mineral content. As such, adequate amounts of both minerals are essential for normal bone development. In addition to the total amount of each mineral in the diet, the balance, or ratio, of calcium to phosphorus is important, because an excess or deficiency of one negatively affects the absorption and metabolism of the other. Ideally, the calcium-to-phosphorus ratio (Ca:P) from all dietary sources in the growing horse should be between 1.5:1 and 2.5:1. Ratios above or below this range can cause problems with bone development.

When it comes to DOD, an excess of phosphorus is more problematic than an excess of calcium. High levels of dietary phosphorus inhibit the absorption of calcium, thereby creating a relative calcium deficiency, even though the diet contains a seemingly adequate amount of calcium. Experimentally, excessive dietary phosphorus greatly increased the incidence of osteochondrosis, whereas excessive dietary calcium did not.[4] Furthermore, adding extra calcium did not protect against the development of osteochondrosis in foals fed an excess of dietary carbohydrate. Although excessive dietary calcium on its own does not appear to increase the risk for DOD, gross excesses of calcium can cause secondary problems by interfering with the absorption of phosphorus, zinc, and iodine.

Incidentally, grains have a Ca:P ratio of less than 0.3:1 (i.e., much more phosphorus than calcium), and many grass hays have a Ca:P ratio of barely 1.5:1. Thus, diets that comprise unfortified grain (e.g., plain oats) and grass hay are inadequate for a growing horse, as they contain too little calcium and a Ca:P ratio that is too low.

Copper. Copper is an essential element in the process of collagen production. Because collagen is a critical structural component of cartilage and bone, adequate dietary copper is important for normal skeletal development. An increased incidence of DOD has been reported in foals born to mares fed low-copper diets and in foals who themselves were fed low-copper diets. Supplementing the pregnant mare's or, later, the foal's diet with copper can lower the incidence of

*American Association of Equine Practitioners, 4075 Iron Works Parkway, Lexington, KY, 40511. www.aaep.org.

DOD on a farm.[3,4] High-copper diets do not completely prevent DOD, however.[4] It is important to remember that copper is only one of several key dietary components required for normal skeletal development. Supplementing a mare's or foal's diet with copper while the diet contains excessive amounts of carbohydrate or has an inappropriate Ca:P ratio is not likely to be effective.

Zinc. Among its many roles, zinc is essential for the normal development of connective tissues and for bone and joint integrity. As with calcium and phosphorus, the ratio of zinc-to-copper (Zn:Cu) is as important as the actual amounts of each mineral in the diet. For normal development, the diet should provide a Zn:Cu ratio of between 3:1 and 4:1.

Diets that are very low in zinc have been linked to DOD but so have diets containing excessive amounts of zinc, as high-zinc diets create a secondary copper deficiency by inhibiting the absorption of copper. Such high-zinc diets are uncommon, unless the diet is oversupplemented with zinc and is marginal or low in copper. Other potential sources of zinc on a farm include galvanized water pipes and fence paint.[5]

Other Minerals. In addition to the minerals already discussed, excessive dietary intake of iodine, selenium, fluoride, and heavy metals such as lead, molybdenum, and cadmium may cause or contribute to DOD. This situation may result from oversupplementation or from environmental contamination.[4,5]

Vitamin D. Vitamin D is essential for normal calcium and phosphorus absorption and metabolism and thus for normal skeletal development. Foals with severe phosphorus or vitamin D deficiency may develop rickets, a disorder in which the bones are so soft that they bend or bow under even normal weight bearing. Fortunately, rickets is rare these days, unless the foal is grossly mismanaged (e.g., fed a completely inappropriate diet and deprived of sunlight).

Movement

The body is designed to move, and movement is essential for normal development. However, the type and amount of movement must be well matched to the current loading capacity of the tissues. With too great a load, a structure may fail at its weakest point. On the other hand, with too little a load, there may be insufficient stimulus for normal development.

Particularly in weanlings and yearlings bred for racing or showing, too little exercise often is coupled with too great an intake of dietary carbohydrates. When these overfed, underactive "hothouse flowers" are then forced to exercise in preparation for a sale or show, lameness is an all-too-common result. As pain induces and perpetuates a protective flexor withdrawal response and otherwise alters the way in which the limb is loaded and used, this situation is ripe for the development of ALDs and FLDs.

Ground Surface. The ground surface rates a mention here, too, because the entire musculoskeletal system, including the hoof, adapts to the environmental conditions to which it is subjected. In the young foal, particularly, extremes should be avoided as much as possible. That includes too hard, too soft, too wet, too dry, too irregular, too slick, too deep, and too abrasive. As the foal matures, its ability to negotiate and tolerate these various surfaces increases, although common sense should prevail when managing horses of any age. A balance must be achieved between avoidance of injury and the application of sufficient stimulation to create and maintain a healthy, resilient musculoskeletal system.

Causes and Contributing Factors for Angular Limb Deformities and Flexural Limb Deformities

Developmental orthopedic disorders, including ALDs and FLDs, can be divided into two broad categories, based on when the defect first appears:

- *Congenital*—present at birth
- *Acquired*—develops as the foal grows (or even later in life)

The underlying or predisposing factors vary somewhat with the category.

Congenital Conditions

By definition, congenital conditions are simply those problems that are present at the time of birth. Some are hereditary (i.e., genetically determined), but others are not and develop during fetal development for reasons other than heredity. Not a lot of research has gone into this area, so at this time we can only speculate that congenital DODs that are not hereditary involve problems with one or more of these factors:

- Uterine environment
- Mare's diet during pregnancy
- Mare's health during pregnancy

Acquired Conditions

Angular or flexural limb deformities that develop after birth can occur for a wide variety of reasons, including the following:

- Inappropriate diet, particularly excessive carbohydrate intake
- Inappropriate type or amount of exercise, especially too much superimposed on too little (e.g., forced exercise in a foal that is raised in confinement)
- Inappropriate hoof care, whether improper trimming or footing
- Musculoskeletal injury or disease that seriously damages part of the growth cartilage; common causes include trauma and infection (primarily in very young foals)
- Musculoskeletal injury or disease that, through pain or dysfunction, alters the way in which the limb is loaded or used
- Injury to a soft tissue structure (muscle, tendon, ligament, joint capsule, fascia) that results in fibrosis (scarring) and functional shortening or restriction of that structure

Heredity probably should be added to that list, as some hereditary conditions or propensities do not manifest until after the foal is born. A common example is osteochondrosis. It often takes the superimposition of improper diet and inappropriate activity to induce clinical disease in these foals. Another example may be the development of club foot in a number of foals sired by the same stallion. The foal may not be born with a flexural deformity, but a club foot may develop later, sometimes within the first few days of life, again typically under the influence of other factors (e.g., inappropriate diet or exercise, pain).

Biomechanical Perspectives

Helen M.S. Davies

Angular and flexural limb deformities occur when there is a mismatch in lengthwise growth between different bones or different parts of the one bone, or when the soft tissues that support the joints are so consistently lax or tight that they cause an abnormal posture through those joints and hence cause the bones to change shape to accommodate the change in loading.

Bones do not grow in length against gravity; it is the cartilage in the growth plate that is growing. It is only when bone is relieved of the effect of gravity (e.g., bed rest, external support, space travel) that it will grow in length once the growth plates are closed. Hence, problems in the lengthwise growth of long bones may cause angular limb deformities in the distal limb only in very young foals or in utero (during fetal development) when the cartilage growth plates are still open and active.

Tight restriction across a growth plate will reduce the ability of the cartilage to expand and hence limit its growth, while tensioning may encourage faster expansion. In this way, differential loading, and especially differences between compression and tension across the bone, are likely to cause changes in the straightness or alignment, or both, of the limb during development. Hence, flexural deformities can develop as a consequence of changes in the pattern of loading of the region as the animal grows.

A change in loading could arise from a number of different situations, including the following: underlying conformational problems, such as tilted or crooked joint surfaces or poorly positioned ligament or tendon attachments; normal differences in bone length between the left and right sides[6]; sources of pain or dysfunction due to physical or other damage; or uneven or otherwise dysfunctional movement habits.

Changes in loading can also occur through lack of, or unbalanced, stimulation from both the physical and emotional environment. How the locomotory structures develop and are maintained depends on the total input from everything the horse normally does. That includes their habitual postures, such as those associated with fear or relaxation; opportunities for exploring the normal range of joints in play; too much stimulation leading to exhaustion; and just about anything else that varies or may be less than optimal in the environment, including pasture companions, nutrition, and the weather.

Superimposed on all this is the effect of the dietary supply of nutrients required for optimal growth and adaptation to changing loads, and the underlying metabolic capabilities of the horse's body to supply these nutrients where and when they are needed.

Mechanical or chemical damage to specific physeal or epiphyseal plates may cause angular limb deformities while the bone is still growing in length. Such deformities are a common consequence of accident or infection of the growing areas of bones, where two bones are present and only one stops growing in length, or a growth plate is damaged on only one side of the limb.

Thus there are potentially four common factors underlying angular and flexural limb deformities: (1) problems or interference with the normal loading of the region, (2) mechanical damage, (3) nutritional restriction or imbalance, and (4) infection.

The third metacarpal bone (the cannon bone) does not grow significantly in length after 10 weeks of age,[7] although the distal growth plates often are still visible radiographically at 6 months of age. Thus deformities of the distal limb that develop after 10 weeks of age must be due to mechanisms not associated with lengthwise bone growth. Once the bones have reached their adult length and all the growth plates have fully ossified, there is no further lengthwise bone growth. However, the bones still change their shapes and relative sizes according to the loading that they receive during normal use. This potential for change persists throughout adult life and involves constant modeling and remodeling of all the bones of the skeleton.

Changes in hoof balance in very young foals causes a very rapid adaptation in the metacarpal bone. Firth et al[8] used lateral hoof wedges in very young foals to demonstrate that increased loading of the medial cortex of the third metacarpal bone (MC3) returned to pretreatment patterns within 10 days. This effect may be confined to young foals in which the metacarpal growth plates are still open; however, bone in general adapts very rapidly to any changes in the loading environment, so a similar effect is likely to occur at any age, although this assumption remains to be tested.

It is likely that bone will model (change shape) to optimize loading within weeks if the loading is outside the range of normal for that region of bone, as long as the new loading does not cause bone damage (in which case adaptation will take longer, as the damaged bone has to be removed first). Hence, hoof angle may have a long-term effect on bone shape at any time during life. Similarly, when there is a lot of pain or restriction around or within a joint, it is likely to lead to some distortion of the associated bones at any time.

Horses appear to be able to tolerate some degree of crookedness (such as offset, rotated, or angled cannon bones) with no apparent effect on their soundness or performance, as long as the joint surfaces of the fetlock and coffin joint are relatively parallel to the ground surface. If the joint surfaces are tilted, especially in the coffin joint, then it may cause unbalanced loading of the limb and may lead to lameness whenever the workload increases or the type of work changes. Furthermore, there is likely to be an accumulation of damage until the horse becomes chronically unsound. The greater the angle of tilt, the more likely it is for this situation to occur. However, this process may take years.

If the fetlock joint is tilted or rotated, then the direction of movement of the digit in the flight phase of the stride is likely to cause interference between the limbs, although there may be no obvious detrimental effects on the horse's soundness. In contrast, a tilt or rotation in the coffin joint is more likely to be associated with a club foot or general clumsiness in the use of that limb and perhaps the development of low articular ringbone (osteoarthritis involving the coffin joint) and chronic, low-level unsoundness.

ANGULAR LIMB DEFORMITIES

Definitions and Mechanisms

Angular limb deformities are those in which the limb deviates from the normal vertical alignment when viewed from the front or the rear. With rare exceptions, the point of deviation is a joint

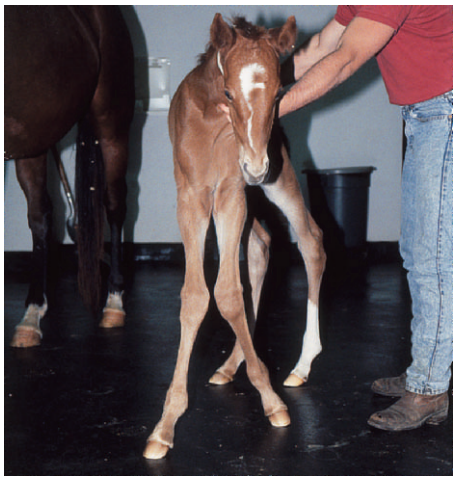

FIGURE 11-3 Severe, bilateral carpal valgus results in a "knock-kneed" foal. *(Courtesy R.F. Redden.)*

FIGURE 11-4 Valgus deformity in one limb and varus deformity in the other results in a "windswept" foal. *(Courtesy R.F. Redden.)*

or its adjacent physis. The specific reason for the deviation is one or more of the following:

- Laxity of the soft tissues that support the sides of the joint
- Unequal bone growth between medial and lateral sides of the physis
- Abnormal ossification of the epiphysis, resulting in a tilted or otherwise abnormally oriented joint surface
- In the knee or hock, incomplete ossification and subsequent collapse of the vulnerable cuboidal bones on one side of the joint

The ALDs are named according to whether the limb below the affected joint deviates toward or away from the midline of the body. *Valgus* is the term used when the lower limb deviates away from the midline. For example, a foal with valgus deformities of both knees appears "knock-kneed," with the feet set wider apart than the knees (Figure 11-3). *Varus* is the opposite: the limb below the pivot point deviates toward the midline. A foal with varus deformities of both knees appears "bow-legged." A foal with a valgus deformity of one knee and a varus deformity of the other knee is called "windswept" (Figure 11-4).

In the vast majority of cases, the deviation is centered at the carpus (knee), the tarsus (hock), or the fetlock (often with coinvolvement of the pastern). The most common ALDs, in descending order, are carpal valgus, tarsal valgus, and fetlock varus. Rotational deformity often accompanies the ALD. An outward rotation tends to be seen with valgus deformities, and an inward rotation with varus deformities. Thus the foal with carpal valgus may stand toed-out, and the foal with fetlock varus may stand toed-in.

Congenital Angular Limb Deformities

Congenital ALDs are common. In fact, many people consider mild, bilateral carpal or tarsal valgus and a toed-out stance to be normal in newborn foals and to resolve without special care as the foal grows. In most cases, congenital ALDs are simply the result of laxity in the soft tissues that support the affected joints. In such cases, it is possible to manually straighten the limb with little effort.

Management

Unless the deviation is severe, these limbs usually straighten just with controlled exercise and provision of good footing. No manipulation of the hoof is necessary in most of these foals. Depending on the circumstances and the facilities available, appropriate exercise for these foals may comprise confinement with the mare in a large foaling stall, with short periods of turnout in a small, level paddock two or three times per day. Good footing for these foals primarily means avoidance of deep bedding and slippery surfaces. Deep bedding requires the young foal to make exaggerated or otherwise abnormal movements to get about the stall. Slippery surfaces encourage the foal's foot to slide out from underneath the limb, potentially worsening the deviation.

Severe congenital ALDs generally do not resolve just with controlled exercise and provision of good footing. If the deviation is severe or if it remains after 1 to 2 weeks of age despite appropriate care, the foal should be managed as described for acquired ALDs.

Incomplete Cuboidal Bone Ossification. In foals that are premature or dysmature (developmentally immature for their gestational age), joint laxity can be severe, and incomplete ossification of the cuboidal bones may also be involved in the deviation. It is important to identify these foals immediately and to monitor ossification radiographically, because exercise without some form of joint support can worsen the problem by allowing crushing of the vulnerable growth cartilage of these immature cuboidal bones. Management of these cases is beyond the scope of this chapter and is discussed elsewhere.[9]

Acquired Angular Limb Deformities

Angular limb deformities that develop or worsen after birth require the involvement of a veterinarian and a farrier. The

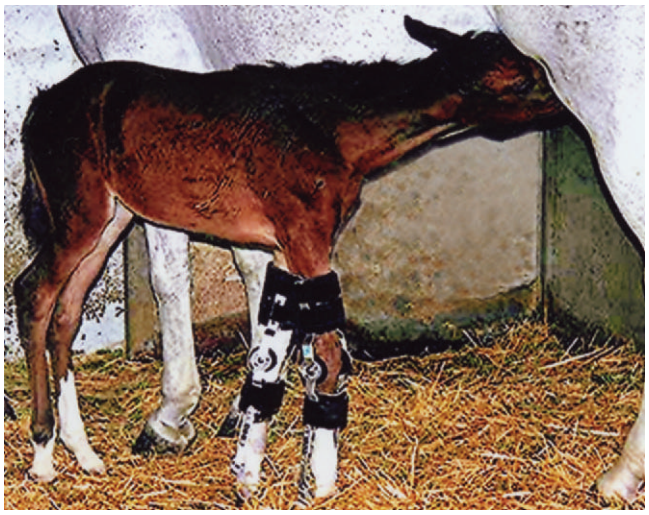

FIGURE 11-5 Management of an angular limb deformity using an orthotic brace. *(Courtesy Valentine Robles, NTD Equine Bracing.)*

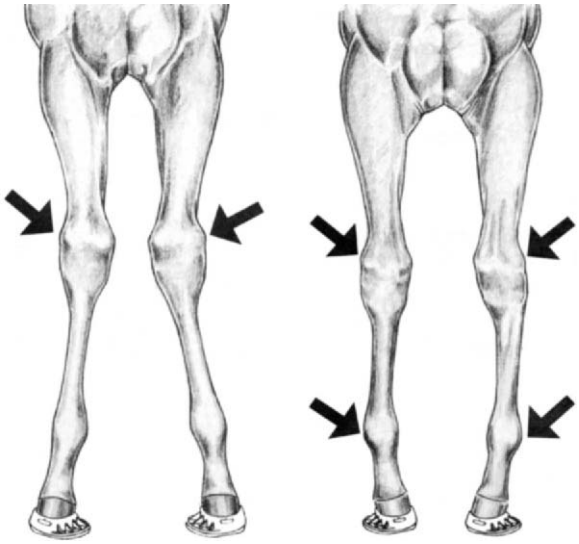

FIGURE 11-6 Lateral or medial hoof extensions can be used to help correct varus and valgus deformities. *(Courtesy R.F. Redden.)*

first step is to determine the cause of the deformity and address it appropriately.

General Management Approach

Most cases self-correct with controlled exercise, attention to footing, proper hoof care, and appropriate management of any underlying problems (e.g., lameness in that limb or the opposite limb). However, if the deviation is the result of permanent damage to part of the physeal growth cartilage, then surgery may be required to optimize the remaining growth from that physis. (Surgical options are discussed elsewhere.[9]) It has been customary to treat moderate to severe ALDs with splinting or casting. Orthotic braces offer a dynamic alternative to these static forms of joint support (Figure 11-5).

In all cases, it is important to identify the deformity early and address it appropriately. The physes adjacent to the fetlock are functionally closed by 3 months of age, so rapid manipulation of their growth is possible only in very young foals. The physes just above the knee and hock close much later; but even so, the sooner the problem is identified and corrected, the better the outcome will be.

Hoof Care

The hoof can be used to improve the alignment of the developing limb. In young foals, careful trimming of the hoof to optimize hoof balance can be effective, when used in conjunction with controlled exercise and attention to footing. Care must be taken, however, not to overtrim the immature foot, something that is easily done with such a small foot and such soft horn. Imbalance of the hoof or soreness caused by excessive trimming can quickly worsen the situation. It is better to trim very small amounts every 1 to 2 weeks than to trim more vigorously but less often. Frequent visits also allow the veterinarian or farrier to monitor the rate of improvement and intervene sooner if change is needed.

Periosteal stripping is a common surgical technique used for the treatment of ALDs in foals, particularly for defects involving the carpus.[9] However, in a recent study in which carpal valgus was surgically created in foals, weekly hoof rasping to maintain mediolateral hoof wall balance was just as effective in correcting the deformity as was periosteal stripping and hoof rasping.[10] As this information is absorbed into routine equine practice, it is hoped that this surgery will be performed less and less, and more attention will be paid to hoof balance and regular hoof care in foals with ALDs.

It has also been common practice to intentionally lower one side of the hoof to address ALDs in foals. However, this practice is counterproductive in the long run, as it worsens the uneven loading of the limb, particularly of the developing physes and epiphyses, and can lead to uneven bone growth (persistent or new ALD, rotational limb deformity, or offset distal limb). With the offset distal limb condition, the limb distal to the joint of interest is more or less vertical, but it is set a little lateral or medial to the long axis of the limb proximal to the joint. In most cases, offset cannons or pasterns are the result of inappropriate trimming of the juvenile foot in an effort to correct an ALD and not a deformity that occurs spontaneously. Instead of lowering one side of the hoof to correct limb deformities, the hoof should be carefully trimmed to achieve and maintain a balanced foot and, if necessary, hoof extensions applied to improve loading of the limb.

Hoof Extensions. Hoof extensions are commonly used to address ALDs in young foals. Several hoof appliances are available that can be glued onto the infant hoof to provide medial or lateral extensions, or toe or heel extensions, depending on the purpose of the device (Figure 11-6).

Medial or lateral extensions (as the individual case requires) have been quite helpful in reducing the need for surgery in foals with ALDs. However, care must be taken not to leave these devices on for more than 2 weeks at a time in young foals, as they limit hoof expansion and can thus impede the growth of the developing foot. Foals wearing hoof extensions of any kind should be monitored very closely by the veterinarian and the farrier.

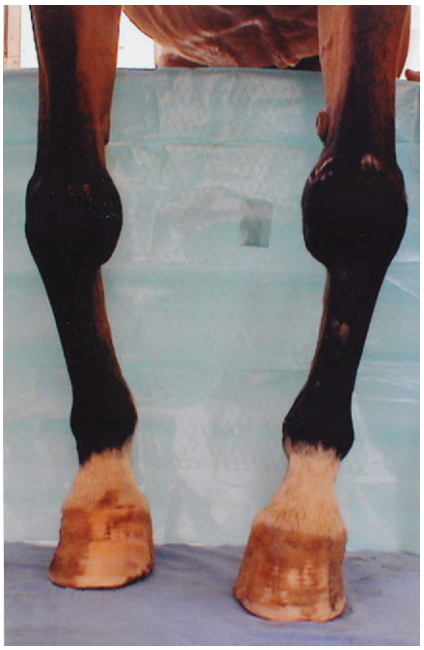

FIGURE 11-7 Uncorrected carpal varus in an older, neglected horse.

Whether to apply the extension on the medial or lateral side of the foot depends on the direction of the deviation. In general, the following rules apply:

- Valgus deformities (in which the lower limb deviates laterally) require medial extensions
- Varus deformities (in which the lower limb deviates medially) require lateral extensions

The objective in each case is to shift the base of support to where it should be under the main axis of the limb. In the case of uncertainty as to which side of the hoof requires an extension, a plumb line can be dropped down the front of the upper leg; where the plumb bob hangs is the side that requires an extension.

As useful as they can be, hoof extensions should not be relied on as the only means of addressing ALDs in foals. It is far better to identify these defects early and address them with controlled exercise and careful, balanced trimming than it is to attempt to manage them once they are established. In Chapter 23, the general manager of a large Thoroughbred breeding farm describes how implementing a broad-based program to improve hoof quality in their horses decreased the incidence of various foot problems that were plaguing their breeding and racing programs. Simply by maintaining a monthly schedule of routine farriery care for all foals, starting at 2 weeks of age, they were able to reduce the need for hoof extensions in their foals.

Management of Angular Limb Deformities in Adult Horses

Angular limb deformities that were not adequately addressed in the first few months of life, or that develop later in life as a result of injury, can still be improved with patience and perseverance (Figure 11-7). As mentioned earlier, bone continually remodels throughout life, in response to the direction and intensity of the loads placed on it. Thus it is possible to improve conformational defects in older foals and even in adult horses. The speed and amount of correction are less than that possible in a young foal, however, so the sooner the defect is addressed with appropriate trimming and shoeing, the better the outcome will be. The best results are obtained when correction is started before the horse reaches skeletal maturity.

Correction of ALDs in older animals should follow the same principles of trimming for balance and judicious use of hoof extensions discussed in the previous section. Hoof extensions can be glued on or welded or forged into the shoe. It is important to make changes gradually, to allow the bone time to remodel. Progress will be slow, and it may take 1 to 2 years of consistent correction to achieve a satisfactory outcome, but the effort is well worth it in the end.

FLEXURAL LIMB DEFORMITIES

Flexural limb deformities are those in which the limb deviates from the normal vertical alignment and normal joint angles, when viewed from the side. In almost all cases, the primary problem involves the soft tissues that support the various joints of the limb. Depending on the defect, and on its severity and chronicity, the problem may simply involve one or more of the flexor muscles (primarily the superficial and deep digital flexors) and the associated ligaments and fascia, or it may also involve ligaments, other fascia, and even the joint capsule and synovial structures of the affected joints.

Flexural limb deformities are divided into two broad categories:

- *Flexor laxity*—in which the supporting tissues are so loose that they allow the joints to hyperextend (e.g., the knee bends backwards, the fetlock sinks, and/or the toe tips up)
- *Flexor contracture*—in which the supporting tissues are so tight that they limit normal extension of the joints, so the horse stands with the affected joints partially flexed

Within each category, the flexural defect can be congenital or acquired.

Before discussing each type of FLD, it is important to note that hyperextension of a joint may, on occasion, be the result of pain involving the joint capsule or the articular surfaces. Young foals with "joint ill" (septic arthritis) have been known to assume this stance, as have those with subsolar abscesses. These conditions develop rapidly, so they should be included in the differential diagnosis list, especially if the hyperextension is sudden in onset.

Flexor Laxity

With flexor laxity, the joints of the lower limb are not sufficiently supported to effectively counteract the hyperextension that occurs simply from the weight of the body through the bony column of the limb. As a result, the knee may bend back slightly ("back at the knee"), the fetlock may drop, and, in severe cases, the pastern and coffin joints may hyperextend to such an extent that the toe tips up and the patient walks on the heel bulbs or even the back of the pastern or fetlock (Figure 11-8).

FIGURE 11-8 Severe flexor laxity in a young foal.

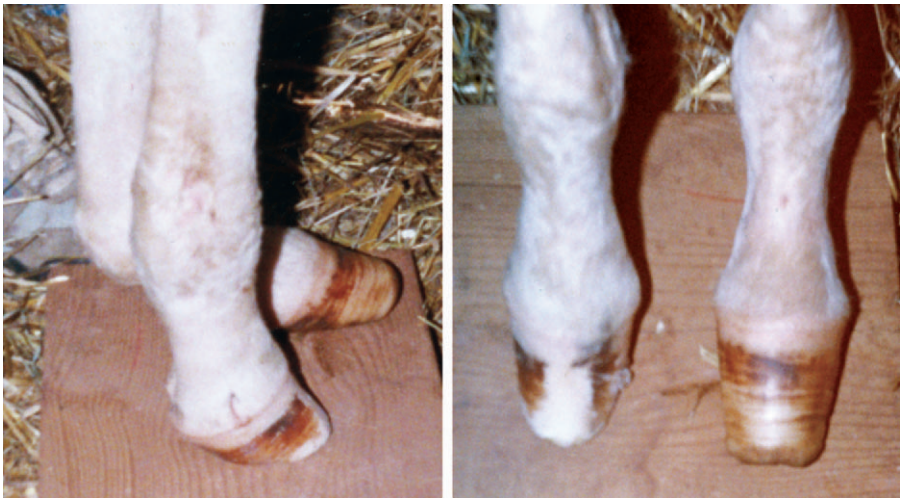

FIGURE 11-9 The foal shown in Figure 11-8, before and after hoof trimming.

Although the name implies that the condition involves a functional defect of the flexor apparatus, it is probably only one of the components of the problem; the suspensory ligaments and extensor tendons may also be involved. Regardless of whether the condition is congenital or acquired, flexor laxity represents a functional imbalance between the long flexors and extensors of the limb (see Chapter 1). As such, it likely has a neuromuscular underpinning, as muscle tone and flexor–extensor balance and synchrony are largely controlled by the nervous system. Thus, activity is an essential component of resolving this abnormality, because the stimulus of normal locomotion encourages normal muscle tone and coordinated function.

Congenital Flexor Laxity

Flexor laxity is quite common in newborn foals. It is particularly common in foals that are premature or dysmature. In these foals, flexor laxity often is combined with incomplete ossification of the cuboidal bones in the knees and/or hocks. The management of these foals is beyond the scope of this chapter, and is discussed elsewhere.[9]

Routine Care. In full-term foals with normal cuboidal bone ossification, flexor laxity typically resolves within a couple of days, just with controlled exercise and provision of good footing, as described for congenital ALDs. It is also important to avoid abrasive surfaces in these foals, as the soft tissues at the heel bulbs and at the back of the pastern and fetlock are easily damaged just by walking around in foals with moderate to severe flexor laxity.

Protective Wraps. If the laxity is so severe that the foal is walking on its heel bulbs or even the back of its pasterns or fetlocks, a light wrap should be used to pad and protect the soft tissues at the back of the digit from abrasion, bruising, and pressure necrosis until the foal is standing and walking normally. A material such as Impact Gel (Impact Gel Corporation, Melrose, Wis.) or the silicone used in prosthetic sleeves makes good padding for this purpose.

It is important, however, not to excessively limit joint mobility in the process. Too much support removes the stimulus for improving flexor muscle tone and thus delays resolution of the laxity. It is also important for the owner or manager to be committed to unwrapping and inspecting the limbs for abrasions, bruising, or pressure necrosis, and then carefully rewrapping them each day until protective wraps are no longer needed.

As the foal's stance improves, more activity can be allowed, and the faster the laxity will resolve. As soon as the foal is strong enough to load the feet in a more normal way, careful hoof trimming to encourage normal ground contact further improves the situation (Figure 11-9). Radiographs of the foot

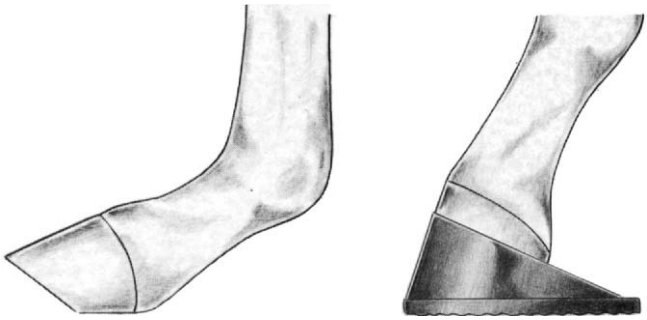

FIGURE 11-10 Heel extensions can be used to encourage normal loading in mild cases of flexor laxity. *(Courtesy R.F. Redden.)*

are very useful for guiding hoof trimming after tendon laxity. These little feet tend to have more sole at the toe than at the heel, and having a radiographic reference allows optimal trimming of the sole and hoof wall once the hoof is in contact with the ground.

Heel Extensions. No special hoof care is required for most young foals with flexor laxity. Mild cases, in which the hoof is in contact with the ground, may benefit from a heel extension (e.g., Dalric D Heel Extension Cuffs [Nanric, Lawrenceburg, Ky.] or a similar home-made device), which is glued on to the hoof to encourage more normal loading of the foot (Figure 11-10).

NOTE: Heel extensions should be used only to treat mild cases of flexor laxity. If heel extensions are used in moderate to severe cases (in which the foal bears weight on the heel bulbs or the pastern/fetlock), tremendous tension is exerted on the hoof capsule by the device, and complete avulsion of the hoof may result.

Acquired Flexor Laxity

Older foals and adult horses often develop mild flexor laxity when a cast or heavy support bandage is used to immobilize the lower limb for more than a couple of weeks at a time. The flexor laxity generally resolves within a week just with controlled exercise and attention to footing, once the cast is finally removed. No special hoof care is required, other than that needed by any foot that has been confined to a cast for weeks.

If the digital hyperextension is so severe that the toe tips up and the horse is bearing the majority of its weight on the heels, then a heel extension can be used temporarily to encourage normal hoof loading while flexor muscle tone normalizes. However, such severe flexor laxity in any horse other than a newborn foal should prompt careful examination for rupture of the deep digital flexor tendon.

Flexor Contracture: General Discussion

With flexor contracture, normal joint extension and thus normal joint angle during stance is prevented by functional shortening or restriction of the supporting soft tissues. Even when the limb is lifted and thus relaxed, it is not possible to manually extend the affected joint to its normal range. The shortening may be dynamic and thus reversible, such as excessive tone in the flexor muscles or further up the chain of muscles in the limb, or it may be static and thus irreversible,

FIGURE 11-11 Carpal contracture in a foal. *(Courtesy R.F. Redden.)*

such as fibrosis (scarring) of a tendon, ligament, fascia, or joint capsule.

Flexor contracture can be congenital or acquired. Specific causes are discussed in the following sections. Regardless of when the contracture manifests, there is almost certainly a neuromuscular component, as discussed earlier for flexor laxity.

Descriptions and Hoof Involvement

Typically, the contracture primarily affects one joint in the limb, so flexor contracture is described according to the principal joint involved: carpal contracture (knee), fetlock contracture, or coffin joint contracture. The pastern joint may also be involved with contracture of the fetlock or the coffin joint. The following stances are typical:

- Carpal contracture—the horse stands with the knee slightly flexed ("over at the knee" or "tied in at the knee"); the foot is placed flat (Figure 11-11)
- Fetlock contracture—the horse stands with a reduced angle at the fetlock (i.e., straight or upright through the fetlock) and often "buckles" or "knuckles" forward at the fetlock; in severe cases, the horse stands with the fetlock permanently knuckled forward (i.e., in a partially flexed position); the foot is placed flat
- Coffin joint contracture—initially, the heels are raised off the ground and the horse bears weight primarily at the toe; unless promptly and aggressively treated, these cases often progress to develop a "club foot" (see p. 218)

With the exception of coffin joint contracture, the hoof is seldom affected to any great degree by the flexor contracture. Manipulation of the hoof can be a component of treatment for fetlock and pastern joint contracture, so the focus of the following discussion is on flexor contracture of the fetlock, pastern, and coffin joints. Management of other types of flexor contracture are discussed elsewhere.[11]

Surgery

A few general comments about surgical management of these conditions are in order. In almost all cases, conservative

management should be given a good try before resorting to surgery. Surgical "release" procedures for the management of flexor contracture are not without risk. Also, one must bear in mind that the surgeon is cutting a structure that is meant to be there; it has a purpose in the overall scheme of musculoskeletal function. Although the body repairs the transected ligament or tendon, damage has been done and the final functional or cosmetic result may be disappointing.

Which surgical procedure to perform and when to perform it is determined by the joint involved, the particular ligaments or tendons primarily involved, and the age of the patient. The best approach is to start with the most conservative surgical procedure; if it does not produce the desired result, a more invasive procedure can then be considered.

A more invasive procedure can be performed during the same surgical event if the initial conservative procedure did not produce the desired result. For example, if cutting a check ligament is going to be effective in correcting the flexor contracture, it will do so immediately, and the surgeon will be able to restore the affected joint to its normal angle then and there (although other measures may be needed to maintain a normal joint angle postoperatively while the limb heals). But if, after cutting the check ligament, the surgeon cannot achieve a normal joint angle, then performing a more invasive procedure should be considered, either during the same surgery or at a later time. When surgery is performed on the standing, sedated horse, the effect of the procedure usually is readily apparent.

The possibility of multiple surgeries and the risks and potential complications of any planned procedures must be thoroughly discussed with the owner beforehand. It is also important that the owner's expectations for the surgery be realistic. It may be that the best possible outcome is a pasture-sound horse or one suitable only for light, noncompetitive activities.

Flexor Contracture of the Fetlock Joint

Congenital Fetlock Contracture

Fetlock contracture is fairly common in newborn foals and in most cases is mild to moderate. These cases usually resolve with controlled exercise and provision of good footing, as discussed for congenital ALDs. Gentle physical therapy (e.g., heat therapy followed by repeated manual flexion and extension of the fetlock) may help, although to be effective it must be performed several times per day. Care must be taken to avoid causing discomfort during physical therapy, as pain induces a protective flexor withdrawal response that is counterproductive. No special hoof care is required in these foals.

Oxytetracycline. Therapy with oxytetracycline (2 to 4 g, diluted in 500 mL of sterile isotonic fluids, and given by slow intravenous infusion) usually is effective in temporarily relieving flexor contracture in young foals. However, it must be combined with controlled exercise and perhaps physical therapy to prevent recurrence of the contracture. In some cases, treatment with oxytetracycline needs to be repeated once or twice to keep the foal on the right track. Moreover, although it can induce relaxation in the affected limbs, oxytetracycline can also cause flexor laxity in the unaffected limbs. This therapy also carries a small but real risk of inducing acute renal failure, so care should be taken to ensure that the foal has normal renal function beforehand.[11]

Unresponsive Cases. Foals that do not respond to these conservative therapies may require the application of a cast, a customized orthotic leg brace, or even a surgical release procedure (usually beginning with desmotomy of one or both of the check ligaments). These treatments are beyond the scope of this chapter.

Acquired Fetlock Contracture

Fetlock contracture can develop at any age after birth, but it more often occurs in horses between 3 months and 2 years of age than in younger foals or adult horses. Although most authors consider this condition to involve just the superficial digital flexor tendon, other structures that either directly or indirectly support the fetlock have often been found to be involved in this syndrome as well.

Diet and Exercise. Genetic predisposition, coupled with excessive carbohydrate intake and inadequate exercise (stall confinement) are primary factors in the development of this condition. In the early stages, a reduction in carbohydrate intake (along with careful attention to mineral intake) and provision of self-regulated exercise (e.g., full-time turnout in a small pasture) may halt the process and return the horse to a normal stance.

Pain Management. Pain somewhere in the limb, whether from osteochondrosis, physitis/epiphysitis, or a nondevelopmental problem (e.g., trauma, infection), can quickly lead to off-loading of the limb and hence fetlock contracture via the flexor withdrawal response. Thus a thorough lameness examination should be part of the diagnostic work-up in every case, as appropriate therapy for any underlying condition is an essential component in managing the resulting fetlock contracture.

Treatment with phenylbutazone or another nonsteroidal anti-inflammatory drug (NSAID) is an important part of the therapeutic plan in most cases of acquired fetlock contracture. However, it is not enough to simply administer NSAIDs to foals with fetlock contracture. A thorough search for any underlying conditions should be conducted before NSAID therapy is commenced.

Damage to one or more of the soft tissue structures that support or comprise the fetlock can result in fibrosis, which may permanently limit the range of joint mobility and perhaps even fix the joint in partial flexion. In such cases, conservative treatment is likely to meet with limited success. Thus, in addition to radiographic examination, ultrasonography is advised if there is a history of trauma to the limb or if the condition is chronic in nature.

Heel Elevation. Elevating the heels with a 10-degree wedged cuff or wedged shoe (Figure 11-12) can, over time, help correct fetlock contracture and avoid the need for surgical intervention. During the period of heel elevation, the horse must be restricted to a stall until the condition is at least 50% improved, as this shoeing system can potentiate other injuries if the horse is allowed unrestricted activity.

Once the condition has improved sufficiently, hand-walking may begin, gradually building up to turnout in a small paddock. Grain is withheld during the period of confinement and restricted activity, and the horse's vitamin and mineral needs should be met in some other way. The degree of heel elevation is slowly decreased once the contracture has substantially improved. When reducing the degree of wedge support, the practitioner should make sure that the fetlock remains in proper alignment following trimming and shoe-

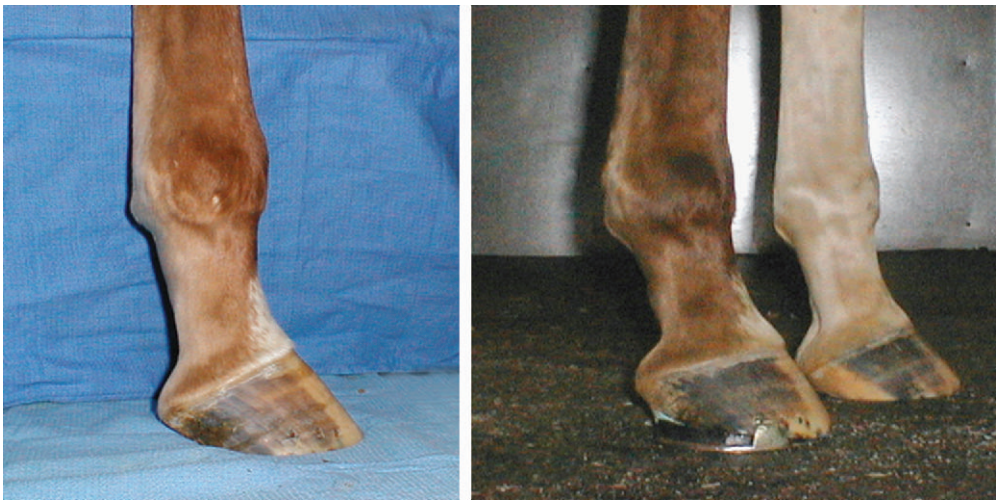

FIGURE 11-12 Elevating the heels with a 10-degree wedged cuff or wedged shoe helps correct acquired fetlock contracture. NOTE: In this horse, the wedge has been lowered to 5 degrees.

ing or replacement of the cuff. If the fetlock angle is abnormal, a return to the original wedge height is in order.

NOTE: Trimming and shoeing a horse with moderate to severe fetlock contracture can be dangerous, as the horse cannot stabilize the fetlock and is prone to collapse onto any unsuspecting handler or farrier. The danger is minimized once one hoof is placed in a 10-degree heel-wedge cuff or shod with that degree of heel elevation. The use of a sling may be required in severe cases.

Patience and Persistence. These two qualities are essential ingredients in the management of acquired fetlock contracture. Regardless of the patient's age at presentation, it may take up to 1 year to fully correct this problem. As long as the horse is younger than 2 years of age when first diagnosed, it is possible to avoid resorting to surgical correction, even with the most severe cases of fetlock contracture. Older horses become surgical candidates if they do not respond to this treatment approach.

Figure 11-13 shows the use of a 10-degree wedge cuff in a 4-year-old horse with fetlock contracture. Despite his age, this horse responded well to wedge support. Cases of severe flexor contracture that are the result of nerve damage are different from the typical case of fetlock contracture discussed here and may require extensive surgery (tenotomies and desmotomies) and arthrodesis of the fetlock. Their management is beyond the scope of this chapter.

Surgery. Many veterinarians opt for surgical release procedures sooner rather than later. Cutting both the superior and inferior check ligaments (i.e., the accessory ligaments of the superficial and deep digital flexor tendons, respectively) can be effective. However, the cosmetic result may be less than satisfactory. More extensive surgical release procedures have been described for cases in which check desmotomies have not been entirely successful. However, the prognosis for future athletic function is greatly diminished when more structures than the check ligaments are transected.

Flexor Contracture of the Pastern Joint

Flexor contracture of the pastern joint may be congenital or acquired. It is often seen in conjunction with flexor contracture of the fetlock joint, but it also occurs as a separate entity (Figure 11-14). In either case, it is readily identified with routine radiography of the digit; the lateromedial view is most informative. Treatment using heel elevation, as described for acquired flexor contracture of the fetlock joint, is recommended.

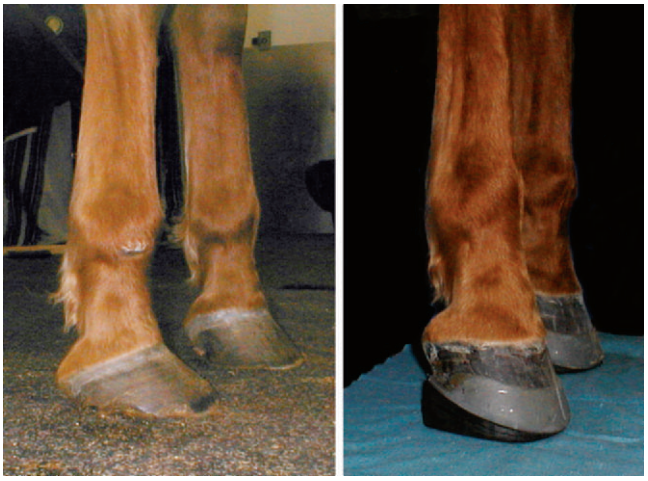

FIGURE 11-13 This 4-year-old horse with fetlock contracture was successfully treated with a 10-degree wedge cuff.

Flexor Contracture of the Coffin Joint: Clubfoot

Flexor contracture of the coffin joint generally is referred to simply as *clubfoot*, although a truly clubbed appearance, in which the hoof develops a boxy shape as the elevated heels grow down to meet the ground, takes time to develop. In the meantime, the hoof may be fairly normal in shape and simply abnormal in orientation and stance (heels raised off the ground, weight-bearing only at the toe, causing loss of hoof wall at the toe). Unless there is concurrent fetlock or carpal contracture, the orientation of the fetlock and knee is normal.

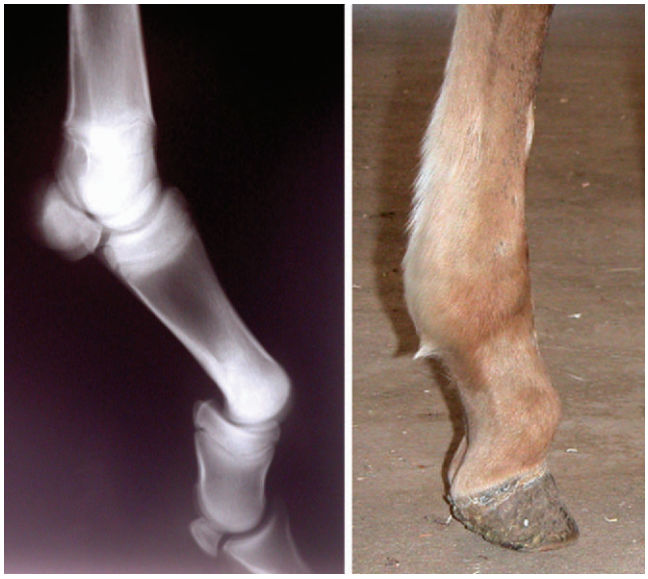

FIGURE 11-14 Acquired flexor contracture of the pastern joint. *(Courtesy Brad Root.)*

Because this condition is so prevalent and of such importance to the function and value of the horse, the following discussion is divided into three separate sections:

- Congenital contracture
- Acquired contracture in young foals
- Club foot in older foals and adult horses

Congenital Coffin Joint Contracture

Coffin joint contracture is relatively uncommon in newborn foals. With this condition, the heels are raised off the ground, although the foot is not truly "clubbed." (NOTE: Newborn foals have a steeper hoof wall angle than older horses; this upright hoof should not be misdiagnosed as a clubfoot. As long as the dorsal hoof wall and the pastern are aligned [i.e., the hoof–pastern axis is normal], and there is no tell-tale dorsal bulge at the coronary band nor any other signs of clubfoot, the foal probably does not have coffin joint contracture.)

Treatment

Initial management of coffin joint contracture in newborn foals is similar to that discussed for congenital fetlock contracture. Unless moderate to severe, the defect usually resolves with only controlled exercise and provision of good footing. Gentle physical therapy and intravenous oxytetracycline may be beneficial in other cases.

It is essential that these foals be monitored closely and the therapeutic approach changed if no improvement is seen within 2 weeks of birth or if the condition recurs or worsens despite treatment. Coffin joint contracture quickly becomes a persistent problem, one that is far better treated early than battled later.

Heel Elevation. Foals that do not respond to conservative treatment within 1 to 2 weeks of birth require more intensive therapy. Elevation of the heels with glue-on wedges (e.g., Dalric Baby wedges; Figure 11-15) can help by temporarily relieving tension in the deep digital flexor tendon. The length of time the heel wedge should be left on the young foot depends on the age of the foal:

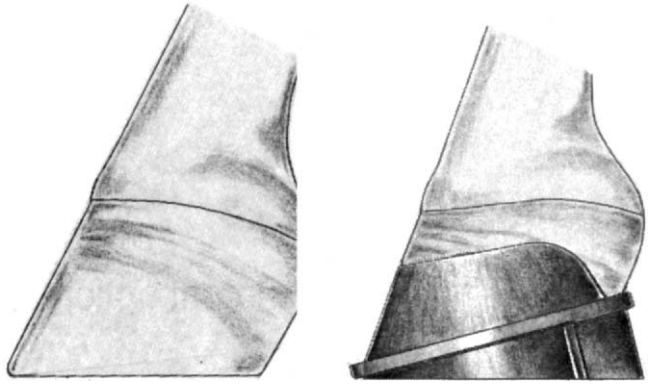

FIGURE 11-15 Use of the Dalric B1 cuff for treatment of coffin joint contracture in a young foal. *(Courtesy R.F. Redden.)*

- Birth to 2 weeks of age—remove after 4 days and re-evaluate in 7 days
- 2 to 4 weeks of age—remove after 10 days and re-evaluate in 7 to 10 days
- 4 to 8 weeks of age—remove after 10 to 14 days and re-evaluate in 7 to 10 days
- 8 to 12 weeks of age—remove after 14 days and re-evaluate in 7 to 10 days
- 3 to 8 months of age—remove after 21 days and re-evaluate in 7 to 10 days

If, after the wedge and cuff is removed and the hoof is trimmed, the foal is not able to comfortably load the properly balanced hoof, then the wedge should be immediately reapplied and left on for the maximum length of time appropriate for the age of the foal before again being re-evaluated. If, on the other hand, the foal can comfortably load the trimmed foot normally, then the wedge and cuff can be left off and the foal re-evaluated in 7 to 10 days, depending on its age.

In the majority of cases, the foal will respond to this treatment, although older foals (those 3 to 8 months of age) may need to wear a wedged shoe or cuff for as long as a year before being able to do without it. By following this approach, surgery can usually be avoided.

Surgery. Foals that fail to respond to this approach may require a surgical release procedure. The first procedure to consider in this situation is desmotomy of the inferior check ligament (the accessory ligament of the deep digital flexor tendon). In severe cases, deep flexor tenotomy is needed.

Acquired Coffin Joint Contracture in Young Foals

Flexor contracture of the coffin joint can develop or worsen at any age, but it is most likely to occur or progress while the foal is actively growing. In the veterinary literature, the period between 3 and 6 months of age apparently is a time of particular vulnerability, the propensity can be established much earlier, often within the first few weeks of life, but it is simply not recognized by the caregivers.

Causes and Contributing Factors

Of all the possible causes and contributing factors, the three most common are as follows:

- Overfeeding of carbohydrates (particularly grain-based feeds and lush pasture) in the nursing mare or the weanling foal
- A source of pain somewhere in the limb
- Neuromuscular dysfunction

In some cases, there appears to be a genetic component as well, with several foals sired by the same stallion or mare developing a clubfoot as they mature, often all involving the same limb (frequently the right forelimb). Generally speaking, there is some evidence to suggest that right forelimb contracture may be hereditary, bilateral contracture may be nutritional, and any single-limb involvement may be neurogenically stimulated through improper agonist/antagonist muscle activity or as a response to pain somewhere in the limb.

Pain from any cause can quickly result in flexor contracture, because it induces a protective flexor withdrawal response, in which the limb is partially off-loaded to spare the painful area. So, careful evaluation of the foal's diet and management and thorough examination for a source of pain somewhere in the affected limb are essential starting points in the management of this problem.

Early Recognition and Evaluation

Acquired coffin joint contracture often begins within a few days of birth; however, this early stage generally is missed by all but the astute owner/manager or veterinarian. The condition usually affects the forelimbs, but it can also occur in the hindlimbs, where it is even more commonly overlooked. Owners should be advised to inspect all four of the foal's feet on a regular basis, with the foal standing on a flat, clean surface. Tell-tale signs of trouble include raised heels that do not touch the ground, a bulge just above the coronary band at the dorsum of the foot, excessive wear of the hoof wall at the toe when the cleaned sole is inspected, and, over time, a dish or concavity in the dorsal face of the hoof wall.

The condition can manifest suddenly and progress very rapidly, or it can deteriorate very slowly, if at all, and be merely a mild deformity that is discernible only to the trained farrier's or veterinarian's eye. Because it can be difficult to predict in advance which way an individual case will go, it is always best to err on the side of caution and treat every case of acquired coffin joint contracture in a foal as a potentially serious condition.

In the process, however, one should be very careful not to mistake a foal with different size feet for one with a clubfoot. Some horses naturally seem to have one flat foot and one normal foot, and it is likely that this conformational asymmetry has a hereditary basis. In comparison to the flat foot, the normal foot may appear clubbed. However, use of a hoof gauge, in addition to lateral radiographs of the digit, helps distinguish this as-yet poorly defined condition from a true clubfoot, which has a hoof angle greater than 60 degrees. (Pony hoof gauges are ideal for measuring hoof angles in foals. These small gauges are available from most farriery supply outlets.)

Treatment

Initial treatment includes the use of NSAID analgesics (small doses, twice daily) for the first week, along with appropriate treatment for any underlying conditions found during evaluation.

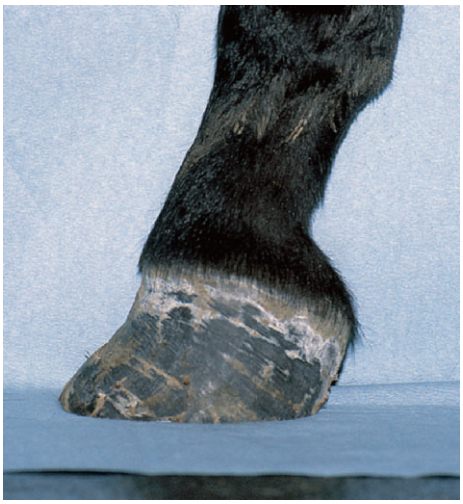

FIGURE 11-16 Grade III clubfoot. Note that the growth rings in the hoof wall are much wider at the heel than at the toe, and the dorsal hoof wall is dished. *(Courtesy R.F. Redden.)*

Heel Elevation. In foals younger than 8 months of age, a Dalric Baby wedged cuff can be glued on, after proper trimming of the hoof (Figure 11-16). Before applying the cuff, the heels should be lowered to approximate a normal hoof angle of 50 to 55 degrees. The length of time the cuff should be left on the immature foot depends on the age of the foal (see guidelines on p. 218).

After the cuff is removed and, if necessary, the hoof is trimmed to once again restore a normal angle (depending on how long the cuff was on the foot), the foal is observed for several minutes before anything more is done. If the heels contact the ground and remain that way after several minutes, then the cuff is left off and the owner is instructed to monitor the foal closely for evidence that the contracture is recurring. (The first signs typically are raising of the heels and loss of hoof wall at the toe.)

If, however, the heels do not touch the ground after the wedge is removed, the wedge should be reapplied. In most cases, one or two applications of the wedged cuff halts the contracture and allows the growth of a normal hoof.

Surgery. In the few instances in which the clubfoot persists in contracting despite treatment (probably due to a neurogenic response), the only reasonable therapeutic option is deep flexor tenotomy. The veterinary literature advises desmotomy of the superior or inferior check ligaments as an early intervention for coffin joint contracture in foals. In some cases of low-grade, slowly developing club foot, this procedure has some value, especially in foals with no heel as found in wet climates. However, immediate application of the Dalric Baby wedge cuff reliably halts the progress of the condition in foals with mild contracture, making surgery unnecessary.

In contrast, for clubfeet that have gone undetected until chronic or in cases in which the contracture is advancing uncontrollably, deep flexor tenotomy is the best course of action. In either situation (chronic or rapidly worsening clubfoot), desmotomy of the check ligaments will not halt the disease process nor allow restoration of a normal coffin joint

angle. Provided that deep flexor tenotomy is accompanied by appropriate postoperative wound care, rehabilitation as the tendon heals, and regular hoof trimming to restore and maintain normal digital alignment, and as long as the foal does not have any other limiting problems, the prognosis for future athletic function after tenotomy is good.

Clubfoot in Older Foals and Adult Horses

Clubfoot that has become chronic can be a very difficult problem to manage. Early recognition, proper diagnostic evaluation, appropriate treatment, and diligent ongoing care and monitoring can prevent, or at least minimize the impact of, chronic contracture in the growing horse. However, when the problem is not addressed until the horse is mature, the propensity for this deformity may remain despite proper treatment and frustrate the very best efforts of the farrier and the veterinarian.

Based on several cases of chronic coffin joint contracture in adult horses that attempted to return to their contracted state even after deep flexor tenotomy and repeated digital realignment, it is possible that the neuromuscular component will confound all attempts to restore the horse to normality, even if the horse responds to appropriate treatment of the digital deformity. It is therefore of critical importance that the contracture be recognized and treated appropriately as soon as the propensity reveals itself, and hopefully before a permanent neuromuscular pattern of contracture is established.

Fortunately, in each of the cases described, the horses were no longer lame after tenotomy and digital realignment and could be used for some level of activity. The palmar angle (i.e., the angle of the palmar margin of P3 relative to the ground surface) achieved with digital realignment in each case was only a few degrees greater than normal, which may have been a key factor in achieving an acceptable functional outcome.

Grading the Clubfoot

Specific treatment for clubfoot depends on the severity and chronicity of the deformity. There are a few different methods for grading the clubfoot. One system grades the deformity based on the physical and radiographic appearance of the foot, using a scale from I (mild) to IV (severe):

Grade I: The hoof angle is 3 to 5 degrees greater than that of the opposite (normal) foot. There is a characteristic fullness just above the coronary band at the dorsum of the foot, caused by subluxation of the coffin joint. Growth rings usually are present in the hoof wall, but unlike with the more severe grades, the rings are the same distance apart at the heels as at the toe.

Grade II: The hoof angle is 5 to 8 degrees greater than that of the opposite foot, and the growth rings in the hoof wall are wider at the heel than at the toe. The heels do not touch the ground when properly trimmed to a normal length.

Grade III: The grade III clubfoot takes the abnormalities of the grade II foot even further, and as a result develops a dish or slight concavity in the dorsal hoof wall (Figure 11-17). In many cases, the growth rings are twice as wide at the heel as they are at the toe. Radiographically, there is demineralization and evidence of microfractures or lipping along the dorsodistal margin (apex) of P3.

FIGURE 11-17 Aplasia of the second phalanx.

Grade IV: The hoof angle is greater than 80 degrees, the coronary band is higher at the heel than at the toe, and the dorsal hoof wall is heavily dished. The sole may be flat or even convex below the apex of P3. Radiographically, there is severe loss of mineral at the apex of P3, so much so that the normally sharp margin may appear rounded. In addition, there is divergence in the width of the dorsal horn-lamellar zone from proximal and distal, similar to that seen in the laminitic hoof (see Chapter 16). The palmar angle of P3 may be as much as 30 degrees. In extreme cases, the horse may be walking on the dorsal surface of the hoof wall.

Treatment Principles

The principles of treatment are the same, regardless of the severity of the deformity. The goal is to achieve normal digital angles (i.e., normal alignment between the first, second, and third phalanges [P1–P2–P3]) and thus normal orientation and loading of P3 relative to the ground. The means of achieving this goal range from simply trimming and shoeing to surgery (deep flexor tenotomy), depending on the severity and chronicity of the defect.

Treating Grade I or II Clubfeet

The goals of treatment can be achieved in horses with grade I or II clubfeet simply with careful trimming and shoeing. Several shoeing options are available for these cases, including "air ride" or "air rail" shoes, or full rocker shoes. The aims of trimming and shoeing are to correct the palmar angle; relieve excessive tension on the deep digital flexor tendon, distal sesamoidean ligaments, and navicular bursa; and restore digital alignment (i.e., P1–P2–P3 orientation).

Grade I. In horses with grade I clubfoot, the specific aim is a palmar angle of 0 to +2 degrees (i.e., a neutral or slightly positive palmar angle) and an unbroken line along the dorsal faces of P1, P2, and P3. This modest change in palmar angle can usually be achieved with a full rocker shoe or a Dan four-point shoe.

Grade II. The main difference between a grade I and a grade II clubfoot is the greater palmar angle in the grade II foot. In horses with a grade II clubfoot, particularly one that may be bordering on being a grade III, it may be necessary to rasp the bearing surface of the foot to create two separate planes, with the breakpoint between the two planes being located at the widest part of the foot (i.e., at the quarters). Rasping the heels back to the widest part of the frog produces a palmar angle of 0 to 2 degrees (confirmed radiographically) and creates two separate flat planes in the hoof, with the main bearing surface being in front of the quarters.

Rasping the bearing surface into two planes makes it easier to achieve the goal of a neutral or slightly positive palmar angle in grade II clubfeet. By restoring more normal loading of the hoof capsule, blood flow is improved to all areas of the hoof; as a result, more normal sole and hoof wall growth can be achieved over successive shoeing cycles.

When two planes are made on the bearing surface of the foot, an air ride shoe is an appropriate choice. (An air ride shoe basically is any shoe that is tooled to conform to the bearing surface of the foot that has been trimmed into two planes and that does not have a heel wedge.) An air rail shoe (i.e., any shoe with a wedged heel, such as the Redden four-point rail shoe [Nanric, Lawrenceburg, Ky.]) should be applied if the horse is not comfortable in an air ride shoe (i.e., is unable to make ground contact with the heels on the trimmed, unshod hoof) or other type of shoe that does not have a wedged heel.

A simpler alternative is to use a flat shoe and a wedge pad (or pads) until the horse is comfortable. Taping the heel wedges to the foot prior to shoeing lets the veterinarian and farrier know how many wedge pads it will take to make that particular horse comfortable. NOTE: Wedging the heels in this manner excessively loads the heels, thereby constricting the bulbar blood supply and weakening the heels. Thus it should be used only as a short-term measure.

Surgery. Surgery is seldom required in horses with grade I or II club feet. Inferior check desmotomy may be considered in the growing horse with a recalcitrant grade I or II club foot, if the horse cannot be made comfortable with the shoeing methods discussed.

Treating Grade III Clubfeet

Rarely can the goals of treatment be achieved in grade III clubfeet through trimming and shoeing alone. These horses generally benefit from a surgical release procedure, in addition to the digital realignment described for grade I and II club feet. If inferior check desmotomy is not immediately successful in allowing a normal coffin joint angle and P3 orientation, then deep flexor tenotomy should be considered.

It is worth noting again that grade III clubfeet have pathologic changes at the apex of P3 that are irreversible. These changes are a direct result of constriction of the circumflex and dorsal circulation by the abnormal orientation of P3. There may also be irreversible tearing of the dorsal laminae, which will result in widening of the white line similar to that seen in the laminitic hoof. Nevertheless, restoring a more normal palmar angle and digital alignment (through surgery, trimming, and shoeing) improves blood flow throughout the hoof, thereby halting the disease process and allowing whatever healing that can occur to take place.

Treating Grade IV Clubfeet

Horses with such a severe and long-standing deformity as a grade IV clubfoot require deep flexor tenotomy, in conjunction with appropriate trimming and shoeing to restore a more normal palmar angle and digital alignment. Even so, the prognosis for future athletic usefulness is guarded (but not unrealistic). By this stage, the apex of P3 is extensively damaged, and the hoof capsule is grossly distorted. It takes a lot of time, patience, and meticulous care to restore these feet to some degree of functional normality.

Heel Support after Tenotomy. When deep flexor tenotomy and digital realignment are performed, it is important not to allow the trimmed heels to contact the ground immediately after surgical release, especially in young horses. Instead, the trimmed heels must be supported in a raised position with a large wedge of sole putty (e.g., Advanced Cushion Support, Equilox, or Grand Circuit Hoof Putty [Nanric, Lawrenceburg, Ky.]) so as to approximate the original height of the heels in the contracted state. The heels are then gradually lowered over the following 1 to 2 weeks by removing a little of the support material each day.

There are two reasons for this precaution. First, the tenotomy generally is performed on the standing, sedated horse, using regional nerve blocks that, in addition to desensitizing the surgical site, also desensitize the hoof. If the horse is permitted to fully bear weight on the trimmed hoof before sensation returns, the supporting soft tissues (which have habituated to the flexor contracture) may suddenly be overloaded and damaged. The distal sesamoidean impar ligament (which anchors the distal margin of the navicular bone to the palmar surface of P3) may be particularly vulnerable. There have been reports of impar ligament rupture when horses with grade III or IV club foot have been allowed to fully bear weight on the trimmed, unsupported foot after deep flexor tenotomy.

Second, tenotomy and digital realignment represent major changes to these grossly deformed limbs, and most patients go through a period of adaptation that lasts 1 to 2 weeks, sometimes longer. Use of the putty as a heel wedge that approximates the original height of the heel allows a small amount of the material to be trimmed away each day, thus gradually lowering the heels, while observing the horse for an increase in lameness. Should the lameness worsen, more putty can be applied to raise the heels back up. After a few days, the process of gradually lowering the heels can begin again.

Investigating Persistent Discomfort. The purpose of any procedure in these cases is to make the horse as comfortable as possible. The underlying reason for aiming to realign P1–P2–P3 and restore a more normal palmar angle is to re-establish blood supply to the compromised areas and more normal loading of the foot. If the horse remains uncomfortable for several weeks after tenotomy and appropriate digital realignment, then a thorough search for other problems should be undertaken.

The following conditions may be found in horses with grade IV club feet and predispose to discomfort: articular damage in the coffin joint, stretching or tearing of the ligaments that support the coffin joint or navicular bone, seroma formation within the soft tissue of the hoof as a result of P3 displacement (which puts pressure on these tissues), and microfractures or bone chips of P3 or the navicular bone. Routine radiography (both soft tissue and bone exposures),

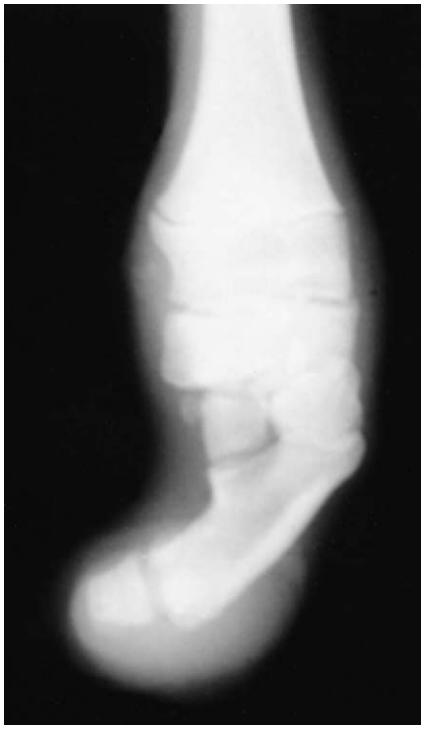

FIGURE 11-18 Hypoplasia of the third metacarpal bone (cannon bone) and aplasia of the digit (all three phalanges). *(Courtesy R. Weiss.)*

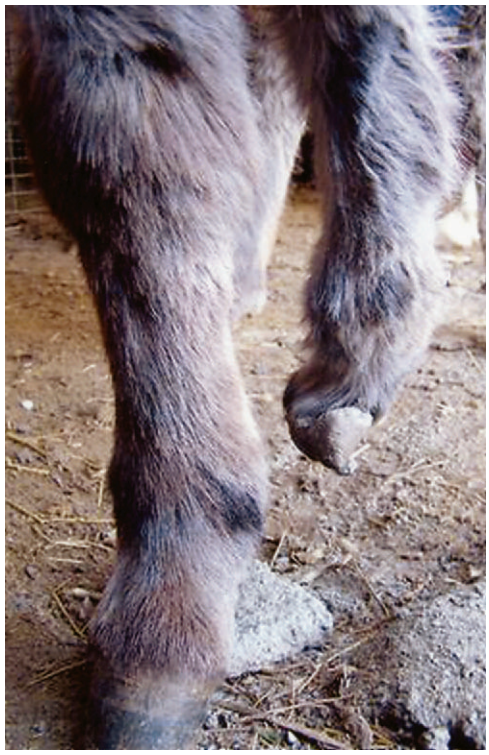

FIGURE 11-19 Photograph of the foal whose radiograph is shown in Figure 11-18. Note the deformed hoof capsule at the end of the aplastic digit. *(Courtesy R. Weiss.)*

venography, and ultrasonography are invaluable in evaluating these patients. These diagnostic tools are discussed in Chapter 10.

HYPOPLASIA OR APLASIA OF THE EQUINE DIGIT

Hypoplasia is incomplete development of a structure, whereas aplasia is complete absence of a structure (Figures 11-18 and 11-19). Hypoplasia and aplasia of part or all of the digit are rare in horses, although there are numerous reports of these conditions in the veterinary literature, perhaps because they are so unusual. Except in the case of hypoplasia or other developmental defects of the navicular bone, these conditions usually are apparent at birth, as they typically are accompanied by visible deformity of the digit. Developmental abnormalities of the navicular bone may not be found unless the foot is radiographed for some reason.

When any foal is born with a misshapen digit that does not fit with the common deformities discussed in this chapter, radiographs should be taken, as it is likely that the foal suffers from hypoplasia or aplasia of one or more of the phalanges (P1, P2, or P3). If the foal is of sufficient economic or sentimental value, attempts can be made to render the limb as functionally useful as possible (Figure 11-20).

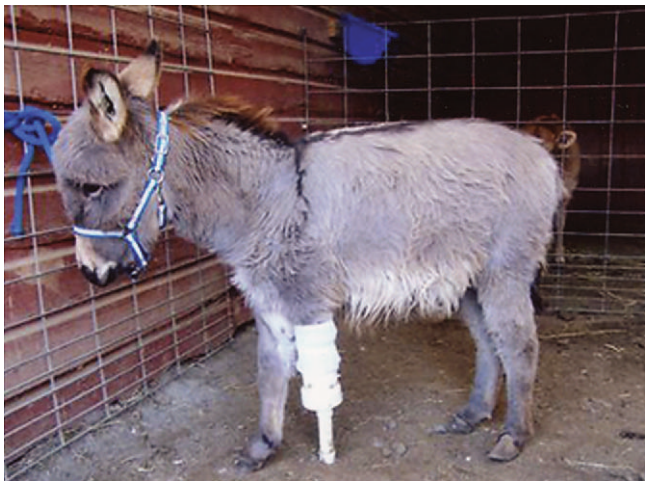

FIGURE 11-20 Prosthetic management of the limb shown in Figures 11-18 and 11-19. *(Courtesy R. Weiss.)*

MISMATCHED FEET

There is a syndrome in which the foal (or horse) has different size feet, specifically one flat foot and one normal foot. This conformational asymmetry may have a hereditary basis, as the propensity often is apparent in young foals. This syndrome should not be confused with clubfoot, as the more upright foot actually is normal (Figure 11-21). Routine radiography (in particular, the lateromedial view) of the foot clears up any confusion, as these feet have a normal palmar angle and normal digital (i.e. P1–P2–P3) alignment.

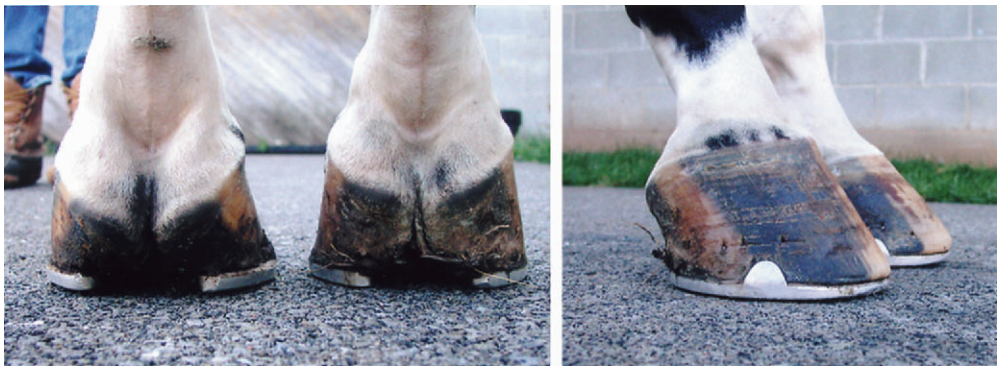

FIGURE 11-21 This horse has mismatched feet, not a club foot. *(Courtesy R.F. Redden.)*

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12

PATHOLOGIC CONDITIONS OF THE EXTERNAL HOOF CAPSULE

LARRY BOOTH and DANIEL WHITE

Injuries to the foot generally appear as lacerations or rope burns across the back of the pastern. Injuries may also appear as an avulsion of a segment of hoof wall or coronary band from entrapment of the foot in a fence (most notably barbed wire) or under a panel, gate, or other sharp metal object. Lacerations to the bottom of the hoof occur from stepping on a sharp object. Wounds to the palmar or plantar aspect of the pastern often transect the digital artery and vein, with varying degrees of blood loss.

INITIAL TREATMENT

Clients should be instructed to apply a clean bandage with pressure to stop any bleeding. The horse should be evaluated for hypotension and hypovolemia as a result of excessive blood loss before any sedation or general anesthesia is administered. Regardless of the cause, the initial approach to treatment consists of the same basic principles of wound management applicable to any lower limb wound in the horse. The biggest problem with wounds to the foot is the almost unavoidable environmental contamination with bedding, dirt, debris, and fecal material. The long-term prognosis for soundness is always a concern, but initially it may be difficult to predict the outcome. Many horses go through a long and protracted healing and rehabilitation period in which they may remain lame for months but yet recover completely. In a retrospective study, 22 of 30 horses with various types of hoof injuries returned to soundness with an average healing time of 3.3 months.[1] In this study, the time delay to definitive treatment was more important to a successful outcome than the extent of the injury. The goals of treatment are to reduce the bacterial contamination of the wound and then surgically reconstruct the area as soon as possible to minimize scarring and preserve function.

LACERATIONS TO THE PASTERN AND HEEL BULB REGION

Heel bulb lacerations deserve special consideration because of their frequent occurrence. Deep wounds that gape when the horse bears weight should not be left to heal on their own because exuberant granulation tissue and delayed healing is the inevitable result (Figure 12-1). These wounds often extend in an arc through the coronary band and into the hoof capsule either at the buttress of the heel or at the quarter or both. The digital cushion is invariably involved to some degree. Deep lacerations to the heel very often transect the digital artery, vein, and nerve. This is of little concern if the wound is unilateral since there is adequate collateral circulation and painful neuromas are very uncommon. The collateral cartilage is often damaged or exposed, but chronic infection leading to quittor is rare if adequate debridement is accomplished. If damage is done to both digital arteries, then the entire hoof capsule may slough as a result of ischemic necrosis of the foot (Figure 12-2).

Wound Preparation

Preparing the hoof capsule for surgery requires a lot of work. The evaluation and preparation of the hoof can be accomplished with the horse standing if the horse is cooperative; if not, the work can be done more efficiently with the horse under general anesthesia. Radiographs should be obtained if the wound appears to involve the bone or deep structures. Broad-spectrum systemic antibiotics are indicated for most deep wounds that may involve the bone, tendon, ligaments, or synovial structures. Tetanus prophylaxis should be routine.

Initially, the wound should be protected by packing with sterile saline-soaked gauze to prevent contamination from the trimming, clipping, and cleaning process. The hoof should be thoroughly trimmed and the hoof wall lightly rasped to allow adequate cleaning and aseptic preparation. Initial cleaning can be accomplished with a stiff brush and lots of soap and water. The hair should be clipped the entire circumference of the limb from the coronary band proximal to the fetlock. A combination of sterile saline gauze packing and wetting the hair or applying water-soluble jelly (Surgilube; E. Fougera & Co, Melville, NY) to the hair along the wound edges will help prevent hair clippings from contaminating the wound. After clipping, the gauze packing is changed, and the entire clipped area should be aseptically prepared in the event it is necessary to perform regional limb perfusion or ingress/egress lavage of the coffin joint or digital sheath. Regardless of whether the goal is simply debridement of a heavily contaminated wound or definitive surgical repair, the surgeon should use sterile technique throughout the procedure.

If vital structures such as the distal phalanx, navicular bone, digital sheath, deep flexor tendon, or distal interphalangeal joint appear to be involved, or if the laceration is deep with considerable contamination, regional limb perfusion should be considered to prevent infection. This procedure is well described[2] and has been shown to provide very high local concentrations of antibiotics to the tissues. An intravenous digital perfusion technique is the easiest to perform. A pneumatic or Esmarch (Medvet International, Libertyville, Ill.) tourniquet should be placed above the fetlock prior to draping of the distal limb and foot. A 20-gauge butterfly catheter or 1-inch over-the-needle catheter attached to a heparinized extension set is inserted into the digital vein at the level of the

FIGURE 12-1 **A,** This heel bulb laceration developed exuberant granulation tissue as a result of inadequate immobilization. **B,** Hypertrophic scarring in a heel bulb laceration.

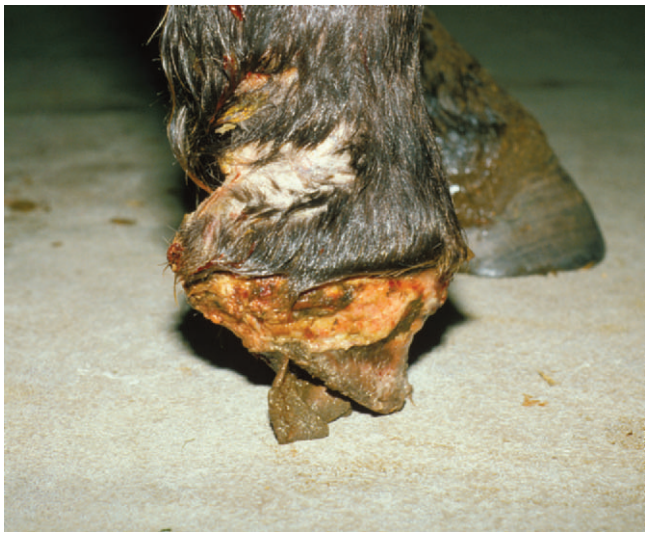


FIGURE 12-2 Trauma to the blood supply to the foot resulted in ischemic necrosis of the hoof capsule.

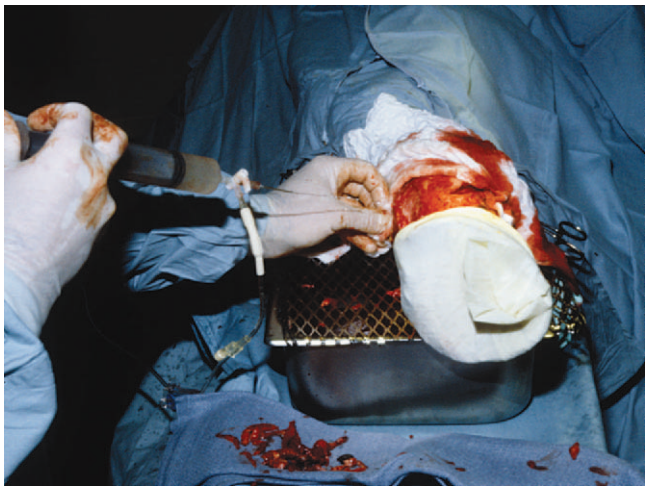


FIGURE 12-3 A 35-mL syringe and an 18-gauge needle can be used to lavage a wound with at least 7 psi of pressure.

proximal sesamoid bones. The digit is then perfused by slow injection of 100 to 200 mg of gentamycin, 125 mg of amikacin, or 1 g of ceftazidime diluted in 60 mL of balanced electrolyte solution over 1 to 2 minutes with the tourniquet inflated or in place. The tourniquet should remain in place for at least 20 to 30 minutes. The perfusion technique should be performed early on in the treatment process so that high levels of antibiotics are in the tissues at the time of wound debridement.

Surgical Management

Once the distal limb is aseptically prepared and draped, involvement of the digital sheath or the coffin joint can be confirmed by synoviocentesis and gross observation of the fluid for cloudiness or by injecting a volume of saline or lactated Ringer's solution into each synovial cavity at a distant location and observing the wound for egress of fluid. If either structure is involved, a large 12-gauge needle or arthroscopic cannula should be used to establish an ingress flush line and a continuous egress flow of lactated Ringer's solution should be established from the wound site. The joint or tendon sheath should be flushed with approximately 4 to 6 L of lactated Ringer's solution. At the same time that the joint or tendon sheath is being lavaged, wound exploration and sharp surgical debridement of all contaminated and devitalized tissue is performed. The constant egress of fluid during the surgical debridement phase prevents contamination of the deeper structures and aids in removing debris and bacteria from the joint or tendon sheath. Copious wound lavage is also performed using a 35-mL syringe and an 18-gauge needle or a high-quality spray bottle that will deliver the fluid at a pressure of at least 7 psi to effectively dislodge bacteria and debris (Figure 12-3). The most common lavage fluids used are 0.1% gentamycin in saline or 0.05% chlorhexidine in distilled water.

If there was minimal or no synovial cavity contamination, and all contaminated and devitalized tissues have been debrided, then primary suture closure should be the goal. Several large, tension-reducing sutures using No. 2 polypropylene in a vertical mattress or far-far-near-near pattern are preplaced but not tied. These tension-reducing sutures serve

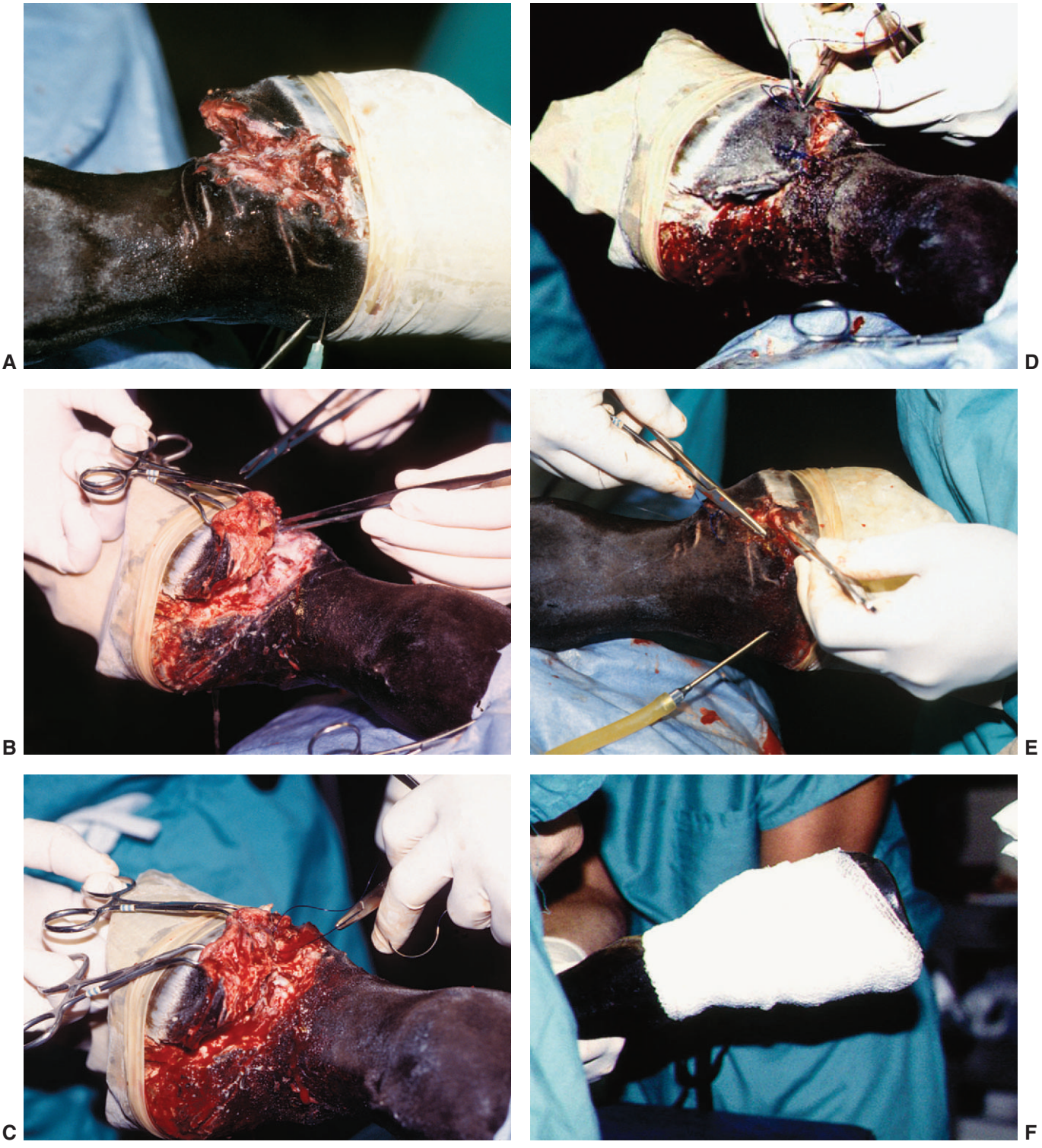


FIGURE 12-4 **A,** This heel bulb laceration has been prepared for sterile surgery. Injection of saline through an 18-gauge needle placed in the front of the joint exited the wound, indicating an open joint. A 12-gauge needle has been inserted into the joint to establish an egress lavage. **B,** The wound is being meticulously debrided with a scalpel while joint lavage is being conducted. **C,** After debridement and lavage, the joint capsule and deeper tissues are sutured. **D** and **E,** The coronary band is carefully reconstructed with 2-0 polypropylene. **F,** The foot is bandaged with a sterile dressing before application of a foot cast.

to protect the repair and stabilize the wound edges. Wound closure is completed with 2-0 or 0 monofilament nylon in a simple interrupted or vertical mattress pattern, and the tension sutures are then tied snuggly enough to just reduce the tension on the primary suture line. Even if criteria are not met for closing the wound, the placement of several large, tension-reducing sutures, tied snuggly enough to stabilize the wound edges, will help provide a cosmetic and functional outcome when combined with a foot cast, as described under Postoperative Management (Figure 12-4).

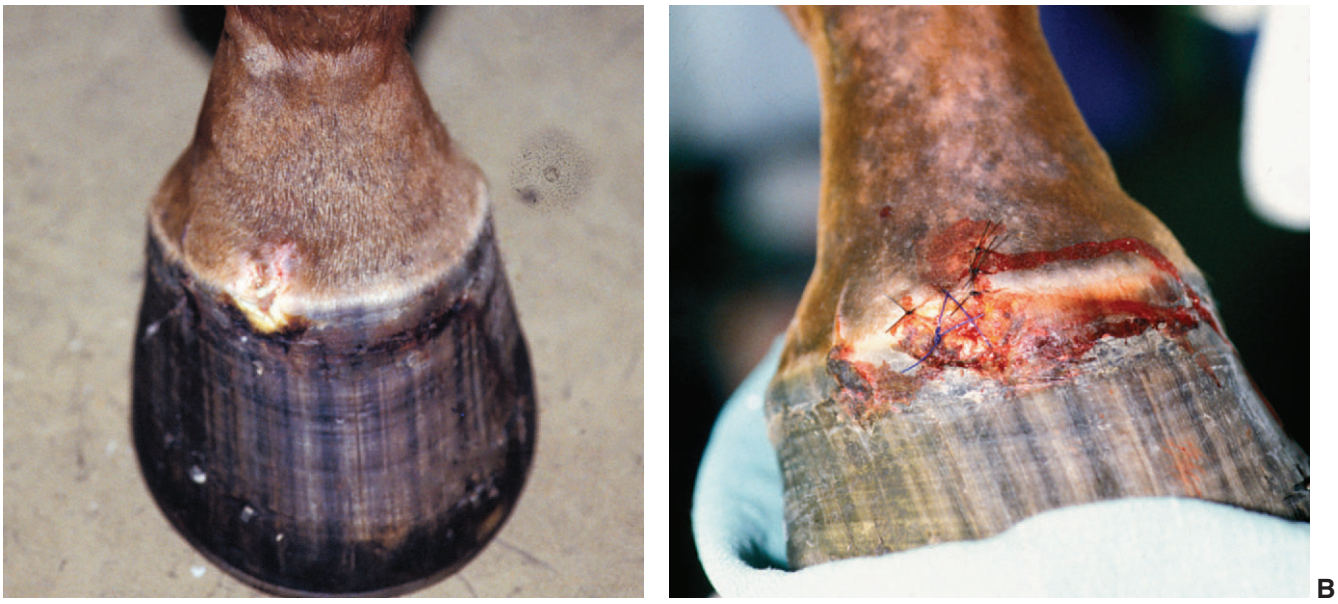


FIGURE 12-5 **A,** Coronary band defect as a result of a wound left to heal by second intention. **B,** The wound has been reconstructed to better oppose the coronary band and lessen the resulting defect.

FIGURE 12-6 **A,** This yearling suffered a heel bulb laceration that extended through the coronary band and into the hoof in the quarter region. The wound has been repaired by suturing the hoof and coronary band. The foot was then immobilized in a cast. **B,** After 2 weeks in a cast, there is minimal scarring.

If the wound extends through the coronary band, every attempt should be made to reconstruct it as accurately as possible to prevent a hoof defect. Reconstruction is accomplished by placing approximating skin sutures as close as possible to the coronary band (Figure 12-5). Alternatively, placing sutures directly into the coronary band to achieve accurate reconstruction does not seem to be a problem.[3] The hoof tissues in the heel region are usually soft enough to allow suture approximation in the reconstruction process (Figure 12-6). In the quarter region, a small thin flap of hoof is often torn loose at the coronet, but the hoof is thin enough to drive a needle through it to allow suture approximation.

Postoperative Management

After surgical reconstruction, the wound is bandaged with a porous, nonadhesive dressing (Adaptic; Johnson & Johnson, New Brunswick, NJ) and several layers of 4 × 4-inch gauze held in place with elastic gauze. A fiberglass foot cast that extends proximally to up to the fetlock is then applied (Figure 12-7). Immobilization in a cast is extremely important to protect the surgical repair.[4,5] The cast should be changed in 2 weeks or sooner, as dictated by the horse's use of the limb. Usually 2 weeks is sufficient time for adequate healing of smaller wounds. More extensive wounds require a second cast and an additional week or two of cast immobilization to prevent

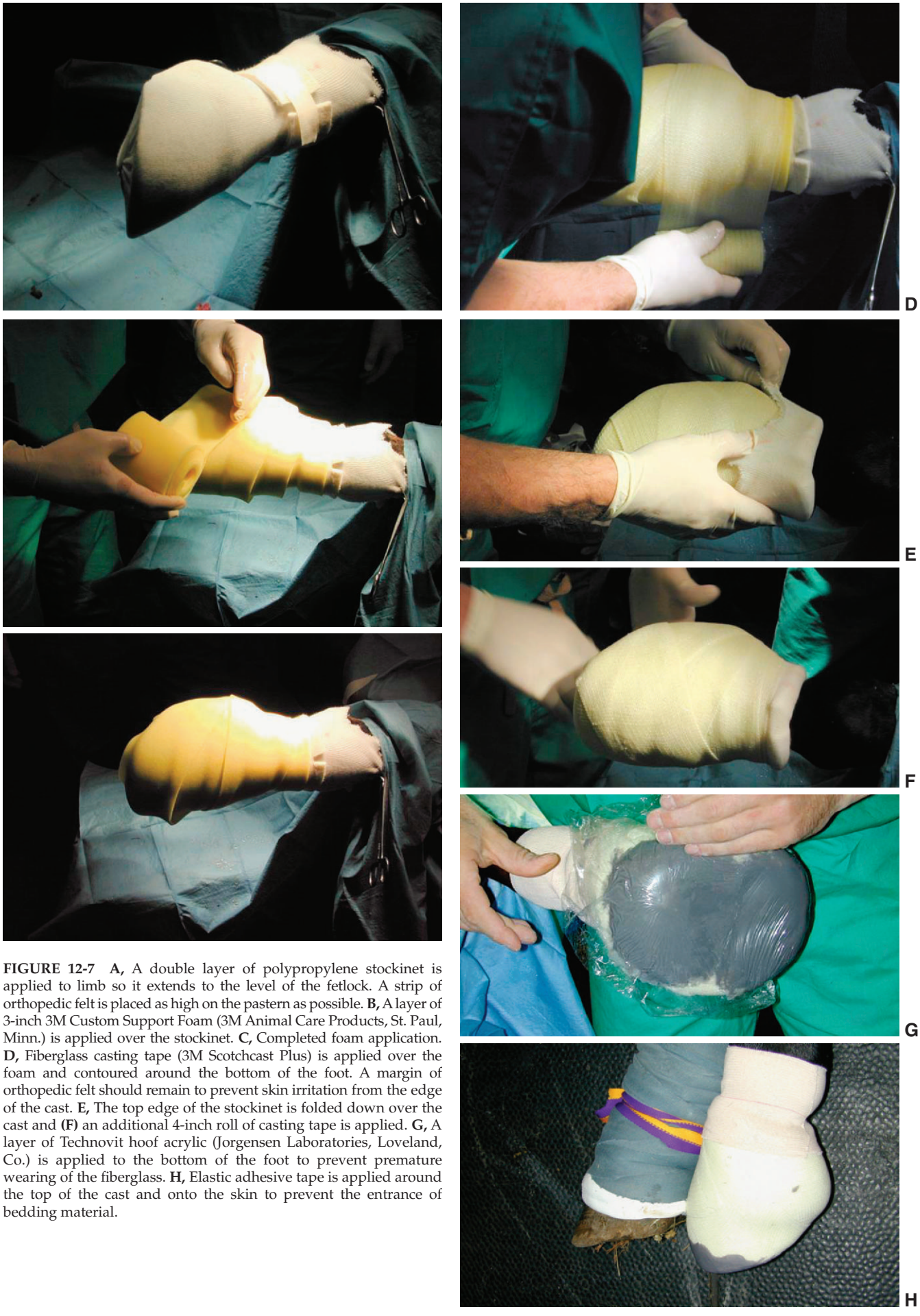


FIGURE 12-7 **A,** A double layer of polypropylene stockinet is applied to limb so it extends to the level of the fetlock. A strip of orthopedic felt is placed as high on the pastern as possible. **B,** A layer of 3-inch 3M Custom Support Foam (3M Animal Care Products, St. Paul, Minn.) is applied over the stockinet. **C,** Completed foam application. **D,** Fiberglass casting tape (3M Scotchcast Plus) is applied over the foam and contoured around the bottom of the foot. A margin of orthopedic felt should remain to prevent skin irritation from the edge of the cast. **E,** The top edge of the stockinet is folded down over the cast and **(F)** an additional 4-inch roll of casting tape is applied. **G,** A layer of Technovit hoof acrylic (Jorgensen Laboratories, Loveland, Co.) is applied to the bottom of the foot to prevent premature wearing of the fiberglass. **H,** Elastic adhesive tape is applied around the top of the cast and onto the skin to prevent the entrance of bedding material.

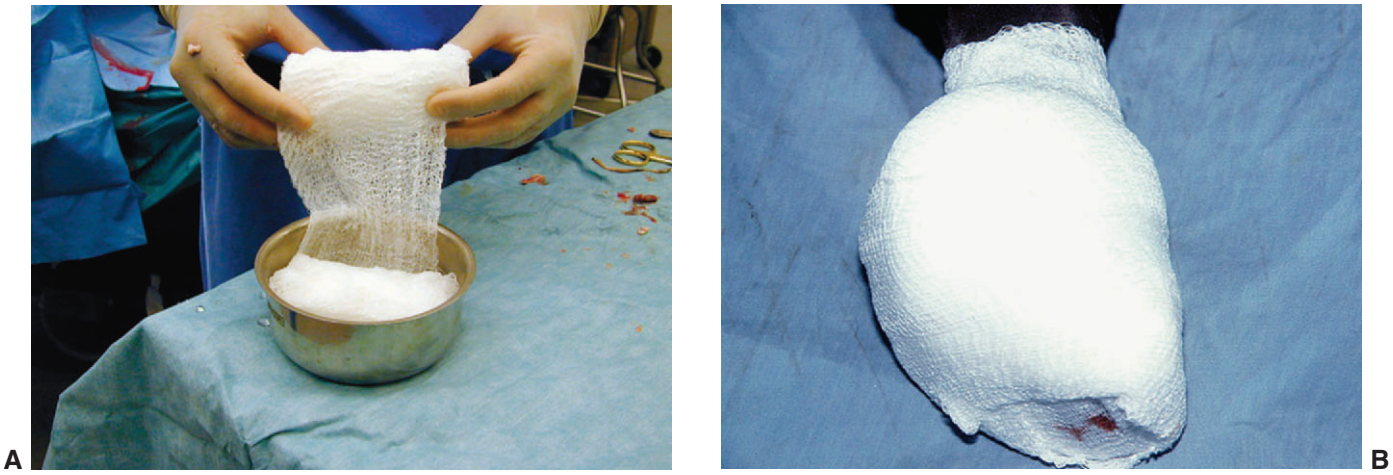


FIGURE 12-8 **A,** A portion of a Kerlix (Kendall Company, Mansfield, Mass.) gauze roll is soaked in saline solution; the excess solution is then squeezed out so the gauze is damp. **B,** The hoof defect is packed with the damp portion of the gauze and the remainder is used to finish bandaging the foot.

dehiscence. Once the cast is removed, the foot should be protected with a bandage for an additional 2 weeks or until the wound is completely healed.

If the wound is grossly contaminated, there is extensive tissue trauma that cannot be completely debrided, or there is established tendon sheath or joint infection, then the best approach is to pack the wound using a wet-to-dry dressing and then bandage the entire foot. The bandage should be changed daily under sterile conditions until the wound has a healthy bed of granulation tissue and there is no sign of infection. Wet-to-dry dressings are an excellent method of wound debridement. A thick, wide mesh gauze (Kerlix; The Kendall Co, Mansfield, Mass.) works best. The gauze comes in its own sterile plastic pouch. The pouch is opened, the gauze roll is removed, and a portion of the gauze is unrolled and dipped into saline solution added to the pouch or a separate sterile bowl (Figure 12-8). The excess fluid is squeezed out so the gauze is damp and not soaking wet. The dampened portion of the gauze is packed into the wound and the remaining roll of dry gauze is used to wrap the foot and secure the dressing in place. The foot bandage is completed with elastic adhesive bandage (Triple pads; Professional Medical Products, Greenwood, SC), and a layer of duct tape is used to provide a waterproof barrier (Figure 12-9). The fluid in the gauze dilutes any viscous exudates and wicks it into the outer layers of the bandage. After 24 hours, the dried gauze is removed along with the resorbed exudates and attached necrotic debris. The process is repeated daily as necessary until a healthy granulation tissue appears in the wound.

Additional joint flushes or regional limb perfusions can be performed as needed on a daily basis on the standing, sedated horse if it is cooperative. However, the usual protocol is to reanesthetize the horse in 3 days and re-evaluate the wound. Further debridement, joint lavage, and limb perfusion is performed if necessary. If the wound appears healthy at that time, then it can be closed as previously described. This delayed closure can be performed at any subsequent point in the healing process when the tissues are deemed healthy. If granulation tissue has started to fill the wound, it should be removed or debulked to facilitate accurate closure of the wound margins. Removing the granulation tissue also removes the bacteria that often persist on the surface. Older heel bulb lacerations with exuberant granulation tissue already present are managed similarly by surgically debulking the granulation tissue, approximating the skin edges with sutures if indicated, as previously described, and applying a foot cast for at least 2 to 3 weeks (see Figure 12-7).

Alternative Management

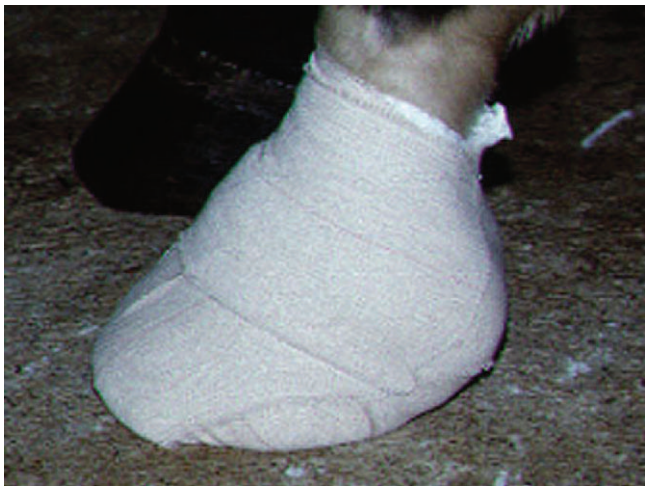
Even working under severe economic constraints, there are still basic things that can be done to benefit the healing process. All but the smallest wounds below the fetlock will be benefit greatly from immobilization in a cast. Primary closure is not always necessary to achieve a functional and cosmetic outcome. This is especially true of heel bulb lacerations. Wound debridement can often be accomplished in the standing sedated horse. Wet-to-dry dressings and a bandage can be used for several days to obtain a relatively healthy wound bed, and finally, a foot cast can also be applied with the horse standing. It is important to remember that a cast is not a definitive form of wound closure, and therefore there is no danger that a cast will somehow prevent adequate wound drainage or trap bacteria in a wound. It is, however, one of the most important things the veterinarian can do to a foot wound to help prevent exuberant granulation tissue (Figures 12-10 and 12-11).

Prognosis

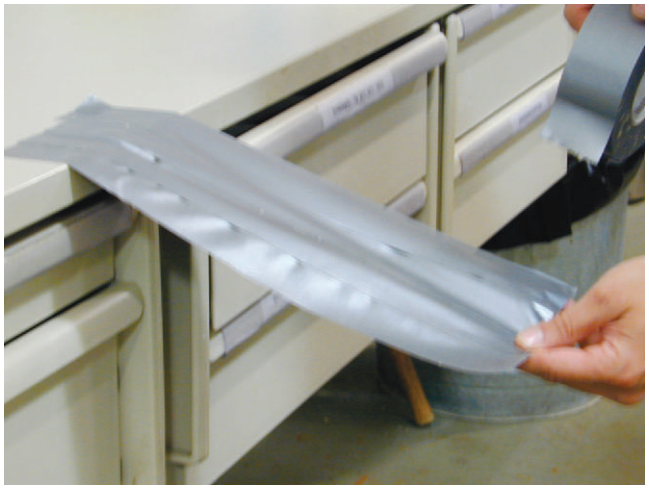
For most lacerations that do not involve vital structures, the prognosis is good for both cosmetic and functional outcomes. Deep lacerations that involve the deep flexor tendon, the tendon sheath, the distal interphalangeal joint, or the podotrochlear apparatus are more problematic and warrant a guarded prognosis for return to performance. Often the progress in these horses is measured in months.[1,3,6]

ROPE INJURIES TO THE PASTERN

When the horse becomes entangled in a rope, the rope generally hooks around the pastern and then as the horse struggles, the rope saws through the skin, producing signifi-



A



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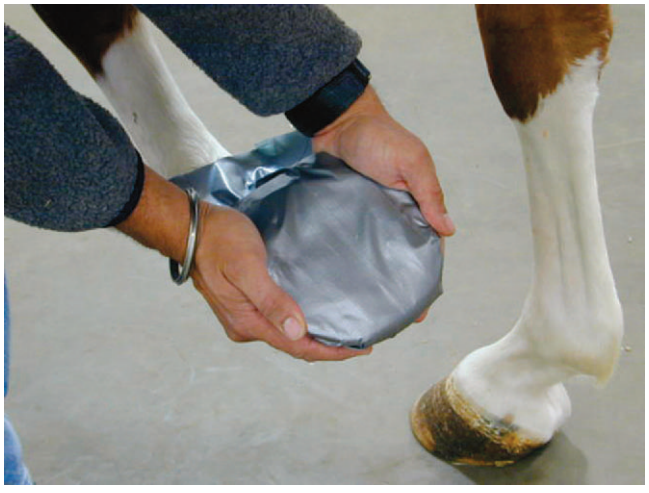
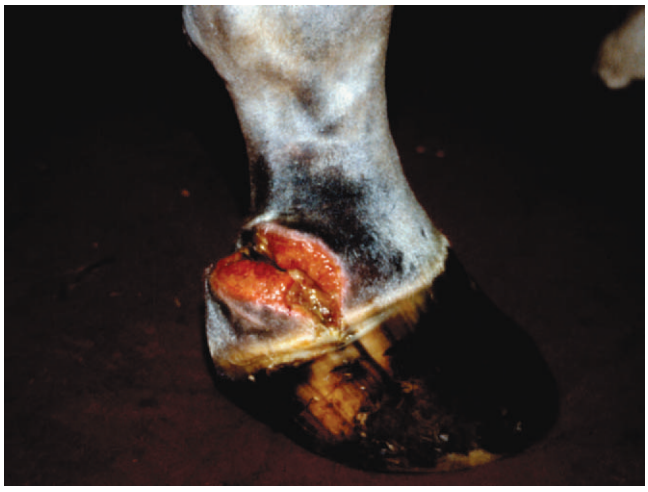


C

FIGURE 12-9 **A,** Elastic adhesive bandage material is used to secure the gauze roll to the foot. **B,** Multiple strips of duct tape are used to create a solid sheet. **C,** The solid sheet of duct tape is applied to the bottom of the foot to provide a waterproof barrier.



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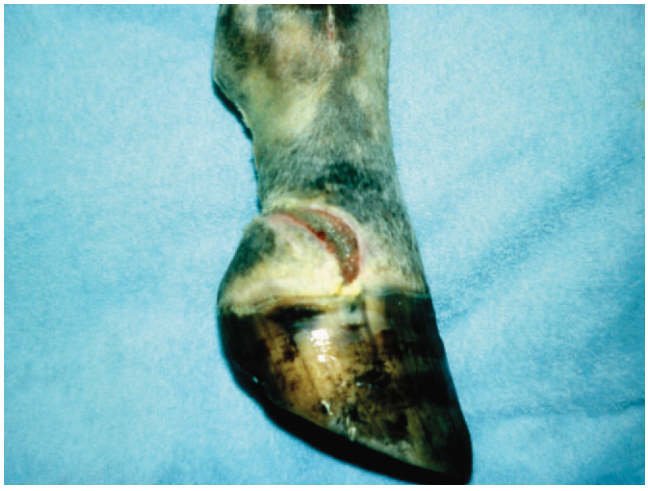


B

C

FIGURE 12-10 **A,** Typical pattern of healing of a heel bulb laceration that is managed by bandaging alone. Note the expanding wound margins as a result of exuberant granulation tissue. **B,** The result of 2 weeks of immobilization in a cast. No other treatment was done. The result would have been even better if the exuberant granulation tissue had been excised and the wound edges were approximated with sutures. **C,** Appearance of the wound 1 week after cast removal. The wound was immobilized in a cast for a total of 3 weeks.

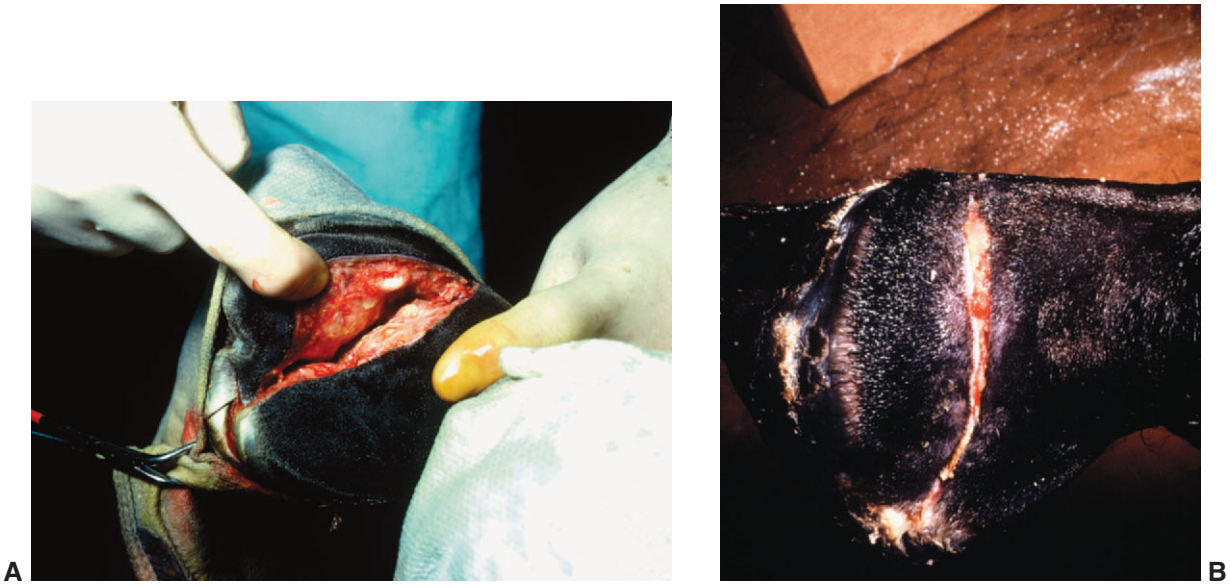


FIGURE 12-11 **A,** This heel bulb laceration went very deep. It was not sutured at all but simply placed in a cast. **B,** Appearance of the wound after 2 weeks in a cast. Note the absence of exuberant granulation tissue and excellent healing.

cant tissue trauma. Because of the destructive nature of the injury, primary surgical repair is contraindicated, because subsequent tissue death from ischemia is common and difficult to detect on initial presentation. One of the reasons these wounds heal slowly is that small strands of the hemp material in the rope often become embedded in the tissues, causing a foreign-body reaction and significantly delaying the healing process. Another reason for slow healing is debridement of partially damaged skin, the blood supply of which has been compromised, often takes a long time, similar to burned skin. Therefore it is very important to carefully surgically debride these wounds of all dead and traumatized tissue. Vigorous scrubbing with a surgical scrub brush will help dislodge any remaining rope debris. Repeated surgical debridement at 2- to 3-day intervals may be necessary until all dead tissue is removed. The foot should be kept in a bandage. Because the wounds are left to heal by second intention, wet-to-dry dressing can also help with the debridement phase. Once a healthy bed of granulation tissue is established, a foot cast is applied to limit movement, protect the wound, and speed up the contraction and epithelialization process. The cast is changed at 2-week intervals as needed (Figure 12-12).

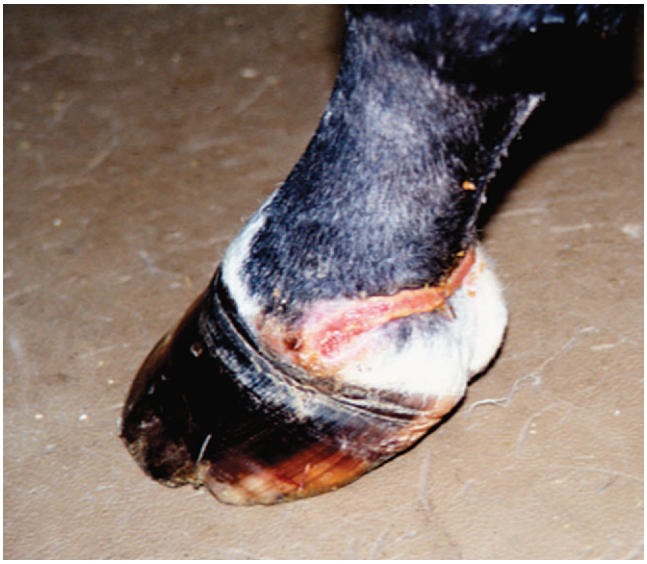


FIGURE 12-12 This full-thickness rope burn was immobilized in a cast for 2 weeks. Exuberant granulation tissue is minimal.

ABRASIONS OR AVULSIONS OF THE CORONET

The coronary band or coronet consists of the coronary corium and coronary papillae with an overlying germinal layer of the coronary epidermis, which is responsible for generating the tubular and intertubular horn of the stratum medium as well as the primary and secondary epidermal laminae of the hoof wall. The stratum medium is the thickest of the three main layers of the hoof, the others being the stratum externum and the stratum internum. Every effort should be made to preserve or reconstruct the coronary band; if the architecture is distorted or the papillae are replaced with scar tissue, then a permanent defect or abnormal horn development is likely to occur.

Abrasions are classified as partial-thickness or superficial wounds resulting from a shearing injury to coronary epithelium.[7] A common cause of a foot abrasion is being stepped on by the opposite foot. Overreaching will also cause this type of injury. Most mild abrasions rapidly re-epithelialize and heal without consequence. This type of wound occasionally disrupts horn production temporarily, leading to a horizontal hoof defect as it grows down (Figure 12-13). In the case of an abrasion or wound that causes a loss of the coronary band cells, there is little to do but allow for secondary-intention

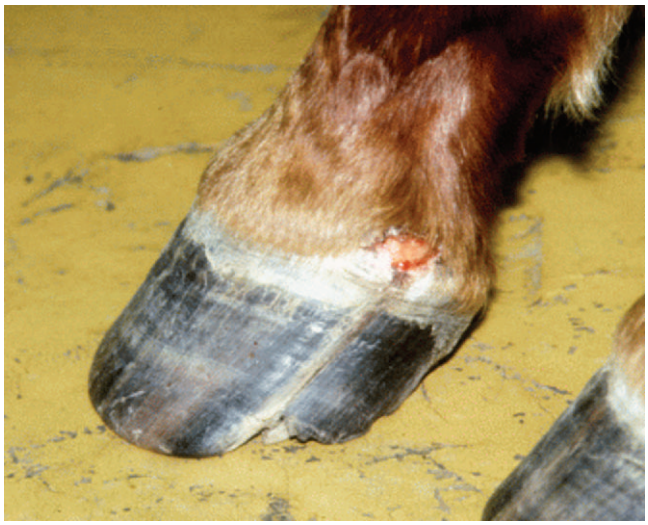


FIGURE 12-13 A chronic wound and damage to the coronary band has produced a hoof defect in this horse.

healing. Often the horse will be left with a scar that produces a chronic hoof defect.

Avulsion injuries that involve the coronary band usually occur by trapping or snagging of the foot on a fence or gate. In these cases, every effort should be made to surgically replace the avulsed segment of coronary band tissue back into its original wound bed. Reconstruction should be attempted even if the wound is several days or several weeks old. The flap of tissue should be sutured down and a foot cast should be applied for at least 2 weeks to provide protection (Figure 12-14).

In those cases in which the avulsion injury involves the skin of the pastern, the coronary band, and the hoof wall (Figure 12-15), it is not uncommon for germinal epithelium derived from the corium to migrate beyond the old boundaries of the coronary band up onto the pastern (Figure 12-16). The germinal epithelium of the hoof will produce keratinized tissue, whereas the skin above the coronary band will produce new epithelialized scar tissue.

AVULSIONS OF THE HOOF

Incomplete Avulsion of the Heel

Horses that step on a sharp object, such as a farming disc, may have a whole segment of hoof and soft tissue torn away

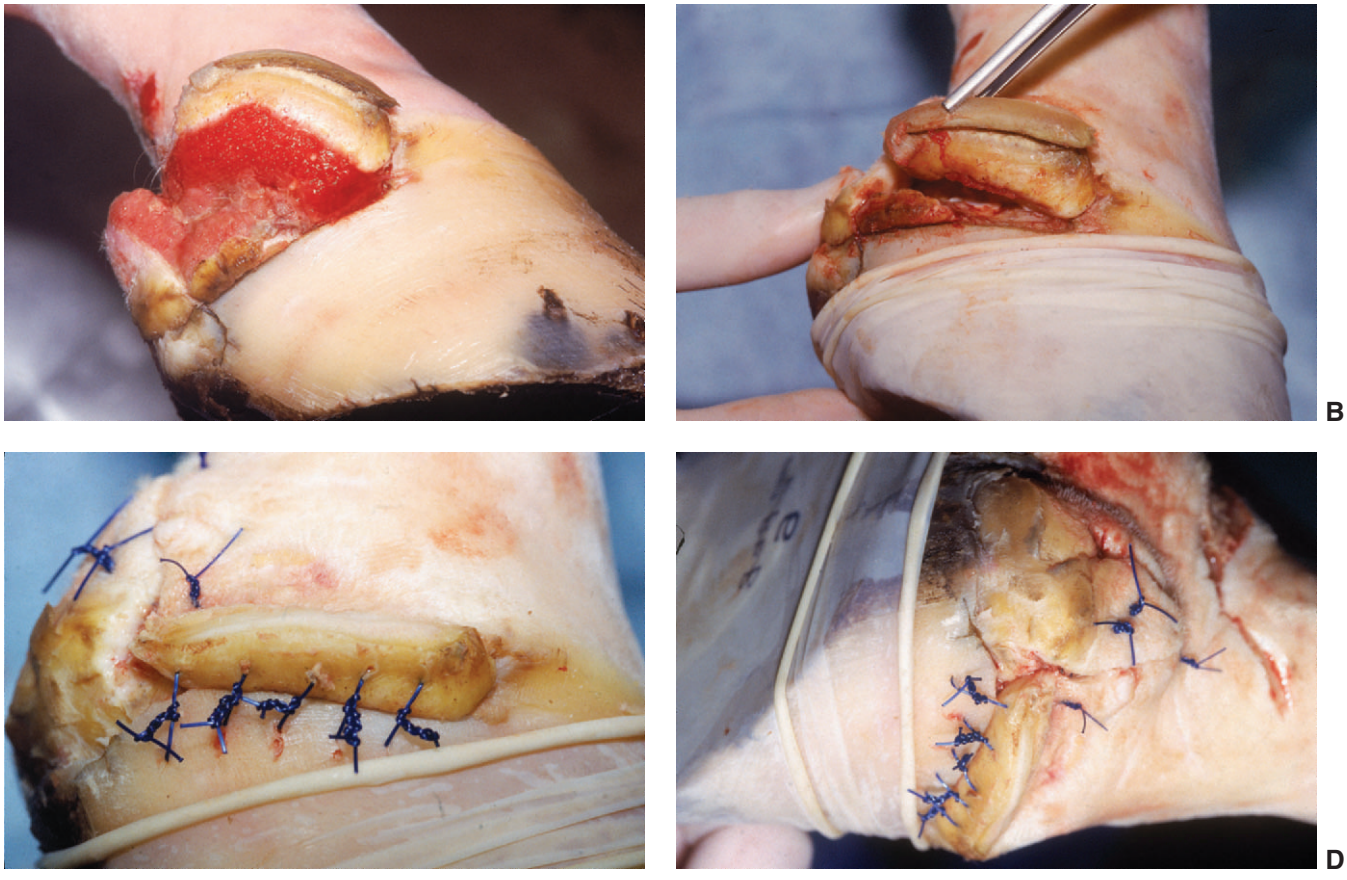


FIGURE 12-14 **A,** This horse had an avulsion injury to the coronary band that was neglected and allowed to heal by second intention. **B,** All exuberant granulation tissue has been removed using a tourniquet to aid visualization. **C,** The coronary band has been reconstructed by placing sutures through the hoof wall. **D,** Appearance of the final repair before casting.

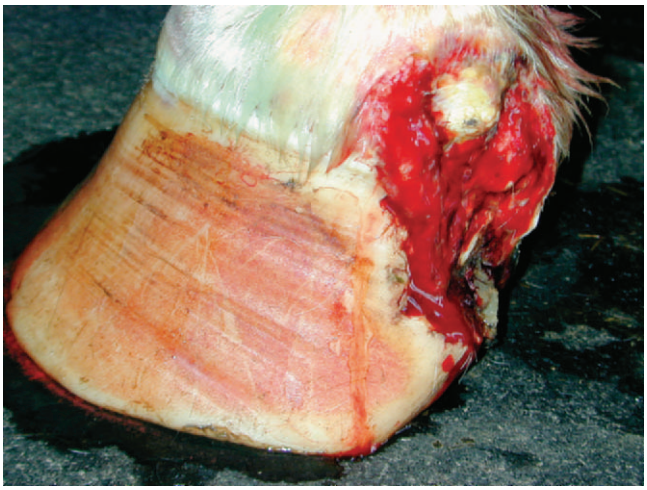


FIGURE 12-15 This avulsion injury involves the skin, coronary band, and hoof wall.

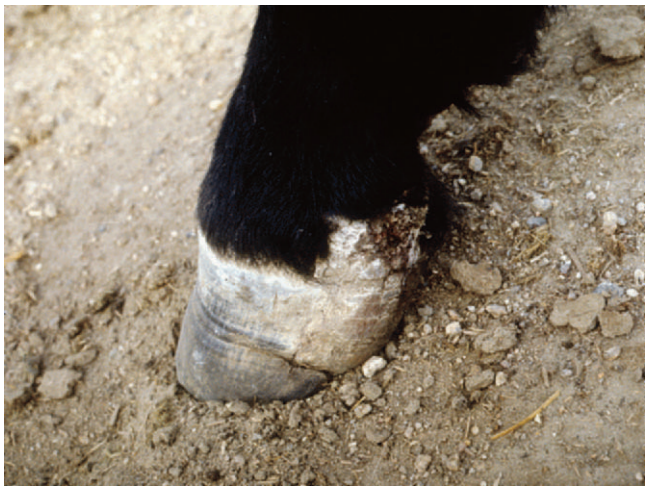


FIGURE 12-16 Keratinized tissue has extended above the coronary band in this healed avulsion injury as a result of migrating germinal epithelium derived from the corium of the hoof.

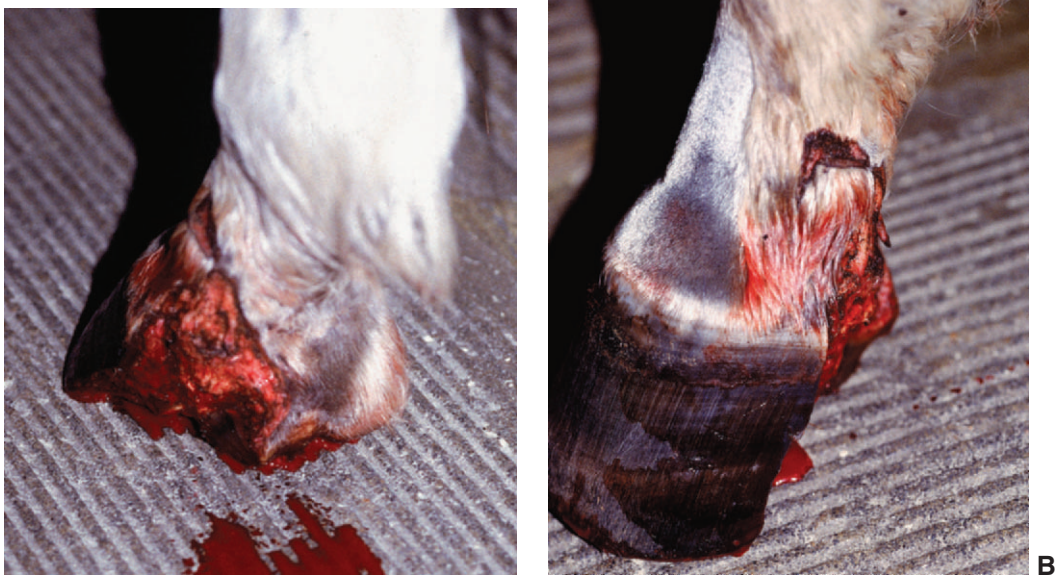


FIGURE 12-17 **A,** Posterior view of a complete avulsion of the hoof wall and heel. **B,** Anterior oblique view of a complete avulsion of the hoof wall and heel.

(Figure 12-17) or an incomplete avulsion of the heel forming a large flap still attached at the coronary band (Figure 12-18). In the case in which the coronary band and underlying hoof wall corium are still attached and viable, the entire hoof segment can be reconstructed by thoroughly cleaning and debriding the wound, replacing the flap of hoof, and anchoring it in place with wire sutures, similar to repairing a hoof crack. Additional stabilization is provided by a foot cast. After cast immobilization for approximately 3 to 4 weeks, an egg-bar shoe with clips can be used to protect the foot until healing is complete. Alternatively, the hoof wall can be stabilized by use of a wrap-around hoof patch (Figure 12-19). A complete foot bandage should be used to protect the wound from contamination. The wound will heal by secondary intention, and over time the hoof crack will grow out.

Incomplete Avulsion of the Hoof Wall

If a segment of hoof has been traumatically stripped away from the underlying dermal lamina, or if the wall is undermined or unstable proximal to the level of the coronet, then the best course is to completely remove the loose segment (Figure 12-20). The procedure can be performed with the horse under general anesthesia in combination with an abaxial nerve block or with the horse standing, using an abaxial nerve block and sedation as needed. The application of an Esmarch tourniquet at the level of the fetlock is helpful in reducing hemorrhage. The foot should be cleaned and prepared for aseptic surgery as previously described. If necessary, the hoof wall is cut with a rotary burr the remainder of the distance from the white line to the coronary band, with special care taken to

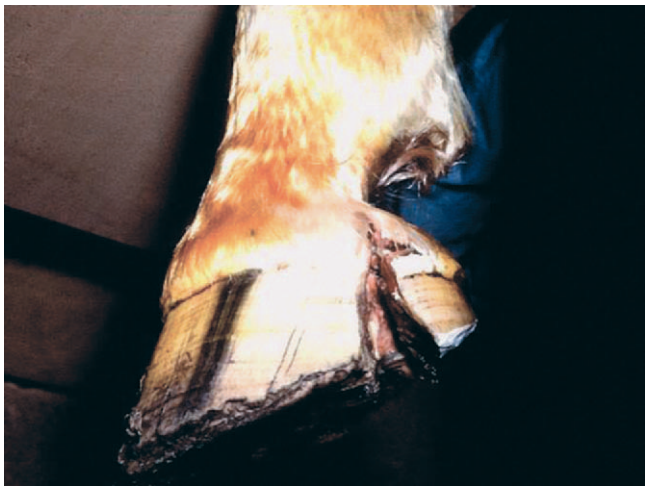


FIGURE 12-18 Incomplete avulsion of the heel caused by stepping on a sharp object. The hoof is still attached at the coronary band and therefore can be reconstructed.

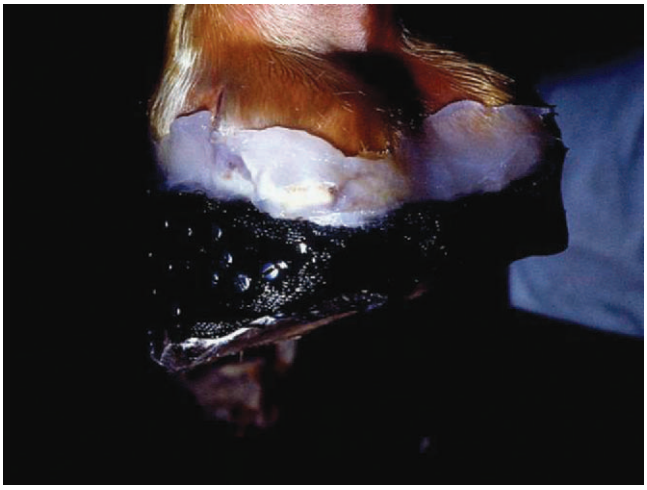


FIGURE 12-19 The hoof flap in Figure 12-18 has been stabilized with a fiberglass hoof patch that is anchored to normal hoof wall and wraps around the heels and is anchored to the opposite normal heel. Healing was uneventful.

extend the cut only to the depth of the nonpigmented stratum internum, avoiding the underlying vascular dermis if possible. The distal margin of the hoof wall is loosened from the sole by creating another cut through the white line. Hoof pullers or vise-grip pliers are then used to grasp the loosened hoof wall and peel it upward to the coronet, gently teasing it free from the papillae of the coronary band (Figure 12-21). A porous, nonadhesive dressing (Adaptic; Johnson & Johnson) is applied to the wound, and the foot is bandaged with a heavy cotton bandage or cotton cellulose pad (Triple pads; Professional Medical Products, Greenwood, SC) and elastic adhesive tape (Elastikon; Johnson & Johnson) snug enough to stop the bleeding before removal of the tourniquet. The bandage is changed after 24 hours and a nonirritating triple antibiotic ointment is applied under a semiocclusive nonadherent pad (Release; Johnson & Johnson) and a protective bandage. The bandage is changed initially at 3- to 5-day intervals and less frequently as healing progresses until keratinization of the

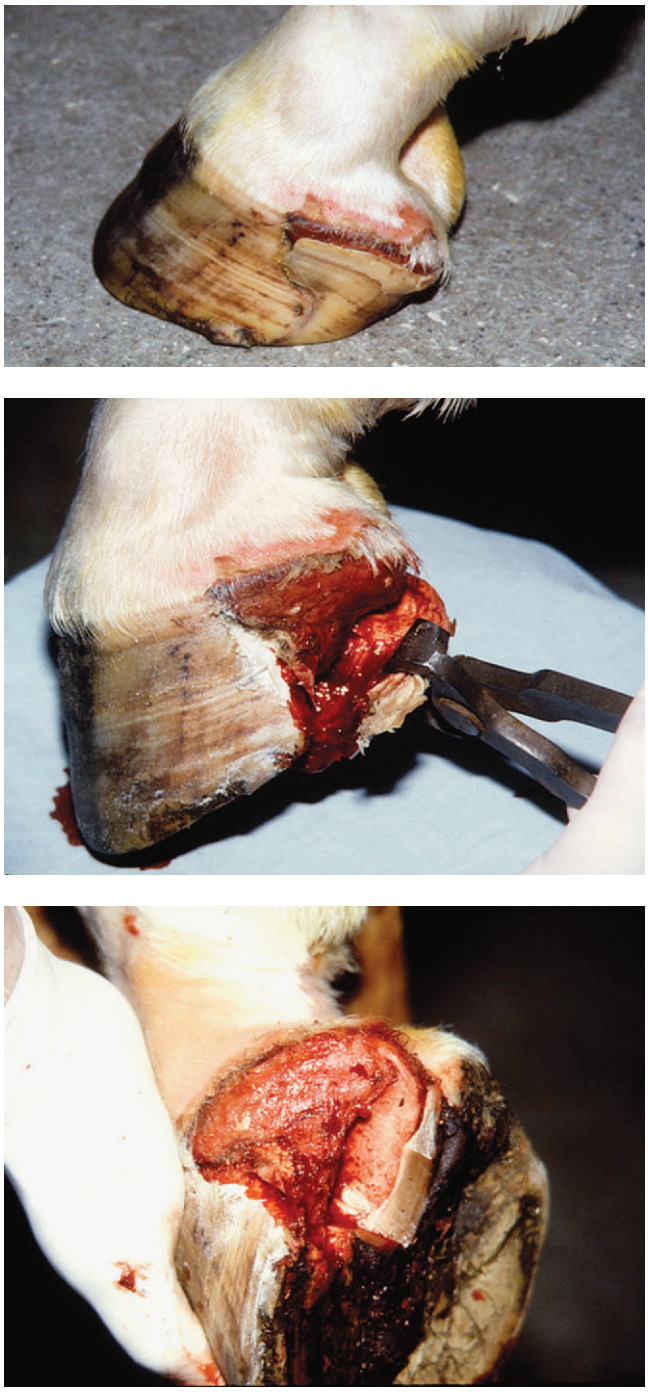


FIGURE 12-20 **A,** The hoof wall in the heel region in this horse has been completely torn away from the underlying dermis. **B,** The loose segment of hoof wall is being removed with a shoe puller. **C,** Appearance of the heel with remaining underlying soft tissues after hoof wall removal.

lamina is complete. In most cases of traumatic avulsion of the hoof wall, the separation occurs between the stratum corneum and the stratum germinativum. The germinal cells of the stratum germinativum of the epidermis and the associated basement membrane remain attached to the underlying dermis. These germinal cells proliferate and enlarge, producing a new horn covering within 10 to 14 days. These cells only have the capacity to replace the stratum internum.

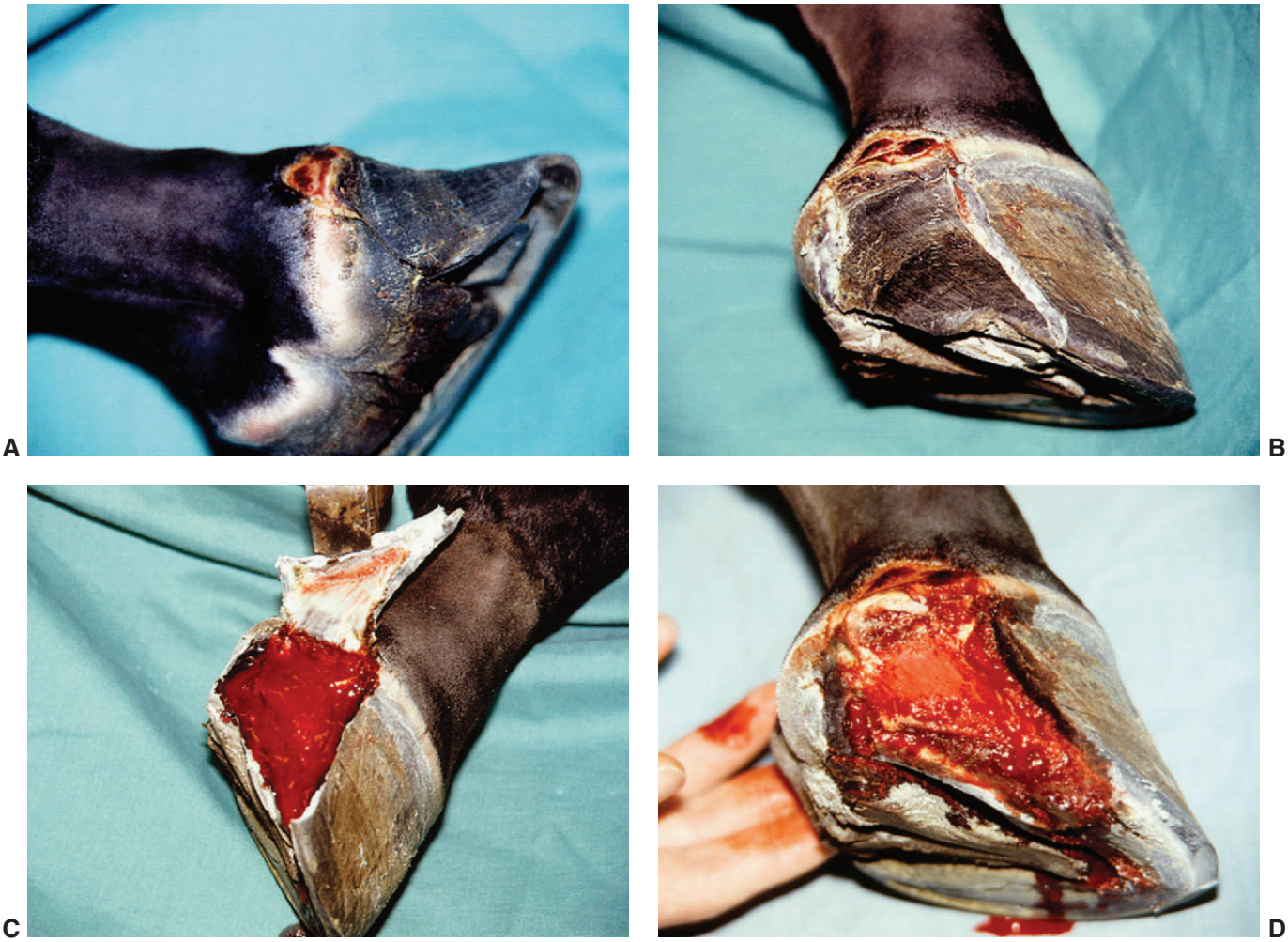


FIGURE 12-21 **A,** Note the crack at the most caudal aspect of the hoof wall. A large portion of the hoof was undermined and loose, and it was separated from the coronary band. **B,** The partially detached segment of hoof is separated from the normal hoof with a rotary burr. **C,** Hoof pullers are used to peel the loosened hoof wall away from the dermal lamina and coronary papillae. **D,** Appearance of the completed hoof wall resection prior to bandaging.

Replacement of the stratum medium and stratum externum necessary to reconstruct the remainder of the hoof wall occurs from the epidermal germinal cells at the coronary band as the hoof grows down.[6,7] Hoof regenerates at a rate of about 1/4 inch per month. Fully keratinized defects can be reconstructed with hoof acrylic if necessary to allow shoeing and return to performance activities.

Complete Avulsion of the Hoof Wall or Heel

If the traumatic injury has resulted in a complete loss of a segment of the hoof, including the primary and secondary epidermal laminae down to dermis, then the wound must heal by a process of granulation tissue formation and epithelialization from migration of germinal epithelium at the periphery of the wound (Figure 12-22).[6,7] Wound contraction cannot occur because of the rigidity of the surrounding hoof capsule, although the author has seen what appears to be contraction in the heel region where presumably the tissues are more compliant (Figure 12-23).

Deep wounds often involve the heel bulb, collateral cartilage, digital cushion, distal interphalangeal joint, or third phalanx (P3). In these cases, early therapeutic intervention is critical to success. Preparation of the foot is as previously described. Systemic antibiotics are often used but are probably of little value. Phenylbutazone is indicated for its analgesic and anti-inflammatory properties.

After aseptic preparation of the foot, the wound should undergo thorough and meticulous debridement and lavage of the exposed tissues to create a healthy wound environment. Joint lavage and regional limb perfusion are indicated if the joint and bone are exposed. Wet-to-dry dressings using saline-soaked gauze are a very helpful adjunct to obtaining a healthy wound bed. A sterile waterproof bandage is essential. The bandage is changed daily until a complete granulation tissue bed is present. Bandage changes should be conducted in a clean environment, keeping the foot elevated or using a clean or sterile towel to set the foot on to prevent contamination. The wound is cleaned with an antiseptic detergent and rinsed with saline. Pressure lavage using a spray bottle and dilute antiseptic or antibiotic solution is helpful in cleaning the surface of the forming granulation tissue. An egg-bar shoe or a heart-bar shoe can be applied as soon as a protective bed of healthy granulation tissue has been established. The

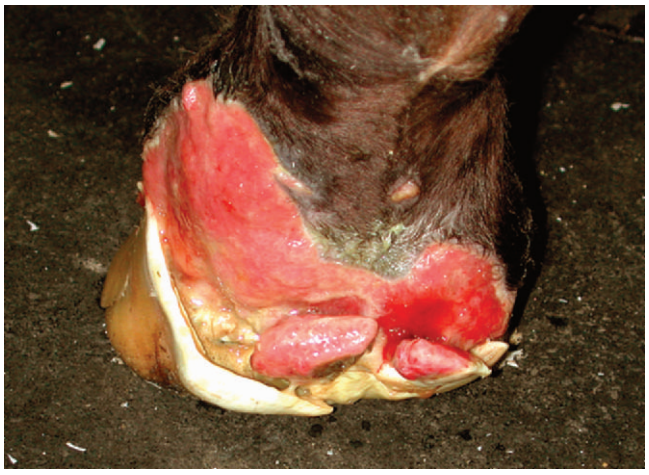


FIGURE 12-22 This avulsion injury to the heel has destroyed all the underlying corium of the hoof wall, resulting in healing by granulation tissue.

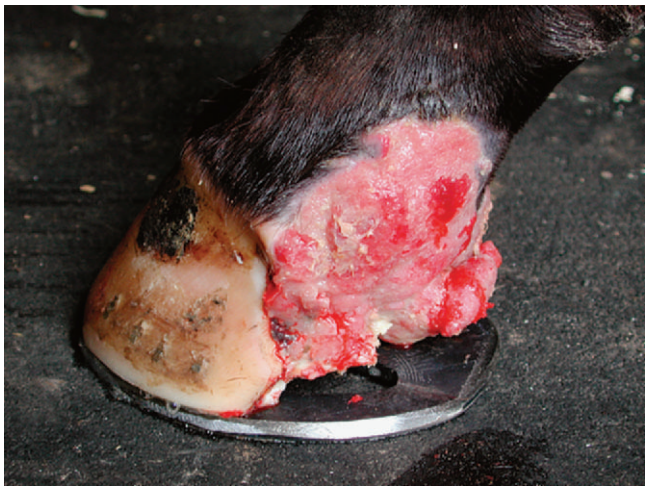


FIGURE 12-24 A heart–bar shoe has been applied to help support the foot in this healing avulsion injury.

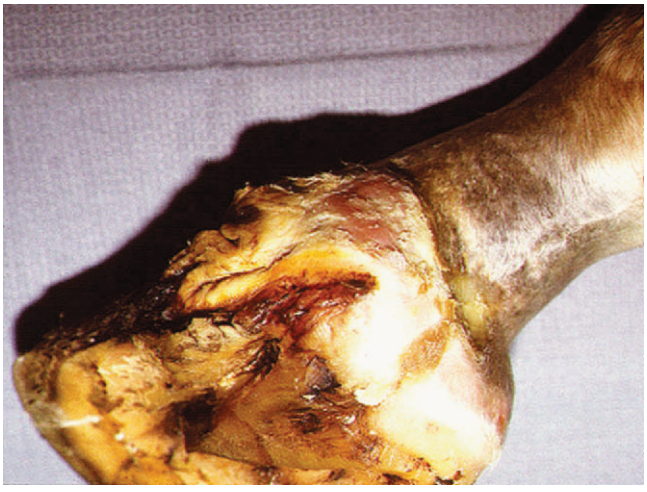


FIGURE 12-23 This horse sustained an avulsion injury similar to the one in Figure 12-22, but not as extensive. Note what appears to be contraction and reapposition of the new proliferating stratum internum after a period of cast immobilization.

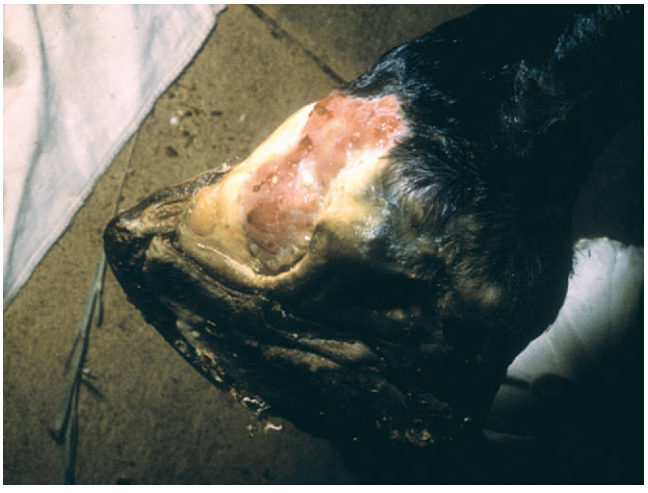


FIGURE 12-25 This wound demonstrates the new proliferating and migrating horn tissue produced by the germinal cells of the epidermal laminae.

type of shoe selected will depend upon the required amount of support and protection (Figure 12-24).

Bandages should be maintained until the germinal cells of the epidermal laminae originating from the wound margins can produce a new horn covering by proliferating and migrating over the granulation tissue bed (Figure 12-25). As healing progresses, the interval between bandage changes can be extended to every 3 to 5 days or longer. Large wounds can take months to heal. Caustic substances can delay the epithelialization and keratinization process and should be avoided. A semiocclusive wound dressing (Release) provides the optimal wound-healing environment. Exuberant granulation tissue can be controlled by excision or the judicious application of triamcinolone ointment or cream. A foot cast can also be used once a granulating wound bed is established and epithelialization has begun. The cast protects the foot from further trauma and provides a suitable wound environment without the expense of frequent bandage changes. The cast should be changed every 2 weeks (Figure 12-26). If the epithelialization ceases or slows because of overmaturation of the granulating wound bed, debridement to stimulate new capillary formation and renew epithelial migration can be performed every 30 to 45 days or as needed.[6] The horse should be confined to a clean, dry stall during this period. Once the wound is healed, a protective egg-bar shoe is usually necessary for the remainder of the horse's life to provide foot support if a segment of the heel has been lost (Figure 12-27).

TRAUMATIC HOOF WALL DEFECTS

Occasionally horses suffer full-thickness hoof wall injuries as a result of a penetrating injury or, more commonly, from surgical trephination of the hoof wall to treat a deep-seated infection or for drainage and curettage of an infected collateral cartilage. The defect will fill in with granulation tissue followed by proliferation and migration of the surrounding laminar epithelium. Only the stratum internum is replaced in

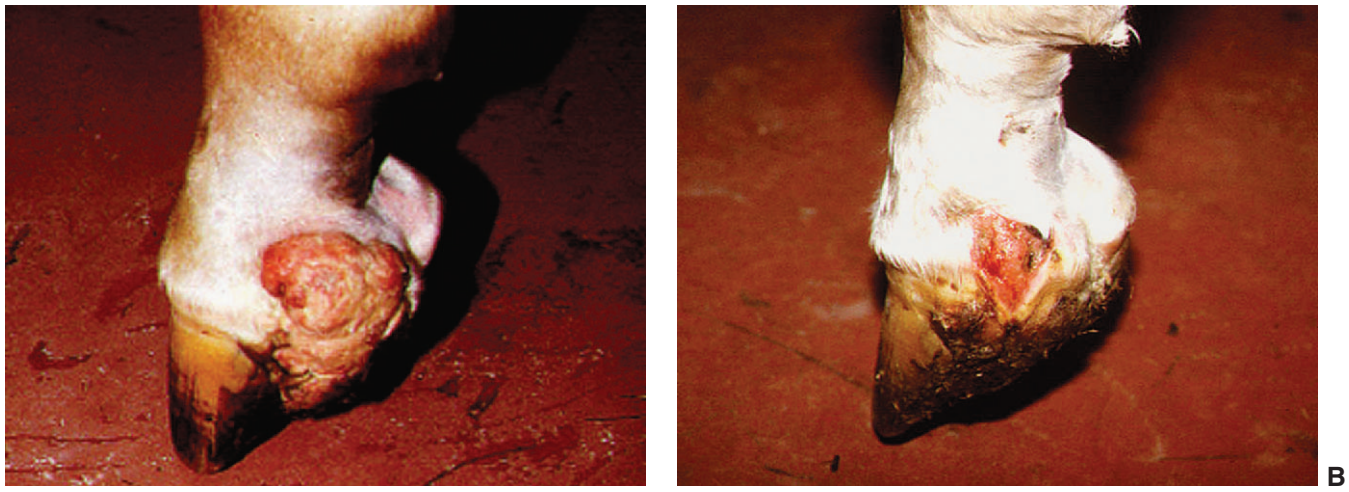


FIGURE 12-26 **A,** This wound was allowed to heal by second intention under a bandage without any attempt to control exuberant granulation tissue. **B,** The same wound after debulking the granulation tissue and immobilizing the foot in a cast for 2 weeks.

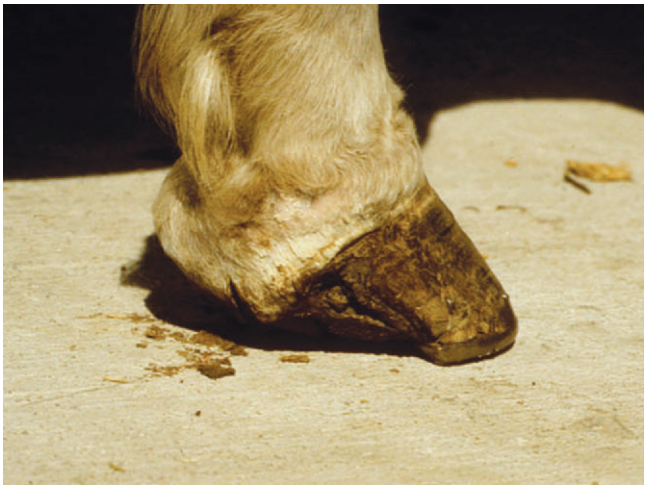


FIGURE 12-27 Note where the shoe ends on this foot. The application of a bar shoe would likely provide more support to the foot.

the initial healing phase. Full restoration of normal hoof wall thickness must await replacement of the stratum externum and stratum medium as the hoof grows down from the coronary band. Keratinized defects can be repaired with a hoof acrylic if desired. The application procedure is described in the section on hoof cracks.

THRUSH

Thrush is the term commonly used to describe a pododermatitis of the frog caused by a bacterial infection. The collateral and central sulci of the frog are invaded by keratolytic bacteria causing degeneration and breakdown of the frog tissue, accompanied by the presence of foul-smelling black exudate. *Fusobacterium necrophorum* is reportedly the most commonly isolated organism, but published data on the true incidence of the presence of this organism and others are lacking.[8,9] This organism is also a cause of equine canker, thus making it very difficult to understand how a single species of bacteria can produce two entirely different clinical and pathologic presentations in the same frog tissue. Obviously the true processes underlying these diseases are poorly understood.

Fusobacterium necrophorum is a gram-negative, obligate anaerobic bacillus. The organism is part of the normal fecal flora of a number of large animal species, including cattle and horses, and it can survive in the soil and environment for months. It is ubiquitous in the environment. *Fusobacterium necrophorum* is classified as an opportunistic pathogen with little ability to penetrate and invade normal epithelial tissue. This classification suggests that there must be predisposing factors to the establishment of infection, such as tissue necrosis from trauma or a poor blood supply that could lower the oxidation-reduction potential, thus providing a favorable environment for the growth of anaerobes. These prerequisites also apply to most of the other anaerobic bacteria that may be involved in this condition. It is also very likely that other species of aerobic bacteria are involved as well, facilitating the invasion of the *Fusobacterium* species or other anaerobes. Once the *Fusobacterium* organism is established, it can release degradative enzymes such as collagenase that would facilitate its spread into the deeper structures of the foot. Invasion into the sensitive structures of the corium of the frog, the digital cushion, and the heel bulb region leads to inflammation and lameness.

Although the reported predisposing cause of thrush is a damp environment and poor stable sanitation,[10,11] most farriers have seen thrush in horses kept in well-managed stables. The opposite is also true: not all horses kept in unsanitary environments, such as feedlots or persistently wet environments, develop thrush. This supports the theory that there is an underlying cause in many cases besides the environment. It is true that many of the gaited horses that wear pads have thrush, but most of these horses also have very long hoofs with contracted heels and a poorly developed frog.[10] It is the authors' opinion that the most common causes of this condition are poor foot health from lack of exercise, lack of proper trimming and general hoof care, or foot imbalance leading to sheared heels.

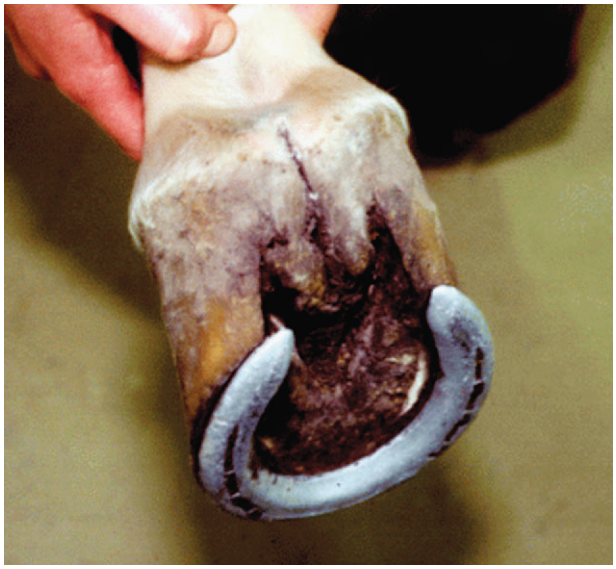


FIGURE 12-28 This horse demonstrates the classic signs of sheared heels and accompanying thrush and degeneration of the frog. Note that the fissure extends through the hairline into the digital cushion.

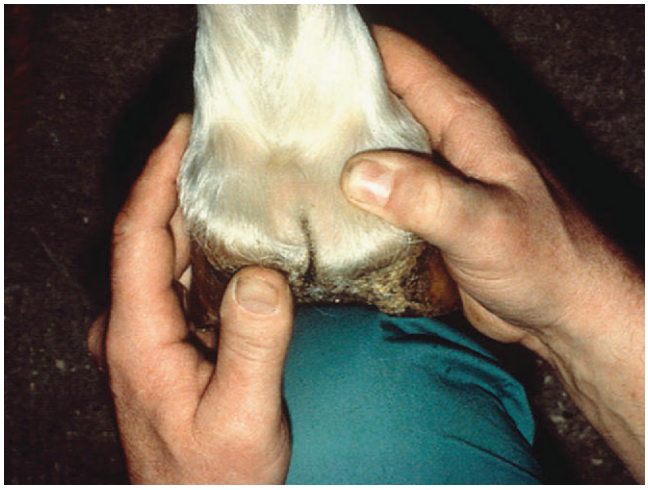


FIGURE 12-29 The heel bulbs can be manipulated independently with sheared heels.

A

B

FIGURE 12-30 This is another case of sheared heels with secondary thrush demonstrating the depth of the fissure using a tongue depressor.

Diagnosis and Clinical Signs

Horses can contract at least three different types of thrush, and it is important to make the distinction since it influences the response to therapy. One type occurs secondary to sheared heels. These horses are usually lame and generally have a very deep cleft or fissure in the central sulcus of the frog that extends down into the sensitive tissues of the heel (Figure 12-28). Invariably the fissure extends through the hairline at the heel into the digital cushion. The heels move independently with simple thumb pressure (Figure 12-29). By definition this is a sheared heel.[12] Insertion of a hoof pick or tongue depressor into the fissure elicits pain and bleeding as it contacts the sensitive tissues of the frog (Figure 12-30). The lameness is alleviated with a palmar digital nerve block. It could be argued which came first, the thrush that destroyed the frog and thus led to the sheared heels, or a hoof imbalance that led to physical shearing damage to the frog and digital cushion, which provided the proper conditions for bacterial invasion and colonization. Both are possible, but the latter scenario seems more plausible in most cases, especially in any horse that shows the characteristic signs of foot imbalance with upward displacement of the medial heel and flaring of the opposite side of the hoof. At a trot and at a walk, these horses usually land on the outside wall of the foot first and then load the medial heel as the weight comes onto the hoof, displacing it proximally.

The second type of thrush is in the horse that fits the first scenario described. The horse is examined with a clinical picture that would be defined as *sheared heels*, but without any evidence of hoof imbalance (Figure 12-31). Long and contracted heels are also common features in these cases.

The third and more common form of thrush can occur without the presence of sheared heels. There is usually a characteristic black, foul discharge from areas of degenerative and undermined frog. The lateral sulci of the frog are more

FIGURE 12-31 This horse had long and contracted heels, with poor frog quality and a central sulcus fissure.

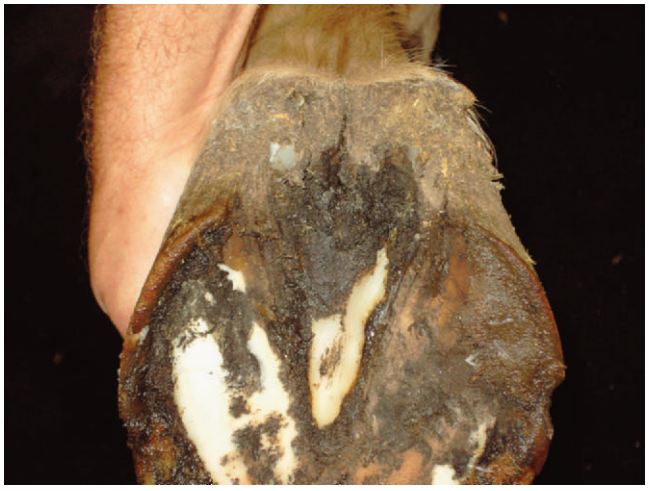


FIGURE 12-32 Typical case of thrush affecting a majority of the frog.

likely to be affected and the central sulcus is often not involved to the same extent as with sheared heels (Figure 12-32). Lameness may or may not be present.

Treatment

Treatment is directed at addressing the primary cause, which includes improving sanitation, providing a dry environment for the hoof, restoring proper balance to the foot, and providing adequate exercise or turn-out to improve the blood flow and general health of the foot. The infection is easy to deal with if the primary cause is eliminated. It is the authors' experience that horses with sheared heels as a result of hoof imbalance have often been treated with topical thrush medications for months without any response, which is understandable in light of the underlying problem (Figure 12-33). The thrush will resolve only after balancing the foot as much as possible and applying a bar shoe to eliminate the shearing forces. Healing is rapid, often without any other treatment.

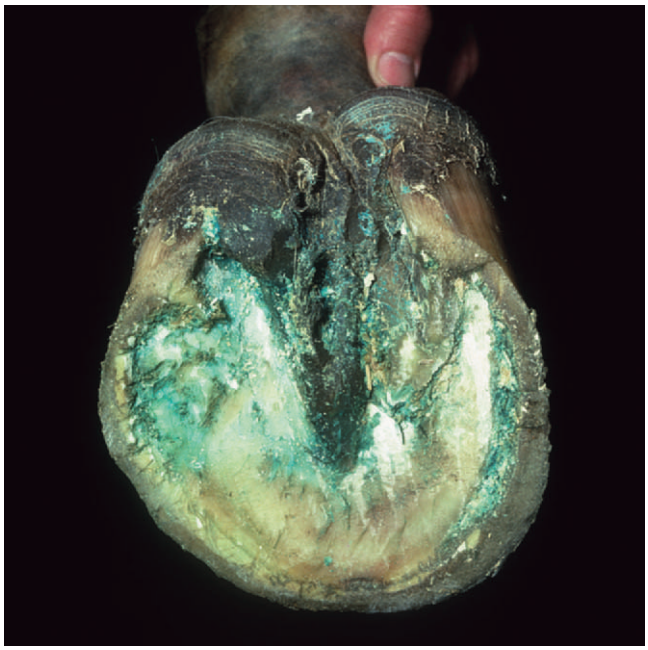


FIGURE 12-33 This horse has been treated with copper sulfate for months with little response. The foot is severely imbalanced.

In the typical case, the frog should be debrided of all loose tags and undermined and necrotic tissue. All pockets and crevices should be opened to allow exposure for cleaning and medicating. There are literally dozens of commercial products on the market for the treatment of thrush. Most products contain caustic materials such as iodine and copper sulfate.[3] Caustic agents should be avoided whenever sensitive tissues are exposed. Metronidazole is the drug of choice for gram-negative anaerobic infections, and it should be effective in most cases if more standard treatments fail. It can be applied topically in a similar manner as described for the treatment of canker. Diluted 1% povidone iodine and 0.5% chlorhexidine are both good antiseptic treatment choices. Chlorine dioxide is also a popular choice, but it is not the same as chlorine bleach. Chlorine dioxide is classified as an oxidizing biocide; it destroys the microorganisms by means of interrupting the food transport along the cell walls. It is commonly used for water purification and disinfection in the food processing industry. It is an unstable gas that is usually packaged as two stable components that require mixing before use. It comes in liquid and gel formulations (White Lightning; Grand Circuit Products, Adelphia, NJ).

The crevice in the frog should be gently cleaned by swabbing with a dry gauze sponge wrapped around a hoof pick. Cotton balls purchased at the drug store can then be soaked in the desired treatment solution and loosely packed into the crevice. This procedure should be repeated daily as needed until the infection is under control. Bandaging the foot is usually not necessary.

HOOF CRACKS

Hoof cracks are fractures of the hoof capsule. The hoof capsule is a highly structured and very complex composite of keratinized epithelial cells.[13] It can withstand large loads

without undergoing permanent deformation. Under normal circumstances, the hoof wall does not approach strains that would result in failure.[14] The stratum medium, which accounts for most of the thickness of the wall, has a unique structure of tubules with an intertubular matrix that is very resistant to fracture and has a unique crack-diversion mechanism to minimize damage.[14-16] Nevertheless, damage seems to occur fairly frequently, although the true incidence remains undocumented.

Cracks are usually named after their location: toe, quarter, heel, or bar. Cracks can be superficial or full-thickness and they can run vertically, horizontally, or angularly through the various layers of the horn. The true cause of many cracks is difficult to discern and may be cause by a combination of factors that can affect the quality of the horn, including environmental conditions, genetics (small feet, long toes, thin walls and soles, and under-run heels), nutrition, shoeing practices, trauma, stable management, level of exercise, and use of the horse. Microscopic examination of horn tubules adjacent to cracks in one study revealed abnormal tubule structure or the presence of empty spaces suggesting damage to the coronary papillae that led to poor horn quality, which probably predisposed the hoof wall to fracture failure.[17]

Etiology

A horse's foot is the product of its environment, which includes stabling and pasture conditions. It has long been assumed by veterinarians and farriers that horses exposed to constantly wet environments or to alternating wet and dry environments are more susceptible to developing hoof cracks. As a result of this concern, several studies have looked at moisture content and its influence on the mechanical properties of the hoof.[18-21] There is a stiffness gradient in the hoof wall that is inversely related to the moisture content.[19] The outer layers of the hoof wall, which are exposed to the environment, are dryer and stiffer than the inner hoof wall. This gradual stiffness and moisture gradient is necessary to safely transfer the large ground contact loads from the hoof to the bone column. Abrupt changes in stiffness between the epidermis and the dermis could lead to higher stresses and potential cyclic loading failure. The moisture gradient is due to the proximity of the various hoof layers to the underlying dermis and its vascular supply, which is the source of the moisture.[14] It is presumed that water moves from the dermis to the stratum internum and out through the stratum medium as a result of a hydrostatic pressure gradient as well as an osmotic gradient.[3] Given this model, it would seem plausible that increasing the level of exercise would increase the moisture content by increasing the circulation.

The physiologic moisture content varies significantly between horses and between different segments of hoof.[22] In general, resistance to fracture was greatest at normal hoof hydration levels, and overhydrated or fully hydrated horn was less stiff and more prone to crack propagation. The outer stratum medium is capable of absorbing water within 24 hours of immersion,[3] suggesting that environmental changes can disrupt the normal moisture gradient in the hoof, potentially weakening it.

The effect of nutrition and dietary supplementation on hoof quality and strength has been difficult to evaluate because of the difficulty in conducting controlled studies that account for all the variables. One controlled study has been able to demonstrate that dietary biotin supplementation at a dose rate of 0.12 mg/kg shows a positive treatment effect on hoof growth after 5 months of biotin supplementation.[23] This study was conducted on match-paired ponies with no obvious hoof capsule abnormalities. The study did not look at the effects of biotin supplementation on the quality or strength of the hoof wall produced. Studies on Lipizzaner horses documented an improvement in hoof quality (less decay and less tendency to develop cracks) and tensile strength, but not growth rate when the horses were fed 20 mg, or 0.04 mg/kg bodyweight, of biotin per day over a 38-month period.[24,25]

Finally, balance is one of the most important aspects of a healthy hoof capsule. A horse that is trimmed out of balance, or has conformational defects that cause exaggerated or unequal landing patterns, creates undo stress on the hoof capsule, causing it to become weak. A foot that is allowed to grow long has increased bending loads and is more subject to failure.

Commonly described causes of toe cracks are injury to the coronary papillae, a severe abscess that undermines the hoof wall and breaks at the coronary band, a compromised white line due to chronic laminitis or long overgrown toes, or mechanical tears such as those occurring with a clubfoot. Quarter cracks are often the result of short shoeing, long toes and under-run heels, mediolateral hoof imbalance, or injury to the coronary band. The hoof wall is generally thinner in the quarters, and cracks in this area usually involve the dermal layer. These cracks also are frequently infected and a cause of lameness. Heels cracks can be caused by trauma, short shoeing, or shoes being left on too long. Bar cracks are less frequent and may be due to lack of trimming, trauma, or shoes left on too long.

Horizontal cracks coursing parallel to the coronet are sometimes termed *blowouts*. They are caused by a disruption in horn production from a blow to the coronet or a severe abscess that drains from the coronet (Figure 12-34). They rarely are a problem, but a blowout in the heel region may need to be shod or trimmed out as it nears ground level to prevent it from snagging and tearing if caught on an object. A blowout in the quarter area may present a challenge to the farrier, when it nears ground level, in trying to place and clinch nails in the hoof capsule.

Vertical cracks can start at the coronary band and extend distally or begin at the ground and extend proximally (Figure 12-35). Cracks are also classified as *superficial* or *full-thickness* (Figure 12-36). Superficial cracks are not associated with pain and lameness because there is no dermal involvement. They are usually associated with feet that have incorrect moisture content, feet that go from wet to dry throughout the day, or a nutritional deficiency. Full-thickness cracks are associated with pain because of dermal involvement. These cracks are unstable; they can spread, pinch, or have vertical movement when the horse moves from non–weight bearing to weight bearing. Generally, the more unstable the crack is, the more pain and lameness are present. Hemorrhaging and purulent discharge may be present and caused by infection (Figure 12-37). The site of pain can be confirmed through simple thumb pressure, the use of hoof testers, or hoof percussion with a hammer. Perineural anesthesia of the palmar

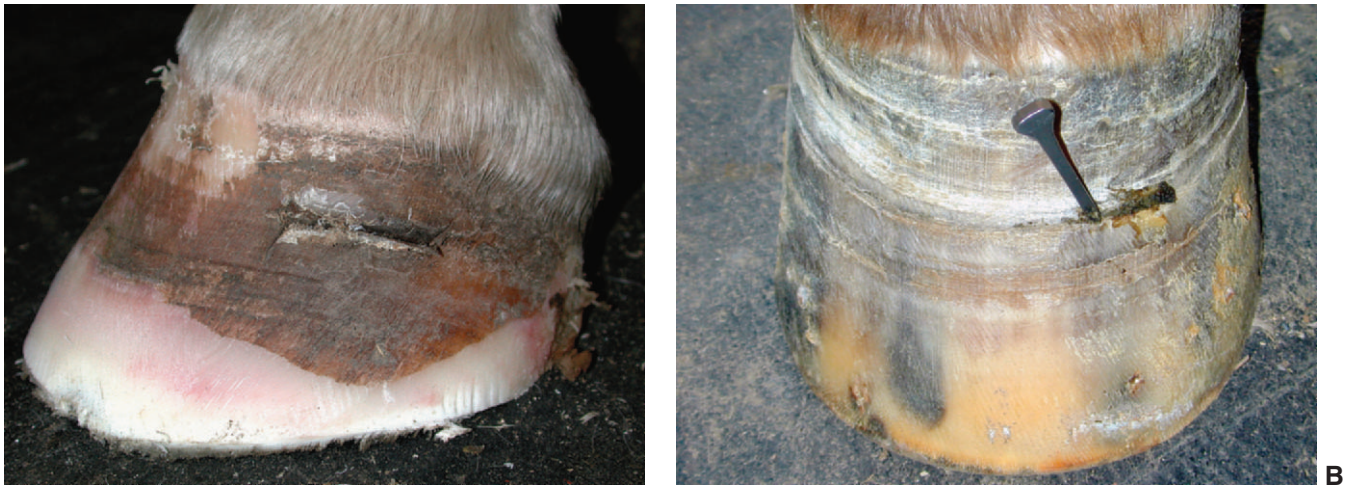


FIGURE 12-34 **A,** Typical appearance of a horizontal hoof crack. **B,** Exploration of a horizontal hoof crack using a shoeing nail.

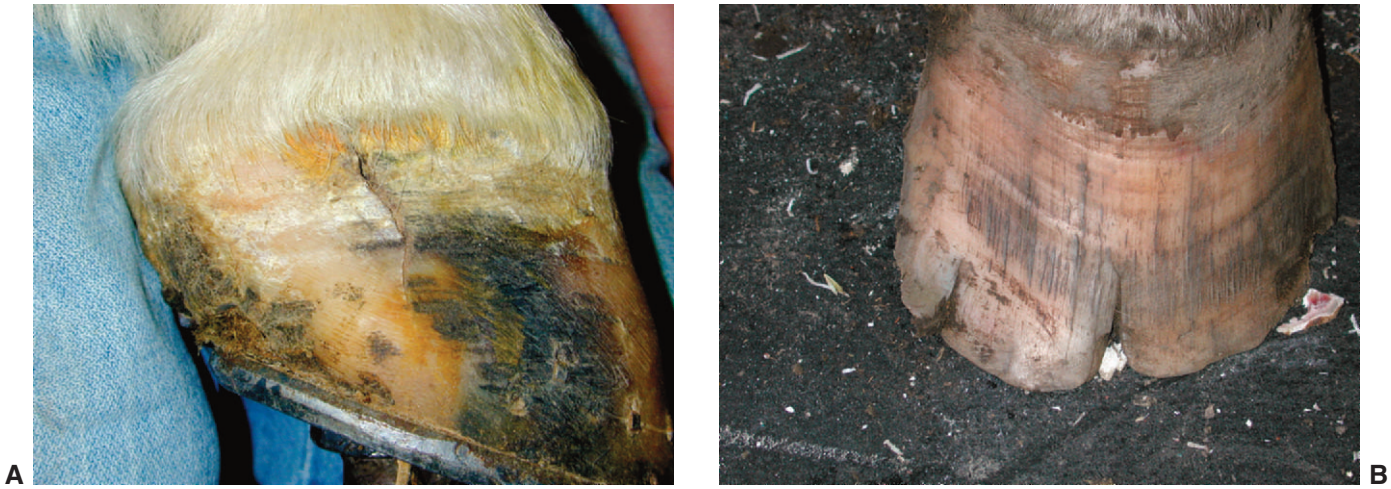


FIGURE 12-35 **A,** This quarter crack begins proximally at the coronary band and extends distally. **B,** This toe crack is superficial; it begins distally and extends proximally.

or plantar nerves on the affected side can help to rule out other contributing causes to the lameness.

Treatment

Treatment of cracks usually begins with an evaluation of the hoof capsule as a whole, looking for quality of the horn, thickness of the hoof wall, moisture content, hoof balance, dermal involvement, and presence of infection. The method of repair depends on the location, depth, and extent of the crack, as well as the degree of instability, thickness of the hoof wall, and whether infection is present.

There is a long history of methods to stabilize complete hoof wall fractures, which suggests there is no one best method that will work in all situations. Because the fractures are usually caused by abnormal stresses and strains on the hoof capsule, balancing the foot to the extent possible and applying a bar shoe to stabilize the foot are the basic principles of therapy (Figure 12-38). Drawing clips on either side of the fracture when applying a shoe should also be applied if feasible. Many different types of materials and appliances have

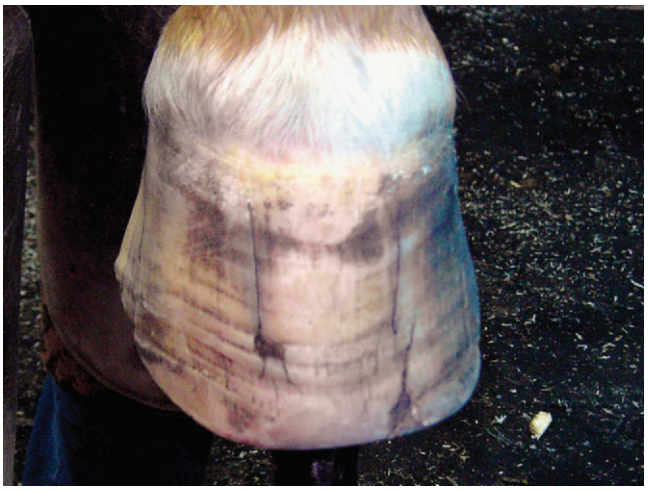


FIGURE 12-36 The cracks in this hoof are superficial and are not causing a lameness problem.

FIGURE 12-37 Note the hemorrhage from this full-thickness quarter crack.

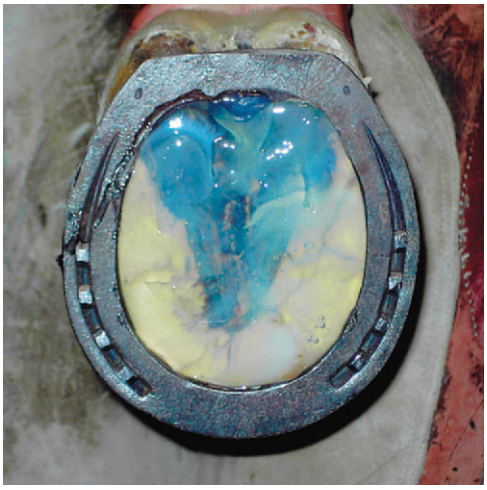


FIGURE 12-39 The use of impression material on the entire surface of the sole may offer additional stabilization by decreasing the drop of the sole during weight bearing.

FIGURE 12-38 A bar shoe with clips used to help stabilize a toe crack.

been used for definitive repair, in various combinations, including fiberglass, radiator hose clamps, nails, screws and wire, steel or brass plates, Kevlar mesh, and clipped shoes.[3,26] Today, the availability of modern hoof repair composites (especially polymethylmethacrylate acrylics [PMMA] and polyurethanes) has replaced most of these older techniques as the preferred method of repair. Today's composites have very high tensile strength, they adhere well to the properly prepared hoof wall, and when combined with wire lacing and fiberglass or Kevlar mesh they can be expected to produce consistently good results in stabilizing even the most challenging hoof capsule fractures.

In the case of full-thickness fractures, the fracture site is opened up using a Dremel tool (Robert Bosch Tool Corporation, Racine, Wis.) and hoof knife and all undermined horn and necrotic tissue is removed. The foot is trimmed and balanced. The foot is then measured and a bar shoe is forged with clips if they can be positioned on either side of the crack. The bar shoe provides a greater base of support to the hoof and decreases the independent vertical movement of the heels. The clips placed on either side of the fracture help prevent expansion of the foot. The use of impression material (Vettec, Horse Care Products, Oxnard, Calif.) on the entire surface of the sole may offer additional stabilization by decreasing the drop of the sole during weight bearing and further decreasing movement of the hoof capsule until the fracture has been replaced with new horn, which can be up to 1 year (Figure 12-39). If the corium is exposed and hemorrhage or infection is present, then the wound should be packed with sugar-dine (table sugar and povidone iodine solution mixed to a paste consistency) or a similar antiseptic drying agent under a bandage until the infection is under control and the crack is dry. This usually can be accomplished in a few days. It is best to postpone definitive treatment with a hoof composite until the tissues are fully keratinized, to protect the underlying tissues from the exothermic heat reaction that takes place as the composite cures. If the horse needs to return to work immediately, the repair can be completed as will be described, with care taken not to seal up the crack, which might allow the infection to spread. Alternatively, a technique of repair can be chosen that does not rely on composites (Figures 12-40).

Most full-thickness hoof capsule fractures that extend for any distance should be stabilized with wire lacing to support the composite repair. The wire behaves like reinforcing rods in concrete, greatly strengthening the repair and providing an anchor for the composite. Careful preparation of the hoof wall is necessary to ensure that the composite does not loosen prematurely. The hoof wall that will be covered with the composite material is lightly sanded to remove the periople and clean and roughen the surface. Scrubbing the hoof wall with acetone or denatured alcohol will remove the lipid layer and allow for better adhesion. For the wire lacing procedure, anesthetizing the foot and sedating the horse is recommended. Entering too close to the dermal layer with the drill bit can cause sudden movements by the horse, resulting in broken bits or injury to personnel. All drilling should be done with the use of a hoof stand to support the foot, and the drill operator should never be off their feet for safety reasons. The steps for repairing a quarter crack and a toe crack are presented as examples (Figures 12-41 and 12-42). Drilling should be

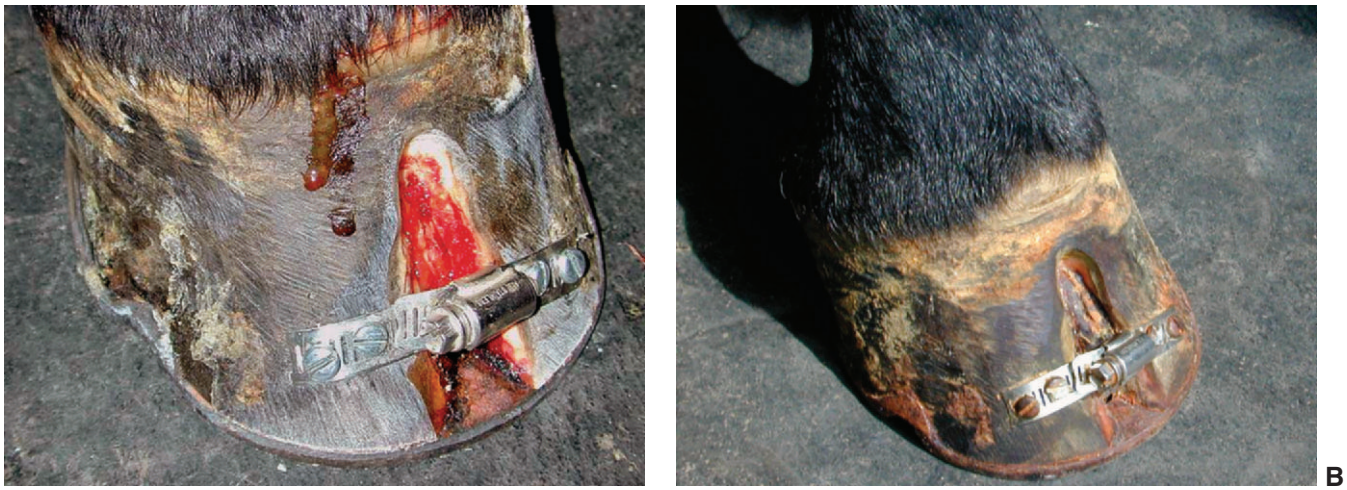


FIGURE 12-40 **A,** This infected toe crack was debrided, a bar shoe with clips was applied, and the crack was stabilized with a radiator hose clamp secured to the hoof wall with screws. **B,** Appearance of the crack 2 weeks after repair.

accomplished as deeply as possible in the insensitive stratum medium to provide a solid anchor for the wire. Brass buttons or plates should be used to prevent the wires from cutting through the hoof material as they are tightened.

One basic rule is to never cover the exposed corium with composites without a layer of Play-Doh (Hasbro, Pawtucket, RI) or putty between the corium and the patch. Openings should always be left for drainage, preferably proximal and distal to the affected area, if there is even a remote chance of recurrent infection. This is accomplished by placing a 1/8-inch rubber or polyethylene tubing along the length of the crack and then covering it with the clay or putty and applying the composite material with or without stabilizing wires. Composite is also available that is impregnated with antibiotics. Once the composite is set, pulling one end of the tubing out ventrally will remove it, leaving behind a track for drainage and application of topical medication or flushing (Figure 12-43).

If the use of a drainage tube is planned, it should be installed under a layer of clay or putty before the wires are placed. Just prior to application of the composite, the hoof is given a final cleaning with acetone. The authors prefer to use a PMMA-based system (Equilox Adhesive System; Equilox International, Inc., Pine Island, Minn.). This system consists of two components that are mixed thoroughly just before application. Gloves should be worn when handling the composite material. The coronary band should be protected with tape. If at all possible, the working environment should be clean, dry, and warm. The mixture normally sets up within 2 to 15 minutes (cold temperatures significantly slow the cure time), so everything needed should be laid out ahead of time. First, a uniform coat of the composite is applied with a wooden tongue depressor to fill the crack and cover the prepared hoof wall. Then the foot is wrapped in a sheet of plastic obstetrics sleeve held in place with Vetrap (3M, St. Paul, Minn.) or other suitable elastic bandage material to provide an oxygen-free environment to speed the set up time. When the repair has hardened, the bandage is removed and the next step is begun, which is the addition of three to six layers of fiberglass cloth (Bondo Corporation, Atlanta, Ga.), depending on the stability needed. The fiberglass pieces should be precut and laid out on a layer of cellophane and impregnated with the PMMA using a tongue depressor to force the composite material into the fabric. They are applied in successive layers after a light coat of the composite is applied to the previous repair. The fiberglass cloth is covered with enough composite to produce a smooth surface, and the area is again covered with plastic and elastic tape to facilitate the final cure. The plastic can be used to smooth the surface of the repair by applying it with pressure and molding it with finger pressure. Once the repair has hardened, it can be shaped and smoothed with a rasp or by light sanding, similar to normal hoof material. Once the repair has fully cured (a few hours in most cases), the horse can return to normal activity.

If the horse's hoof lacks sufficient wall thickness to allow the use of wire or other metal implants, a technique of drilling multiple shallow holes in the hoof wall on either side of the crack can be used to allow the hoof composite to better anchor to the hoof capsule to prevent shear failure. Undercutting the hoof wall on either side of the crack will also help anchor the composite to the wall. Reinforcement of the repair with fiberglass cloth is as described in Figure 12-44.

Cracks starting at the ground are usually the result of overgrown feet or a white line that has been compromised. If they are limited in their extent, composite repair is not always necessary. This is especially true if the cracks are not full thickness. The objective is to stabilize the hoof capsule to prevent further progression of the crack. Stabilization can usually be accomplished by trimming away the excess horn and balancing the foot and applying a bar shoe with clips drawn on either side of the crack. Most cracks continue microscopically beyond the visible crack. Because of that, a half-circle groove must be burned 1/4 inch above the crack and must be burned the full depth of the crack to help stop its progression after the foot has been trimmed and balanced. A specially forged tool is used to burn the half-groove (Figure 12-45). These cracks follow up the hoof wall between the horn tubules. Once the crack enters the burned groove, the crack is isolated. One note of caution: this method should never be used within 3/4 inch of the hairline, to prevent damage to the coronary papillae.

Cracks beginning at the coronary band and extending downward can be difficult to treat. Every effort should be

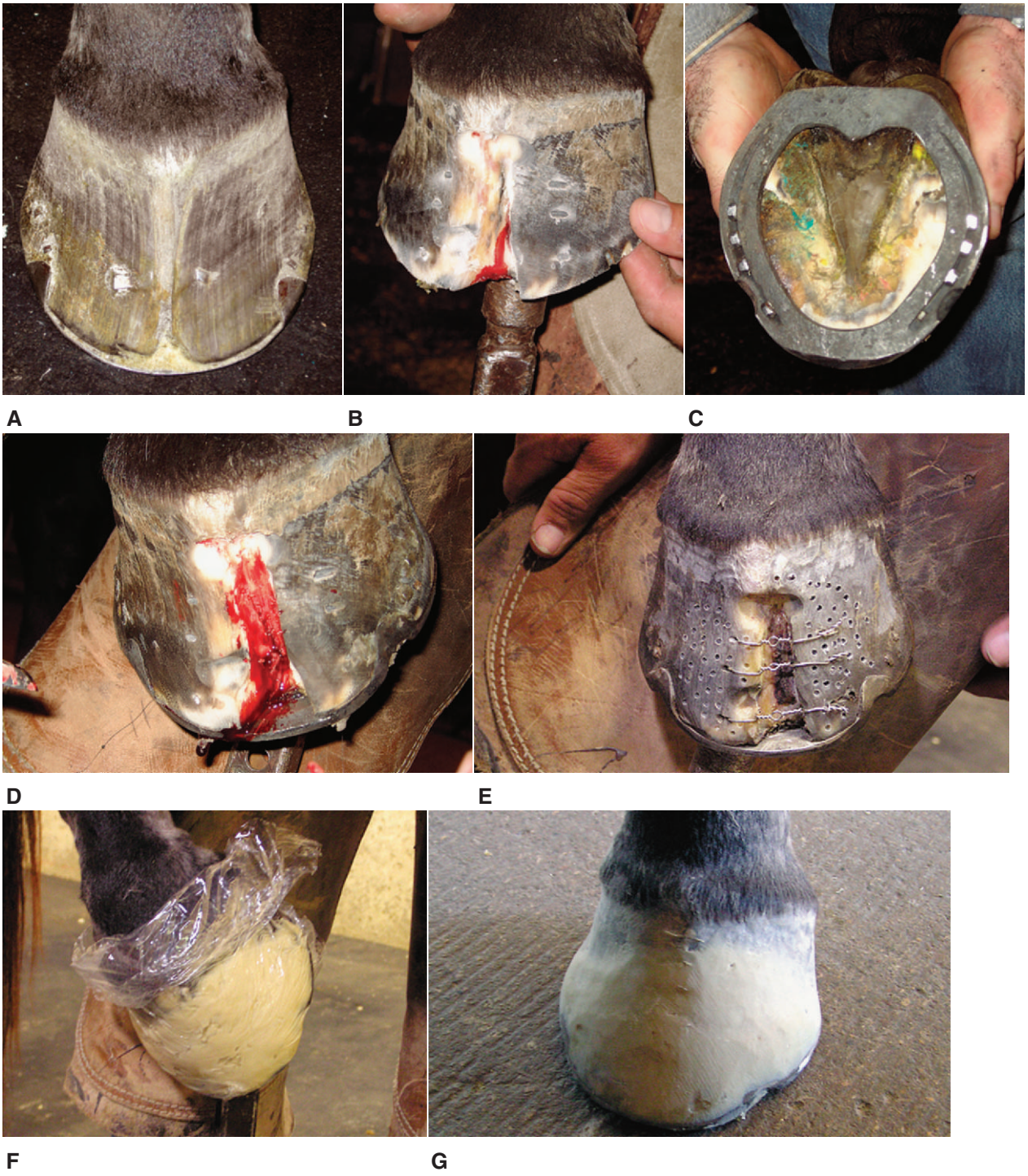


FIGURE 12-41 **A,** This toe crack was repaired with a bar shoe and clips, but the use of a shoeing nail to stabilize the crack was inadequate; the crack remained and horse continued to be lame. **B,** The crack has been initially debrided with a motorized burr. **C,** A bar shoe with clips has been applied. **D,** Further debridement of all damaged and necrotic lamina is accomplished with a scalpel. The crack was wired, and the foot was treated with sugar-dine and a bandage for several days. **E,** After 2 weeks, the crack is dry, cornified, and ready for repair with a composite. Additional wires were placed before the composite was applied. Holes have been drilled in the hoof wall to help anchor the composite material. **F,** After the application of a final layer of composite material, the foot is wrapped in a sheet of plastic obstetrics sleeve held in place with Vetrap or other suitable elastic bandage material to provide an oxygen-free environment to speed the set up time. **G,** Appearance of the final repair.

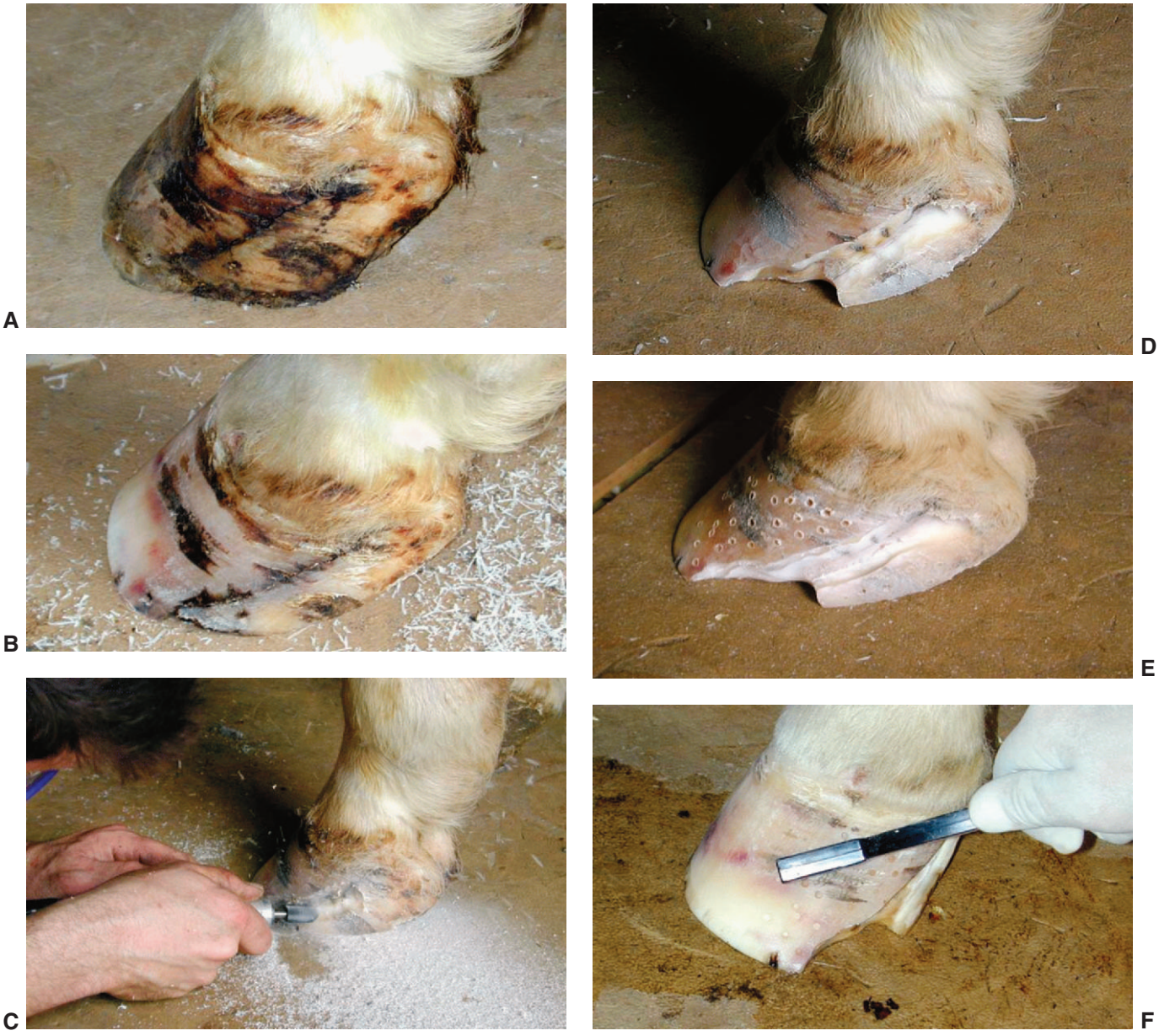
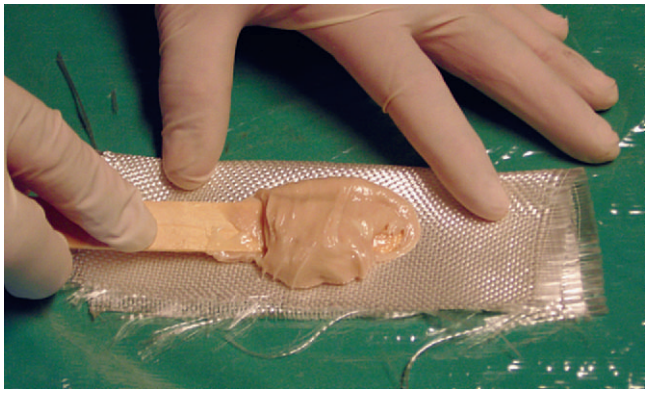


FIGURE 12-42 **A,** Appearance of a full-thickness quarter crack before repair. **B,** The hoof wall is rasped to begin preparation for repair. **C,** The crack is cleaned out with a motorized burr. **D,** Completion of the initial hoof preparation for acrylic repair. **E,** Small holes are drilled into the stratum medium to help anchor the composite material to the hoof wall. **F,** The hoof wall is thoroughly cleaned with acetone to remove any grease, lipids, or oil that would interfere with the bonding of the composite material to the hoof wall.

made to correct any imbalances in the foot. The combination of PMMA hoof composite and a bar shoe may provide the stability needed for adequate healing (Figure 12-46).

Many heel cracks can be debrided and repaired with one of the methods previously described. Heel cracks that begin at the ground surface and do not run full length to the coronet can be floated (Figure 12-47): the portion of the heel caudal to the fracture site can be rasped so there is no contact with the bar shoe. Leaving it in a non–weight-bearing state will help minimize movement. Floating heel cracks that originate at the coronet may cause additional shearing and separation of the fracture site. The use of a full support system, such as a heart-bar or straight-bar shoe in conjunction with a rigid sole support impression composite, will help prevent this occurrence. Alternatively, the hoof capsule caudal to the heel crack can be completely removed in circumstances in which horn quality, horn thickness, infection, or prior patch failure compromises the repair. The hoof capsule is stripped off, as discussed under avulsion injuries or by using a Dremel tool to burr down the hoof capsule to the nonpigmented stratum internum (Figure 12-48). Burring is the preferred method because there is less bleeding and damage to the dermal layer of hoof. Generally, the heel is replaced with new horn in 4 to 6 months. As the heel grows down, the most caudal edge will contact the shoe or ground surface first. This leading edge should be trimmed back as necessary until the remainder of the new hoof reaches the ground surface so that it does not cause a point of stress. With the aid of a bar shoe and

G



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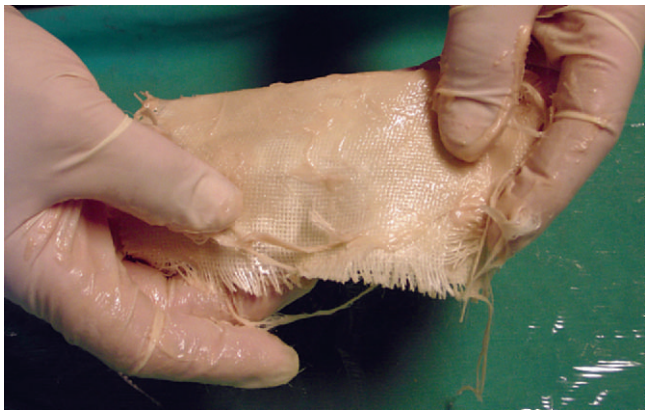

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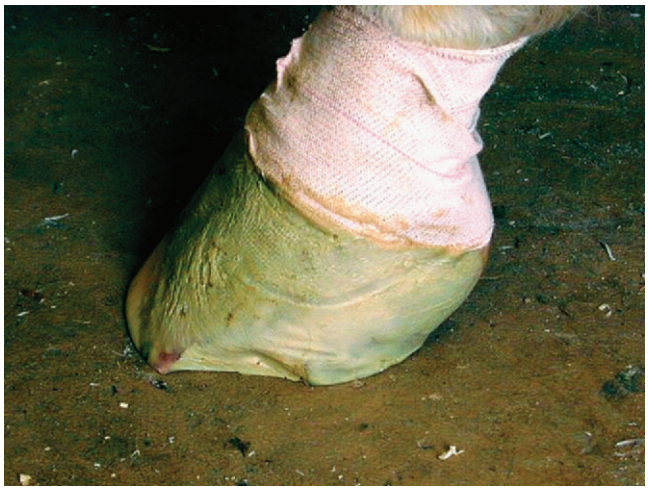

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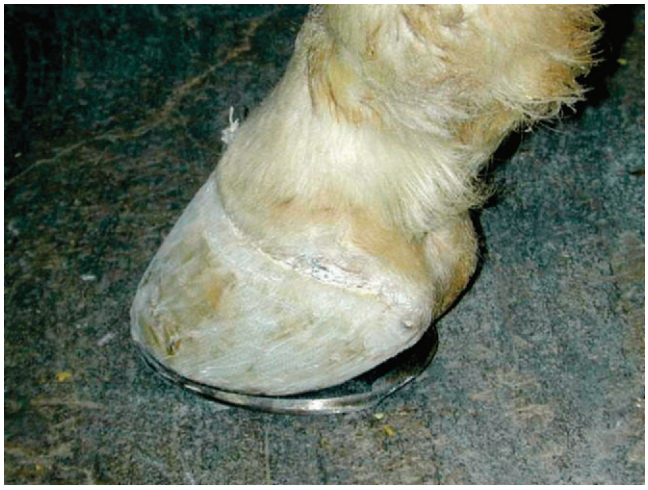


K

FIGURE 12-42, cont'd **G,** Layers of fiberglass cloth are cut to size and laid out on a layer of plastic. **H,** The fiberglass cloth is impregnated with the composite material using a wooden toe depressor. **I,** Five layers of fiberglass cloth are ready to be applied to the hoof wall. **J,** Several layers of composite material and fiberglass cloth have been applied. **K,** Appearance of the hoof after completion of the composite repair and the application of a bar shoe.

impression material, the horse will replace the resected area and will eventually be able to return to work.

EQUINE CANKER

Canker is an infrequently seen, hypertrophic, moist dermatitis of the frog and the bulbs of the heel. Specifically, it has been characterized as a gram-negative bacterial infection of the stratum germinativum of the epidermis of the frog.[27,28] The infection results in abnormal keratin production (dyskeratosis) recognized as white, filamentous fronds of soft vegetative horn.

Clinical Signs

Typically, the affected frog tissue is covered with a white caseous exudate resembling cottage cheese.[29] There may be a foul odor. Initially, horses may not be lame, but in advanced cases, the infection can extend to the sole and wall at the heels or produce enough inflammation of the underlying dermis to produce pain.[27] Pain also results from exposure of the underlying sensitive tissues after loss of the keratinized frog. All breeds are affected, but draft horses seem to be overrepresented, and the condition can occur in more than one foot at the same time. It is usually associated with chronically wet and unhygienic conditions that produce a favorable environment for bacterial growth[28] but can be seen in horses in a good stable environment.[30]

Initially, the disease can be confused with thrush. Thrush causes a destruction of the frog and usually involves the central, lateral, or medial sulci of the frog, as opposed to the proliferation of unkeratinized epithelial tissue, beginning anywhere in the frog, in canker. Canker can also manifest as a focal area of granulation tissue within the frog.[30] If the diagnosis is unclear, a biopsy is indicated because early recogni-

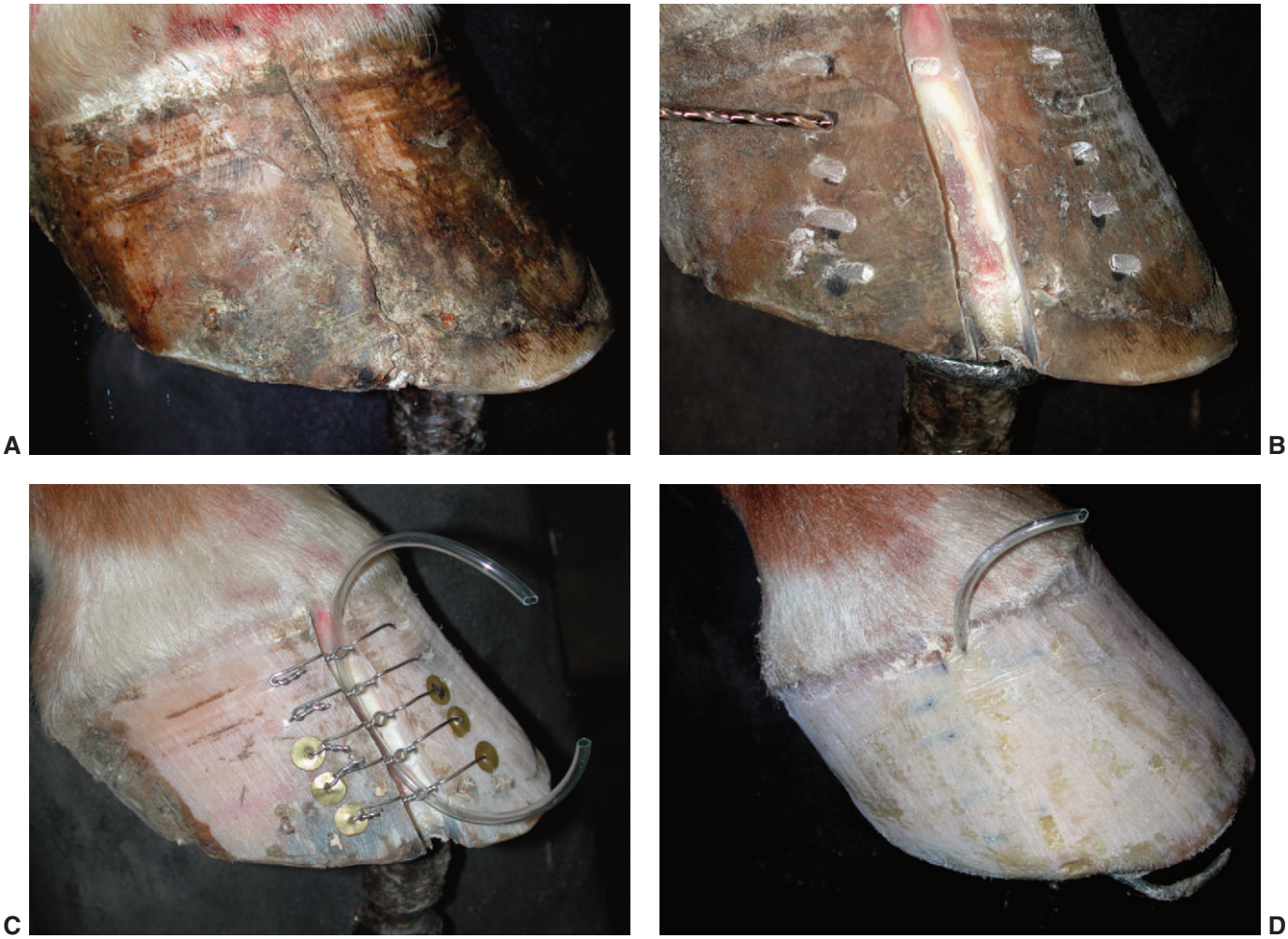


FIGURE 12-43 **A,** Quarter crack before repair. **B,** The crack has been debrided and holes are being drilled for the application of wires. **C,** Wires are in place and a drainage tube has been inserted because of the appearance of moisture and infection in the deep recesses of the crack. **D,** Completion of the composite repair before application of a shoe. The plastic tube will be removed to allow a hole for flushing and application of medication.

FIGURE 12-44 **A,** The hoof wall in this horse is very thin, making it difficult to safely use wires. **B,** Instead of trying to use wires, the wall has been drilled with multiple shallow holes to resist shear failure of the composite hoof wall bond. **C,** Appearance of the completed repair with PMMA hoof composite.

FIGURE 12-45 A half-round groove has been burned into the hoof to stop the progression of a superficial toe crack.

tion is important to successful management of the disease. A 6-mm biopsy punch will take an adequate sample. The site should first be debrided of necrotic debris and the sample should be deep enough to include the dermis. Several samples can be taken and at least one should include the juncture of normal and abnormal tissue. Canker is recognized histologically as a chronic pododermatitis of the deep germinal layers of the epidermis causing a focal, proliferative, papillary hyperplasia of the epidermis, with minimal involvement of the dermis. Cultures are not that helpful, since they typically yield a variety of organisms, both aerobic and anaerobic. *Fusobaterium necrophorus* is the most commonly isolated organism.[28,31]

Treatment

To date, the best treatment results have come from initially cleaning the foot; conservative debridement of obviously diseased tissue; application of topical metronidazole; and maintenance of a clean, dry foot environment using bandages. Bandages should be changed daily. At each bandage change, the foot is cleaned with a surgical scrub detergent followed by topical application of metronidazole or metronidazole combined with 10% benzoyl peroxide in acetone until the disease is resolved.[28,30] Debridement can be accomplished with the horse either standing, using regional local anesthesia and sedation, or under general anesthesia. In either case, a tourniquet should be applied at the level of the fetlock using a latex bandage or strip of rubber inner tube to control bleeding, since the affected tissue is highly vascular. This makes debridement much easier by allowing good visualization of the demarcation of normal and abnormal tissues. All necrotic, proliferative, and poorly cornified tissue should be removed down to normal-appearing corium using a scalpel, a sharp hoof knife, electrocautery, or laser ablation. Cryotherapy using liquid nitrogen has also been used, but great caution should be used to prevent destruction of the germinal epithelium.[29,30] Recent case studies[28,30] make it clear that because the infection is usually confined to the epithelium, debridement should be conservative to preserve the germinal layers of the epithelium necessary for optimal healing. The

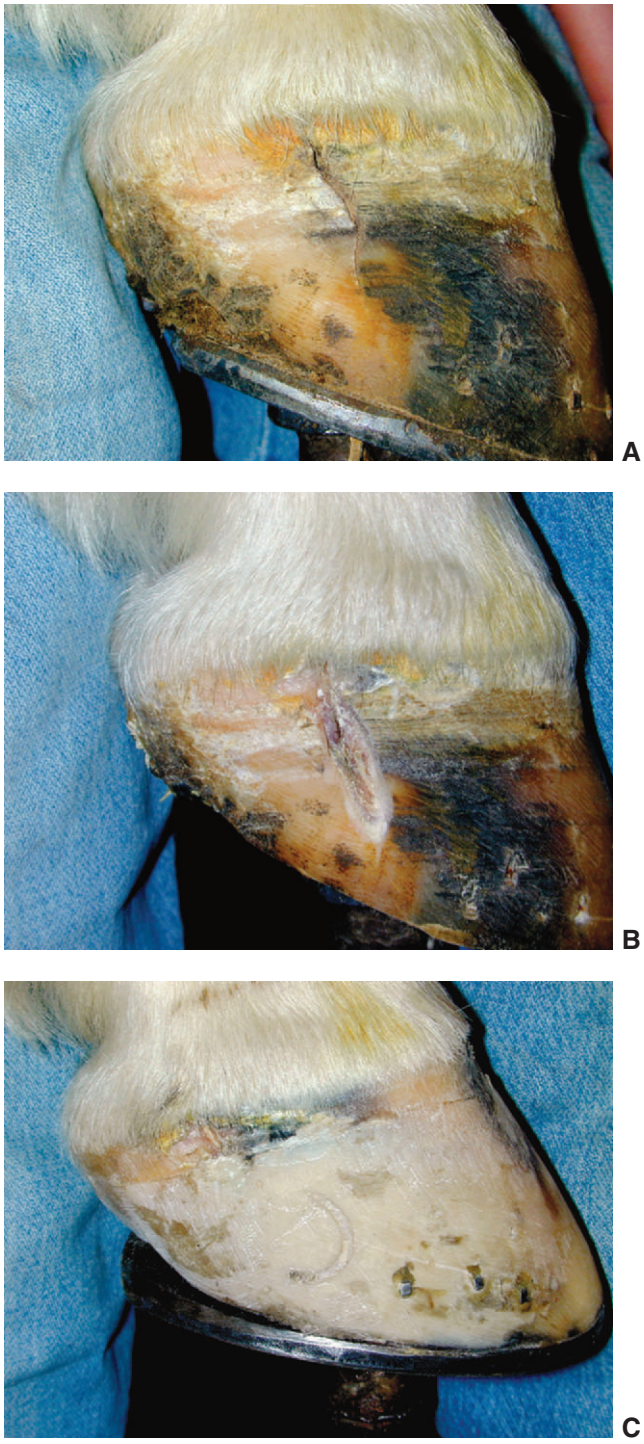


FIGURE 12-46 **A,** Appearance of a proximal quarter crack. **B,** Appearance of the crack after debridement. **C,** Appearance of the foot after composite repair and application of a bar shoe.

application of caustic agents is contraindicated for the same reason. Minor debridement should be repeated as necessary. Topical metronidazole, compounded as a 2% ointment, is used because of its efficacy against gram-negative anaerobic bacteria.[31] A solution of 10% benzoyl peroxide in acetone, combined with several crushed metronidazole tablets, has produced consistent success in 56 consecutive cases.[30] Systemic antibiotics, including oral trimethoprim sulfa and

metronidazole, have been used,[29,31] but topical therapy is usually sufficient to effect a cure.

Time to resolution averages about one month, and the prognosis for recovery is good since the replacement horn is usually functional, which is significantly better than previous treatment regimens that advocated aggressive debridement and the application of harsh antiseptics and astringents, which ultimately damaged the germinal tissues and delayed healing.[28]

WHITE LINE DISEASE

White line disease was reportedly first described in the mid-1980s,[16] and the first published article on this condition appeared in 1990.[32] Since then, investigators have been unable to identify a single definitive cause. The term *white line disease* is anatomically misleading, since the condition often extends proximal to the white line or white zone, which is the junction between the wall and the sole. It manifests as a progressive keratinolytic process within the deep layers of the stratum medium at the juncture of the stratum internum (stratum lamellatum) that results in degeneration and separation of the hoof wall. Fissures, cracks, and degeneration of the white line or white zone, which is normally yellow in color, and fraying and separation of the horny wall are usually visible on the solar surface of the hoof.

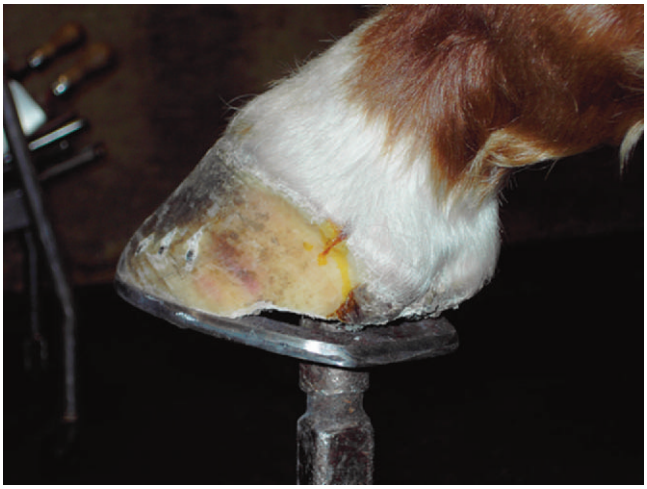
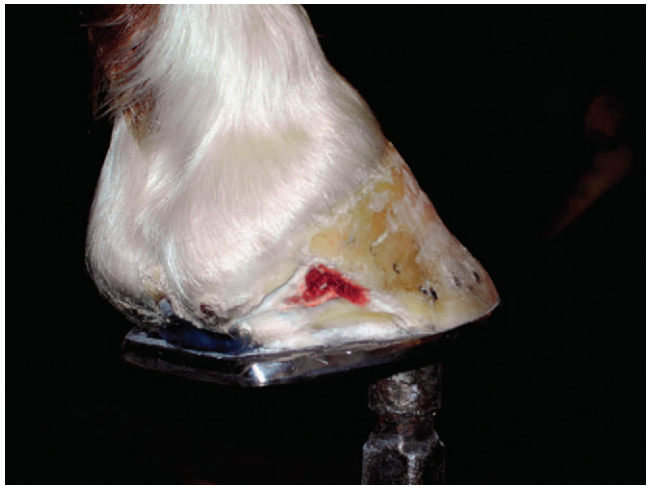


FIGURE 12-47 Proximal heel crack treated by floating the heel and applying a bar shoe in conjunction with impression material on the sole.

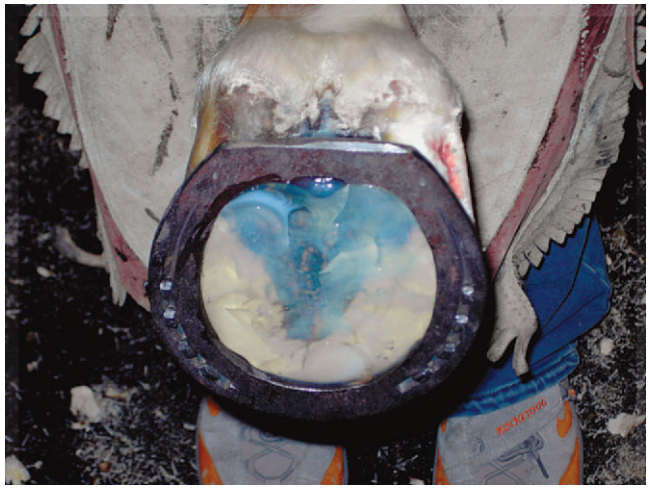
Etiology

Keratinolytic bacteria and fungi have been incriminated as the cause of white line disease, but given the number and variety of organisms isolated, they are probably secondary opportunists. In one study that examined the relationship between keratinopathogenic fungi and healthy and diseased hooves, 732 isolates were made from 187 horses. Of the 732 isolates, 452 were classified as nonpathogenic, 57 isolates were considered keratinopathogenic fungi, and 223 isolates were of uncertain pathogenicity. Of interest was that 80% of the samples from horses with hoof horn lesions (classified as poor horn quality, cracks, white line disease, brittleness, parakeratosis, and bruising) and 66.7% of the samples from horses with poor horn quality contained fungi from only the keratinopathogenic group, whereas only 8.9% of the samples from normal hooves grew keratinopathogenic fungi.[33] The predominate fungus isolated also appears to vary between regions of the world, with the genus *Scedosporium* most common in Japan[34] and *Scopularopsis* more commonly isolated in the United States and central Europe.[35]

The most likely scenario is that there is some predisposing factor, whether nutritional deficiency, environmental factors (excessive dry or wet conditions), or mechanical factors such as neglected overgrown feet, long toes, chronic laminitis, and hoof imbalances, that leads to damage to the hoof wall and associated white zone, with subsequent invasion and colonization by bacteria and fungi.[36,37] Once the bacterium or fungus becomes established, it is probable that it is a primary cause of progression. The term *onychomycosis* has been suggested for cases in which fungi are isolated[34]; however, the term is generally used to refer to a nailbed infection of the fingernails and toes in humans and the toes in dogs. In the horse, the hoof, although made up of keratin like the nails and



A



B

FIGURE 12-48 **A,** Appearance of a heel crack after removal of the caudal portion of the hoof wall by burring down to the stratum internum. **B,** A bar shoe with impression material is used to provide support.

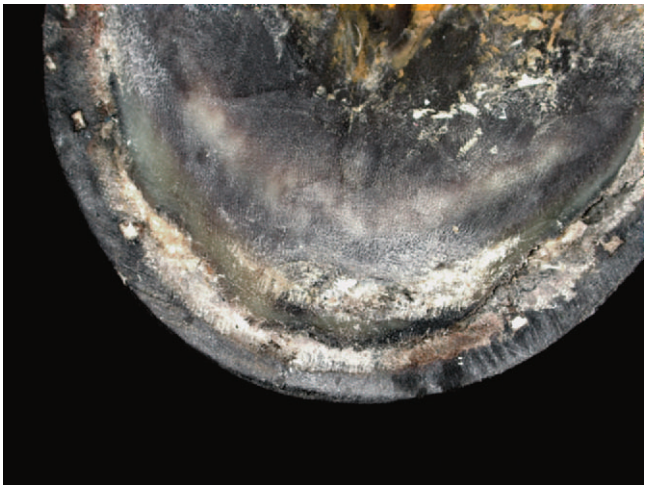


FIGURE 12-49 Characteristic appearance of local separation of the hoof wall and sole at the white line in the toe region.

FIGURE 12-50 Exploration of the extent of undermining of the wall is noted using a shoeing nail.

claws, has a uniquely different structure and the infection begins at the distal portion of the hoof rather that the germinative portion of the coronary band, so the term may not be entirely appropriate.

There does not appear to be any sex or breed predilection, but the incidence does appear to increase with age in some populations.[38] One or multiple hooves can be involved, but the disease more commonly affects the front feet. It is reportedly more common in hot humid areas of the United States,[32] presumably as a result of weakening of the white line from excessive moisture, allowing the entrance of foreign material. The toe and quarter regions are most often affected.

Pathology

Biopsies of the involved hoof sections show degeneration of the keratin and in some cases the presence of invading fungi.[39] Inflammation is absent because the problem is usually confined to the dead keratin of the hoof wall, although the occasional extension of the lesions into the dermis has been reported.[37]

FIGURE 12-51 Depth of undermining present at the toe.

Clinical Signs

White line disease is most commonly an incidental finding at the time of routine trimming and shoeing. It appears as a localized separation at the white line and further exploration with a probe or shoeing nail often reveals undermining of the hoof wall, which produces a characteristic hollow sound when the hoof wall is percussed with a shoeing hammer (Figures 12-49 to 12-51). The white line often appears softer and wider than normal, with a gray to black chalky appearance. Hoof tester response is generally negative and the horses are rarely lame from this condition unless the hoof wall separation has progressed to the point of causing instability of the pedal bone/hoof interface or the lesions have extended down into the sensitive laminae of the stratum internum.

Diagnosis

The diagnosis is usually made on the basis of the characteristic clinical signs. Fungal and bacterial cultures are of little value, since mixed populations of organisms are to be expected as part of the normal hoof environment. If the horse is lame or the separation is extensive, radiographs are indicated to determine the presence of P3 rotation. A series of oblique views and a true lateral view using a soft tissue radiographic technique will help delineate the extent of the involvement. The separation is easily recognized as an air density between the hoof wall and the dermis of P3 (Figure 12-52). Extensive undermining with rotation of P3 should be differentiated from laminitis. With white line disease, the lamina are not involved, so the degree of pain is less, and the lucent line visible on radiographs is filled with air and degenerated horn, not fluid, hemorrhage, or lamellar wedge tissue, which usually is present in cases of acute and chronic laminitis. The air lucency on radiographs is also closer to the hoof wall than in cases of laminitis, and it usually extends completely to the ground surface, which again is not characteristic of laminitis. The separation in white line disease can also be eccentric to the midline, depending on the location of the lesion.[32]

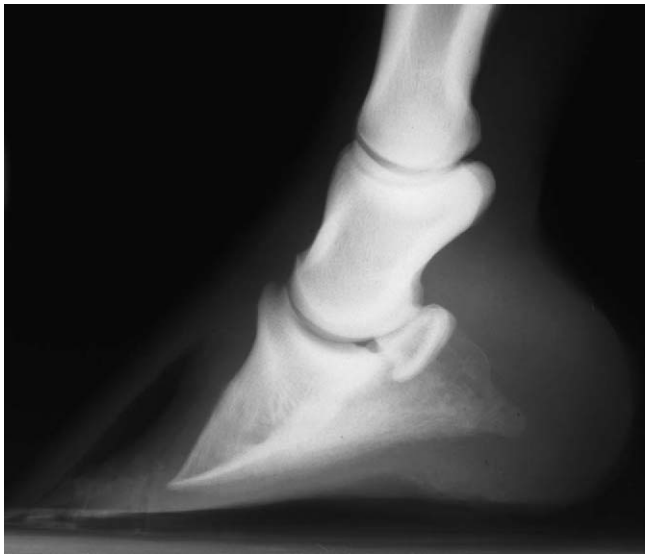


FIGURE 12-52 Lateral radiograph of the foot demonstrating the characteristic appearance of an extensive hoof wall separation at the toe that resulted in rotation of the third phalanx. Note the large air density.

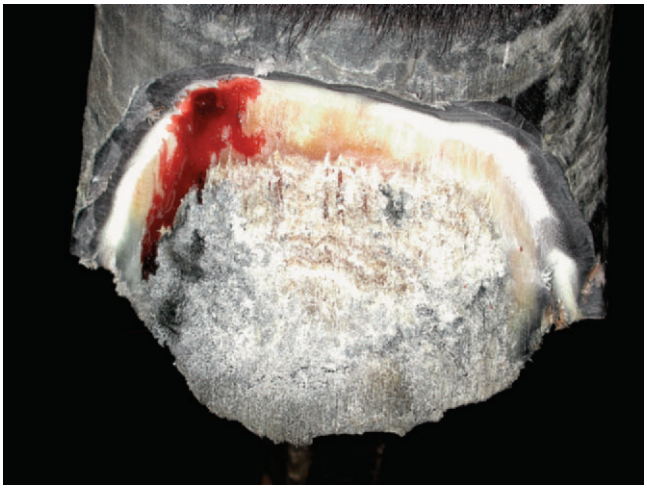


FIGURE 12-53 The foot shown in Figure 12-51 after complete debridement of the loose separated wall back to normal healthy tissue.

Treatment

Treatment consists of complete removal of all undermined hoof wall and thorough debridement and cleaning of all cracks and fissures. The goal is exposure and removal of all diseased hoof wall (Figure 12-53). The use of topical disinfectants and astringents such as merthiolate or 2% iodine is optional and does not seem to provide a distinct advantage over debridement and frequent cleaning alone. The staining effect of merthiolate may help to identify the extent of abnormal hoof within cracks and fissures when debridement is performed. Cleaning is facilitated by the use of a wire brush. Repeated debridement at the time of shoeing resets is often necessary. Success is signaled by the appearance of normal, healthy keratinized tissue produced by the underlying germinal epidermal cells of the stratum internum and the absence of any further undermining or fissures in the hoof wall.

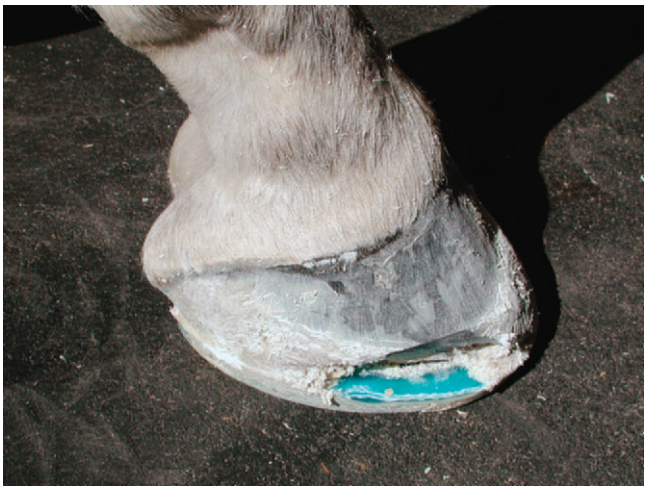


FIGURE 12-54 A portion of the hoof wall at the quarter was removed in this case, necessitating the use of a glue-on shoe.

FIGURE 12-55 All the diseased hoof has been removed and the defect is being allowed to grow out on its own.

The use of a shoe is usually indicated to protect the exposed sole and lamellar tissues, as well as the juncture of normal and resected hoof wall. Since most defects are present in the toe region, a shoe that moves the breakover back toward the frog as much as possible will provide protection from shearing stresses at the time of breakover. A bar shoe is indicated for stabilizing the hoof when large segments of the hoof wall are removed. Glue on shoes may be necessary if the condition of the hoof wall precludes the use of nails (Figures 12-54 and 12-55). If extensive undermining has allowed P3 rotation, shoeing should be performed as described for laminitis (see Chapter 18). If the disease has resulted in sufficient hoof wall separation to affect the stability of the underlying dermal structures and bone, then the horse should be rested until the hoof wall has grown out sufficiently. Alternatively, once the disease is completely eliminated and the resected portions of hoof wall have fully keratinized with normal-appearing hoof material, then the remaining hoof wall defects can be reconstructed with one of the available hoof acrylics. This will also allow an early return to work, although covering the defects with acrylic can delay recognition of recurrence of the problem and possibly exacerbate it.

Biotin supplementation at a dose rate of 0.12 mg/kg bodyweight for an extended period may be helpful in improving the quality of the horn that is produced. Providing a suitable environment, including a clean dry stall, and avoiding extremes of moisture is also important. The horse should not be turned out in wet inclement weather or early in the morning when the dew is heavy. Dry brittle hooves may benefit from the application of a lanolin-based hoof dressing. The prognosis for recovery is good in most cases, depending on the underlying cause, but recurrence is possible, even a year or two later.

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13
PATHOLOGIC CONDITIONS INVOLVING THE INTERNAL STRUCTURES OF THE FOOT

W. RICH REDDING

Lameness associated with structures inside the hoof capsule can be difficult to accurately diagnose. Careful clinical examination, including timed and sequential nerve and joint blocks, are necessary to isolate the area or site of pain. Advanced imaging (radiography, thermography, diagnostic ultrasonography, nuclear scintigraphy, computed tomography, magnetic resonance imaging) is then necessary to determine which structures may be involved. The appropriate treatment strategy for the specific condition and the individual horse is dictated by these findings. Clinical examination, diagnostic anesthesia, and diagnostic imaging of the digit are discussed in Section II.

SURGERY TO THE FOOT

With several of the conditions discussed in this chapter, surgical intervention can be an appropriate component of treatment. Some general comments on surgery to the equine foot are therefore in order. Any surgical procedure performed on the foot should follow a thorough diagnostic evaluation to ascertain that surgery is necessary. The fact that a procedure *can* be performed does not mean that it *should* be performed.

Once surgery is deemed appropriate in a particular case, related factors should be carefully evaluated before proceeding with surgery. The horse's temperament and the specific technique to be undertaken dictate whether the procedure can be performed with the horse standing or under general anesthesia. Many surgical procedures involving the foot can be performed in the standing horse. There is, however, a higher likelihood of contamination when foot surgery is performed with the horse standing, as surgical conditions are less controlled. Minimizing patient movement during the procedure helps minimize contamination. Even so, contamination of the environment with feces and urine can seriously compromise sterile technique. Experience of the surgeon and careful preoperative planning to minimize surgical time also are critical in avoiding complications during and after the surgery.

Chemical Restraint for Standing Procedures

Standing procedures require some form of patient restraint, usually a combination of physical and chemical means. The intensity of the surgical stimulus, technical assistance available, the facilities, and the expected duration of the procedure should dictate the type of restraint used. Chemical restraint can be provided by a variety of drugs and should be a routine part of every invasive procedure performed in the horse. Within the context of this discussion, with any procedure that will create surgical stimulation, the horse will benefit from the use of a drug or drug combination that has good analgesic and sedative properties.

The ultimate goal of chemical sedation is to optimize the drug's desirable effects while diminishing its detrimental effects. For instance, to achieve an adequate level of analgesia with xylazine or detomidine, the plane of sedation may have to be increased to the point of inducing unconsciousness. Also, with these alpha-adrenergic drugs, the level of sedation can be deceiving; horses can be aroused out of this sedative state and can place remarkably accurate kicks when stimulated. This fact necessitates a drug combination that optimizes the analgesic effects of xylazine and/or detomidine while providing an adequate level of sedation.

Minor invasive procedures of short duration can often be accomplished using a combination of xylazine (0.55 mg/kg IV) and acepromazine (0.05 mg/kg IV). If analgesia is insufficient with this combination, another dose of xylazine or the addition of butorphanol (0.05 mg/kg IV) frequently is all that is necessary.

If more intense pain is expected during a short procedure, then a combination of xylazine (0.5 to 1.1 mg/kg IV) and butorphanol (0.05 mg/kg IV) works well. With any procedure expected to take longer than 20 minutes, detomidine (0.01 to 0.04 mg/kg IV) should be used in place of xylazine. Detomidine is a longer-acting and more potent alpha-adrenergic receptor agonist than xylazine. (It should be kept in mind, however, that the analgesic effects of the alpha-adrenergics generally last only one half to two thirds as long as the duration of their sedative effects.) NOTE: When used in combination with butorphanol, the detomidine dose should be reduced to 0.01 mg/kg.

Addition of Regional Anesthesia

Whichever method of sedation is chosen, it is best in most procedures to supplement the analgesic effects of the intravenous drugs with regional anesthesia (nerve blocks and/or ring block). With the use of local anesthetics for surgical anesthesia, three points are important: (1) whenever possible, regional anesthesia should be used proximal to the surgical site instead of local infiltration at the surgical site, because local anesthetic can distort the tissues into which it is instilled and can potentiate infection; (2) if local infiltration must be used, then strict aseptic technique must be maintained when injecting; and (3) anesthetics that contain epinephrine should not be used, as epinephrine can also potentiate infection. It is advisable to use a new bottle of local anesthetic solution for

each surgery. Adding amikacin (250 mg of amikacin for each 10 mL of anesthetic) helps minimize contamination at the injection site.

Complete desensitization of the forefoot can usually be achieved with a palmar nerve block applied at the level of the proximal sesamoid bones (i.e., an abaxial sesamoidean nerve block). In the hindfoot, it generally is necessary to perform either a plantar digital nerve block at the level of the proximal sesamoids or a low six-point nerve block to completely desensitize the foot.

Regional anesthesia can also be used to augment general anesthesia. Doing so reduces the requirement for inhalant anesthetic agents and therefore the plane of anesthesia necessary to perform the surgery, and diminishing the total amount of inhalant used enhances recovery from anesthesia.

Preparation for Surgery

Preoperative antibiotics (e.g., procaine penicillin at 22,000 IU/kg IM q12h plus gentamicin at 6.6 mg/kg IV or IM q24h) should be administered 1 to 2 hours before surgery and continued for 24 hours postoperatively. Sterile preparation of the distal limb is accomplished with a 5-minute scrub using chlorhexidine gluconate (Hibiclens), followed by an alcohol rinse. It is advisable to scrub the injection site for 3 minutes before injecting the local anesthetic agent.

In most instances, placement of a tourniquet will help avoid excessive bleeding. The tourniquet can be as simple as a self-adhesive bandage (e.g., Vetrap [3M, St. Paul, Minn.]) wrapped around the fetlock to compress the digital arteries against the proximal sesamoid bones (Figure 13-1). While the tourniquet is in place, regional perfusion with an antibiotic can be performed for the treatment of contaminated wounds (see p. 278).

Other Considerations

The decision to perform surgery should be dictated by the necessity for surgical intervention, the horse's health, the specific procedure to be performed, and the comfort level and skill of the surgeon. To determine whether a procedure is suitable or appropriate, the surgeon needs to first consider the horse's health. Pregnancy, severe pulmonary abnormalities, excessively large horses (draft breeds), hyperkalemic periodic paralysis (HyPP), or any prior stress may preclude the use of general anesthesia and dictate that a standing procedure be performed.

The surgeon also needs to understand the expectations of the client and educate the client when these expectations are unrealistic. Understanding the client's expectations may direct the surgeon to do a more radical procedure for salvage of an animal or a more conservative procedure aimed at restoring soundness.

The skill and experience of the surgeon performing the procedure should ultimately dictate whether surgery is done in the field or at a referral surgical facility. Skill must be developed and is usually achieved by practicing a procedure first on cadaver limbs and thereafter on clinical cases. Developing a routine expedites most procedures.

Further, it is the surgeon's job to understand and assess the risk for complications and to educate the client about the likelihood of such complications. Anyone attempting to develop experience in surgical procedures should learn and understand the axioms "Surgery equals complications" and "Ignorance is bliss." The decision to perform a specific procedure should be weighed against the risk to the patient and the possibility for complications. The higher the likelihood for complications, the greater the need to refer the case to a surgical facility.

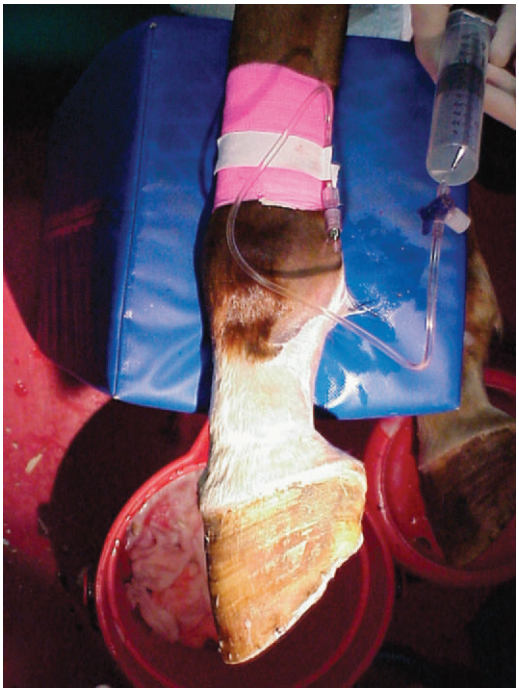


FIGURE 13-1 Placement of a tourniquet at the level of the sesamoid can be used for surgery or regional perfusion of the digit.

Postoperative care of the horse is one of the most important aspects of surgery. Relying on the client to recognize and manage problems that occur postoperatively is asking for trouble. The best way to manage problems is to be proactive and prevent them. Complications occur despite good technique, but the surgeon should work hard to prevent complications. It is usually the experienced surgeon, who has had to manage many types of complications, who is most aggressive at preventing them. This experience allows the surgeon to more accurately predict the short-term outcome as well as the long-term success.

NONINFECTIOUS JOINT DISORDERS

In the following sections, noninfectious processes that cause clinical problems involving the proximal or distal interphalangeal joints are discussed. Septic arthritis is covered in the later section on Infectious Processes.

Medical Therapy for Noninfectious Joint Disease

Treatment for noninfectious joint disease often involves the administration of antiinflammatory or chondroprotective agents, or both. Options include the following:

- Nonsteroidal antiinflammatory drugs (NSAIDs), such as phenylbutazone and flunixin meglumine; these agents usually are given systemically (orally or intravenously), although some are available in topical formulations
- Corticosteroids, such as triamcinolone (Vetalog) and methylprednisolone (Depo-Medrol); these agents usually are given intraarticularly
- Hyaluronic acid (e.g., Legend), either intraarticularly or intravenously (q7d for three treatments)
- Polysulfated glycosaminoglycans (PSGAGs; e.g., Adequan), either intraarticularly or intramuscularly (q5d for seven treatments)

Inflammation of a joint can be managed medically with one or a combination of these products. There is a risk for complications with the long-term systemic use of NSAIDs, so only a short course of therapy is usually recommended with these drugs. Joint inflammation may be more effectively managed with intraarticular administration of an antiinflammatory/chondroprotective agent (corticosteroid, hyaluronic acid, or PSGAG).

Each of these drugs has a place in intraarticular medication of the joint, but corticosteroids are the most potent; they have such a profound effect that they should almost always be included. Most clinicians prefer to use a combination of hyaluronic acid (20 to 40 mg per joint) and triamcinolone (6 to 9 mg per joint). Whenever a corticosteroid or PSGAG is used as part of an intraarticular injection, an antimicrobial agent (e.g., amikacin, 125 to 250 mg) should be added to decrease the risk of joint infection.

Synovitis of the Distal Interphalangeal Joint

Primary synovitis of the distal interphalangeal (DIP; or coffin) joint is a common problem in athletic horses.[1] It most often involves the forelimbs and can affect one or both feet. Arthrosis of the DIP joint is reported as a significant cause of lameness in racing Quarterhorses.[2] Repeated concussion has been suggested as an underlying cause in these and other athletes. Craniocaudal or lateromedial imbalance of the foot may be a contributing factor in many cases. The surface on which the horse trains can also influence the severity of the clinical signs.

Many injuries that occur in racehorses are caused by hyperextension of the joints, particularly in the forelimbs. It seems likely that hyperextension might also be a factor in synovitis of the DIP joint. In addition to hoof imbalance, many affected horses are shod with toe grabs, which effectively lengthen the toe and predispose to hyperextension of this joint.

Clinical Findings

The lameness associated with synovitis of the DIP joint seems to manifest insidiously and most times is not severe. Clinical examination usually reveals palpable effusion in the dorsal pouch of the DIP joint (just proximal to the coronary band, on either side of the extensor tendon). Most affected horses have moderate foot imbalance. A complete lameness examination, including distal limb flexion, extension stress test, and careful hoof tester assessment, is warranted to correctly diagnose this problem. A flexion test of the digit may be moderately positive. Note: Acute, severe lameness with rapid development of DIP joint effusion should be evaluated carefully for more severe problems such as articular fractures, sepsis, or severe collateral ligament injury.

Diagnosis

Intraarticular anesthesia of the DIP joint rapidly eliminates the lameness in most cases. The severity of the synovitis usually is reflected by changes in the synovial fluid, which can range from more cellular (cloudy) than normal, to fibrinous and possibly even hemorrhagic.

Radiography Radiographic examination of the foot is indicated to rule out other causes of pain that may be blocked by intraarticular anesthesia. In the early stages of this condition, however, when synovitis is considered the only problem, there typically are no significant radiographic changes.

It has been speculated that the synovial infoldings of the DIP joint that are seen radiographically as larger-than-normal radiolucent shapes along the distal margin of the navicular bone are associated with arthrosis of the DIP joint. If that is true, then it calls into question the cause and significance of radiographically apparent changes at the distal border of the navicular bone (often diagnosed as navicular disease) in young racehorses.

Ultrasonography Diagnostic ultrasonographic examination can be helpful in assessing many joint injuries, because synovial effusion is easily identified sonographically. However, the hoof capsule does not transmit sound and so precludes sonographic examination of much of the DIP joint. The window to the DIP joint is limited to the soft tissue around the coronary band and to the distopalmar recesses in the pastern (between the collateral cartilages).

Although thickening of the thin synovial membrane (i.e., synovitis) is difficult to appreciate sonographically, careful evaluation of the joint capsule can be useful in demonstrating capsular thickening or damage. The redundancy of the joint capsule should be eliminated during the examination by altering the limb position. For example, the dorsal aspect of the joint should be examined while the digit is flexed, which tenses the dorsal portion of the joint capsule.

Magnetic Resonance Imaging Magnetic resonance imaging (MRI) is a very sensitive tool for evaluating the DIP joint and its soft tissues. Schneider et al.[3] reported on the clinical presentation and MRI findings for more than 150 horses that underwent MRI of the distal limb. An increase in the amount of synovial fluid in the DIP joint was considered a sign of synovitis/arthritis. Interestingly, most of the horses with synovial effusion also had abnormal findings in the navicular bone or its supporting soft tissues.

Differentiating DIP joint synovitis from early navicular disease is difficult and may not be possible with standard imaging techniques. It is hoped that MRI will add to understanding of these disease processes and any relationship between them. It may well be that the same forces that create navicular changes also lead to DIP joint arthritis.

Treatment and Prognosis

Primary synovitis can progress to osteoarthritis if the underlying cause is not resolved and the insult to the joint continues. Treatment is therefore directed at correcting any shoeing abnormalities and foot imbalance. Corrective shoeing is greatly assisted by referring to a complete radiographic study of the digit, to assess the position of the third phalanx

(P3) within the hoof capsule and to carefully evaluate the DIP joint and navicular bone. If necessary, intraarticular injection of hyaluronic acid and triamcinolone can be very effective at reducing the acute inflammatory response (see p. 255).

If the horse responds to shoeing adjustments and does not require repeated intraarticular injections to control the inflammation, then the prognosis is reasonably good. However, if the shoeing adjustments and injections are not effective over the long term, then the prognosis is guarded for the horse in its present discipline, and a change to a less stressful discipline may be indicated.

Osteoarthritis of the Distal Interphalangeal Joint

Osteoarthritis of the DIP joint is a degenerative disease that can occur secondary to a number of different problems.

Causes and Contributing Factors

In young horses, osteoarthritis can occur secondary to osteochondrosis (see the following section on osseous cyst–like lesions). In any horse, it can result from a traumatic injury that causes significant instability of the joint, such as severe damage to a collateral ligament. Over time, any horse can develop osteoarthritis as a consequence of "use trauma." Poor conformation can accelerate degenerative changes that result from osteochondrosis, damage to the joint's supporting structures (e.g., tearing of the joint capsule, collateral ligaments, or other tendon or ligamentous structures), or repetitive use by overloading parts of the articular surface.

Hyperextension Injuries In horses that perform at speed, hyperextension of the DIP joint can create osteochondral fractures on the dorsal surface of the joint. Hyperextension may also damage the palmar/plantar soft tissues, including the deep digital flexor tendon, suspensory ligaments of the navicular bone, and distal sesamoidean impar ligament. These injuries can significantly affect the prognosis for return to performance because they predispose to osteoarthritis of the DIP joint.

Intraarticular Injections Intraarticular injection of corticosteroids (especially methylprednisolone), when repeated too frequently, may lead to degenerative changes in the articular cartilage. This problem may be more likely in joints that also have osteochondral fragmentation of the articular surface.

Any intraarticular injection carries an inherent risk for infection. The DIP joint is probably at higher risk for infection than most other joints because of its proximity to an environment inherent with fecal contamination. Thus, great care must be taken when performing intraarticular injection of the DIP joint. Unfortunately, iatrogenic infections frequently involve *Staphylococcus aureus* (a bacterium that usually is indicative of human contamination), which can be particularly damaging to articular cartilage. Corticosteroids and PSGAGs carry a higher risk for infection than most other substances given by intraarticular injection, as both cause immunocompromise within the joint.

Clinical Findings

Early cases of DIP joint osteoarthritis can show joint effusion and lameness, which resolves with rest but returns with exercise. Intraarticular medication can be very effective in controlling the inflammation associated with DIP joint synovitis and early osteoarthritis, but it can have adverse effects if the inflammatory insult is not resolved. Thus, progression of degenerative changes in the joint should be suspected when the treatment regimen for primary synovitis becomes less effective over time or the duration of clinical efficacy decreases (i.e., the frequency between injections is decreasing).

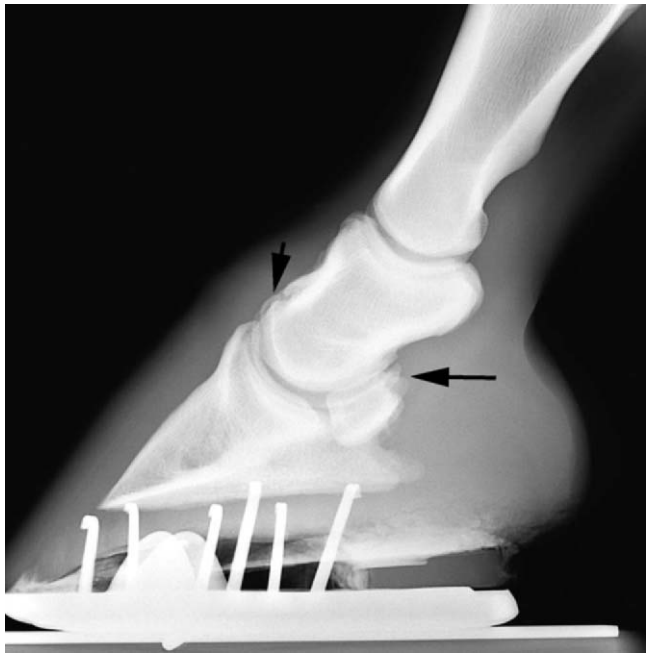


FIGURE 13-2 Radiographically apparent degenerative joint disease of the coffin joint arthritis is manifested by osteophyte formation on the distodorsal aspect of P2 or at the proximal border of the navicular bone.

Diagnosis

Radiography Whatever the cause, osteoarthritis leads to thinning of the articular cartilage, particularly in areas of high load. This cartilage thinning can be appreciated radiographically as narrowing of the joint space. Along with cartilage thinning, periarticular remodeling creates osteophytes and/or enthesiophytes at the joint margins. These changes are most often seen on the distodorsal and palmar aspects of the second phalanx (P2) and on the proximal surface of the navicular bone (Figure 13-2). Unfortunately, narrowing of the joint space and periarticular osteophytes and enthesiophytes can be seen radiographically only in the more advanced stages of disease. Joint capsule thickening, with or without metaplasia, also is a common feature of osteoarthritis; however, these changes are not apparent radiographically.

Ultrasonography Sonographic evaluation early in the course of a joint problem can help to identify the early changes that occur with osteoarthritis that may not be evident radiographically. Calcification at the joint capsule attachments often is the earliest sonographic indication of osteoarthritis. Cartilage thinning can be difficult to appreciate sonographically. A study in humans showed that ultrasonography is useful in identifying early osteoarthritis of the knee.[4] Changes included a decrease in cartilage thickness and blurring of cartilage margins, which made cartilage measurements less precise. In

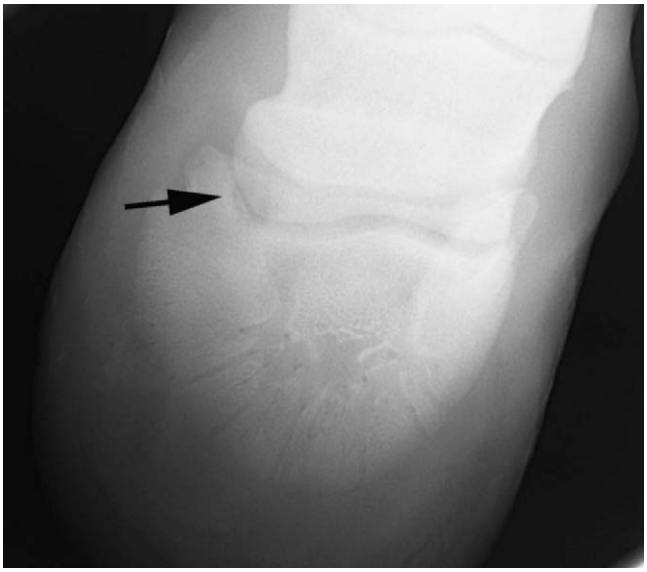


FIGURE 13-3 Subchondral bone cyst in the third phalanx (*arrow*). This cyst is located at a more abaxial position than usual.

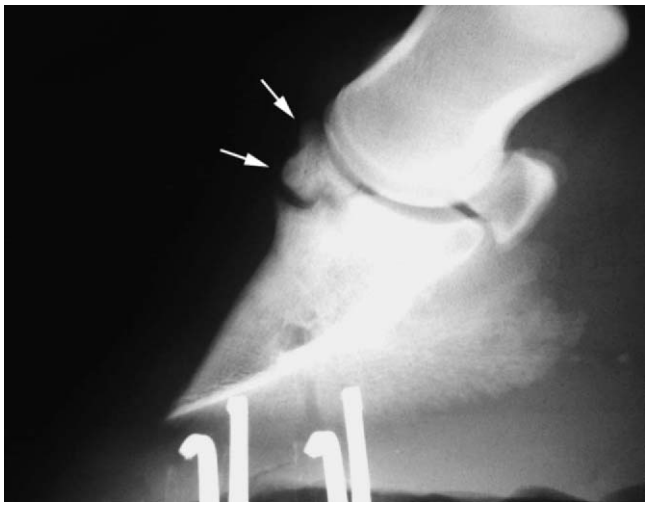


FIGURE 13-4 Extensor process fracture in a young horse. In some cases, subchondral bone cysts are associated with these large extensor process fractures.

general, ratings of clarity and sharpness correlated better with clinical status than did absolute thickness of the articular cartilage.

Other Imaging Modalities Nuclear scintigraphy can be helpful in identifying the DIP joint as a source of lameness. Degenerative joint disease, however, may not be readily apparent, as bone remodeling may be minimal in this joint, especially in sedentary adult horses. The definitive imaging modality in this situation is MRI. It may show generalized irregularity of the cartilage surfaces and loss of signal within the articular cartilage.

Treatment and Prognosis

Treatment of osteoarthritis involving the DIP joint depends on the extent of degenerative changes in the joint as well as the expectations for performance. Intraarticular injections of corticosteroids and/or PSGAGs may be effective for early osteoarthritis. When the changes are too advanced and intraarticular injections are no longer effective, arthrodesis may be worth considering. However, arthrodesis is primarily for salvage; most often the goal merely is sufficient soundness for breeding or comfort at pasture (i.e., pasture soundness).

Osseous Cyst–Like Lesions of the Distal Interphalangeal Joint

Subchondral bone cysts have been reported in many locations in the horse's body, including P3 (Figure 13-3). In most instances, they are not true cysts because they do not have a secretory lining; thus they are more appropriately called osseous cyst–like lesions. Depending on the stage of development, they may have a well-demarcated rim of sclerotic bone and a cavity that, on gross analysis, is filled with fibrous tissue.

Within P3, the most common site for these lesions is in the subchondral bone on or near the midline. They can also be found in the extensor process, where they may be associated with fracture of the extensor process (Figure 13-4). The cyst can communicate with the joint space via a narrow neck of variable size.

The cause of this lesion is unclear, but trauma and osteochondrosis have both been proposed. It is thought that there must be some form of primary cartilage damage in conjunction with subchondral bone damage that occurs from a single traumatic incident or from some form of repetitive injury. Primary cartilage damage can occur due to defects in endochondral ossification associated with osteochondrosis. The retained cartilage may develop avascular necrosis, and subsequent bone resorption may lead to cyst formation. Synovial fluid is thought to be mechanically pumped into the subchondral defect, which remodels into a cystic structure.

Alternatively, an injury that causes subchondral bone damage can lead to bone necrosis, which may result in secondary collapse of the overlying articular cartilage. These cysts frequently develop at sites of maximal load on the articular surface, which lends support to this theory.

Clinical Findings

The clinical presentation typically involves a unilateral, acute, moderate to severe lameness. Occasionally, the cyst is an incidental finding in a mature animal. When lameness is present, an intraarticular block of the DIP joint usually is effective in diminishing or eliminating the lameness.

Diagnosis

Radiographic examination, including special projections as needed, should be performed to identify the cystlike lesion and to demonstrate its precise location, size, and communication with the joint space. Contrast arthrography may be required to confirm communication between the cystic lesion and the joint space. In some cases, fluoroscopy has been more effective than routine radiography at defining the cyst location and dimensions because the x-ray beam can be moved in real time during the examination.

Ultrasonographic examination of cystic lesions in the DIP joint has been unrewarding because of the limited window available to visualize the articular surface of P3. Nuclear scintigraphy is helpful in defining the extent of bone turnover associated with the lesion. However, long-standing cases

may not have high bone turnover and therefore the lesion may not be apparent on bone scan.

Treatment

Treatment options are limited. Conservative therapy is directed at managing the associated synovitis that results from the expulsion of inflammatory debris from the cyst into the joint space. Therapy includes intraarticular corticosteroids (triamcinolone) and hyaluronic acid. However, the duration of effect is variable and unpredictable. The frequency of repeat injection should be carefully monitored; injection should not be repeated indiscriminately, because repeated intraarticular use of corticosteroids can precipitate or accelerate osteoarthritis.

Surgery Surgical management is dictated by the accessibility of the cyst cavity. If the cyst is thought to be accessible, then an arthroscopic approach can be attempted. Extensor process fractures associated with these cystic lesions can usually be accessed arthroscopically. Cystic lesions in the central portion of P3 can occasionally be accessed arthroscopically, but access to this area usually is limited. In those cases, a more aggressive surgical approach may be required in which the cyst is accessed by drilling through a hole created in the hoof wall. This procedure is called *extracapsular cyst enucleation*.

Intralesional Corticosteroids Some clinicians have recently proposed that injecting corticosteroids directly into the cystic lesion may be as effective as surgical intervention, without some of the potential complications of surgery. If the cystic opening can be visualized sonographically, then guiding a needle into the cyst may be possible. If the cyst is not visible with the use of diagnostic ultrasonography, then arthroscopic visualization for needle placement may be necessary to direct corticosteroid injection.

Prognosis

The prognosis for subchondral cystic lesions is somewhat unpredictable. These lesions can be found incidentally in mature horses that have no history of lameness. On the other hand, young horses with these lesions that present with acute lameness localized to the DIP joint should be followed carefully. In most instances, these horses are candidates for surgical or intralesional therapy. The prognosis then is influenced by the accessibility of the cyst to treatment. In any horse with these cystic lesions, concomitant degenerative changes in the DIP joint carry a poor prognosis.

Osteoarthritis of the Proximal Interphalangeal Joint

The proximal interphalangeal (PIP; pastern) joint is mentioned here for two reasons. First, the PIP joint may be partially or completely desensitized by anesthesia of the digital nerves, owing to retrograde flow of anesthetic along the neurovascular bundle. If the block improves or resolves the lameness and the possibility of a problem involving the PIP joint is not considered, then the results of a palmar digital nerve block may be misinterpreted. It is therefore important to isolate problems involving the PIP joint using intraarticular anesthesia.

Second, the PIP joint can be damaged in wounds involving the digit. It is close to the hoof and to the DIP joint and for most of its circumference it has minimal soft tissue cover. This possibility must be considered and the PIP joint carefully evaluated to ensure appropriate treatment and a successful outcome.

Causes and Contributing Factors

The causes of osteoarthritis of the PIP joint are similar to those discussed for osteoarthritis of the DIP joint. (NOTE: Primary synovitis of the PIP joint is not recognized as a clinical entity, probably because this joint has a lower range of motion than the DIP joint.)

The most common causes of osteoarthritis of the PIP joint are osteochondrosis, infection, and traumatic injury (including fracture of the proximal or middle phalanx). Wounds to this area are common and frequently involve the PIP joint (Figure 13-5) and/or the digital flexor tendon sheath. (When either structure is contaminated, severe lameness results and the situation requires aggressive treatment; see p. 260) In some Quarterhorse lines, there appears to be a heritable component to osteoarthritis of the PIP joint.

Osteochondrosis in the PIP joint is manifested either as osseous cyst–like lesions or as osteochondral fragmentation. Either type of lesion can occur at the distal end of the first phalanx (P1) or at the proximal end of P2. Osteochondral fragmentation is discussed separately in the next section. Osseous cyst–like lesions are discussed here because of their propensity to cause osteoarthritis.

Clinical Findings

Mild, intermittent lameness can be seen with early osteoarthritis, whereas the lameness may be severe with advanced osteoarthritis. Clinical examination usually allows localization of the lameness to the pastern. Contour changes are easily seen around this joint because there is minimal soft tissue cover. Distal limb flexion usually is positive.

Diagnosis

Palmar digital nerve blocks occasionally alleviate lameness associated with the PIP joint. More often, an abaxial sesamoidean (i.e., low palmar) nerve block or a low four- or six-point regional nerve block may be necessary to eliminate the lameness. (These blocks are discussed in Chapter 9.) Intraarticular anesthesia is more accurate in identifying the PIP joint as a site of lameness and should be used to confirm that this joint is a problem.

Radiography Radiographic changes are similar to those described earlier for osteoarthritis of the DIP joint. Osteoarthritis of the PIP joint usually is associated with new bone production on the dorsomedial and dorsolateral aspects of the joint (Figure 13-6). Fractures of the medial or lateral palmar/plantar tuberosities on the proximal aspect of P2 are evident radiographically, with appropriate positioning; they may also create palpable contour changes at the back of the joint.

Radiolucent areas in the center of the articular surface at the distal end of P1 are a relatively common radiographic finding and are thought to be an incidental finding. In contrast, cystlike lesions in the medial or lateral condyles are more likely to be clinically relevant, particularly if the cystic cavity appears to communicate with the joint space (Figure 13-7). Many of these cysts are not diagnosed until there is advanced osteoarthritis. Some of these osseous cyst–like lesions do not communicate with the joint, but they may still cause lameness.

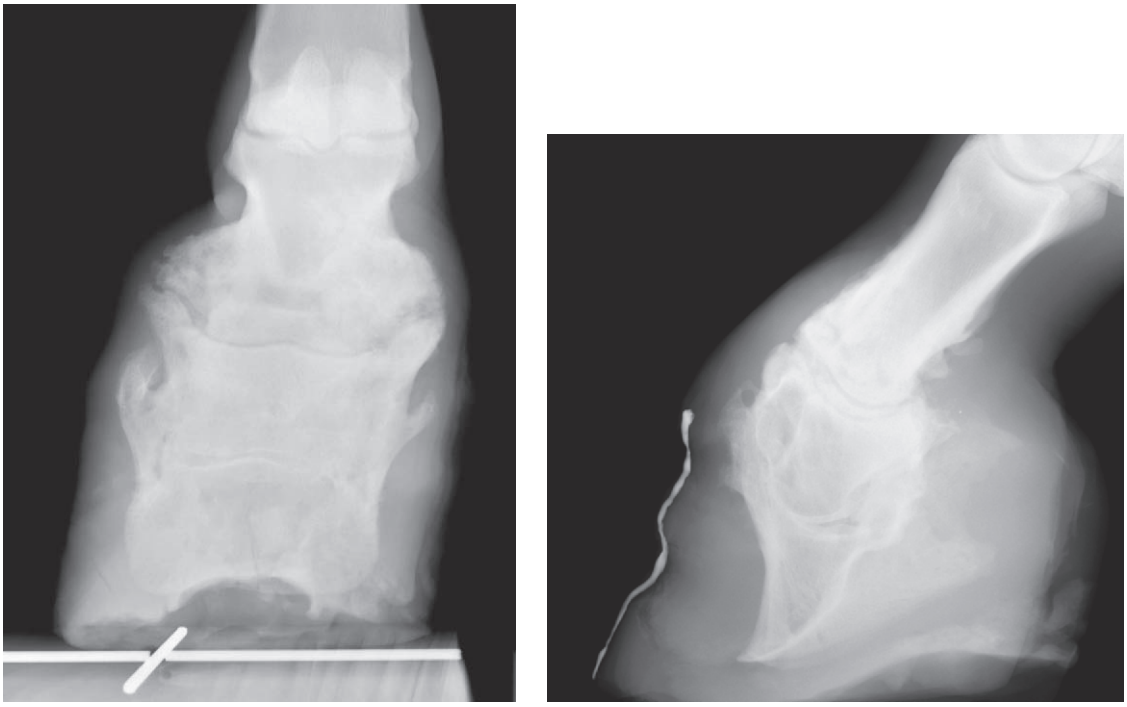


FIGURE 13-5 Severe degenerative joint disease of the proximal and distal interphalangeal joint associated with a wound to the pastern.

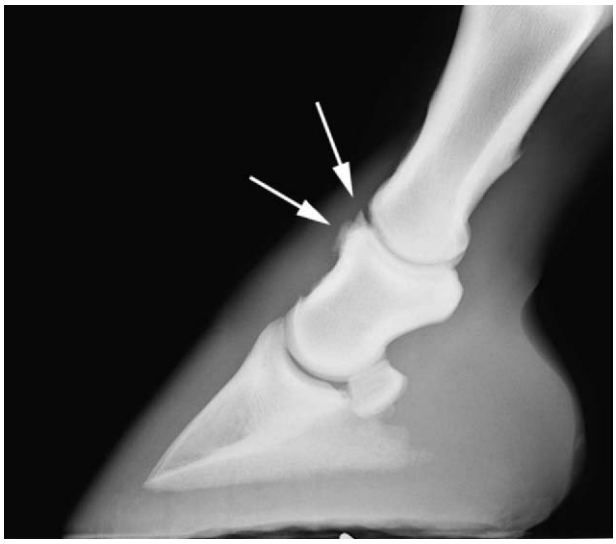


FIGURE 13-6 Degenerative joint disease of the pastern joint.

Treatment

Intraarticular injection of corticosteroids or PSGAGs, or both, may help in the short term, but some cases will still progress to severe osteoarthritis. Nonresponsive cases are candidates for surgical arthrodesis of the PIP joint. Several arthrodesis techniques have been reported, but currently the most common procedure involves the application of a four-hole, narrow dynamic compression plate, in combination with two parallel transarticular screws, placed in lag fashion across the joint (Figure 13-8). The third screw in the plate also is positioned so that the screw crosses the joint in lag fashion. The patient is placed in a half-limb cast; the cast is changed after 2 weeks and is removed after another 2 weeks.

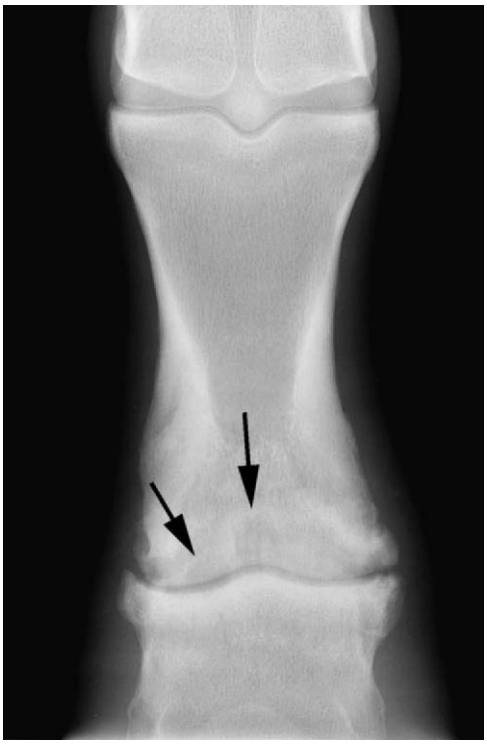


FIGURE 13-7 Osseous cyst–like lesions in the proximal interphalangeal joint with severe degenerative joint disease.

Osseous cyst–like lesions that do not communicate with the joint space may be candidates for extracapsular enucleation. Under fluoroscopic or radiographic guidance, a hole is drilled through the cortex of the bone into the cyst, and the cavity is then curetted. It is also possible to inject these cystic lesions with corticosteroids.

Prognosis

The prognosis for soundness without surgical arthrodesis is guarded, so surgery is recommended in most cases. Return to soundness can take up a year after the procedure, however. The prognosis for soundness after arthrodesis is better for hindlimbs than for forelimbs.

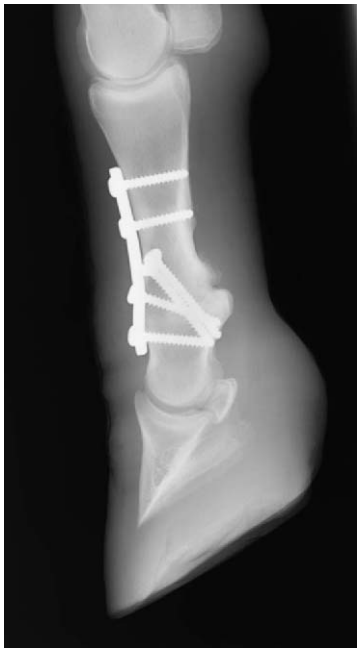


FIGURE 13-8 Arthodesis of the proximal interphalangeal joint with a four-hole narrow dynamic compression plate and two transarticular screws.

Osteochondral Fragmentation of the Proximal Interphalangeal Joint

Osteochondrosis involving the PIP joint frequently is associated with lameness. Osseous cyst–like lesions and osteochondral fragmentation can occur on either articular surface (P1 or P2).[5] Osseous cystlike lesions are discussed in the preceding section because they frequently cause osteoarthritis, which requires surgical arthrodesis to restore soundness.

Clinical Findings and Diagnosis

Osteochondrosis with fragmentation causes variable degrees of lameness. The condition can be bilateral, although the lameness may be worse in one limb. Most horses are mildly lame in the worst-affected limb and have some swelling (joint effusion) associated with the clinically affected joints. Distal limb flexion tests may yield positive results, particularly on the affected side. In most cases, the lameness is alleviated by intraarticular anesthesia of the PIP joint. These fragments are readily apparent on a routine radiographic study of the pastern (Figure 13-9).

Treatment

Horses with mild lameness can be treated in the short term with intraarticular medications (see p. 255). In cases with more severe lameness, and those in which the lameness is unresponsive to intraarticular therapy, arthroscopic removal of the osteochondral fragment should be considered.[6,7] Osteoarthritis is a possibility if the fragments are left in place.

Prognosis

Few clinical cases have been reported, but those that were amenable to arthroscopic removal of the fragments became sound. Larger fractures of the palmar/plantar eminences can sometimes be repaired with lag screws, but most of these cases are candidates for arthrodesis of the PIP joint.

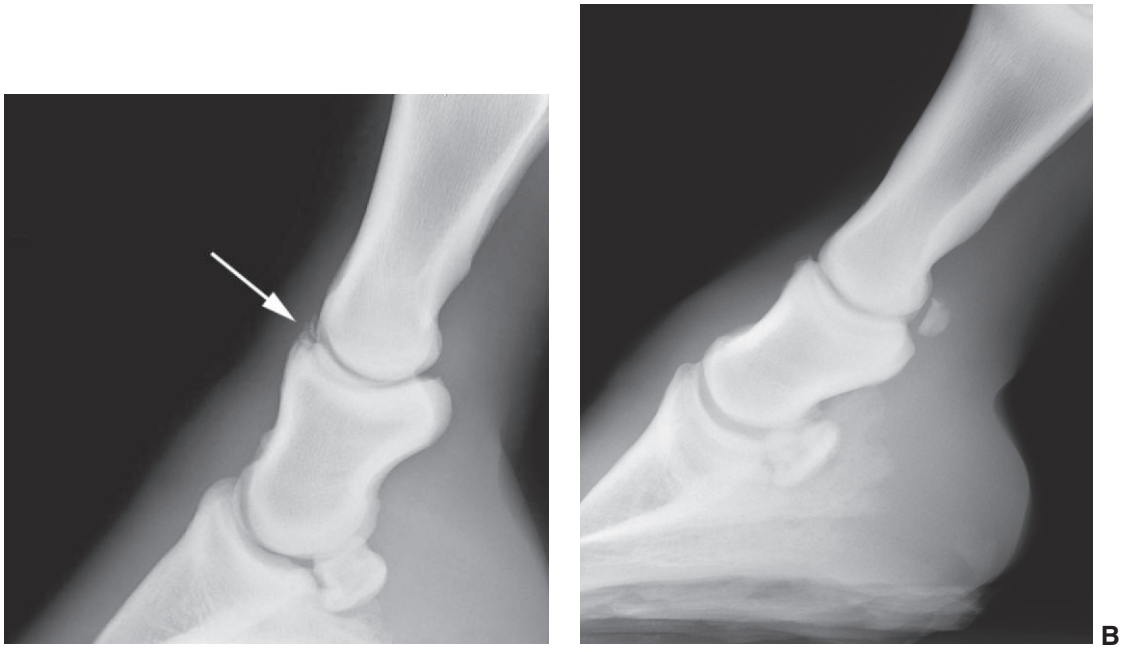


FIGURE 13-9 Osteochondral fragmentation of the second phalanx (P2). **A,** Dorsal P2. **B,** Palmar P2.

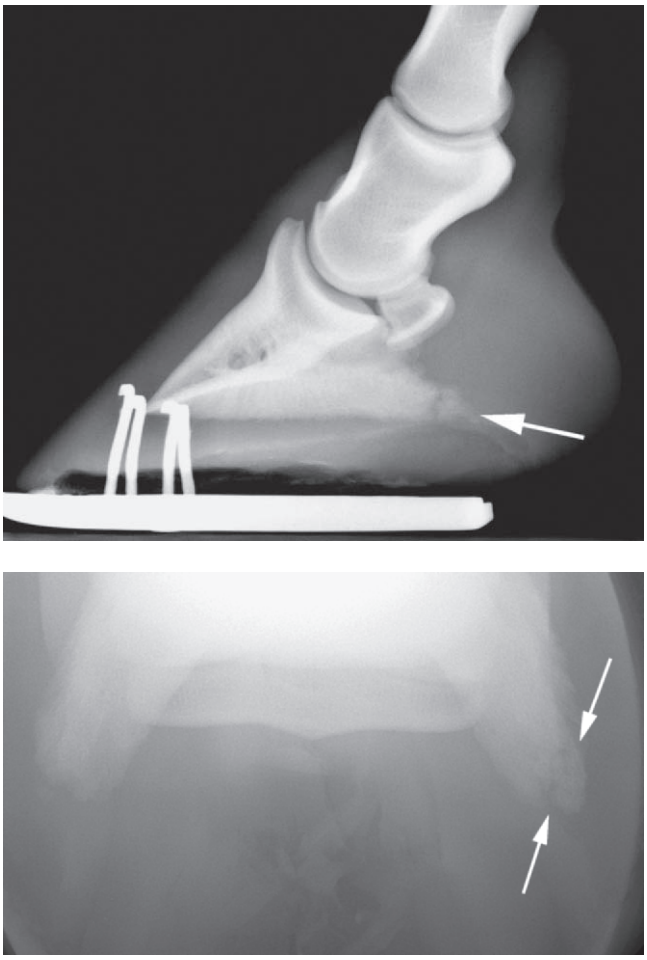


FIGURE 13-10 Type I fracture of the third phalanx *(arrows)*.

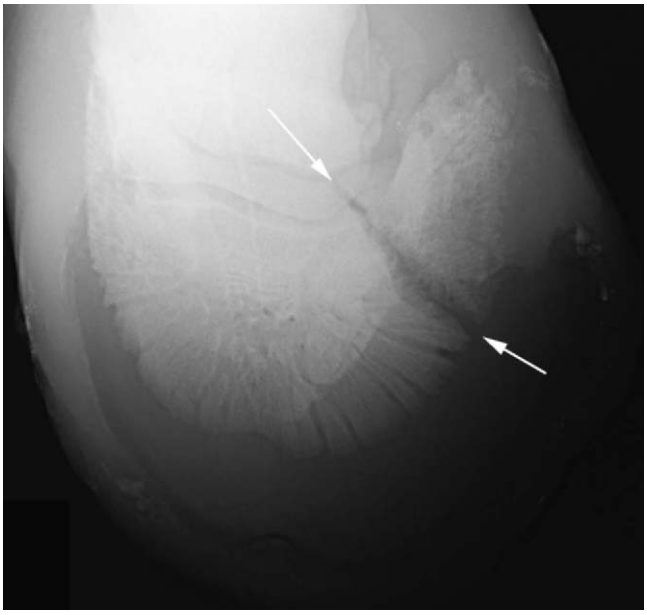


FIGURE 13-11 Type II fracture of third phalanx (P3)—the most common distal phalanx fracture in the horse.

BONE DISORDERS

Fractures of the Third Phalanx

Fractures of the distal, or third, phalanx are relatively uncommon. Overall, these fractures are most common in racehorses, particularly Thoroughbreds and Standardbreds. The age, breed, and athletic use of the horse play significant roles in determining which type of P3 fracture is most likely to occur. Laminitis, pedal osteitis, osteomyelitis, and unbalanced trimming or shoeing can predispose to P3 fractures. Behavioral problems, such as pawing or kicking the stall wall, may also predispose an individual horse to one of these fractures.

Fracture Types

Fractures of P3 are classified into one of seven different types, according to fracture configuration:

- Type I—nonarticular fracture involving the palmar/plantar process (or wing) of P3 (Figure 13-10)
- Type II—sagittal articular fracture, extending obliquely from the DIP joint to the solar margin on the medial or lateral side of P3 (Figure 13-11)
- Type III—midsagittal articular fracture that divides P3 into approximately equal halves (Figure 13-12)
- Type IV—osteochondral fragments off the extensor process of P3; these fractures involve the joint surface (Figures 13-13)
- Type V—comminuted fracture, usually secondary to osteomyelitis of P3 (which most often is a sequela of laminitis) (Figure 13-14)
- Type VI—nonarticular fracture at the solar margin of P3
- Type VII—nonarticular fracture at the solar margin, specifically in the palmar/plantar process of a foal (Figure 13-15)

Overall, type II fractures are the most commonly diagnosed P3 fracture configuration.

General Clinical Findings

Clinical signs associated with P3 fractures are not specific for the fracture type. The associated lameness ranges from mild to severe. With the more substantial fractures (e.g., types II and III), the lameness may progress after the initial injury as hemorrhage and inflammation increase swelling within the rigid hoof capsule. With articular fractures, the lameness frequently progresses to severe, non–weight-bearing lameness (grade 5/5).

An increased digital pulse typically is found in the affected limb, along with hoof tester sensitivity around the fracture site. Some horses are sensitive across the entire foot because of the intense swelling within the hoof capsule. In other cases, the horse may not respond to hoof testers at all, particularly if the fracture is small and the horse has a hard, thick sole. If the fracture involves the DIP joint, then manipulation of the digit may elicit a moderate pain response.

General Diagnostic Protocol

Radiographic examination is indicated in any case of lameness associated with the foot. Familiarity with the complex anatomy of the foot and the range of normal variations seen

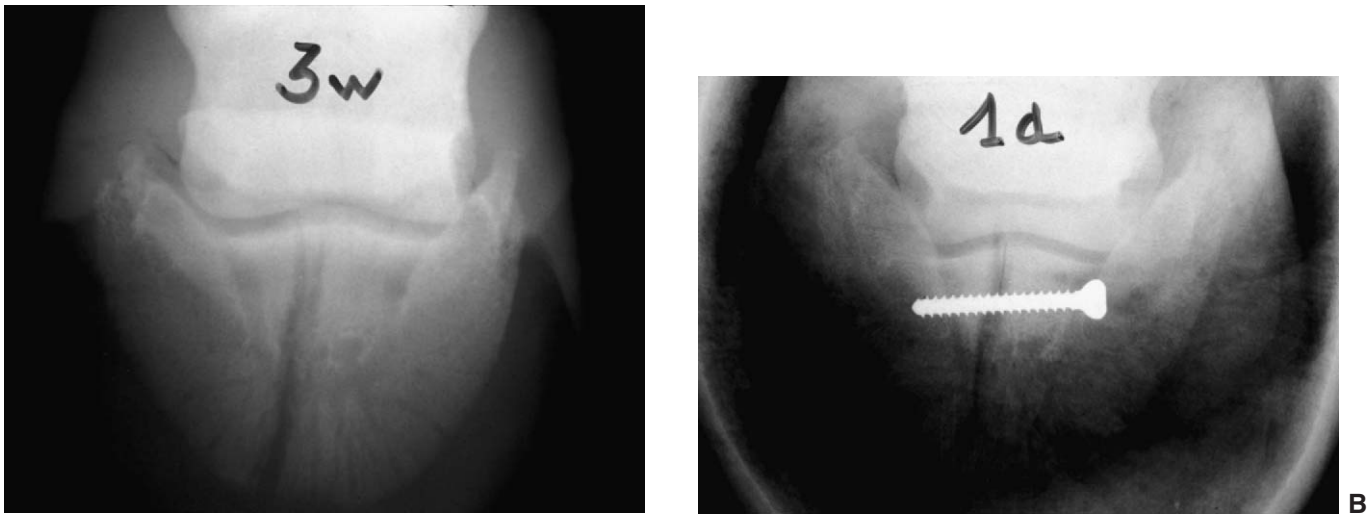


FIGURE 13-12 **A,** Type III fracture of third phalanx (P3); sagittal fracture of P3. **B,** Type III fracture repair using a single lag screw. *(Courtesy Michael Schramme.)*

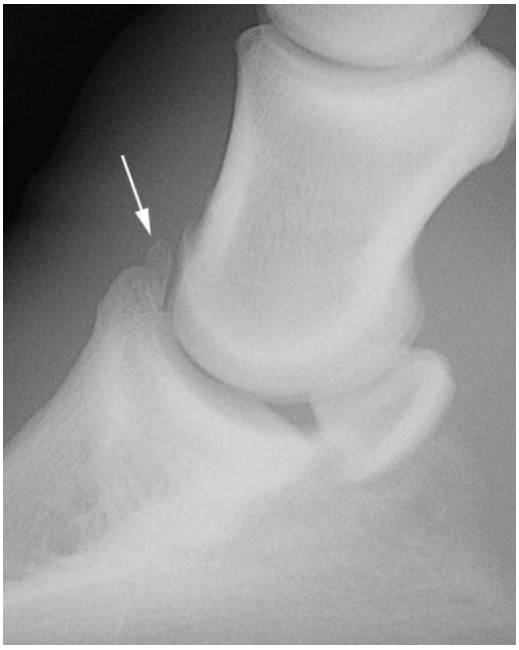


FIGURE 13-13 Type IV fracture of third phalanx (P3) or a separate center of ossification on the extensor process of P3.

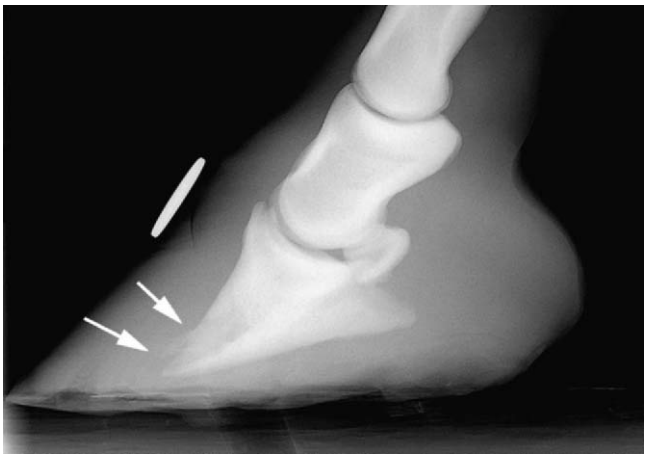


FIGURE 13-14 Type V fracture of the third phalanx (P3): comminuted fracture, usually secondary to osteomyelitis.

in many of the anatomical structures in the foot is critical for accurate diagnosis. Artifacts are easily created if the foot is not properly prepared; they must be recognized and eliminated if misdiagnosis is to be avoided. Radiography of the foot is discussed in detail in Chapter 10.

Radiographs are most helpful in identifying a fracture line when the x-ray beam is projected parallel to the fracture plane. Thus it frequently is necessary to obtain a standard radiographic study of the foot and additional oblique views to highlight the medial and lateral wings of P3.

If no fracture is apparent yet clinical signs and nerve blocks indicate a significant problem in the foot, then more diagnostic information is needed. The DIP joint and navicular bursa should be aspirated and samples submitted for fluid

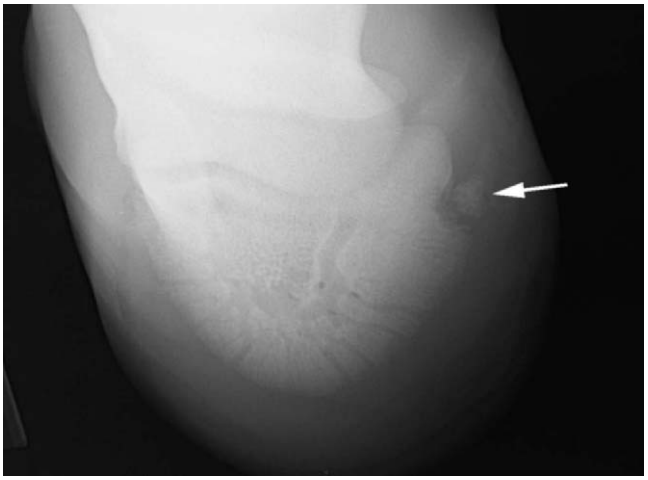


FIGURE 13-15 Type VII fractures are nonarticular fractures at the solar margin of the palmar/plantar processes in foals.

analysis. If there is no indication that these structures are involved, then radiographs should be repeated in 10 to 14 days. A fracture line may be visible at that time when none was apparent initially, owing to bone resorption in the intervening days. Nuclear scintigraphy can be useful in the interim, as it will reveal active bone remodeling.

Type I Fractures

Type I fractures are nonarticular fractures of the palmar/plantar process of P3 (see Figure 13-10). They are most common in the forelimb. These fractures are more prevalent in racehorses and typically are found on the lateral aspect of the left forelimb or the medial aspect of the right forelimb. The cause is thought to be direct trauma or possibly repetitive trauma from an imbalanced or improperly shod foot.

Clinical Findings

The clinical signs can be severe initially, but the lameness may improve more rapidly with these fractures than with other types of substantial P3 fracture. The lameness can often be eliminated by blocking the relevant palmar digital nerve (i.e., medial or lateral, depending on which wing is fractured).

Treatment

Treatment typically involves rest, trimming for proper hoof balance, and application of a bar shoe with quarter clips. Although extended periods of rest are not necessary for these fractures to heal, use of a protective bar shoe is advisable for 9 to 12 months after injury. If the fracture is refractory to conservative management, or when time constraints on the performance horse (especially racehorses) are a factor, then a unilateral palmar digital neurectomy can be performed to restore soundness.

Prognosis

The prognosis for future athletic use in horses with type I fractures generally is good.

Types II and III

Type II and III sagittal fractures (see Figures 13-11 and 13-12) are probably the most common fracture configurations. They are most often found in the forelimbs of racehorses and other sport horses. These fractures can cause more pain than type I fractures and for longer periods of time after injury. Effusion in the DIP joint may be marked if there is a large articular component to the fracture. If arthrocentesis reveals hemorrhagic synovial fluid, then an articular fracture is likely. In some cases, the lameness is improved with intraarticular anesthesia of the DIP joint.

Conservative Treatment

Rest and some form of restrictive coaptation device (a hoof cast or bar shoes with quarter clips) can be effective. The goal is to minimize motion at the fracture site. This goal can be accomplished with a bar shoe that does not contact the frog and has clips drawn substantially up the hoof wall and placed perpendicular to the fracture plane. In addition to the bar shoe, the solar surface of the foot is packed with a firm polyurethane material. Shoes with this packing material can be difficult to remove but nevertheless should be reset every 4 to 6 weeks. Alternatively, a rim shoe fitted to the foot and filled with acrylic or polyurethane can be used to minimize motion throughout the hoof capsule.

With type II and III fractures that are managed conservatively, it can take 6 to 12 months for the fracture to appear healed radiographically. The fracture line disappears from distal to proximal, from solar margin to articular surface. The last area to heal is the subchondral bone just below the articular cartilage. Some authors recommend supportive shoeing for the rest of the horse's athletic career. The rationale is that fracture healing is insufficient to support the stress of race training, and that expansion of the hoof capsule during weight bearing concentrates loading forces at the previous fracture site.[8]

Surgical Repair

Type II and III fractures can be repaired surgically using single lag-screw fixation (see Figure 13-12, *B*). The horses in which this approach is most warranted are mature, large-breed horses with radiographic evidence of a step deformity at the articular surface. Type III fractures are more amenable to surgical repair because the relatively equal halves of P3 allow screw placement more perpendicular to the fracture plane.

Preoperative radiographs should be carefully analyzed with respect to screw size, screw placement, and depth of hoof wall. Radiographic or fluoroscopic guidance is essential for proper placement of the screw. If fluoroscopy is not available, survey radiographs with radiopaque markers placed on the hoof wall as reference points can be used to determine the appropriate site for screw placement. A hole 10 mm in diameter is drilled through the hoof wall to expose the surface of P3. A glide hole is drilled parallel to the articular surface in the near portion of the bone. A radiograph is then taken to assess placement of the drill bit in the glide hole.

In most instances, a 5.5-mm–diameter screw should be used. A drill sleeve insert is used to drill the thread hole in the far portion of the bone. The thread hole is measured for depth and a screw 5 mm shorter than the entire depth of the hole is selected. The thread hole is tapped and the screw placed to compress the two parts of P3 together in an attempt to eliminate the step deformity at the articular surface. (NOTE: The glide hole does not need to be countersunk, as P3 is relatively soft and the surface collapses under the compression of the screwhead as the screw is tightened.)

Most technical failures occur because the hole in the hoof wall is not sealed properly and the screwhead becomes contaminated before it is covered with granulation tissue. Infection of the screw may compromise the fixation before complete bone healing occurs. Furthermore, infection may seed the fracture line and allow entry of bacteria into the DIP joint. Polymethylmethacrylate impregnated with antibiotics can be used to seal the hoof wall defect. As with horses managed conservatively, it is prudent to keep these horses in corrective shoes for the rest of their athletic career.

Prognosis

The prognosis for future athletic performance in horses with type II or III fractures has been reported to be guarded, although it probably depends on the size of the articular component, whether the integrity of the articular surface can be restored, and the age, size, and temperament of the horse.

Healing is likely to be more rapid and complete in younger horses. In one study involving horses 1 to 2 years of age that were managed conservatively, 50% of the horses were sound after a 12-month break from training.[9]

Type IV Fractures

Type IV fractures are extensor process fractures. Two distinct causes have been proposed. One is hyperextension of the DIP joint (which typically is an injury of horses that perform at high speed). The other is incomplete ossification of the extensor process, which is a secondary ossification center for P3; these fractures may be bilateral and do not necessarily cause a clinical problem (see Figure 13-13). Fractures of the extensor process vary in size and in the extent of their articular involvement (see Figure 13-14). Surgical intervention may be indicated if clinical signs (lameness, joint effusion) indicate that the fragment is causing a problem.

Diagnosis

Extensor process fractures are easiest to see radiographically on the lateromedial projection of the foot. Most extensor process fragments are considered to be separate centers of ossification and are located proximal to the main portion of the extensor process. In occasional cases involving bilateral extensor process fractures, there is cystic cavitation of the extensor process.

Diagnostic ultrasonography is helpful in assessing the inflammatory reaction associated with the fracture or separate ossification center. Sonographic evidence of thickening of the joint capsule and proliferation of the synovium around the fragment indicates that the fragment is creating an inflammatory reaction. This information is useful in determining how to manage the problem (conservatively or surgically). Diagnostic ultrasonography is also useful in determining which side of the joint the fragment lies on, which is important for surgical planning.

Treatment

If the fragment is found to be clinically significant, then surgical removal should be contemplated. It is important, however, to first evaluate the DIP joint for signs of osteoarthritis. Horses that are developing osteoarthritis are not candidates for surgical removal of the fragment unless the client understands that the joint will have to be medicated postoperatively to maintain soundness and that these changes can seriously affect long-term soundness.

Horses with acute fractures or fragments without associated osteoarthritis are candidates for arthroscopic removal. With very large fragments, internal fixation with a small lag screw may be warranted, with fracture reduction directed arthroscopically.

Prognosis

The prognosis for future athletic performance generally is good for horses with separate centers of ossification, provided that the fragment is not creating cartilage damage due to its size. Many surgeons are removing these fragments as a preventive measure, but many more are removing them prior to the sale of the horse. Acute extensor process fragments also have a good prognosis, provided they can be removed easily. Larger fragments that require surgical reconstruction carry a more guarded prognosis. The prognosis is guarded for either condition when the horse has already developed osteoarthritis.

Type V Fractures

Type V fractures are comminuted fractures of P3 (see Figure 13-14). They most often occur in association with an infectious process, such as osteomyelitis (septic pedal osteitis; see p. 276). Once osteomyelitis is established, the associated bone resorption may weaken P3 enough to allow it to fracture under even normal loading. Clinical signs are similar to those seen with other infectious processes involving the foot.

Diagnosis

It may be difficult to define the fracture area unless a radiograph is taken of the specific location. Special radiographic techniques, such as contrast fistulograms, may assist in identifying the location of the fracture and determining the extent of the infectious process.

Treatment

When osteomyelitis is present or suspected, surgical debridement of the infected bone and any sequestra is indicated. It may be necessary to remove a considerable amount of sole or wall to gain sufficient exposure for removal of all the infected bone. Diseased bone usually is easily differentiated from normal bone with a curette, because it is very soft and discolored. Comminuted fractures that result from penetrating wounds to the foot are discussed on p. 275. Removal of any devitalized or grossly contaminated bone is important in managing these injuries.

Prognosis

The prognosis for type V fractures depends on the underlying cause and on whether the DIP joint is involved. Comminuted, infected fractures that communicate with the DIP joint require aggressive therapy and carry a poor prognosis.

Type VI Fractures

Type VI fractures are nonarticular fractures at the solar margin of P3 (see Figure 13-15). These fractures usually occur secondary to other foot conditions that cause bone resorption, such as laminitis or pedal osteitis. However, they can be primary, as a result of trauma.

Diagnosis

Solar margin fractures can be missed on standard radiographic projections of the foot if the technique is not adjusted appropriately. It is imperative that the shoe be removed to visualize the entire solar margin of P3. Routine 65-degree dorsopalmar/plantar projections are effective in demonstrating the solar margin, provided a soft technique is used.

Treatment

Conservative treatment involving the application of a wide-web shoe with a concave solar surface, with or without pads, is sufficient in most cases.

Prognosis

The prognosis is good for traumatically induced (i.e., primary) solar margin fractures, because they are uncompli-

cated and heal well. The prognosis for type VI fractures that are secondary to other foot conditions is guarded, unless the primary problem can be managed effectively.

Type VII Fractures

Type VII fractures are nonarticular fractures at the solar margin of the palmar/plantar processes in foals (see Figure 13-15). These fractures are sometimes called *ossicles* or *osseous bodies*. They can cause various degrees of lameness and may lead to a club foot conformation. Some authors consider these ossicles to be incidental findings, as they are believed to be secondary centers of ossification. However, Kaneps et al.[10] demonstrated histologically and microradiographically that these lesions are, in fact, fractures. Conservative management, primarily involving restricted exercise and appropriate trimming for optimal hoof balance, generally is all that is required for complete healing.

Subchondral Bone Trauma

Some horses present clinically very much like those with a P3 fracture, yet there is no radiographic evidence of a fracture, either immediately or days after the onset of lameness. Nuclear scintigraphy reveals intense radionuclide uptake in P3, focally or in a semicircular area of the subchondral bone. It is currently assumed that this lesion is the result of trauma to the subchondral bone and may be a work-specific injury associated with stress remodeling, possibly exacerbated by mediolateral imbalance in the foot. This problem is discussed further on p. 266.

Fractures of the Navicular Bone

Navicular bone fractures are relatively rare. They are more common in the forelimbs than in the hindlimbs. These fractures can be comminuted or simple; most are simple vertical fractures that run in a sagittal plane on one side or other of the central ridge (Figure 13-16). (Avulsion fractures of the navicular bone in the region of the distal sesamoidean impar ligament are discussed later.)

In most cases of acute fracture, there is a history of blunt trauma, such as kicking the stall or landing on a solid object. It is speculated that navicular disease and the associated demineralization may predispose the navicular bone to sagittal fractures. It has also been suggested that navicular bone fractures are created by sudden and severe unequal pressure by P2 and P3 on the navicular bone during hyperextension of the DIP joint. Colles[11] reported on 40 horses with sagittal fractures of the navicular bone; in 39 cases, the fracture could be related to a specific traumatic incident, and in the other case the horse had previously undergone digital neurectomy.

It has further been proposed that navicular bone fractures might be associated with the pull of the suspensory ligaments of the navicular bone when a sudden force pushes P2 in a palmar direction. The navicular bone is thicker in its center (particularly over the sagittal ridge), so the bone is more likely to fracture to one side of the midline.

The navicular bone does not have a periosteal covering, so bone repair is limited to fibrous union and endosteal ossification. Because navicular fractures do not heal with callus formation, they may be more prone to instability, and thus

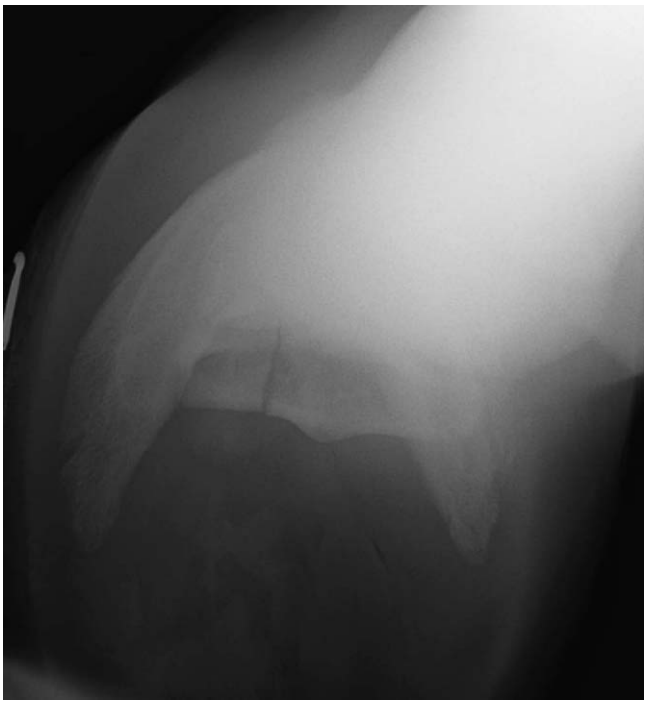


FIGURE 13-16 Sagittal fracture of the navicular bone.

persistent lameness, than fractures in other regions. Incomplete healing may be further complicated by constant motion at the fracture site.

Clinical Findings and Diagnosis

Horses with acute navicular bone fractures may be moderately to severely lame. Horses with chronic fractures may be lame only at a trot, although in many cases the lameness persists. Palmar digital nerve blocks substantially improve or resolve the lameness in most cases. The majority of navicular bone fractures may be definitively diagnosed by routine radiography, provided the foot is adequately prepared (to avoid artifacts) and high-quality radiographs are obtained. The 65-degree dorsopalmar/plantar view typically is the most informative of the routine projections.

Bipartite/Tripartite Navicular Bones It is important to distinguish navicular bone fractures from the congenital condition in which the navicular bone appears radiographically to be in two (bipartite) or three (tripartite) pieces (Figure 13-17). Unlike navicular fractures, this condition usually is bilateral and is present from the time of birth. If these horses are lame, the lameness is more typical of horses with navicular disease than of those with navicular bone fractures. Radiographically, the bipartite/tripartite navicular bones have one or two lucent lines and symmetrical pieces of bone. The bone edges are smooth and there is a wide radiolucent region between the pieces.

Treatment

Acute navicular bone fractures usually are treated conservatively, with rest and the application of a bar shoe with quarter clips. One recommendation is to trim the foot to achieve cranial-caudal balance and then apply four 3-degree wedge pads to raise the heels. Stall rest is recommended for 2 months, followed by hand-walking exercise for 2 months. The shoe

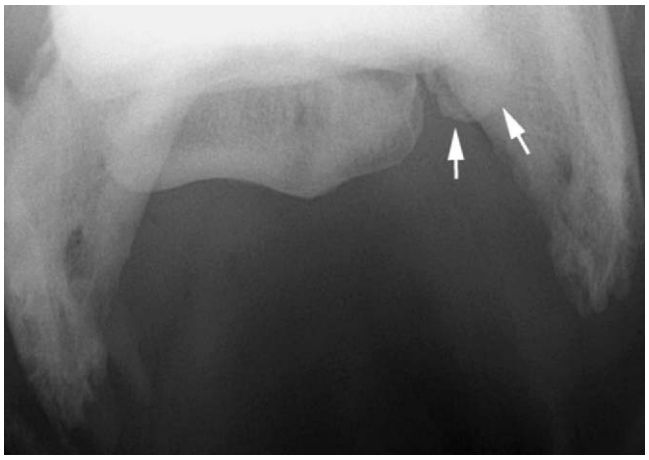


FIGURE 13-17 Bipartite navicular bone.

is reset every 4 weeks, with removal of one pad at each shoeing.[12]

Lag screw fixation of navicular bone fractures has shown promising results.[11] With this technique, a drill jig is used to position the foot. Once positioned, careful radiographic control or fluoroscopic guidance directs accurate placement of a long, 3.5-mm lag screw across the fracture (Figures 13-21 and 13-22). Potential complications include splitting of the fragment, inability to stabilize the fragment with resultant step deformity, and poor stabilization of the fracture.

Prognosis

The prognosis for future athletic performance in horses with navicular bone fractures is guarded. Regardless of whether the fracture is managed conservatively or surgically, neurectomy may be indicated in horses that are still lame after 6 to 12 months.

Subchondral Bone Trauma of the Third Phalanx

Subchondral bone trauma involving P3 is a recently recognized clinical condition that can be definitively diagnosed only with nuclear scintigraphy or MRI. It occurs predominantly in racehorses and occasionally in other sporthorses. Subchondral bone trauma may be a precursor to P3 fracture or possibly to subchondral bone cyst. Dyson[1] has suggested that these reactions are not single-episode injuries but rather stress reactions and bone remodeling.

Clinical Findings and Diagnosis

Most cases involve forelimb lameness that is localized to the foot. Intraarticular anesthesia has little effect on the lameness, and radiographs fail to demonstrate any significant abnormality at initial presentation. Nuclear scintigraphy is necessary to identify the characteristic bone reaction pattern, which may be focal or more widespread across the DIP joint. MRI may allow definitive diagnosis of focal lesions.

Treatment

Conservative treatment is indicated and includes rest and some form of restrictive coaptation device for the foot (a hoof cast or bar shoes with quarter clips). This goal can be accomplished with a bar shoe that does not contact the frog and has clips drawn substantially up the hoof wall. In addition to the bar shoe, the solar surface of the foot is packed with a firm polyurethane material. Shoes with this packing material can be difficult to remove but nevertheless should be reset every 4 to 6 weeks. Alternatively, a rim shoe fitted to the foot and filled with acrylic or polyurethane can be used to minimize motion throughout the hoof capsule.

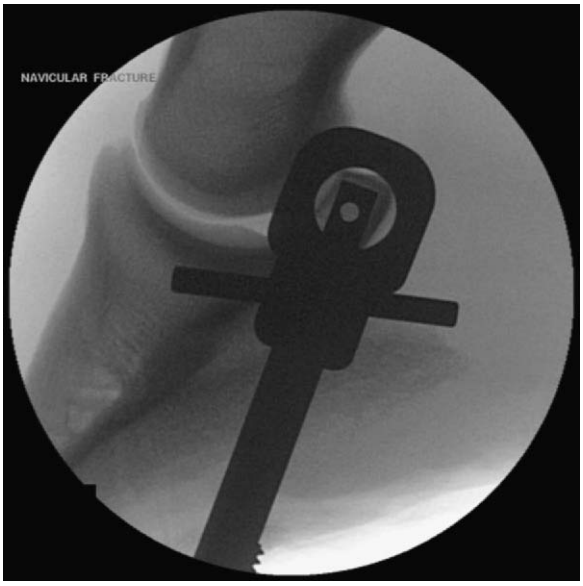


FIGURE 13-18 Fluoroscopic image of a navicular bone fracture being repaired with a jig to assist in screw placement.

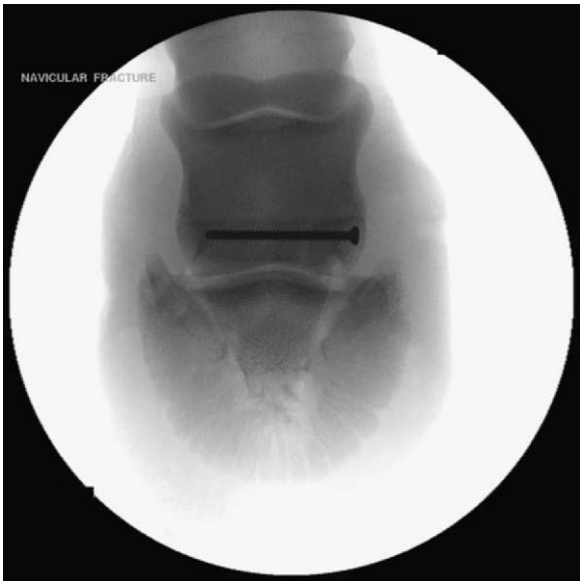


FIGURE 13-19 Final repair of the navicular bone fracture shown in Figure 13-16.

Prognosis

The prognosis appears to be good with an appropriate period of rest and restricted activity. Subchondral injury may lead to cyst formation, so reexamination should include a complete

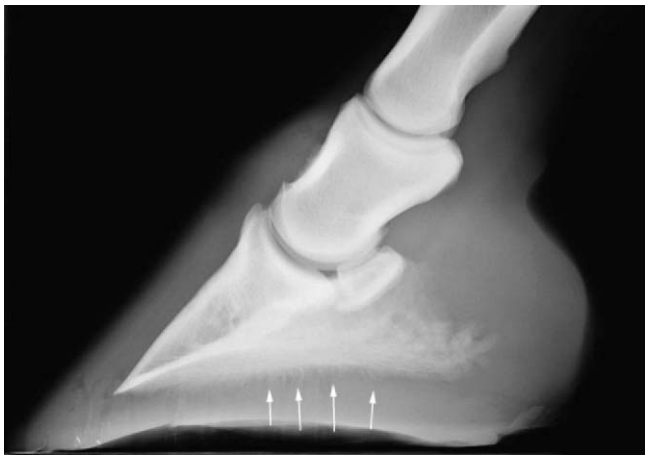


FIGURE 13-20 Radiographic appearance of pedal osteitis.

radiographic study to evaluate the subchondral bone for cyst development, as well as a repeat bone scan.

Nonseptic Pedal Osteitis

Pedal osteitis is a descriptive term and not a definitive diagnosis. It has been defined as demineralization of the solar margin of P3, as a result of inflammation.[13] Pedal osteitis has historically been a radiographic diagnosis, evident as focal or diffuse resorption of bone around the solar margin of P3 and widening of the vascular channels, with or without abnormal lucent areas in the palmar processes and associated new bone production (Figure 13-20). Currently, this diagnosis is supported by a nuclear scintigraphic pattern of bone remodeling in the periphery of the foot (Figure 13-21).

Causes and Contributing Factors

This term *pedal osteitis* probably describes the result of a number of different insults that cause hyperemia or focal pressure, or both, at the solar margin of P3. Persistent inflammation in this region of the foot is the most likely cause of bone loss and reactive new bone production. Once these radiographically apparent changes have occurred, they seem to be permanent and may not continue to be an active clinical problem.

Pedal osteitis can be a primary condition, but it is usually secondary to other conditions, including laminitis, focal submural sepsis, type VI fractures of P3, chronic subsolar bruising, flexural deformity of the DIP joint (clubfoot), keratomas, and puncture wounds. It usually occurs in the forefeet, probably because of the increased concussive forces in these feet compared with the hindfeet. Histopathologic changes are thought to be similar to those seen with laminitis and are associated with abnormal structure in the epidermal and dermal laminae of the distal hoof wall and sole, primarily at the toe and wing of P3. Demineralization can be severe and predisposes to solar margin fractures.

Clinical Findings and Diagnosis

Pedal osteitis can be difficult to definitively diagnose and requires clinical, radiographic, and scintigraphic evidence to support the diagnosis. It has historically been diagnosed with properly exposed and positioned radiographs, but radiography should not be the definitive diagnostic tool.

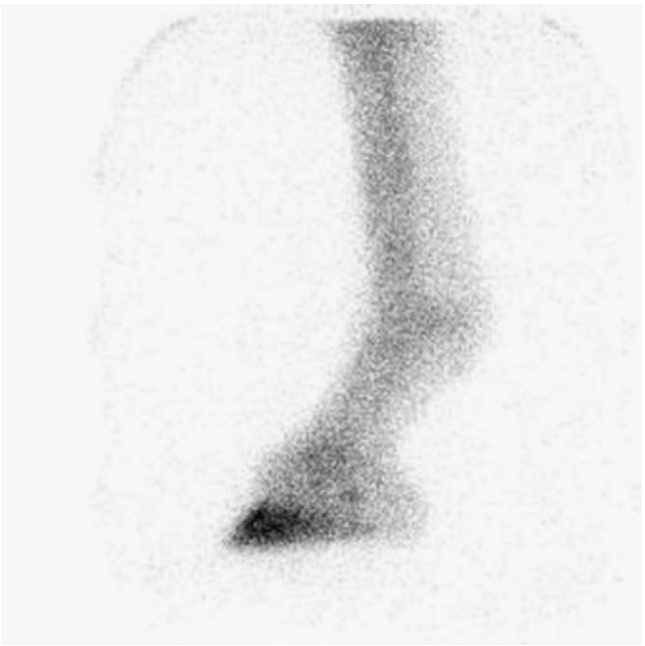


FIGURE 13-21 Scintigraphic appearance of pedal osteitis.

Radiographic signs of disease (demineralization of P3, wide vascular channels, and irregular new bone formation along the solar margin) can easily be missed in overexposed radiographs. Standard P3 technique 65-degree dorsopalmar oblique, lateromedial, and 45-degree palmaroproximal/palmarodistal oblique (skyline) projections are useful to assess the solar margin of P3.

Nuclear scintigraphic examination with solar views of the foot are diagnostic for bone remodeling along the solar margin. Bone remodeling is usually bilaterally symmetrical but can be more intense in one foot.

Treatment and Prognosis

Treatment is determined by the underlying cause.[14] Some authors suggest that reducing concussion in the foot using a wide-web shoe with a concave solar surface, with or without full pads, is the treatment of choice. However, this strategy is likely to be effective only in cases of primary pedal osteitis or chronic solar bruising.

Osteitis of the Palmar Processes

Radiographically, the palmar processes of P3 have substantial remodeling changes in some horses.[1] The normal appearance of these structures is quite variable, so it is necessary to have an understanding of normal variations to identify abnormalities. The radiographic changes seen with osteitis of the palmar processes are similar to those described in the preceding section for horses with nonseptic pedal osteitis (Figure 13-22). Increased bone remodeling at the heels seen on nuclear scintigraphic examination supports this diagnosis (Figure 13-23).

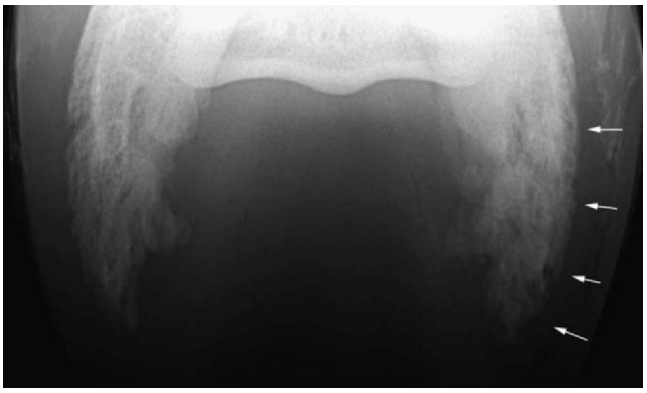


FIGURE 13-22 Radiographic appearance of pedal osteitis of the palmar process.

FIGURE 13-23 Scintigraphic appearance of pedal osteitis of the palmar process.

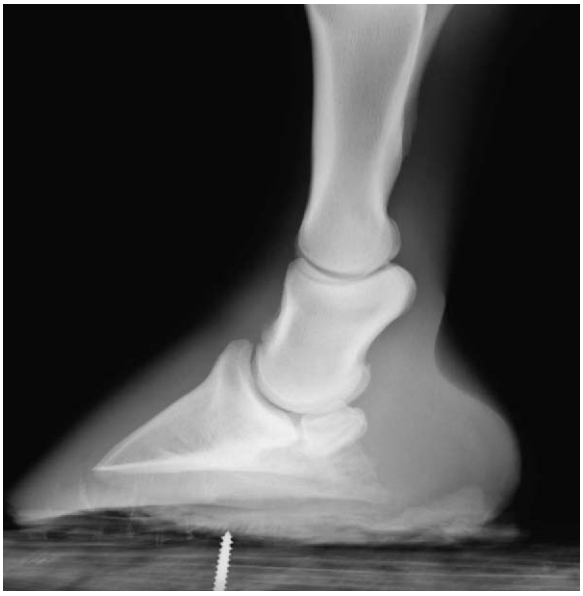


FIGURE 13-24 Long-toe/low-heel foot conformation with 0-degree or negative palmar angle.

Clinical Findings and Diagnosis

Osteitis of the palmar processes is most often seen in horses with the long toe/low heel foot conformation and flat, compressible soles. This condition most often occurs in the forefeet. It can be difficult to determine whether this condition is a primary cause of lameness or part of the "heel pain" complex (see Chapter 14).

Radiographs of the foot should include a lateromedial projection (with the beam centered just above the bearing surface of the foot), a 65-degree dorsopalmar oblique view, and a palmaroproximal/palmarodistal oblique (skyline) view. The lateromedial view is particularly useful for determining the palmar angle of P3 relative to the ground surface; it is also helpful for assessing medial-lateral balance (see Chapter 14). Low-heeled horses with this condition frequently have a 0-degree or negative palmar angle (Figure 13-24).

The 65-degree dorsopalmar and skyline oblique views may reveal discrete circular lucencies within the palmar processes that can occur with this condition. In addition, the trabecular pattern of the palmar process may be more obvious because of generalized demineralization. Nuclear scintigraphy may be necessary to determine whether these changes are primary or associated with other clinically significant changes in the navicular bone, navicular bursa, or deep digital flexor tendon.

Treatment and Prognosis

Treatment is directed at correcting the foot imbalance. The prognosis depends on how successful this correction has been. Chronic changes in the hoof capsule can be difficult to correct.

Navicular Disease

Owing to its typical clinical presentation of "heel pain," navicular disease, or navicular syndrome, is discussed at length in Chapter 14.

TENDON AND LIGAMENT DISORDERS

Desmitis of the Collateral Ligaments of the Distal Interphalangeal Joint

Injury to the collateral ligaments of the DIP joint has only recently been reported as a cause of acute lameness, owing to the expanded clinical use of diagnostic ultrasonography with high-frequency transducers.[15] More recently, MRI has improved the diagnostic accuracy of identifying collateral desmitis of the DIP joint.

These ligaments originate in depressions on the distal aspect of P2 and insert in small depressions on the dorsomedial and dorsolateral aspects of P3 (see Chapter 1). They support the DIP joint during movement primarily in the sagittal plane but also in the frontal and transverse planes. Asymmetrical foot placement can create lateral or medial rotation and sliding of P3 with respect to P2.[16] It also creates torque, with P2 rotating and the higher side moving in a palmar direction. These forces create tension in the collateral ligaments that, when excessive, can result in ligament damage.

Clinical Findings and Diagnosis

This condition occurs most often in the forelimb, and in the medial collateral ligament more often than the lateral.[17] Seldom are there localizing signs on clinical examination that are suggestive of collateral ligament injury. Most affected horses are moderately lame; typically, the lameness worsens when the horse is walked in a circle with the lame limb to the inside. Palmar digital nerve block improves the lameness in most cases. Improvement with a DIP joint block is more variable, with positive results reported to occur in only 40% of cases[17] or in 75% of cases,[15] depending on the study.

Radiography Radiographic examination often does not demonstrate any changes relative to collateral ligament injury. In one study, in which collateral desmitis was diagnosed using MRI, only 2 of 18 horses had radiographically detectable lesions.[17] In another study, however, in which the diagnosis was based on radiographic and ultrasonographic findings, 10 of 20 horses had radiographic lesions.[15] (This disparity may explain the difference in response to intraarticular anesthesia between these studies.)

When present, radiographic findings consist primarily of enthesiophyte formation at the origin and/or insertion of the damaged collateral ligament. On occasion, radiographic examination has revealed a well-defined radiolucent area associated with the insertion site on P3 (Figure 13-25).

Nuclear Scintigraphy Nuclear scintigraphy may reveal bone remodeling at the origin or insertion site of the damaged collateral ligaments (Figure 13-26). However, scintigraphy may not demonstrate any changes relative to the collateral ligaments. In one study, increased radionuclide uptake on the affected site was found in only 60% of horses with collateral desmitis diagnosed using MRI.[17] Those authors found that the intensity and distribution of uptake in horses with collateral ligament injuries was similar to the findings in horses with palmar process fractures, so appropriate radiographic studies must be used to rule out a palmar process fracture in horses with these scintigraphic findings.

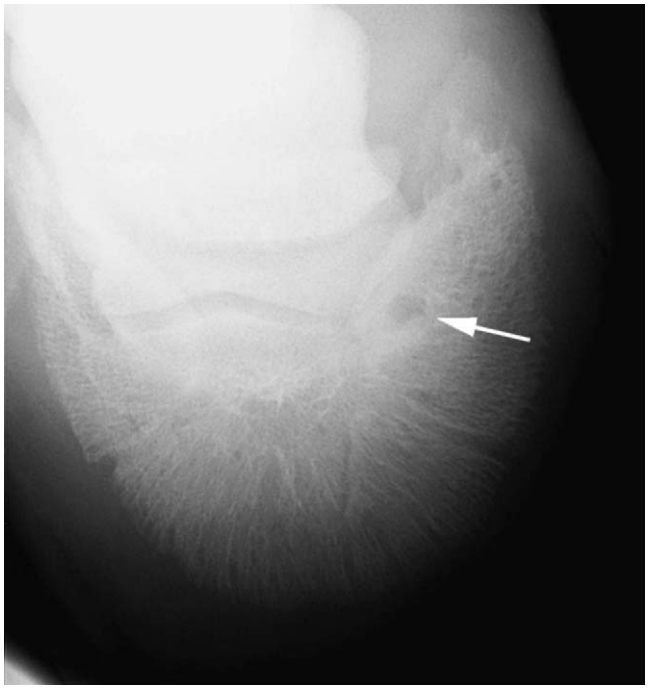


FIGURE 13-25 Ovoid lucency associated with the distal attachment of the collateral ligament of the distal interphalangeal joint.

FIGURE 13-26 Nuclear scintigraphic examination of a collateral ligament desmitis of the distal interphalangeal joint. Radiographs were necessary to rule out a palmar process fracture as these two clinical conditions of the foot can appear quite similar.

Ultrasonography For the clinician without access to MRI, diagnostic ultrasonography can be used to assess the origin and, depending on foot conformation and coronary band position, part of the body of the collateral ligaments. Obtaining good-quality images can be difficult, however, and

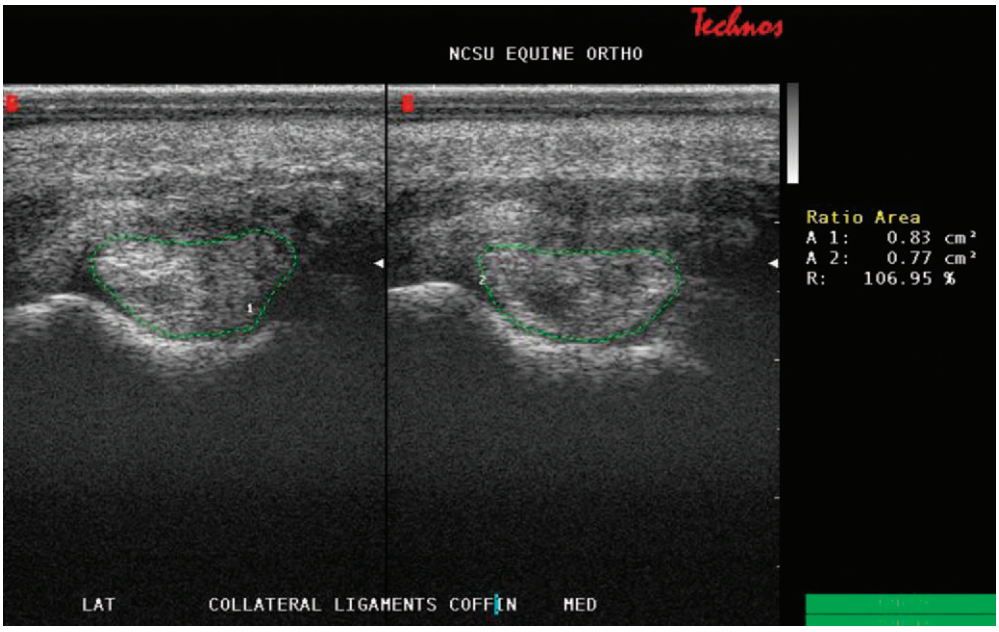


FIGURE 13-27 Sonographic examination of a horse with collateral desmitis of the distal interphalangeal joint. Both ligaments are shown for comparison.

requires diligence. Placing a 3-degree wedge pad under the opposite side of the foot being examined can assist with evaluating the collateral ligament on the nonwedged side. The opposite foot should always be used for comparison of size, shape, and consistency. Collateral desmitis is recognized sonographically when the body of the ligament has an increased cross-sectional area and reduced echogenicity or a discrete hypoechoic lesion, when compared with the normal side and with the opposite foot (Figure 13-27).

Unfortunately, the insertion sites on P3 cannot be assessed sonographically, and this limitation should be recognized. It is therefore prudent to perform radiography and, if possible, nuclear scintigraphy in every case in which collateral desmitis is suspected yet sonographic findings are inconclusive.

Treatment

Treatment includes rest, antiinflammatory therapy, and shoeing adjustments. Shock-wave therapy has been recommended, because the ligament insertion sites can be affected (i.e., this condition can be an insertional desmopathy). At present, however, only anecdotal information exists about the effectiveness of shock-wave therapy for this condition. It may be that only the proximal sites are accessible to shock-wave therapy, so careful case selection is necessary. One author recommends the fabrication and use of a shoe that has a wide web on the affected side of the foot and a normal web on the unaffected side of the foot.[16]

Prognosis

The prognosis for return to athletic use is guarded in most cases. Typically, the horses that have recovered well had mild injuries and were given a sufficient period of rest and appropriate rehabilitation. The prognosis probably is determined by the site and extent of the injury. However, these criteria may be difficult to accurately determine with current imaging techniques. This area is one in which MRI is very helpful.

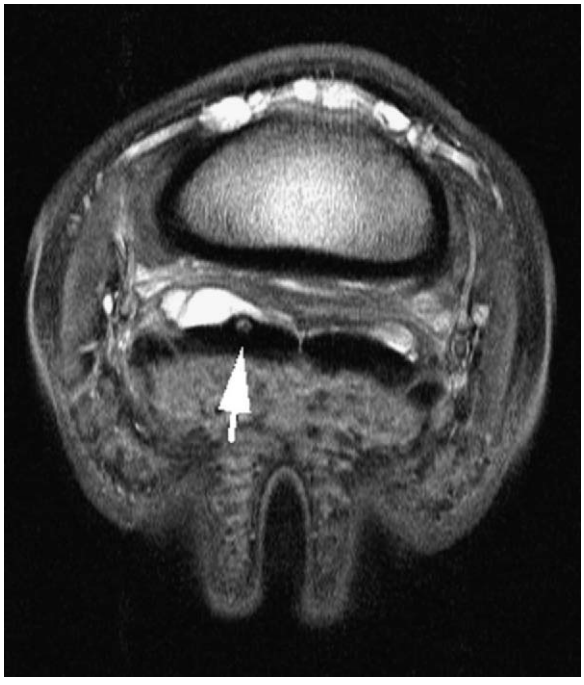


FIGURE 13-28 Magnetic resonance imaging examination of a horse with lameness that was blocked out by a palmar digital nerve block. The deep digital flexor tendon has a large core defect just proximal to the navicular bone.

Primary Deep Digital Flexor Tendonitis

With the expanded use of nuclear scintigraphy and MRI in clinical practice, tendonitis of the deep digital flexor tendon (DDFT) at the level of the digit has become recognized as a primary clinical entity (Figure 13-28). Previously, it was thought that deep digital flexor (DDF) tendonitis at this level was secondary to the changes seen with navicular disease,

particularly changes involving the flexor surface of the navicular bone.[18] It is now clear that the DDFT can be damaged at this level without other pathologic conditions being present. The clinical presentation and imaging results are different from what is typically seen with navicular disease, and treatment for the two conditions likewise differs.

The anatomy of the palmar aspect of the digit is discussed in detail and illustrated in Chapter 1. Bowker[19] recently demonstrated changes in P3 at the attachment of the DDFT and the distal sesamoidean impar ligament (DSIL) in horses previously diagnosed with navicular disease. These changes were demonstrated by an increase in safronin O staining across the entire area of attachment and extending for several millimeters from the point of attachment into the DDFT.

Increased production of proteoglycans in tendons and ligaments occurs when the tissues are stressed by compressive forces. Some of the specimens from horses with navicular disease were found to have cartilage formation in the DSIL and DDFT.[19] Bowker hypothesized that progressive stress in the tissues of this region may contribute to the onset of clinical signs typical of navicular disease. Based on these findings, it may be supposed that some of the cases of DDF tendonitis at this level are not separate entities but are part of the complex of changes seen with navicular disease. Some authors, however, have reported DDF tendonitis proximal to the navicular bone as a separate entity (i.e., primary tendonitis).[20,21] Those authors made the distinction using MRI in horses with foot pain.

Clinical Findings

Although primary DDF tendonitis has been identified in horses from many different disciplines, lameness is most often seen in horses that jump. The lameness often is acute, severe, and usually confined to one forefoot. Clinical examination contributes little to localizing the problem to the distal portion of the DDFT. The severity of the lameness typically increases when the horse is lunged and when worked on soft footing. The extension stress test (in which the DIP joint is extended by raising the toe for 30 to 60 seconds before the horse is trotted away) is positive in most cases.

Diagnosis

Palmar digital nerve block usually improves the lameness. However, as the DDFT lesion can extend proximally, beyond the foot, an abaxial sesamoidean nerve block is required to improve the lameness in some cases. Intraarticular anesthesia of the DIP joint inconsistently improves the lameness.

Intrathecal Anesthesia Intrathecal anesthesia of the digital flexor tendon sheath reportedly improves the lameness.[20] However, this technique needs further evaluation. In particular, it is necessary to identify which other structures could be anesthetized and thus confuse interpretation of the results. Also, because most affected horses do not have obvious effusion in the digital flexor tendon sheath, it can be difficult to inject into the sheath. Furthermore, intrathecal anesthesia may not completely abolish the lameness if the lesion is distal to the tendon sheath (e.g., an insertional tenopathy).

This procedure is made easier by approaching the sheath from the palmar aspect, just distal to the palmar annular ligament (see Chapter 1). An 18-gauge, 1.5-inch needle is directed proximally along the abaxial border of the flexor tendon. Fluid is obtained in some horses, which confirms placement of the needle within the sheath. Most often, however, ease of injection of the anesthetic solution is the only indicator that the sheath has been entered successfully.

If a palmar digital nerve block improved the lameness, then the digital flexor tendon sheath should be anesthetized. If digital sheath anesthesia improves the lameness, then a course of intrathecal treatment may be indicated.

Imaging Confirmation of DDF tendonitis as a cause of heel pain requires an MRI examination. Ultrasonographic examination of the DDFT as far distally as possible may reveal asymmetry of the tendon's lobes, which may suggest a clinical problem more distally in the tendon (Figure 13-29). However, MRI is necessary to more accurately differentiate primary DDF tendonitis from tendonitis associated with navicular disease.

Treatment

Intrathecal injection of a corticosteroid (triamcinolone or methylprednisolone) plus hyaluronic acid may be effective in treating chronic DDF tendonitis at this level. Corticosteroids are contraindicated in cases of acute injury, however, because they can inhibit collagen formation in a healing tendon. A period of rest and rehabilitation also is indicated for horses with acute injuries.

Prognosis

At this time, it appears that horses confirmed with MRI to have primary DDF tendonitis have a guarded prognosis. However, because this condition has only recently been diagnosed, more cases need to be followed and different treatment protocols evaluated.

Flexural Deformity of the Distal Interphalangeal Joint

Flexural deformity of the DIP joint is common in growing horses. With this condition, the DIP joint is maintained in a flexed position. In most cases, it is primarily associated with the DDFT and secondarily with other soft tissues within the digit.

Causes and Contributing Factors

Flexural deformities can be congenital (relatively uncommon) or acquired (common). The cause is poorly understood and probably is multifactorial. There are two widely held theories for the development of these deformities: (1) a discrepancy between bone growth and tendon and ligament growth, and (2) contracture of the muscle-tendon unit of the DDFT in response to pain.

Most flexural deformities of the DIP joint are thought to be part of the developmental orthopedic disease complex, which includes osteochondrosis, angular limb deformities, physitis, and cervical vertebral malarticulation. Rapid skeletal growth predominantly involves an increase in bone length and is primarily determined by genetics and nutrition. With the first theory of flexural deformity, bone is thought to grow more rapidly than tendon or ligament.

Rapid growth, however, is not always a factor in flexural deformity of the DIP joint. The other major factor is a painful condition that causes contracture of the DDF muscle (flexor

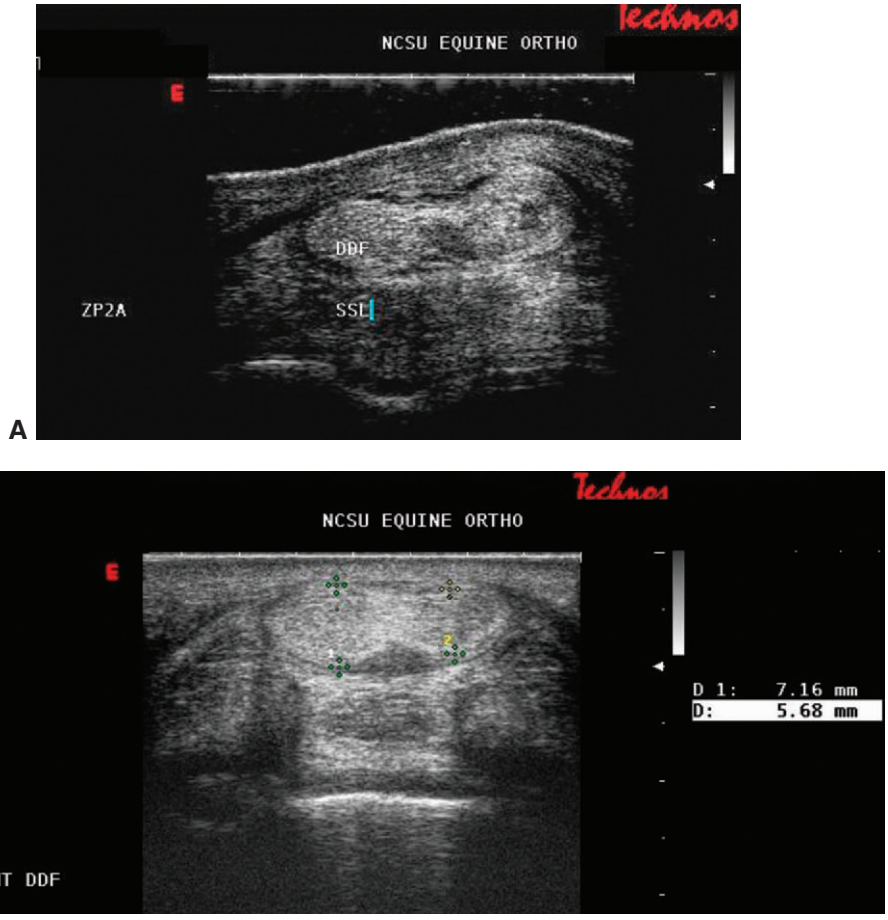


FIGURE 13-29 Sonographic examination of the deep digital flexor tendon at the most distal aspect of the pastern (zone 2PB). Note the asymmetry between the two lobes, indicating tendonitis of the enlarged lobe.

withdrawal reflex) in that limb. Painful conditions include subsolar abscess or severe bruising, P3 fracture (ossicle formation), osteochondrosis, physitis, and puncture wounds. Acute contracture initiated by the flexor withdrawal reflex is thought to create the deformity, which becomes chronic once other soft tissues in the foot (e.g., the joint capsule at the palmar/plantar aspect of the DIP joint) become shortened.

Clinical Findings

Flexural deformity of the DIP joint typically begins in foals 1 to 6 months of age. Most often it occurs in one of the forelimbs. In some breeds and family lines, the condition appears to be heritable, as there is a higher than normal incidence of DIP joint deformity in these foals, and it occurs with regularity in the same foot. In any case, the dorsal hoof wall assumes a more vertical orientation and, in acute cases, the heels may be elevated off the ground. In more chronic cases, the heels grow down, giving the hoof a boxy appearance (Figure 13-30). With chronicity, the toe begins to wear, which can create flaring of the wall at the toe.

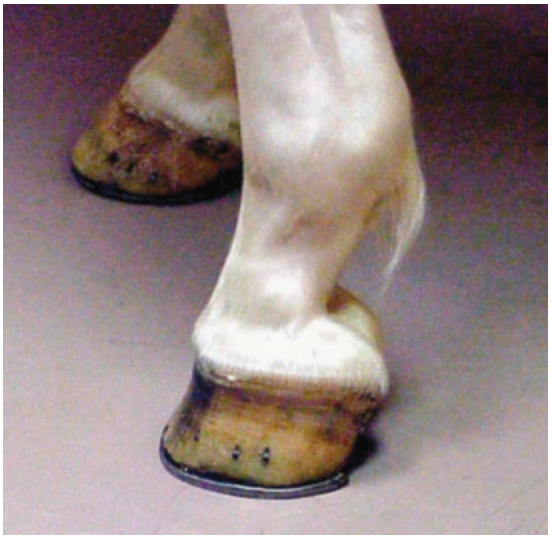


FIGURE 13-30 Flexural deformity of the distal interphalangeal joint in a Quarterhorse yearling.

Diagnosis and Staging

These deformities can be graded or staged based on the hoof wall angle at the toe.[22] Stage I deformities have a dorsal hoof wall angle of 90 degrees or less. In other words, the hoof wall at the toe is not quite or only just vertical. Stage II deformities have a dorsal hoof wall angle of greater than 90 degrees. In these feet, the hoof wall at the toe has proceeded beyond vertical.

Radiographs of the foot can also assist in staging the deformity and evaluating the chronicity of the condition. Changes in the position of P3 within the hoof capsule and other inflammatory changes that may have occurred to P3 due to chronic tearing of the dorsal laminae (e.g., dorsodistal lipping of P3) are apparent on a lateromedial view of the foot.

Treatment and Prognosis

Early recognition of the deformity gives medical therapy the best chance of success. Initial medical treatment should be conservative and include restricted exercise, NSAID therapy, and nutritional management (particularly if no obvious source of pain is identified). Treatment of the underlying cause of pain is critical in foals in which the flexural deformity is secondary to a painful condition.

In foals with chronic deformities, a controlled exercise program in conjunction with NSAID therapy may allow gradual stretching of the joint capsule and the DDFT. Physical therapy can also be useful; exercises may include hopping and creating forced extension of the joint by placing the toe on a block and picking up the opposite limb. However, care must be taken not to overdo these exercises and create more pain.

In some mild cases, the deformity can be managed simply by trimming the heels on a regular basis. More chronic deformities may require the application of toe extensions, which are thought to increase the tension in the DDFT by delaying breakover. Simple glue-on toe extensions are preferable to glue-on shoes because they do little to restrict the rest of the foot in the growing foal.

Surgery Stage I deformities that have not responded to conservative management usually are amenable to transection of the inferior check ligament. This procedure carries a good prognosis for future performance, provided it is done relatively early in the development of the condition and early in the life of the foal.

Stage II deformities usually require a more aggressive approach. An inferior check desmotomy is performed initially. If it is unsuccessful or only minimally effective, then complete transection of the DDFT is necessary to correct the flexural deformity. However, complete transection can compromise soundness in the performance horse, so the prognosis is guarded in these foals.

Flexural deformity of the DIP joint is discussed further in Chapter 11.

INFECTIOUS PROCESSES

Bacterial infection of the tissues within the hoof most often results from a penetrating wound to the foot. These wounds can cause severe lameness and may pose a diagnostic and therapeutic challenge. They are serious and even life-threatening when they involve the DIP joint, navicular bursa, or digital sheath. Early recognition of involvement of a synovial structure and rapid institution of aggressive therapy are critical to a successful outcome.

Bacteria can be introduced into a synovial cavity via an open wound, a self-sealing puncture, hematogenous seeding, extension of perisynovial infection, or iatrogenically. Infection is most likely to become established when foreign material or devitalized tissue is present. A large inoculum of bacteria and the involvement of virulent bacteria further increase the potential for infection to become established.

These infections become particularly problematic when bacteria proliferate within the synovial cavity and colonize the synovial lining. Bacterial pathogens and the immune response of the host stimulate the release of enzymes, which causes further destruction of the synovial tissues. Fibrin and cellular debris produced in the inflammatory response fill the joint and act as a barrier to synovial diffusion, and they limit access of systemically administered drugs to the joint space. Thus treatment must be directed at eliminating any foreign material, reducing bacterial numbers, removing contaminated and devitalized material, and neutralizing and eliminating inflammatory enzymes and other inflammatory products.

Septic Arthritis of the Distal Interphalangeal Joint

Septic processes involving the DIP joint (and even the PIP joint) usually result from traumatic injuries to the foot, most often a puncture wound or laceration that involves the coronary band. Iatrogenic infections do occur and most often are associated with intraarticular injection of corticosteroids. The DIP joint can also be contaminated by a puncture wound to the frog. This situation is discussed later, in the section on penetrating injuries to the navicular area.

History

An accurate history should be obtained to determine the time elapsed since the injury or intraarticular injection, and whether there are any preexisting foot problems. (For example, neurectomy of the affected foot can predispose to the development of septic arthritis in the DIP joint by allowing subsolar infections to progress to osteomyelitis and possibly extend to the joint.) The clinician should recognize that the use of systemic antibiotics and antiinflammatory medications can effectively mask a severe lameness and minimize swelling associated with septic arthritis.

Clinical Findings

Clinical examination should include careful inspection of the frog, sole, and coronary band for evidence of a wound. Marked distal limb swelling may be present, making it difficult to accurately identify surface landmarks. Actively draining wounds, similar to those seen with drainage from a foot abscess, can occur. However, it may be necessary to clip the coronary band and pastern to find a puncture site.

Diagnosis

Radiography If a radiopaque foreign body is still in place, then a radiograph should be taken before the object is removed to determine the depth of penetration (Figure 13-31). In the absence of a foreign body, a survey radiographic study should be performed. Evidence of joint space widening suggests sepsis with fluid accumulation. Osteochondral fragments or foreign material may be apparent close to the puncture or wound or within the joint space (Figure 13-32). Osteomyelitis of P3 may be evident in chronic cases. A contrast fistulogram or arthrogram can help determine joint involvement (Figure 13-33), although arthrocentesis with joint fluid analysis may be more useful than arthrography.

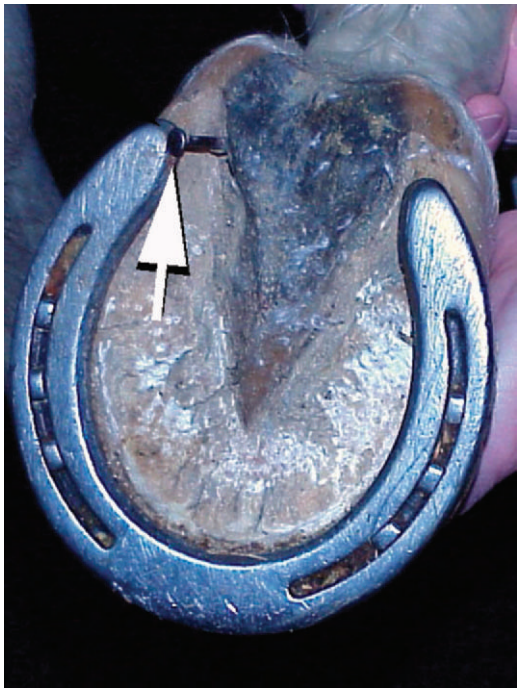


FIGURE 13-31 A nail has penetrated the frog in this horse (*arrow*). A complete radiographic study of the foot, with the nail in place, is critical to determining which structures might be involved.

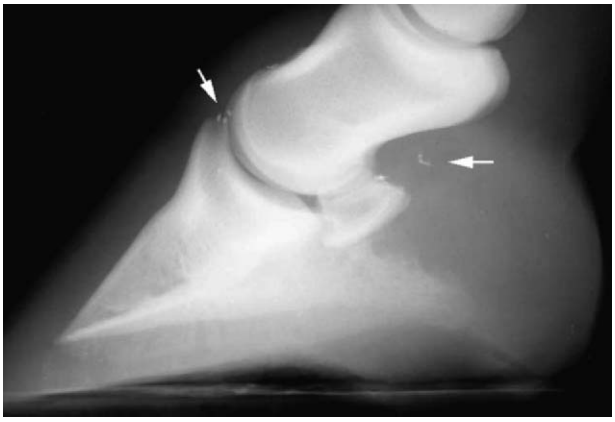


FIGURE 13-32 Foreign material may be present in the joint space. Rust from a nail that had penetrated the frog is seen as radiopaque foreign material dispersed throughout the distal interphalangeal joint (*arrows*).

Ultrasonography Diagnostic ultrasonography can be useful to identify synovial distention and assess the character of the synovial fluid. An increase in cellularity and fibrin content in the synovial fluid increases its echogenicity. The presence of gas shadows suggests either an open joint space or the presence of gas-producing organisms in the joint fluid. The puncture site or wound itself should be evaluated sonographically to determine the specific structures involved. Puncture wounds involving the tendons and ligaments around the joint can significantly affect the prognosis.

Exploration Deep puncture wounds that penetrate the corium require surgical debridement to determine the full extent of the injury. Differentiating septic arthritis from less severe punctures to the dermal laminae can be difficult, however. Septic arthritis should be suspected in the case of any puncture wound that is close to the joint capsule. Failure to examine puncture wounds for possible contamination and septic arthritis of the distal interphalangeal joint will delay appropriate therapy and diminish the prognosis for soundness.

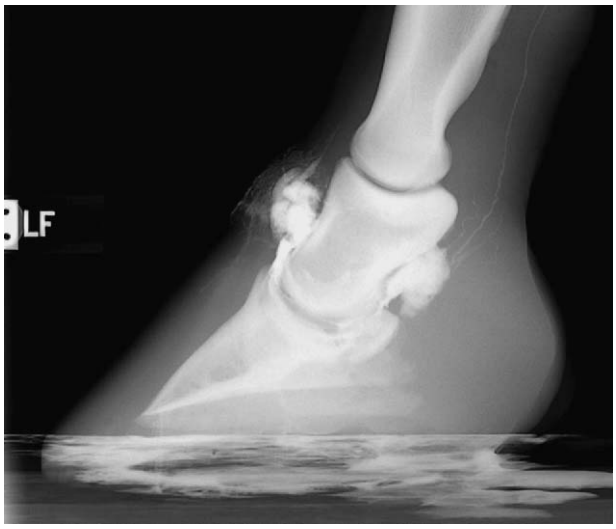


FIGURE 13-33 Contrast arthrogram of the distal interphalangeal joint in a horse with a puncture wound to the frog. The contrast material can be seen on the wooden positioning block, indicating leakage from the joint, along the puncture tract, and out the bottom of the foot. This radiograph confirms that the original puncture wound penetrated the joint.

Arthrocentesis Aseptic collection of a joint fluid sample at a site remote from the wound is recommended in all cases. An increase in total cell count (>30,000 cells/μL) with a predominance of neutrophils and an elevated total protein concentration (≥3 g/dL) are good indicators of sepsis. Gram staining of the joint fluid may demonstrate free bacteria in the joint fluid. Regardless, the fluid should be submitted for bacterial culture and antibiotic sensitivity testing.

After sample collection, it is advisable to assess the integrity of the joint capsule by injecting sterile balanced electrolyte solution into the joint. The fluid should be injected under pressure and the wound assessed for fluid leakage. Fluid seen exiting the wound is evidence of capsular disruption.

Treatment and Prognosis

Confirmation of joint contamination indicates the need for aggressive medical and surgical therapy. Principles common to treating any closed-space infection should be incorporated:

- Copious lavage and drainage of the joint space
- Systemic antimicrobial therapy; broad-spectrum initially and then specific therapy as directed by culture and sensitivity results
- Intrasynovial therapy with an appropriate antibiotic
- Regional perfusion of an appropriate antibiotic

Antibiotic-impregnated polymethylmethacrylate beads or cylinders can also be used to increase the local delivery of antibiotics.

Surgery Although arthrotomy alone can be performed to evaluate and treat the contaminated joint space, arthroscopy

allows more complete evaluation of the septic joint. Whether arthrotomy or arthroscopy is used, osteochondral fragments and areas of osteomyelitis should be removed and/or debrided. Synovectomy is indicated to remove as much pannus and devitalized tissue as possible. Even when arthroscopy is used, it is advisable to enlarge the arthroscopic portals at the end of the procedure to create an arthrotomy wound. Arthrotomy is necessary for drainage of purulent material and daily large-volume lavage of the joint space.

A recent report evaluated the use of endoscopic lavage alone in the treatment of septic joints, tendon sheaths, and bursae. Endoscopy portals and traumatic wounds were closed primarily after lavage. Follow-up information on the 118 horses revealed a 90% survival rate, with return to athletic function in 81% of horses.[23] In this population of cases, it was the early institution of treatment that allowed this protocol to be successful. This approach is unlikely to be successful in horses with more chronic wounds that contain large amounts of fibrin and gross contamination.

Long-term survival in chronic cases of septic arthritis is considered poor (only around 40% in one study).[24,25] Ankylosis occurs with a high frequency and may ultimately result in pasture soundness. Use of cancellous bone grafts can help encourage ankylosis if that is the goal.

Penetrating Injuries to the Navicular Area

Penetrating injuries to the frog can extend to the navicular bursa, DIP joint, and digital flexor tendon sheath. These injuries are considered career-ending and possibly even life-threatening, because sepsis associated with any of these synovial structures carries a poor prognosis.

Clinical Findings

Most foot wounds that penetrate the dermal laminae produce acute signs of moderate to severe lameness. Puncture wounds that contaminate the bursa or DIP joint, and thus penetrate the frog, digital cushion, and DDFT, cause severe lameness. Even with careful inspection, it can be difficult to determine where the puncture site is, because the frog has a tendency to collapse around the tract on removal of the offending object.

Diagnosis

As discussed in the preceding section on septic arthritis of the DIP joint, exhaustive diagnostic evaluation is necessary to determine the depth of penetration and the structures involved. If a foreign body is still in place, then a survey film can help determine the depth of penetration and the structures involved.

When the object is no longer in situ but careful evaluation of the foot reveals the puncture site, then a metal probe can be used to evaluate the course and extent of the wound. The wound and surrounding horn should be thoroughly cleaned and aseptically prepared before a sterile probe is placed in the tract, and care must be taken to not penetrate normal tissue. A radiograph can be taken with the probe in place to define the extent of penetration.

Contrast Radiography Contrast fistulography or contrast arthrography can be especially useful in determining the extent of the injury. Contrast arthrography of the DIP joint or digital sheath may demonstrate a lack of integrity in the joint capsule or sheath by leakage of contrast material into the tissues along the puncture tract (see Figure 13-33).

Ultrasonography Diagnostic ultrasonography can be useful in assessing the extent of the injury in cases of wounds that involve the frog. If the foreign body has been removed, then careful evaluation through the frog may demonstrate gas shadows in the underlying soft tissues. The presence of gas shadows in the navicular bursa or DIP joint is confirmation of penetration and probable contamination. Diagnostic ultrasonography can also be used in conjunction with probe placement, because a metal probe creates a shadowing artifact that can be followed to determine the depth and proximity to key structures.

Synoviocentesis Samples of synovial fluid from the DIP joint, digital flexor tendon sheath, and navicular bursa should be obtained and submitted for total and differential blood cell counts, assessment of total protein concentration, and bacterial culture and antibiotic sensitivity testing. Even if one or more of these structures is contaminated, the prognosis is improved by early diagnosis and immediate and aggressive medical and surgical therapy.

Treatment

Medical therapy is similar to that described in the preceding section for septic arthritis of the DIP joint. It includes broad-spectrum systemic antibiotics, copious lavage of the synovial spaces involved, regional perfusion of antibiotics, and appropriate surgical debridement and lavage.

Surgery Diagnostic findings dictate which surgical procedure is performed. Puncture wounds to the sole can be explored and debrided with the horse standing. An area of sole 1 to 2 cm in diameter should be removed around the puncture site so that the tract can be completely explored. Where the wound penetrated the solar corium, the affected corium should be removed by sharp dissection. If there is bone involvement and possibly osteomyelitis, the abnormal bone should be removed by curettage. With wounds involving the frog that are not thought to involve a synovial structure, the cornified tissue overlying the puncture site should be removed to the full extent of the tract. A probe can be used to guide dissection.

In wounds that are thought to involve the navicular bursa, an endoscopic approach to the bursa is recommended for evaluation, debridement, and lavage. This procedure must be performed with the horse under general anesthesia. At the same time, regional perfusion with an appropriate antibiotic can be performed. The approach for endoscopic examination of the navicular bursa is described elsewhere.[26]

In addition to lavage of the bursa, the puncture wound through the frog is debrided by sharp dissection and all devitalized tissue is resected. After completion of the procedure, an antibiotic is injected into the bursa (and, if indicated, into the DIP joint). Occasionally, when extensive debridement of the wound is necessary, an opening through the DDFT must be made. These horses need to be managed much like those that undergo "streetnail" surgery.

Streetnail Surgery Historically, infections of the navicular bursa have been managed with a procedure known as *streetnail surgery*. This procedure involves cutting a funnel-shaped window in the frog and through the digital cushion to expose the DDFT. In the process, all devitalized tissue around the puncture wound is debrided (Figure 13-34). If the punc-

FIGURE 13-34 The streetnail procedure involves cutting a window through the frog to expose the deep flexor tendon.

ture wound appears to continue through the DDFT, then a longitudinal incision that separates the tendon fibers is made in the DDFT to allow exposure of the navicular bursa. Any portion of the tendon that appears necrotic or devitalized is resected. The navicular bursa is opened and lavaged.

Careful placement of the window through the DDFT over the flexor cortex of the navicular bone is critical to avoid entering the DIP joint distal to the navicular bone (through the impar ligament) or the palmar/plantar pouch of the DIP joint or the digital flexor tendon sheath proximal to the navicular bone. The flexor tendon sheath and DIP joint should be distended to determine whether there has been inadvertent penetration of either structure.

Postoperative care is critical with this procedure. A sterile bandage should be maintained over the incision site and changed daily. When the DIP joint or digital sheath is also involved, lavage of the joint and sheath and both regional perfusion and intraarticular or intrathecal antibiotic administration should be performed daily for a minimum of 3 days and then as necessary until clinical improvement is seen. A cancellous bone graft can be packed into the wound to obliterate the dead space, prevent ascending contamination, and provide a scaffolding into which cells can migrate during wound healing. Convalescence after the streetnail procedure is much longer than for horses treated using endoscopic lavage of the navicular bursa.

Lavage Alone When financial constraints limit more involved therapy, transcutaneous lavage of the navicular bursa, with ingress and egress of fluid and antibiotic solution via an 18-gauge, 3.5-inch spinal needle, can be attempted (Figure 13-35). However, it is important to impress upon the client that this procedure is likely to be effective only in early cases; and even then, the success rate is much lower than for surgical exploration and lavage.

Prognosis

In an early report, horses treated with appropriate surgical debridement within 4 days after injury had a reasonably good prognosis. Another author has reported good success with arthroscopic exploration of the navicular bursa in lieu of the more aggressive streetnail procedure. Horses with cases involving a hindlimb are more likely to return to previous activities than those with cases involving a forelimb. When the DDFT is involved, the prognosis is more guarded. The most common mistake made in the management of these cases is the use of a conservative approach.

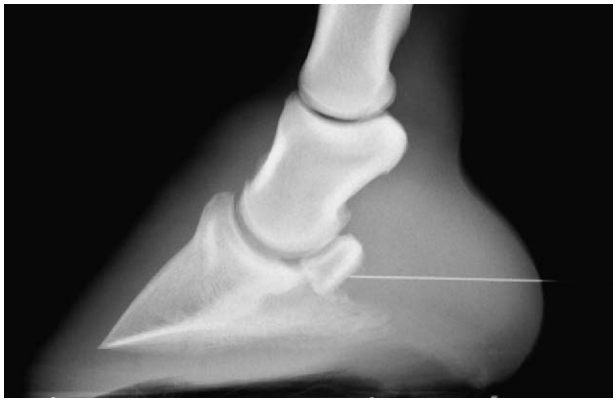


FIGURE 13-35 Needle placement in the navicular bursa for fluid collection and for through-and-through lavage.

Septic Pedal Osteitis

Septic pedal osteitis involves bone lysis of P3 as a result of bacterial infection. The presence of purulent exudate differentiates this condition from nonseptic pedal osteitis.[27] The most common cause is direct introduction of contaminated material via a penetrating wound. Chronic soft tissue infection (e.g., subsolar abscess) can also extend into the bone. Septic pedal osteitis is a recognized sequela of chronic laminitis. Occasionally, chronic infection of P3 leads to the development of a bony sequestrum.

Clinical Findings and Diagnosis

Clinical examination usually reveals a draining tract that, on exploration, leads to P3. Radiographs should be taken to assess the extent of bone involvement and to evaluate the affected area for the presence of a foreign body or sequestrum (Figure 13-36). If necessary, a fistulogram can be obtained to evaluate the area more thoroughly. A venogram may be indicated to assess blood flow to the affected area (see Chapter 17).

Treatment and Prognosis

Surgical drainage and debridement of the infected bone and soft tissue is indicated. Removal of all infected material is important for resolution. The approach should follow the draining tract (if present) and allow adequate exposure for the removal of all affected tissue and for ventral drainage of exudate (Figure 13-37). Complete resection of the draining tract is important. With a curette, it usually is easy to distinguish between normal and abnormal bone (which is soft and discolored, relative to healthy bone). Up to 25% of P3 can be removed without adversely affecting bone integrity.[27]

The horse should be administered systemic antibiotics and NSAIDs before surgery. Tourniquet placement and regional perfusion with antibiotics should be performed prior to surgery. It may be helpful to culture the infected bone before starting antibiotic therapy, although a mixed growth of

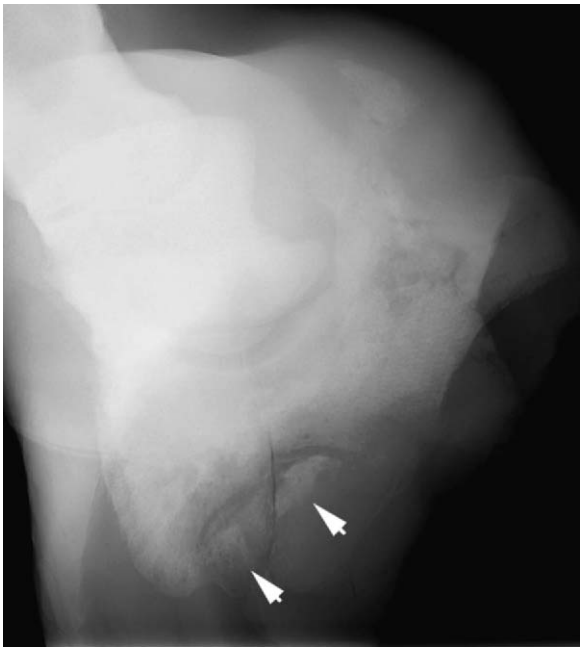


FIGURE 13-36 Septic pedal osteitis with sequestra formation.

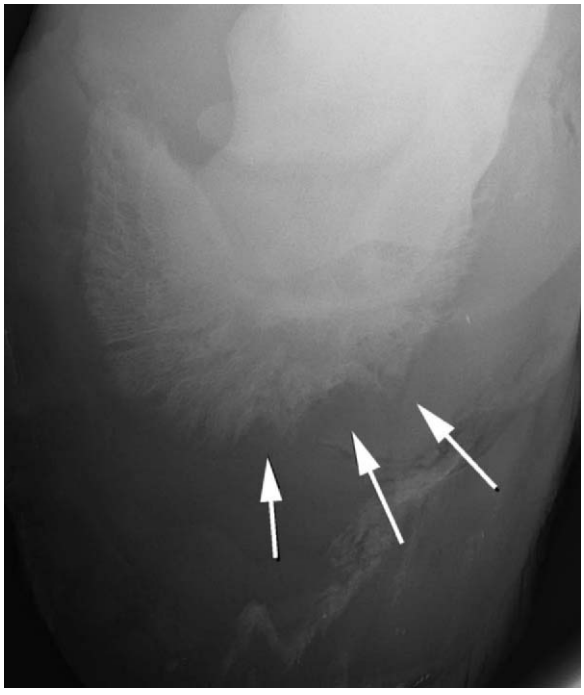


FIGURE 13-37 Septic pedal osteitis.

several bacterial species can be expected. After surgery, a sterile bandage is maintained during recovery and changed daily. Daily inspection of the surgery site is helpful to determine whether further debridement is necessary. After 5 to 7 days, a hospital plate can be placed on the foot to assist in daily wound care. The prognosis for soundness depends on the cause of the infection, its duration, and the adequacy of surgical debridement.

Infection of the Collateral Cartilages (Quittor)

Infection of the collateral cartilages of the foot is called *quittor*. Lacerations, puncture wounds, abscesses, and occasionally hoof wall cracks can involve the collateral cartilages of the foot, potentially causing cartilage necrosis, which may lead to infection of the cartilage. Quittor is most common in draft breeds.

Clinical Findings and Diagnosis

Chronic abscessation with intermittent purulent discharge from the infected cartilage is the usual clinical course. The diagnosis is based on the clinical signs of swelling and drainage from the affected cartilage. The primary differential diagnosis is chronic foot abscess. However, the drainage site for quittor usually is above the coronary band, whereas most submural abscesses ("gravel") drain from the coronary band itself. The lameness can be severe, especially when pressure increases from the accumulation of purulent material in the infected structures. But as with foot abscesses, once drainage occurs the lameness seems to diminish.

Treatment

The collateral cartilages have a poor blood supply, so healing of these tissues is slow. Furthermore, because most of the cartilage lies within the hoof capsule, it is difficult to establish effective drainage. Thus quittor is a surgical disease. The treatment of choice is surgical excision of all infected tissue and establishment of adequate ventral drainage, in conjunction with broad-spectrum antimicrobial therapy. The wound can be cultured, but it is likely to grow a mixed population of bacteria. Culture of the infected cartilage once it is removed at surgery will provide more accurate culture and sensitivity data.

A proximally based, curved incision is made to access the infected cartilage. Meticulous dissection is necessary, because the palmar pouch of the DIP joint is located just axial to the collateral cartilages. Honnas recommends placing the foot in traction, so as to place tension on the joint capsule, thereby retracting it away from the area of dissection.[28] Surgical dissection can also be assisted by injecting new methylene blue into the draining tract to clearly identify the affected tissue. Complete removal of all diseased cartilage may necessitate removal of a portion of the proximal hoof wall, which can be performed with a Dremel tool (Robert Bosch Tool Corporation, Racine, Wis.) or trephine, while taking care to preserve the germinal tissue of the coronary band.

Once the dissection is complete, the DIP joint should be distended with sterile balanced electrolyte solution and the wound assessed for fluid leakage that would indicate loss of integrity in the joint capsule. If the DIP joint was invaded, then the prognosis is decreased and the DIP joint should be treated as if contaminated. If the joint capsule can be closed, then an attempt should be made to do so. If closure is not possible, the wound should be treated as an open arthrotomy, as described for septic arthritis. The skin incision is closed primarily, if possible.

Prognosis

The prognosis is guarded, as it can be difficult to remove all of the infected tissue. The incision is at risk of dehiscence, which can complicate those cases in which the DIP joint was invaded while attempting to remove the affected tissue.

Intravenous Regional Perfusion for Septic Processes in the Digit

Infection can be a serious complication in wounds involving the foot. Foot infections can be difficult to treat because they often are polymicrobial, the organisms may be resistant to multiple commonly used antibiotics, and the infected area may be poorly vascularized (owing to its inherent structure or because swelling of the infected soft tissues within the rigid hoof capsule impedes vascular flow). Infection is enhanced in the presence of damaged tissue, hematoma formation, avascular bone, or foreign material (including soil and fecal matter).

Sepsis, vascular compromise, and a drop in pH as a consequence of inflammation and ischemia may prevent adequate delivery or activity of antibiotics in the infected tissue. Furthermore, vascular compromise increases the risk of sequestrum formation, which can promote bacterial proliferation.

Intravenous regional perfusion (IVRP) involves the delivery of an antibiotic to a selected region of the limb via the venous system. The infused volume is delivered under pressure to ensure distribution of the fluid to all vascular spaces in the region distal to the tourniquet. Retention of the antibiotic in the venous space for several minutes allows diffusion into surrounding tissues that may otherwise have inadequate blood flow. During IVRP of the distal limb, it is possible to achieve antibiotic concentrations in the tissues that are 25 to 50 times the minimum inhibitory concentration required to kill most pathogenic bacteria.[29] Thus, with this technique it is possible to achieve therapeutic concentrations of antibiotic even in necrotic tissue.

Technique

Regional perfusion of the digit can be performed in the standing horse.[30] A tourniquet or Esmarch bandage is applied to the fetlock, and the skin over the medial or lateral digital vein is aseptically prepared (Figure 13-38). A catheter is aseptically placed in the digital vein; most clinicians use a 20-gauge catheter, placed in the lateral digital vein. (Some clinicians place the catheter before applying the tourniquet.) An extension set is attached to the catheter, and infusion is begun.

Ideally, the antibiotic chosen is determined by culture and sensitivity results. Frequently, however, the results are not available when the first perfusion is performed. The clinician must therefore rely on clinical judgment and select an appropriate antibiotic based on the most likely organism or organisms involved. The antibiotics most commonly used for IVRP include amikacin (1 g), gentamicin (1 g), potassium penicillin (10 million units), timentin (1 g), and cephazolin (1 to 2 g). Whichever antibiotic is selected, the amount to be delivered (e.g., 1 g amikacin) is diluted in 20 mL of sterile balanced electrolyte solution. The antibiotic solution is infused over 30 to 60 seconds, but the tourniquet is left in place for a total of 20 to 30 minutes before it is removed.

Intravenous regional perfusion can be performed as a single treatment or repeated as often as necessary until clinical improvement is seen or the patency of the digital veins becomes compromised. Although uncommon with careful technique, the most common complication with IVRP is injury to the vasculature and soft tissues either from catheterization or perivascular leakage of the solution and subsequent local reaction.

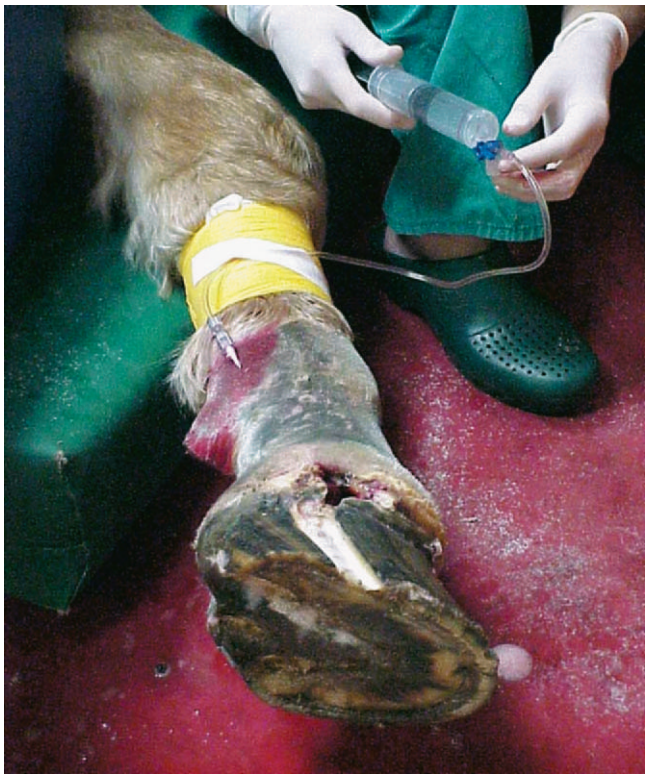


FIGURE 13-38 Regional perfusion of the digit is performed by placing a tourniquet at the fetlock.

Intraosseous Infusion

Intraosseous regional perfusion is an alternative to IVRP. It has the advantage of avoiding the repeated use of regional vessels. With this technique, the antibiotic solution is infused into the intraosseous space. After aseptic skin preparation, local anesthesia, and a stab incision through the skin and periosteum, a 4-mm–diameter hole is drilled through the cortex of the bone adjacent to the septic process. An intraosseous infusion needle or the male adaptor of an intravenous extension set is wedged into the hole and the antibiotic solution is infused into the medullary cavity. Unfortunately, the bones of the digit can be difficult to access for this procedure.

OTHER DISEASE PROCESSES

Keratoma

Keratoma is a benign, keratin-containing soft tissue mass that develops between the hoof wall and P3. Keratomas are thought to originate from the epidermal horn-producing cells of the coronary band,[31] although these lesions have not been extensively studied histologically. Keratomas have been described as having an outer epidermal layer and a core of dense, laminated keratin, with no evidence of inflammatory infiltrate.[31] The lack of consistent histologic evaluation makes it difficult to determine a common cause. Previous trauma and chronic irritation have been proposed as possible causes.

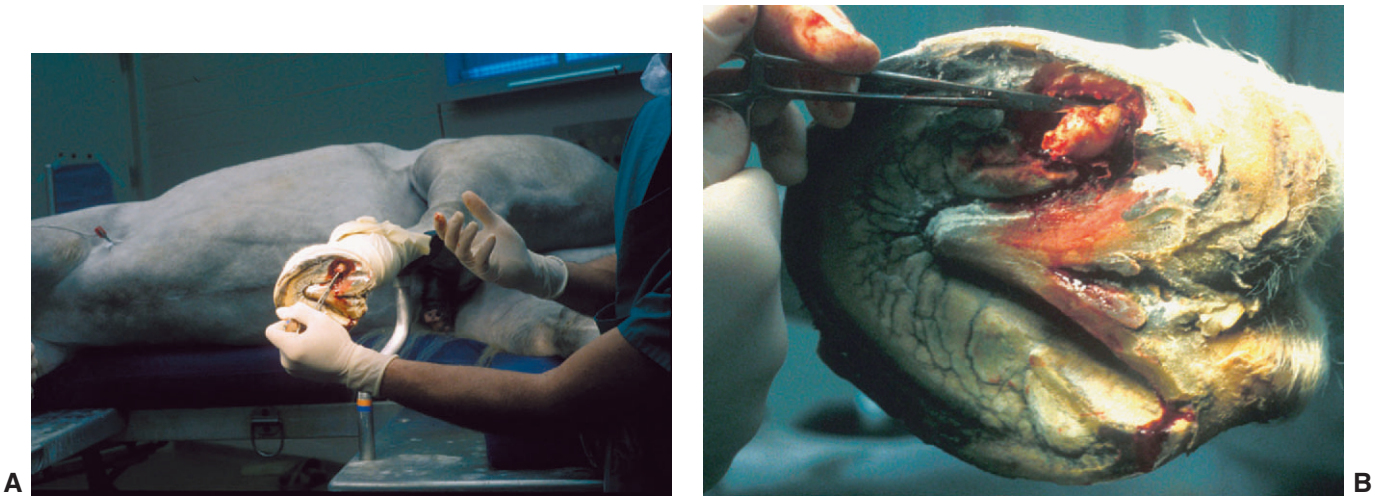


FIGURE 13-39 This keratoma was surgically removed via a window in the solar surface of the foot.

Keratomas act as space-occupying masses within the hoof capsule. Because they usually are contained within the epidermal lamellae on the inner aspect of the hoof wall, an area of bone resorption can be seen radiographically in most cases, most often at the toe.[32-35] In some cases, the keratoma extends to the solar surface of the foot, where it is evident at the white line.

Clinical Findings

Keratoma is an uncommon cause of lameness. Clinical signs are variable and may be related to the size of the keratoma. The lameness can be mild to moderate, depending on the degree of impingement on the sensitive laminae. Severe lameness may be caused by disruption of normal hoof architecture with subsequent abscess formation. The lameness may become progressively worse as the keratoma grows in size, increasing pressure on the sensitive laminae.

Keratomas can change the shape of the coronary band (i.e., create a bulge) in the affected area and, in more advanced cases, in the overlying hoof wall. In keratomas that extend down to the bearing surface of the wall, thoroughly cleaning the sole may demonstrate an island of horn 1 to 2 cm in diameter in the white line and deviation of the white line at that location toward the center of the foot (Figure 13-39).

Diagnosis

Palmar digital nerve block typically improves the lameness associated with a keratoma at the quarters, but it may be necessary to perform an abaxial sesamoidean nerve block to improve the lameness if the keratoma is located at the toe. Radiographic examination often demonstrates a smoothly demarcated radiolucent defect in the solar margin of P3. Standard 65-degree dorsopalmar views are useful to identify this bony change (Figure 13-40). Differential diagnoses include septic or nonseptic pedal osteitis, osseous cyst–like lesions, and benign or malignant neoplasms.

If a keratoma is suspected but there are no supporting radiographic findings, then nuclear scintigraphy may be helpful, because active bone remodeling will be apparent as increased radionuclide uptake. Diagnostic ultrasonography can also be helpful in identifying keratomas at the coronary band.[34]

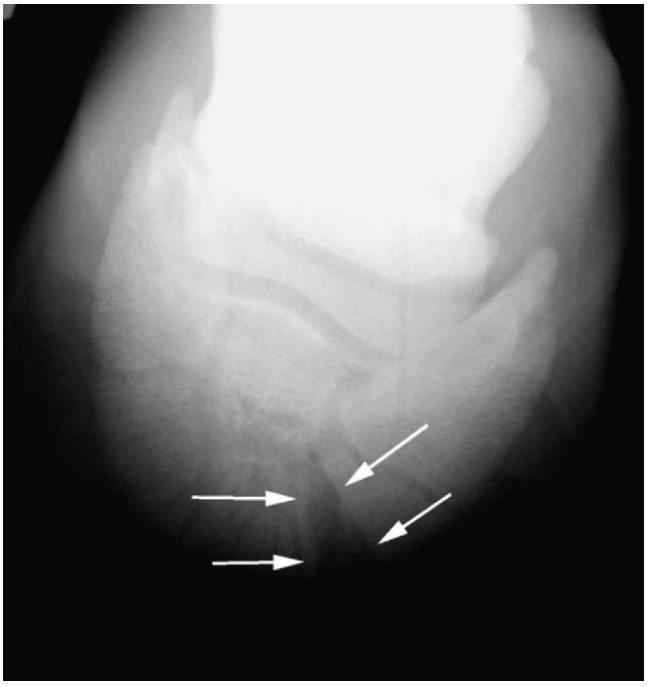


FIGURE 13-40 The keratoma acts as a space-occupying mass; the resulting bone resorption creates a radiolucent area on the third phalanx (P3).

Treatment

The treatment of choice is surgical removal of the keratoma, which is accomplished by removal of the hoof wall over the mass. Historically, a full-length strip of hoof wall over the keratoma is removed. However, removing this much of the wall can create excessive instability in the hoof wall and prolong convalescence.

If the keratoma can be isolated to a specific area using radiography or ultrasonography (at the coronary band), then the hoof wall at just that site can be removed to allow access to the mass. Removal of the overlying horn can be accomplished using a Dremel tool or a trephine. If more hoof wall needs to be removed, then the Dremel tool can be used to access and remove the entire keratoma (Figure 13-41).

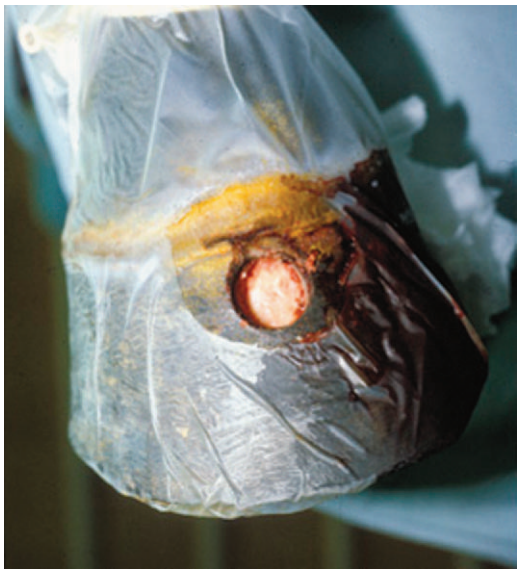


FIGURE 13-41 This keratoma was approached using a trephine to remove the hoof wall over the mass.

Management of the hoof wall defect is as described for any wound to the foot (see Chapter 12). Stability can be increased by the use of a bar shoe with clips. Once adequate keratinization of the defect has occurred, then more stability can be provided by reconstruction of the hoof wall with composite adhesives.

Prognosis

The prognosis for future athletic performance is good, although recurrence of the mass and return of the lameness can occur if the entire lesion was not resected.

Neoplasia

Neoplasia involving the equine foot is rare. Of the few cases reported in the literature, melanoma is the most common type of neoplasm affecting the foot.[36-38] Squamous cell carcinoma involving the foot also is reported.[39,40]

Melanoma

Although this dermal tumor is commonly found in other parts of the horse's body, it is reported only rarely in the foot. While most melanomas in the horse are benign or only minimally invasive, those reported to involve the foot invariably have been invasive (i.e., malignant melanoma).[36-38]

In cases of melanoma involving the foot, the presenting complaint was a draining tract or hoof wall defect.[36-38] The clinical course was protracted—as long as 2 years in some cases. In each case, radiography revealed marked osteolysis of P3. Thus, cases of recurrent foot abscess with marked osteolysis of P3 should be carefully evaluated for the possibility of neoplasia.[38]

Surgical biopsy of the tissue in the area of bone resorption is indicated in horses with this clinical picture. Extensive removal of the abnormal tissue is the treatment of choice; however, it has not been curative in the few reported cases. All reported cases eventually resulted in euthanasia. Surgical resection, local chemotherapeutics, immunotherapeutics, or a combination of therapies may yield better results.[38]

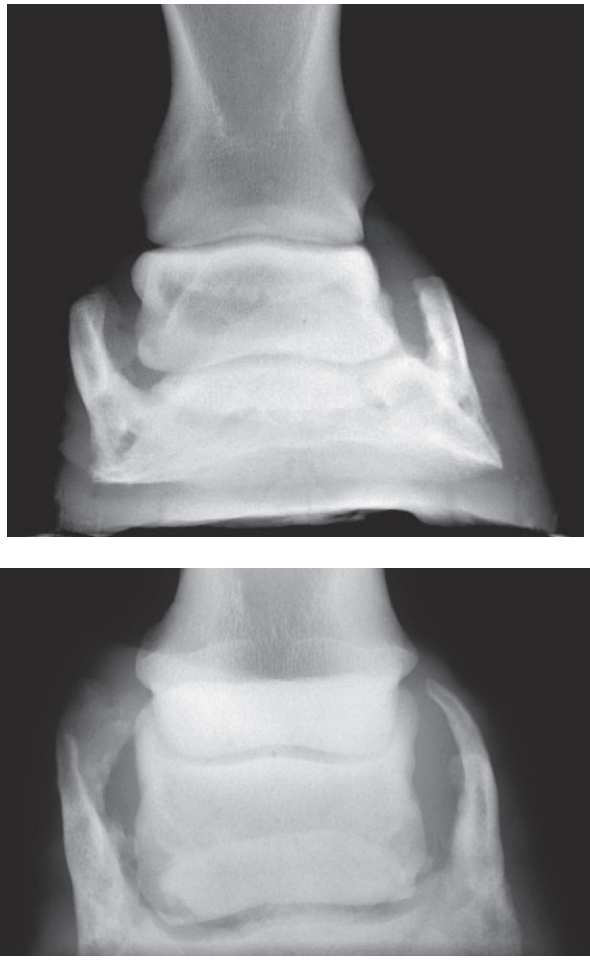


FIGURE 13-42 Radiographically apparent ossification of the collateral cartilages (sidebone).

Ossification of the Collateral Cartilages (Sidebone)

Ossification of the collateral cartilages is called *sidebone*. The degree of ossification can vary considerably from horse to horse. Ossification occurs more often in the forefeet and more often on the lateral cartilage than on the medial. Ossification tends to be more extensive in larger breeds than in lighter breeds; draft breeds have an unusually high incidence of sidebone.

Sidebone can develop secondary to traumatic wounds that involve the collateral cartilage. Most often, however, the cause is unknown. Chronic trauma from poor foot conformation, poor shoeing, or imbalanced feet has been implicated as a predisposing factor.

Clinical Findings and Diagnosis

Lameness associated with sidebone is rare. For sidebone to be considered as a source of lameness, there should be clinical evidence of pain, heat, and swelling involving the cartilage and radiographic evidence of abnormal ossification of the cartilage (Figure 13-42). Fractures of the ossified cartilage can occur, but this lesion must not be confused with radiolucent

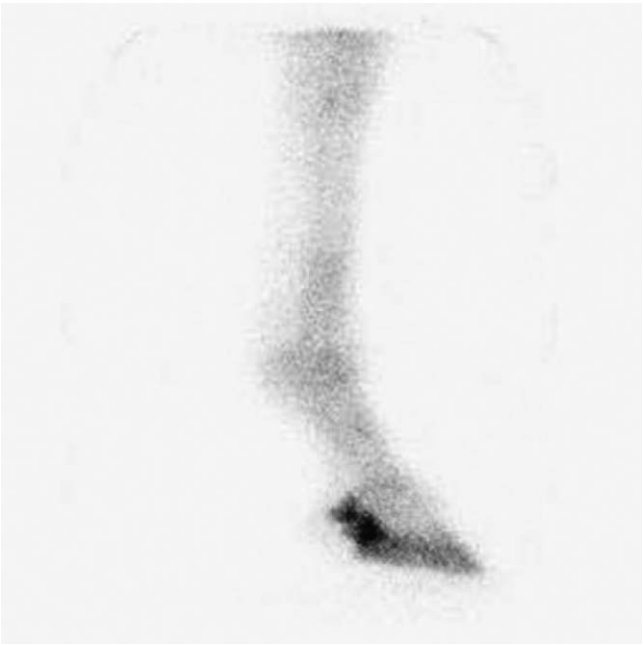


FIGURE 13-43 Nuclear scintigraphy of a fractured sidebone, with bone remodeling.

lines between separate centers of ossification that may be found in the collateral cartilages.

Diagnosis of clinically active sidebone is necessary before treatment is instituted. Blocking the palmar digital nerve on the same side of the foot as the ossified cartilage should eliminate the lameness if it is caused by the sidebone. However, improvement of the lameness after nerve block is merely suggestive of clinically active sidebone; it is not confirmation of a clinical problem. Nuclear scintigraphy can be helpful by establishing the presence of active, intense bone remodeling associated with a fracture of the sidebone (Figure 13-43).

Treatment and Prognosis

Treatment of acute injuries to the collateral cartilages should include rest and anti-inflammatory therapy. Chronic lameness associated with sidebone may be managed using a shoe with slippered heels, in conjunction with rasping or grooving of the wall below the cartilage. Collateral cartilage fractures have been managed with shock-wave therapy with some success. In unresponsive cases of lameness associated with sidebone, a unilateral digital neurectomy may be performed. The prognosis for clinically significant sidebone depends on the underlying cause and whether that problem can be effectively managed.

SUBSOLAR ABSCESS

W. Tyler Brady

A subsolar abscess (foot abscess) is a condition in which a localized pocket of infection accumulates between the internal surface of the sole and the solar corium. It usually courses between these two layers and erupts at the level of the heel bulbs, often causing the horse great discomfort and obvious lameness. Sometimes it is confused with a "gravel,"

FIGURE 13-44 This abscess found at the point of the frog before the wound was flushed.

which travels proximally between the laminar corium and the white line to exit the coronary band.[41] Subsolar abscesses can be very rewarding to treat, meeting with great success, but if left untreated can affect other structures in the foot, leading to chronic problems and serious lameness issues.

Subsolar abscesses form when the structural integrity of the foot is compromised, allowing debris and bacteria to proliferate in the sensitive structures. This may be preceded by a close nail, hoof wall defects, chronic laminitis, or abnormally wet conditions allowing the sole to become excessively pliable. Horses usually have an acute onset of moderate to marked lameness and may or may not be weight-bearing.

Upon examination, it is not uncommon to find swelling in the distal limb of the affected foot. Typically, hydrotherapy and a support wrap of the limb will resolve this swelling once the abscess is treated. Palpation of the limb may reveal tenderness at the level of the heel bulbs or the coronary band. Often, if the abscess is near the skin surface, hair loss, heat, localized swelling, and tenderness are evident. The foot may seem warm, and an elevated digital pulse may also be present.[42] Percussion of the hoof wall with a farrier's hammer or a closed set of hoof testers may point out a general area in which the horse is sensitive.[43] The foot should be cleaned and the superficial layers of sole should be pared with a hoof knife to reveal evidence of a draining tract or wound.[42] Hoof testers can then be applied to localize areas of tenderness. Because there is usually a marked pain response, hoof testers should be applied lightly at first.

When the most sensitive area is localized, many times digital pressure with the thumbs can further isolate the area to investigate. Using a sharp hoof knife or abscess knife, the examiner cuts into the area using a circular motion to allow for adequate drainage of the abscess. Caution should be taken not to remove more tissue than is needed or healing time will be prolonged. Periodically, a horse does not respond to hoof testers, which may be due to thick hoof walls or an extremely hard foot. In such a case, it may be helpful to poultice the foot for 12 to 24 hours.[44] Another option is to soak the foot in warm water and magnesium sulfate 15 to 20 minutes daily for 1 or 2 days and repeat the examination process (Figures 13-44 and 13-45).

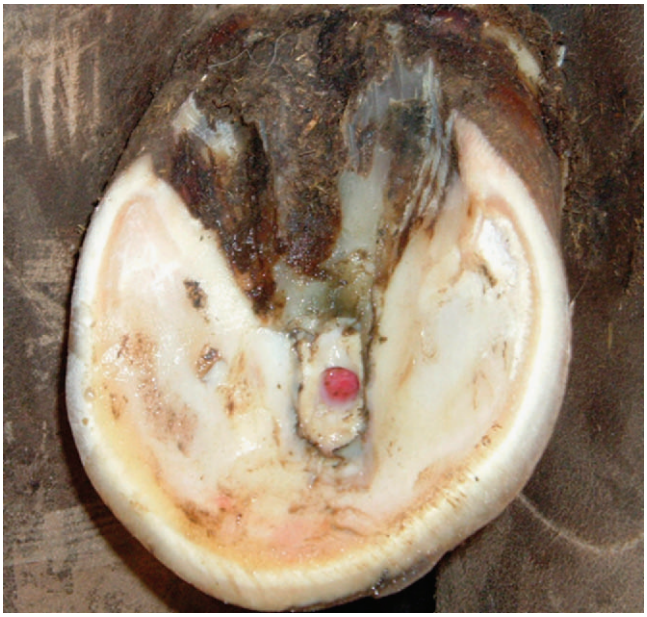


FIGURE 13-45 The foot in Figure 13-44 has been cleaned and irrigated.

In a situation in which the abscess is not easily discovered, radiographic examination may reveal a fluid or gas line between the internal surface of the sole and the distal phalanx. A minimum of two views taken at 90-degree angles to each other should be obtained for a thorough examination[42]; most commonly, lateromedial and dorsopalmar or dorsoplantar views are used.

Once the abscess is opened for drainage, the wound is flushed with an antiseptic wash and bandaged. Daily foot soaks for 3 to 4 days in warm water and magnesium sulfate may help to draw out infection and reduce any swelling in the structures above the foot. This has questionable value to some practitioners.[45] Common antiseptics include dilute iodine or chlorhexidine solutions. Bandages can be made of gauze pads or cotton and duct tape. Many horse owners choose to use a diaper as a foot bandage, but they can be costly. Most horses will show significant improvement within the first 24 hours after draining of the abscess if unexpected structures are not affected. If the sole is undermined so extensively that healing may be delayed, a hospital plate may be indicated to allow enough time for granulation to take place. Packing the foot with antiseptic solution–laden cotton or gauze pads will help in keeping the wound clean. Drying agents such as 2% formalin, 2% phenol, or tincture of iodine can also be considered.[45]

Because a subsolar abscess is typically a local infection, systemic antibiotics are not usually administered. If there is significant swelling in the distal limb and there is evidence of cellulitis, systemic antibiotics should be provided. Most bacteria entering the foot are *Clostridium* spp.; therefore tetanus prophylaxis should be updated if not current.[46] Nonsteroidal antiinflammatory agents such as phenylbutazone or flunixin meglumine should be used as needed, but sparingly due to possible side effects.

Most uncomplicated subsolar abscesses heal within 5 to 7 days and should not affect the overall long-term performance of the horse. If a horse presents with repeated foot abscesses, the overall health, shoeing, training, and environmental conditions should be explored thoroughly.

VENOGRAPHY AS A TOOL FOR GUIDING SURGERY TO THE FOOT

Bruce E. Lyle

Surgical exploration of the internal structures of the foot strikes fear in owners and trainers, making the recommendation difficult for the attending veterinarian and treatment acceptance difficult for those in charge. Options are limited and time is of the essence to return the horse to soundness. A thoughtful analysis of the pathophysiology affecting the foot is enhanced by venography, and venography provides the veterinarian with a visual aide for the owner or trainer and a roadmap for surgical exploration. Early recognition of submural disorders results in less bone and soft tissue being removed, shorter surgery time, limited scar formation, and less likelihood of the effects of chronic pain, including creation or exacerbation of contralateral limb laminitis. The diagnosis and treatment of coffin bone fractures is well documented and is discussed previously in this chapter. This section focuses on assisting the reader in early recognition of septic osteitis of the third phalanx in laminitic and nonlaminitic patients.

As discussed in Chapter 17, venography offers a world of discovery within the hoof capsule. An important benefit of venography is that it allows recognition of submural disorders before they become evident with plain-film radiography. Interpretation of the findings provides tremendous challenge and opportunity in equal measure. MRI may surpass venography in the amount of detail it provides, but the expense and limited availability of MRI mean that venography will continue to be a valuable tool for the clinician wishing to pursue diagnosis and treatment of foot disorders in horses.

Submural foot conditions are discussed at length earlier in this chapter. This section focuses on early recognition and treatment of septic osteitis of P3, using venography to guide evaluation and treatment decisions. Nonlaminitic horses and those with laminitis are discussed separately. A case with much the same presentation involving a submural keratoma also is presented to further illustrate the utility of venography in differentiating this cause of severe lameness and purulent drainage from septic osteitis. (The principles, technique, and applications of venography are discussed in Chapter 17.)

Septic Pedal Osteitis

Septic pedal osteitis can result from a variety of insults to P3 and its overlying corium. A common denominator, regardless of the cause, is altered blood flow to or from the affected area of the foot. Hence, venography is a tremendously useful tool in early recognition of septic pedal osteitis, because it can highlight the vascular defect several days, and even weeks, before bony lesions become apparent radiographically.

Disease Process and Pathophysiology

The venogram helps elucidate the pathophysiologies at play in the disease. The initiating cause (contusion, penetration, or

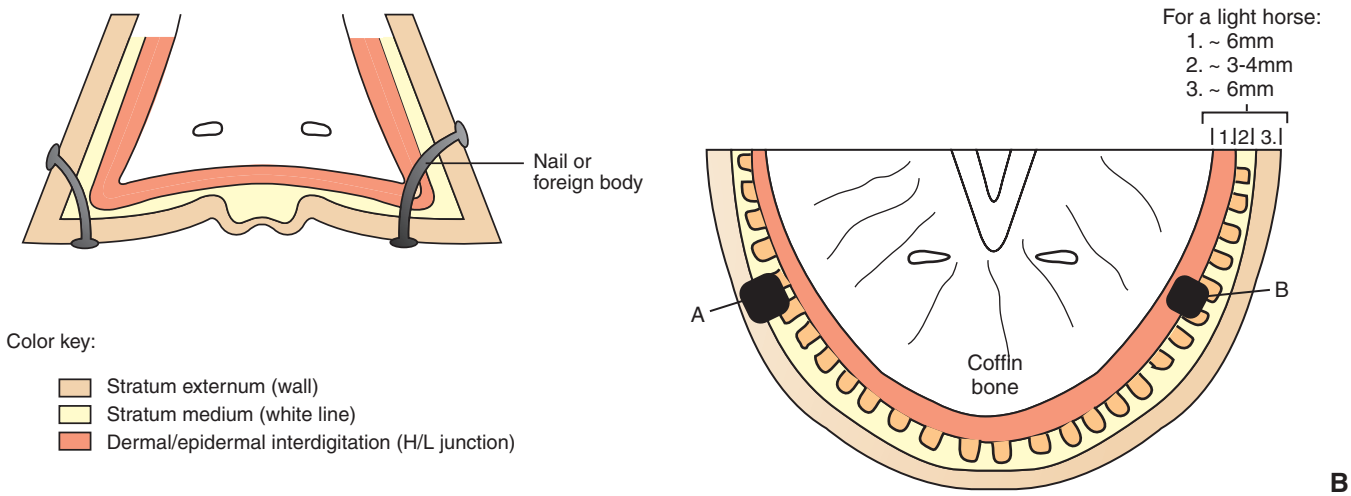


FIGURE 13-46 **A,** A nail or other foreign body insult creates the potential for osteitis when it penetrates deep to the dermal-epidermal interface. **B,** Insults (*A*) into the dermal-epidermal interface can cause hemorrhage and abscessation. Deep *(B)* infections and osteitis/osteomyelitis occur when the insult is axial to the dermal lamellar interfacing.

thrombi) can create vascular compression, shear, or blockage with a resulting inflammatory cascade. Swelling is trying to occur within the confines of the hoof capsule, causing pain and further vascular compromise in the local environment (Figure 13-46). As the inflammatory process proceeds, arterial pressures may remain high enough to provide blood to the affected area; however, tissue destruction and increased interstitial pressures cause venous compression and decreased fluid drainage, thus contributing to localized tissue necrosis and pus production. This may well lead to pressures high enough to inhibit arterial influx, creating even more pain until relief is granted. Corium death or tearing from the insult result in the local production of granulation or scar tissue, the quality of which is determined by the degree of sepsis in the local environment. Persisting pathology results in continued swelling peripheral to the lesion. The longer the delay is in removal of the necrotic bone, the more extensively the soft tissues are affected, and the less likely it is for a full return to soundness, as the peripheral swelling tends to migrate proximally, resulting in varying degrees of destruction of the outer horn generating cells at the coronary band.

This event is more devastating and progressive in the laminitic foot than in the nonlaminitic foot. The increase in the horn-lamellar zone generally does not progress in the nonlaminitic foot with septic osteitis, whereas the laminitic or founder foot continues to swell and destabilize as a result of loss of local nutrition to the laminae, the parietal and circumflex vessels of which are compromised in cases of severe, septic laminitis. The degree of soft tissue and bone destruction can likely be affected by the introduced organisms, their enzymatic capabilities, and the degree of immune response incited.

Limitations of Antibiotic Therapy

Venography provides convincing evidence as to why antibiotics are inadequate as the only therapy in cases of septic pedal osteitis. The hallmark lesion on venography is an absence of contrast material (a void or filling defect) in the region of the infected or necrotic bone (see Figure 13-49). Achieving effective levels of drug would seem unlikely in these areas with compromised circulation and avascular bone.

Venogram versus Fistulogram

Fistulograms, using a tom cat catheter, can be a useful adjunct to plain-film radiography in the assessment of persistent draining tracts, especially with wounds that fail to heal completely after several weeks. Fistulograms do not provide anywhere near the amount of useful information that a good venogram does, however.

The fistulogram can outline the course of a draining tract and thus guide surgical exploration of the tract. However, the tract opening may be far removed from the primary site of infection. Furthermore, the presence of a draining tract is an indication of chronicity, and the goal is to intervene before the disease process advances this far. Any introduction of a catheter or probe should be done using aseptic technique, so as to prevent the introduction of more potentially pathogenic bacteria into an already compromised environment.

SEPTIC PEDAL OSTEITIS IN NONLAMINITIC PATIENTS

Septic osteitis involving P3 can result from blunt trauma, from penetrating injuries to the foot, or as a result of bacterial embolization from another site of sepsis in the body. It is likely that a scar or other insult significant enough to impede normal circulation can predispose the site to colonization.

Infectious osteitis can affect any age, breed, sex, or discipline of horse. Foals may be affected, but infectious osteitis in foals is uncommon in the author's experience when compared with sepsis of other sites in foals and with the incidence of coffin bone infections in horses of training or breeding age (Figure 13-47).

History and Clinical Presentation

The most common presentation is that of a penetrating wound to the foot, usually from a misplaced shoe nail (however careful the farrier may have been). In cases

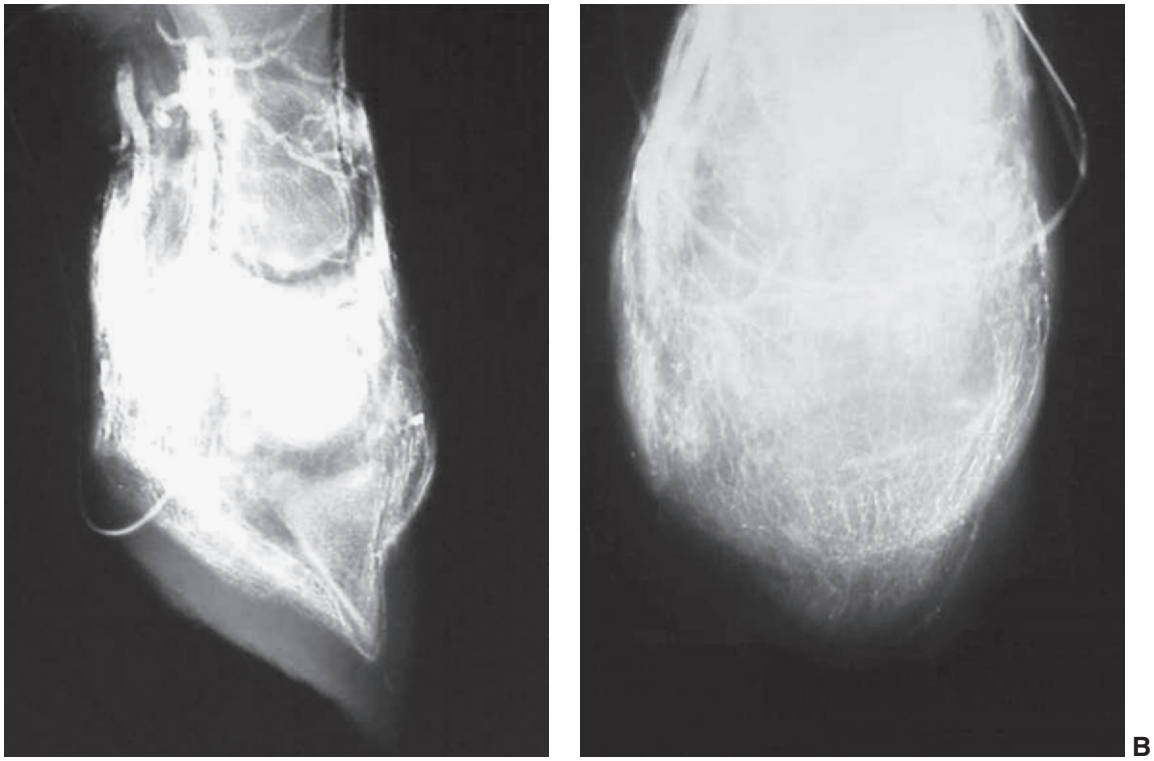


FIGURE 13-47 **A,** This lateral medial venogram is of a 1-week-old foal and looks basically normal, although there is questionable loss of dye density about halfway between the opaque tubing below the heel and the apex of the coffin bone. Note the size of the coffin joint. **B,** This solar margin view of the venogram shows the affected side void of dye in the circumflex with loss of dye density continuing toward the wing. On the original film, a small sequestra could be made out and confirmed with additional views (not shown). The infection and demineralization extended to the coffin joint and navicular bone. Follow-up radiographs showed severe new bone formation and pathologic fracture of the navicular bone. The foal healed, was able to be turned out with its mother, and as a yearling was pasture sound with no special care. To the author's knowledge, it has not been trained but is still pasture sound.

involving a "hot nail," the history usually indicates that the horse was shod 3 to 7 days before the lameness first appeared. The vast majority of "hot nails" and, for that matter, other foreign body penetrations to the foot resolve uneventfully once the offending object is removed and the foot is treated appropriately within 24 hours of the onset of lameness. Appropriate treatment generally includes debridement and drainage of the puncture tract and topical antibiotic or poultice therapy.

In the case of deep infections, often the original insult occurred deep to the dermal/epidermal interfacing. Another possible cause is an insult to the laminar interfacing that was left untreated for 48 to 72 hours in hopes that the lesion would create its own drainage port. The infectious process causes corium inflammation, and pain from this is interpreted to be from an abscess; however, the insult has not been walled off as is the case with abscessation. In two cases in the author's experience, the veterinarian or farrier had inserted a needle into the suspect area (through the sole) in the hope of finding an abscess. This procedure is to be discouraged, because there is a significant risk of carrying infectious agents into the closed environment of the hoof, which may introduce sepsis, or contribute to it, especially in the already compromised underlying soft tissue. A more troublesome subset of cases are those in which the foreign object penetrated to the bone and seeded the damaged bone and soft tissue with bacteria. If the veterinarian is able to remove the penetrating object and assess the damage initially, additional diagnostics are likely not necessary. Often, however, the owner or trainer has pulled out the foreign body, and venography, or sonography if in the frog area, is necessary to determine the depth to which the injury occurred.

The typical presentation in these cases is moderate to severe lameness (grade 3 to 5 out of 5) that does not improve with routine treatment for a suspected foot abscess, even when the regimen includes systemic antibiotic therapy.

Horses with lesions involving vascular abnormalities (e.g., bruising and/or thrombus formation from contusions or over-reaching injuries) often have a history of acute, severe lameness (grade 4 out of 5) that failed to improve with bute and poulticing, and with no obvious penetrating injury. Depending on the duration of the problem, drainage may be evident, as may deformation of the capsule, coronary band, or hairline above the coronary band.

Clinical Evaluation

Gentle use of hoof testers can help localize the site of pain. However, a positive response may be present over more than half of the foot and perhaps even the entire foot if the insult was severe or long-standing. Hoof tester response will also be a function of hoof mass and sole depth. That is, a horse with 25 mm sole shadow or sole depth (which can be verified during the radiographic part of the examination) is much less likely to respond to a hoof tester than is the flat-footed, shallow-soled (≤10 mm) horse.

Infection or avascular necrosis of P3 should be strongly suspected in any case of unilateral lameness that is localized to the foot, does not resolve in 10 to 14 days, is not accompanied by distortion of the hoof wall, or is accompanied by purulent exudate at the coronary band or through an opening made by the veterinarian or farrier. (NOTE: Distortion of the overlying hoof wall can develop if the problem is allowed to persist untreated for 3 to 6 weeks. This condition is more likely with septic osteitis in the quarter or heel area.)

Fever (rectal temperature of 39.4° to 40° C [103° to 104° F]) may be found, but it is not uncommon for the rectal temperature to be normal or only slightly elevated (in the range of 38.2° to 38.9° C [100.8° to 102° F]), especially in horses receiving phenylbutazone. These horses may still be moderately lame (grade 2 to 3 out of 5), despite NSAID therapy. If the history and presenting signs are suggestive of foot sepsis, then further investigation is warranted. When foot sepsis is severe enough to cause signs of systemic illness, the white blood cell count and plasma fibrinogen concentration may be elevated. This may be a function of the bacteria involved rather than the location of the sepsis. Typically, however, septic pedal osteitis does not cause any abnormalities on the hemogram.

Eruption of a draining tract above the hairline (i.e., proximal to the hoof wall) can be seen with septic pedal osteitis, but it can also occur with quittor (sepsis or necrosis of the ungual cartilage), sepsis or necrosis of the deep digital flexor tendon, sepsis involving the navicular bone or bursa, and necrotizing corns or compressed bar disease. The presence of a draining tract with any of these conditions signifies an advanced pathologic condition. Venography, ultrasonography, and now MRI can help identify these problems earlier in their course, thereby allowing less invasive treatment (even when surgery is necessary) than is required when diagnosis and treatment are delayed.

Diagnostic Imaging

Regardless of the cause of septic pedal osteitis, early recognition and prompt treatment are essential to minimize incited and iatrogenic damage to the foot. In addition to careful physical examination of the foot, diagnostic imaging is important for identifying and characterizing these lesions. Both plain-film (i.e., routine) radiography and venography are important in early identification of these lesions and in surgical planning.

Routine Radiography

Routine radiography is important in these cases, but its limitations must be recognized. Early in the course of septic pedal osteitis, plain-film radiography is most useful for ruling out P3 fractures. In more long-standing cases (>14 days), abnormalities that can be identified on plain films include demineralization, sequestration, pathologic fractures, gas lines, and asymmetrical horn-lamellar (H-L) zones (Figures 13-48 and 13-49).

Radiographs taken early in the course of disease can be difficult to interpret if there is a physical defect in the wall or sole in the area of interest caused by exploration by the veterinarian or farrier. Even when there is not a large defect in the hoof capsule, it can be difficult to evaluate the margin of P3 on most views unless there is sequestration or marked demineralization, which generally takes 14 to 20 days to become evident radiographically. Even so, radiographic changes are not necessarily evident in 2 to 3 weeks. Prior to recognizing and beginning to understand the usefulness of venography, it took this author 6 to 8 weeks to recognize enough bone change to pursue aggressive surgical debridement. Delaying surgical debridement may allow more extensive destruction to occur, increase the risk of contralateral limb laminitis, and require such extensive tissue removal that the hoof capsule, P3, or even the coffin joint is destabilized (Figure 13-50).

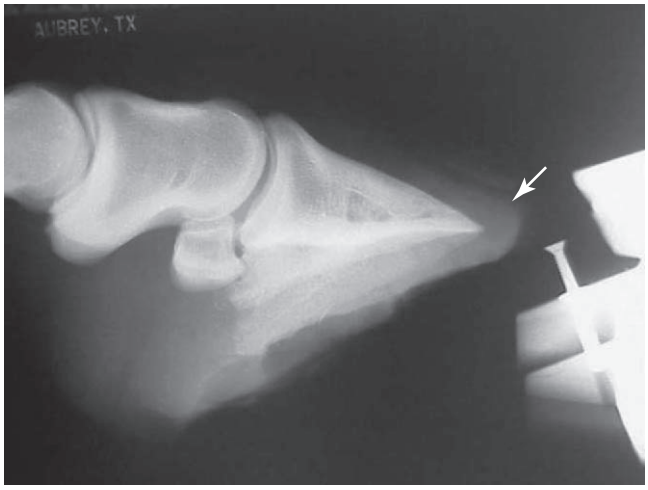


FIGURE 13-48 Note the gas density between the horn and the corium. This is a result of secondary laminar necrosis. Note that the "L" of the "HL" zone is the same as the sole shadow, suggesting that this disease process is different than laminitis.

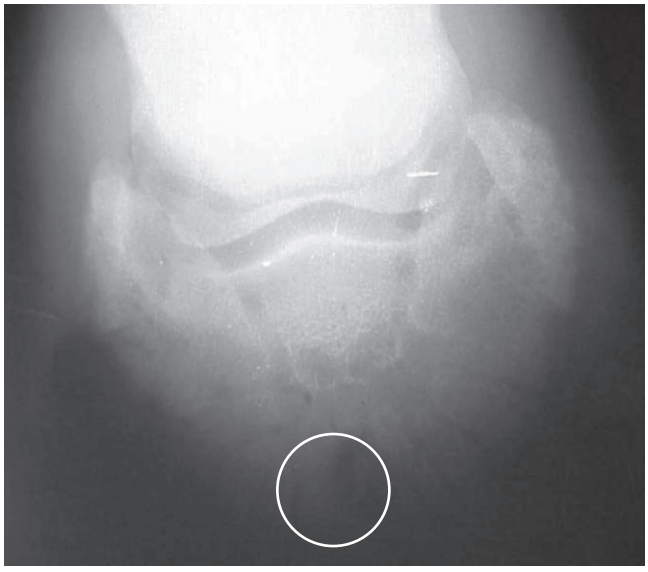


FIGURE 13-49 This solar margin anteroposterior view shows a well-demarcated sequestrate in the same case as seen in Figure 13-50.

Venography

Venography is a sensitive indicator of the need for surgical exploration and it encourages earlier intervention than when

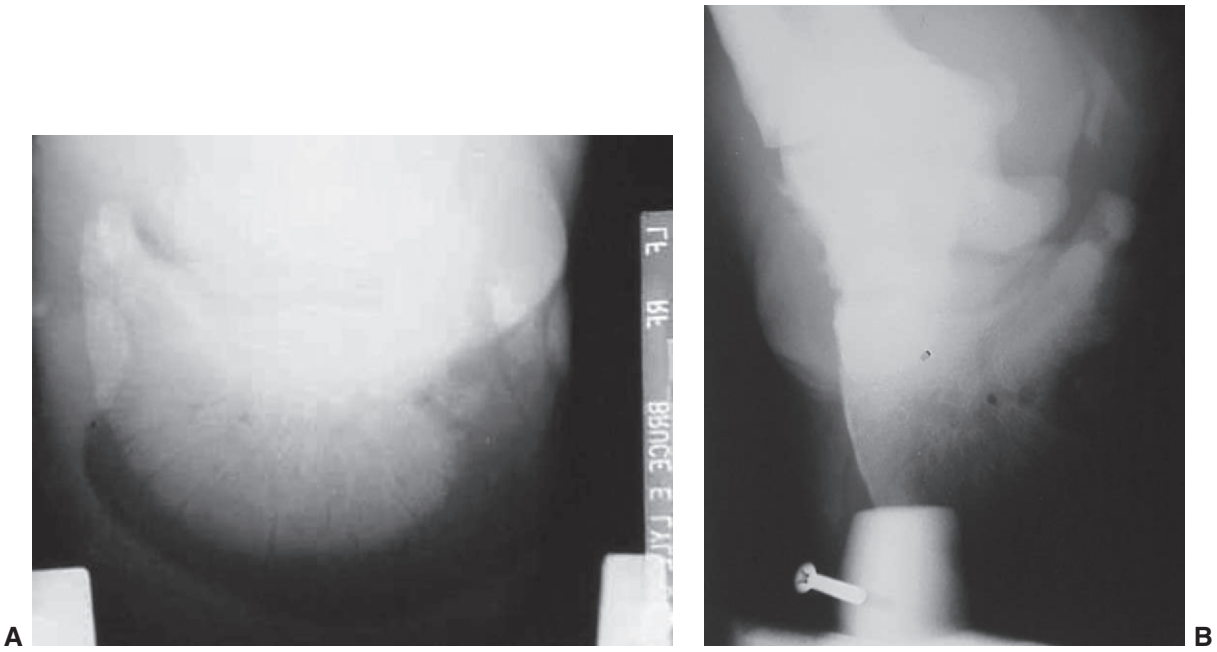


FIGURE 13-50 **A,** Margin view showing extensive osteolysis in the quarter region of the coffin bone wing. A sequestrum is visible and the borders of the bone are lytic and proliferative, suggesting the presence of osteomyelitis. Note the linear distance from the solar margin to the coffin joint is reduced by about 50% as a result of the disease process. **B,** This 60-degree dorsopalmar oblique view shows the sequestrum, osteolysis, and osteoproliferation. The radiopaque area directly above the lesion consists of thick, swollen skin folds—another characteristic of osteomyelitis of the third phalanx and/or the navicular bone.

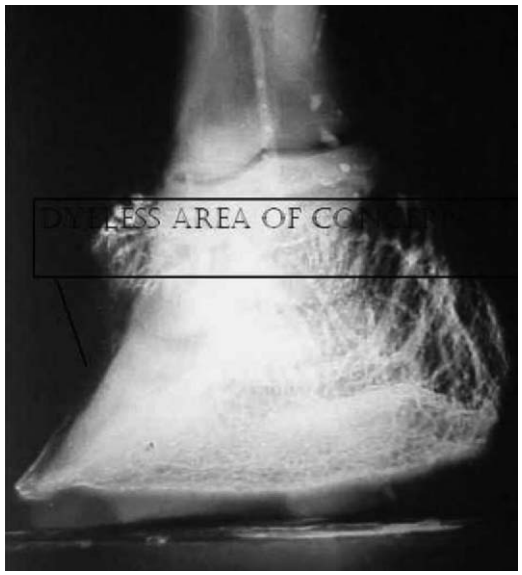


FIGURE 13-51 The loss of dye midway up the coffin bone suggests a submural problem rather than a subsolar one.

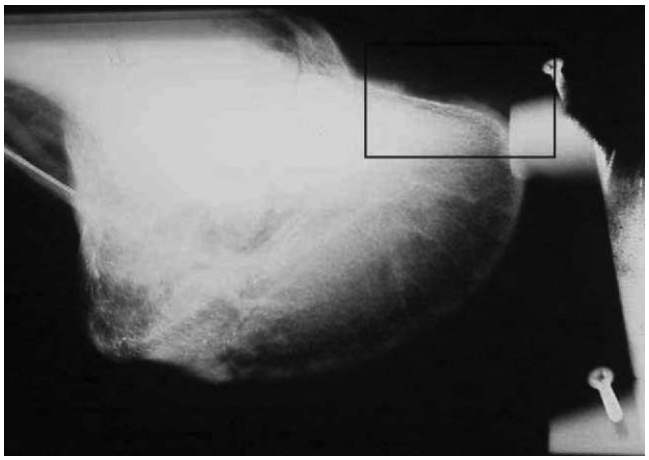


FIGURE 13-52 In this nail venogram with laminar void, the oblique view demonstrates the dyve void above the circumflex zone, suggesting a surgical approach through the wall rather than through the sole.

plain films are used alone. The technique is the same as that described for use in the laminitic patient (see Chapter 17).

Radiographic Views

The area most commonly affected in septic pedal osteitis is the region of the circumflex vessels, although defects can occur anywhere in the vascular network (Figure 13-51). This area is best evaluated by taking multiple views with the horse standing on a 3- to 4-inch–high wooden block. Several projections are taken circumferentially, including lateromedial, dorsopalmar/plantar, and various oblique views.

Depending on the conformation of the foot, a solar margin view, taken with the plate and x-ray beam perpendicular to one another, may also be helpful. Oblique views with the digit in the flexed position should be included to localize lesions associated with the quarters and wings of P3 (Figures 13-52 and 13-53), although these projections are not particularly helpful in directing the surgical approach.

Surgical Procedure

Septic pedal osteitis is a surgical lesion; it requires surgical exploration, debridement, and drainage. Healing time is related to lesion size, so early recognition and intervention is

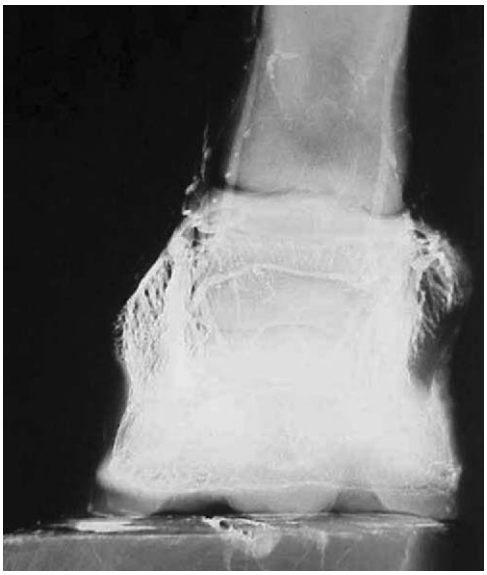


FIGURE 13-53 This view provides little surgical direction, but it is interesting when compared with a venogram of a sinker with osteitis.

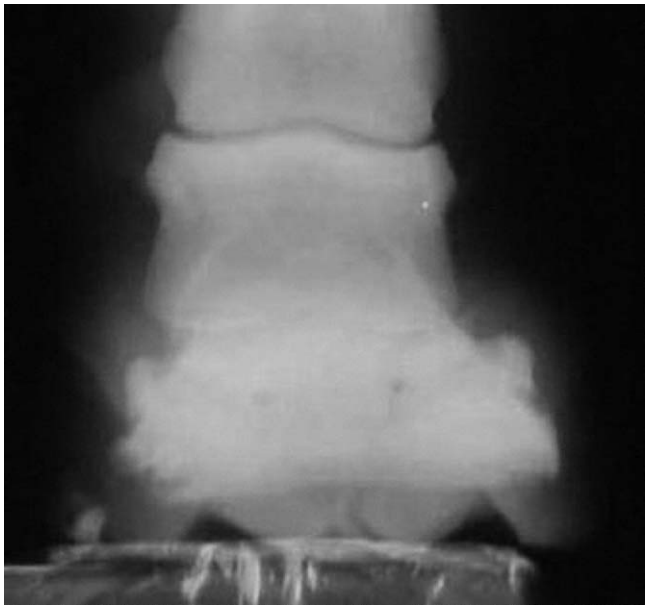


FIGURE 13-54 This flat-standing anterior-palmar view demonstrates lysis on the affected side versus the normal side. The H-L zone is minimally thickened compared to sinker cases.

important in returning the horse to soundness as quickly as possible. The biggest problem caused by a delay in surgical intervention is the tendency for bone deep to the lesion to become malacotic, beyond what is recognizable on plain films or venograms. Debridement of P3 then becomes so extensive that the coffin joint, collateral cartilage, DDFT, and navicular bursa are at risk of being invaded. Stability of P3 can become so compromised that secondary wing fractures or coffin joint luxation, or both, can occur (Figure 13-54). These cases then become candidates for phalangeal amputation. In contrast, intervention early in the course of the disease can be very rewarding.

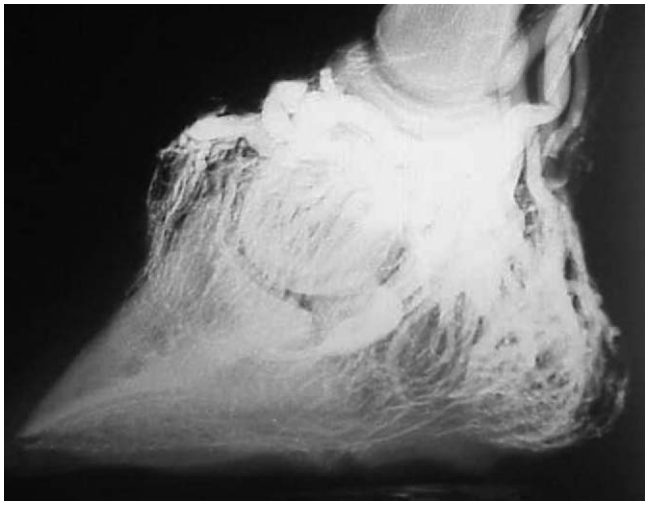


FIGURE 13-55 Same case as show in Figure 13-54 with sequestrate. The dorsal distal laminar vessel void suggested that a mural approach would be better than a solar approach.

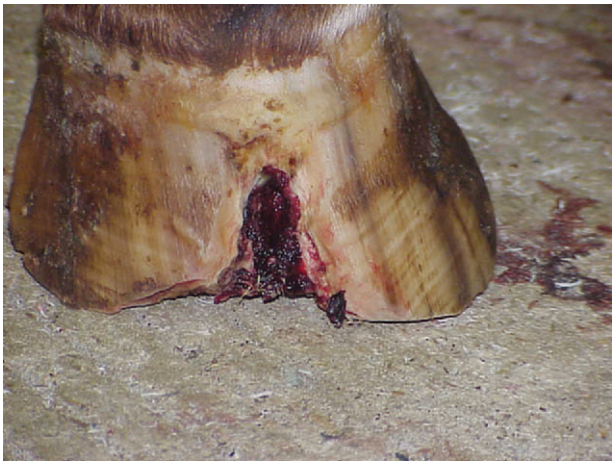


FIGURE 13-56 Approach to the dorsal sequestrate.

Restraint and Anesthesia

In the vast majority of cases, the surgery can be performed with the horse standing, using sedation and regional anesthesia, although lesions on the medial side of the foot can be difficult to access safely if extensive debridement is necessary. General anesthesia is preferred in foals. Detomidine, with or without butorphanol, provides good sedation for standing procedures. Although small lesions on one side of the foot could be debrided after desensitizing only the palmar digital nerve on the affected side, it is preferable to desensitize both medial and lateral sides at the level of the proximal sesamoid bones.

Surgical Approach

Some authors advocate approaching septic pedal osteitis lesions through the sole.[47] However, venography has been very helpful in demonstrating that, in many cases, a transmural approach is best (Figures 13-55 and 13-56). The horn at the wall–sole junction is removed with half-round nippers to the level of the dermal wall–sole junction. A hoof knife or a rotary burr is then used to debride the wall further proxi-

FIGURE 13-57 Preoperative Dremel (Robert Bosch Tool Corporation, Racine, Wis.) work. A rotary burr and hoof knife have been used to approach the affected soft tissue and bone. The tomcat catheter was placed to provide a visual aid during hoof wall removal. Note there is no tract visible at or above the hairline.

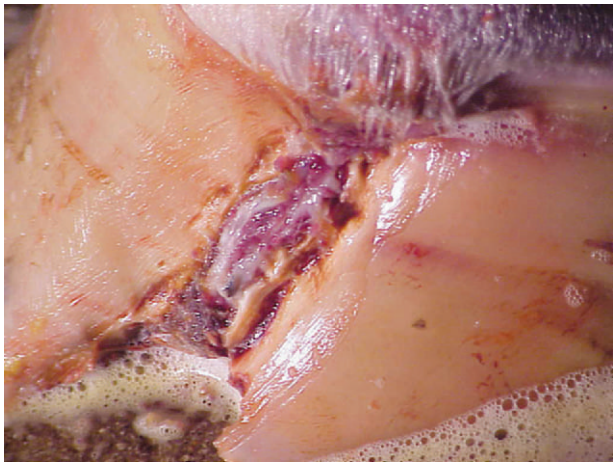


FIGURE 13-58 Preoperative tourniquet and scrubbing of the hoof.

mally to provide adequate exposure and removal of all diseased tissue (Figure 13-57).

It is common for the margin of P3 to be primarily affected, with the infection migrating proximally beneath one or more of the dermal lamellae. It can extend proximally all the way to the coronary band; in rare cases, it may even be necessary to remove a small area of coronary band to expose all of the damaged tissue.

During this initial approach, swelling of the affected dermal lamellae and dermal–epidermal detachment over the lesion site can be appreciated. A tourniquet (e.g., an Esmarch bandage or a length of bicycle inner tube) is applied around the fetlock at this time, and the hoof is scrubbed for surgery (Figure 13-58).

Surgical Debridement

If the lesion is detected and operated on early in the course of the disease, the infected tissue can be removed precisely with a scalpel (preferably, a No. 11 or No. 15 blade on a No. 3 handle) (Figure 13-59). The diseased corium peels easily away from P3.

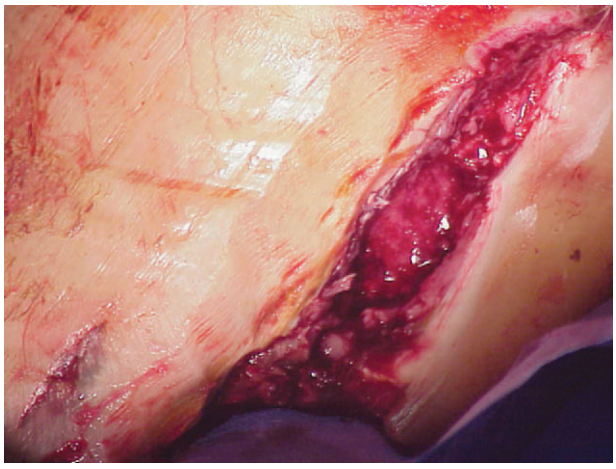


FIGURE 13-59 The dermal laminae have been incised along the edges of the wall and sharply dissected out. Note the purulence beneath the laminae.

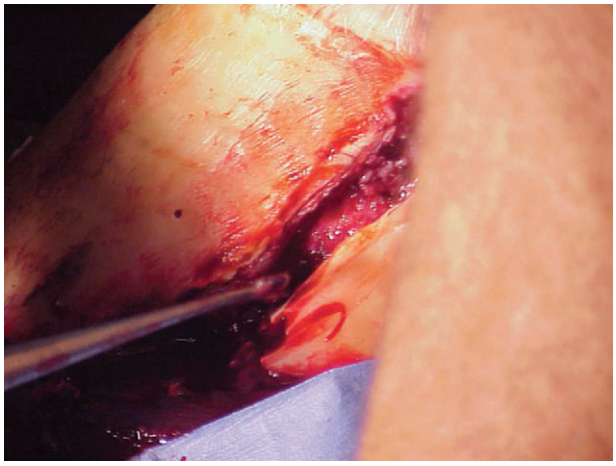


FIGURE 13-60 The process of curettage.

(A CO_2 surgical laser may prove to be superior to sharp dissection using a scalpel, although laser on bone can create its own subset of problems.)

Careful curettage is then performed to remove as much of the diseased bone as possible. Infected bone is soft (malacotic) and is easily removed (Figure 13-60). Sequestra at the margin of the bone can be removed with rongeurs and then curetted down to hard, healthy bone. (Healthy bone, corium, and epidermal lamellae are difficult to separate from one another, whereas infected and necrotic tissues are separated without much effort.) Samples of the diseased tissue should be submitted for aerobic and anaerobic bacterial culture and antibiotic sensitivity testing.

After satisfactory bone debridement, the soft tissue is debrided to create a tapered effect into the wound bed, and the wall is tapered into this. This minimizes a pinching effect of the wall on the soft tissue as a result of postoperative swelling (Figure 13-61).

Initial Wound Management

Once debridement is completed, amikacin (250 to 500 mg) is applied to some gauze sponges that have been soaked in

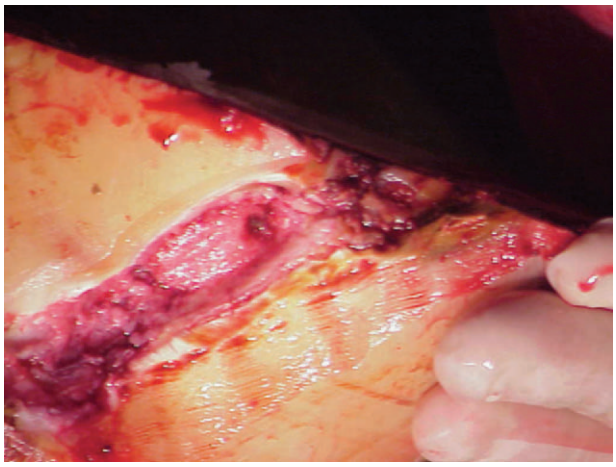


FIGURE 13-61 The curetted defect with beveled edges. Healthy bone is white, hard, and difficult to remove. The affected area is malacotic and, although grossly distinguishable, this small area would be difficult to recognize on radiographs.

dimethyl sulfoxide (DMSO) or directly onto the surgical site and the medicated sponges are packed into the defect. Additional DMSO-soaked sponges are placed over the defect so that sufficient pressure can be applied to limit hemorrhage once the tourniquet is removed. The gauze sponges are held in place with a few layers of self-adhesive bandage (e.g., Vetrap) and the tourniquet is then removed.

A well-padded bandage (e.g., thick cotton Combine dressing, secured with Vetrap and Elastikon) is applied to the digit, incorporating both the hoof and the pastern. It is often necessary to double-bandage the hoof to assist with hemostasis and absorb any initial hemorrhage. Once the horse is returned to its stall, it should be tied until the local anesthetic has diminished.

Medical Therapy

Systemic, broad-spectrum antibiotic therapy (e.g., penicillin plus gentamicin or amikacin) should begin immediately while awaiting results of culture and sensitivity tests. Phenylbutazone can be administered at a typical dose of 1.5 to 2 g IV q12h for the average 1000-pound horse. Tetanus prophylaxis also is important. Antibiotic therapy can be altered according to bacteriology results obtained from the surgical biopsy. Regional perfusion is an option, although in this author's experience it has not been satisfactory preoperatively and has not seemed necessary postoperatively. Regional perfusion can be performed at the time of surgery, after tourniquet placement, as this probably provides a high level of antibiotic at the surgical site.

Postoperative Care

Postoperative care consists of continued systemic antibiotic and antiinflammatory therapy and bandaging with topical antibiotic and antiinflammatory preparations. Systemic antibiotic therapy is continued for at least 10 days and typically for 3 to 4 weeks. Phenylbutazone is given for the first 3 days, 1.5 g b.i.d. for a 900- to 1200-pound horse; if needed, it can be continued at a lower dose for 7 to 10 days. If the infected tissue has been completely removed and there are no other problems, then the horse should be comfortable on the foot within 48 hours after surgery and should remain sound. The need for additional debridement usually becomes apparent within the first 7 days after surgery and is characterized by a return of or worsening of the lameness, despite medical therapy. Early intervention, thorough debridement, and use of the amikacin and DMSO-soaked packing has greatly reduced the need for additional debridement in the author's practice.

Initial Bandage Changes

The bandage is first removed 24 to 48 hours after surgery and replaced with the same type of dressing. Thereafter, the bandage is changed daily or every other day until granulation of the bone and soft tissue defect is complete. Changing the bandage this frequently assists with superficial debridement of the wound bed and encourages granulation of the wound. Granulation tissue grows in from the edges of the wound (i.e., from the adjacent dermis) and from the site of bone curettage itself. This pattern of healing is usually obvious by 5 to 7 days postoperatively.

Postoperatively, the surgical site usually is sensitive to touch until it has fully granulated. A stomping foot is not a healthy thing for the surgeon or the patient, especially in foundered horses or after extensive debridement in nonlaminitic cases. Regional anesthesia (e.g., unilateral abaxial sesamoidean block) can facilitate bandage changes; however, it often carries some risk, as these horses quickly come to hate needles during the course of treatment. Spraying 1 to 3 mL of mepivacaine on the wound bed works well if it is done 3 to 5 minutes before repacking the defect. Ethylene chloride sprays have proven unsatisfactory. This topical anesthetic allows for much easier wound packing and bandaging.

Uniform filling of the defect with granulation tissue is followed by keratinization of the outer layer of the granulation bed, which begins at the wound margins. Provided that keratinization is beginning by day 6 to 8 after surgery and the horse is walking comfortably, the bandage can be replaced with a hoof cast. Another option would be a shoe and flanged hospital plate. The properly applied cast has been shown to require less effort on the caretaker's part, provide less risk to the horse, and provide greater comfort and an astonishing healing environment for surgical and nonsurgical wounds of this nature.

Hoof Cast

At this stage of recovery, the cornifying wound bed should be covered with gauze sponges soaked in povidone-iodine solution and then a hoof cast should be applied that extends to the pastern. The iodine-soaked sponges are secured with a few layers of Vetrap, and a layer of Combine dressing is applied over this wrap. A 36- to 40-inch length of 14-inch wide Combiroll (Franklin-Williams Co, Lexington, Ky.) is cut in half lengthwise, wrapped uniformly around the hoof and pastern, and secured with Vetrap and Elastikon (Johnson and Johnson, Skillman, NJ). A final layer of cotton cast padding (Specialist Cast Padding; BSN Medical, Brierfield, England) is added to prevent the fiberglass casting tape from sticking to the bandage.

Three or four rolls of 4-inch fiberglass casting tape are used for the average-sized adult horse, with the cast ending just below the fetlock. A rubber pad or aluminum plate can be applied to the bottom of the cast to decrease wear. A few layers of Elastikon around the top of the cast and over the

fetlock prevent bedding from getting down into the cast.

Hoof Care. Before surgery, the hoof should be trimmed to remove any excess length of wall and, ideally, to create a 0-degree palmar/plantar angle and thus even weight bearing across the entire solar plane of P3. With lesions involving the caudal quarter of the wing of the coffin bone, it can be advantageous to provide a 15- to 20-degree palmar angle in the form of a stack of padding if there is substantial risk in shoe application, or in the form of an elevated heel shoe. An example would be fitting a keg shoe to the perimeter of the foot and then cutting the branch of the affected side on a line slightly behind Duckett's dot. Using another shoe or bar stock, the cut branch is connected to the opposite heel, creating a positive palmar angle during stance and preventing palmar rotation of the heel by having the fulcrum of the weld caudal to Duckett's dot. The sole and dead space should be filled with two-part dental impression material (Advanced Cushion Support; Nanric, Lawrenceburg, Ky.). The cast can be applied over this, or bandaging can continue on small lesions.

There are several orthotic formulations that can benefit the healing environment. In choosing an application, several points should be kept in mind: The horse should be treated without a block. The shoe should maintain the horse in the same or better degree of comfort as before application. Casting should improve any lameness even more. The veterinarian should be cognizant of the palmar/plantar angle of P3, where the injury or insult is, and the conformation of the horse. At this time there is no cookbook recipe for every combination of these ingredients, so it is important that the veterinarian be thoughtful and adaptable in the approach to the healing environment he or she provides.

Valuable lessens in what hurts can be learned by watching the submural motion that occurs with each phase of the stride and the horse's response. It is very challenging and important to provide internal stability without so much external pressure so as to create pressure ischemia. In this author's experience, casting has seemed far superior to any shoe alone.

Recovery is enhanced and complications in the contralateral limb are lessened by use of a properly applied heel wedge on the limb opposite the cast. Options include the Modified Ultimate wedge (Nanric, Lawrenceburg, Ky.) and a two-pad stack of Horse Trax gel pads (Impact Gel Co, Melrose, Wis.). The goal is to equalize limb length while a cast is on the affected limb, thereby allowing more uniform weight bearing on both feet.

Cast Removal. The cast is left on for 3 to 4 weeks, during which time the horse is confined to a stall. The cast and bandage material are then removed; the hoof wall around the perimeter of the defect is carefully trimmed with a sharp hoof knife to prevent pinching of the cornifying tissue, and a bandage is reapplied. The bandage is changed every 3 to 6 days until this level of protection is no longer necessary.

Alternatively, a second cast can be applied; however, in most cases recasting is not necessary. Typically, the defect is stable and almost completely keratinized by 4 weeks after surgery, and it takes only another 4 to 6 weeks to mature enough to be stable for athletic activity. If, however, the hoof wall resection was so extensive that it reached or extended into the coronary band, then it is best to allow the new horn growth to reach the level of the distal third of P3 before allowing turnout or returning the horse to training. The recovery time in these horses usually is 14 to 16 weeks.

SEPTIC PEDAL OSTEITIS IN LAMINITIC HORSES

Septic osteitis of the third phalanx can be one sequela to and a reason for advanced, complicated, uncompensated laminitis/founder cases. This complication has often been regarded as a dire circumstance and has led and can lead to the death or euthanasia of the horse. However, because of the myriad possible complications and interpretations of events occurring in the foundered foot, definitive diagnosis of septic pedal osteitis can be difficult. As a result, either the necessary surgery is not recommended or unnecessary surgery is undertaken in an exploratory fashion. Similar surgical debridement of septic pedal osteitis in laminitic horses may be indicated. Signs indicating surgical debridement may manifest as unrelenting pain and lowered response to therapeutic mechanical shoeing or deep digital tenotomy.

Radiographic Findings

In addition to the various radiographic abnormalities that can be found in the laminitic foot (see Chapter 18), septic pedal osteitis is accompanied by an increase of the dorsal H-L zone and of the H-L zone in the affected area (heel or quarter). This swelling progresses over weeks rather than days and may be observed medially, laterally, dorsally or palmar/plantarly, or any combination thereof. The finding of progressively increasing H-L zone is not exclusive to the laminitic foot affected by bone sepsis, but localized bone sepsis may be the reason the foot does not reach a compensated state in spite of appropriate medical and mechanical efforts, justifying further diagnostic evaluation.

Venographic Findings

Venography has been the most helpful modality in deciding whether to pursue a surgical course in cases of septic pedal osteitis in laminitic horses. Venographic findings are described in the earlier section on nonlaminitic patients. Areas of bone not encased by the normally dense vascular web should be resected when accompanied by purulence and progressive deterioration of the foot and patient.

It is not uncommon to see the apex of P3 beyond the vascular bed on the lateral to medial view of the venogram. This is an indication for deep flexor tenotomy. It is not an indication for coffin bone curettage unless there is accompanying purulent exudate at either the solar margin or the coronary band. In cases with no to minimal progressive swelling and no purulent exudates but imperfect venograms, bone debridement should probably not be pursued. These cases can be dealt with mechanically in most instances; however, vascularly compromised areas can become septic and may require debridement at some time. Broad-spectrum antibiotics can be beneficial in helping to avoid surgery. These cases invariably show radiographic evidence of bone loss while healing. Currently, these cases are left to resorb the bone on their own, but future pursuits may include surgery to this area to speed healing and improve return to function.

Surgical Approach and Postoperative Treatment

In this author's experience, debridement of septic pedal osteitis is most useful in the first 10 to 150 days of laminitis onset. It may not be recommended at this time to operate in

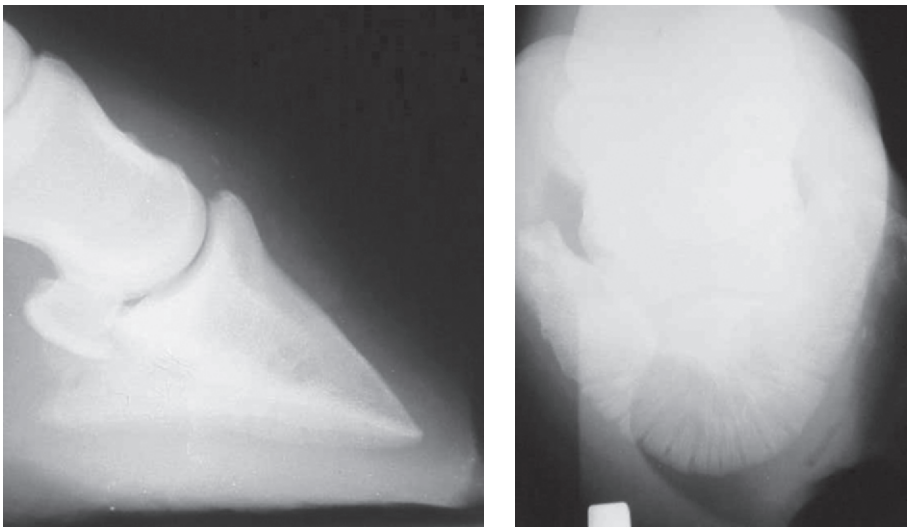


FIGURE 13-62 Radiographs of a mural keratoma. A very slight soft tissue change can be detected below the extensor process on the lateral-medial projection. Only a gas density off the toe can be seen on the solar margin view.

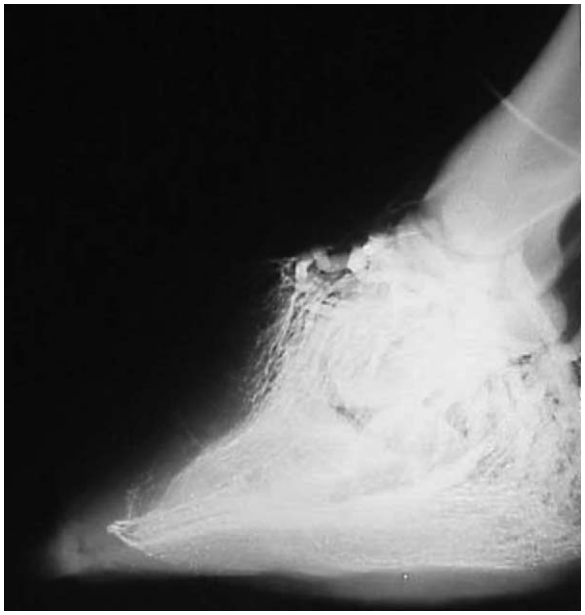


FIGURE 13-63 This lateral medial venogram projection shows (1) the coffin bone totally encased within the venous network; (2) disorganized but not particularly stretched vessels arising from the circumflex vein and from the terminal arch vessels; and (3) most interestingly, the vessels growing from the coronary plexus, rather than following the "waterfall" over the extensor process, going parallel to the horizontal and terminating into something that turned out to be an approximately 2.5 × 1.2-cm keratoma. This finding suggests that a keratoma does have a blood supply.

the very chronic (3- to 14-year-old) case with bone loss back to or approaching the foramina of the terminal arch. As with chronic septic osteitis of the nonlaminitic foot, but to an even greater degree, the remaining shelf of bone is inadequate to support the weight of the horse and is likely to fracture sagitally, leaving the second phalanx (P2) as the ground contact bone. The most extensive debridement performed in a horse whose foot then returned to a normal, sound, functional state involved the removal of about 50% of the circumference of the solar margin, about 7 to 10 mm deep of P3 on a hindfoot. Other cases have involved more extensive debridement, and the foot appeared to be doing well; however, in these cases the horse died or was euthanized because of other systemic complications of the disase process.

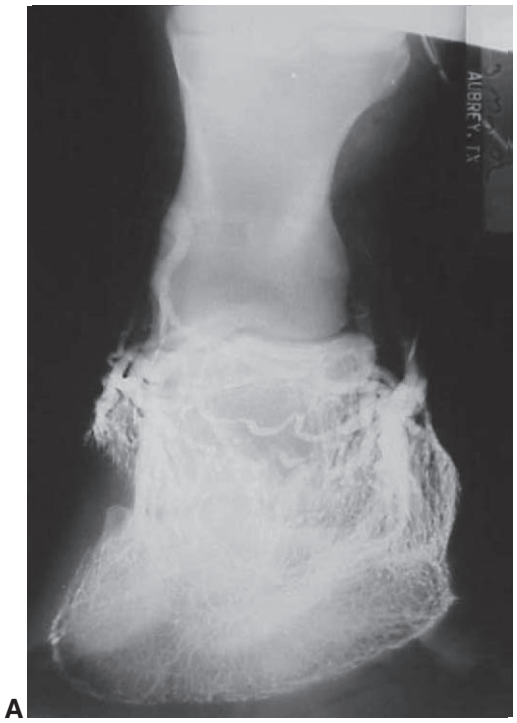
The surgical approach and postoperative care for septic pedal osteitis in laminitic cases are similar to that described earlier for nonlaminitic patients, but laminitic patients may require more intense care and longer healing time because of the abnormalities caused by the laminits.

Venography for Detection of Keratomas

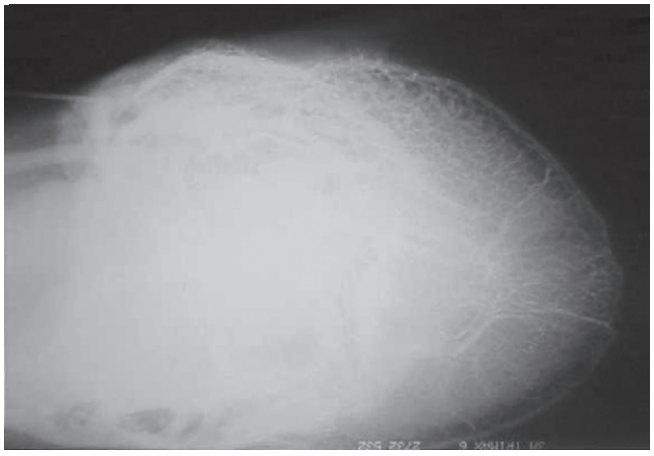
Venography can also be used to identify soft tissue masses within the hoof that have not had time or are not in a position to cause radiographic changes to P3. Keratomas are one such lesion. The horse with the keratoma illustrated in Figures 13-62 to 13-64 presented with exudation above the hair line, capsular deformation, severe lameness (grade 4 out of 5), and a history of "seedy toe." Plain radiographs showed no obvious bony abnormality, although some spotty opacities were seen near the H-L interface along the proximal surface of P3 on the lateromedial view (Figure 13-62).

Osteitis was suggested, so a venogram was obtained. The findings suggested the presence of a soft tissue mass beneath the hoof wall (Figures 13-63 and 13-64). The suspect area was exposed through the dorsal hoof wall, and the abnormal tissue was excised. Histopathologic examination confirmed it to be a keratoma. Recovery was uneventful and the horse was able to be returned to pasture 12 weeks after surgery.

This case further illustrates how helpful venography can be in the early identification of submural diseases, injuries, and other insults. It allows a greater degree of confidence in decision-making when considering or proceeding with invasive procedures involving the horse's foot. Better problem identification coupled with more expedient, less invasive, and more focused surgery provides a superior alternative to the horse, caretaker, and veterinarian dealing with these lesions before substantial bone compromise occurs.



A



B

FIGURE 13-64 **A,** This oblique view catches a void in dye slightly off center of the toe. Note that the laminar zone of the coffin bone has no deficit, and the visible bone shows no lysis or proliferation, which should be occurring in the time line of the process. This leads to high suspicion of a mass, scar, or foreign body. **B,** This view depicts a relatively normal circumflex system and terminal arch. With no defects seen in the distal one third to one half of the venogram, a proximal wall removal and approach was taken surgically, and the distal wall was able to be maintained intact. Without venography, the entire length of toe wall would have been removed in search of the mass.

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14
CAUDAL HOOF LAMENESS

TRACY A. TURNER

HISTORY

Any discussion of caudal hoof lameness must begin with a discussion of historical perspective. This condition was first described by Bridges in 1751; at that time he called it "coffin joint lameness."[1-4] This description referred to the entire lower joint of the leg, including surrounding structures, rather than to any specific part of the anatomy. The pathologic findings associated with the lameness were not described until 1819, when Moorcraft wrote the results of his study of the navicular bone.[4] A decade later, in the 1830s, because of the pathologic findings, the condition was renamed "navicular joint lameness" by Turner. Little changed in the basic knowledge concerning this disease for 100 years until 1929, when the first radiographic examination of the navicular bone was performed.[3,5] This development opened a whole new method to study this condition from an ante mortem standpoint. In 1935, Oxspring published the first description of the radiographic changes in the navicular bone associated with navicular disease.[3] Little new information developed over the next 40 years until the late 1970s, when renewed interest and new technology allowed clinicians to reassess their opinions regarding this lameness condition.

Today, navicular disease is known as the leading cause of chronic lameness in the horse.[6] However, the name has changed again. First, it became apparent that these cases were not all alike although they did have similar clinical signs, so the condition was called *navicular syndrome*.[7-9] However, it became apparent that not all these cases involved the navicular bone. The common clinical sign of these cases was that the lameness would be eliminated by a palmar digital nerve block; hence the lameness became known as *palmar foot pain syndrome* or *caudal hoof lameness*. For the purposes of this chapter, the following descriptions are used: *caudal hoof lameness* describes the lameness that is improved by palmar digital analgesia, *navicular pain* refers to pain originating from the navicular bone and or its adjacent ligaments, and *palmar foot pain* refers to all caudal hoof lameness that is not caused by navicular pain.

PATHOGENESIS

Lameness in the caudal region of the horse's foot accounts for more than one third of all cases of chronic lameness in the horse.[3,6,10,11] There are numerous causes of pain in the caudal aspect of the foot of the horse. These causes can be arbitrarily divided into conditions of the hoof wall and horn-producing tissues, conditions of the third phalanx (P3), and conditions of the podotrochlear region.[12] Hoof problems would include hoof wall defects, such as cracks or clefts that involve the sensitive tissue; any laminar tearing, separation, or inflammation; contusions of the hoof causing bruising or corn formation; abscess formation; and pododermatitis (thrush or canker). Third phalanx lameness blocked out by palmar digital anesthesia would include wing fractures, marginal fractures, solar fractures, or deep digital flexor insertional tenopathy. Conditions of the podotroclear region have been reported to include distal interphalangeal (DIP) synovitis, deep digital flexor tendinitis, desmitis of the impar (distal navicular ligament) or collateral sesamoidean ligaments, navicular osteitis or osteopathy, and vascular disease. The common denominator of all these conditions is that they are characterized by pain that can be localized to the caudal aspect of the hoof. Trauma or repeated trauma (wear and tear) is another factor common to each of these problems. The purpose of this section is to describe the leading theories of podotrochlear region pain.

Before a reasonable theory on the pathogenesis of any problem can be addressed, one must understand the pathology that is associated with the condition. Among the most commonly noted gross pathologic findings with navicular syndrome are thinning and erosions of the flexor fibrocartilage, but it must be mentioned that these are also seen in asymptomatic horses.[6,7,13-15] Synovial fossae are invaginations of synovial tissue into the navicular bone.[16,17] These invaginations provide direct communications between the medullary cavity of the navicular bone and the DIP joint. In cases of navicular syndrome, these fossae are commonly dilated; however, as with the thinning fibrocartilage, this change is also seen in nonlame horses. Enthesiophyte formation within the proximal navicular suspensory ligament (collateral sesamoidean ligament) and within the impar ligament is frequently seen pathologically.[7,15] These lesions probably represent tearing or damage along the insertion of these ligaments to the navicular bone. In long-standing cases, adhesions of the deep flexor tendon to the navicular bone are seen.[7,15,18]

Histologically, degeneration of the fibrocartilage covering the distal half of the flexor surface of the navicular bone is a common finding in older horses with and without signs of navicular syndrome.[3,13,15,19-21] Changes have been noted as early as 3 years of age in athletic horses and are found commonly in horses 8 years of age and older.[15,19] The primary change noted is fibrillation of the fibrocartilage, loss of staining for proteoglycan, death of superficial chondrocytes, and chondrone formation by adjacent viable chondrocytes.[3,13,15,19-21]

Recent microscopic studies comparing the synovial fossa of normal horses and horses affected by navicular disease could not elicit any substantial differences between the two groups.[16,17] This finding indicates that although enlarged synovial fossa has frequently been associated with navicular disease, the lesion is probably secondary and is not important in the causation of disease. In another study, the distal arterial blood supply to the navicular bone was partially occluded.[22] This occlusion caused arteriographic and histologic signs that resemble changes that occur in horses with navicular disease.

The changes were increased bone remodeling, shifting of the arterial pattern, formation of collateral vessels, and increased connective tissue in the synovial membrane and synovial fossa. However, they were unable to cause lameness or produce any radiographic changes within the navicular bone. This would suggest that vascular changes are important in the pathogenesis of navicular disease, but the ischemia is not of importance as a cause of the lameness. In other words, some other injury occurs that causes the lameness and then secondary changes occur within the vascular system.

In an unrelated study using transmission and grid electron microscopy, changes over the flexor fibrocartilage and deep flexor tendon were studied and comparisons were made between normal horses, horses with tenopathy, and horses with navicular syndrome.[13] This study showed that tendon fraying can occur in normal horses, but this fraying becomes more noticeable in horses with navicular disease. Also noted in this study was that the extents of flexor fibrocartilage wear correlated with the amount of tendon damage. This finding suggests that the flexor fibrocartilage wear may actually be caused by tendon damage and subsequent damage to the flexor surface.

Most theories can be grouped into one of three categories: vascular or ischemic theory, biomechanical stresses, or degenerative cartilage disease.[7,10,20,23,24] The first theory, ischemia, proposes that thrombosis of the distal navicular artery causes ischemia in adjacent bone. Ischemia causes pain that would lead to lameness. In response to the thrombosis, an increase in the number of arteriolar anastomoses and the number of arterioles leaving the distal nutrient foramina occur.[10,19,25] Increased vascularization around the foramina leads to an osteoporosis at the tip of the foramina, thus causing a characteristic shape change. Subsequent revascularization of the ischemic area would result in remission of the lameness. However, revascularization is slow and may not keep pace with the occlusive changes; this results in a slowly progressive lameness. If enough vascular thrombosis occurs at the same time, the ischemia progresses to a localized necrosis. Necrosis in the subchondral area of the flexor surface could break through the flexor fibrocartilage and lead to adhesions of the deep flexor tendon to the navicular bone. This theory more than any other explained the clinical nature of the lameness and also explained the radiographic changes that were associated with navicular disease. Unfortunately, further research has failed to corroborate this theory.[7,17,22] No one has been able to confirm the presence of thrombosis or infarcts in horses affected with navicular syndrome; changes in the blood flow to the bone have been recognized, but not occlusion. In one study, the navicular bone's distal nutrient arteries were occluded[22]; however, this did not cause lameness or any radiographic changes within the bone. Histologically, a change was noted in the vascular supply of the bone similar to that noted in navicular syndrome, but simple occlusion failed to produce pain. As a result, it is thought that vascular problems may play a role in the lameness but are not the primary cause.

The second, or biomechanical, category contains several theories that support biomechanical causes of navicular syndrome.[23] The concussion theory proposed by Adams hypothesized that repetitious pressure of the navicular bone against the deep digital flexor tendon caused navicular bursitis.[24] The bursitis is painful and the hyperemia caused by the bursitis causes decalcification of the navicular bone. Rooney proposed a similar theory.[23] In his theory, he speculates that vibrations between the deep flexor tendon and the navicular bone during locomotion cause damage to the flexor fibrocartilage and subsequent degeneration of the deep flexor tendon. Hoof concussion and poor hoof conformation, in particular broken-back hoof axis, are major predisposing factors to the vibrations.[23] In brief, this theory indicates that out-of-phase rotations of the fetlock and coffin joints cause an abnormal increase in the compressive force of the deep digital flexor tendon and navicular bone. The increased compression causes increased friction between the bone and tendon that result in fibrillation of the surfaces of both structures. The cause of the out-of-phase rotation is toe-first hoof impact with the ground. However, studies by other researchers indicate that broken-back hoof axis occurs only about 25% of the time.[26] In addition, there is little explanation as to what causes the toe-first landing. Correlations have been drawn between the occurrence of hoof imbalance and navicular lameness. This study indicates that the hoof imbalances cause abnormal biomechanical stresses on the feet that eventually lead to navicular disease.

Meier showed active bone remodeling and eventual sclerosis of the trabeculae of the navicular bone.[20] The sclerosis became worse with time and severity of lameness. It was the opinion of the researcher that the key to navicular disease's chronicity is this remodeling change and as the bone becomes sclerotic, the less likely it becomes that the horse will not be lame. However, the author admits to not knowing why. These theories taken as a whole may shed more light on the process; that is, obviously the navicular region is stressed in some manner, very likely due to a hoof imbalance or an improper loading of the limb for some reason. These abnormal forces over time stimulate remodeling of the navicular bone and eventual replacement of the normal trabeculae of the bone with a sclerotic, thickened, adynamic bone that cannot accept stress and becomes chronically painful.

A new area of interest in this respect has been with DIP joint pressures.[27] Hertsch has shown that horses with navicular disease typically have higher pressures in their DIP joint than do normal horses. The exact role this may play in navicular disease was unknown until recently, when Turner was able to show that the DIP joint and the navicular medullary cavity communicate (Turner, unpublished data, 1982-2005). As pressures increase within the joint, the pressures within the navicular bone increase. When pressures in the joint are returned to normal, pressures within the navicular bone were slow to decrease and remained elevated. It may be that chronically elevated pressures within the DIP joint cause increased intraosseous pressures within the navicular bone. Over time the increased intraosseous pressures could be expected to cause remodeling changes in the trabeculae of the navicular bone. Unfortunately, these theories fail to explain why one horse undergoing the same concussion does not develop the disease while another horse does.

The third theory involves navicular syndrome being a type of degenerative joint disease.[7] This theory holds that the fundamental lesion involved in navicular disease is a degenerative disorder. The degenerative changes involving the bone are initiated and promoted by excessive and sustained forces of compression exerted against the distal half of the navicular bone. Abnormal forces from faulty conformation

are considered the etiologic factor in this biomechanical disorder. It is thought that the conformational flaws result in biomechanical stresses on the navicular bone that cause remodeling changes. As long as the remodeling is slow, no problems occur. But when the biomechanical stresses exceed physiologic levels, pathologic processes begin. These processes lead to the development of degenerative disease in the fibrocartilage of the flexor surface. This leads to changes in the underlying subchondral bone and bone marrow. The changes in the bone lead to edema and subsequent venous hypertension and bone-related pain within the navicular bone. Researchers have indicated a close similarity between navicular syndrome and other degenerative conditions, specifically high ringbone and bone spavin. Although this theory does explain many of the histologic changes noted in horses with navicular syndrome, it does not explain the clinical features of the disease, with the exception of pain, nor does it explain how similar histologic changes can be present in nonlame horses.

Since none of these theories fully explain navicular disease, it would seem reasonable that the true pathogenesis is not known or that the pathogenesis may be a combination of these theories. A multiple pathogenesis theory seems most reasonable. In this theory, damage to the navicular bone, any of its supporting structures, or the deep digital flexor tendon can cause pain and lameness.[3] The anatomic position of the supporting ligaments makes them prone to injury by excessive biomechanical stress that could lead to tearing.[28] Similar biomechanical stresses could lead to tendon injury as well. In this case, excessive tension on the tendon with subsequent tearing of the tendon vasculature, local ischemia, and necrosis of the tendon would occur. The roughened tendon surface could then cause damage to the flexor fibrocartilage. The excessive stress could lead to damage, as hypothesized in either Rooney's theory or the degeneration theory. Biomechanical forces could cause strain within the DIP joint.[29,30] The joint responds by increasing the amount of joint fluid, which leads to increasing pressures within the joint. Excessive joint pressures then affect the interosseous pressure within the navicular bone, which causes pain and the subsequent changes within the medullary cavity that have been associated with navicular disease.

Many aspects of these theories are yet to be proven. Pathologic, clinical, and research findings indicate that many aspects of navicular disease cannot be easily explained. One major problem is that many investigators cannot agree on the nature of the disease. Once the nature of the disease is defined, then more specific research can be performed. This may be accomplished by simply defining navicular disease as lameness due to pain in the navicular bone. Some authors believe that by using differentiating analgesic blocks, pain in the navicular bone can be determined.[31]

DIAGNOSING CAUDAL HOOF LAMENESS

There are numerous causes of pain in the palmar aspect of the foot of the horse.[11,31] These causes can be divided arbitrarily into conditions of the hoof wall and horn-producing tissues, conditions of P3, and conditions of the podotrochlear region.[12] Hoof problems include hoof wall defects, such as cracks or clefts that involve the sensitive tissue; laminar tearing, separation, or inflammation; contusions of the hoof causing bruising or corn formation; abscess formation; and pododermatitis (thrush or canker). Third phalanx lamenesses blocked out by palmar digital anesthesia include wing fractures, marginal fractures, solar fractures, and deep digital flexor insertional tenopathy. Conditions of the podotrochlear region have been reported to include DIP synovitis, deep digital flexor tendinitis, desmitis of the impar (distal navicular ligament) or collateral sesamoidean ligaments, navicular osteitis or osteopathy, and vascular disease. The common denominator of all these conditions is that they are characterized by pain that can be localized to the caudal aspect of the hoof.

The first step in developing a logical approach to the treatment of caudal hoof lameness is an accurate assessment of the pain and careful evaluation of hoof structure that may predispose to or cause the pain.[11,32-34] The lameness must be characterized by viewing the horse walking, trotting, and trotting in a circle. Once the lameness is characterized, further diagnostics can be divided into (1) response to diagnostic manipulative tests, (2) response of the lameness to diagnostic analgesia, (3) imaging of the foot, and (4) evaluation of the hoof.[12,24]

Diagnostic Tests

Four diagnostic tests should be performed: hoof tester examination, distal limb flexion, hoof extension wedge test, and palmar hoof wedge test.[11,12] A positive response to any of these tests is important, but a negative response is equivocal and does not rule out any problem. The hoof should be examined with hoof testers.[12] Developing a routine for the examination is important. One method is to begin at the heel on the examiner's left side and work around the hoof in a clockwise fashion. Beginning with the bar, the examiner moves to the heel, to the quarter, and then to the toe, then back toward the heel on the examiner's right. The progress of the testers should be spaced at approximately 1-inch intervals, being sure to include each exit point of the shoeing nails. Next the testers are placed in each of the collateral sulci and across the hoof to the opposite hoof wall (a suggestion is to progressively move the hoof tester along the hoof wall caudal to cranial to check for alterations in the pain response); then the testers are placed in the central sulcus to the hoof wall at the toe and then across the heels. Finally, using the hammer, the examiner gently raps the structures on the bearing surface of the sole and frog. The examiner should keep in mind that hoof testers are essential but certainly not foolproof. The response the examiner gets on hoof testers is dependent on many factors, such as the hardness of the wall, depth of the hoof, thickness of the hoof, and stoicism of the horse.

Distal limb flexion test can exacerbate lameness if any of the three distal joints of the leg are affected by synovitis or osteoarthritis.[11,12,31,33] A positive response could also be expected by any condition that causes induration of the tissues of the foot. This has been shown to be positive in more than 95% of horses with navicular disease.

The hoof extension test is performed by elevating the toe with a block, holding the opposite limb, and trotting the horse away after 60 seconds (Figure 14-1).[11,12,31,33] The palmar hoof wedge test is performed by placing the block under the palmar two thirds of the frog and forcing the horse to stand

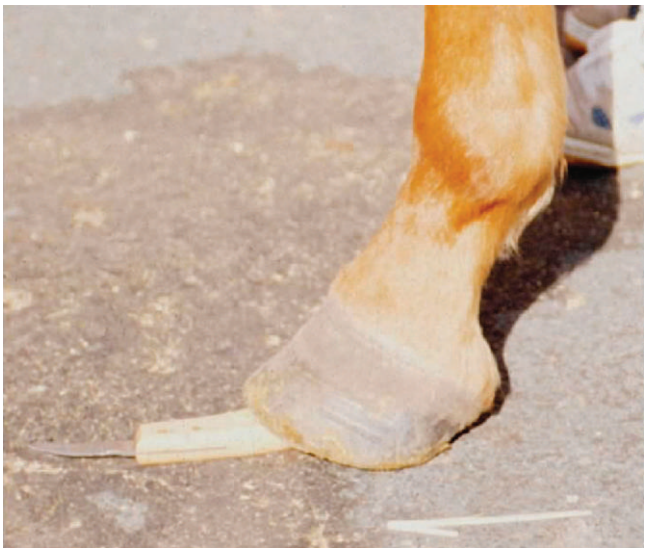

FIGURE 14-1 Hoof extension test is performed by positioning a block of wood under the toe, holding the opposite leg up for 1 minute, and then trotting the horse away.

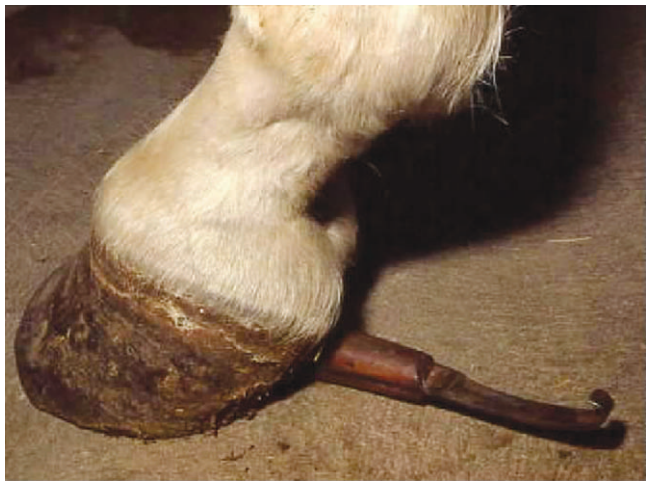

FIGURE 14-2 Frog wedge test is performed by positioning a block of wood under the frog, holding the opposite leg up for 1 minute, and then trotting the horse away.

on that foot (Figure 14-2). The test can be further modified so that the wedge can be placed under either heel to determine whether the pressure there causes exacerbation of the lameness.

Diagnostic Analgesia

By definition, caudal hoof lameness will be improved by at least 90% after perineural anesthesia of the palmar digital nerves, but palmar digital analgesia does not help differentiate these lamenesses. Anesthesia of the DIP joint or the podotrochlear bursa are additional procedures that provide information about palmar hoof pain. In a study reported by Dyson, in 95% of the horses examined using DIP and bursa anesthesia, significant new information about the pain the horse exhibited was realized.[35] The pain relief by anesthesia to any of these three regions has been shown to overlap. The DIP joint and podotrochlear bursa do not communicate, and yet the results of injecting anesthetic into these synovial cavities are similar.[36,37] Both cavities have in common the navicular bone, the impar ligament, and the collateral sesamoidean ligament (proximal suspensory ligament of the navicular bone). The neuroreceptors for the navicular bone are in those two ligaments and they can be anesthetized from either synovial cavity. Furthermore, Bowker has shown that the palmar digital nerve is in very close proximity to the medial and lateral limits of the bursa and the nerve may be anesthetized at this level whenever the bursa is injected.[28] Schumacher et al. showed that the interpretation of diagnostic analgesia into the DIP joint must be done carefully.[38] They performed an experiment in which they induced lameness by forcing setscrews into the sole of a horse's foot. The subsequent lameness was alleviated by DIP analgesia; this led the authors to conclude that local anesthetic into the DIP joint could alleviate pain in the sole. This information was contradictory to what this author (Turner) had observed. In a separate experiment, a Dremel tool (Robert Bosch Tool Corporation, Racine, Wis.) was used to expose sensitive sole on horses' feet.[27] The exposure was adequate so that "pin pricking" of the exposed area would cause pain. The DIP joint was then injected with anesthetic solution and the sole remained sensitive. In contrast, when the palmar digital nerve was anesthetized the sole was desensitized. One other observation was that the sole was not as sensitive as the author thought it would be. This observation led to the conclusion that the setscrew model creates lameness by causing the horse to shift its weight away from the pressure; that is, the horse abnormally transfers weight to different structures in the foot to avoid the pressure caused by the setscrews. In this case, the abnormal positioning causes stress in the caudal joint capsule of the DIP joint; therefore the pain the horse perceives as a result of the setscrews is via the DIP joint.

It has been shown by using the response of the horse to the three different analgesic injections that horses with caudal hoof lameness can be divided into five groups: (1) those horses with navicular region pain (desensitized by DIP analgesia and bursa analgesia, as well as palmar digital analgesia); (2) those with DIP pain (desensitized by DIP analgesia, as well as palmar digital analgesia but not bursa analgesia); (3) those that are not desensitized by DIP analgesia but are desensitized by bursa analgesia, as well as palmar digital analgesia; (4) those that are improved by either DIP or bursa analgesia but are not sound, but are sound after palmar digital analgesia; and (5) those that are not desensitized by either DIP or bursal analgesia but are desensitized by palmar digital analgesia (Figure 14-3).[31] It has also been noted recently that injection of the podotrochlear bursa can be very difficult and that it is quite easy to inject the DIP joint instead.[18] Not only is radiographic control necessary to successfully perform this block but adding contrast media to the anesthetic to prove the limits of the block is also necessary. This necessity has led to a new method of assessing navicular pathology, by evaluating the cartilage of the flexor surface of the navicular bone by contrast arthrography. This will be discussed in the following section.

Imaging

To get a good radiographic overview of the navicular bone and caudal hoof, a minimum of five radiographic projections

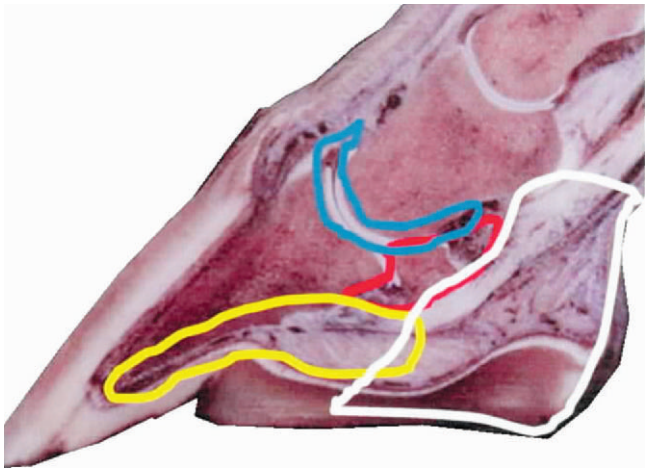

FIGURE 14-3 Areas blocked by combination procedures: (1) navicular pain blocked by both distal interphalangeal (DIP) joint analgesia and bursa analgesia (*red*); (2) DIP pain blocked by DIP analgesia and no effect with bursa analgesia (*blue*); (3) solar third phalanx insertional deep digital flexor tendon blocked by bursa analgesia but no effect with DIP analgesia (*yellow*); (4) multiple areas of pain improved by DIP analgesia but not sound and improved by bursal analgesia but not sound; (5) heel/quarter pain showing no improvement with either DIP analgesia or bursa analgesia (*white*).

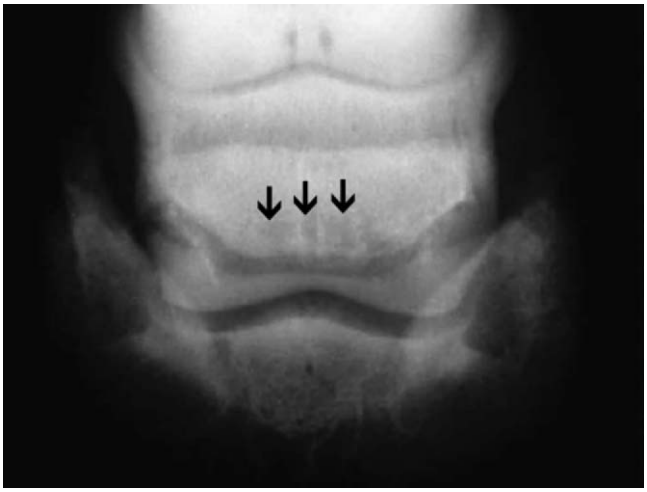

FIGURE 14-4 Enlarged synovial fossa (*arrows*) seen along the distal border of the navicular bone.

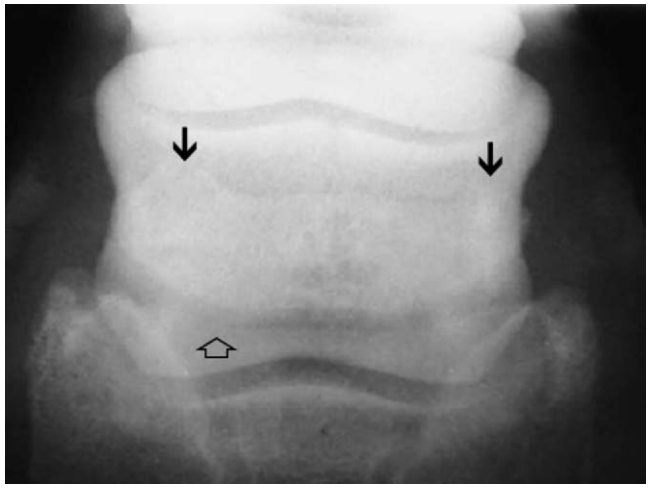

FIGURE 14-5 Enthesophyte formation on the proximal border (*solid arrows*) and enthesophyte formation in the impar ligament (*open arrow*).

of each foot should be made. These include the dorsal 65-degree proximal to palmarodistal oblique projection (D65Pr-PaDiO), the dorsal 45-degree proximal to palmarodistal oblique projection (D45Pr-PaDiO), the lateral to medial (LM) projection, the palmaroproximal to palmarodistal oblique projection (PaPr-PaDiO), and a horizontal dorsopalmar (DP) projection.[3,8,12,24,39-41] These projections are needed to evaluate the borders and architecture of the distal and second phalanges, the navicular bone, and the DIP and proximal interphalangeal joints. Specific changes to evaluate within the navicular bone include enlarged synovial fossa, enthesiopathy, cystlike formations, and changes of the flexor cortical region. Radiographic examination is the imaging method most often used to assess osseous changes in the distal sesamoid bone and P3. These changes, with the exception of fractures, are usually not pathognomonic but do provide insight into damage that has occurred to the foot.

Enlarged synovial fossa is a common finding. Reports vary but most accept that around 85% of horses presented for caudal hoof lameness show this change within the navicular bone.[8] These changes are best seen on the dorsal 65-degree proximal to palmarodistal oblique projection. Synovial fossa are typically observed along the distal border of the navicular bone (Figure 14-4). The appearance can vary from small inverted cones to areas that appear like lollipops and all stages in between.* The inverted cones are considered normal and the lollipops are considered evidence of navicular remodeling. The problem is that further scrutiny has shown that this change also occurs under normal circumstances; in fact, Quarterhorses and Thoroughbreds show the change normally starting around the age of 6 years old, and warmblood breeds may show it as early as 3 years of age.[8] The occurrence of these changes happens in nonlame horses as frequently as in lame horses. Furthermore, recent research has shown there is no predictability between the presence of these findings and the eventual development of caudal hoof lameness (Turner, unpublished data, 1982-2005). Because of this discrepancy, it has been this author's opinion to consider what this change indicates rather than whether this change confirms a disease.[11] In other words, the implications of this finding should be considered relative to all the clinical findings instead of using the change or lack thereof to confirm or rule out navicular problems. It has been shown that this change is due to bone resorption in naturally occurring depressions in the distal aspect of the navicular bone to accommodate more DIP synovial tissue.

Enthesiophyte formation associated with navicular problems involves calcification of either the collateral sesamoidean ligament (proximal suspensory ligament) or the impar ligament (Figure 14-5).[3,8,11,24,41] These changes are best evaluated on the D65Pr-PaDiO projection and can be confirmed on the lateral to medial projection. These changes occur in about 20% to 25% of horses with caudal hoof lameness and with the

*References 3, 8, 11, 17, 24, 41.

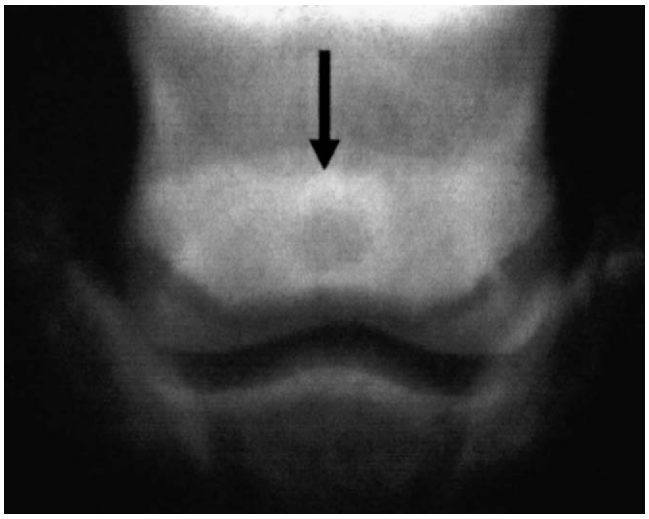

FIGURE 14-6 Cystlike formation in the navicular bone (*arrow*).

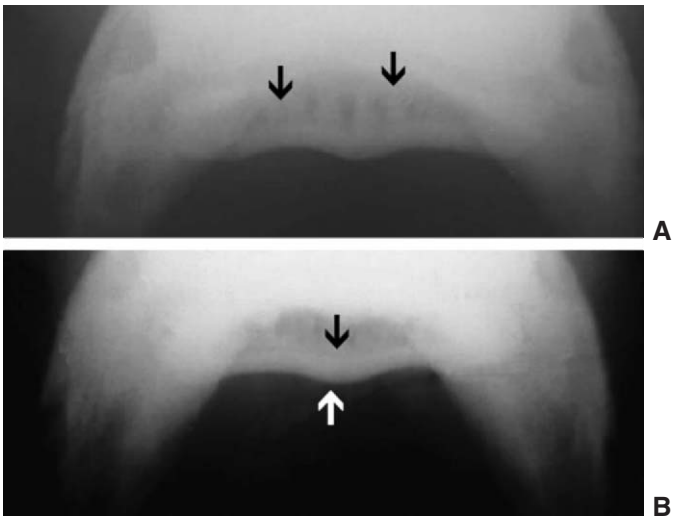

FIGURE 14-7 Medullary sclerosis identified on the flexor view. **A**, Sclerosis is seen when the medullary cavity (*arrows*) is approaching the same density as the flexor cortex. **B**, Comparison view showing clear demarcation on both sides of the flexor cortex (*arrows*).

same frequency in nonlame horses.[8] The change most likely is due to stress on or injury to the ligaments and subsequent calcification of the ligament.

Cystlike formations within the navicular bone are caused by focal decalcification of the navicular bone; usually this occurs within the flexor cortical bone (Figure 14-6).[3,8,11,24,41] These changes should be evaluated by using two projections, the D65Pr-PaDiO and the PaPr-PaDiO. There are many causes of this finding, including cystic invaginations of the flexor surface, focal areas of decalcification, and adhesions between the navicular bone and deep flexor tendon. Again, these changes occur in lame horses with the same frequency as in nonlame horses.[8]

A navicular bone change that is considered more pathognomonic for chronic remodeling change on the navicular bone is sclerosis of the medullary cavity (Figure 14-7).[3,8,11,24,41] This change can only be assessed using the PaPr-PaDiO projection. Positioning is critical; normally there should be a well defined demarcation between the flexor cortical bone and the medullary cavity. Sclerosis is identified when all or part of the medullary cavity achieves the same density as the flexor cortex.

A study of 1917 German auction horses between the ages of 3 and 8 years indicated that 97% of the horses had one or more radiographic signs of navicular pathology.[40] In another study of 220 5- to 9-month-old foals, 25% had radiographic changes of the navicular bone.[42] This indicates that the navicular bone is very actively remodeling as stresses such as growth are applied to it. This was confirmed by a study of scintigraphic and radiographic findings in horses with navicular disease (Turner, unpublished data, 1982-2005).[43] The study showed that scintigraphy showed activity prior to radiographic changes and that scintigraphic changes were often noted early in the course of the disease before radiographic changes. Because scintigraphy is an indicator of remodeling, this shows that the bone is responding to stresses.

Contrast navicular bursography is a new radiographic technique that indicates pathologic lesions in the flexor cortex region 60% more often than plain-film radiography (Figure 14-8).[18] Injection into the bursa is made from the palmar surface with the limb flexed at the carpus. Aseptic injection

FIGURE 14-8 Normal navicular busogram. Note the even black space between the flexor cortex and white line (contrast material). The black space is the flexor fibrocartilage.

techniques are used to inject a 3-mL mixture of 1:1 contrast material and local anesthetic solution. The landmarks for needle insertion are just proximal to the central sulcus of the frog with the needle directed in line with the apex of the frog and in a direction parallel to the ground surface of the hoof (Figure 14-9). A 20-gauge, 3.5-inch needle is used. The needle is inserted until resistance is encountered; this is usually at two thirds the length of the needle. If the needle is inserted further, it usually indicates incorrect placement. A lateral radiograph of the hoof is taken to confirm the position of the needle prior to injection. Ideally the needle should be midway between the proximal and distal borders. Once needle position is confirmed, the bursa is injected with the contrast mixture and a second lateral hoof radiograph is taken to confirm filling of the bursa. If the bursa has been injected, then a palmaroproximal-palmarodistal oblique projection of the navicular bone is obtained.

A distinct line of contrast material juxtaposed to the deep digital flexor tendon is normally separated from the navicular cortical bone by a layer of radiolucent fibrocartilage.[18] Five basic findings are noted with contrast navicular bursography: (1) normal flexor fibrocartilage; (2) thinning or erosions of the flexor fibrocartilage (confirmed by postmortem examination in three cases) (Figure 14-10); (3) complete focal loss of the dye column thought to be due to flexor tendon adhesions to the bone (Figure 14-11); (4) presence of flexor subchondral bone cystic defects, which is noted as focal

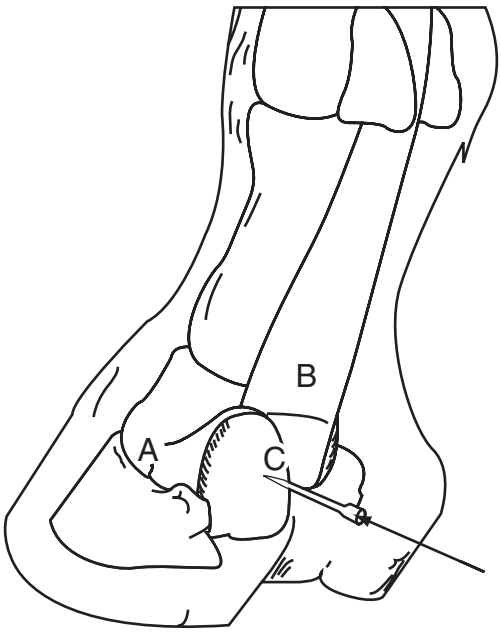

FIGURE 14-9 Outline for injection into the bursa. *A,* Collateral cartilage; *B,* deep digital flexor tendon; *C,* navicular bursa. *Arrow* indicates the placement of the needle.

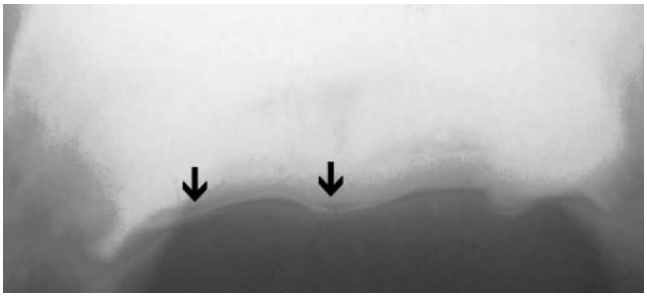

FIGURE 14-10 Erosions and thinning of cartilage. The contrast material is in contact with the flexor surface along most of its border. The *arrows* indicate areas of flexor cartilage.

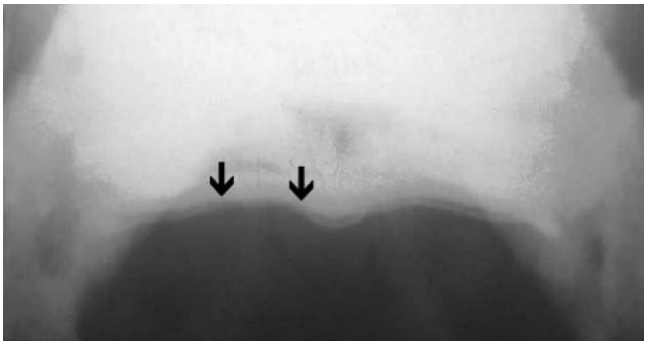

FIGURE 14-11 Bursogram showing a partial loss of the dye column (between *arrows*). This indicates an adhesion of the deep flexor tendon to the bone in this spot.

filling of the flexor cortical area with contrast (Figure 14-12); and (5) fibrillation of the deep flexor tendon, which is noted as filling defects along the bursal surface of the deep flexor tendon (Figure 14-13). Normal fibrocartilage was seen in 13% of the examinations. Thinning or erosions of the flexor

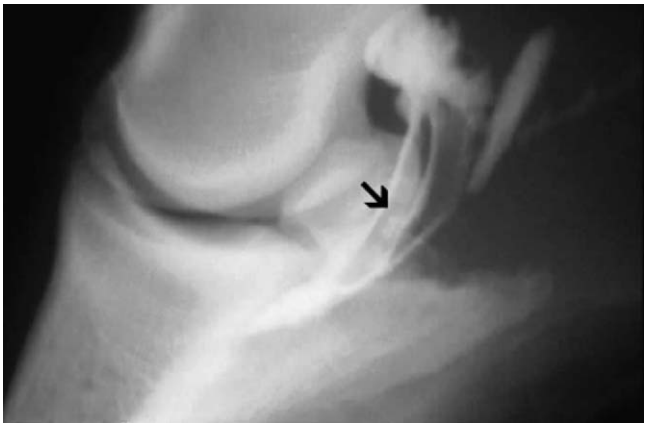

FIGURE 14-12 Lateral bursogram showing a filling defect (*arrow*) into the flexor cortex.

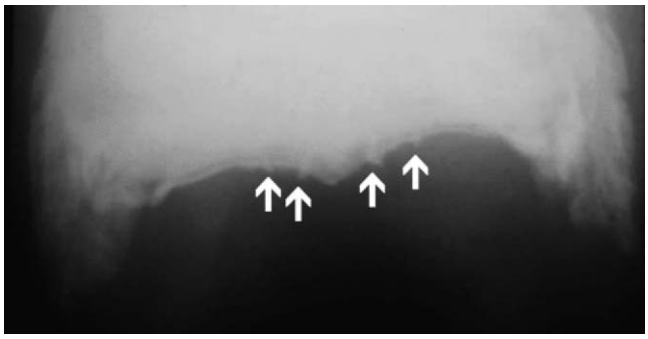

FIGURE 14-13 Bursogram showing fibrillation of the tendon (*arrows*).

fibrocartilage is seen in 69% of the bursograms. Adhesions (loss of the dye column) are noted in 8% of the cases. Filling defects of the navicular flexor surface are noted in only 2% of the horses. Fibrillation of the deep flexor tendon is recognized in 21% of the horses. Horses with normal flexor cartilage are more likely to have navicular pain (8 of 97) rather than palmar foot pain (5 of 97). This is in contrast to horses with cartilage thinning or erosions in which horses with palmar foot pain were more likely to exhibit this change (38/97 for palmar foot pain versus 29/97 for navicular pain). Horses thought to have adhesions all were in the navicular pain group, and there was no difference in horses with flexor filling defects (1/97 with palmar foot pain and 1/97 with navicular pain). Horses showing tendon fibrillation were also more likely to show palmar foot pain (14/97) rather than navicular pain (6/97).

Navicular bursography was developed out of the need to confirm injection of local anesthetic into the bursa.[18,31,33] The bursa is not only a small space but is also in close proximity to other synovial structures, such as the distal interphalangeal joint or distal tendon sheath. Because of this, the bursa can be difficult to inject. Use of this technique allows the practitioner to know bursa injection has occurred.

Interestingly, normal flexor fibrocartilage is noted more frequently in horses thought to have navicular pain.[18] This finding tends to refute the long believed premise that navicular disease begins as damage to the flexor fibrocartilage or at the very least suggests that there may be more than one pathogenesis of navicular pain. Following these cases with sub-

sequent bursography would be a method to follow the pathogenesis of these cases.

The most common change noted on the bursogram is that of flexor cartilage thinning.[18] However, when horses that were lame due to navicular pain were compared with horses that were lame due to other pain in the palmar foot, there was no statistical difference between the two groups, although the change occurs more frequently in the group with palmar foot pain. It could be that this change is a normal wearing process or it may be either a primary or secondary response.

Adhesion formation is noted only in horses with navicular pain, whereas flexor filling defects occurred equally in horses with navicular as well as other causes of palmar foot pain.[18] This indicates that the adhesions are associated with navicular pain but that defects on the flexor surface may be developmental and have no effect on pain. Lesions of the flexor surface of the deep flexor tendon have been noted and some investigators have suggested that this may be an early navicular pathologic lesion.[13] However, in one study fraying of the tendon was seen more than two times more often in the palmar foot pain group.[18] In most cases, tendon fraying was associated with flexor fibrocartilage thinning and erosions (12 of 20 cases). At this time, no speculation can be made about which came first.

Overall, navicular bursography is a simple technique that can be used to confirm injection into the navicular bursa and can also give valuable new information regarding pathologic lesions in the region of the navicular bone.[18] Changes seen via contrast navicular bursography represent stages of pathologic damage and allow more timely therapeutic intervention and more accurate prognostication. The author's approach to these types of cases is to develop a treatment strategy based on the individual case needs rather than a treatment formula. Bursography has improved the ability to identify pathologic lesions such as flexor cartilage erosions and to utilize therapy such as chondroprotective agents. Identification of tendon injuries causes concern for tendinitis, and strict rest to allow healing of the tendon can be instituted. The identification of adhesions has been a grave prognostic indicator for conservative management. This technique also provides a means to study the pathogenesis of navicular disease or navicular pathology because it provides important information about anatomical structures that earlier could only be evaluated post mortem. By utilizing this technique, understanding of navicular problems can be advanced.

Recently it has become possible to examine the podotrochlear region (deep flexor tendon, navicular bursa, and impar ligament) sonographically (Figure 14-14).[44,45] To examine the podotrochlea, the superficial horn must be pared from the frog to expose soft, spongy frog tissue. Next, sonographic gel is liberally applied to the frog. The ultrasound transducer is then applied to the frog. Images of the podotrochlea are apparent from the center of the frog to the apex. A 7.5-MHz probe provides the best image.

Generally, at the center third of the frog, the flexor surface of the navicular bone is readily noticeable as a hyperechoic line (Figure 14-15).[44] The bursa is seen as a hypoechoic (fluid-filled) region juxtaposed to the navicular bone. The deep flexor fibers can be seen curving around the bone. As the probe is moved toward the apex of the frog, the distal aspect of the navicular bone can be identified, as can the intersection between the deep flexor tendon and the impar ligament. As the probe reaches the apex of the frog, the deep flexor tendon's insertion on P3 becomes apparent.

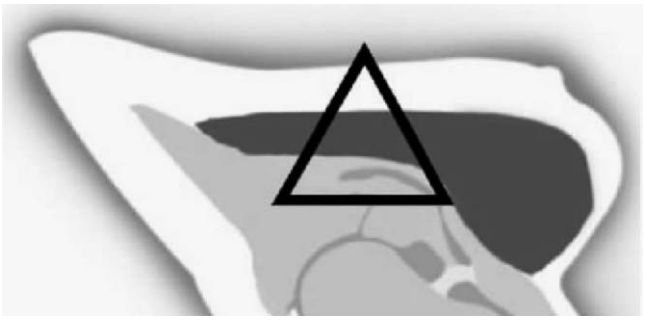

FIGURE 14-14 Diagram of the positioning for songraphy through the frog.

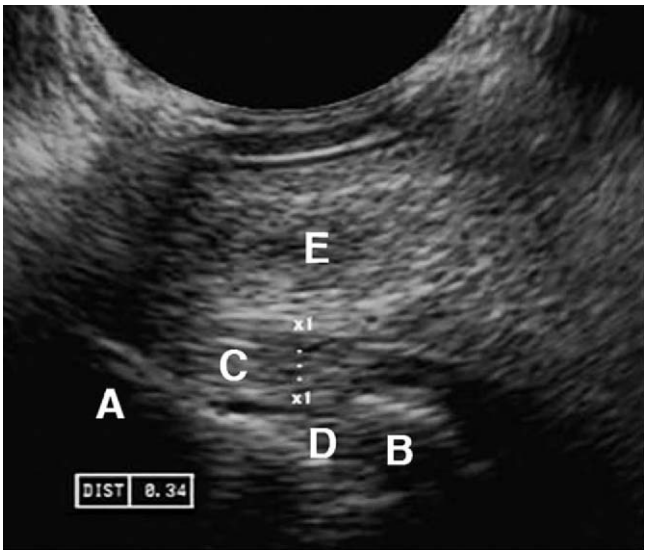

FIGURE 14-15 Sonogram through the horse's frog. *A,* Coffin bone; *B,* navicular bone; *C,* deep flexor tendon; *D,* impar ligament; *E,* digital cushion.

Ultrasonography is an excellent means to visualize soft tissue structures. However, examination of the foot has been limited to the pastern because the hoof capsule serves as a barrier to examination of the hoof. The proximal regions of the navicular bone could be examined if one had a special probe that would fit between the bulbs of the horse's heels.[45] This gives no information, however, as to what may be occurring further distally. The frog, because of its high water content, can serve as the hoof's "stand off." Removing the hard, outer layers of the frog's epidermis exposes tissue that can transmit sound waves, allowing the examiner to see these distal tissues.

Ultrasonographic examination of the DIP collateral ligaments and joint capsule can be performed utilizing either a 7.5-MHz convex linear or straight linear probe.[45] The area of the coronary band was clipped and the ligaments were located at the 10 o'clock and 2 o'clock positions along the hair hoof interface (Figure 14-16). Ultrasonography is the method of choice to diagnose DIP collateral desmitis.[46] All cases need to be evaluated for cross-sectional area, fiber alignment, and echogenicity. Routine examination at 30-day intervals can be used to regulate the recovery process. Using this approach,

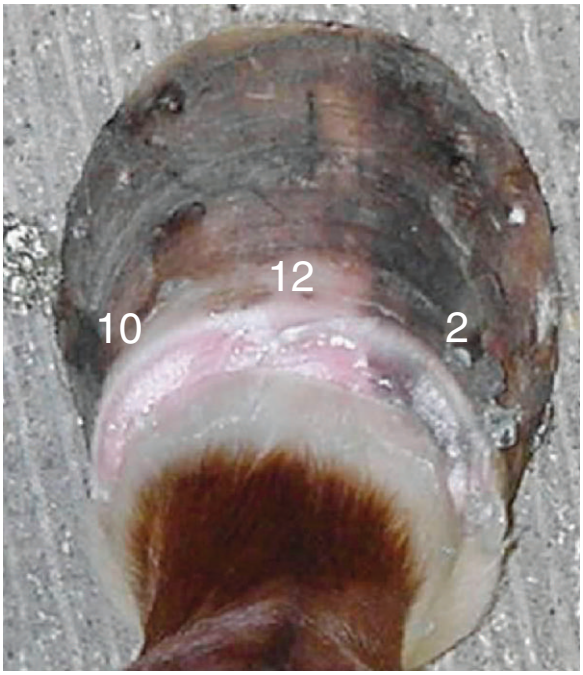

FIGURE 14-16 View of the hoof showing the locations of the collateral ligaments at the 10 o'clock and 2 o'clock positions.

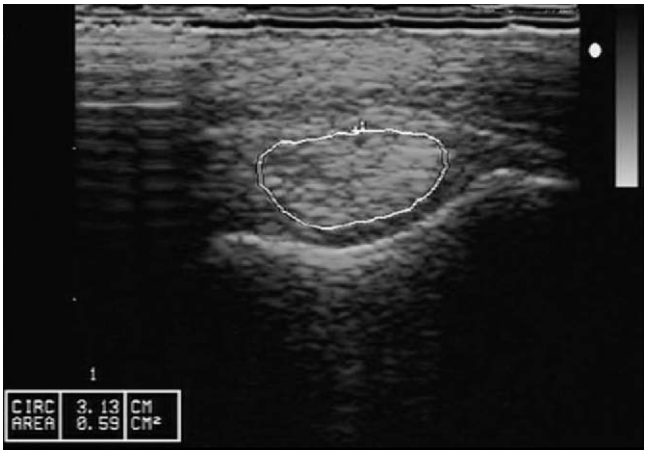

FIGURE 14-17 Sonogram of the distal interphalangeal collateral ligament. It is most easily located by finding the crescent shape along the short pastern, then locating the ligament in the depression (*white outline*).

the ligament can be followed for decreasing cross-sectional area, improvement in fiber alignment, and echogenicity.

Cross-sectional areas of diseased collateral ligaments of the DIP joint vary widely ranging from 0.75 cm^2 to 1.04 cm^2 (normal is 0.62 cm^2) (Figure 14-17).[45,46] Diseased ligaments show various amounts of hypoechogenicity and parallel fiber malalignment. A few horses exhibit a specific anechoic core lesion, and on a rare occasion a horse will nearly completely rupture the collateral ligament. There is no difference in the occurrence of injury to the medial or lateral collateral ligament; about 25% of the time both ligaments are affected.

Scintigraphy is a technique that measures gamma ray emission from a radioactive nuclide agent injected into the animal.[43,47] The technique provides information on relative vascularity and rate of tissue metabolism. This is particularly useful in studying bone pathologic lesions and can help differentiate sites of injury in the foot.

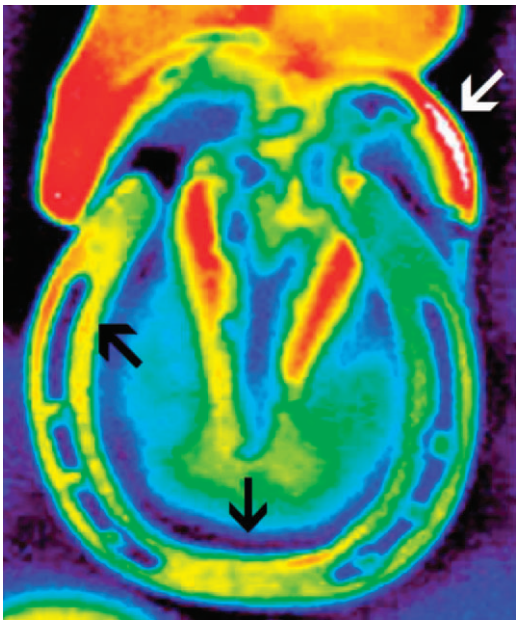

FIGURE 14-18 The medial side of the shoe has more heat, indicating more friction on that part of the shoe (*black arrows*). This may be caused by inflammation in the lateral heel (*white arrow*).

Thermography provides information regarding skin temperature.[48] Navicular pain does not have a specific thermal pattern, but thermography can identify several aspects of the syndrome that are helpful in differentiating various conditions making up the syndrome. Thermography can help determine whether poor hoof blood flow is associated with the lameness, it can help assess hoof balance, and it can determine whether there is evidence of inflammation in the hoof capsule or sole or whether there is inflammation in other regions of the pastern that may account for the lameness.[48,49]

Thermography can qualitatively test blood flow to the foot and determine whether a decrease in blood flow is associated with the lameness.[49] For blood flow assessment, the foot is thermographically evaluated before and after exercise. The normal horse will sustain a 0.5° C increase in temperature of the foot after exercise, but the horse with low or decreased blood flow does not sustain this increase in the foot. This observation is helpful therapeutically by more specifically identifying horses that need rheologic drug therapy. In contrast, focal inflammatory conditions of the hoof such as abscesses, bruises, or fractures are characterized by focal areas of increased temperature.[48] Exercise in these cases only intensifies the "hot spot."

In addition, thermography is useful in assessing hoof balance.[48] In this manner, the heat pattern corresponds to the stress patterns placed on the horse's foot. By examining the heat patterns of the shoe, the examiner can determine where the horse is landing and can also use this information to help differentiate inflammation from stress. When heat in the sole or hoof wall corresponds to the hot area of the shoe, this is most likely stressed induced. However, if a "hot spot" occurs opposite the hot area of the shoe, this particular hot spot is inflammatory (Figure 14-18).

Inflammatory conditions of the hoof wall and sole are seen as focal hot spots.[48] Cracks may or may not show heat, depending on whether they are a problem. Hoof cracks that cause pain invariably invade the sensitive tissues and will be associated with increased heat. Cracks that are not clinical are simply cold, signifying separation between that hoof wall and the deep sensitive tissues. The sole thermographically is usually relatively cool; the first sign of inflammation in P3 is the finding of a crescent-shaped area of increased heat anterior to the apex of the frog. As inflammation progresses, the size and intensity of the heat pattern increases.

Finally, thermography can be used to determine whether other painful conditions of the pastern may be causing the lameness rather than the hoof. In these cases, the pastern region shows increased heat, which gives the examiner evidence that the inflammation is there rather than in the hoof.[48]

Hoof Assessment

A subjective and objective assessment of the hoof should be made. As with any physical examination, this is not simply measuring a few parameters and determining where on the scale of normality they fall, but rather it is a systematic evaluation of the hoof capsule and the structures within.[12] The examination tells the examiner about the general health of the hoof, the stresses that have been placed on it, and how the hoof has responded to these stresses. The hoof is a dynamic structure that grows continuously and therefore has the ability to deform continuously to stresses that are applied to it. The physical examination of the hoof determines what the hoof has to tell the examiner.

The examination begins simply by the examiner's looking at the hoof, preferably from sufficient distance to compare all four feet at once.[12] The size, shape, toe length, heel length, hoof pastern axis, and position of each foot relative to the each limb and to each other are assessed. This is the best time to evaluate the horse's balance, that is, the differences in each of the horse's legs and how the horse stands on the hoof. One could consider this a conformational analysis, but in fact the examiner is simply evaluating the position of the hoof on the end of the limb. This feature must be evaluated from three directions, the front (dorsal), the side (lateral), and the back (palmar/plantar). From the front, the hoof needs to be assessed for symmetry and alignment. Is the hoof centered under the cannon bone or is it offset? If the hoof is offset, then the stresses on the hoof will change. Does the hoof rotate on the leg (toe-in or toe-out)? If it does rotate, where does it rotate—from, knee, fetlock, pastern, or hoof? This will determine where the torque is occurring on the hoof. Does the ground surface of the hoof appear symmetrical? If not, this indicates stresses on the hoof. Most commonly, one sees the medial wall more upright. Is the coronary band straight and parallel to the ground surface? If not, this indicates a stress on the wall below the coronet (Figure 14-19).

The next factor to observe is the hoof alignment,[50] which is viewed from the dorsal and lateral aspect. The average horse (60%) will have a hoof angle between 50 and 55 degrees. The hoof axis is how the cannon bone, pastern, and hoof line up. Ideally, when the horse is standing square, the cannon bone, pastern, and hoof line up straight as seen from the front. From the side, the pastern and hoof should be straight with the angle created by the dorsal hoof wall, the same angle as

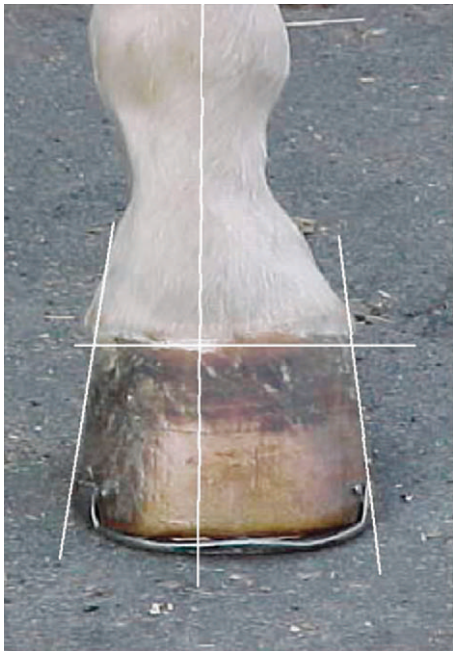

FIGURE 14-19 Well-aligned left foot from the frontal plane. The foot is slightly offset laterally.

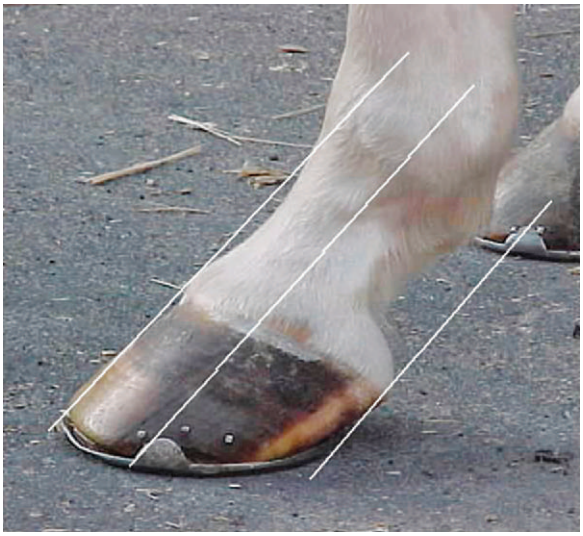

FIGURE 14-20 Relatively well-aligned hoof from the lateral aspect.

the pastern and the angle of the heels within 5 degrees of the angle of the toe (Figure 14-20). Horses that have a low hoof angle, compared to the pastern, have a broken-back hoof axis and fall into a group of horses called *long toe/low heel.* On the other hand, horses with a steep hoof and sloping pastern have a broken-forward axis and are called *clubby.* Unfortunately, horses do not normally stand with their cannon bones perpendicular, so evaluation of hoof alignment must be done with the horse standing comfortably. The purpose is not to determine right and wrong but to determine what is comfortable for the horse.

The next area to evaluate is the shape and levelness of the hoof.[9,51] Generally the front hoof should be round or circular in shape, whereas the rear hoof is more triangular or pear-

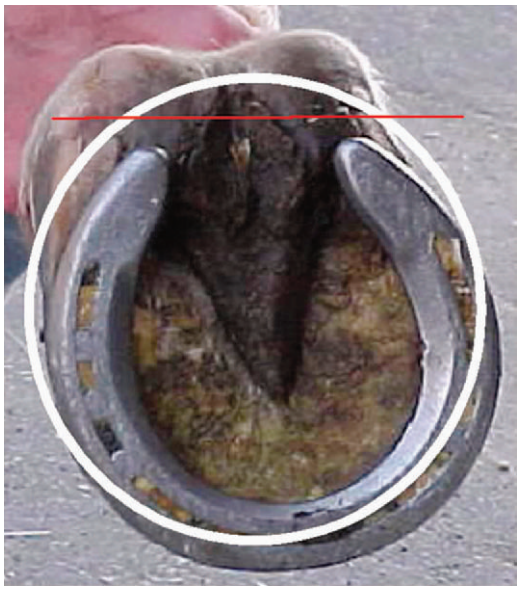

FIGURE 14-21 Assessment of heel support as seen from the sole. The *circle* shows that the foot support is offset laterally; ideally the shoe should be moved toward the white circle. The *red line* indicates the base of the frog where the heels of the shoe should be.

shaped. Front and rear hooves should be shaped like inverted cones. Both hooves should be evaluated for differences in length and width. Hooves of equal width and length tend to look circular, but as the length becomes greater than the width, the hoof wall in the quarters becomes more upright and the stresses on the hoof will naturally be different. The levelness of the hoof has two aspects. One, is the ground-bearing surface flat? This determines how evenly the hoof wall will bear weight. Two, is the ground-bearing surface perpendicular to the upper limb? This determines how the leg is loaded during weight bearing. These factors are the basis for determining medial to lateral hoof orientation.

The final observation is to evaluate the heel support (Figure 14-21).[9] This is done by evaluating the location of the ground-bearing surface of the heels relative to the remaining hoof capsule, relative to the pastern and relative to the fetlock and cannon bone. Does the ground-bearing surface provide sufficient support to the palmar (plantar) aspect of the digit? Are the heels of the hoof centered under the cannon bone (from the palmar/plantar aspect) or are they offset? This can be important in determining how the horse loads the heels, whether the heels are landing simultaneously, or whether one heel strikes before the other. These observations are helpful for the examiner to understand how the hoof capsule has grown and remodeled to adapt to the forces on it.

Watching the horse at a walk will enhance the previous observations.[12] Observing the foot in motion should determine the manner in which the horse lands and breaks over, as well as the path of the foot during the flight phase of the stride. Toe-first landing or excessively heel-first landing indicates either compensation for pain or dorsopalmar hoof imbalance. Similarly, medial or excessively lateral heel/quarter-first landing suggest either compensation for limb conformation or pain leading to mediolateral hoof imbalance. The flight of the foot during the stride is correlated with rotational deviation of the limb and imbalance of the foot. The horse that wings-in or "dishes" is either toed-out or breaking over the inside toe. Conversely, the horse that paddles or wings-out is either toed-in or breaking over the outside toe.

Once these observations are made, the examiner needs to make a closer evaluation of the hoof.[12] This evaluation needs to be performed first with the horse in weight-bearing position and then with the foot in non–weight-bearing position; this begins by palpating the pastern.[12] Palpation for any obvious heat, pain, or swelling goes without saying. More subtly, the examiner needs to palpate the bones and tendinous structures. Generally the flexor tendons are not as wide as the pastern bones and there is a fingers-width difference medially and laterally. The examiner should follow the tendons down the leg until they disappear at the heel bulb.

Next the digital arteries, vein, and nerve are palpated.[12] It is normal to feel a digital pulse but not a bounding pulse, which is abnormal and an indicator of foot inflammation. The strength of the pulse can be compared to other limbs if there is any doubt. A symmetric abnormal pulse indicates generalized inflammation, whereas an asymmetric pulse indicates the inflammatory process on the side of the stronger pulse. In addition, the skin should to be carefully palpated for the presence of neurectomy scars.

Palpation is then continued to the coronet (hairline/hoof capsule junction).[12] One technique is to palpate from the central toe region caudally on the medial and lateral aspects. Normally one should appreciate a "spongy" feel to this area, and deviation such as swelling, discharge, focal pain or heat, or absence of tissue (loss of sponginess or a "trough") should be examined more closely. The examiner should feel that the hairline forms a smooth edge with the hoof capsule. Any area where the hoof capsule is prominent indicates an area of stress. The author believes that these edges indicate a proximal movement of the hoof capsule ("jamming") into the hairline. In many breeds, particularly in the gaited breeds that carry longer lengths of hoof, this seems to be normal. As the edge becomes more prominent, the examiner can be sure that the vertical distance from the hairline to the extensor process of P3 is increasing (measurement that is made from a lateral radiograph).

From the coronet, the examiner moves to the collateral cartilages, where they are palpated and manipulated.[12] The palmar and proximal edges should be easily defined. The thickness, density, and pliability of the cartilages need to be assessed. Palpation of this area not only will determine if there is any pain but also can give an impression of the flexibility of the hoof. For instance, generally speaking a very stiff, inflexible collateral cartilage is associated with a narrow, upright hoof. On the other hand, flimsy cartilages are commonly seen in the hoof with collapsed heels and a narrow, convex frog.

The entire hoof wall must be examined for the presence of cracks, fissures, bulges, growth abnormalities, focal heat, wall loss, or breakage.[12] A high percentage of quarter and heel cracks begin as small very fine fissures at the coronet. They may extend less than 1 cm distally and are easily missed if this area is not carefully examined. In fact, it is advised to pay particularly close attention to any area of the hairline that is not straight. These small fissures are a definite cause of foot pain and usually associated with deeper injury to the coronet and lamina below.

The exit of all shoeing nails from the hoof capsule needs to be evaluated.[12] The higher the exit point is, the more likely it is that the nail is impinging on sensitive tissue. This is an excellent time to use the hammer and gently percuss the hoof wall to determine wall defects, hollow sounds, or painful areas.

From this point, it is natural to begin manipulating the foot in the non–weight-bearing position.[12] The examiner begins by cleaning the bottom of the hoof, using the dull side of a hoof knife. Next, the examiner turns the hoof knife to use the blade to lightly pare away any debris that obscures an accurate visualization of the frog, sulci of the frog, sole, and white line if the horse is unshod. Once the foot is clean, it should be examined in its entirety. The frog should be examined for size, shape, and consistency and to determine whether it is securely attached to the underlying tissue and its sulci (collateral and central). The examiner needs to determine how much of the structure could actually bear weight and how much represents loose tissue. It is the author's opinion that the frog should be a resilient, rubbery substance versus a hard, flaky substance. The frog should be nearly even with the ground surface of the hoof wall, particularly the caudal two thirds of the frog. The frog should not be recessed deep into the sulci of the foot, nor should the frog be convex at its apex. The receded frog is often associated with upright narrow feet, whereas the convex frog is associated with weak and underrun heels. The author has long thought that this conformation is associated with a poorly constructed digital cushion and therefore a poor hoof support mechanism, but this has yet be proven.

The medial and lateral bars of the foot usually require light paring with a hoof knife to appreciate problems such as bar cracks.[12] The bars should not be pared totally, as this weakens the foot. The entire sole of the foot should be carefully examined for fissures, punctures, consistency, discoloration (bruising), and the degree of concavity. The shape of the sole should be concave. If it is not, then the sole will be either flat or convex. A flat sole may signify either poor hoof conformation (a weak hoof) or coffin bone displacement. A concave sole, however, indicates a displaced coffin bone. The consistency (relative degree of stiffness) is usually determined using digital pressure as well as hoof testers. At this point, it is necessary to evaluate the texture of the sole. By grasping the quarters with the fingers, the thumbs can be used to gently press on the sole. If the sole moves under this pressure, it is thin and the examiner knows that there is little space between the coffin bone and the outside environment. On the other hand, if the sole does not move, the examiner knows there is at least some thickness and depth to the sole. The true sole depth can be determined later via radiography.

The white line is examined to determine its width and character.[12] The white line is usually wider at the toe and gradually tapers to a thinner structure as it approaches the heels. It is best visualized after either light paring with the hoof knife or light rasping of the superficial portion of the foot. It is used to demarcate the insensitive hoof from the sensitive hoof for the purpose of driving horseshoe nails. Everything outside the white line is insensitive; everything inside is considered sensitive. Widening of this area represents stress and separation of the laminar hoof wall from coronary hoof wall. The deeper the separation goes, the more severe the injury is. This separation can be seen anywhere on the solar surface and indicates a bending force on the wall that is pulling the wall away from the coffin bone. Most frequently, this separation is seen at the toe and is referred to as "seedy toe" because it looks like small seeds could fit between the spaces created by the separation.

The bulbs of the heels are examined to determine their relative position to one another.[12] The strength of this tissue is assessed manually by attempting to distract the two bulbs from one another in a vertical direction. The heel bulbs should be digitally explored for the presence of swelling, heat, pain, or separation at the coronet. The central sulcus of the frog needs to be examined and probed to determine its depth. Normally this should be a shallow depression of no more than a centimeter. If the sulcus goes deeper, it can be due to either very serious thrush or loss of structural support in the heel bulbs (the heel bulbs can be distracted in opposite vertical directions).

Next, the examiner should lightly support the limb at the metacarpus (metatarsus) and allow the foot to drop naturally.[12] The line of vision should be positioned so that the examiner can appreciate foot balance and levelness of the walls. The levelness of the hoof has two aspects. One, is the ground-bearing surface flat? This determines how evenly the hoof wall will bear weight. Two, is the ground-bearing surface perpendicular to the upper limb? This determines how the leg is loaded during weight bearing (hoof balance). The entire ground surface of the foot should be examined to determine the divisions of the hoof (toe, quarters, and heels) and their proportions. Generally the front hoof should be circular in shape, whereas the rear hoof is more triangular or pear-shaped. Front and rear hooves should be shaped like inverted cones. By imagining a line drawn through the axial center of the limb, which transects the ground surface of the foot, the examiner can determine the relative proportion of medial and lateral foot to this imaginary line. For example, a given foot may demonstrate a unilateral medial heel contraction in combination with a flared lateral quarter and toe (diagonal imbalance).

The palpation of the cartilages of the distal phalanx and the coronet is repeated.[12] Bringing the limb forward and flexing the toe facilitate palpation in the region of the extensor process of the distal phalanx region and the associated distal interphalangeal joint. The thumbs can then be pressed over this area to feel for joint distension, heat, or pain. The foot also should be rotated (twisted) medial and lateral around the vertical axis of the pastern. A normal range of motion allows for 10 to 15 degrees of rotation each way. Injury to the joint capsule, injury to the collateral ligaments, or chronic navicular pain tends to reduce this motion. Likewise distal limb flexion should reveal 30 to 45 degrees of excursion. Again, injury to the joint capsule, injury to the collateral ligaments, or chronic navicular pain tends to reduce this motion.

If the horse is shod, the examination should include the following additions.[12] First, the examiner determines the security of the shoe to the foot by gently rapping the shoe at 1-inch intervals with a shoeing hammer. Note should be made of the shoe type as well as the presence or absence of additions such as toe grabs, block heels, trailers, and so on. The examiner should carefully determine if abnormal shoe wear exists and carefully record the findings, as it is easy to forget subtle discoveries that may ultimately determine how the horse should be treated or shod.

As part of the overall evaluation of these horses, an objective assessment of hoof balance is important.[12,49] Eleven measurements are made of each foot. The horse's weight is determined with a weight tape or scale. Seven measurements are made of the hoof length with a tape measure: medial and lateral heel lengths, medial and lateral quarter lengths, dorsomedial and dorsolateral toe lengths, and sagittal toe length. These measurements are recorded on a graph to illustrate the general shape of the foot. In addition, the frog's length and width are measured at their longest and widest points. The hoof circumference immediately below the coronary band, and the hoof angle (using a hoof gauge) are also measured. From these measurements, two additional measurements can be calculated: the frog ratio (frog width divided by length) and the body size to hoof area (horse's weight in pounds multiplied by 12.56 and divided by square of the hoof wall circumference in inches).

A dorsopalmar (or plantar) and a lateral radiograph of the hoof can also be used to determine valuable information about hoof balance.[50] The horse must be standing with the metacarpus (or metatarsus) perpendicular to the ground, which can most easily be determined by either the use of a level placed against the cannon bone or the use of a weighted string to align the leg. The radiographic beam should be horizontal and centered on the hoof. Resting the horse's foot on a block to raise the hoof off the ground facilitates these exposures (the opposite limb should be similarly elevated). For easy identification of landmarks, radiopaque markers can be attached to the hoof. A wire or radiopaque paste placed sagittally (midline) along the toe from the coronary band to the ground, a thumb tack in the apex of the frog, and thumb tacks in the most caudal point of the ground contact of each heel emphasize these areas on the radiographs, making their identification much easier.

After plotting the hoof wall lengths, one should have a curve that reflects the shape of the hoof.[50] For a hoof of average hoof angle (48 to 55 degrees), flattening of the plotted curve indicates that the heels are underrun. A flat curve would also be expected for very upright hooves (greater than 60 degrees). Generally speaking, the three measurements at the toe should be equal. The measurements at the quarter are usually 1 to 2 cm shorter than at the toe (for the average hoof). The heel length should generally be about one third of the toe length. Another factor to evaluate is the size (weight) of the horse versus its foot size. Guidelines have been made relative to toe hoof length.[52] The average toe length is a function of the horse's weight. The average pleasure horse weighing 800 to 900 pounds (360 to 400 kg) should have 3 inches (7.6 cm) of hoof length at the toe. A horse weighing 950 to 1050 pounds (430 to 480 kg) should have 3.25 inches (8.25 cm) of hoof length, and horses weighing 1150 to 1250 pounds (520 to 570 kg) should have 3.5 inches (8.9 cm) in length. Another rule of thumb that has been suggested is to add or subtract $^1/_8$ inch of length for every 200 pounds using 1000-pound horse carrying 3.25 inches of length. Obviously these are only guidelines. A measure that has been an excellent aid to assessment is comparing the horse's weight to the coronary band circumference.[50] By using the formula of the horse's weight multiplied by 12.56 and divided by the square of the coronary circumference, a weight/hoof-area ratio is determined; 99.5% of horses have a ratio of 78 lb/in^2 (5.5 kg/cm^2). Horses with a higher ratio are too heavy for their hoof size. The measurements of the frog length and width are used to determine whether the hoof is contracted. Normally the frog width should be two thirds its length. Furthermore, the frog length should be two thirds of the solar length (heel bulbs to toe).

Examination of the lateral and dorsopalmar (or dorsoplantar) radiographs provides excellent pictorial evidence of imbalance.[50] The lateral radiograph should be evaluated for the second phalanx (P2) and P3 alignment, which gives insight into the presence of a broken-hoof axis. In addition, the alignment between P3 and the hoof wall should be assessed. If the hoof wall and dorsal surface of P3 are not parallel, the functional hoof angle can be determined by measuring the angle of the dorsal surface of P3 with the ground. Usually the slope of the heels can be seen on the radiograph and can also be used to determine whether the heels are underrun.

The dorsopalmar/plantar (DP) radiographic projection should be assessed for joint alignment, medial and lateral hoof wall lengths, and foot symmetry.[50] Joint alignment is determined by examining the symmetry of the joint space. Misalignment is present if one side of the joint is narrower. This phenomenon can also be caused by poor positioning, in which case all three of the lower leg joints (fetlock, pastern, and coffin) will be affected. The hoof wall length can be measured directly from the radiograph. The symmetry of weight bearing can be predicted in a similar manner.

Differential Diagnosis

Through thorough examination of the horse affected by pain in the palmar region of the foot, a more precise diagnosis can be made, whether the diagnosis reflects injury to the hoof capsule, third phalanx, or podotrochlear region. Treatment then should be based on the type of injury. There are differences in the clinical presentation of navicular region pain (NRP) and palmar foot pain (PFP). Navicular region pain would be applied only to those horses for which the lameness is eliminated by both DIP analgesia and bursa analgesia; PFP describes all other cases. A differential diagnosis for NRP may include navicular osteitis, navicular bursitis, desmopathy of the proximal suspensory of the navicular bone, desmopathy of the impar ligament, and navicular bone fracture.[31] A differential for the syndrome for other causes of PFP (not obviously differentiated) are deep flexor tendonitis, insertional tenopathy of the deep flexor tendon, distal interphalangeal arthrosis, sole bruising, and marginal third phalanx fractures.

In a prospective study performed at the University of Minnesota, approximately 54% of the cases seen are affected by NRP and 46% by other sources of PFP. In this study, no diagnostic test was pathognomonic for navicular pain.[33] Distal limb flexion has been suggested by many authors to be of importance in the differentiation of navicular disease. In this study, 87.5% of the horses responded to this test. This is in agreement with observations made by Wright.[34] When the horses are grouped according to their response to diagnostic analgesia, the NRP group was positive in 88% of the cases while the PFP group was positive in 87%.[33] This indicates that the test is good for exacerbating pain in the palmar hoof but does not help in the differentiation.

The frog wedge test is thought to exert pressure directly on the navicular bone, similar to hoof testers, but is thought

to be more accurate because the horse's weight exerts the pressure rather than the examiner-induced pressure.[33] About 75% of the horses in the aforementioned study responded to this test, but 76% from the NRP group and 74% from PFP group responded, again indicating no difference.

The toe wedge test was positive in 45 of the 80 horses (56%).[33] This figure is higher than that reported by Wright.[34] However, the test was of no help in differentiating pain. Fifty-five percent were positive in the NRP group and 58% were positive in the PFP group.

Hoof tester examination over the frog is considered by some clinicians as almost pathognomonic for navicular pain.[24,39] In this study, however, hoof tester examination was found not only to be less sensitive than other manipulative tests for navicular pain, but other types of palmar heel pain were also more likely to respond to the hoof tester examination over the frog than horses with navicular pain.[33]

It is clear that the diagnosis of navicular disease must be made based on the response to diagnostic analgesia.[31,33] Several reports have indicated that the response to these blocks in horses with navicular disease can be variable,[28,35,38,53] but in those studies the definition of navicular disease either lacks specificity or the diagnostic criteria lack specificity. The Minnesota study defines the disease based on the location of pain, either the navicular bone and its surrounding ligaments (NRP) or other causes of pain in the palmar hoof (PFP).[31,33] Taken individually, the analgesic blocks have variable responses. Palmar digital nerve analgesia eliminated the majority of the pain in every horse in this study. Therefore, its sensitivity is 100% for NRP. However, sensitivity is an inappropriate assessment because the analgesic blocks were criteria for grouping in the study. Specificity of the analgesic blocks does provide interesting information. Palmar digital analgesia had specificity for NRP of 0, whereas, DIP analgesia had a specificity of 87% and podotrochlear bursa (PB) analgesia has a specificity of only 59%. This indicates that the single most accurate diagnostic test for navicular pain is DIP analgesia. According to this study, if the horse's lameness markedly improves after the DIP analgesia, there is an 89% chance that the horse has pain in the navicular region.[33] This assumes that the horse also markedly improves with palmar digital analgesia. Based on the evidence of this study, it appears that if a horse responds profoundly to DIP analgesia, the horse either has DIP pain or navicular pain. This finding is in agreement with observations Schebitz made 30 years ago.[3] The PB block, which in the past was thought to block only the bursa, appears to have more effect. In the Minnesota study, nine horses responded profoundly to PB analgesia but had little or no response to DIP analgesia. This indicates that the navicular bone is not the source of pain. Further, scintigraphy indicated increased activity in the third phalanx in each of these cases.[31] The palmar digital nerve comes in very close proximity to the bursa, and it seems likely that PB analgesia will either desensitize the navicular area or the solar surface, including the insertion of the deep flexor tendon, of the third phalanx.

Use of the PB contrast study has provided new information regarding the flexor cartilage, the presence of adhesions between the deep flexor tendon and navicular bone, and possible tendon damage.[18,31,33] Adhesions between the deep flexor tendon and navicular bone were seen as space-occupying lesions in the dye column across the flexor surface of the bone. In each of the cases in which this was noted the horse had navicular pain. Tendon damage was noted when the dye filled small defects in the tendon. This finding was present only in the palmar heel pain group of horses. Flexor cartilage damage was evidenced by the loss of cartilage on the flexor surface. This finding was noted equally in horses with navicular pain and the group with other causes of palmar heel pain. This suggests that flexor cartilage erosion is probably of little consequence or at least highly variable in causing navicular bone pain.

Scintigraphy has been shown to be an excellent imaging method to help identify navicular disease.[54] The Minnesota study confirmed that scintigraphy is useful in the diagnosis of navicular pain but it is not pathognomonic.[31,33] Scintigraphy must be cautiously interpreted. Thirty-six percent of the PFP horses showed increased uptake of the radionuclide within the navicular bone and 20% of the NRP horses did not have increased uptake (Turner, unpublished data, 1982-2005). This finding indicates that there can be navicular region pain without increased navicular bone remodeling and that navicular bone remodeling is a component of some cases of PHP.

Reduced circulation has been considered a component of the pathogenesis of navicular syndrome.* A unique method to determine qualitative differences in the blood flow has been used that involves measuring skin temperatures before and after exercise in the region of the pastern.[49] The Minnesota study has shown that decreased blood flow is more commonly associated with PFP rather than NRP.[31,33] Logically, poor blood flow to the foot should affect the entire foot rather than the navicular bone. This agrees with the one clinical study in which occluded blood supply to the navicular bone did not result in lameness.

The Minnesota study has helped to show that the clinical findings associated with navicular region pain are similar to those seen in horses with palmar heel pain.[31,33] The clinician should be able to differentiate navicular bone pain from other causes of palmar heel pain using appropriate nerve blocks. The more accurately or the more specifically those cases are defined, the better the conclusions are that one can draw from a clinical series of cases. The poor definition of cases explains in part the high variability of treatments for navicular disease. Further, it is logical to assume that the more precise the diagnosis is, the better the therapeutic and prognostic decisions that can be made.

Thorough examination of the horse affected with navicular syndrome is important not only to determine that the horse has the syndrome but also to try to determine which type of disease process is at work.[32] Treatment then should be based on the type of injury. Responses can be broken down according to the following results[31,33]:

- Palmar digital analgesia improves all these horses greater than 90%.
- Horses that improve greater than 90% with DIP analgesia and greater than 90% with navicular bursa suffer navicular region pain (about 50% of the cases).
- Horses that improve greater than 90% with DIP analgesia, but in which navicular bursa analgesia has no effect, suffer from DIP joint pain (about 10% of the cases).

*References 6, 10, 19, 25, 49, 55.

- Horses that show no effect from DIP analgesia, but in which navicular bursa analgesia improves the lameness greater than 90%, suffer from distal deep flexor pain, deep flexor insertional tenopathy, or solar third phalanx pain (about 10% of the cases).
- Horses that improve some, but not significantly from either DIP or navicular bursa analgesia, have mixed sources of pain (about 25% of the cases).
- Horse that are not effected by either DIP or navicular bursa analgesia suffer from deep flexor pain or heel/quarter hoof capsule pain (about 5% of the cases).

TREATMENT

Thorough examination of the horse affected with caudal hoof lameness syndrome is important not only to determine that the horse has the syndrome but also to try to determine which type of disease process is at work.[31] Treatment then should be based on the type of injury.

The treatments of navicular syndrome vary widely, which probably reflects the treatment of multiple causes.* By determining the most likely cause of the syndrome, the most specific problem can be treated. The treatment of caudal hoof lameness is as controversial as any aspect of this syndrome. However, it has been shown that correct shoeing should be the basis of all treatment. Any medicinal or surgical therapy should be an adjunct to shoeing.

The most successful approach to shoeing is that based on individual case needs rather than a standard formula.[9,11,51,59,61] The following principles should be followed: (1) correct any preexisting problems of the hoof, such as underrun heels, contracted heels, sheared heels, mismatched hoof angles, broken hoof/pastern axis; (2) use all weight-bearing structures of the foot; (3) allow for hoof expansion; (4) decrease the work of moving the foot. Shoeing is most effective when corrections are made within the first 10 months of lameness, with up to 96% success rate. In contrast, when shoeing changes are not made until after 1 year of lameness, only 56% of the cases have been successfully treated.

These principles can be accomplished using many different methods and techniques. Shoeing is of utmost importance in dealing with hoof pain causing the signs associated with navicular syndrome or remodeling of the bone (osseous form). It is necessary to ensure proper hoof balance and support to eliminate the pain and stop or decrease the stresses that are causing the problem.

Six hoof balance abnormalities have been described: broken hoof axis, underrun heels, contracted heels, shear heels, mismatched hoof angles, and small feet.[26] Some authors have attempted to define these hoof abnormalities objectively. A broken hoof axis exists when the slopes of the pastern and hoof are not the same. This condition is further defined as *broken-back* when the hoof angle is lower than the pastern angle and as *broken-forward* when the hoof angle is steeper than the pastern angle. Underrun heels have been defined as angle of the heels of 5 degrees less than the toe angle. A contracted heel is defined as frog width less than 67% of the frog length. Sheared heels are defined as a disparity between the medial and lateral heel lengths of 0.5 cm or more. Small feet (small feet to body size) are defined as a weight/hoof-area ratio of greater than 78 lb/in^2.

Numerous factors contribute to the balance of an equine hoof.[52] Toe length is important because it determines the length of the lever arm over which the limb rotates and the timing of hoof lift. Hence, a long toe, that would delay breakover, could be expected to increase the pressure of the deep flexor tendon over the navicular bone, increase the tension on the proximal suspensory ligament of the navicular bone, and increase the dorsal rim pressure on the joints of the leg. The optimal toe length has not been determined. Toe length will to a certain extent be dictated by the use of the horse as well as its height and weight. Guidelines have been described that relate toe length to body weight. A graph of hoof measurements documents disparities in hoof wall length between feet.[50] Lengthening one hoof over its opposite has been suggested as a treatment for limb length disparity in the horse. However, this condition has not been scientifically documented in the horse. It has been the author's experience that apparent limb length disparities are more commonly due to mild flexural deformities (contracted tendon) rather than actual differences in limb length and that this condition is most commonly manifested as mismatched hoof angles. One study indicated that 28% of normal performance horses might be affected in this manner.[26] This can be documented most easily using lateral radiographs.

The hoof angle should be the same as the hoof axis.[49,52] Using the lateral radiographic projection, the ideal hoof angulation to properly align the second and third phalanges can be measured accurately (Figure 14-22). The appropriate correction can be determined by measuring the degree of malalignment (flexion or extension) present in the coffin joint and raising or lowering the hoof angle that amount. For instance, if the lateral radiographic projection showed 4-degree flexion of the coffin joint, then the hoof angle should be lowered 4 degrees. In most cases, the aligned hoof axis is 52 ± 2 degrees for the front feet and 55 ± 2 degrees for the back feet. Intentional lowering of the hoof angle has been used to attempt to increase stride length in racehorses, but studies have shown that this is not effective; therefore, there is no reason not to shoe for a correct hoof axis, and a broken hoof axis can predispose to lameness problems and has been associated with a greater risk of breakdown in racehorses.

In addition to hoof axis deviations, the lateral radiographic projection can be used to document problems of heel support,[50] such as underrun heels. In horses with hoof angles between 50 and 55 degrees, the hoof length graph also documents underrun heels if the ratio of toe length to heel length is less than 3:1. Either drawing a bisecting line through the metacarpus to the ground or measuring the appropriate position on the radiograph can determine the proper position of the heels. Where these lines contact the ground is the point where the heels should be. From a practical standpoint, the heel-ground contact should be even with the base of the frog. Underrun heels are the most commonly encountered hoof abnormality. In one study of foot related lameness, this condition was found in 77% of the horses and in another study of normal performance horses it was found in 52% of the horses. The necessity of correcting underrun heels has been well documented. If left uncorrected, the underrun condition can cause alterations in hoof wall growth that can be very difficult to correct, and it can predispose the horse to

*References 6, 9, 11, 21, 23, 24, 30, 51, 55-62

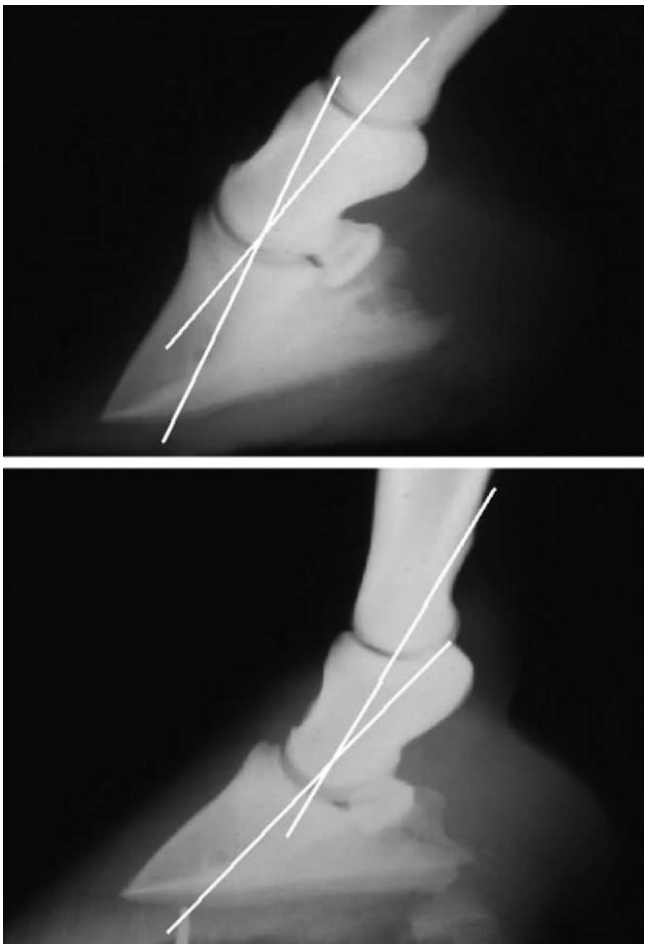

FIGURE 14-22 Lateral radiographs. The angle of the bisecting lines indicates the amount of correction necessary to make the alignment straight.

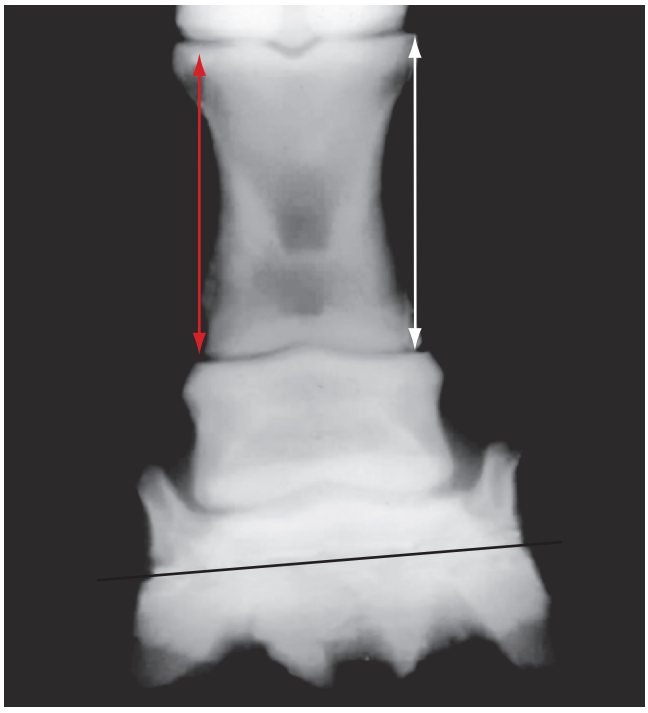

FIGURE 14-23 Dorsopalmar radiograph illustrating one common type of medial to lateral imbalance. The coffin bone can be seen to be not parallel to the ground surface (*black line*). This indicates one side of the hoof capsule is longer than the other. One can also assess the medial and lateral lengths of the pastern bones (*red and white arrows*). If these lengths are not the same, they reflect a conformational rotation in the limb. These rotations cannot be easily corrected.

lameness problems that range from bruised heels to navicular syndrome.

One of the most difficult parameters to assess is the hoof's ability to expand.[50] Applied clinical studies have shown that the ratio of frog length to width is useful for this purpose; when the frog's width is at least two thirds its length, the hoof has normal expansive abilities. When the frog is narrow, hoof expansion is reduced. Whether this is a function of frog pressure is not known, although both reduced and excessive frog pressure have been shown to cause hoof contracture. Identification of a narrow frog should alert the clinician that steps need to be taken to promote hoof expansion. These may vary from simply ensuring proper heel support to encouraging hoof expansion through the use of slipper heels.

Medial/lateral imbalance or shear heels have been shown to cause, or predispose to, a number of hoof-related lameness.[50] Medial/lateral balance can be assessed by both the hoof measurements and the radiographic examination. The graph of hoof wall measurements will clearly show if one side of the hoof is longer than the other. The obvious correction is to make the walls equal, although it is not always that simple. The dorsopalmar radiograph will also clearly demonstrate any imbalance (Figure 14-23). Since this projection also shows the effect the imbalance has on the coffin joint, this radiograph can be used to emphasize the need for correction. The magnification in most radiographs makes even subtle disparities more obvious. It is accepted that conformation can alter this balance. The radiograph will help determine whether the imbalance is hoof related or conformational. Hoof-related imbalances will show medial/lateral hoof length disparities, and the first and second phalanges can be bisected equally. If the medial/lateral disparity is conformationally related the first and second phalanges will appear oblique on the dorsopalmar/plantar radiograph.

The final assessment of balance is the weight of the horse in proportion to its feet.[50] Small feet have been a commonly described problem, particularly in Quarterhorses, that predispose the horse to lameness. One study identified small feet as an indicator of poor prognosis in the treatment of navicular syndrome. Most descriptions of what actually constitutes a small foot are quite subjective. However, studies have been performed using simplistic formulas to make this assessment objective.[26] These studies measured the circumference of the hoof immediately below the coronary band. This was done to get a rough idea of the hoof cross-sectional area. This was then compared to the horse's weight and statistical analysis was performed. A ratio of 78 pounds per square inch was determined to be the maximum weight to hoof area ratio for a normal performance horse. The steps to determine this number have been simplified to the following formula:

$$12.56 \times wt/C^2$$

where *wt* is weight in pounds and *C* is circumference in inches.

Once identified, a high weight/hoof-area ratio can be used to show a client that the horse should lose weight.[50] In addition, it can be used to show the necessity of fitting a shoe as fully as is practical to produce the largest surface area possible for that particular horse's hoof.

The author believes there is a hierarchy of hoof problems.[9,51,59] The most important problems relate to heel support and involve not simply adding a shoe but improving the ability of the quarters and heels of the hoof capsule to bear weight. The next most important issues, in descending order, are medial to lateral balance, improving the ability of the hoof to expand, and body size to foot size mismatches. The least important of the hoof problems appears to be hoof pastern axis. Oddly enough, this is the one most commonly and easily treated.

These hoof balance issues have importance relative to prognosis. Caudal hoof lameness treated with shoeing alone within the first 10 months of lameness has been 97% successful in managing the lameness.[9,59] However, in horses that have been lame for 1 year or more shoeing is only 54% successful. The presence of underrun, contracted, and sheared heels in the feet makes it four times less likely to be successful. Finally, among horses with a weight/hoof-area ratio of 83 lb/in^2 or more, none were successfully treated with shoeing.

Horses that respond to coffin joint anesthesia should be treated for inflammation of that joint.[11,58,62] This treatment may include systemic nonsteroidal antiinflammatory therapy, but intraarticular therapy or specific joint therapy should also be considered. The use of hyaluronic acid and corticosteroids as antiinflammatories within the joint is well documented. A combination of high–molecular-weight hyaluronic acid (10 to 20 mg) and triamcinolone (8 mg) injected intraarticularly followed by a second shot of hyaluronic acid in 2 weeks is a recommended approach. In addition, the use of intraarticular or intramuscular polysulfated glycosaminoglycans has been useful in the control of joint disease, especially if cartilage damage is suspected (500 mg IM, every 4 days for 7 injections). Cartilage damage, at least on the flexor surface, can most easily be assessed by contrast navicular bursagraphy.

Occasionally horses affected with coffin joint synovitis also have a chronic broken-forward hoof axis. Many of these cases appear to be mild flexural deformities. Because of the malarticulation of the short pastern and coffin bones, the joint remains inflamed despite therapy. In these cases, inferior check desmotomy to allow correction of the broken-forward axis has been very useful in treatment of these types of cases.[60,63]

Treatment of vascular forms of the disease will need to be treated with vasoactive drugs.* Four drugs have been used for this purpose. Warfarin is used to improve the circulation to the podotrochlea by increasing the one-stage prothrombin time by 20% up to 50% if improvement not seen within 8 weeks.[6,10,19,25] The drug is administered orally at a dose of 0.2 mg/kg daily (total daily dose 6–85 mg daily to the 500-kg horse). Vitamin K_1 must be available at all times because of the possibility of fatal hemorrhage caused by warfarin. This is also why this drug has fallen out of favor.

Isoxsuprine is the most common drug used to increase the circulation to the podotrochlea,[55] although there is some controversy as to the effectiveness of oral administration.[64] It is administered at a dose of 0.6 to 1.2 mg/kg twice a day until the horse is sound, then administration is decreased to once a day for 2 weeks then further decreased to every other day. The author's approach to using this drug has been to give 1.2 mg/kg bid for 2 weeks, followed by 1.2 mg/kg once a day for 1 week, and then 0.6 mg/kg once a day for 1 week.[11] The drug is discontinued after the fourth week and the effect is reassessed. If the horse becomes lame after the drug has been discontinued, it is restarted at 1.2 mg/kg once a day then reduced weekly to the minimum effective dose.

Other drugs have been studied. Metrenperone is a serotonin antagonist and increases circulation.[11] It has been used at a dose of 0.1 mg/kg b.i.d. However, the drug has not been shown to be as efficacious as isoxsuprine. A new drug that is showing promise is pentoxifylline, which increases red blood cell deformability and decreases blood viscosity, thus aiding circulation.[57] The drug is administered at a dose of 4.5 to 7 mg/kg t.i.d. Clinical trials in Canada have shown much promise, but the research failed to determine which patient profile is best suited to this treatment.

Some surgeries have been suggested to be useful in the treatment of vascular forms of the disease. Palmar digital neurectomy causes vasodilation and the effect lasts as long as that of neurectomy.[11] Fasciotomy of the palmar digital nerve has also been suggested, but the effect does not last and may cause more damage to the nerve.[65]

In cases in which desmitis of the navicular suspensory ligament is suspected there are basically two treatment alternatives. Treatment is designed to reduce strain on the ligament. This can be achieved by either raising the heels of the horse's foot or by cutting the collateral sesamiodean ligaments. Collateral sesamoidean desmotomy is a surgery that has become popular in Europe and has been effective on selected cases of navicular syndrome.[11,30] The surgical approach is made just proximal to the collateral cartilages, just cranial to the digital vein. A 2-cm incision is made, the vein is retracted in a palmar direction, and the collateral sesamoidean ligament can be located as it courses proximally and dorsally over the short pastern bone. A hemostat is used to dissect around the ligament and then transection is performed. Closure is standard. The horse is allowed to rest for 2 weeks for skin incision healing, and then it is returned to work.

Similarly, when the deep flexor tendon is involved, raising the heels of the hoof will decrease strain on the tendon.[9,11,51,59] In addition, desmotomy of the inferior check ligament has also recently been shown to be effective in treatment of these cases.[61,63] If tendonitis is diagnosed, however, the tendon needs to be rested.

Podotrochlear bursa lavage has been suggested for the treatment of true cases of navicular bursitis.[11] Ingress and egress needles are placed in the bursa and isotonic fluid is flushed through the bursa to remove any inflammatory debris.

When all other treatments have failed or have not had the desired affect, palmar digital neurectomy remains a viable treatment alternative.[11,56] Numerous techniques are available but all follow some basic rules. First, the neurectomy will not improve the lameness any more than a palmar digital nerve block. Therefore, it is highly recommended that the nerves be anesthetized with the owner/rider present so that he or she can decide whether the horse has sufficiently improved. Second, neuromas are a common problem but can be avoided by atraumatic surgical technique. Atraumatic surgery can really only be learned by practice. Neuroma

*References 6, 10, 11, 19, 25, 55, 57.

formation can be decreased by allowing the surgical wounds to heal as well as possible before the horse returns to work. This usually requires 4 to 6 weeks' rest after the surgery. Third, the horse will lose skin sensation in the back half of its foot and probably lose all or most of its sole sensation, although it will always know where the foot is. The foot therefore should be protected somehow, usually by a pad.

There are other treatments for caudal hoof pain, but these treatments are the core of the therapy that is available. Drugs that affect bone remodeling are being tested, new analgesic drugs are under investigation, and extracorporeal shockwave are being tested. Where these treatments will fit in the treatment of caudal hoof pain has yet to be determined.

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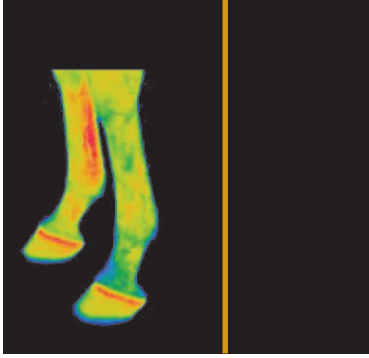

Section IV
LAMINITIS

15
LAMINITIS PATHOPHYSIOLOGY

CHRISTOPHER C. POLLITT

PHASES OF LAMINITIS

A developmental phase, during which lamellar separation is triggered, precedes the appearance of the foot pain (the acute phase) of laminitis. The developmental phase may be as short as 8 to 12 hours, as in the case of laminitis caused by exposure to the water-soluble toxins of black walnut *(Juglans nigra)* heartwood shavings,[1] or as long as 30 to 40 hours, as in the case of excessive ingestion of high-starch grain.[2-4] During the developmental phase and before the clinical appearance of foot pain, the horse or pony usually experiences a problem with one or more of the following systems: gastrointestinal, respiratory, reproductive, renal, endocrine, musculoskeletal, integumentary, and immune. Multisystemic aberrations in organs anatomically remote from the foot result in the lamellar tissues of the feet being exposed to factors that lead to separation and disorganization of lamellar anatomy.

The exact nature of the laminitis trigger factors, apparently reaching the lamellar tissues via the circulation, has yet to be elucidated. Sometimes no developmental phase can be recognized; the horse or pony is discovered in the acute phase with no apparent ill-health or inciting problem occurring beforehand. Obesity and related endocrinopathic problems have recently been incriminated in the pathogenesis of this insidious form of laminitis.[5,6] Grass founder can also appear without warning, and this has now been linked to seasonal variations in the concentration of the soluble sugar fructan by temperate pasture species.[7,8] Fructan can suddenly reach very high concentrations in the stems of grass and trigger a laminitis-inducing gastrointestinal disturbance when consumed by horses and ponies. That laminitis can be induced by such sugars has been verified experimentally using oligofructose, a closely related compound.[9] The parenteral injection of potent, long-acting corticosteroid preparations for the treatment of skin disease may precipitate iatrogenic acute laminitis.[10]

It is important to realize that the process initiating the destruction of the lamellar attachment apparatus begins to operate during the developmental phase before the first clinical sign of laminitis (i.e., foot pain) is apparent. During the developmental phase, the specific problems of the horse often have to be attended to urgently (e.g., acute abdomen, grain overload acidosis, electrolyte imbalance, rhabdomyolysis, retained placenta) and unfortunately the feet are often left out of the therapeutic equation until the first signs of foot pain (shifting weight from one foot to the other) appear. By the time foot pain is apparent, the lamellar pathologic process is underway. In other words, foot pain is the clinical sign that lamellar disintegration is occurring. To wait and see whether foot pain is the sequel to a metabolic crisis is to miss the opportunity to prevent or at least ameliorate lamellar pathology. There is a good correlation between the severity of laminitis histopathology, as seen with the microscope, and the degree of lameness (using the Obel grading system[2]) shown by the horse.[3] When a horse first starts to show laminitic pain, the anatomy of the hoof wall lamellae is being destroyed. The higher the lameness grade is, the more severe the microscopic damage. Any activity that places stress on an already weakened lamellar attachment apparatus (e.g., forced exercise) causes further damage and is contraindicated. The use of nerve blocks to eliminate pain will also encourage locomotion and precipitate more damage.

LAMINITIS HISTOLOGIC GRADING SYSTEM

As laminitis develops, a sequence of histopathologic changes occurs. Three grades of histologic laminitis have been identified based on the degree of severity of the changes. Making the lamellar basement membrane clearly visible is important and requires staining lamellar tissues with either periodic acid-Schiff, or periodic acid silver methanamine stains, or with

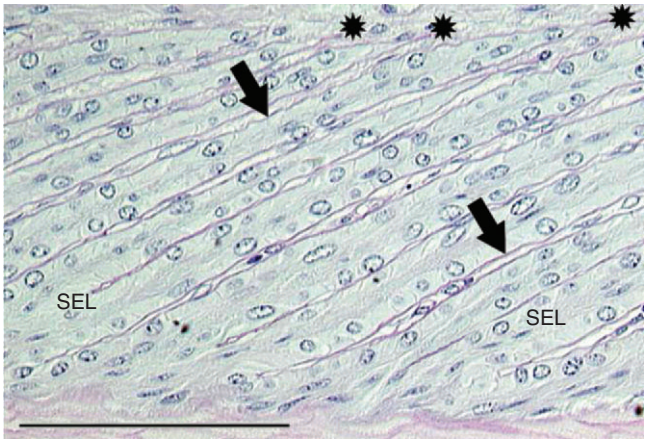

FIGURE 15-1 Grade 1 histologic laminitis. Micrograph showing hoof lamellar tissues stained to highlight the basement membrane (BM). The BM (*arrow*) is stained dark magenta. At the now tapered tips of the secondary epidermal lamellae (SELs), the BM has lifted away (*stars*) from the underlying basal cells. Between the SEL bases, the BM is in its normal position, close to the primary epidermal lamella (PEL). (Periodic acid-Schiff stain; bar = 10 μm.)

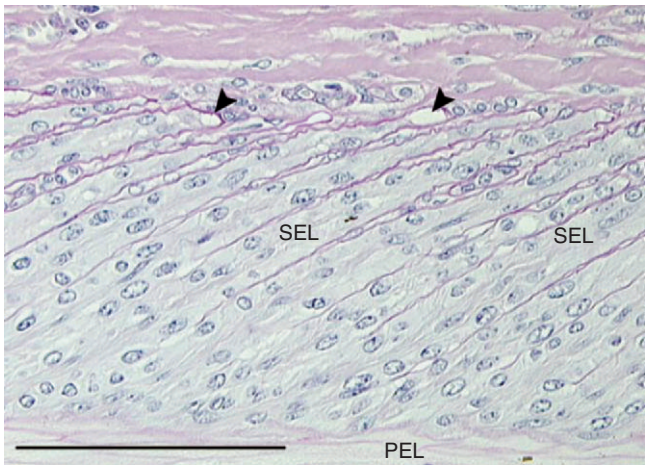

FIGURE 15-2 Micrograph showing hoof lamellar tissues with histologic grade 2 laminitis. The basement membrane (BM) is stained dark magenta. At the tips of the now pointed secondary epidermal lamellae (SELs), the BM has continued to lift from the underlying basal cells to form empty, teat-shaped caps (*arrowheads*). The BM has disappeared from adjacent SEL bases and there is little connective tissue and few capillaries between. The lamellar BM is no longer close to the primary epidermal lamella (PEL). (Periodic acid-Schiff stain; bar = 10 μm.)

immunohistochemical methods using basement membrane–(BM) specific antibodies.

Grade 1 Histologic Laminitis

During the developmental phase, lamellar basal and parabasal cells lose their normal shape, become elongated, and appear to slide over one another and, as a consequence, the secondary epidermal lamellae (SELs) become attenuated with tapering, instead of club-shaped, tips.[3,11] While this is going on, the BM of the SEL loses its attachment to the basal cells. This is first noticeable at the tips of the SELs, where small teat-shaped bubbles of loose BM form. To render this detectable by light microscopy, the tissues should be stained with periodic acid Schiff, or periodic acid silver methanamine stains (Figure 15-1).

Grade 2 Histologic Laminitis

With disadhesion occurring between the lamellar BM and the SEL basal cells, the BM is drawn further away with each cycle of foot loading by the horse. The lamellar basement membrane is now absent between the bases of adjacent SELs.[12,13] The BM retracts from between the SELs and takes with it secondary dermal lamellar connective tissue and secondary dermal lamellar capillaries (Figure 15-2). The loss of these capillaries may explain why resistance to blood flow was increased 3.5 times (the bounding digital pulse) in horses during early laminitis[14] and why blood appears to bypass the lamellar capillary bed through dilated arteriovenous anastomoses in horses with acute laminitis.[13] Both of these phenomena are now placed after the triggering of matrix metalloproteinase (MMP) production and occur as a consequence of it. The epidermal basal cells that have lost their BM attachment do not appear to undergo necrosis, at least initially, and clump together to form amorphous, BM-free masses on either side of the primary lamellar axis.

Grade 3 Histologic Laminitis

In laminitis, the worse case scenario is rapid and total BM separation from all the epidermal lamellae. Sheets of BM peel away to form aggregations of loose isolated BM in the connective tissue adjoining the lamellae. The epidermal lamellar cells are left as isolated columns with no connection with the dermal connective tissue. The lamellar tips slide away from the BM connective tissue attachments, at first microscopically; but as the degree of separation increases, the distance between hoof and distal phalanx becomes measurable in millimeters (Figures 15-3, 15-4, and 15-5). This is manifest clinically as the "sinker." Since the BM is the key structure bridging the epidermis of the hoof to the connective tissue of the distal phalanx, it follows that the wholesale loss and disorganization of the lamellar BM inexorably leads to the failure of hoof anatomy so characteristic of the chronic stage of laminitis.

PATHOPHYSIOLOGY OF LAMINITIS

The spectacular disintegration of the lamellar attachment apparatus, initiated during the development phase of laminitis, compromises a normally robust and trouble-free hoof–distal phalanx attachment apparatus in a surprisingly short period of time. Logic suggests that it is normally a tightly controlled metabolic process that is thrown into disarray to cause the lamellar-specific lesion of laminitis during its developmental phase.[9]

The enzymatic remodeling of the epidermal lamellae, assumed to be mandatory if the continually proliferating stratum medium of the hoof wall[15] is to move past the stationary distal phalanx, could be accidentally recruited in the pathogenesis of the laminitis disease process. Enzymes capable of destroying key components of the lamellar attachment apparatus have been isolated from normal lamellar tissues[16]

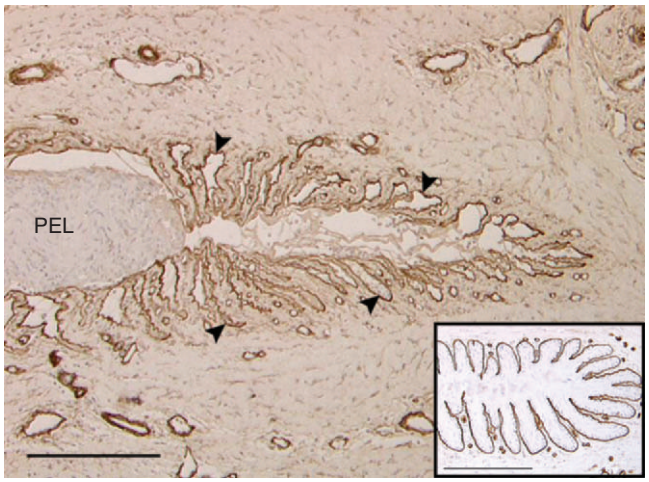

FIGURE 15-3 Grade 3 histologic laminitis. The basement membrane of a lamellar tip is highlighted by staining. The tip of the primary epidermal lamella (PEL) has completely detached from its basement membrane. The PEL basal cells are now an unattached, amorphous mass. Collapsed tubes of basement membrane, now empty of epidermal cells, are still attached to connective tissue (*arrowheads*). The PEL has already moved 0.03 mm from its dermal compartment and soon the distance will be measurable in millimeters on a radiograph. The inset shows a normal lamellar tip, immunostained the same way. (Type IV collagen immunostain; bars = 10 μm.)

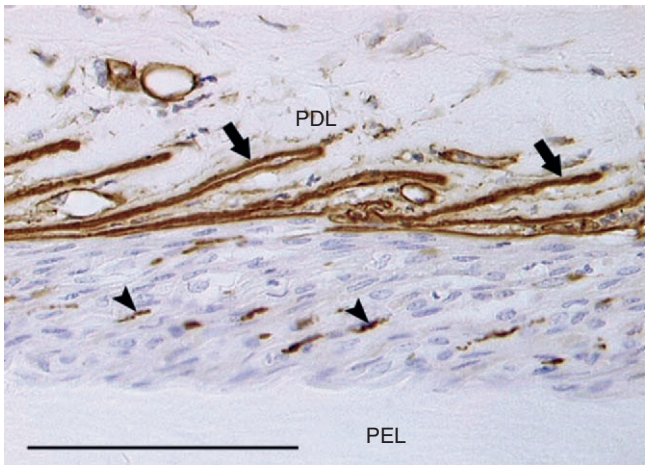

FIGURE 15-4 Grade 3 histologic laminitis. Only remnants (*arrowheads*) of the basement membrane (BM) remain between the now disorganized secondary epidermal lamellae. Most of the lamellar epidermal cells have coalesced into an amorphous mass no longer effectively attached to any connective tissue. The remainder of the lamellar BM lies free, in strands (*arrows*), among the connective tissue of the primary epidermal lamella (PEL). (Type IV collagen immunostain; bar = 10 μm.) *PDL,* Primary dermal lamina.

and in increased quantities from lamellar tissues affected by laminitis.[17] The enzymes are metalloproteinase-2 and metalloproteinase-9 (MMP-2 and MMP-9), also found in a wide range of human and animal remodeling tissues such as bone, joints, and endometrium as well as in metastasizing malignant tumors.[18]

It is assumed that lamellar MMP activity is constantly responding to the stresses and strains of normal equine life as well as to constant growth. When called for, sufficient MMP is manufactured locally, to release epidermal cell-to-cell and cell-to-BM attachments, as required, to maintain the correct shape and orientation of the hoof lamellae. From time to time, injury to the BM would require its lysis and reconstruction. The controlled release of specific MMP inhibitors keeps this remodeling process in equilibrium and the hoof lamellae and the hoof itself slowly migrate past the stationary basal cells firmly attached to their underlying basement membrane that is in turn attached to the connective tissue of the distal phalanx.

The epidermal cells of other species have been shown to readily increase their production of MMP when exposed to cytokines. Cultures of human oral mucosal keratinocytes respond to the addition of tumor necrosis factor, interleukin-1, and transforming growth factor-1 by increasing production of MMP-9.[19] Lamellar tissues affected by laminitis also increase transcription of MMP[16] and produce MMPs in their active forms,[17] but whether in response to circulating cytokines or to some other trigger factor is yet to be established. Evidence from in vitro studies, using equine lamellar explants, suggests that lamellar MMPs are not activated by exposure to cytokines.[20]

The enzymatic theory of laminitis etiology, based on lamellar MMP activation, challenges the alternate view that laminitis develops because of a vascular pathologic condition affecting the circulation of the foot. A current theory is that venoconstriction and high hydrostatic interstitial fluid pressure (compartment syndrome) impede the flow of blood in the lamellar microcirculation to cause ischemic necrosis of epidermal lamellae.[14] Epidermal cell necrosis, intravascular coagulation, and edema were not identified in sections made from tissue in the early stages of laminitis.[3] The vessels in the primary dermal lamella, even the smallest, were generally dilated without evidence of microvascular thrombi.[21] Further, no abnormalities in the systemic coagulation and fibrinolytic cascades are found in horses with carbohydrate-induced acute laminitis.[22] The gross anatomical appearance of freshly dissected laminitis tissue is one of dryness. Sometimes the lamellae peel apart. Tissues affected by a compartment syndrome exude fluid.

How do the trigger factors of laminitis reach the lamellae? There is now strong evidence from three independent experimental sources that the foot circulation during the developmental phase of laminitis is dilated. [23-25] Laminitis does not occur if the foot is in a state of vasoconstriction during the developmental phase, suggesting that the trigger factors will cause laminitis only if they reach the lamellar tissues via dilated blood vessels at a high enough concentration and over a long enough time period. It follows that therapy aimed at keeping the feet of horses in danger of developing laminitis as cool as possible—and therefore vasoconstricted—is logical. Indeed, acute laminitis can be prevented in a single, cooled limb while laminitis develops in the three remaining limbs maintained at room temperature.[26] Horses, unlike humans, do not regard extremely cold feet as uncomfortable and can tolerate having their feet in iced water for days with no discomfort or ill effect.[27] Scintigraphic studies comparing the circulation of iced feet versus normal feet showed profound vasoconstriction in the cold feet (80.5% of precooled values).[28]

What are the laminitis trigger factors? Since the carbohydrate overload model of laminitis is characterized by endotoxin production, it would seem a safe presumption that macrophages in the circulation, peritoneal cavity, and elsewhere in the

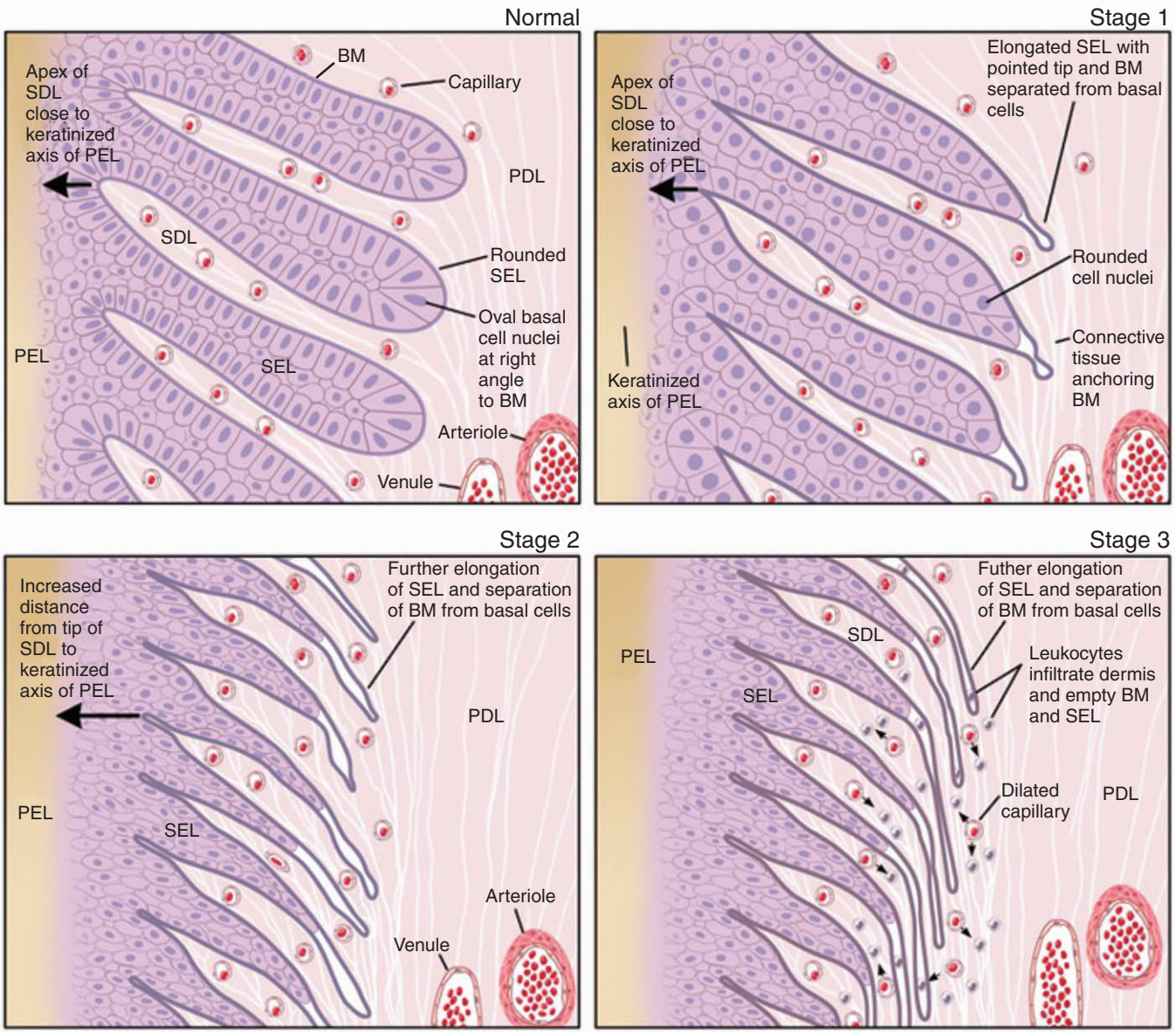

FIGURE 15-5 Diagrams showing normal lamellar histology and three grades of laminitis histopathology in order of increasing severity. *BM,* Basement membrane; *SDL,* secondary dermal lamina; *PDL,* primary dermal lamina; *PEL,* primary epidermal lamella; *SEL,* secondary

body would be subject to endotoxin stimulation just as they are during other acute gastrointestinal diseases.[29] Mononuclear phagocytes express tumor necrosis factor along with other cytokines such as interleukin within minutes of exposure to endotoxin. The cytokine cascade originating from an acute abdomen is responsible for most of the pathologic effects of endotoxemia. However, laminitis has never been triggered by the experimental administration of endotoxin into the bloodstream[30] or the peritoneal cavity, and the actual trigger factors of laminitis remain unidentified. What appears certain in the light of recent research is that the lamellar disintegration of laminitis is mediated by the uncontrolled release of excess MMP.[16]

An in vitro model for equine laminitis has been developed using small explants of tissue taken from the inner hoof wall of normal, freshly killed, abattoir horses. [17,20] Each explant consists of stratum medium, the lamellar layer, and the sublamellar connective tissue. After incubation for 48 hours in tissue culture medium plus the laminitis trigger factor under investigation, each explant is subjected to tension. The force required to separate epidermal from dermal lamellae is recorded. When dermal-epidermal lamellar separation occurs readily, as in field cases of laminitis, the tissue is considered to have developed in vitro laminitis. Lamellar explants can be cultured for up to 7 days in normal medium and no lamellar separation occurs. It is virtually impossible to separate normal lamellar explants. One event that readily causes separation of lamellar explants is MMP activation. The addition to the culture medium of the organomercurial compound aminophenylmercuric acetate, a well-known nonphysiologic MMP activator, readily induces explant lamellar separation. Treatment of lamellar explants with aminophenylmercuric acetate (APMA) is the in vitro laminitis control against which naturally occurring laminitis induction factors can be measured. The presence or absence of MMP activation in explant super-

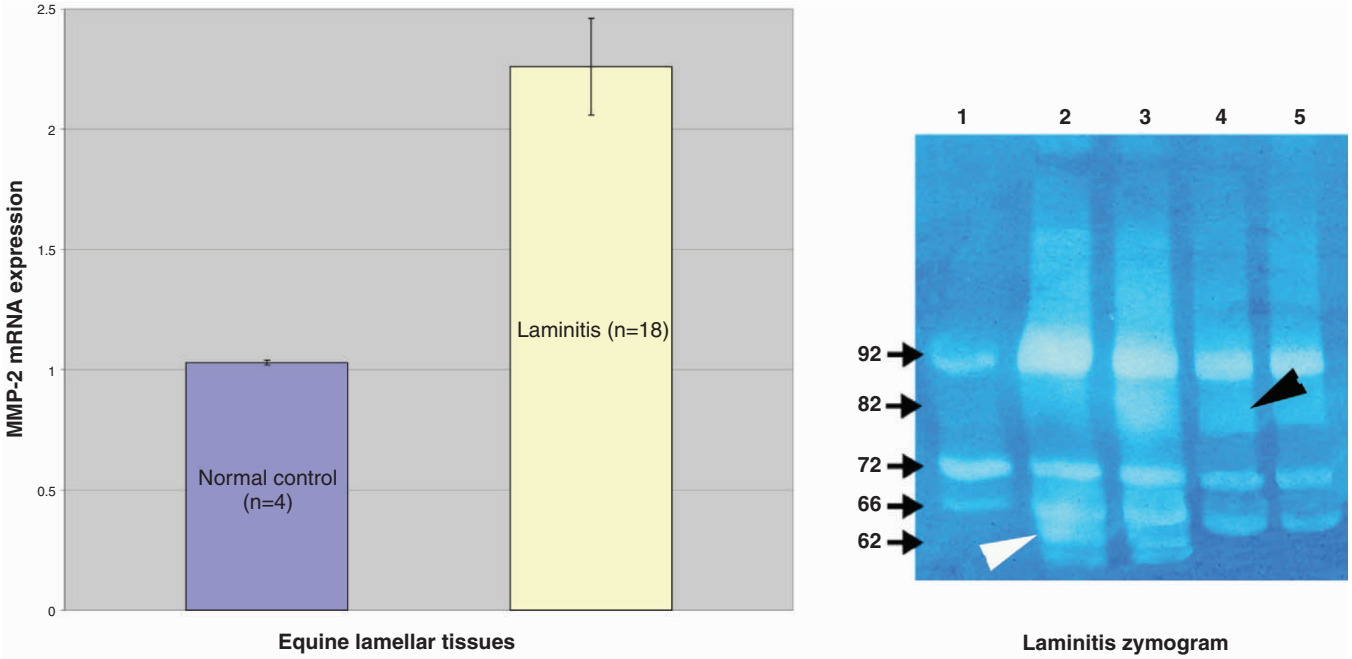

FIGURE 15-6 Graph (*left*) showing the significantly different ($P<0.01$) mean values of matrix metalloprotein (MMP)-2 expression between four normal hooves and 18 laminitis-affected hooves. Polyacrylamide gel zymography (gel contains 0.1% gelatin) of lamellar explants from a horse with laminitis (*right*). Lane 1, normal hoof explant supernatant. Lanes 2 and 3, laminitis forehoof explant supernatants. Lanes 4 and 5, laminitis hindhoof explant supernatants. Molecular weights are derived from standards (not shown). There is a significant increase in the amount of active MMP-9 (82 kDa; *black arrowhead*) and MMP-2 (62 kDa; *white arrowhead*).

natants is detected zymographically using gelatin polyacrylamide electrophoresis, and all explant tissues are fixed and examined histologically. Histologic sections show a clear zone of complete separation between the basement membrane and the basal cells of the epidermal lamellae. This is a characteristic of in vitro laminitis and resembles the basement membrane lesion of natural in vivo laminitis.

The in vitro laminitis explant model has been used to investigate most of the proposed causes of equine laminitis. The equine lamellae have proved resistant to virtually all known cytokines, tissue factors, and prostaglandins. Gram-negative bacterial endotoxin, extract of black walnut *(Juglans nigra),* and even anaerobic culture conditions fail to induce lamellar separation or significant MMP activation. There is one notable exception, however. A factor present in the supernatant of cultures of *Streptococcus bovis* isolated from the equine cecum activates equine hoof MMP-2 and causes lamellar separation.[20] During grain overload, *S. bovis* is the principal microorganism responsible for the rapid fermentation of carbohydrate to lactic acid in the equine hindgut. In the presence of virtually unlimited substrate, its population explodes exponentially. The role of the *S. bovis* MMP activator in natural cases of equine laminitis is currently being investigated.[20] If it crosses the mucosal barrier of the hindgut and enters the circulation, it may be a "cause" of laminitis (at least in the carbohydrate overload model). In other words, it may be an exogenous laminitis trigger factor.

The activity of tissue MMPs has long been shown to correlate strongly with the degree of malignancy and invasiveness of lethal human tumors such as malignant melanoma and breast and colon cancer. The gene responsible for MMP-2 expression in lamellar hoof was recently cloned.[16] Horses with acute laminitis show increased expression of the MMP-2 gene 48 hours after alimentary carbohydrate overload (Figure 15-6).

For mean MMP-2 gene expression to have doubled by the time lameness is manifest implies that the factors signaling the increased expression have been present for some time. This places perturbation of MMP equilibrium early in the cascade of events leading to the foot pain of acute, clinical laminitis. Indeed, biopsies of lamellar tissue taken at 24, 36, and 48 hours (Wattle and Pollitt, unpublished data) all showed some of the histopathologic features described in the published laminitis grading systems.[3,31] At 24 hours, lamellae had intact BMs, but SELs were attenuated with round basal cell nuclei. At 36 hours, SEL attenuation had progressed and SEL basal cells with rounded nuclei were disorganized; SEL tips were pointed instead of rounded. Only at 48 hours was the BM not attached to SEL basal cells, suggesting that the disadhesion process commenced somewhere between 36 and 48 hours. However, the molecular and biochemical events contributing to BM disattachment, as evidenced by nuclear rounding and SEL attenuation, were in place by 24 hours. The basement membrane lesion of laminitis is insidious and well underway by the time clinicians are aware of laminitis foot pain. Any preventive[26] or treatment strategies must be in place before overt foot pain develops if horses are to survive the development phase of laminitis without significant lamellar damage.

A wide range of chemical agents are capable of inhibiting MMP activity both in vitro and in vivo.[32] One of these (Batimastat, or BB-94; British Biotech, Oxford) blocks the

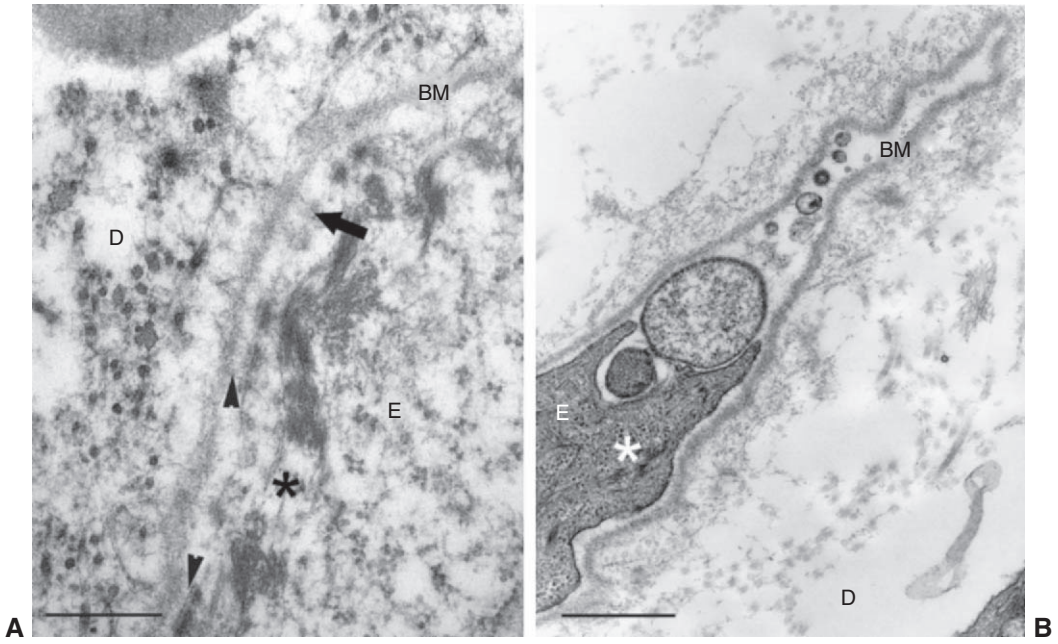

FIGURE 15-7 Transmission electron micrographs of secondary epidermal lamellae (SELs) at the onset of acute laminitis. **A,** Many hemidesmosomes (*arrow*) are absent or faded. Anchoring filaments (*arrowheads*) are present where hemidesmosomes are still relatively normal. Loss and disruption of hemidesmosomes is accompanied by the commencement of basement membrane (BM) separation and damage of the cytoskeleton (*star*). (Bar = 500 nm.) **B,** The BM has separated from the attenuated, pointed SEL tip and formed a typical, empty BM enclosed bubble. There are few recognizable hemidesmosomes and only fragments of cytoskeleton (*star*). (Bar = 200 nm.) *D,* dermis; *E,* epidermal basal cell.

activity of the laminitis MMPs in vitro and has the potential to be a useful tool in the prevention and management of acute laminitis.[17] Trials to test whether MMP inhibitors can prevent or ameliorate field cases of laminitis are currently underway in the Australian Equine Laminitis Research Unit at The University of Queensland.

ULTRASTRUCTURE OF LAMINITIS

Laminitis studied by transmission electron microscopy (TEM) and immunofluorescence microscopy (IFM) has provided new insight into the mechanism of the disease. The hemidesmosome (HD) is the attachment plaque responsible for maintaining contact between the SEL basal cell and its underlying BM. In lamellar SEL samples taken at the onset of acute laminitis, many HDs are absent or disrupted (Figure 15-7). Loss and disruption of HDs is accompanied by BM separation, cytoskeleton damage, and rounding of the basal cell nucleus.

The magnitude of HD loss in SEL basal cells affected by laminitis directly correlates to the dose of carbohydrate used to induce it[11]; these data support a bacterial pathogenesis of laminitis.[20] Presumably greater amounts of substrate support more microbes that produce higher concentrations of laminitis trigger factors.

Hoof lamellae cultured in vitro separate under tension and the intracytoplasmic components of their HDs denucleate and fade if not provided with sufficient glucose[33]; this is a mechanism that may be operating in vivo when toxemia and the various endocrinopathies associated with laminitis limit the supply of lamellar glucose. Activation of constituent lamellar MMPs also causes lamellar separation under tension but without affecting HD ultrastructure. Activated MMPs appear to cleave laminin5 anchoring filaments and set the BM adrift; this is also a process now shown to occur in vivo.[31]

The dependence of sound lamellar architecture on the structural integrity of HDs is well illustrated when foals are born lacking just one of their HD proteins. A 45-day-old Quarterhorse foal showed the clinical signs and gross pathologic appearance of chronic laminitis since birth.[34] Characteristic laminitis histopathologic features were present only in the front feet. In all feet, TEM showed that the intracytoplasmic plaques of lamellar hemidesmosomes were small, misshapen, and not associated with the cytoskeleton. In all feet, IFM showed the hemidesmosomal, intracytoplasmic plaque protein, plectin, was absent. The foal was a rare case of congenital epidermolysis bullosa simplex, an inherited failure to express plectin. Lacking plectin, the cytoskeleton and hemidesmosomes of the hoof lamellae were unstable, resulting in laminitis when the front feet first bore weight. The loss of even a single protein causes structural failure and laminitis. The presence of histologic lesions only in the front feet illustrated the influence of weight distribution on laminitis severity and supports the validity of preventive strategies that unload the feet.

Ideas on laminitis pathophysiology abound,[35] and this review has focused on the MMP, enzymatic theory of laminitis pathogenesis, a hypothesis that depends on the generation of circulating toxins or proinflammatory mediators (laminitis trigger factors) in the gastrointestinal (carbohydrate overload) or reproductive (septic metritis) tracts. A weakness of this hypothesis is the question of how laminitis trigger factors can pass through the lung, kidney, and liver without inducing significant pathologic lesions.[36] Perhaps it all comes down to the unique anatomy of digitigrade equids. MMP activation and basement membrane disadhesion may

be ubiquitous to the epithelia of many organs, but without the influence of weight bearing any resultant pathologic condition is transient. However, weight-bearing BMs such as those within the lamellar dermal epidermal interface of horse's feet separate under tension, a process that may escalate into a cascade of ever-increasing severity. The validity of this proposition will be tested when veterinary researchers learn how to unload the feet of horses during the developmental phase of laminitis.

Acknowledgments

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16
GRADING THE LAMINITIC HORSE

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The objective of a clinical grading system such as the one described in this chapter is to provide, as accurately as possible, an overview of the diagnosis, treatment, and prognosis for the particular disease process. In other words, the following system for grading the laminitic horse was developed as a logical way of determining "what you have, what you can do with it, and what your chances are of full recovery."

This grading system evolved over time and many hundreds of laminitis cases, encompassing acute and chronic conditions as well as horses of various ages, breeds, medical history, and diagnoses at the time of presentation. However, it is important to note that no grading system is infallible or "set in stone." Once a grade has been assigned to a particular horse, it is subject to change, especially during an acute laminitic episode or acute exacerbation of a chronic case. To accurately grade the laminitic horse, each of the following provides essential information:

- Physical examination of the entire horse
- Plain and contrast-enhanced (venographic) radiographs of the foot
- Cumulative history of the case

PHYSICAL EXAMINATION

In addition to routine physical examination findings—such as vital signs (rectal temperature, heart rate, and respiratory rate), hydration status, and conjunctival color (Figure 16-1)—several other observations are pertinent when evaluating the laminitic horse, including the following:

- The horse's stance
- Degree of mobility
- Body condition score
- Evidence of trauma (e.g., presence and severity of abrasions or decubital ulcers)
- Rate and quality of the digital pulses (e.g., are the digital pulses more forceful than normal in any of the limbs? and are those pulses simply a bit stronger than normal or are they bounding?) (Figure 16-2)
- Severity of pain (see Chapter 20)
- Presence and frequency of foot pumping (repeated raising and lowering of the painful foot/feet)
- Visible or palpable abnormalities of the coronary band or hoof capsule (wall and sole)
- Whether the horse is willing to pick up each foot for the examiner (the horse will be reluctant to support all its weight on the most painful foot)

As regards foot pumping, horses with bilateral laminitis repeatedly raise one foot, place it back down, and then raise the other foot in a continuous effort to relieve the load (constriction to the hoof vasculature), first on one foot and then the other. The interval between foot lifts is an indicator of the severity of the foot pain (see Chapter 20).

RADIOGRAPHY

Routine radiography (plain films) and contrast-enhanced radiography (venograms) are each important in grading the severity of digital pathologic lesions related to laminitis. The protocols for interpreting plain films and venograms are summarized in Figures 16-3 and 16-4, respectively, and discussed in more detail in Chapter 17.

Plain Films

Several objective and subjective evaluations can be made from plain films that were produced using exposure factors that allow assessment of the soft tissues within the hoof capsule (see Chapter 10). The following indices on the lateromedial view are particularly important in grading the laminitic horse:

Palmar angle of the third phalanx (P3)—the angle of the palmar margin of P3 relative to the bearing surface of the foot

Horn-lamellar (H-L) zone width—the distance between the dorsal surface of P3 and the outer surface of the dorsal hoof wall (encompassing both the horn and lamellar layers of tissue). H-L zone width is measured at two locations: just distal to the base of the extensor process and at the dorsal-distal margin (tip) of P3. These two values are expressed as a ratio of proximal and distal measurements (e.g., 19/19 mm)

Extensor process–coronary band distance—the vertical distance between the top of the extensor process of P3 and the proximal limit of the dorsal hoof (wall where the coronary band meets the hairline)

Sole depth at the tip (SDT) and the wing (SDW) of P3—the vertical distance between the palmar margin of P3, and the outer surface of the sole. Sole depth is measured at two points, the tip and the wing of P3), taking into account the cup of the sole. These two values can be expressed as a ratio of SDT to SDW (e.g., 20/23 mm)

Soft tissues—nature and severity of any radiographic abnormalities in the tissues between the surfaces of P3 and the outer surfaces of the hoof capsule; this encompasses both the horn and the corium of the dorsal hoof wall and sole

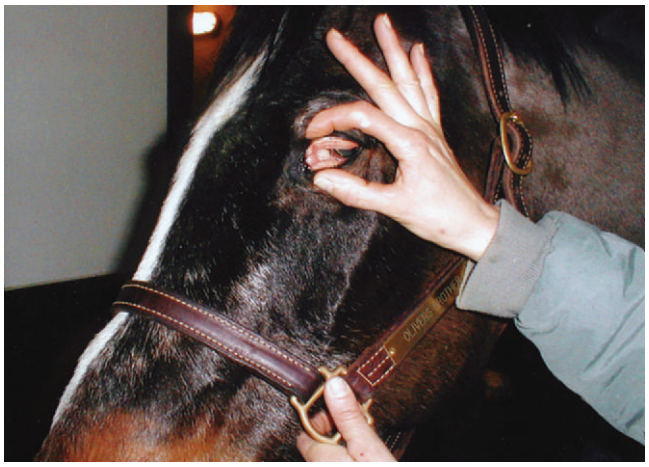

FIGURE 16-1 Using conjunctival color to assess degree of pain or high blood pressure.

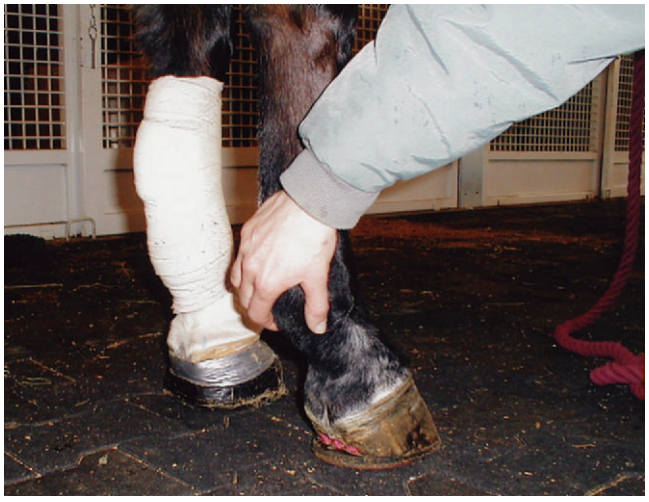

FIGURE 16-2 Assessing bounding digital artery pulses.

Venograms

Venography of the equine digit is discussed in more detail in Chapter 17. When grading the laminitic horse, it is important to carefully evaluate each of the following regions of the digital vasculature for filling deficits:

- Coronary circulation
- Coronal cascade
- Terminal arch
- Dorsal circulation and papillae
- Bulbar circulation
- Circumflex vessels and fimbriae

Examples of filling deficits in these regions are shown later in this chapter.

CUMULATIVE HISTORY

When it comes to grading the laminitic horse, the more information one has about the horse, the better. In addition to the signalment (e.g., age, breed, gender), the following information is particularly important:

- Duration of current ownership
- Date when the laminitic episode began (is the disease process acute or chronic?)
- Findings on previous radiographs (ideally, the new examiner should see the radiographs themselves)
- Previous medical therapy
- Previous farrier interventions
- Results of any blood tests or other diagnostic tests (e.g., has the cause of the disease process been determined?)
- Nutritional status, including supplements given

FIGURE 16-3 Horse with a grade II laminitic event demonstrating difficulty with lateral movements.

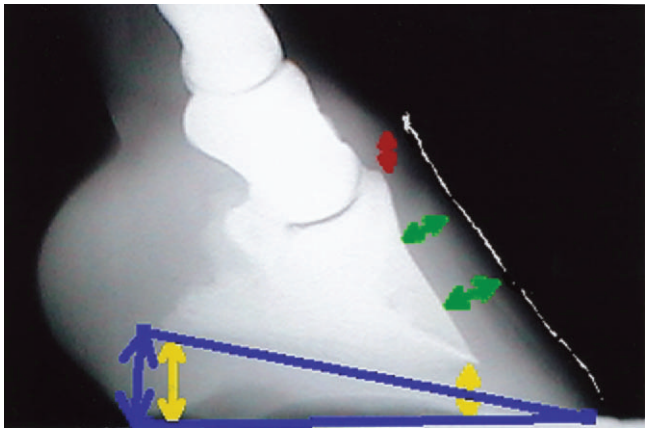

FIGURE 16-4 Radiograph of a grade I laminitic event.

Table 16-1 Grading the Laminitic Horse: Clinical Criteria

Clinical Findings	Grade I	Grade II	Grade III	Grade IV
Foot pain	Mild	Moderate	Severe; spends much of the time recumbent	Extreme; recumbent most or all of the time
Gait	Walks willingly; discomfort may be noted only on tight turns	Walks slowly; reluctant to make tight turns	Walks forward reluctantly, refuses to turn	Stands and walks only with great difficulty
External hoof pathologic changes	None initially; circumferential ring on hoof wall may appear as wall grows down	None initially; circumferential ring on hoof wall as wall grows; sole bruising seen after a few trims	None initially	*Cranial rotation:* often includes coronary band rupture and sole penetration by P3; *FSS:* may be none initially or may have rupture at heel bulbs and frog, flat and friable sole, moveable hoof capsule, with or without coronary band hemorrhage

FSS, Fatal sinker syndrome; *P3,* third phalanx.

GRADING SYSTEM

Many systems have been used, and continue to be used, around the world for grading the laminitic horse. The system used by the author and presented in this section is concise and allows a quick determination of the disease status. This system incorporates the findings of physical examination, plain films, and venograms to grade the severity of the condition on a scale from I (mild) to IV (severe). It is based in part on the physical and radiographic parameters developed by Ric Redden.

Normal Radiographic Characteristics

The grading system is founded on a good understanding of what a normal, healthy foot looks like. It is important to note that, in this context, "normal" is a range rather than a single value. For most of the indices used in this grading system, the normal range encompasses a few degrees or millimeters on either side of the average value given.

In most light horse breeds, a normal foot in an adult horse has the following characteristics:

- Positive palmar angle (PA) of 3 to 5 degrees
- H-L zone width of 15 to 19 mm
- Extensor process–coronary band (EP-CB) distance of approximately 14 mm
- SDT/SDW ratio of approximately 20/23 mm
- No abnormalities seen in the soft tissues
- Solar cup (i.e., concavity to the shape of the sole) present under the digital corium

These normal parameters are the basis for the radiographic component of the following laminitis grading system. Clinical, radiographic, and venographic criteria for each of the four grades are summarized in Tables 16-1, 16-2, and 16-3, respectively.

Grade I

Clinical Findings

Foot pain generally is mild; in fact, discomfort may be noted only on tight turns or figure-eight patterns. These horses walk willingly and have no external pathologic hoof changes related to the acute laminitic condition (see Figure 16-3). Over time, a demarcating ring around the entire circumference of the hoof wall may become visible as it grows down from the coronary band.

Radiographic Findings

Plain Films. Typical abnormalities found on plain soft-tissue films are summarized in Table 16-2 and illustrated in Figure 16-4.

Venogram. The venogram may be unremarkable, although minor slipper-like distortion ("slippering") or "wisping" of the vasculature at the dorsal-distal tip of P3 (the dorsal and circumflex vessels) may be seen in horses with chronic laminitis (Figure 16-5).

Despite these vascular changes, the relationship between P3 and the circumflex circulation is normal, or nearly so (i.e., the distal margin of P3 is located within the circumflex vasculature, not dropped below it, as occurs with more severe grades).

Expected Outcome

Digital realignment and elevation of the heels usually returns the horse to its former level of activity within a few weeks or months. Use of an air ride or platano shoe may work as well as heel wedges once the horse is ready for and can tolerate shoeing.

Grade II

Clinical Findings

The horse walks forward slowly but is reluctant to make tight turns or tight figure-eight patterns. There is no external pathologic hoof lesion at the beginning of disease process. Later, a demarcating ring becomes visible as it grows down from the coronary band; this ring often is deeper and larger than that seen in horses with grade I laminitis. Bruising of the solar corium in front of the frog is evident after a few trimmings of the hoof (Figure 16-6).

Radiographic Findings

Plain Films. Typical abnormalities found on plain soft-tissue films are summarized in Table 16-2 and illustrated in Figure 16-7.

Table 16-2 Grading the Laminitic Horse: Radiographic Criteria*

Radiographic Indices	Grade I	Grade II	Grade III	Grade IV
Positive palmar angle	5-9 degrees	10-14 degrees	≥15 degrees	*Cranial rotation:* >15 degrees, often 20-30 degrees *FSS:* may be 0 degrees or negative (see Figure 16-13)
H-L zone width	Approximately 16/20 mm	Approximately 20/25 mm	Approximately 25/30 mm	*Cranial rotation:* approximately 25/30 mm *FSS:* proximal and distal measurements are the same and are ≥30/30 mm
EP-CB distance	Within normal limits	May be increased slightly (e.g., to 15-16 mm) as dorsal surface of EP tips distally (NOTE: this does not indicate sinking)	Increased	*Cranial rotation:* increased *FSS:* increased, but must be interpreted with H-L zone width (≥30/30) and decreased SDT/SDW
SDT/SDW	Both within a few mm of normal	SDT decreased (may be 10-12 mm); SDW increased	SDT greatly decreased; SDW greatly increased	*Cranial rotation:* as for grade III; SDT may be ≤0 if P3 has penetrated the sole *FSS:* both values greatly decreased equally
Soft tissue changes	Limited to a subtle lucent line dorsal to the dorsal surface of P3	May have wider lucent line than grade I; lucent area may also be present distally, under tip of P3	Disorganization of dorsal laminae and solar corium with increased lucency in both areas	*Cranial rotation:* marked lucency along dorsal face of P3 (wider distally); sole may be open and P3 exposed *FSS:* see text

CP, Coronary band; *EP,* extensor process; *FSS,* fatal sinker syndrome; *H-L,* horn-lamellar; *P3,* third phalanx; *SDT,* sole depth at tip of P3; *SDW,* sole depth at wings of P3.
*Using indices readily measured on lateral soft-tissue detail plain films of the foot.

Table 16-3 Grading the Laminitic Horse: Venographic Criteria*

Vascular Region†	Grade I	Grade II	Grade III	Grade IV
Coronal cascade	NSA	Constriction by the tipped extensor process of P3	Marked congestion just proximal to extensor process of P3	*Cranial rotation:* marked congestion *FSS:* marked compression
Dorsal lamellar and circumflex vasculature	*Acute:* NSA *Chronic:* minor slippering *Acute-chronic:* wisping (see text)	*Acute:* dorsal circulation may be decreased; circumflex vessels at tip of P3 may be cavitated (see text) *Chronic:* wisping of dorsal circumflex vessels	Dorsal circulation decreased, fimbriae and papillae shortened or absent; constriction of circumflex vessels at tip of P3	*Cranial rotation:* decreased or absent circulation in dorsal lamellae and circumflex vessels at tip of P3 *FSS:* filling in both areas markedly decreased or absent
Terminal arch	NSA	NSA	NSA	*FSS:* may be markedly decreased (even absent in severe cases)
Bulbar circulation	NSA	NSA	NSA	*FSS:* either congested or absent
Position of P3 relative to circumflex vessels	Normal	Normal	Distal margin of P3 partially or entirely below vessels	Distal margin of P3 below vessels

FSS, Fatal sinker syndrome; *NSA,* no significant abnormalities; *P3,* third phalanx.
*Presence and severity of filling deficits in the digital circulation.
†See Chapter 18 for further explanation.

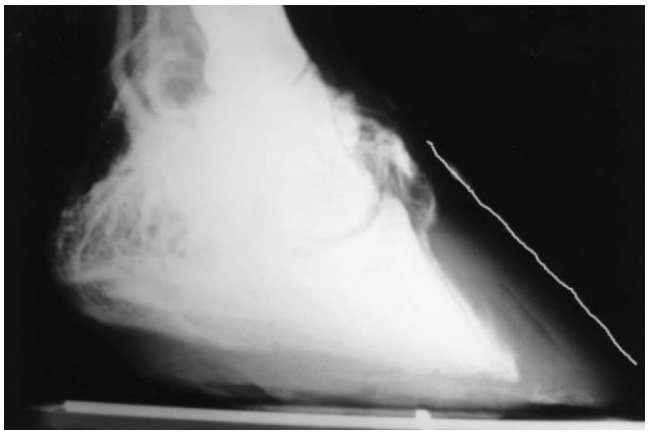

FIGURE 16-5 Venogram of a grade I laminitic event.

FIGURE 16-6 Solar hematoma in grade II laminitic event.

Venogram. There is constriction of the coronal cascade from pressure by the extensor process as it tips forward. In addition, the dorsal circulation may be decreased, and the circumflex vasculature at the dorsal-distal tip of P3 may show cavitations from blood pooling, seroma, or hematoma formation. The distal margin of P3 remains within the boundaries of the circumflex circulation. Wisping of the dorsal circumflex circulation may be evident if the case is chronic (Figure 16-8).

Expected Outcome

Return to the former level of athletic performance is possible with diligent care (repeated digital realignment and heel elevation or rocker shoeing for at least 6 months).

Grade III

Clinical Findings

These horses are obviously in pain. They walk forward reluctantly and refuse to turn, and they often spend a good deal of time recumbent. Despite the severity of clinical signs, there is no external pathologic lesion at the onset of the disease process.

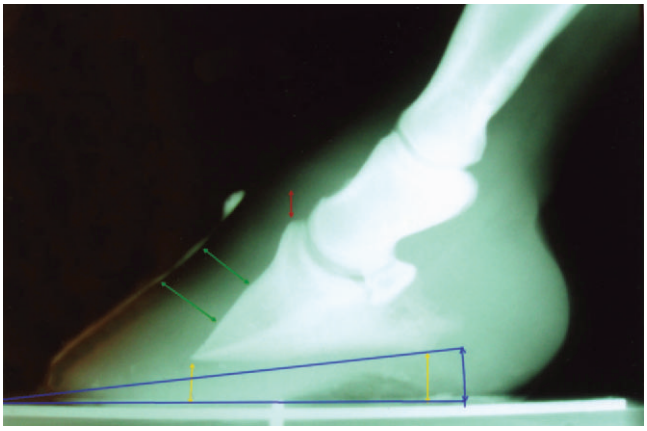

FIGURE 16-7 Radiograph of a grade II laminitic event.

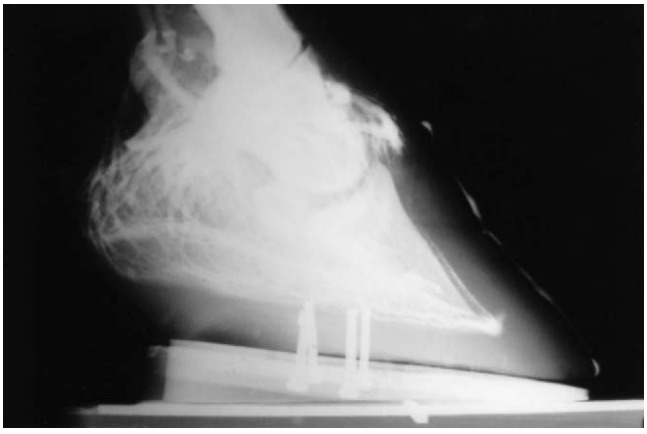

FIGURE 16-8 Venogram of a grade II laminitic event.

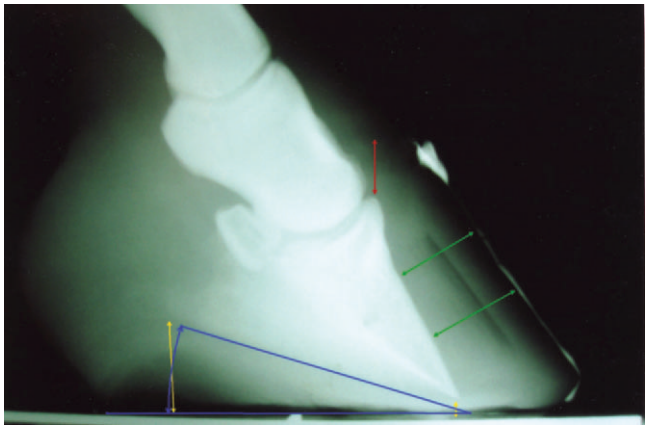

FIGURE 16-9 Radiograph of a grade III laminitic event.

Radiographic Findings

Plain Films. Typical abnormalities found on plain soft-tissue films are summarized in Table 16-2 and illustrated in Figure 16-9.

Venogram. There is marked congestion of the coronal cascade just proximal to the extensor process and constriction of the circumflex vessels at the dorsal-distal tip of P3. In addition, the dorsal circulation is decreased, and the fimbriae and papillae are absent or decreased in length. The distal

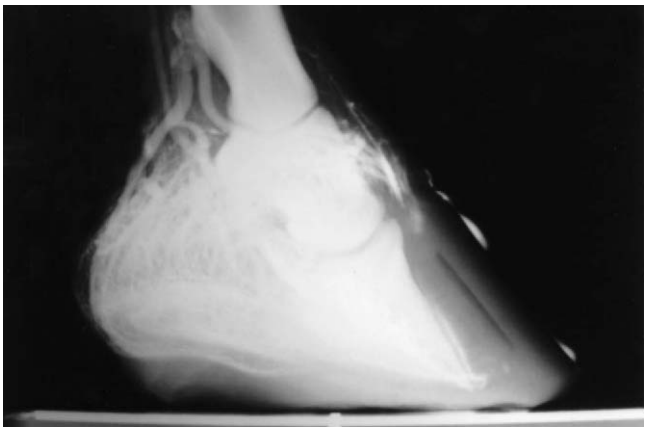

FIGURE 16-10 Venogram of a grade III laminitic event.

margin of P3 may be partially or entirely below the circumflex circulation (Figure 16-10).

Expected Outcome

It is important to understand that horses with grade III laminitis usually progress to grade IV unless rigorous intervention is undertaken. Deep digital flexor (DDF) tenotomy and digital realignment are recommended for horses with grade III laminitis. These horses may still return to their former level of athletic performance within 2 years of the laminitic episode, depending on the horse's age, the chronicity of the disease process, and whether arthritic changes or other unrelated but limiting factors are present.

Grade IV

Clinical Findings

Horses with grade IV laminitis are in extreme pain. They are recumbent for most of the time and stand only with great difficulty. Walking is nearly impossible for these horses, and many walk primarily on their hindlegs (unless the disease process is restricted to the hindfeet).

It is important to differentiate between horses with cranial rotation of P3 (i.e., a positive PA), and those with distal displacement of P3 (i.e., sinkers), because fatal sinker syndrome (FSS) manifests somewhat differently and has a poorer prognosis.

Cranial Rotation. In grade IV cases with cranial rotation, external pathologic findings often include rupture of the coronary band (which typically occurs dorsally or medially) (Figure 16-11) and penetration of the sole by the distal margin of P3 and its digital corium (Figure 16-12), with exposure of the solar corium at that location. Cranial rotation or increased positive palmar angle is an important component of all grades of laminitis.

NOTE: Cranial rotation should be differentiated from caudal rotation or a negative palmar angle. Horses with a history of caudal rotation will show a normal (e.g., 19/19) or slightly reversed H-L zone width (e.g., 19/18) during the worst phases of laminitis, and this finding can be confusing to the diagnostician.

Fatal Sinker Syndrome. Cases of FSS may or may not show external pathologic changes at onset. Quittor-type lesions (drainage from the lateral cartilages proximal to the

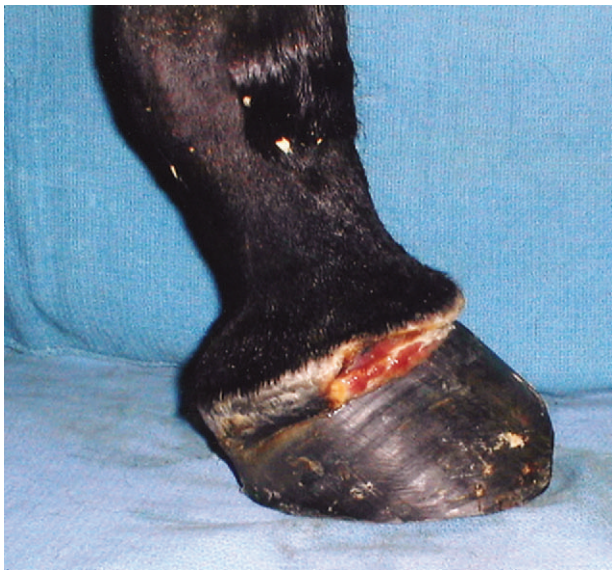

FIGURE 16-11 Example of a common coronary band rupture seen in grade IV laminitic events.

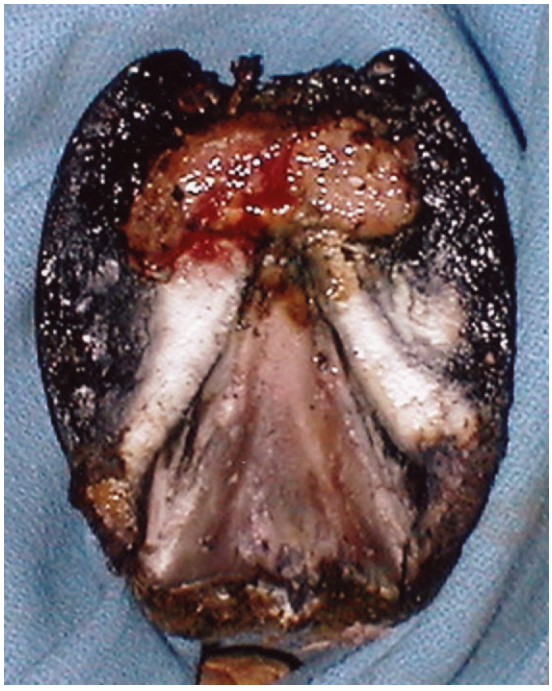

FIGURE 16-12 Solar prolapse with penetrated digital corium in a grade IV cranial rotation.

medial and lateral coronary band) and rupture at the heel bulbs and frog (Figure 16-13) are common as pressure is put on bulbar circulation by the second (P2) and third (P3) phalanges sliding backward and down within the hoof capsule. The sole may be flat and friable, and the entire hoof capsule often is weak and easily moveable, separate from the digit. In advanced cases of FSS, swelling of the coronary band proximal to the fetlock is an indicator of circulatory loss to the hoof (Figure 16-14) and carries a poor prognosis.

NOTE: A depression at the coronary band is not a good diagnostic or prognostic indicator, unless it is present around

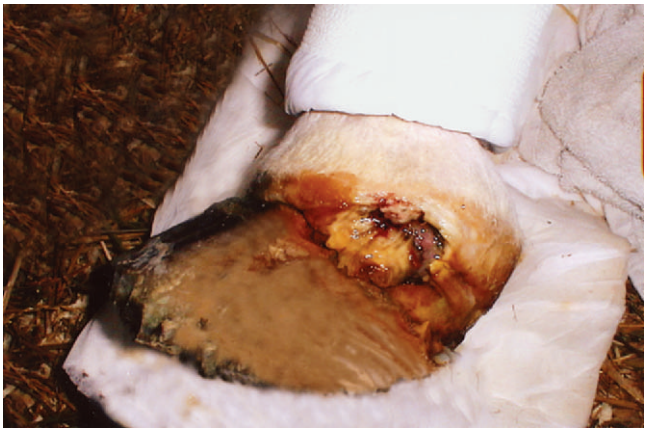

FIGURE 16-13 Quittor-like lesions common in fatal sinker syndrome from pressure applied to bulbar circulation by P3 moving down and back into the hoof capsule.

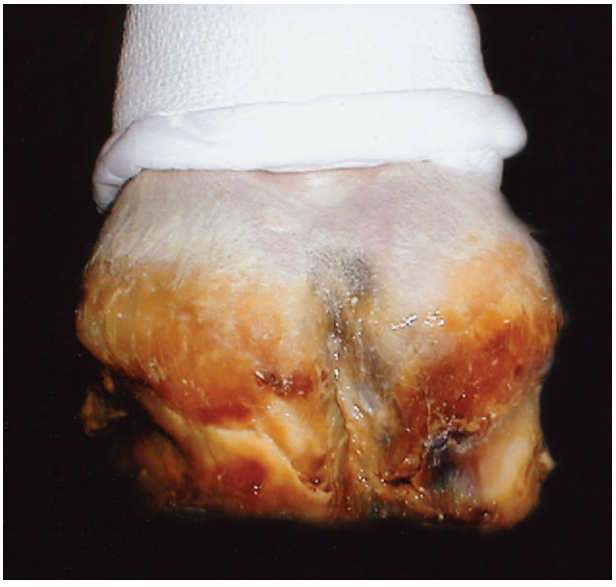

FIGURE 16-14 Congestion of circulation in the pastern and heel bulb region common in cases of fatal sinker syndrome. The swelling, if left unchecked, will continue proximally up the leg.

the entire circumference of the hoof. In horses with severe, acute FSS, there may also be hemorrhage from the coronary band (rupture of the coronal vessels).

Radiographic Findings

Plain Films. Typical abnormalities found on plain soft-tissue films are summarized in Table 16-2 and illustrated in Figures 16-15 and 16-16. One of the radiographic hallmarks of FSS is that both the SDT and the SDW are greatly decreased, more or less equally so, as the distal phalanges move down and back within the hoof capsule. Despite this marked displacement of P3, however, soft-tissue changes may be minimal or limited to just a very wide lucency dorsal to the face of P3; this area (the H-L zone) may be equal in width from proximal to distal. NOTE: There may be cranial rotation prior to the onset of FSS.

Venogram. Abnormalities in horses with dorsal rotation include marked congestion of the coronal cascade and

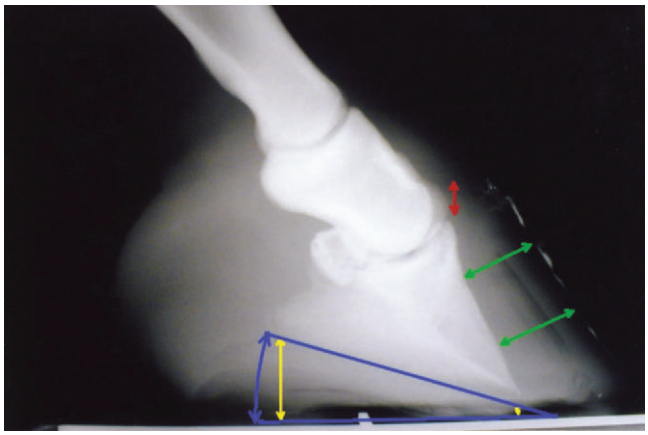

FIGURE 16-15 Radiograph of grade IV cranial rotation laminitic event. Note the solar prolapse.

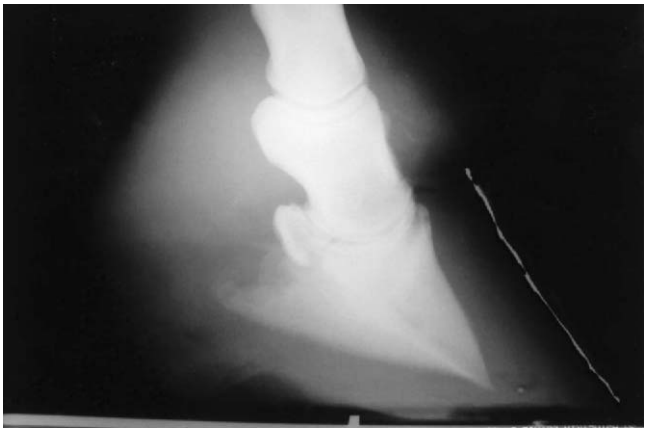

FIGURE 16-16 Radiograph of a case of fatal sinker syndrome. Note the congestion and swelling of tissues proximal to the coronary band.

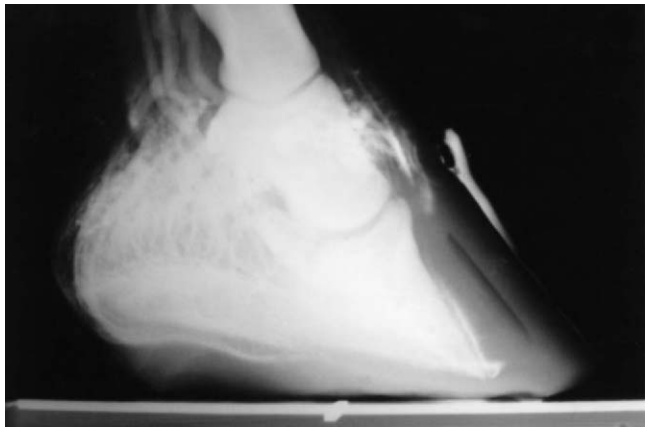

FIGURE 16-17 Venogram of a cranial rotation grade IV laminitic event.

decreased or absent circulation in the dorsal lamellae and circumflex vessels at the dorsal-distal tip of P3. The distal margin of P3 lies below the circumflex circulation (Figure 16-17).

In horses with FSS, there is marked compression of the coronal cascade and markedly decreased or absent dorsal and circumflex circulation; circulation in the terminal arch may also

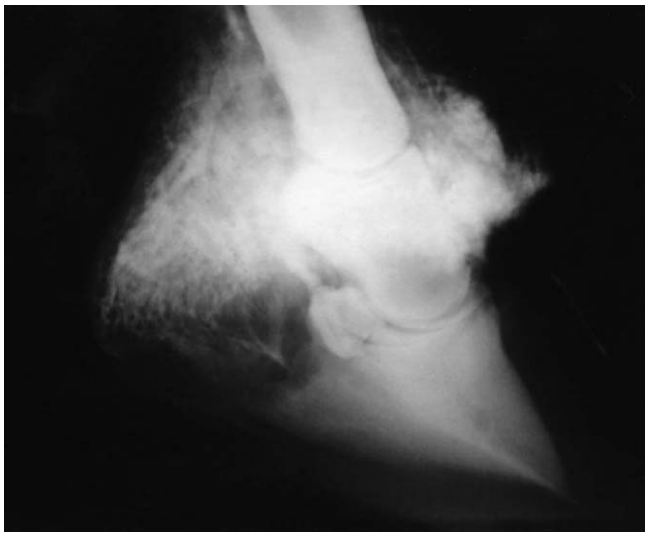

FIGURE 16-18 Venogram of a case of fatal sinker syndrome.

be markedly decreased. The bulbar circulation is either congested or absent. In severe cases, there is a complete absence of contrast material below the coronary band (Figure 16-18).

Expected Outcome

Cranial Rotation. Most horses with cranial rotation do well with DDF tenotomy, digital realignment, and meticulous care during the long recovery period (see Chapter 19). It is possible for these horses to return to athletic function, provided they have no other abnormalities (e.g., arthritic conditions) that would prevent them from doing so.

Return to athletic performance is totally dependent on two factors: (1) how quickly appropriate treatment is instituted, and (2) the individual horse's ability to heal. Many older horses survive to become pasture sound or able to perform, but at a lower level than before the laminitic episode. Nevertheless, most horses with grade IV laminitis are euthanized once external pathologic lesions are seen, which is unacceptable to this author.

Fatal Sinker Syndrome. For a successful outcome, horses with FSS require transcortical pinning and casting plus hoof wall ablation (Figures 16-19 and 16-20). Depending on the condition of the pins, the cast is removed and the pins are taken out 3 to 11 weeks after surgery. The limb is then recast (without pins) for another 3 to 6 weeks.

This procedure is still in the research phase, and thus far return to use in surviving horses has been limited to breeding, not athletic performance. Transcortical pinning should not be attempted on horses with soft cannon bones (e.g., from lengthy recumbency) or with a history of cannon bone fracture, because fracture at the level of the pins is a very real threat during the recovery period. When transcortical pinning and hoof wall ablation is not an option, DDF tenotomy and digital manipulation using glue-on shoes may yield acceptable results.

FIGURE 16-19 Example of transcortical pinning for fatal sinker syndrome.

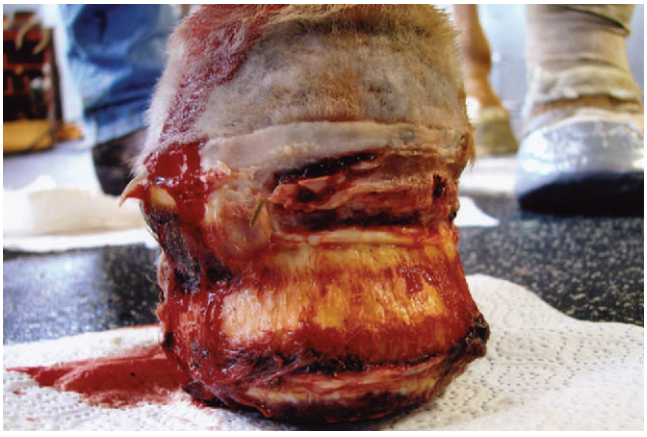

FIGURE 16-20 Hoof wall ablation for grade IV fatal sinker syndrome.

Regardless of the procedure used, horses with FSS are at high risk and require high maintenance. They require intensive medical and nursing care, and residual pathologic lesions and lameness are common. These cases have a long-term recumbency pattern, so meticulous nursing care is critical to success (see Chapter 19).

17
THE DIGITAL VENOGRAM

AMY RUCKER

Laminitis is defined as the failure of the attachment between the distal phalanx (coffin bone or third phalanx [P3]) and the inner hoof wall.[1] Assessment of the laminitic horse is based on the history of lameness in combination with physical evaluation and radiographic examination.[2,3] Prognosis for recovery is determined by historical, physical, and radiographic evaluations as well as response to therapy and findings by newer techniques such as dorsal lamellar biopsy.[4-6] Unfortunately, these traditional methods of evaluating laminitis have limitations and debatable significance. For example, veterinarians disagree about the significance of the degree of lameness, types of radiographic changes, and amount of coffin bone rotation as indicators of the disease severity and prognosis.[2-9] Furthermore, many times the severely lame, acutely laminitic horse presents with no radiographic evidence of displacement of the coffin bone. Horses with no or relatively minor degrees of displacement may have devastating changes in the vascular supply to the foot. The venogram helps the veterinarian decide how aggressively to treat the laminitis, or if the prognosis is grave and euthanasia should be recommended.

The digital venogram is a diagnostic procedure using contrast-enhanced radiography to assess the degree of vascular compromise in the laminitic horse. Contrast medium is injected into the digital veins, resulting in retrograde filling of the arteries. Areas of abnormal or reduced perfusion indicate pathologic changes within the soft tissues of the foot. The technique for performing venography in a standing horse was developed by Pollitt and Redden in the early 1990s and has subsequently been refined and published.[10-12]

The venogram is both sensitive and repeatable when evaluating an individual horse's foot vasculature. In a controlled study of six horses without foot pathologic lesions, bilateral forelimb venograms were used to demonstrate normal perfusion patterns as well as observe changes induced by shoeing with a 5-degree wedged-heel egg-bar shoe. The horse's feet were prepared for shoeing and venograms were obtained at day 0. Subsequently, five of the horses were shod with the egg-bar shoes and all horses were returned to pasture. At day 14, venograms were obtained again and the shoes were pulled post-venogram. Final venograms were taken on day 28. The appearance of each foot's vasculature was repeatable; there was no significant difference in perfusion parameters between day 0, 14, and 28.[12] Furthermore, this study was performed over a period of 3 months during temperature ranges from 15.5° C (60° F) to below freezing, but fluctuation in ambient temperatures did not appear to affect the venogram.

In the pathologic foot, venograms allow evaluation of contrast distribution and identification of areas of reduced perfusion. In a study comparing vascular perfusion casts of normal versus chronic laminitic feet, it was demonstrated that defects in submural laminar circulation, coronary perfusion, and solar circulation were worse in horses with clinically refractive laminitis.[14] These areas can be evaluated in vivo by the digital venogram, because perfusion deficits are apparent in the laminitic horse but absent in horses without laminitis. [15] Venographic images of horses with no evidence of laminitis and horses with clinical and radiographic evidence of chronic laminitis were compared in a blinded independent evaluator study. Evaluators graded five areas of perfusion within the feet and concluded that there was a difference between the images from the normal and the chronically laminitic horses. Furthermore, the coronary plexus, dorsal lamellae, and circumflex vascular areas were consistently normal in the nonlaminitic horses but abnormal within the chronic laminitic feet.[15] Finally, a study was done monitoring venographic changes of horses as they progressed through the acute and into the chronic phase of laminitis.[16] Venograms were obtained on 10 clinically normal horses and then at 48 hours, 7 days, and between 4 and 12 weeks after induction of laminitis. These evaluators did not detect venographic changes during the acute phase (48 hours) but did note changes within sublamellar and circumflex vascular beds at day 7 and at the final venogram; these changes correlated with clinical lameness and displacement of P3.[16]

PREPARATION FOR THE VENOGRAM

Preparing for the venogram includes identifying a safe, quiet area with a flat surface. It is essential that the examiner obtain quality radiographs and consistently use the same techniques, so that films can be compared.[17] Variations in technique can create artifacts, and incorrect interpretations may lead to erroneous conclusions. A standardized routine is necessary to gain the most information from the procedure.

Two 12-mL Luer lock syringes are filled with diatrizoate sodium (Renografin 60; ER Squibb & Sons, Princeton, NJ; or Hypaque-76; Nycomed, Princeton, NJ) contrast medium. A foot that is 10 cm wide will fill with 20 mL of contrast material; a 14-cm foot requires 24 mL. A 21-gauge, 3/4-inch (1.9 cm) butterfly catheter with 30.5-cm tubing (Abbott Laboratories, Abbott Park, Ill.), 10.2 cm and 5.1 cm tape (Elastikon; Johnson & Johnson, New Brunswick, NJ), gauze squares, inner-tube tourniquet, and a pair of mosquito hemostats are needed for the venogram. Radiographic equipment includes a radiography machine, a minimum of five cassettes, a grid, barium paste for the dorsal hoof wall marker, alcohol-saturated gauze to remove the barium from the hoof wall, protective aprons and gloves, and wooden radiograph positioning blocks (Wooden Positioning Block; Nanric, Versailles, Ky.).

Each person helping with the venogram should clearly understand his or her contributions before the procedure is attempted. The team should include a horse holder who observes the behavior of the horse, not the venogram

procedure. This person warns if the horse is going to move, keeps the horse standing in place on the radiograph blocks during the venogram, and does not allow the head to drift to either side, which would cause artifacts in the venogram from uneven loading of the feet. The holder times the venogram—after injection of the contrast material, all radiographs should be taken within 30 to 45 seconds. If the procedure takes longer, then the time is recorded and this is considered when interpreting the radiographs. The veterinarian injects the contrast material, takes radiographs, and manages radiographic technique settings. The assistant hands syringes of contrast medium as needed, places cassettes in the correct position, and removes barium from the dorsal hoof wall while the veterinarian changes the machine from lateral to dorsal position.

The horse is tranquilized with detomidine hydrochloride (0.002 to 0.01 mg/kg IV; Dormosedan, Pfizer Animal Health, Exton, Pa.). The horse must be quiet but not overly sedated, so that it will stand still without wobbling. The foot is meticulously prepared for baseline radiographs (0-degree dorsopalmar and lateral), which assess the radiographic technique needed for the venogram. The beam is centered at the level of the solar surface of the coffin bone. Lateral radiographs are taken with a zero film-to-subject distance for elimination of magnification. This ensures accurate measurements of the osseous, soft tissue, and vascular structures. On initial presentation, radiographs are taken with the horse in the presenting shoes or barefoot. If the horse has acute laminitis, or if it has been in a therapeutic shoe that elevates the palmar angle of P3, once the presenting shoe is removed the foot is placed in a plastic elevated heel shoe for the procedure.

Local anesthesia of the medial and lateral palmar digital nerves at the level of the proximal sesamoid is achieved using 1 to 3 mL of mepivicaine 2% (Polocaine; Astra Zeneca, Wilmington, Del.) per site. (An excess of mepivicaine 2% must not be used, as it may make the application of a tight tourniquet difficult.) Both front feet are blocked to ensure that the horse will stand squarely during the procedure. The medial and lateral pasterns in the area of the palmar digital vein are clipped and aseptically prepared.

OBTAINING THE VENOGRAM

Radiograph blocks are positioned in the normal stance of the horse, and the horse stands on them. The cannon bones should be vertical to the ground. The medial edge of the foot wall should be at the medial edge of the radiographic foot block (Wooden Positioning Block). Sedation is assessed before the procedure begins.

Barium is applied to the dorsal wall, and alcohol preparations are placed near the foot. The distance for lateral and dorsopalmar views is marked on floor, and the machine is positioned for lateral radiographs.

Unexposed cassettes are located next to the assistant for immediate access. All marker windows are in the same location. The grid is in place with the second and third cassette. The veterinarian should be comfortable with the technique chart used for the radiographs, and the assistant should understand which views will require the grid.

The veterinarian kneels dorsolateral to the foot. The machine is lateral to the foot, and the assistant is palmarolateral. A length of 9.2 cm tape (Elastikon), is wrapped around the fetlock. If the veterinarian is right-handed, the tape is wrapped in a clockwise direction when viewed from the front. (This allows the "stronger" right hand to tightly pull the tourniquet from medial to lateral across the palmar surface of the distal fetlock.) The tape does not come down onto the pastern. Care must be taken to not distort the skin or underlying tissue. The tape is cut on the lateral aspect of the leg. One end of the inner-tube tourniquet is taped beneath the tag of tape. After the tourniquet is tightly applied, it is secured with several wraps of 5.1 cm tape (Elastikon). On the dorsolateral aspect, a 3-inch tag end is left loose.

The butterfly catheter cap and needle cover are removed. The veterinarian's inside arm wraps around the leg in a dorsomedial to palmar direction, and the veterinarian's inside shoulder applies gentle pressure to the horse's dorsal forearm and carpus. This will encourage the horse to stand quietly but also immediately alert the veterinarian if the horse unloads the foot before it picks up the leg. The catheter is inserted into the lateral palmar digital vein at midpastern. When blood flows freely from the vein, the assistant lightly attaches a syringe filled with contrast medium. The contrast material should be injected in a minimal amount of time. The veterinarian injects the contrast material with the medial hand, while the lateral hand lightly applies digital pressure to the distal vein and needle. Pressure is necessary to prevent the vein from blowing (perivascular hemorrhage and vein clotting caused by needle or catheter trauma) as the valves are forced open.

The assistant removes the empty first syringe and lightly attaches the second syringe. He or she then grabs the hemostats with the tips of the fingers or palm and readies the first cassette. As the second syringe of contrast material is injected, the veterinarian uses his or her medial arm to shift the weight of the horse off and then back onto the foot, slightly flexing the limb and allowing relaxation of the deep digital flexor tendon.

When the veterinarian is finished injecting the second syringe, the assistant immediately clamps hemostats onto the tubing. The veterinarian removes the syringe and tapes the catheter and hemostat under the tag of tape, which is proximal to the radiographic field.

All radiographs should be taken within 30 seconds. The veterinarian exposes a lateral view film, and then adjusts the technique and takes a lateral grid shot. The assistant removes the barium from the dorsal hoof wall while the veterinarian moves the machine to the 0-degree dorsopalmar position. A dorsopalmar view with a grid is followed by a dorsopalmar view. A final lateral view is taken to evaluate any areas that are perfused late in the venogram.

The tourniquet and then catheter are removed. The palmar digital veins are covered with dry gauze in a light pressure bandage. The bandage is removed 15 minutes after application.

TROUBLESHOOTING THE VENOGRAM

If the horse is allowed to move its head, it will probably step off both blocks. If the horse steps off the block with the off leg, the holder must continue to hold the horse's head in position. The assistant should quickly replace the off foot onto the block. The veterinarian is responsible for control of the venogram foot. He or she should be able to anticipate the horse's

movement through its carpus, which is in contact with the veterinarian's shoulder. The arm of the veterinarian that is wrapped around the medial aspect of the horse's leg will control the leg's movement and replace the foot onto the block without interrupting the procedure. When the veterinarian rocks the knee while injecting the second syringe of contrast, he or she must be careful to unload the leg without signaling the horse to pick up the foot.

Catheterization may be difficult. The vein must be identified before the tourniquet is applied. Occasionally the vein is not apparent with the tourniquet in place and catheterization before applying the tourniquet or catheterization of the medial vein may be necessary. If the vein is not prominent on the pastern due to distal limb edema, catheterization of the vein must occur at the level of the sesamoids. The tourniquet is applied at the most proximal level of the sesamoids and the vein is catheterized just distal to the sesamoids.

The skin thickness should be considered: legs with distal limb edema or the legs of thick-skinned draft horses are more difficult to catheterize and require needle insertion at a greater angle. Older horses have tortuous, fragile veins that require finesse.

During catheterization, if blood starts to flow from the syringe and then stops, acquisition of the venogram must not be attempted. Instead, the needle should be backed out 1 to 2 mm, in case it is resting against the opposite wall of the vein. The needle may need to be redirected, but subsequent venipunctures will leak contrast material into the subcutaneous tissue and should be minimized. Perivascular contrast is signaled by coolness of the skin and distension of the skin perivascularly. If needle placement is questionable, pressure on the syringe plunger should be released; if blood flows retrograde into the catheter tubing, the needle is in the vein. If the vein is blown, radiographs should be taken immediately without further injection. (The venogram will not be ideal, because of inadequate volume, but it will give some information.)

When injecting the contrast media, 12-mL syringes are used because the injection resistance associated with perivascular injection is not detectable with a larger syringe. Care should be taken to keep the needle in the vein. The veterinarian should avoid stretching the catheter tubing, especially when changing syringes and clamping the catheter. If the catheter comes out and the vein starts to blow, the films should be taken quickly, before contrast volume is lost from the foot. And finally, if the horse is not totally weight bearing on the block after the injection is complete, the radiographs should be taken anyway, and a note made in the records so that the lack of load is considered when the venogram is interpreted.

Five radiograph films are suggested. Two "lighter" (lower KVp) images best visualize the soft tissue detail, whereas films taken at higher kilovolt peak or milliampere-seconds allow visualization of the denser structures, such as the terminal arch. If computed or digital systems are used, some systems require only two images to be taken (one lateral and one dorsopalmar view), whereas others necessitate the standard five view series to obtain optimal detail. Digital and computed radiography systems vary in their resolution, both within the processing of the images obtained and also between computer monitors. The solar papillae of the circumflex vasculature are at risk of not being observable on these systems. The examiner should make certain that before interpreting a venogram on a pathologic foot that he or she reviews venographic images made on healthy feet with thick soles and observes distinct solar papillae on the images.

Anatomy of the Venogram

The bone and soft tissue of the foot are supplied by the palmar digital arteries and their numerous branches (Figure 17-1).[18-21] The vascular anatomy is important because areas of the foot are perfused by more than one source. The arteries of the foot form anastomoses; they do not perfuse an area and terminate. If one area of vasculature is compromised, the affected tissue will also be compromised but is not necessarily ischemic. For example, the coronary plexus (supplied by the dorsal branch of the second phalanx [P2] and the coronary artery), terminal arch, and circumflex vessels supply the dorsal lamellae. Damage to or compression of any one of these areas will reduce, but may not eliminate, perfusion of the laminae. The heel area is seldom affected in mild cases due to its many sources of blood. If all vessels within the hoof are compromised, the bulbar artery, which originates at the level of the proximal and middle phalanges, may still perfuse the palmar coronary corium, digital cushion, cuneate corium, and lamellar corium of the heel and bars.

The vessels of the foot are evaluated using five areas of focus (Figures 17-1 to 17-4). (NOTE: Measurements are typical of a light-breed horse such as a Quarterhorse or Thoroughbred.) In the pastern, the palmar digital vessels are uniform in diameter, and smooth-walled veins are distinguished from the muscular walled arteries. The terminal arch (see *A* in Figures 17-1 to 17-4) of the palmar digital arteries and veins is apparent within the center of P3.

Branches from the terminal arch to the dorsal lamellae and circumflex vessels are evident but difficult to distinguish individually. The dorsal lamellar vessels (see *B* in Figures 17-1 to 17-4) form anastomoses with the coronary plexus on the dorsal aspect of the extensor process of P3. Proximally, the dorsal lamellar vessels are within 3 to 4 mm of the dorsal border of P3. At the tip of P3, the vessels extend distally and dorsally for 4 to 6 mm, where they form anastomoses with the circumflex vessels. Dermal papillae are evident as they supply the toe and sole.

The coronary plexus (see *C* in Figures 17-1 to 17-4) is located proximal and dorsal to the extensor process of P3. The size and location of the coronary plexus depends on the conformation of the foot and the vertical distance separating the extensor process from the coronet. The dorsal branch of the middle phalanx is evident at the proximal half of P2. At a distance of 5 to 10 mm distally, the coronal artery and vein are evident dorsal to the common digital extensor tendon and the extensor process. The coronal vessels appear to course around the pulvinus coronae, creating a defect in the coronary crescent, or a "waterfall" appearance.[22] The vessels on the dorsal aspect of the pulvinus coronae supply dermal papillae in the coronet, and then join the dorsal lamellar vessels.

On the palmar aspect of P3, the circumflex vessels (see Figures 17-1 to 17-4, *D*) extend from the anastomoses, with the dorsal lamellar vessels at the apex of P3 to the heel. The circumflex vessels are 3 to 5 mm distal to the palmar aspect of P3, and dermal papillae may extend 5 mm distally if the horse has a thick sole. The dermal papillae are individual arteriovenous anastomoses that supply the tubules of the sole corium. A horse with a thin sole (≤10 mm) seldom has papil-

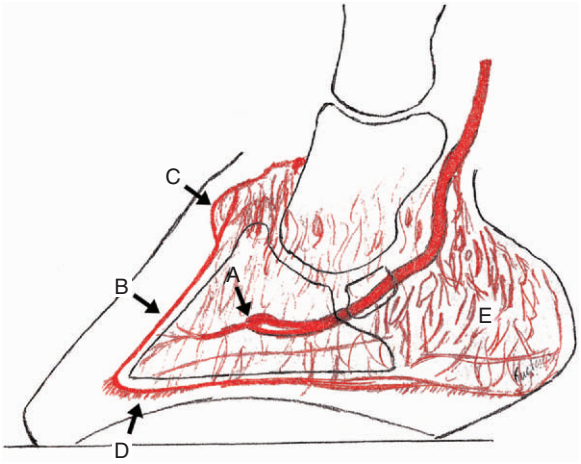

FIGURE 17-1 Diagram of vasculature, lateral view. *A,* Terminal arch; *B,* dorsal lamellar vessels; *C,* coronary plexus; *D,* circumflex vessels; *E,* heel perfusion.

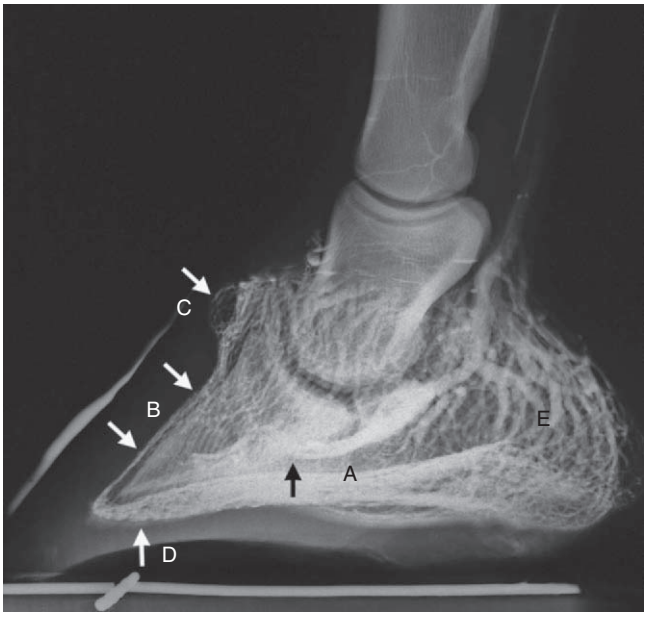

FIGURE 17-3 Lateral venogram of a normal foot. Note that the contrast material has distended both arteries and veins and that the radiograph is a two-dimensional representation; medial and lateral paired vessels overlap. *A,* Terminal arch; *B,* dorsal lamellar vessels; *C,* coronary plexus; *D,* circumflex vessels.

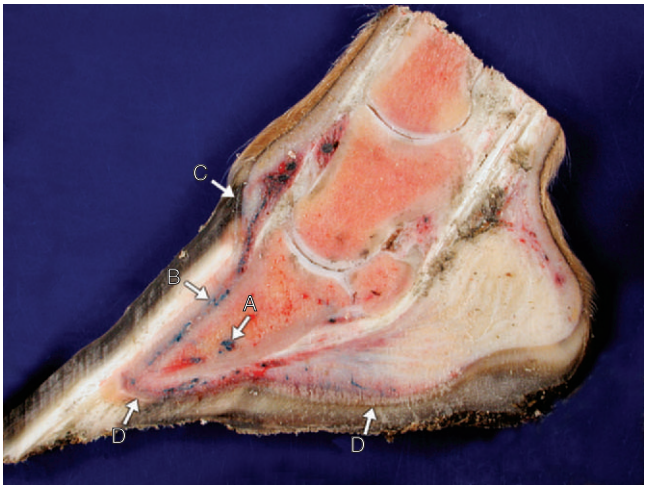

FIGURE 17-2 Median sagittal section of a fresh foot. Blue latex was injected in the palmar digital veins and has partially filled the vasculature. (NOTE: Only the vessels at the midline of the foot are evident.) *A,* Terminal arch; *B,* dorsal lamellar vessels; *C,* coronary plexus; *D,* circumflex vessels. *(Specimen prepared with assistance from Bobby Colley.)*

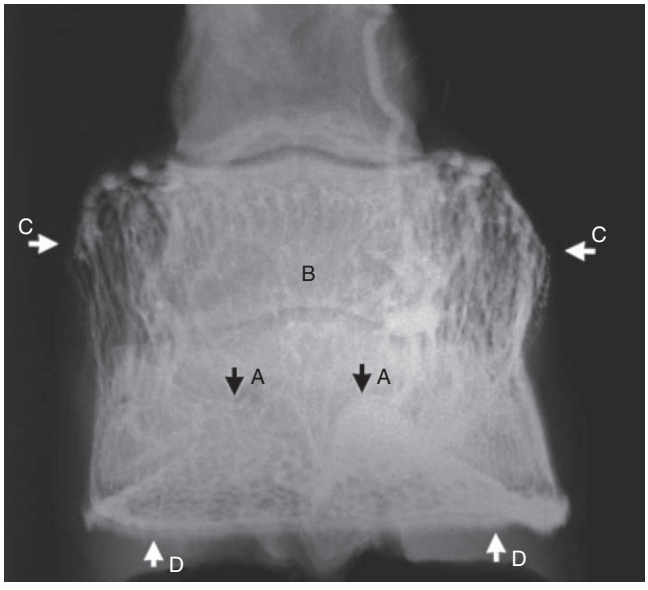

FIGURE 17-4 Zero-degree dorsopalmar venogram of a normal foot. *A,* Terminal arch; *B,* dorsal lamellar vessels; *C,* coronary plexus; *D,* circumflex vessels.

lae evident beneath the anterior half of P3. Most horses have papillae evident as they supply the frog and heel corium.

The heel perfusion (see Figure 17-1, *E*) is a complicated web comprising the bulbar, coronal, and dorsal branch of P3 arterial supply and the venous drainage. It is impossible to distinguish individual arteries and veins in this region of the venogram.

Zero-Degree Dorsopalmar Venogram

The dorsal and palmar branches of the middle phalanx and the terminal arch (see Figure 17-4, *A*) are readily apparent as the medial and lateral vessels form anastomoses. The circumflex vessels (see Figure 17-4, *D*) and solar papillae are also apparent distal and peripheral to the solar margin of P3. The circumflex vessels form anastomoses proximally with the lateral hoof wall lamellae and then the coronary plexus (see Figure 17-4, *C*). The medial and lateral coronets of a balanced foot without compression will appear similar but are seldom symmetrical. If conformation or positioning loads one side of the foot more than the other, the loaded coronary area will lack contrast. Typically the horse bears more load on the medial aspect of the foot, and that coronet has less contrast than the lateral.

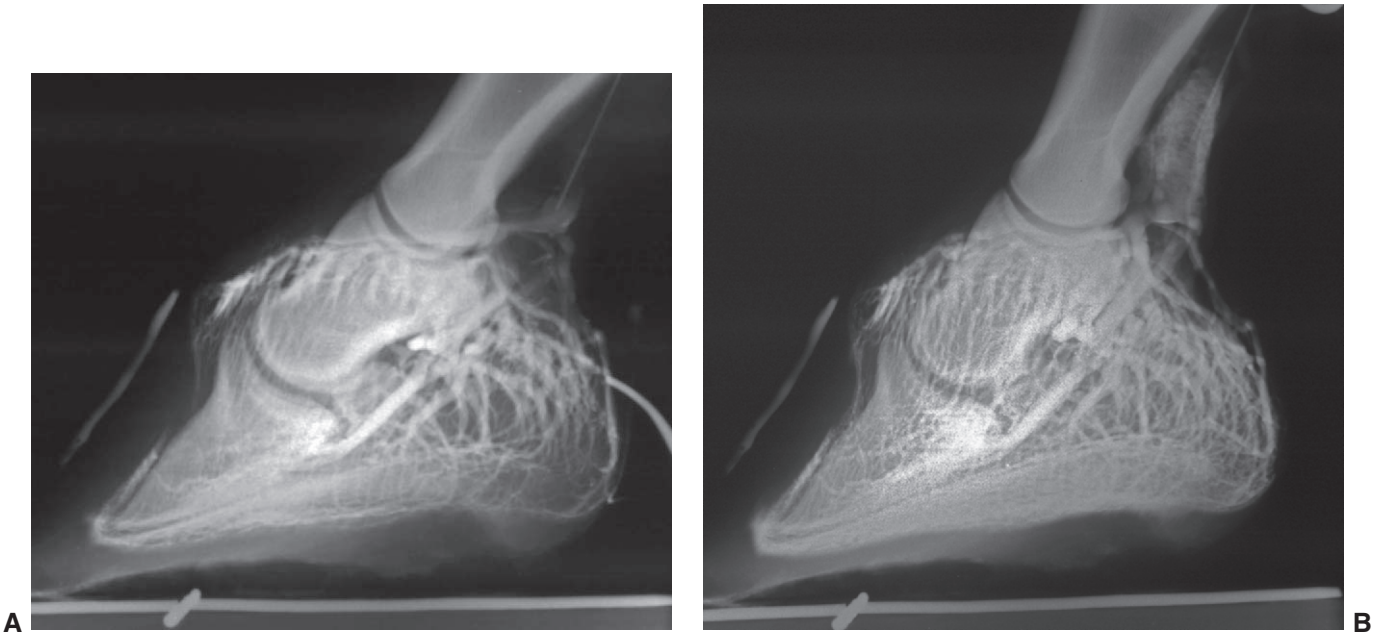

FIGURE 17-5 Venogram with inadequate contrast volume. **A,** This 5-inch–diameter foot was injected with 10 mL of contrast medium. **B,** The same foot after an additional 10 mL of contrast medium was injected.

Normal Variations

The anatomy of the vasculature of the foot does not vary between horses, but mild variations do occur between ages and breeds. Practitioners must establish what is normal for their unique clientele. For example, the 2- to 12-year-old American Thoroughbred has a dorsal hoof wall that is 16 mm when measured from the outer wall to the dorsal aspect of the coffin bone, and the dorsal lamellar vessels are located within 3 to 4 mm of P3. One normal-footed 6-year-old Percheron mare had a 25-mm dorsal wall and the lamellar vessels were within 5 mm of the dorsal aspect of P3. Furthermore, the heel vasculature of the draft foot is much denser than that of the low-heeled Thoroughbred.

Because heel perfusion arises from multiple sources that are palmar in origin, pathologic conditions of the foot seldom affect the appearance of the heel vasculature on venograms. On a normal foot, however, lateral radiographs of a heel loaded by multiple wedge pads will appear mildly compromised. Raising the heel 10 degrees or more reduces the vascular fill in the palmar heel vessels and compresses the heel, frog, and sole papillae from the widest point of the foot to the most palmar aspect. The appearance of the dorsal lamellae, coronary plexus, and terminal arch remain unchanged.

Artifacts

Perivascular contrast is the most common artifact. It may result from multiple venipunctures while catheterizing the vein, movement of the horse during the radiographic process, or the catheter coming out of the vein. A large pool of contrast is evident on both the lateral and dorsopalmar views at the level of the catheter in the palmar digital vein. The amount of leaked contrast material must be taken into consideration, as it may result in inadequate fill of the foot's vasculature.

Inadequate volume of contrast medium, which may be confused with poor perfusion, results from perivascular leakage, loosened catheter clamps, or incorrect calculations in the volume needed for the venogram (Figure 17-5). A characteristic narrowing of the blood vessels and overall lack of contrast indicates that volume, rather than perfusion, is the problem. Vessels have a "tree limb" appearance, tapering and decreasing in diameter in the distal aspect of the foot.

Tourniquet failure is the most common cause of inadequate volume. Normally compressed blood does not allow contrast near the tourniquet. If the tourniquet is loose, radiographs reveal contrast adjacent and proximal to the tourniquet.

Excessive time during contrast medium injection and film exposure results in contrast leaking into the corium and dermis (Figure 17-6). A fuzzy appearance at the margins of the circumflex vessels and dorsal laminae results if more than 30 to 45 seconds are taken for radiographs. This artifact can be confused with leakage into chronic scar tissue. If more than 45 seconds elapse before radiographs are taken, the entire venogram begins to appear fuzzy. It is imperative that each venogram be timed to distinguish between pathologic change and artifact.

A fully loaded foot with a long toe may not have contrast medium filling in the dorsal laminae if the leg is not unloaded after injection of contrast material. This artifact may be difficult to induce in the healthy foot but easy to demonstrate in the laminitic foot.

THE PATHOLOGIC FOOT

Reduction in vessel fill on venograms may result from various conditions, including shearing of vessels and tissues as P3 displaces, tissue edema, increased sympathetic tone, vascular spasm, and arterial embolization. Compression of vasculature

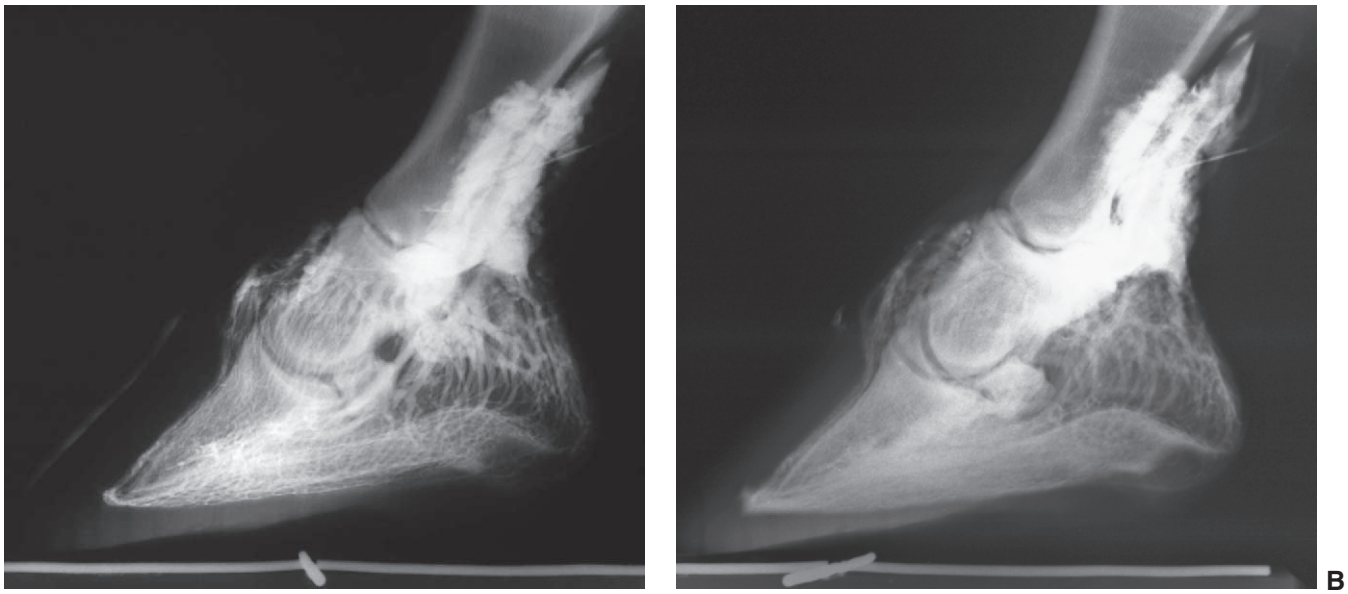

FIGURE 17-6 **A,** Lateral venogram taken within 30 seconds of injecting the contrast medium. The vasculature has crisp margins. **B,** Venogram taken 120 seconds after injection. Note the overall haziness as contrast material leaks into the tissues, including the perivascular contrast at the catheter site.

by loading forces on unstable tissue within the hoof capsule appears to be the most significant cause, because more significant deficits will be seen in a mechanically unstable foot under weight-bearing conditions than when the image is obtained when the foot is not bearing weight.[16] Weight-bearing images are the most accurate representation of digital perfusion, because most horses are standing and bearing weight for a significant part of their day. Furthermore, the venogram is a blueprint for therapy: treatment is directed to reduce compression or forces within the contrast-deficit areas while loading the perfused structures of the foot.

Terminal Arch

Before entering the coffin bone, the palmar digital arteries and veins course along the palmar aspect of the phalanges. Within the coffin bone, the terminal arch and its small branches are protected by a boney canal (Figure 17-7). These factors provide protection to the main blood supply of the foot. As P3 displaces, most of the damaged structures are in the anterior portion of the foot, leaving these palmar vessels undisturbed.

The grid radiographs are typically the best films for viewing the terminal arch within P3 because of the amount of radiation needed to penetrate the bone and the overlying contrast within the vasculature at the widest point of the foot. With displacement of the coffin bone, decreased contrast may be evident in other areas of the foot, while the terminal arch remains. As the entire coffin bone displaces, however, the terminal arch will also be disrupted (Figure 17-8).

In cases of chronic laminitis with extensive destruction of the coffin bone, the canal within P3 is eventually vulnerable. Once the coffin bone has eroded to this point, horses do poorly because of painful osteomyelitis. Furthermore, ischemia results in a corium that cannot grow any quality or quantity of protective sole (Figure 17-9).

Dorsal Lamellar Vessels

The dorsal lamellar vessels are perfused by branches from the terminal arch as well as anastomoses with the coronary plexus proximally and the circumflex vessels distally. As P3 displaces away from the epidermal laminae, blood vessels within the subdermal laminae are disrupted (resulting in hemorrhage into the submural space) or compressed (resulting in deficits in sublamellar circulation). Acute pooling appears to be triangular if the majority of the displacement is rotational, or rectangular if the entire P3 is displacing, as is the case with a sinker (Figure 17-10). Initial decreases in perfusion may progress to complete lack of perfusion within the hoof capsule if all support gives way and P3 collapses or sinks into the capsule.

The lamellar tissue is able to heal somewhat. The lateral venogram allows assessment of that progress. Initially the venogram shows feathering of contrast material into the detached lamellar area, and a thin line of contrast is evident at the most dorsal aspect of the feathering. As the tissue further heals, the feathering is reduced and the dorsal line is no longer evident. Eventually, feathering of only 3 to 4 mm may be noticed at the distal aspect of the dorsal lamellar vasculature where it connects with the circumflex vessels by anastomosis (Figure 17-11).

Coronary Plexus

On the lateral venogram, the coronary plexus appears as a round structure with a void in the center. As the coffin bone displaces, shearing forces within the tissue narrow the

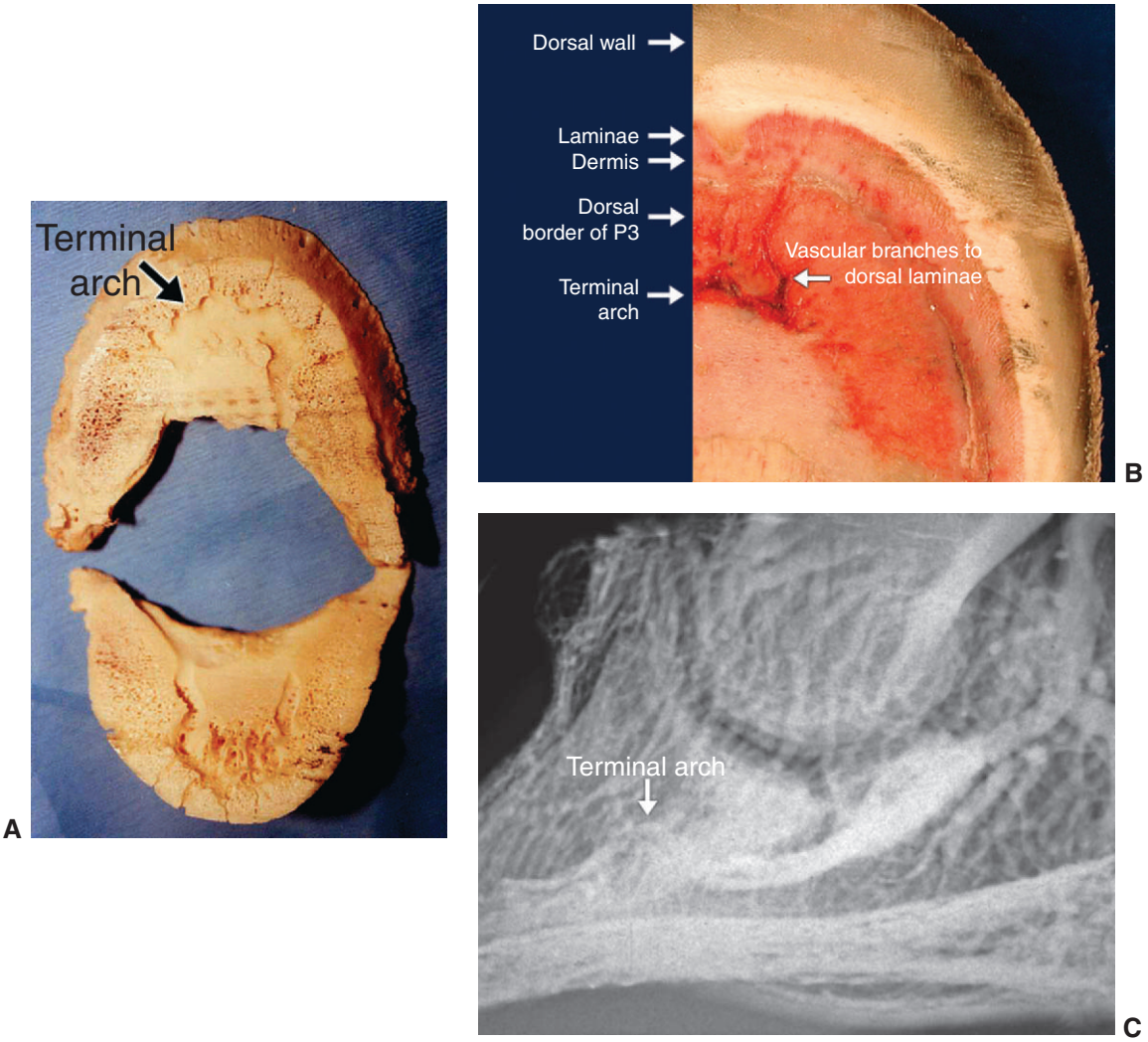

FIGURE 17-7 **A,** Section of a normal coffin bone. The bony canal of the terminal arch of the palmar digital arteries and veins is readily apparent. **B,** This fresh hoof was cut in half in the median sagittal plane and then cut through the terminal arch to demonstrate the branches to the dorsal lamellar blood supply. **C**, The terminal arch on a lateral venogram.

appearance. Additional compression by the extensor process dorsally and the ungual cartilages medially and laterally may eliminate coronary perfusion entirely (Figure 17-12).

Circumflex Vessels

The circumflex vessels are often the first to become compromised with the onset of laminitis. The most sensitive indicator of load may be the sole papillae. Many racing Thoroughbreds have a thin sole that is less than 10 mm in depth when measured just distal to the apex of P3. These horses may be sound on the track but lame on hard or rocky ground. Venograms of these horses will reveal perfusion of the entire circumflex vessel but no dermal papillae. Conversely, a Quarterhorse living on pasture may have a thick sole, at least 20 mm in depth. This venogram will reveal perfusion of the circumflex vessels as well as dermal papillae that extend up to 5 mm distally into the sole corium (Figure 17-13).

In obtaining a venogram on a horse that is suspected of entering the acute phase of laminitis, the sole depth and whether the horse is expected to have visible dermal papillae should be considered. The normal papillae are parallel to the dorsal face of P3; as the coffin bone displaces caudally, the papillae may take on a folded appearance, and it may appear that the tubules are curved and shortened. The next thing to be noticed is a loss of papillae, particularly in the anterior half of the foot distal to the apex of P3. With further displacement, there is a void of contrast distal to the apex of the coffin bone. When the coffin bone displaces distal to the circumflex vessels, there is a "flipped-up" or folded appearance of the circumflex vessel where they form an anastomosis with the dorsal lamellar vessels at the apex of P3 (Figure 17-14). With healing, this area may revascularize, but the folded appearance of the anastomoses remains.

With significant displacement, the margin of P3 moves past the circumflex vessels, compressing them between the

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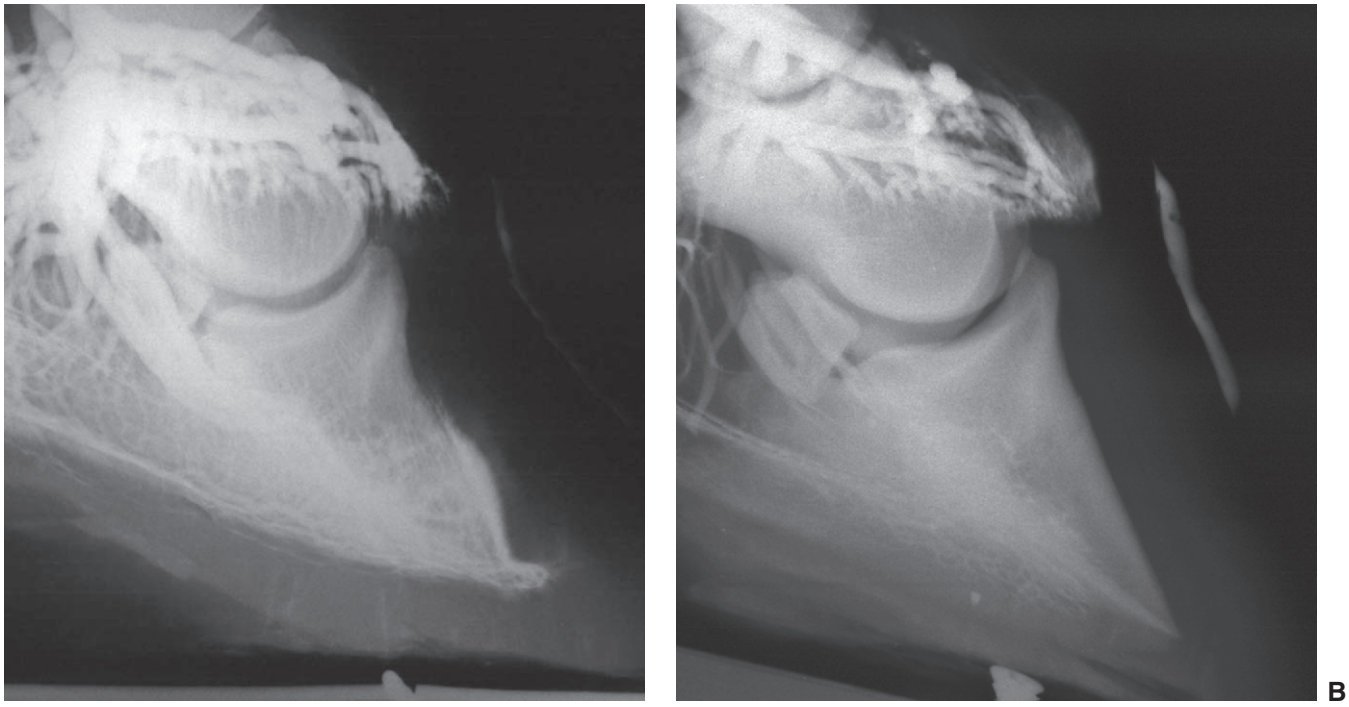


FIGURE 17-8 **A,** Displacement of the coffin bone has resulted in a lack of perfusion in the coronary plexus as well as reduction in the circumflex vessels. The terminal arch remains perfused and supplies the dorsal laminae, unlike the sinker in **B.**

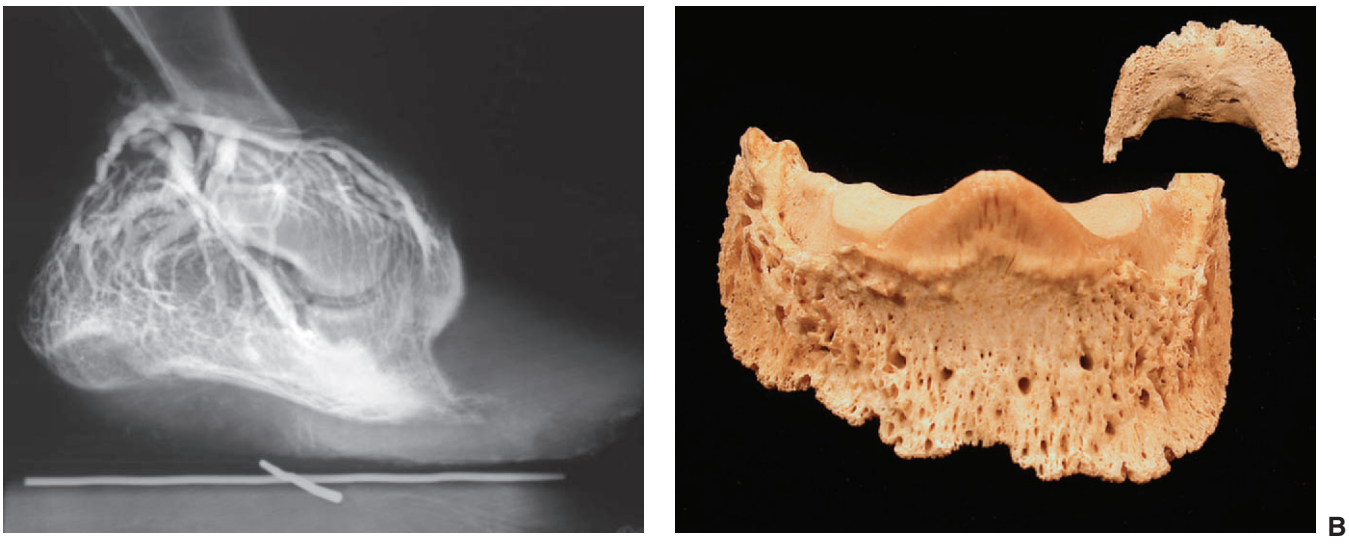
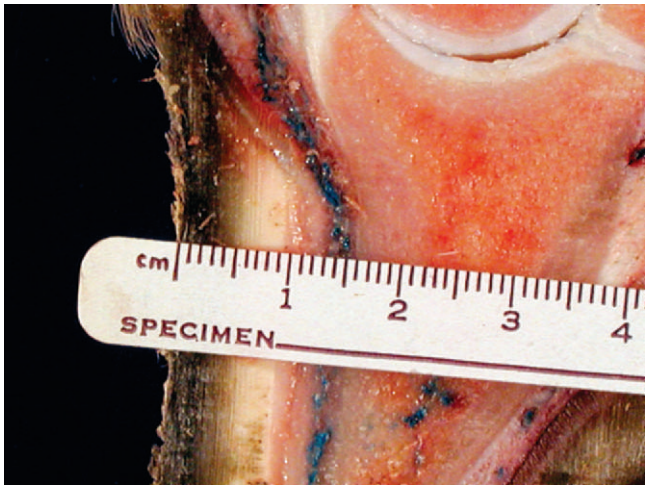


FIGURE 17-9 This 7-year-old Percheron mare presented with chronic laminitis. She was placed in rocker shoes and was able to grow enough sole to be comfortable for a year, which allowed her to raise her recipient foal. However, substantial sole growth needed for surviving in a pasture environment could not be achieved. **A,** Lateral venogram depicting adequate perfusion of the foot with the exception of inadequate contrast distal to the apex of the third phalanx (P3). **B,** Section of P3 postmortem, revealing the osteomyelitis and erosion of P3 to the terminal arch. Note that the damage is more extensive on the medial aspect of P3, which was more heavily loaded in vivo.



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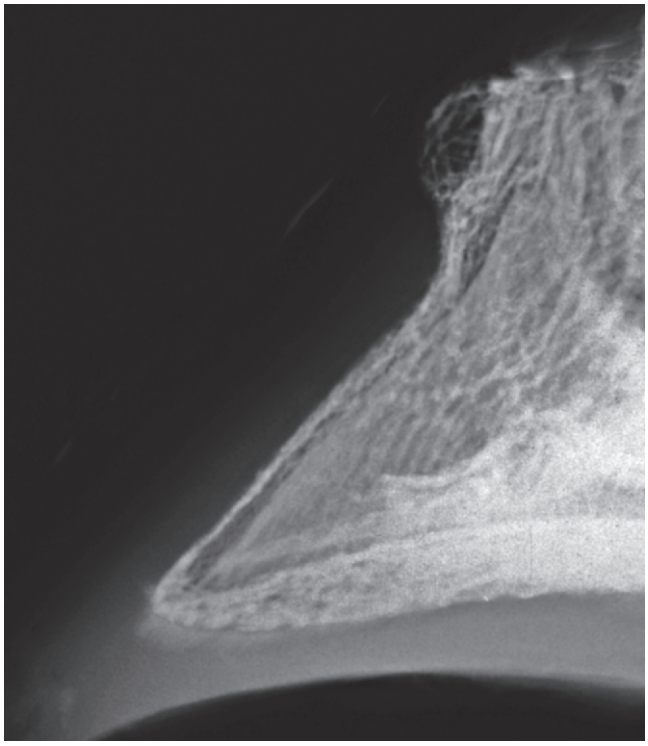

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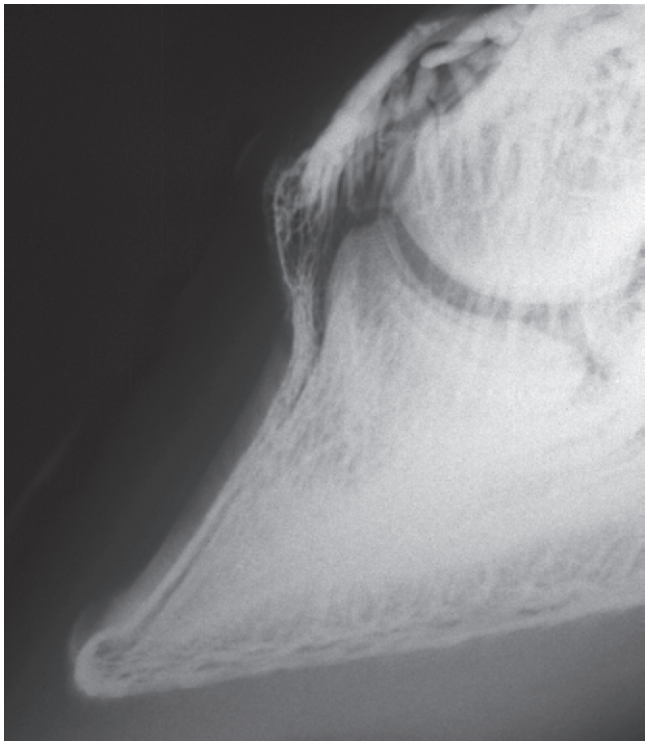

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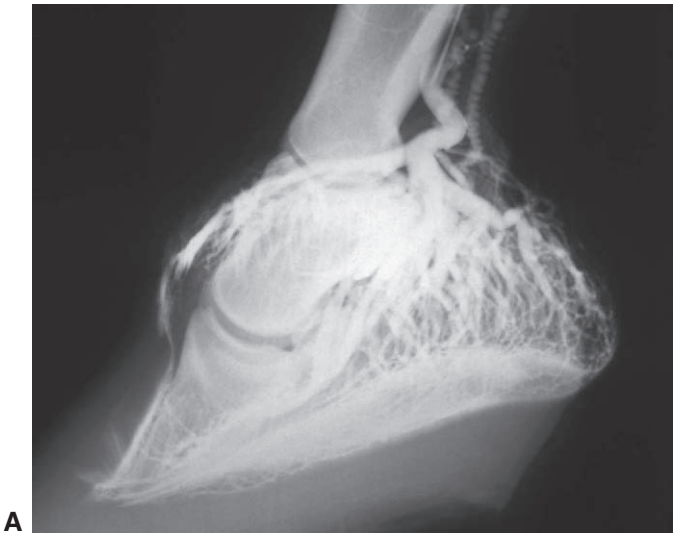
FIGURE 17-10 **A** and **B,** Normal dorsal lamellar vessels are within 4 mm of the dorsal aspect of the third phalanx (P3) and are uniform in appearance. With acute displacement of P3, pooling of contrast material in the area of the detached epidermal and dermal laminae can be seen. If only the distal aspect detaches, a triangular pool of contrast is evident. **C,** As the entire P3 displaces distally, a rectangular pool is evident at the dorsal aspect of P3 on the lateral view. (Also note the folding of the circumflex vessels at the apex of P3 as P3 begins to descend to rest on the sole corium. Although this 800-pound Arabian stallion had a thick sole (18 mm), sole papillae are no longer evident.)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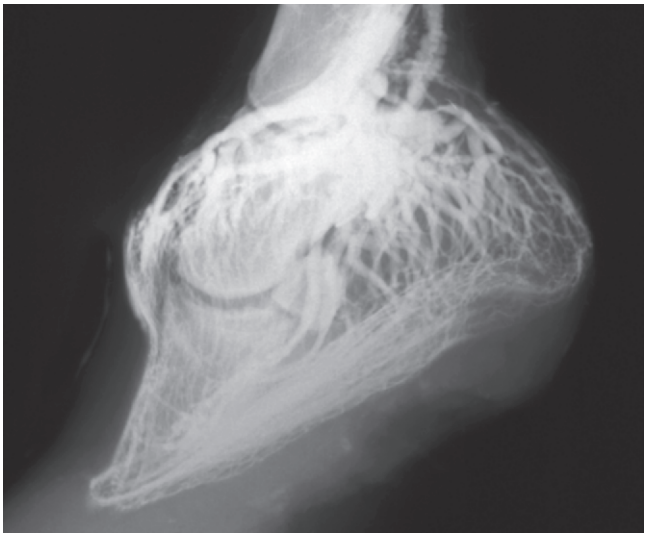
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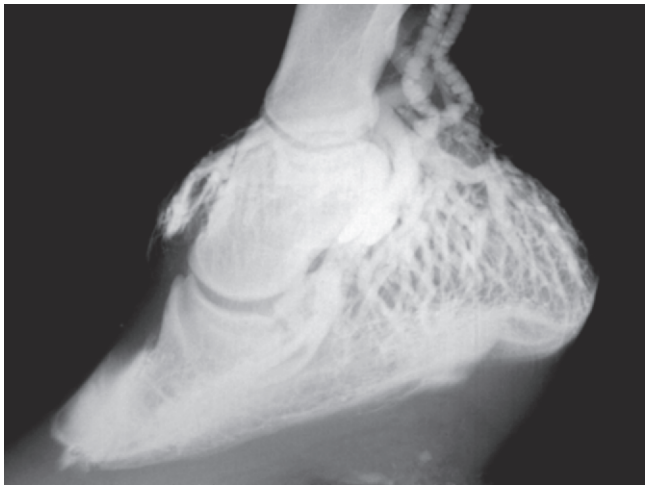
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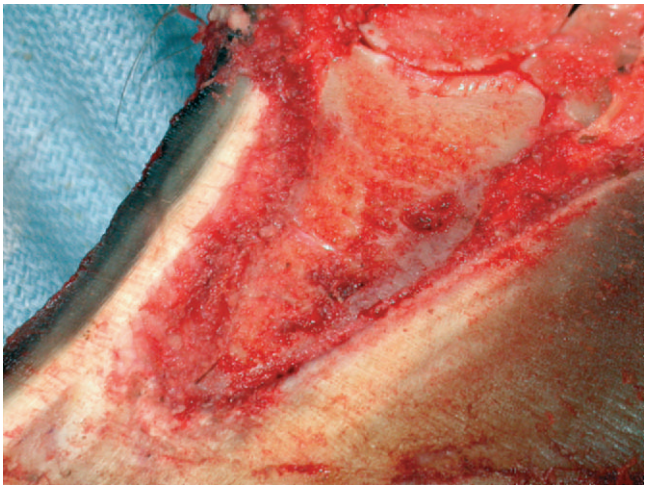
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FIGURE 17-11 This 18-year-old American Saddlebred mare presented with chronic laminitis secondary to Cushing disease. She had periodic flare-ups of laminitis and had lameness of Obel grade IV bilaterally while receiving 4 g of phenylbutazone daily with 15 mm of sole on the left front foot at the initial examination. **A,** Venograms revealed feathering of contrast into the dorsal lamellar area and displacement of P3 distal to the circumflex vessels. The mare was shod with Redden's rocker rail shoes (Redden Aluminum Four Point Rail Shoes; Nanric, Versailles, Ky.) with Advanced Cushion Support (Redden; Nanric, Versailles, Ky.). Six weeks later, the mare had Obel grade I lameness and her sole depth had increased to 22 mm at reset. **B,** At her third reset, the mare was sound and the circumflex vessels and dorsal lamellar tissue had become repaired and were stable. **C,** Two months later, the mare developed acute Obel grade III lameness. No displacement of the coffin bone was evident on plain-film radiographs, but venograms revealed new instability in the lamellar tissue. **D,** The owner eventually elected euthanasia, and a median sagittal section of the foot reveals the unstable dorsal lamellar scar.

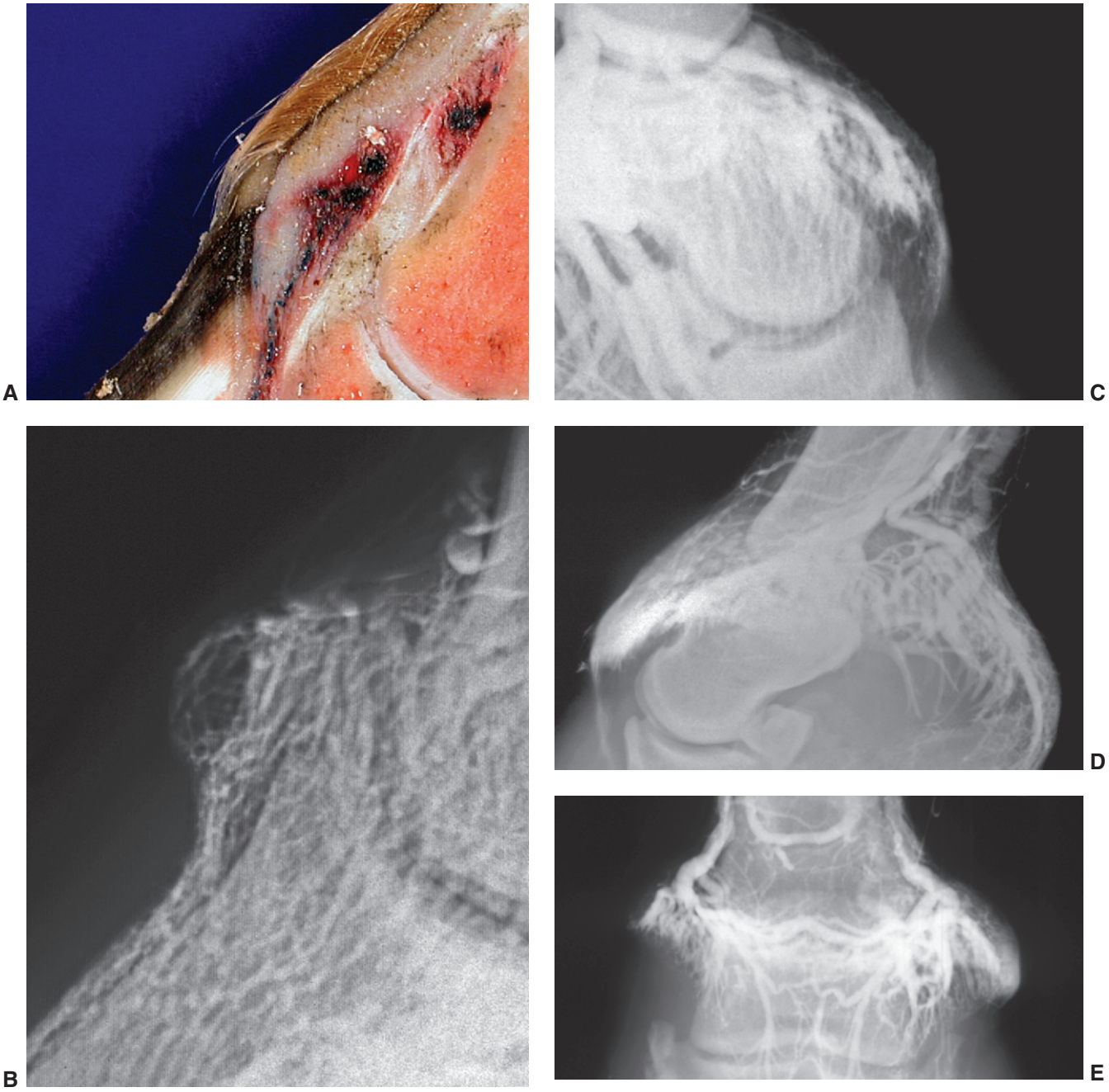


FIGURE 17-12 **A** and **B,** The normal coronary plexus is formed by the vessels coursing around the pulvinis coronalis cartilage. **C,** With displacement, the appearance changes as tissues are compressed or shear. **D,** The "waterfall" appearance is lost as the third phalanx (P3) further displaces, creating a plain line of demarcation at the coronary band with no contrast distally. **E,** The 0-degree dorsopalmar venogram of a foot with severe displacement has perfusion to the coronary band with a plain line of demarcation beneath which there is no contrast.

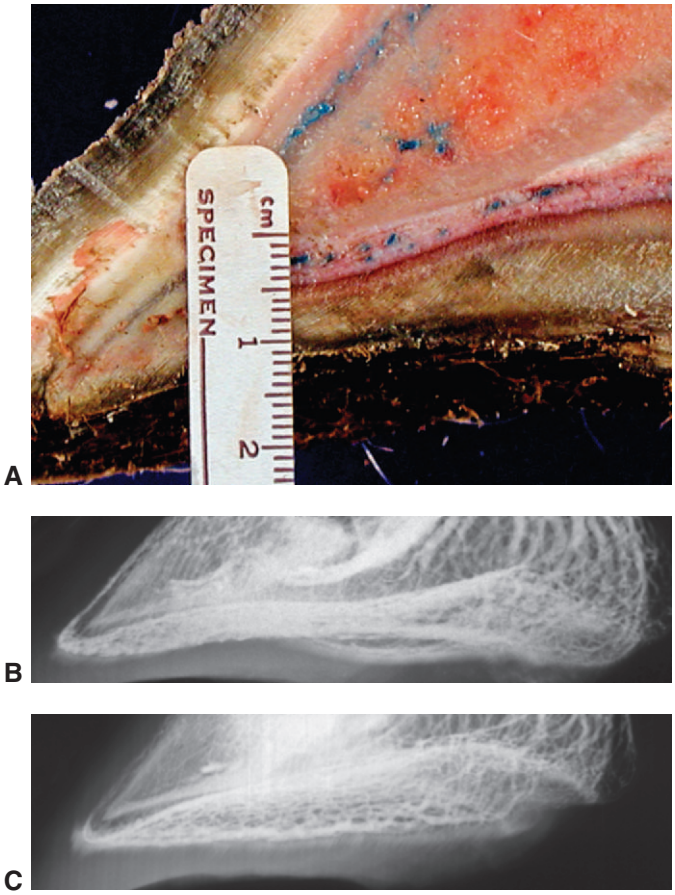


FIGURE 17-13 **A,** This foot was cut in a median sagittal plane, allowing visualization of the 5 mm of sole corium. **B,** Circumflex vasculature of a healthy Thoroughbred foot, which is adequately perfused but does not have the depth of dermal sole papillae seen on the thick-soled foot of a Quarterhorse **(C).**

wall and dorsal surface of P3. The sole corium perfusion is eliminated as the anterior portion of P3 loads the tissue and interrupts the blood supply from the circumflex vessels. (The corium of the heel rarely appears affected unless the entire coffin bone displaces distally.)

The depth and amount of circumflex vessel and sole corium perfusion may vary in an unstable foot depending on the degree of damage and the palmar angle of the coffin bone. If the heel of the hoof is elevated 20 degrees, an improvement in solar perfusion may be evident (Figure 17-15). The strain of the deep digital flexor tendon is reduced when the palmar angle of the coffin bone is increased.[23,24] Wedging the heel of a sound foot will decrease the volume of contrast in the heel from the widest point of the foot caudally. With an unstable foot, the heel perfusion is also reduced, but as one reduces the palmarodistal pull of the apex of P3 onto the sole corium, perfusion may improve in the anterior portion of the hoof. Extensive rotational displacement of P3 may result in penetration of the sole by the coffin bone; contrast material is often seen escaping from the hoof capsule.

Heel Vasculature

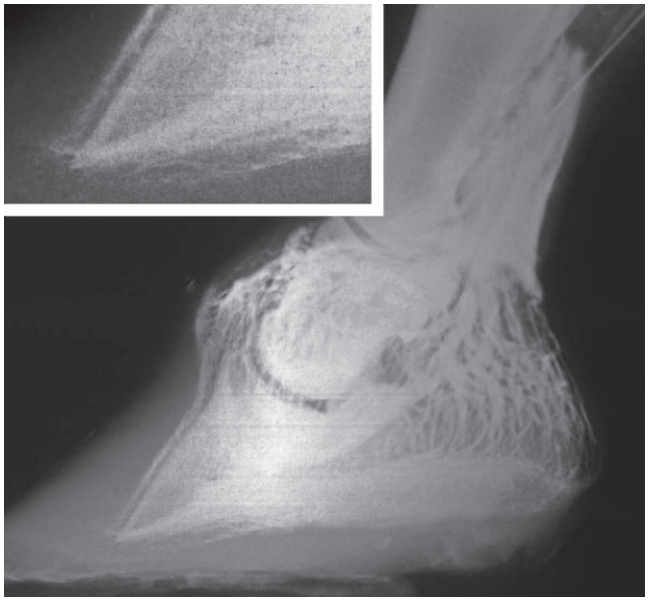
Because the bulbar artery arises in the palmar pastern to supply the heel, and because the majority of the tissue damage and mechanical load of the laminitic foot occurs with displacement of P3 in the anterior half of the foot capsule, the heel vasculature is seldom reduced (see Figures 17-11, *A;* 17-14, *A;* and 17-15, *B*). However, with severe distal displacement of P3 even the heel perfusion is eliminated (Figure 17-16).

CLINICAL APPLICATION

The following case presentation is an example of utilizing venograms in the clinical setting to help make decisions regarding treatment and evaluate progress.

Fanny was an 18-year-old American Saddlebred mare that developed laminitis. She had been treated medically and placed in a sand-lined stall, but showed no significant improvement over a 2-week period. Upon examination, she had bounding digital pulses, was bilaterally lame (worse on the left front foot), and had displacement of the coffin bones on radiographs. The front feet were trimmed and placed in Modified Ultimate Shoes with Advanced Cushion Support (Redden, Nanric, Versailles, Ky). Fanny's feet were reset at 6-week intervals. She received 1 g phenylbutazone twice daily. She had increased sole depth and improved soundness but continued to favor the left forelimb. Sixteen weeks after her initial presentation, the owners called to report that Fanny was reluctant to bear weight on the left forelimb and that an abscess had broken at the lateral coronary band. Three days later her lameness had not improved.

On reexamination, the left foot had new medial growth but no lateral growth, and the coronet was prolapsed over the lateral wall with serum evident at the coronet separation, which extended from the lateral toe to the heel. The coronet was warm and painful to palpation. Radiographs revealed no significant change from those taken 4 weeks previously, but I was skeptical that Fanny was suffering from a simple abscess. Venograms were obtained and demonstrated a sinker pattern with minimal perfusion occurring distal to the coronet (Figure 17-17). I recommend treating sinkers by stripping the wall and supporting the weight of the horse using pins placed through the cannon bones into a transfixation cast. The theory is that a sinker's hoof wall acts as a tourniquet, enhancing vascular stasis and restricting swelling of the inflamed and edematous corium and laminar tissue. Removing the wall eliminates the compartmentalization injury and allows reperfusion of the tissue. If the cellular damage is not too extensive, the foot may recover and grow a new wall with some attachment at the repaired laminae. This is obviously an expensive procedure requiring a life-long care commitment by the owner in hopes of a pasture-sound horse. Fanny had two things that made me question if total wall removal was indicated: (1) the contrast on the venogram extended adjacent to the tourniquet, as can happen with tourniquet failure; and (2) the possibility of her having a pinched lateral wall (as evidenced by the lack of new lateral hoof growth) that developed a serum pocket and caused the coronary prolapse.



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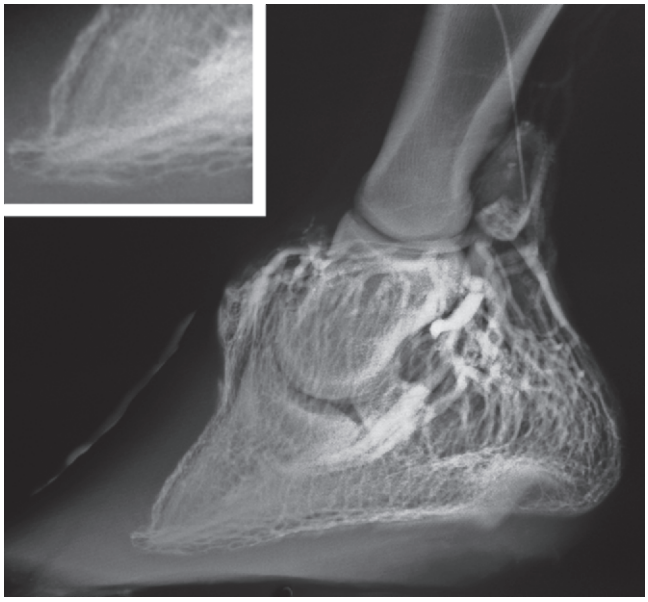

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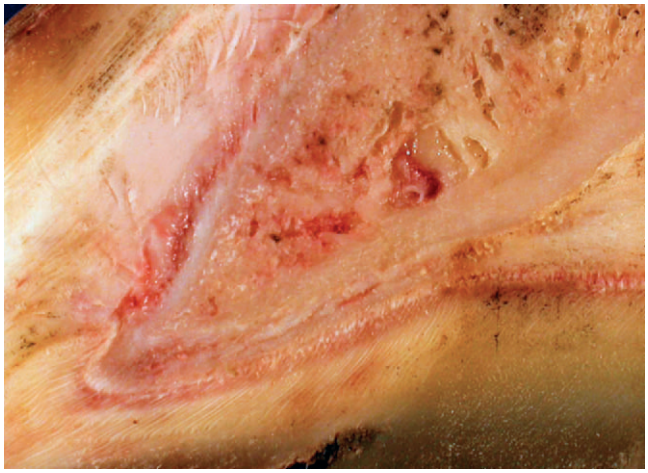


C

FIGURE 17-14 This 7-year-old Appaloosa mare presented with acute laminitis secondary to grass foundering. **A,** Displacement of the apex of the third phalanx (P3) distal to the circumflex vessels with a folding of the circumflex-laminar anastomoses is evident, as is complete lack of contrast distal to the apex of P3. The mare was initially treated with antiinflamatory drugs, hoof trimming, and solar packing with 2-inch Styrofoam. As her condition improved, she was trimmed infrequently and returned to free-choice pasture. Eighteen months later, the mare was euthanized, based on the owner's unwillingness to treat recurrent foot abscesses at the widened white line of the dorsal lamellae. **B,** The venogram revealed that both the coffin bone and the vasculature had remodeled. Perfusion of the dorsal lamellae and circumflex vessels is apparent, as is a folded appearance of the anastomoses typical of a repaired vasculature. **C,** A median sagittal section of the foot is presented.

I hoped that by removing a portion of the lateral wall we could eliminate a significant amount of inflammation and reestablish perfusion without making the valiant effort of removing the entire wall.

The proximal half of the wall was removed from the lateral heel to the lateral toe pillar with only minimal hemorrhage. The edematous laminae were gently massaged until blood supply resumed, the area was packed with saline-soaked gauze and the Modified Ultimate shoe replaced.

Three days later, new venograms were obtained (Figure 17-18). Only the lateral half of the foot had reperfused, indicating the need for complete wall stripping and transfixation casting. Forty-two days later, Fanny was placed in a sling to partially support her weight, and the transfixation cast was removed. The foot was growing nicely with cornified laminae apparent in the resected wall area and new wall growth evident at the coronary band (Figure 17-19).

Fanny continued to do well on the casted left forelimb, but favored she it and overloaded the right forelimb. As two months passed, we continued to note soft tissue radiolucencies in the sole of the right foot where Fanny developed serum pockets. Eventually venograms revealed a significant vascular compromise and we removed the Modified Ultimate shoe, shod Fanny with a 5-degree wedged heel aluminum shoe, and performed a deep digital flexor tenotomy (Figure 17-20). Because I have noted that tenotomized horses grow more toe than heel, I took venograms to monitor the progress of revascularization. The Modified Ultimate Shoe reduces the strain of the deep digital flexor tendon and loads the heels. The tenotomy completely eliminates all pull of the dorsal aspect of P3 away from the dorsal laminae and also eliminates the downward pull onto the sole corium. However, the "lift" of the heel is also eliminated and it "settles" into the hoof capsule, loading the relatively healthier heel tissue. Three months after the pin cast, a deep digital flexor tenotomy was performed on the left forelimb. Six months after wall stripping, the left foot had grown out nicely (Figure 17-21).

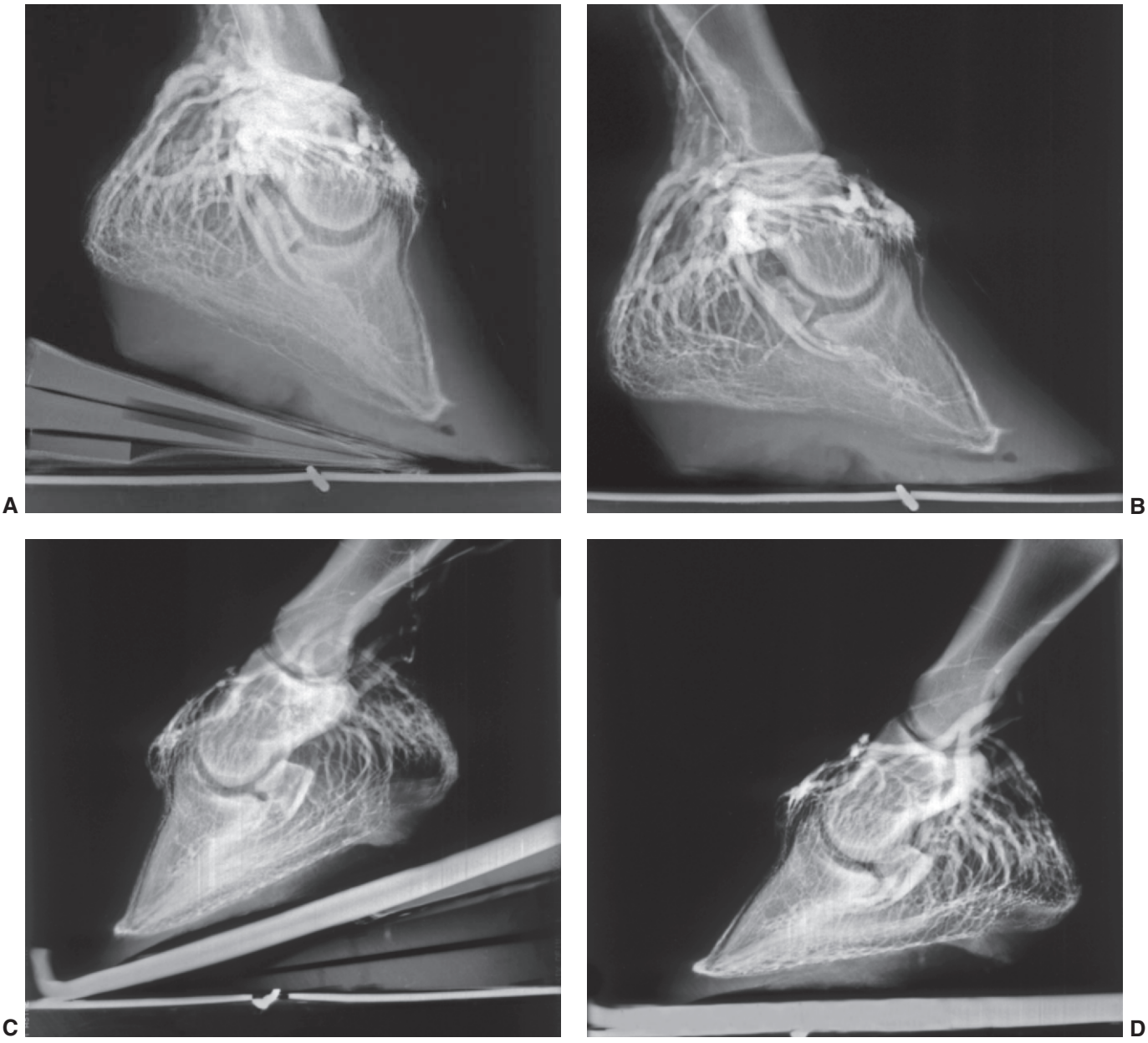


FIGURE 17-15 A 20-year-old American Saddlebred gelding presented with acute laminitis. Solar perfusion distal to the anterior portion of P3 is increased when the heel is elevated **(A)** compared to zero degrees **(B)**. In a healthy foot, perfusion does not change in the anterior half of the foot with heel elevation. **C** and **D,** There is no difference, with the exception of reduced heel perfusion, between the elevated and the loaded condition. *(Courtesy R.F. Redden.)*

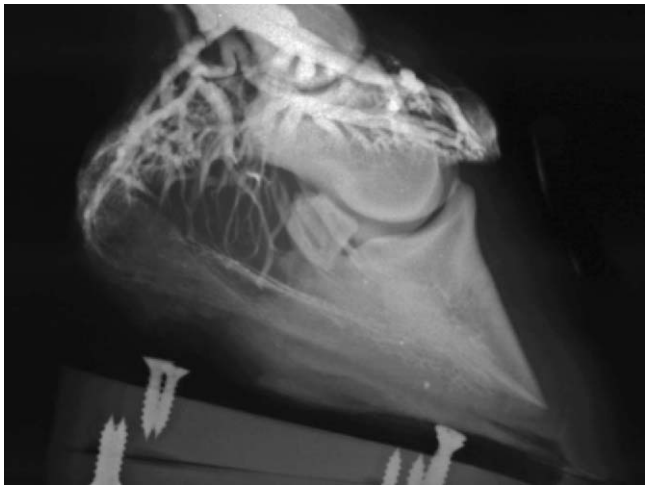
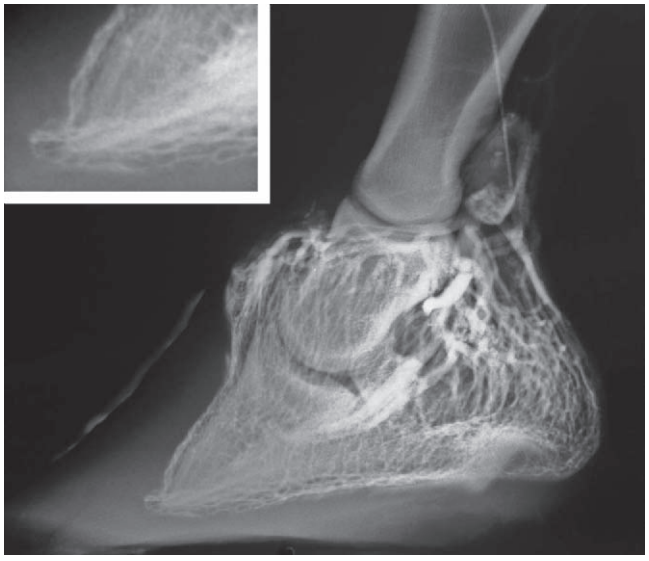


FIGURE 17-16 This sinker has inadequate perfusion distal to the coronet in all areas, including the heel vasculature.



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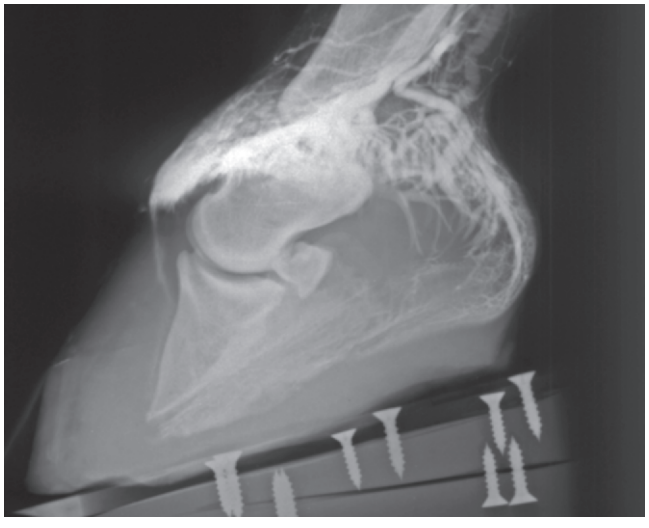

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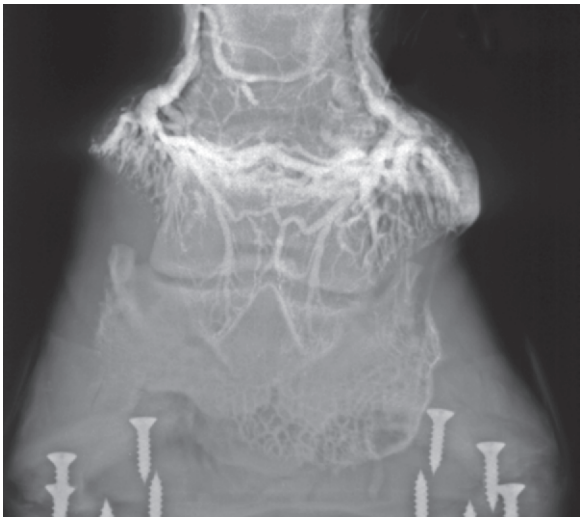

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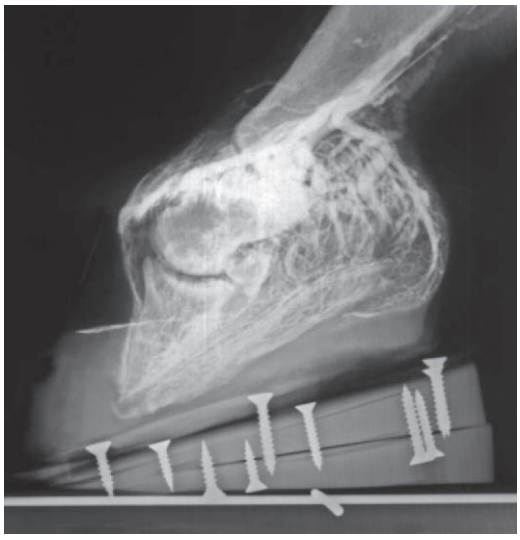
FIGURE 17-17 Venograms of Fanny's left front foot (in the Modified Ultimate shoes) reveal inadequate perfusion distal to the coronet on both the lateral **(B)** and 0-degree dorsopalmar view **(C).** A minimal amount of contrast is evident at the dorsal lamellae, in the caudal circumflex vessels, and in the palmar heel. Stark lack of contrast is apparent at the coronary plexus and the terminal arch.



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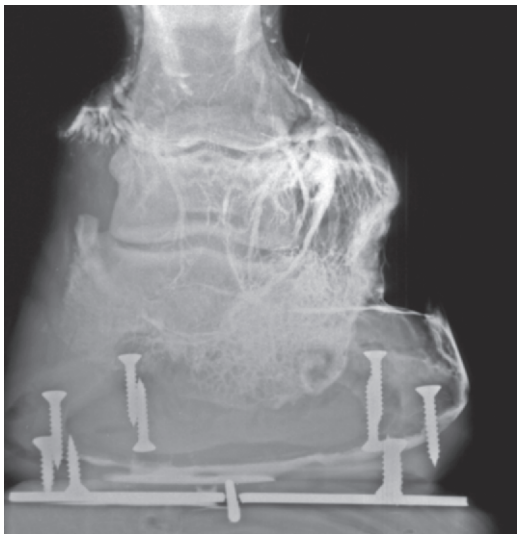

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FIGURE 17-18 **A,** The second set of venograms reveal lateral perfusion of the terminal arch, coronary plexus, lamellae, circumflex vessels, and heel region. **B,** The dorsopalmar view confirms a continued lack of perfusion of the medial foot. Note on both venograms and the photo of the foot that where the wall had been resected, contrast material began running from the laminae. **C,** Within 1 minute, contrast material was pooling on the wall and shoe, then running out onto the ground.

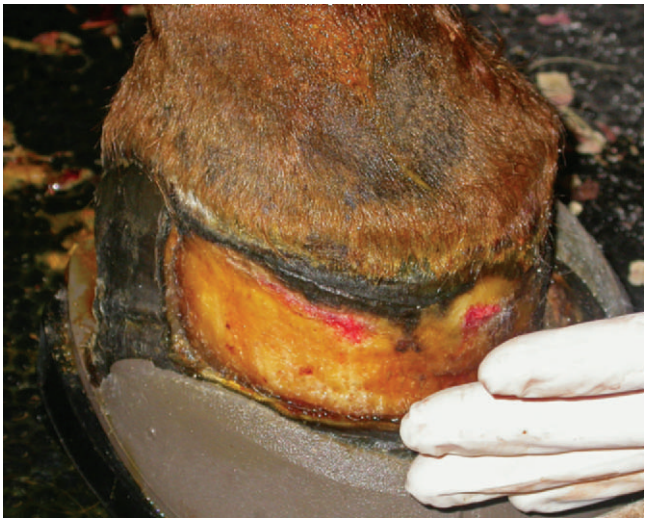

FIGURE 17-19 Dorsolateral view of the cornified laminae and wall growth 42 days after wall stripping.

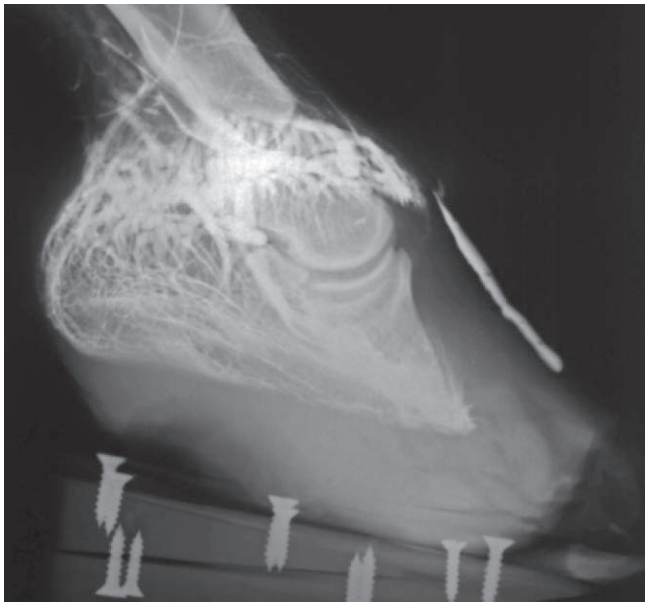

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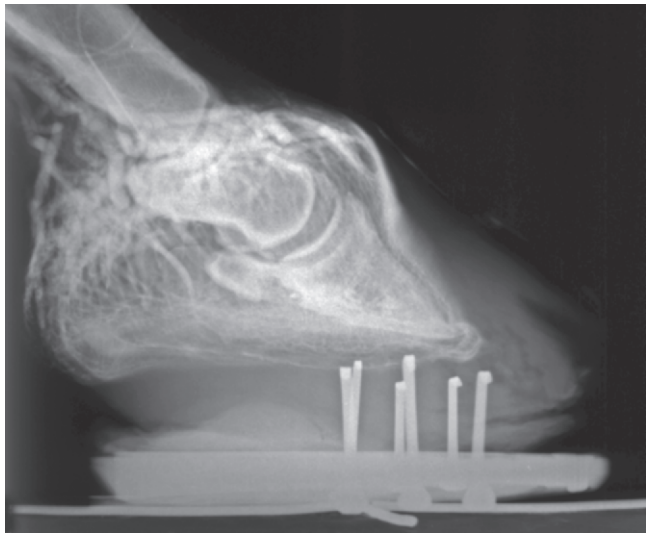
FIGURE 17-20 **A,** On the lateral view, Fanny's right foot has reduced perfusion at the coronary plexus and circumflex vessels. The proximal dorsal lamellar perfusion is reduced, and distal feathering of contrast into the laminae is observed.



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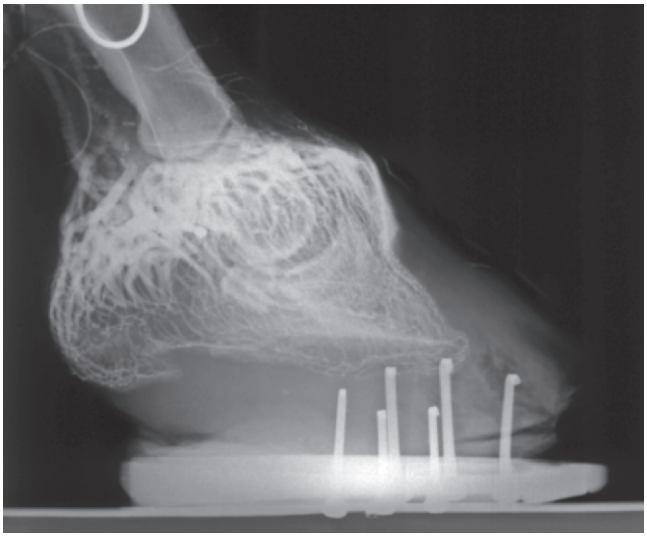

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FIGURE 17-20, cont'd **B,** The foot was shod, a tenotomy performed, and a new venogram obtained 8 days later. At that time, perfusion had returned to the dorsal lamellar and circumflex vessels. **C**, Sixteen days postoperatively, a final venogram was obtained and loading of the palmar circumflex vessels and vasculature of the heel was evident.

SAFETY

Of more than 911 venograms obtained by the author's practice by 2002, 180 were obtained on normal or sound horses. No detrimental effects were noted. Perivascular injection resulted in subcutaneous contrast accumulation and localized edema that resolved within 3 to 24 hours. One previously sound horse developed a transient lameness 24 hours after undergoing a venogram procedure: a sole bruise was identified and attributed to turn-out on rocky ground before resolution of the peripheral nerve block.

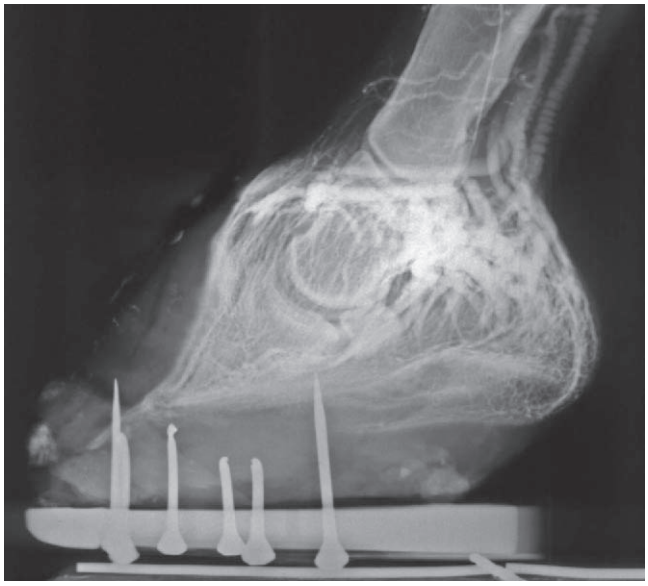
The most important safety consideration in obtaining venograms is preservation of venous integrity in laminitic horses, which already have compromised circulation. The technique should be mastered on sound horses before it is attempted on a horse with a pathologic condition. Also, locally anesthetized pathologic feet should not be mechanically challenged during the venogram procedure. Shoes are removed, and then the feet are placed in an elevated heel shoe (Redden Aluminum Four Point Rail Shoes) for the duration of the procedure. With elevation of the heel, the deep digital flexor tendon tension is reduced, decreasing the pull of P3 away from the dorsal laminae and onto the sole corium.[23,24] There are anecdotal reports of horses having complications secondary to venogram procedures that are incorrectly performed; if perineural anesthesia or venipunctures disrupt the vasculature of the foot, or a horse with an unstable coffin bone is left standing with blocked feet for a prolonged amount of time, this will have a deleterious effect on the already compromised tissues. Again, it is strongly recommended that this technique be perfected on a normal horse before it is attempted on a horse that has active pathologic processes.

Venograms are routinely obtained using hyperosmolar contrast agents. These agents contain 370 mg iodine/mL, or 37% iodine, which creates an osmolality of 2016 mOsm/kg (Renografin 60 or Hypaque-76). Although isotonic contrast agents (Omnipaque; Nycomed, Princeton, NJ) are available, their cost is considerably higher and their use has not been shown to be preferable. The safety of injecting hyperosmolar agents into the foot has been questioned, but in the author's experience this diffusion gradient is not harmful. In an informal safety study, venograms were obtained on seven horses that were subsequently euthanized for reasons unrelated to their feet. The left or right forefoot was selected randomly for each venogram, with the other foot left untreated as a control for comparison. The horses were euthanized 2 hours to 8 weeks after venograms were obtained. Horses that had undergone venography several weeks before euthanasia had no grossly discernable clinical differences between the right and left feet. At euthanasia, dorsal hoof wall biopsy samples of both feet were taken, using the technique described by Hood, for subsequent evaluation by a pathologist.[5] Histopathologic examination failed to distinguish between venogram and control samples; no microscopic lesions could be attributed to the venogram procedure.

DISCUSSION

The venogram procedure is relatively inexpensive, has minimal side effects, can be performed efficiently, and requires only standard radiographic equipment present in most equine hospitals. With a small amount of practice, a team of people can become proficient at obtaining the venogram and maximizing the amount of information they obtain.

Venogram images closely correlate with both clinical and postmortem findings. The venogram is advantageous because it is a record of the vascular status of the foot, which is of primary importance in managing laminitis. Plain-film radiographs are often inconclusive in laminitic cases; venographic images define vascular pathologic lesions and suggest the degree of soft tissue compromise in even the mildest of cases. If consistent radiographic techniques are used, comparison over time and assessment of healing or disease progression is possible. The digital venogram has been an invaluable tool in designing treatment regimens and offering prognoses.



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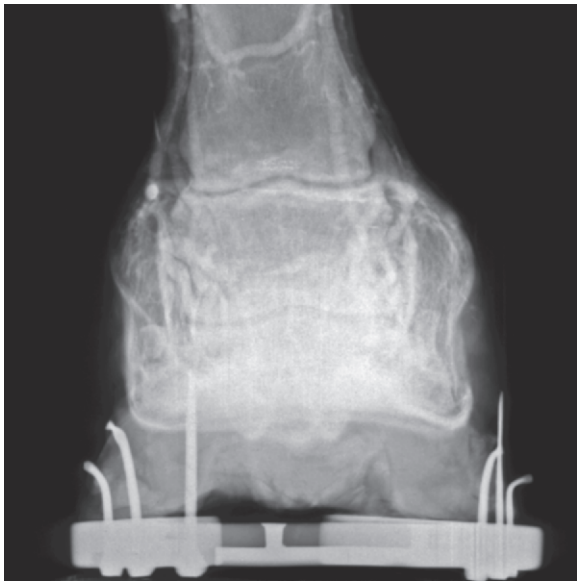

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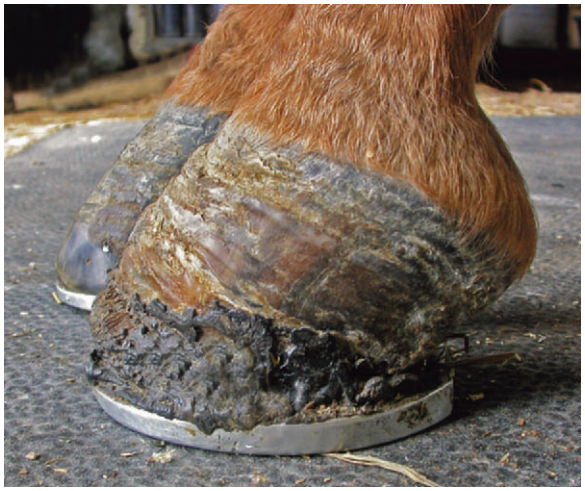

C

FIGURE 17-21 The left foot has adequate perfusion on the venogram (**A** and **B**) and has adequate wall and sole growth **(C).**

Acknowledgment

The author would like to thank Howard Wilson for his assistance editing the images for this chapter.

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18

AN APPROACH TO THE TREATMENT OF THE LAMINITIC HORSE

ANDREA E. FLOYD

Much has been written and many approaches have been tried for the treatment of the laminitic horse. In this chapter, an approach is described that has worked successfully in the author's hands for the majority of cases treated. The pitfalls and shortcomings of any treatment and particularly with this approach are discussed in detail. It is extremely important to understand that the treatment protocol chosen should be one founded on successful cases built on years of using and improving the protocol. It is not expected, nor is it fair to expect, that a general equine practitioner should understand the intricacies of the intensive labor associated with grade IV cases; nor is it expected that the general equine practitioner will want to take on the task of treatment, which is arduous and round-the-clock for a period that easily ranges from 6 to 8 months. Rather, this chapter should be read as a treatise, providing insight into the grade IV case and its outcome. Should general equine practitioners desire to undertake these intense cases, they will be armed with a working protocol. Should they decide to refer these cases to a podiatry facility, they will be armed with the knowledge of what a grade IV case is and how important it is to refer these cases early in the disease process. This chapter deals with the severely laminitic horse (the Grade IV case), as the other grades of laminitis have been discussed in Chapter 16.

Within the last few years, a multitude of over-the-counter remedies have appeared that claim to treat even the worst case of laminitis. There have been herbals, minerals, mud, shoes, boots, sole pads, creams, ointments, vitamins, machines, and therapies, all "guaranteed" to treat laminitis. Let it be said here with all gravity, there is only one successful treatment for laminitis: Restoration of blood flow to the affected tissues through realignment of the digital column and re-establishment of a normal palmar angle. This will stimulate the production of healthy lamina and solar corium.

There are two types of grade IV laminitis:

- The "cranial rotation," or increased palmar angle case
- The "sinker," or fatal sinker syndrome (FSS) case

These cases can be simple "cranial rotations" or "sinkers," or they can be a combination of both types to some degree. The two types are discussed separately in this chapter.

TREATING THE INCREASED PALMAR ANGLE (CRANIAL ROTATION) GRADE IV ACUTE LAMINITIC CASE

Symptoms

The grade IV severely laminitic horse is in extreme pain on presentation, with an elevated heart rate, hyperemic conjunctivas, bounding digital pulses, and anxious facial expression. It makes every attempt to avoid turning or using the toes when walking forward. The general practitioner may also be presented with the laminitic horse that is unable or unwilling to rise.

Visual inspection of the hooves may reveal puffy coronary bands and coronary band separation with oozing serum or purulent blood-tinged exudates. The sole of the hoof may be flat, domed (convex) over the distal dorsal tip of the third phalanx, or ruptured with exposed sensitive solar corium (Figure 18-1). (NOTE: The exposed corium is fleshy and pink and resembles proud flesh [granulation tissue]; however, it is not proud flesh but a viable structure that is very painful once exposed to the air.)

Radiograph

When viewing the lateral radiograph of the hoof capsule, the palmar angle is usually greater than 19 degrees, and the sole depth at the toe (SDT) is reduced or 0 mm in depth (an SDT of 10 to 15 mm or greater is normal). The extensor process to coronary band measurement (EP-CB) is increased (an increased EP-CB is often erroneously diagnosed as FSS), as is the horn-lamellar (H-L) zone measurement, with the proximal measurement being smaller than the distal measurement. Another measurement that the author uses is the wing-to-bulb measurement (Figure 18-2). This measurement is used in all cases that are possible septic or sinker (FSS) cases. (See Chapter 16 for a review of the measurement protocol used by this author). (NOTE: Certain breeds of horses such as American Quarterhorses, Appaloosas, Paints, and other breeds with predominant Quarterhorse breeding may have started with a negative palmar angle [Figure 18-3], which is very important. Many cases involve a symptomatic horse in acute pain that

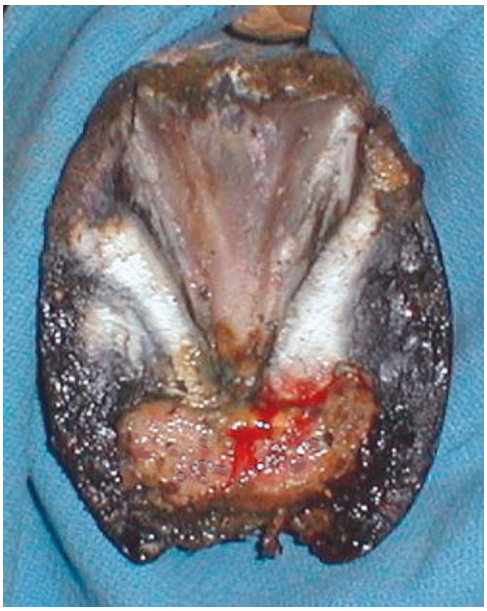

FIGURE 18-1 The corium, once exposed, resembles granulation tissue and is extremely painful to palpation.

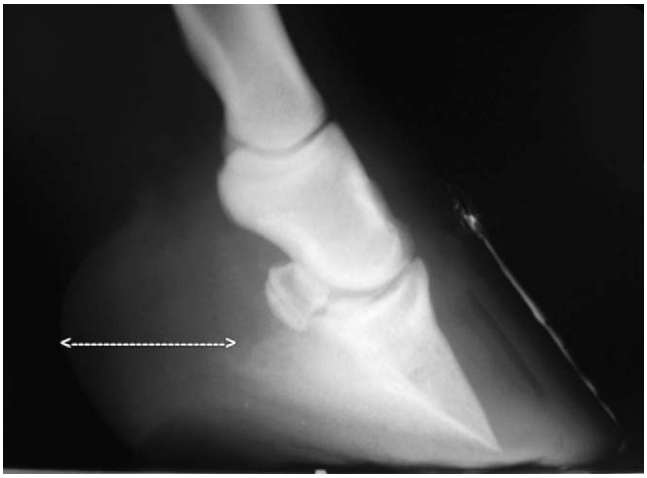

FIGURE 18-2 The wing-to-bulb measurement will aid in diagnosing fatal sinker syndrome, as the third phalanx slides down and backward within the hoof capsule.

has a normal palmar angle and H-L zone. If the horse had a negative palmar angle as a normal physiologic feature, then the pathologic change to a normal palmar angle and H-L zone would be an excruciatingly painful event.)

The Venogram

Venograms should be obtained immediately on the standing, sedated horse (see Chapter 17 for the anatomy of the venogram). Once the extensor process starts to dip distally with the increased palmar angle, impingement of the coronal cascade and the proximal portion of the dorsal circulation will be evident on the venogram. If the loss of venous supply is severe, no blood will be visible in this area (Figure 18-4). The dorsal circulation may be greatly reduced. The H-L zone increases in length with the amount of edema in the vascular

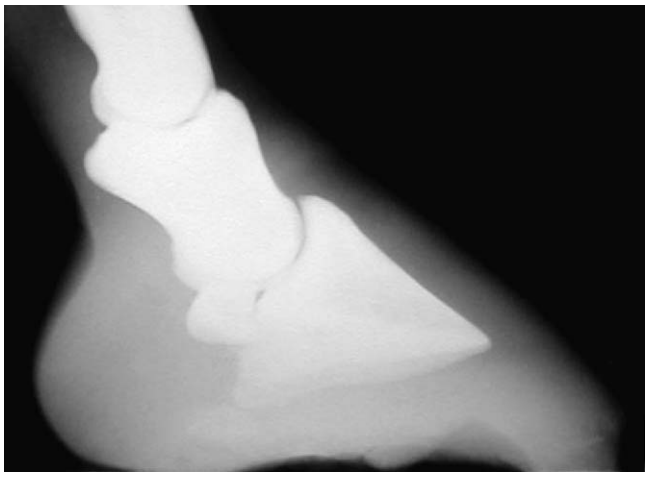

FIGURE 18-3 The majority of horse breeds containing Quarter-horse genes have a negative palmar angle or 0-degree palmar angle.

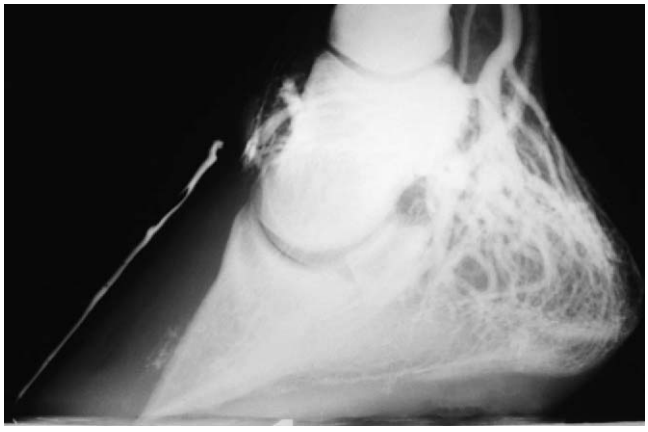

FIGURE 18-4 Venogram showing impingement of the coronal cascade and proximal dorsal circulation. This is caused by the downward pressure of the extensor process of the third phalanx. Also visible is the distal margin of the third phalanx below the circumflex circulation.

tissues; greater than 20 mm of H-L zone indicates edema. Chronic previous episodes show as a "wisping" on the venogram where the blood is flowing into the separated dermal and epidermal lamina (Figure 18-5). The dorsal and circumflex circulation often appear "looped" as the distal margin of the third phalanx (P3) rotates downward out of the circulation (Figure 18-6). This tip of P3 is often visible below the circulation (see Figures 18-4 and 18-6). The bulbar circulation is usually full and prominent. The solar fimbriae in the anterior portion of the circumflex circulation are greatly shortened or absent. All vessels are usually apparent emanating from the terminal arch. On the dorsopalmar venographic view, it is normal to see a slightly different pattern between the medial and lateral aspects of the dorsal and circumflex circulation (Figure 18-7). The medial aspect of the venous filling often appears less prominent than the lateral but is always fully visible unless medial listing is occurring. Medial listing is not FSS but rather is a loss of the integrity of the lamina in the dorsomedial area of the hoof. With medial listing , the entire bony column can be seen to be "sinking" to the medial side and there is a loss of blood flow below the

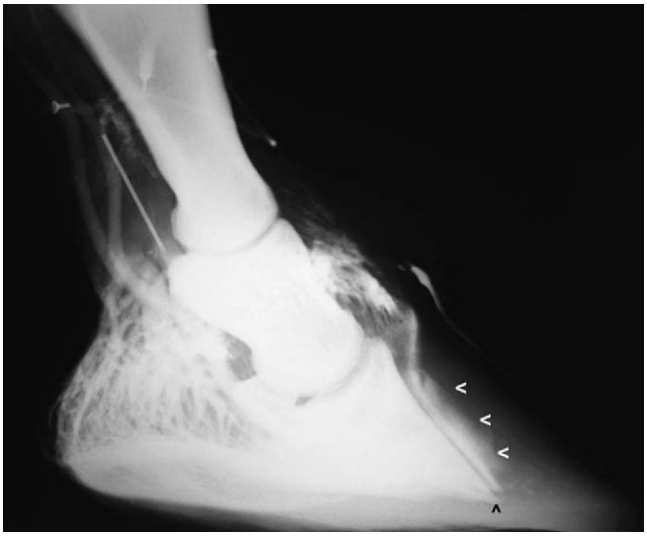

FIGURE 18-5 Wisping (*white arrowheads*) of the dorsal circulation in a horse with chronic episodes of laminitis. *Wisping* is a term used to denote the circulation visible within the stretched lamina of the horn-lamellar zone. The *black arrowhead* points to the distal margin of the third phalanx seen protruding below the circumflex circulation.

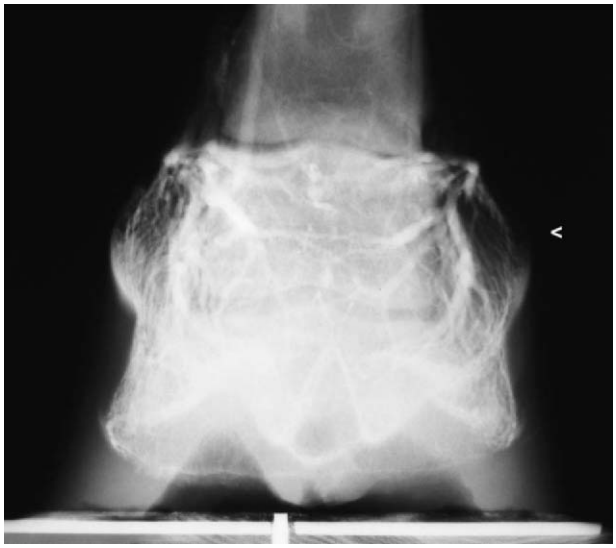

FIGURE 18-7 The *white arrowhead* points to the medial aspect of the coronal cascade and the proximal dorsal circulation. The medial aspect normally appears to have less venographic anatomical presence than the lateral aspect.

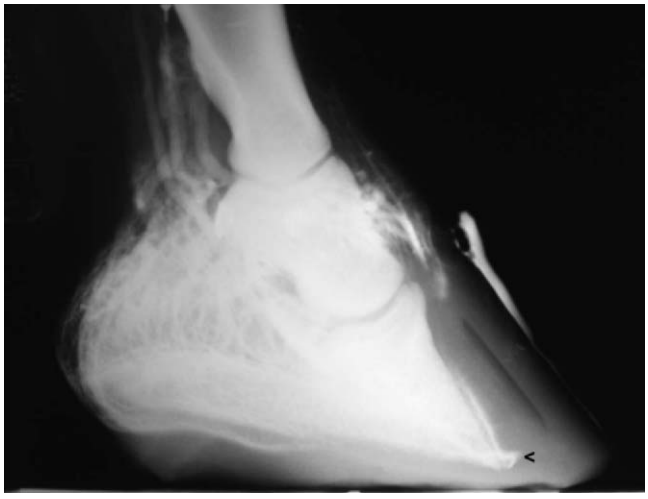

FIGURE 18-6 The looping appearance of the circumflex and distal aspect of the dorsal circulation that occurs when the distal margin of the third phalanx moves downward in the hoof capsule.

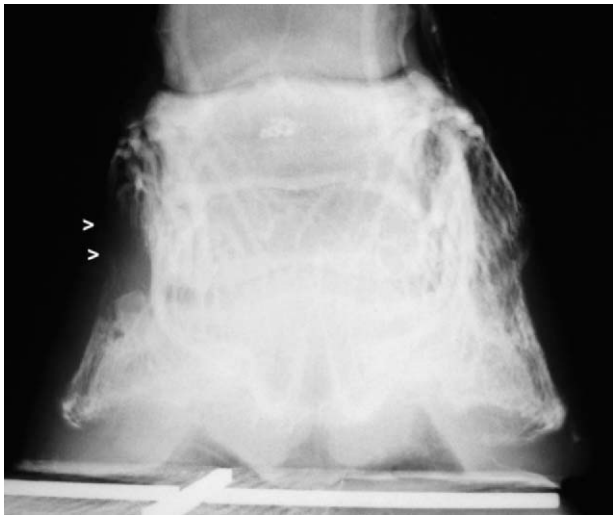

FIGURE 18-8 A dorsopalmar venogram that shows circulatory compression of the proximal portion of the dorsal circulation (*white arrowheads*). This condition is due to medial listing of the digital column.

coronal cascade. In severe cases, the coronal cascade is no longer apparent (Figure 18-8).

The Prognosis

At the onset of a case workup, whether it is an acute or a chronic laminitic case, it is very difficult to make a prognosis based on the accumulated symptoms and venographic and radiographic evidence; rather these tools should be used to form a treatment or surgical plan for the recovery of the animal. Many cases have been observed that were considered doubtful for full recovery and were given a poor prognosis, yet these horses did recover with a diligent treatment plan. All horses can survive, unless a thromboembolic event occurs in more than one foot causing loss of the digital column. Recovery may necessitate an extraordinary effort, such as transcortical pinning with hoof wall ablation or, finally, amputation.

From the perspective of pain and the horse's welfare, the question has been posed: When is enough enough? Indeed, there is a painful period during the recovery process, but it has been the author's experience that it is a relatively short period that can be managed through a good pain relief protocol. The author also thinks that too many horses are euthanized with minimal laminitic problems because of the lack of a good treatment plan for recovery.

In the long run, lack of knowledge and financial considerations may be the ultimate reasons for success or failure. Therefore it is recommended that veterinarians use these diagnostic tools to formulate recovery, not discourage it.

Evaluating the Data and Formulating the Treatment Plan

Horses with a standing heart rate of 80 to 100 beats/min and a recumbent heart rate of 60 to 70 beats/min are in serious crisis. These horses usually show marked gas or serum pockets on the H-L zone with perforated soles or convex (domed) soles. Venograms show marked reduction in blood flow through the coronal cascade and dorsal and circumflex circulation. These cases require immediate midcannon tenotomies and digital realignment. If realignment cannot be achieved through the hoof capsule, then shoeing to achieve a 0-degree palmar angle is mandatory (see under Realignment Process). Horses with chronic laminitic usually have normal physiologic values upon presentation; pain is their most significant issue. Horses exhibiting FSS require immediate surgery.

When making the evaluation, past history, past radiographs, the on-site radiographs and venograms, and the current physiologic status of the horse must all be taken into consideration. If the measurement protocol (see Chapter 16) is consistently used, data gathering and treatment planning will become a methodical scientific approach, instead of a plan based on guesswork.

Addressing Hoof Pain and Sepsis

After surgical or mechanical correction, the horse remains recumbent for a length of time, usually a few weeks. If the horse remains recumbent beyond that time period, this indicates that there are other issues that need to be addressed.

Often the swelling and seroma formation within the H-L zone causes painful pressure on the sensitive lamina and requires establishing an opening to allow drainage, or the hoof capsule and its sensitive structures will continue to devitalize. If serum pockets are noted on the radiographs, the most opportune place for the opening is at the dorsal white line. This can be accomplished by rasping the dorsal hoof wall-sole juncture at a 45-degree angle until the damaged tissue has been reached. A careful excision of the tissue with a White Line Loop Knife (Paul Mitchell, Pinehurst Forge, Tasmania, Australia) (Figure 18-9) while continuing at a 45-degree angle establishes drainage of the seroma. If drainage has not been successful, the seroma will break from the coronary band. This should be avoided if at all possible.

Medial sinking also causes seroma formation, coronary band rupture, hoof wall death, and subsequent contracture of the dead hoof wall. Hoof wall death and contracture cause further impingement on the blood supply as the hoof shrinks from lack of circulation. The impinging hoof wall must be removed in a similar conservative fashion.

The hoof wall should be thinned with a Dremel (Robert Bosch Tool Corporation, Racine, Wis.) or half-round nippers until the innermost white color of the internal hoof capsule is reached, then the veterinarian should feel for pliability of the hoof wall. This continues until serum starts to ooze through the pores of the hoof wall, which establishes drainage without predisposing the hoof to sloughing from loss of architecture or exposing the sensitive lamina to further infection. NOTE: Any time soft tissue is exposed on the hoof, whether at the coronary band, at the sole, or through hoof wall resection, the tissue must be properly bandaged and appropriate antibiotics administered. Bandaging should never cause restriction of blood flow (see Chapter 19 for bandaging techniques).

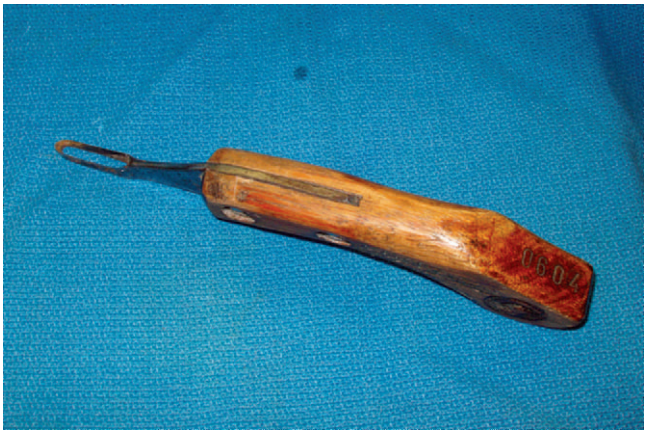

FIGURE 18-9 Photograph of a white line knife that is about the diameter of the white line. The small size of the knife allows for conservative access to seromas or abscesses.

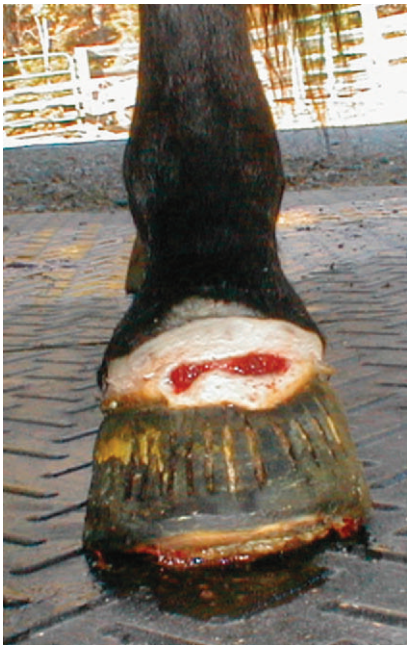

FIGURE 18-10 A coronary band rupture with a minor resection of the hoof wall distal to the rupture. This lesion is in the final stages of healing. Note the vertical grooving of the hoof capsule; the grooves were placed with a dremel to the depth of pliable tissue. This method allows for expansion of the hoof capsule.

Impingement with dead hoof wall at the open coronary band requires removal of the hoof wall just below the coronary band. In this case, full removal of the hoof wall is necessary just below the open area extending the full width of the rupture and distally for approximately an inch (Figure 18-10). This will stop the compression of the coronal cascade and dorsal circulation. Figure 18-11 shows a venogram indicating the compression of the proximal dorsal circulation and coronal cascade of the same hoof in Figure 18-10. The use of the venogram helps to determine the areas of soft tissue that

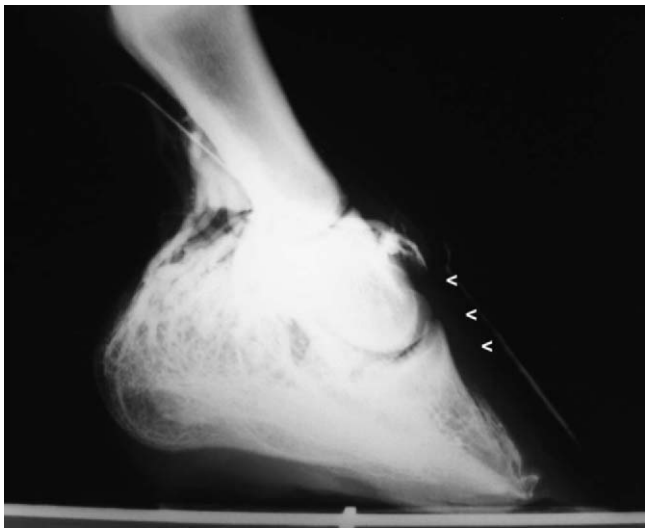

FIGURE 18-11 This venogram shows the loss of circulation in the distal coronal cascade and the proximal aspect of the dorsal circulation, causing the coronary band rupture seen in Figure 18-10.

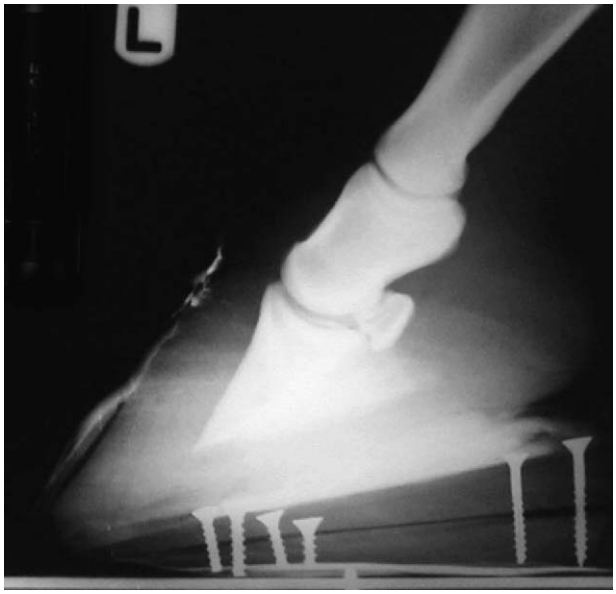

FIGURE 18-12 Radiograph showing the use of a Modified Ultimate shoe (Nanric, Lawrenceburg, Ky.). The hoof has been rasped to approximate a 0-degree palmar angle. In some cases, deep flexor tenotomy surgery can be avoided if the sole depth can be increased to 15 mm or greater in 6 weeks with elevated wedge appliances.

are internally problematic and will eventually become visible external pathologic lesions.

New hoof growth from the coronary band starts within a few weeks. Foot soaks in Epsom salts and chlorhexadine solution will keep the open wounds clean and hasten the cornification of the area. Cryotherapy with sequential compression is used on all acute cases, followed by ice wraps, which can be left on indefinitely (see Chapter 19 for more information).

In hooves that continue to deteriorate and appear septic, regional perfusions should be performed using the appropriate antibiotic. A tourniquet is placed on the fetlock after an abaxial nerve block with Carbocaine-V (Pharmacia and Upjohn Co, Kalamazoo, Mich.) (similar to the procedure for obtaining venograms; see Chapter 17). This procedure helps start the recuperative process of the internal tissues.

TREATING THE CHRONICALLY LAMINITIC (INCREASED PALMAR ANGLE) HORSE

Horses that have had repeated bouts of laminitis, but have not been presented for treatment during the acute phase, have all of the symptoms mentioned earlier, with the possible exception of acute phase coronary or solar lesions. Findings on venograms and radiographs are also similar, but P3 shows bone resorption and loss from the chronic malalignment of P3. The "slipper" appearance of the distal dorsal P3 is a result of circulatory loss and bone fragility. Microfracturing of the distal tip will lead to eventual bone loss in this area.

These cases can be divided into two types:

- Those with more than 10 mm of SDT
- Those with less than 10 mm SDT

All cases, with the possible exception of the FSS horse, have extreme deep flexor tension, which must be relieved. Digital realignment must be achieved with either of the therapies described here.

Those cases with more than 10 mm of SDT and a palmar angle of less than 15 degrees can be fitted with 10-degree wedges with rockered solar surfaces, such as the Redden Modified Ultimate shoe (Nanric, Lawrenceburg, Ky.) (Figure 18-12) in an attempt to relieve the deep flexor pull and as a consequence relieve compression of the dorsal circumflex circulation. The hoof must first be prepared by lowering the heels as much as possible to approximate a 0-degree palmar angle. A good rule of thumb when attempting to use wedges as opposed to the surgical tenotomy method is, whatever heel is removed must be added back in the form of wedges, and then a little more should be added. Follow-up radiographs should be taken at 2, 4, and 6 weeks. If the wedges are promoting sole growth, the SDT will increase to 15 mm or greater within a 6-week period. Obtaining radiographs on a biweekly basis will let the veterinarian track whether gains are being made in sole depth, or whether other options (such as tenotomy) should be sought. Digital realignment can be continued through rasping of the bearing surface of the hoof (approximating a 0-degree palmar angle each time) every 6 weeks while the horse is in the wedges. With time, the alignment and circulation should be restored and soundness regained. If soundness has not been achieved, a midcannon tenotomy is suggested.

Horses that present with less than 10 mm of sole and greater than 15 degrees of palmar angle are candidates for midcannon tenotomy because the compression of the circulatory system of the hoof is unlikely to be restored through mechanical means. NOTE: The first tenotomy should always be performed at midcannon, which allows for a low cannon. Lastly, a midpastern tenotomy should be performed in the future should the first attempts fail for any reason. Tenotomies as salvage procedures should always be accompanied by immediate digital realignment. The only reason to perform a tenotomy is to give maximum movement of the hoof

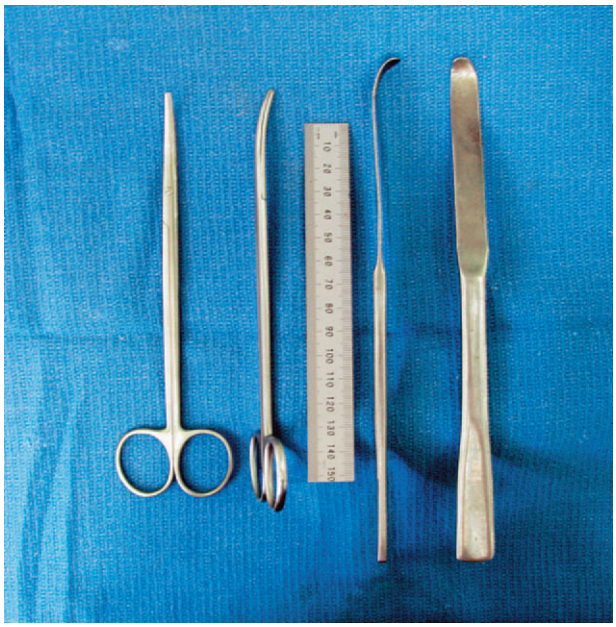

FIGURE 18-13 The most important instruments in the sterile midcannon tenotomy pack are high-quality Metzenbaum scissors and hand-made tendon blades.

capsule in the dorsopalmar (front to back) direction, thus allowing for realignment of the digital column.

The tenotomy procedure is only 1% of the cure, as it allows for the continual realignment of the digital column over a period of 6 to 12 weeks. Once the tendons heal, with proper realignment, the digital column should remain in position. This does not mean, however, that careful attention to the alignment process can be ignored in the future. The hoof, digital column, tendons, and muscles all have memory (to a lesser or greater degree) of the laminitic event and will attempt to reverse the curative process for the life of the horse in most cases. It is paramount that before the surgery, realignment, and care of any laminitic horse are undertaken, the attending farrier and veterinarian understand the needs of the hooves that confront them.

Midcannon Tenotomy for the Acute and Chronic Grade IV Case

It is worthwhile here to discuss the tenotomy procedure in some detail. The author performs this procedure on the standing, sedated horse, regardless of the tenotomy site. Laying the horse down under general anesthesia for this relatively simple procedure is unnecessary and may cause more harm as the horse struggles to regain its feet after recumbency.

Supplies Needed

Several sets of surgical instruments should be on hand that are fabricated or dedicated strictly to the tenotomy procedure (Figure 18-13). Each sterile pack should include:

- Two No. 15 scalpel blades and handles
- One Olsen-Hagar cutting suture instrument with tungsten blades
- Two thumb forceps, one being a 7 × 8-tooth Brown, the other being a 1 × 2 Adson forceps
- Two 5-inch curved hemostats to control skin hemorrhage
- Two high-quality, 8-inch, curved Metzenbaum scissors
- Two specially fabricated tendon blades
- One towel clamp
- Sterile nonwoven 3 × 3-inch gauze pads
- Two packs of 3-0 polydioxinone suture
- Powdered aerosol antibiotic
- Sterile 4-inch gauze rolls
- Sterile 12- or 14-inch combine roll (depending on the length of the leg), elastic bandage roll such as Vetrap (3M Vetrap Bandaging Tape; 3M Animal Care Products, St. Paul, Minn.) or Coflex (Andover Healthcare, Salisbury, Mass.).

Procedure

The day before the scheduled surgery, administration of antibiotics and phenylbutazone begins. Phenylbutazone is generally administered intravenously for 5 days after surgery, unless the pain continues for a longer period (see Chapter 19 for further information).

Sedation is achieved with an adequate dosage of Dormosedan (Pfizer Animal Health, New York, NY) (0.25 mg/450 kg IV) and Torbugesic (Fort Dodge Animal Health, Division of Wyeth, Fort Dodge, Iowa) (0.5 mg/450 kg IV). Repeated lower doses of Dormosedan may be necessary should the horse become restless later in the procedure.

The horse is placed on Redden Modified Ultimate wedges. Lifting the heels will take the tension off the deep flexor tendon and allow the surgical procedure to be performed with the horse standing comfortably on all four feet. Attempting to raise the leg to make the tendon more accessible may result in the horse falling while sedated.

A ring block is applied distal to the carpus with Carbocaine-V, around the proximal portion of the third metacarpal or metatarsal bone. The author prefers a ring block, as there may be aberrant nerve pathways.

An incision is made over the deep flexor tendon in the midcannon region until the fibers of the tendon are visible. Incising directly over the lateral aspect of the tendon will avoid the volar nerve that usually lies dorsal to the deep flexor. The incision length should be as small as possible; a length of 1 inch is preferred. The Metzenbaum scissors are inserted around the tendon dorsally and then palmarly (or plantarly) carefully advanced, in a closed position and hugging the tendon, until the medial aspect of the leg is reached. The jaws of the scissors are opened to separate the fascia from the tendon, and the scissors are removed, with the jaws held open. No movement should be made with the jaws of the scissors once they have been opened. Any sawing action may cut important structures. Once the tendon is free of all fascia, the tendon blades are introduced into the incision and around the tendon, one dorsal to the tendon and one palmar to the tendon between the superficial and deep tendon structures. The tendon blades will "mate"; the handles can then be opened, bringing the isolated deep flexor into view. A fresh scalpel blade and handle dedicated to the transection of the tendon is used. A sterile gloved finger can then be introduced into the incision to guarantee complete transaction of the tendon. Sterile 3-0

polydioxinone suture is used to close the incision with a blanket pattern. A powdered antibiotic such as a nitrofurazone topical powder is puffed thickly over the sutures. The incision is then covered with sterile 3 × 3-inch nonwoven gauzes, folded to create an area of light compression on both sides of the skin over the transected tendon. This is then covered with a sterile 4-inch gauze roll, followed by a sterile 12- to 14-inch combine roll and bandage material such as Vetrap or Coflex. The bandage is left in place for 3 days and then all dressings are removed and the incision site is medicated and rebandaged. Bandage changes take place every week thereafter for 3 weeks and then the sutures are removed at 21 days. A better cosmetic appearance is achieved if the sutures are left in for this length of time. Once the sutures are removed, the scar is covered daily with a lanolin-antibiotic ointment until the scar is no longer visible.

Complications Associated with the Deep Flexor Tenotomy

The following problems can occur with a deep flexor tenotomy:

- The deep flexor tendon was not severed completely, causing severe pain.
- The volar nerve was severed, causing a non–weight-bearing phase or hyperextension of the pastern and fetlock.
- The palmar vein or artery is severed, causing sloughing of the hoof capsule and gangrene.
- The basic principles of surgery, never to exceed "time, trash, or trauma," are broken. Infractions cause sepsis, adhesions, and complications greater than the original laminitic insult.

Pitfalls of the Tenotomy, Before and After Surgery

Thorough palpation of the tendons in the surgical area is strongly recommended, followed by an ultrasonographic examination of the structures involved should adhesions be a considered a possible complication.

Cases involving Quarterhorse-type horses should be evaluated as to possible negative palmar angles prior to the laminitic insult. A high, midcannon tenotomy is strongly recommended in these cases, transecting the deep flexor tendon proximal to the inferior check ligament. Performing the procedure at this level will help control the tendency toward negative palmar angle and severely crushed heels, as maintaining the inferior check ligament to deep flexor attachment will offer some stability to the digital column.

Infection at the surgical site can occur. Adhesions resulting from infections will reverse all benefits obtained from the tenotomy procedure. Performing a tenotomy without immediate realignment of the digital column will cause comfort for a short period of time but will ultimately lead to failure. Horses that are recumbent for long periods of time after the tenotomy procedure will have shortened tendons and a tendency to revert to the digital column condition they had before the realignment stage. Rehabilitation of the tendons after a deep flexor tenotomy through controlled exercise will produce less adhesion formation and a stronger tendon and will lessen the chance of tendon contracture, resulting in continued rotation of the coffin bone or P3.

Realignment Process

There are still many veterinarians and farriers who are not aware of the necessity for realignment of the digital column. There are also many who do not believe this can be achieved. Radiographic evaluation of the horse that undergoes tenotomy should be performed frequently, preferably 1 week after surgery and then every 2 weeks until the alignment remains stable. After alignment stability has been achieved, radiographs are taken at the time of each hoof trimming on a 4-week schedule.

Realigning the hoof after a tenotomy is the most important procedure for obtaining permanent healthy results. If the horse has sufficient heel length to allow manipulation of the hoof to a 0-degree palmar angle, the following description applies. It is strongly suggested that another lateral radiograph of the hoof be taken after the tenotomy procedure and before the realignment procedure, because many horses start the realignment process on their own.

- The frog should be removed to the point that a healthy appearance and depth are achieved.
- Once the frog is correctly removed, the heels will appear too far forward on the bearing surface of the foot.
- The quarters of the hoof should be located and a line made with a permanent marker just behind the quarters. This will help avoid rasping forward of this point. The heels should be rasped back to the widest point of the frog found at the bulbs of the hoof.
- The foot is put down. It will appear at this point that the toe is pointing upward and has no contact with the ground.
- Another radiograph is taken. The distal dorsal first, second, and third phalanxes (P1, P2, and P3) will all be in a straight line if sufficient heel has been removed, if not, then not enough frog has been removed to find true alignment, or rasping has gone beyond the quarters of the hoof. More heel should be removed if necessary and the hoof radiographed again.
- If exposed solar corium is present, the hoof should be wrapped as described previously to avoid further contamination.

In cases in which there is not sufficient heel length to allow realignment of the digital column, then a shoe must be applied to achieve a 0-degree palmar angle. This is performed with a flat, aluminum shoe that is shorter than the length of the hoof from toe to heel, to approximate the proper breakover of P3. The heels of the shoe should be fit tight to the heels of the hoof or extending slightly behind the heel. The quarters or middle of the shoe should fall under the middle of the distal portion of P3. The shoe is applied with a reverse wedge of sole putty material being placed with 0 degrees of putty at the heels extending forward past the apex of the frog, getting larger at the toe. The triangular, or wedge, shape is achieved with more putty at the toe than at the heel. The resulting radiograph should demonstrate a 0-degree palmar angle when measuring the space created with the sole of the horse, combined with the wedge of putty (Figure 18-14).

A close look at the post-tenotomy radiograph will allow the veterinarian to approximate visually the amount of putty that will be required in the toe region. The shoe is then pressed down into the putty firmly at the heels until very little putty is under the shoe at the heels. The extra putty is pushed into and under the shoe with the fingertips, allowing for an area the width of half of the web of the shoe to be free of putty for a firm attachment with a shoe adhesive such as Grand Circuit Pro Fast Patch (Grand Circuit Products, Freehold, NJ) or Equilox (Equilox International, Pine Island, Minn.) between the hoof and shoe. The adhesive is applied with intermittent strips of fiberglass to give a firm bond. The adhesive should be applied from the heels to the toe. The adhesive should be completely set, as should the hoof putty, before the foot is allowed to bear weight. Once the shoeing is complete, a crescent shape of hoof putty should be removed from the sole from the apex of the frog to in front of the quarters. No sole putty should be in contact with exposed soft tissue (solar corium). A radiograph should then be taken to make sure a 0-degree palmar angle is achieved (see Figure 18-14).

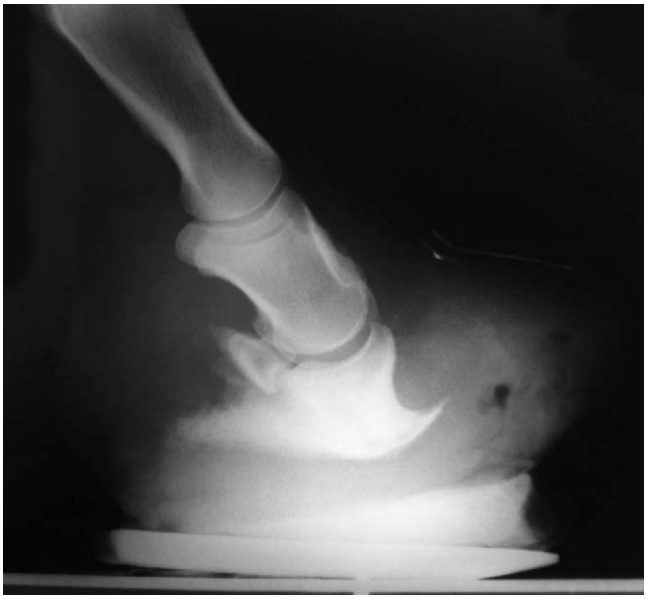

FIGURE 18-14 After the flexor tenotomy procedure, if a 0-degree palmar angle has not been achieved through manipulation of the hoof capsule, a flat shoe is glued to the hoof utilizing a reverse wedge of sole putty to achieve the correct palmar angle and digital alignment.

NOTE: A 0-degree palmar angle puts the circulation of the hoof into neutral, that is, no part of the circulation is under compression, allowing for rapid sole growth.

The shoe is reapplied as soon as it has deteriorated to the point of falling off or 4 to 6 weeks after the tenotomy procedure, if more sole depth is needed. If the horse has an exposed solar corium, it should be aseptically bandaged with appropriate antibiotics given, and the site should be cleaned and the bandage changed daily. Radiographs should be repeatedly taken to monitor the need for adjustment.

Several cases showing the realignment procedure are illustrated in Figures 18-15 to 18-17. Figure 18-15 shows the digital alignment of a grade IV laminitic case before and 4 weeks after a deep flexor tenotomy. Figure 18-16 shows a case that presented with an SDT of 33 mm, an H-L zone of 31/36, an EP-CB of 23, a positive palmar angle of 25 degrees. This case is mentioned later in the chapter. The horse had such superior quality hoof mass that no pathologic lesion ever presented externally, instead, the tendons and ligaments suffered with pitting edema and internal hemorrhage around the flexor tendons, the suspensory apparatus, and the inferior check ligaments as demonstrated by Doppler ultrasonography. Owing to the adhesions around the deep flexors in the cannon bone region, a pastern level tenotomy was performed. The measurements after the tenotomy surgery and digital realignment were SDT of 31 mm, E-CB of 13 mm, and positive palmar angle of 8 degrees. The H-L zone is irrelevant at this point, as it will be reduced following radiographic guidance over the course of several weeks. Dark areas were seen dorsal to the distal P3, which are areas of hemorrhage from compression of the distal dorsal and circumflex circulation but

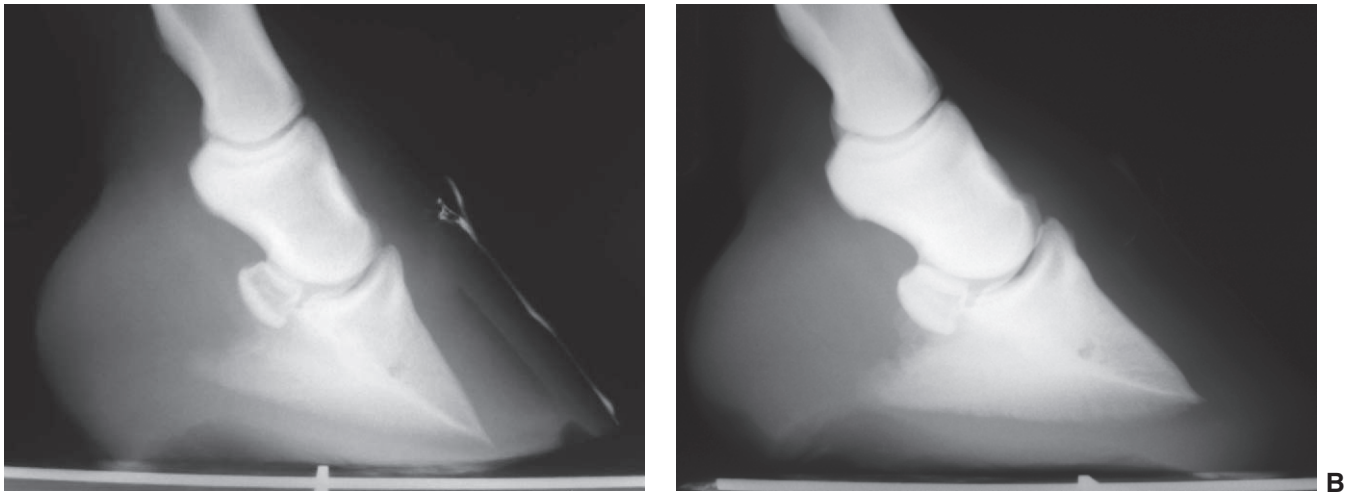

FIGURE 18-15 **A,** Radiograph of a foot taken before deep flexor surgery and digital realignment. Note the gas or seroma line in the hornlamellar zone and the decreased sole depth at the tip of the third phalanx. **B,** Radiograph of the same hoof 8 weeks after surgery and digital realignment. Note the 0-degree palmar angle.

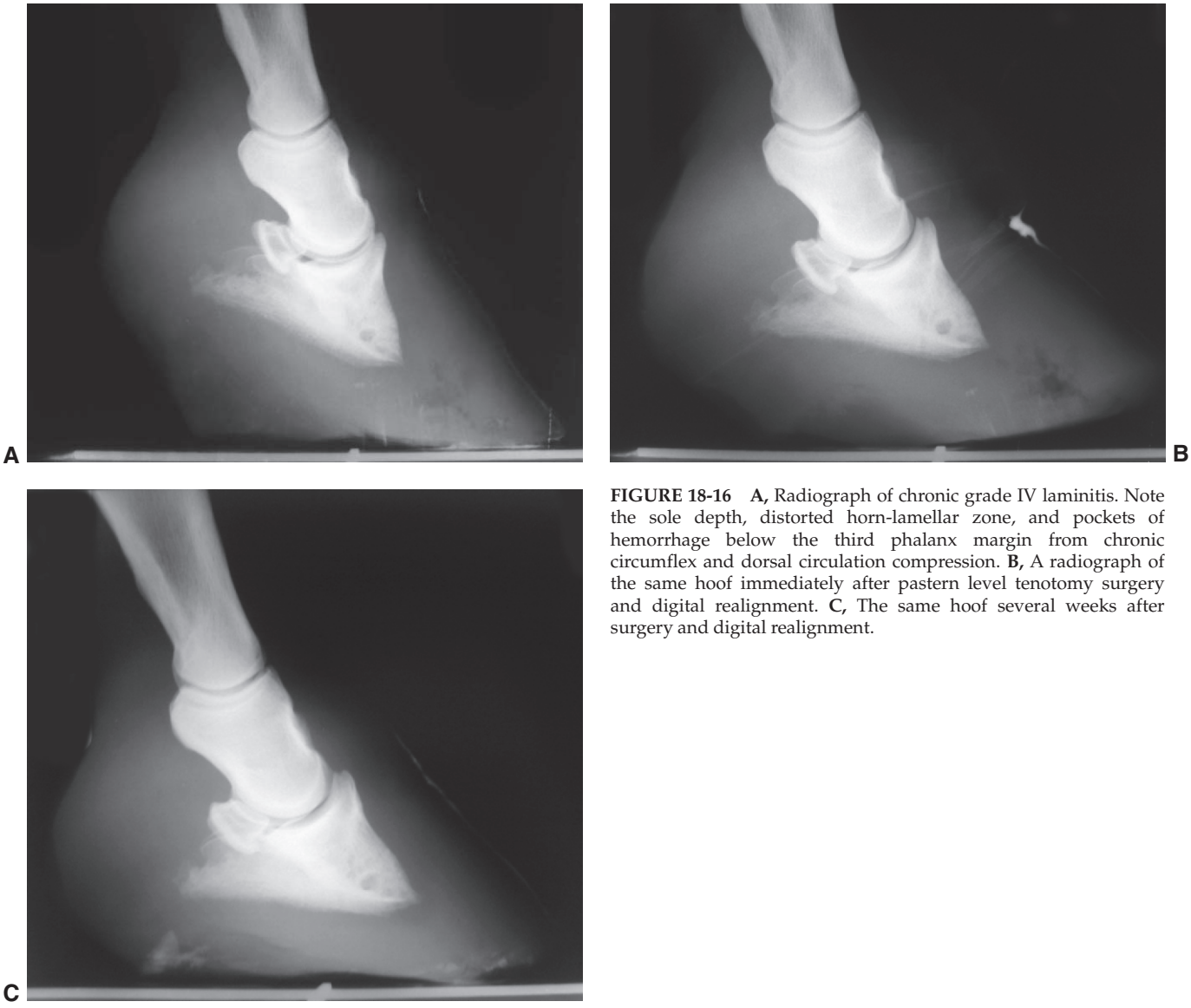

FIGURE 18-16 **A,** Radiograph of chronic grade IV laminitis. Note the sole depth, distorted horn-lamellar zone, and pockets of hemorrhage below the third phalanx margin from chronic circumflex and dorsal circulation compression. **B,** A radiograph of the same hoof immediately after pastern level tenotomy surgery and digital realignment. **C,** The same hoof several weeks after surgery and digital realignment.

have been removed through remodeling of the dorsal hoof wall and solar surface by 8 weeks after tenotomy and digital realignment.

Figure 18-17 shows radiographs of a chronic grade IV laminitic case. At the time of surgery, the gelding had an SDT of 19 mm, an H-L zone of 23/35, an EP-CB of 18 mm, and a positive palmar angle of 23 degrees. At 3 weeks after midcannon deep flexor tenotomy surgery and digital realignment, the dorsal hoof wall of the toe was still dished somewhat and the solar toe surface was still off the ground. Six weeks after deep flexor surgery and digital realignment, the solar surface was flat to the ground surface and the dish in the dorsal hoof wall was removed. The measurements at this time were SDT of 25 mm, EP-CB of 11 mm, an H-L zone of 25/26, and a positive palmar angle of 6 degrees.

The Postsurgical Rehabilitative Phase

Hand walking should commence once the horse is comfortable enough to stand for a normal length of time in the stall. Horses with fragile hoof capsules should not be walked until all lesions are in the healing (cornification) phase. Horses unwilling to walk are not ready.

Walking should start with short ambles (150 feet) once a day until strength and agility increase. Flat soft surfaces that allow for wide turns are required in the early stages. As the horse's condition improves, the distances are increased and short periods of trotting are introduced. All horses are treated as returning athletes and are braced and rebandaged after the workouts. Sequential cryocompression is frequently used before rebandaging. Water tread mills are discouraged as the only exercise mode, because they promote shortening of the shoulder muscle groups. Free exercisers such as the Euroxciser (Euro Gait-Master, Los Angeles, Calif.) are an excellent way to continue the exercise program beyond the handler's capability of hand walking and trotting. Paddock turnout is then added to the workouts. Turnouts should start on flat level surfaces and then continue on hillsides to strengthen the collateral ligaments, in a lateral and medial direction.

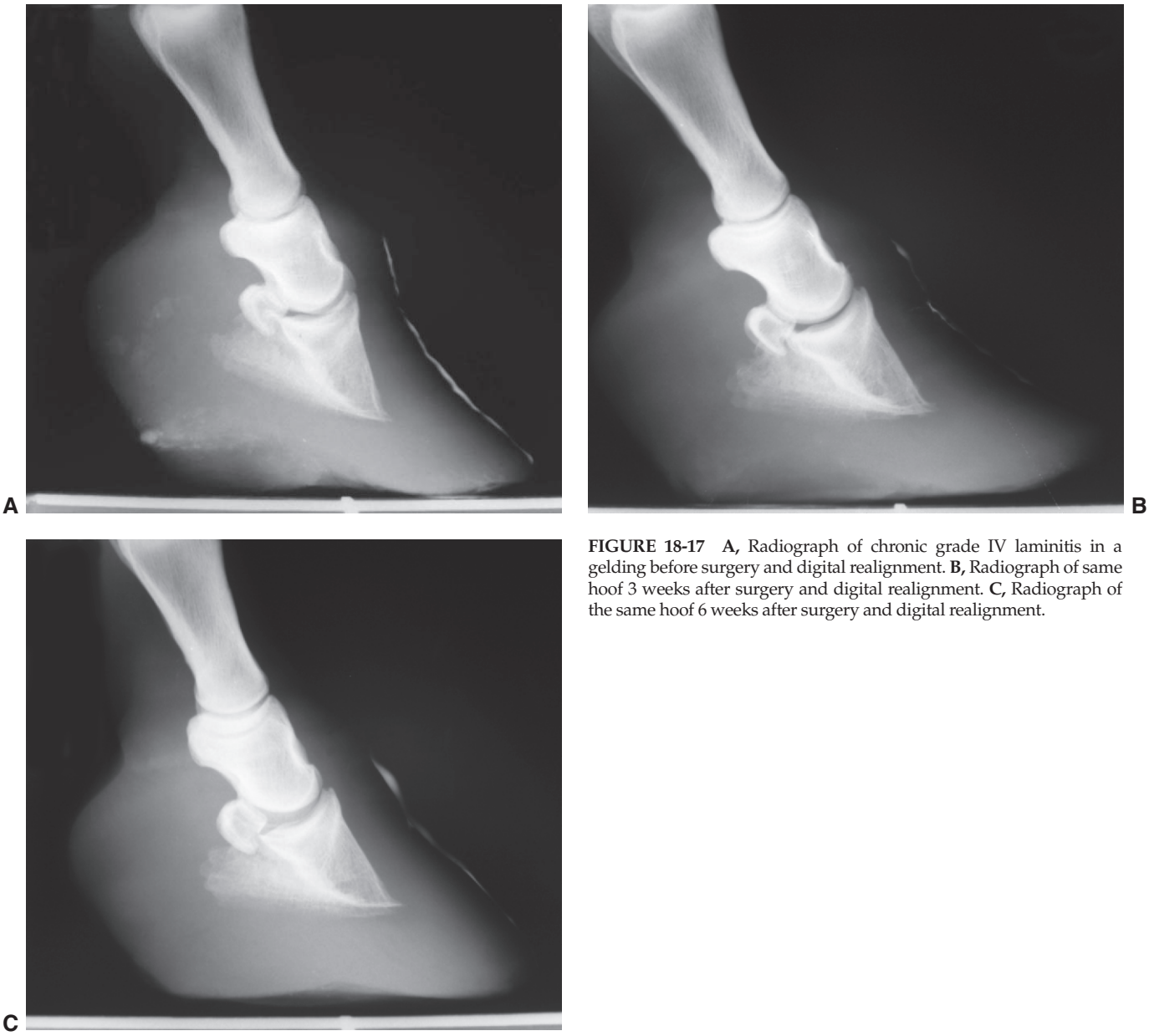

FIGURE 18-17 **A,** Radiograph of chronic grade IV laminitis in a gelding before surgery and digital realignment. **B,** Radiograph of same hoof 3 weeks after surgery and digital realignment. **C,** Radiograph of the same hoof 6 weeks after surgery and digital realignment.

THE FATAL SINKER SYNDROME HORSE

The FSS horse is the most disheartening of all cases to encounter. The majority of cases seen by the author have been in mares after septic metritis secondary to retained placenta. These mares are acutely septic and present many times too late for treatment. These cases require immediate referral to a podiatry hospital. Thrombi, emboli, disseminated intravascular coagulation, and renal failure are common after retained placenta.

The other horses at risk are those with flat, pancake feet that have lived their lives with circulatory compression of the soles. They are the horses that constantly need pads to be sound and never grow any solar depth or heels.

Symptoms

Symptoms of FSS include acute pain on presentation with a heart rate of greater than 80 to 100 beats/min, injected conjunctivas, increased blood pressure, edematous coronary bands, pasterns, and fetlocks, bounding digital pulses, an anxious expression, and a desire to remain recumbent.

Radiograph

Radiographs may have to be obtained on the recumbent horse. Using the radiographic block with the attached cassette, while holding the leg in a position approximating weight-bearing will give a fairly accurate assessment of the disease process, even while the horse is lying down. The FSS horse should never be asked to stand for any length of time.

In the FSS horse, the H-L zone measurement is increased (a finding of 25-35 mm is not unrealistic) and the proximal and distal measurements are often the same, such as 30/30 mm. The SDT and SDW (sole depth at the wing of P3) measurements are greatly reduced and close to being the same, such as 3/4 mm. The EP-CB is greatly increased. The palmar angle is reduced. The bulb to heel measurement is reduced. There may appear

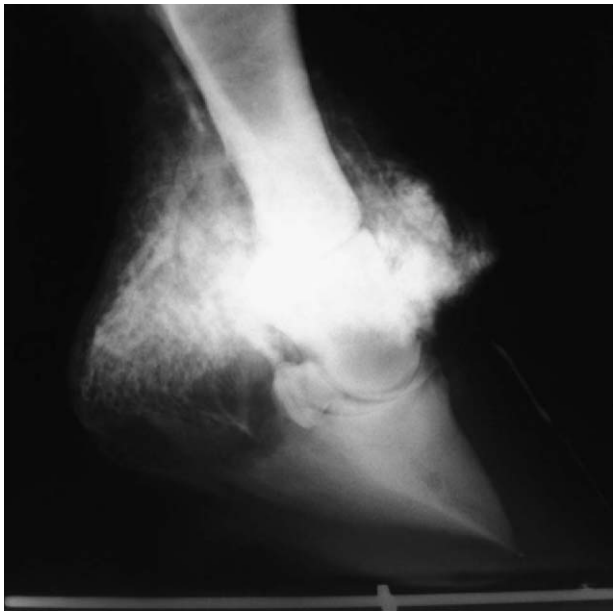

FIGURE 18-18 Venogram of a hoof showing fatal sinker syndrome.

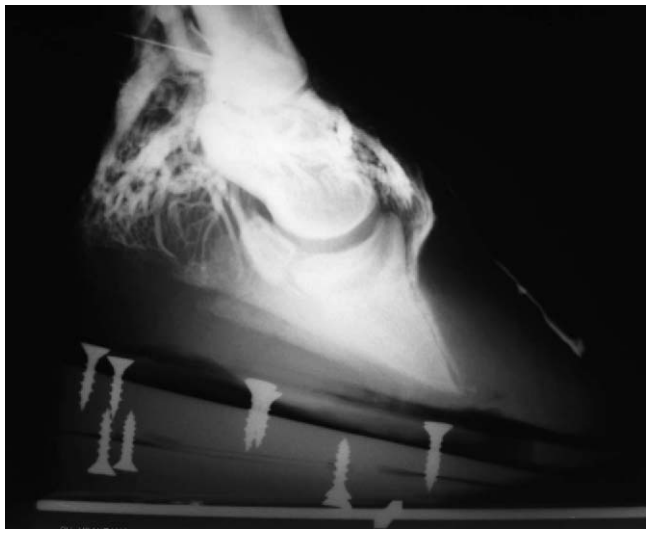

FIGURE 18-19 Venogram of horse showing fatal sinker syndrome with trace circulatory filling.

to have been a slight cranial rotation at the beginning of the disease process, as observed through a triangular gas line (lamellar wedge) or through the H-L zone.

Venogram

The venogram of the FSS horse is definitive for diagnosis. There is a complete lack of circulation distal to the coronary band (Figure 18-18), or only trace amounts of the various circulatory paths visible, as in Figure 18-19, where the dorsal circulation and coronal cascade are still visible but the circumflex artery and a portion of the bulbar circulation are absent.

Treatment

Immediate hoof wall ablation is necessary, preceded by transcortical pinning of the third metacarpal bone (Figure 18-20) and, finally, casting of the limb to allow the foot to be held in a non–weight-bearing position. The author prefers to perform this procedure on the standing, sedated horse that is in a sling.

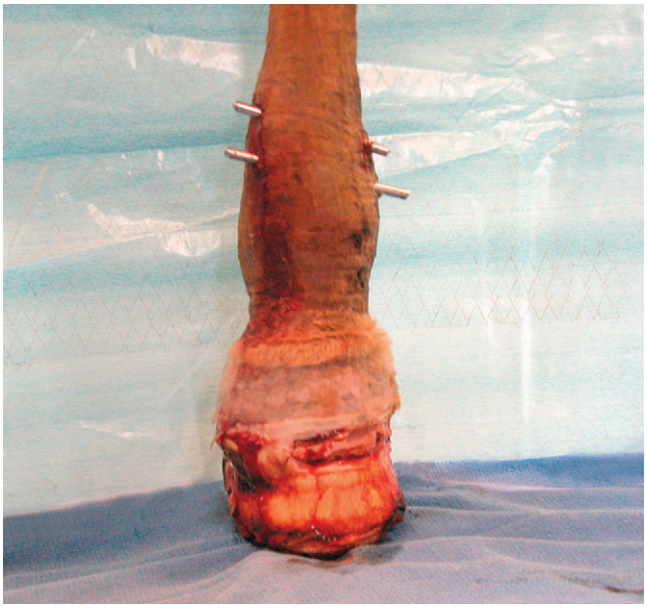

FIGURE 18-20 Photograph of horse from Figure 18-18 showing total hoof wall ablation and transcortical pinning prior to cast application. The horse was sedated and blocked, and is standing in a sling.

Additional Considerations

Some important issues involve hoof tissue quality and the original digital alignment of the column prior to the laminitic process. All equine practitioners should seriously consider incorporating yearly lateral soft-tissue hoof radiographs into their spring vaccination program. Historical data as to the quality of the hoof, sole depth, and digital alignment are extremely important to the equine podiatrist. These data provide a basis for comparison and can help the veterinarian make a treatment plan and prognosis.

Flat, thin-soled hooves with caudal rotation (negative palmar angle) will always have this tendency, as the circulation has been chronically impaired. For example, a horse with heel pain will likely not be able to withstand the application of a 10-degree wedge cuff and will not achieve any pain relief. This type of horse hurts palmarly from heel pain and dorsally from the laminitic insult. The thin-soled hoof is more likely to experience some degree of FSS or medial sinking because there is insufficient sole to keep the circumflex circulation from complete collapse due to a chronically impaired circulatory pattern.

The clubfooted laminitic horse has a larger bulbar tissue mass and an increased palmar angle, SDW, EP-CB, and H-L zone. The SDT may be insufficient to allow patent dorsal circumflex circulation. The clubfooted horse appears as a chronically laminitic horse when using the measurement protocols described here. Venograms are necessary to distinguish the

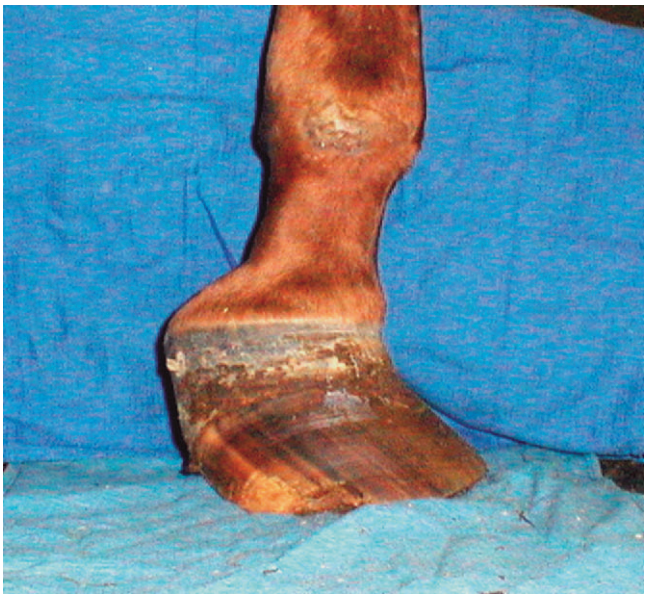

FIGURE 18-21 Photograph showing fetlock dorsiflexion due to tendon contracture from a chronic grade IV laminitic case.

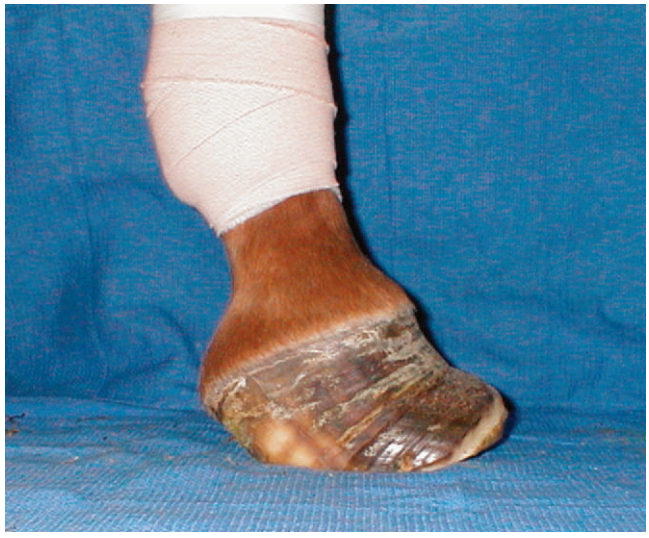

FIGURE 18-22 Photograph of the same horse as in Figure 18-21 showing correction of fetlock dorsiflexion immediately following a midcannon deep flexor tenotomy and digital realignment.

acute laminitic bout from the chronic club-footed disease process.

NOTE: The clubfooted horse has chronic muscle and tendon contracture that is very difficult to override medically or surgically. The memory of the contracture persists and the author has not found a method yet to alleviate the body's need to return to said condition. All clubfooted horses can be helped, but the clubbed foot will always carry a slightly higher palmar angle than the other foot and will have the tendency to grow heel mass faster than toe mass.

A horse with degenerative joint disease suffering from articular lesions or arthritis or having suffered the same from malalignment of the digital column from chronic laminitis has trouble regaining proper alignment. Dorsiflexion of the fetlock may be observed after an attempt at realignment. Not all cases of dorsiflexion of the fetlock are a result of degenerative joint disease; many that present with this type of problem and are chronically laminitic will return to normal alignment once the heels are reduced through realignment after the tenotomy procedure (Figures 18-21 and 18-22). The author has seen one instance of a horse that had superior quality sole (>5 mm) and hoof wall tissue that kept the acute laminitis from penetrating the sole. The severe tension of the deep flexors, however, caused tearing, hemorrhage, fibrosis, and adhesions between the deep and superficial flexor tendons and the suspensory ligaments. The inferior check ligaments were under such strain as to become quadrupled in size. The legs were enlarged, filled with pitting edema, and painful to palpation. Doppler ultrasonography revealed hemorrhage surrounding all of the aforementioned tissues.

Horses that have had severe widening of the H-L zone have a distorted dorsal horn of poor quality with a very thick white line. This condition requires continual remodeling for the rest of the horse's life. As long as the correct digital alignment and palmar angle are achieved, the horse will remain sound and the dorsal hoof wall will be seen as simply a cosmetic fix.

Many changes take place over the course of several months internally, within the hoof capsule, and externally, to the existing cornified tissues. Attention must be paid to the continual battle of returning the hoof and the digital column to a normal anatomy, condition, and usefulness.

Constant supervision and observation of the recovering laminitic horse in a hospital setting gives the best chance of recovery and return to soundness.

19

ENVIRONMENTAL MANAGEMENT OF THE SEVERELY LAMINITIC HORSE

ANDREA E. FLOYD

GOALS AND EXPECTATIONS

Managing the recovery of the severely laminitic horse (grade III or IV) is a long-term process that requires an extensive knowledge of digital biomechanics, intensive medical therapy, common sense, and imaginative manipulation of the psychological well-being of the individual.

Horses with hoof disease that causes such a degree of pain that they are unable to stand for long periods of time inevitably develop serious musculoskeletal injury from prolonged recumbency. The most obvious issue to be addressed, then, is how to get these horses back on their feet as soon as possible. Pain management with antiinflammatory medications has its limitations, as high dosages of these drugs can cause or aggravate gastric ulcers and kidney damage. Biomechanical adjustment of the digits, as described in Chapter XXX, therefore is the key element in the management of severely laminitic horses.

It is important to note that even the properly managed laminitic digit will remain painful during the immediate recovery period. However, it is rare for these horses to remain recumbent for longer than 2 months after appropriate biomechanical adjustment of the digits. Horses that do not begin standing within this timeframe require further evaluation for one or more of the following scenarios: (1) the biomechanical needs of the digits have not been adequately met, (2) the horse has experienced so much muscle atrophy that it is now too weak to stand, and (3) the horse has other health or hoof issues that have not been identified or addressed.

Provided that blood work has shown that the horse does not have gastrointestinal, renal, or liver issues, and the biomechanical needs of the digits have been properly addressed, it is likely that the recumbent horse is unwilling to rise on its own to seek food and water and to defecate and urinate because of its unstable (yet recovering) hoof capsules. In that case, ways must be sought to make the recumbent horse comfortable while the hoof heals.

The goal is to keep the horse as healthy as possible during the recumbent phase. There are several issues that need to be addressed in the management of these horses. Each is discussed separately below.

STALL

When housing a severely laminitic horse during the recovery period, the factors that need to be considered with regard to the stall include stall size, wall covering, flooring, lighting, rigging, climate control, and patient visibility. (Bedding is discussed in the following section.)

Stall Size and Wall Covering

Laminitic horses are uncoordinated and need extra space when attempting to rise. The minimum dimensions for a suitable box stall are 16 × 16 feet, which is larger than the average stall.

The walls should consist of a sturdy but somewhat flexible material that will not cause injury to the horse should it fall or bounce against the wall while struggling to rise. One good option is a wall built of $^{3}/_{4}$-inch tongue-and-groove composite board, covered with $^{1}/_{4}$-inch siding Styrofoam and overlaid with stainless steel sheets (22- to 18-gauge, 4 × 8-foot sheets). This type of wall provides an easy-to-disinfect, maintenance-free surface that is shockproof should the horse strike it or fall against it (Figure 19-1).

Flooring

Floor construction that makes use of natural drainage through limestone gravel is ideal, as it aids in disinfection and helps keep bacterial numbers low in the immediate environment. When constructing stalls that may be used for the long-term care of recumbent patients, large sump holes (4 × 4 feet) can be incorporated that are drilled into the ground and filled with heavy gravel. Smaller gravel is then spread 6 inches deep across the entire stall and overlaid with a 12-inch–deep layer of gravel sand. Rubber mats ($^{3}/_{4}$-inch thick) are laid on top of the sand for a long-lasting floor. Ideally, the surface of the rubber mats lies 12 inches below the level of the hall floor, allowing a thick layer of appropriate bedding material to be laid on top (see p. 362).

Lighting

Incandescent light should be provided in all stalls. Incandescent light is preferable to fluorescent light because it does not distort mucous membrane color the way fluorescent lighting can. Placing light fixtures on all walls, just below the ceiling, allows good illumination for examination and treatment of the horse at any time of the day or night. Provided at least 400 watts of light (e.g., four 100-watt light bulbs) are used, incandescent light supplies sufficient candle power to approximate moderate daylight. Using waterproof light fixtures facilitates thorough power washing of the stall between patients (Figure 19-2).

FIGURE 19-1 This stall wall is easy to disinfect, maintenance free, and safe for the horse.

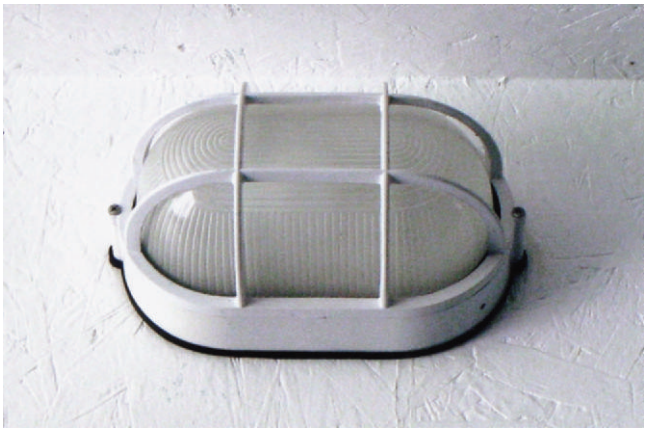

FIGURE 19-2 Waterproof, caged light fixtures that use incandescent lamps are best.

Rigging

The stall rigging includes permanent fixtures such as anchor rings for tying the horse and hooks for hanging bags of intravenous (IV) fluids. Anchor rings should be spaced around the stall wall about 5 feet above the floor so that a lead rope can be run through the ring to assist the horse to stand if necessary (Figure 19-3). Permanent rigging for IV fluids should also be available. A pulley-and-rope system that can be lowered and raised in the center of the stall by an attendant outside the stall is ideal (Figure 19-4). An overhead rail or hook for supporting a horse in a sling also is invaluable. Although not considered stall rigging per se, bars or rails at the horse's hip height, placed at strategic locations in the stall,

FIGURE 19-3 Anchor rings in the stall wall can be used to assist a recumbent horse to stand.

allow the horse to stand with its hind end partially supported should it choose (Figure 19-5).

Climate Control

Keeping the temperature in the stall within a comfortable range is essential for stress management in recumbent horses with severe laminitis. These pain-ridden horses have a higher body temperature than normal and they may sweat profusely, especially on the down side. These horses develop more decubital ulcers and generally appear more stressed unless the temperature in the stall is kept below 16° C (60° F), ideally closer to 4° C (40° F).

Unless ambient conditions are sufficiently cool, air-conditioning should be provided to keep the temperature in the stall within this range. In planning suitable accommodations for these horses, the stalls should be custom-designed with separate cooling systems that have removable return filters that can be changed daily and individual dehumidifying systems. Having zone controls so that individual stalls can be cooled as needed keeps maintenance and electricity costs down.

Patient Visibility

It is essential that the entire horse be readily visible to the hospital staff during the early recovery (i.e., intensive care) phase. Later in the horse's recovery, such easy visibility allows casual observation of the horse as the staff pass by while engaged in other routine activities. The use of heavy metal wire stall fronts that run from floor to ceiling answers this requirement (Figure 19-6).

It is also highly recommended that a video camera system that transmits both image and sound in real time be installed, with a camera in each stall, so that each horse can be monitored from a central area. This system allows observation of the horse without human interference, as these horses often present different symptoms during the early recovery phase when viewed undisturbed. It also allows close monitoring during a medical crisis and immediate backup should a staff member be working with a horse and require assistance.

FIGURE 19-4 Intravenous fluid bags can be raised or lowered from outside the stall using this simple pulley system.

FIGURE 19-5 Rump rails often are used by laminitic horses to help support their weight while standing.

FIGURE 19-6 Full-length, heavy-duty mesh stall fronts allow an unobstructed view of the whole horse, including its feet.

BEDDING

The stall bedding must be carefully chosen and meticulously tended to limit moisture- and pressure-induced damage to the skin and underlying musculoskeletal structures in recumbent horses. The tissues most prone to damage are those overlying the tuber coxae, greater trochanter of the femur, lateral aspect of the stifle, mid rib cage, and lateral aspect of the first two cervical vertebrae. The lateral aspects of the elbow, hock, carpus, fetlock, coronary band, and facial bones also are easily abraded and denuded of flesh if not properly protected.

Many types of bedding have been used for recumbent horses, including sawdust, shavings, straw, hay, Styrofoam peanuts, peat moss, sand, and shredded newspaper. A suitable bedding material for this purpose has the following properties:

- Provides adequate padding for the bony prominences (e.g., tuber coxae)
- Inhibits bacterial proliferation (or at least does not promote bacterial growth)
- Is dry and sufficiently absorbent that it wicks away sweat, urine, and fecal moisture from the horse's skin
- Does not attract flies
- Is not so bulky that it makes walking in the stall difficult
- Is not so easily moved that it allows the recumbent body or legs to contact the stall floor as the horse shifts about

The specific bedding material that is most appropriate in any given circumstance depends, to a large extent, on the ambulatory status of the horse. If the horse is able to stand, it may need nothing more than a 12- to 18-inch–deep bed of sawdust. In horses that are recumbent for at least 12 hours a day, a mixture of sawdust and soft shavings is preferred. This combination of materials gives loft to the bed and prevents decubital ulcers in the moderately ambulatory horse. If skin abrasion or the onset of a decubital ulcer is noticed, the bedding should be changed to "intensive care bedding."

Intensive Care Bedding

Intensive care bedding consists of an absorbent underlayer of sawdust or shavings (6 to 12 inches deep) and a top layer of soft barley or wheat straw (24 inches deep) that is laid down in a thatched pattern (Figure 19-7). Sawdust generally does not shift under the horse, and thatching the straw helps keep it from shifting.

The recumbent horse moves very little and its body weight causes the bedding to compact, creating areas of pressure on the body. For this reason, the horse should be encouraged to rise several times a day (at least every 4 hours) so that the straw can be rethatched and wet spots replaced with clean, dry bedding. This approach also allows blood flow to be restored to the pressure points on the down side of the horse's body. The horse should then be assisted and encouraged to lie down on the opposite side of the body, if possible. (NOTE: The laminitic horse will preferentially lie on the side with the least painful digit, as the down limb receives the most stress upon rising.)

Use of sawdust or sand as the uppermost layer of bedding for horses that are recumbent most or all of the time causes decubital ulcers, as these materials are abrasive. Peat moss is not ideal for these horses, as it may allow bacterial proliferation and thus contamination of surface wounds and it can have a negative effect on the respiratory tract. Styrofoam peanuts also are unacceptable for this purpose, as they "float" out from underneath the horse and do not provide adequate muscular support or absorbency.

FIGURE 19-7 "Intensive care bedding" for recumbent patients consists of a deep underlayer of sawdust or shavings and a thick top layer of straw, laid down in a thatched pattern (*inset*) to prevent it from shifting under the horse.

Shredded newspaper, when used in sufficient quantity, is somewhat disinfectant; however, it is difficult to find an adequate supply of this material, and when wet it compacts and adheres to the body. Straw is far superior to all other bedding materials for cushioning, absorbency, skin protection, and muscular support. However, it attracts flies, especially when soiled, so use of straw requires extra vigilance to prevent problems with flies.

FEEDING

The belief that caloric intake should be reduced in laminitic horses often proves to be erroneous, particularly in horses with severe laminitis. Any sick horse needs sufficient calories and other nutrients to meet its basic metabolic requirements and the increased demands for tissue repair. The specific nutritional requirements for the individual patient thus depend, in part, on the severity of the laminitic event, in other words, the severity of tissue damage, debility, and associated catabolism.

The diet for the laminitic horse must be tailored to the metabolic needs of the individual patient, factoring in total calories, protein, fats, carbohydrates, minerals, vitamins, electrolytes, and water requirements for the disease severity and stage of the recovery process. Even though horses with grade III or IV laminitis are recumbent most of the time, their energy and protein requirements are higher than those of a fully mobile horse with grade I or II laminitis, because these severely pain-ridden, debilitated horses are in a markedly catabolic state. Muscle mass is lost quickly in these horses, owing to the combined effects of pain, stress, inappetance, and prolonged recumbency. Extra calories in the form of fat and addition of a high-quality protein source are required to meet the nutritional needs of these horses and help reverse the weight loss.

Feeding the Recumbent Horse

Horses that remain recumbent must be hand-fed. A balanced diet that consists of 14% to 16% crude protein and provides

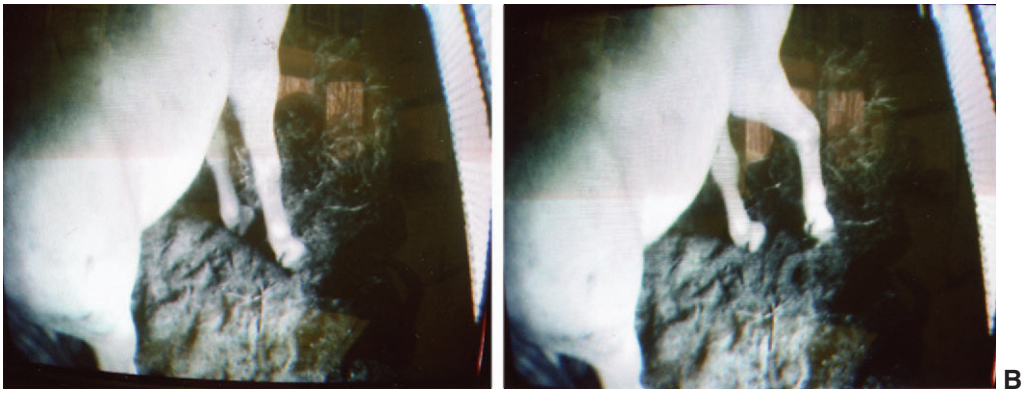

FIGURE 19-8 Video images of a horse exhibiting foot pumping. (Video cameras allow observation of the horse without altering its behavior in response to the presence of a human.)

extra calories in the form of fat (e.g., balanced rice bran or vegetable oil) is recommended. Liquid vitamins and electrolytes can be added to the basic ration as needed. Beet pulp, linseed meal, flaxseed, and kelp also are good additives for meeting the nutritional needs of these horses.

The meal should be fed as a warm gruel, with molasses, water, and salt added. Extra salt, up to 3 tablespoons per meal, increases water consumption. Because recumbent horses do not move about enough to keep the large colon functioning well, increasing the horse's water consumption helps soften the stool and prevent impaction colic. The horse should be hand-fed this gruel meal four times per day. The horse will drink the gruel and, as more water is added, the entire meal is consumed as a drink.

High-quality, appealing hay should be kept near the horse's head (i.e., within reach) at all times. Fresh, clean water is offered at least eight times per day, during the daytime and at night. Carrots, apples, fresh grass, blackberry leaves, plantain, blue lettuce, chickweed, clover (in small quantities), and any other nontoxic local pasture herbs that are in the usual grazing habitat of the horse should also be offered during the day. Interestingly, the roots of fresh grass and the attached clods of dirt are attractive to these horses and perhaps fill a particular nutritional need.

Between the gruel feedings, various grains are provided in tubs placed near the horse's head for free-choice consumption during the day. Varying the choice helps keep the horse's interest and appetite piqued.

PAIN MANAGEMENT

Horses with grade III or IV laminitis are in a significant amount of pain. With experience, it becomes possible to determine fairly accurately whether the manifestations of pain in a particular horse are associated with improvement or deterioration in the horse's condition.

Pain as a Diagnostic Tool

An ambulatory horse will show the observer not only which foot hurts the most at that point in time but also which portion of the foot hurts the most. The horse tries to unload the most painful part of the foot, so toe bearing signifies heel pain, heel bearing signifies toe pain, and "tight-rope walking" (in which the lateral quarters alone are weight-bearing) signifies pain at the medial quarters. A sudden change in the weight-bearing pattern of a recovering patient should prompt careful inspection of the foot, repeated radiographs and venograms, and, if warranted, digital derotation by further minor adjustments to the hoof capsule.

Gauging Pain Severity

Other observations that help determine the severity of pain the patient is experiencing include foot pumping (in ambulatory patients), body temperature, heart rate, and mucous membrane color.

Foot Pumping

An ambulatory horse will attempt to unload the painful areas of the hoof by "foot pumping"—picking up the foot and putting it down, then doing the same with the opposite foot, repeating this process ad infinitum (Figure 19-8). Presumably, foot pumping provides some relief to the damaged tissues and the digital circulation, helping to restore blood flow in the affected areas while the foot is unloaded. Counting the number of seconds between pumps is useful in determining the degree of discomfort and whether the condition is improving or deteriorating. At the start of the recovery phase, after digital derotation or surgery, the time between pumps is only a few seconds. As the tissue heals, the pumping becomes progressively less frequent, until finally the horse is able to stand without pumping the affected feet.

Body Temperature

In horses experiencing severe pain, the body temperature may be elevated by as much as 1.1° C (2° F) above normal. Care must be taken to differentiate between stress-induced hyperthermia and disease-related pyrexia. This distinction can be difficult to make clinically, especially in the recumbent horse that is developing septic myositis (see p. 367) and occult decubital abscessation.

Heart Rate

The heart rate (HR) is a good indicator of pain severity. The HR should be measured at the time of admission, while the horse is standing, and again several hours later when

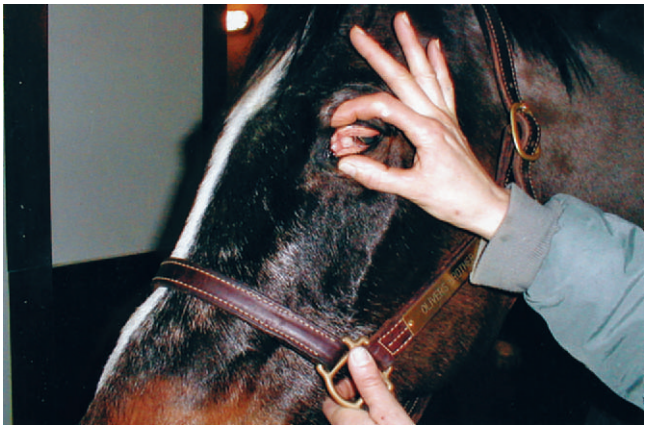

FIGURE 19-9 The color of the conjunctiva is useful in determining pain severity. The membranes become more injected as the heart rate and blood pressure increase in response to severe pain.

the horse is recumbent. Typically, the HR is 20 to 30 beats/min higher in the acutely pain-ridden, standing horse than when that same horse is recumbent. A horse with grade I or II laminitis typically has a standing HR of 60 beats/min and a recumbent HR of 40 beats/min. A horse with grade III or IV laminitis may have a standing HR of 80 to 100 beats/min and a recumbent HR of 60 to 80 beats/min.

Mucous Membrane Color

The color of the mucous membranes (e.g., the conjunctiva) can also be useful in determining the severity of pain (Figure 19-9). In the normal horse, the conjunctiva is light pink, and on close inspection the small vessels are barely visible. With the elevation in heart rate and blood pressure that accompanies moderate pain (HR approximately 60 beats/min), the conjunctiva becomes a darker pink; in horses in severe pain (HR 80 to 100 beats/min), the conjunctiva becomes quite red.

Medical Management of Pain

Nonsteroidal Antiinflammatory Drugs

Nonsteroidal antiinflammatory drugs (NSAIDs) are the most widely used form of pain management in laminitic horses. After surgery and digital derotation, the use of NSAIDs for 5 to 7 days at moderate dosages (e.g., phenylbutazone at 1 to 2 grams PO or IV q12h for a 1000 lb [450 kg] horse) is acceptable. However, use of these drugs interferes with monitoring of the horse's progress after surgery, particularly the type and degree of pain, so continued use of NSAIDs is not recommended.

Horses with disease-related pyrexia can be medicated with a low dose of flunixin meglumine or phenylbutazone at a dosage sufficient to keep the body temperature below 39° C (102° F). Of course, appropriate antibiotic therapy is also given in these cases. Whenever NSAIDs must be given for more than 1 week at a time, it is strongly recommended that gastric mucosal protectants such as cimetidine, ranitidine, or omeprazole (Ulcergard or Gastrogard; Merial, Athens, Ga.) be used concurrently.

Acepromazine

Acepromazine malate can be a useful medication in severely pain-ridden, laminitic horses. It has no analgesic properties, but it increases digital blood flow and helps relieve the muscle spasms that contribute to the bodywide pain these horses experience from continually trying to unload their most painful limbs. Acepromazine also reduces the degree of anxiety these horses experience. The injectable form of acepromazine can be given orally at a dose of 40 mg (2 mL of a 20 mg/mL solution) for a 1000-pound horse, and repeated every 6 hours (40 mg PO qid) in the acute phase and, if needed, every 12 hours (40 mg PO bid) later in the recovery phase.

Other Medications

Many other medications can be used for pain management in severely laminitic horses. They include the less commonly used NSAIDs (e.g., ketoprofen, aspirin), alpha-2 receptor agonist tranquilizers (xylazine, detomidine), opiates (e.g., butorphanol, morphine, fentanyl), topical nitroglycerine (e.g., transdermal patches), and lidocaine drips (i.e., constant-rate infusion). The method that is generating much interest and some research currently is the use of fentanyl (Duragesic) transdermal patches. However, the author has not found them to be of benefit in severely laminitic horses.

Nerve Blocks

Nerve blocks have been suggested for short-term pain relief in laminitic horses. However, if the horse's pain is not controlled with NSAIDs at the start of the recovery phase, then the problems in the digit have not been completely addressed and the situation is a deteriorating one that needs immediate investigation. Without thorough evaluation and appropriate management, these cases inevitably rapidly decline, leaving the veterinarian with a recumbent grade IV laminitic horse.

BANDAGING

In horses with severe laminitis, there are numerous instances in which it is appropriate to apply some sort of bandage to the digit or lower limb. Regardless of the particular reasons or methods, there is one basic rule that must always be followed: pad and protect without causing vasoconstriction.

The most appropriate bandage materials and method will depend on what the treating veterinarian is trying to achieve. The bandage may be required to meet one or more of the following needs:

- Pad tissues exposed through rupture or invasion of the hoof capsule or skin
- Protect those tissues from the environment (in particular, prevent bacterial invasion) during granulation and cornification
- Keep topical medications in place
- Support the tendons and ligaments of the lower limb

Before discussing the specific bandaging techniques for ambulatory horses and recumbent horses, it is important to make two comments about changing the bandages on horses with severe laminitis: (1) a recumbent horse should never be asked to stand for a bandage change; and (2) a standing horse with bilateral laminitis should never be asked to lift a painful foot and bear all its weight on the other painful foot, because this will cause further deterioration in the loaded foot. Learning to bandage the affected limbs on a recumbent horse is thus of paramount importance.

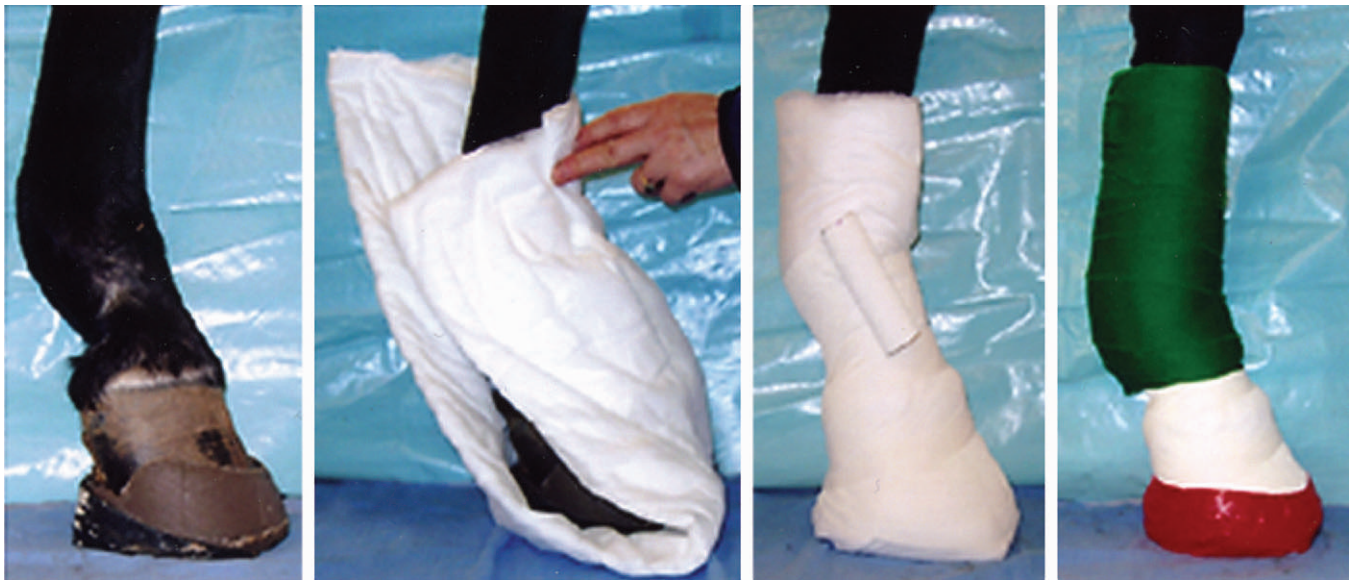

FIGURE 19-10 The Redden Ultimate Wedge (Nanric, Versailles, Ky.) is bandaged in place with a generous layer of cotton padding.

Bandaging with Redden Ultimate Wedges

The Redden Ultimate Wedge (RUW; Nanric, Versailles, Ky.) is one of the most important advances in acute care of the severely laminitic horse. However, proper application of the RUW to the damaged digit is of extreme importance. The digital vasculature must at no time be constricted or compromised by the bandage.

A simple technique using a thick layer of roll cotton (e.g., Combine dressing; Franklin-Williams, Lexington, Ky.) and an adhesive (e.g., Elastikon [Johnson & Johnson, New Brunswick, NJ]) or self-adhesive (e.g., Vetrap [3M Animal Care Products, St. Paul, Minn.], Coflex [Andover Healthcare, Salisbury, Mass.]) bandage provides a secure means of attaching the RUW without causing vascular compromise. This bandage can also be extended up to the level of the carpus or tarsus, thus providing extra support to the lower limb.

The Combine dressing is cut to length—long enough to run from the base of the carpus/tarsus, down the front of the leg, under the foot (with the RUW in place), and up the back of the leg to the base of the carpus/tarsus—and applied to the leg, being overlapped down the length of the cannon to form a double layer of protection for the cannon bone and tendons. The bandage is started midcannon and wrapped distally to include the bottom of the RUW and then rolled proximally until it reaches the top of the Combine dressing. The bearing surface of the RUW, now covered with Combine and bandage, can be covered with duct tape for extra protection and durability (Figure 19-10).

The RUW is recommended for all acute laminitis cases. Laminitic horses that are going to be transported to a referral facility particularly benefit from this system, as it relieves tension in the deep digital flexor and thus further rotation of the third phalanx.

Support Wraps

Distal Limb

Support wraps on the lower limb should be applied to all four legs in horses with severe laminitis. In recumbent horses, the lateral aspects of the cannons, fetlocks, and pasterns are prone to abrasion from the stall floor, and a support wrap protects these areas. Also, laminitic horses place abnormal strain on the entire suspensory apparatus of the limb, particularly the lower limb (extensor and flexor tendons, suspensory ligament, collateral ligaments), and support wraps are invaluable in providing support to these structures. Five-inch nylon leg wraps used with No-Bow (Wrangler, Greensboro, NC) leg cottons are superior in their ability to stay in place, remain free of bedding, and prevent constriction (Figure 19-11).

Proximal Limb

Horses that are recumbent most or all of the time often develop abrasions on the lateral aspect of the elbow and hock. It is difficult to suitably protect the elbow area with a bandage. The hock, however, can be protected with a bandage. Medication is first applied to the hock wound, then a Combine dressing is taped in place with 4-inch Elastikon. A sheep's wool spider bandage is placed over the Combine wrap and tied in place. Another layer of Combine and adhesive bandage is then applied. This protective hock bandage remains in place for a longer time if it is securely incorporated into the support wrap on the lower limb.

Bandaging after Tenotomy

After deep digital flexor tenotomy and digital derotation, a well-padded support wrap (e.g., Combine and 4-inch adhesive bandage) should be applied to the cannon, incorporating a sterile dressing over the surgical site, until the incision has healed. The support wrap should be maintained until the skin sutures or staples are removed, the wrap being changed as often as necessary during that time. (NOTE: Leaving the sutures in for 14 days and staples for 21 days is recommended. Removal of the sutures or staples any earlier can result in an unattractive surgical scar.)

Powdered nitrofurazone can be applied liberally over the incision at each bandage change. Not only does the antibiotic powder absorb any serous ooze from the incision, but it also appears to promote tissue repair and thus minimizes scarring

FIGURE 19-11 A 5-inch nylon leg wrap over a No-Bow (Wrangler, Greensboro, NC) leg quilt provides good protection and support to the lower limb.

at the site. Unless there is a clear indication to remove the bandage sooner, the surgical wrap should be changed for the first time 3 days after surgery, and then weekly until the sutures are removed. Thereafter, the legs should be wrapped with a support bandage that consists of a No-Bow wrap and nylon stall bandages.

Should the fetlock angle decrease (i.e., the pastern appear more upright than normal) at any point postoperatively, bandaging should cease immediately. This change in fetlock angle may be caused by excessive fibrosis and contracture at the tenotomy site. In these cases, leaving the leg unwrapped and initiating light hand-walking usually halts or slows this process.

Bandaging for Acute Coronary Band Rupture

Coronary band (CB) rupture is a common occurrence in horses with grade III or IV laminitis. These cases require comprehensive medical management (see Chapter 18), which includes application of topical medication and a padded wrap to protect the ruptured coronary band from contamination and external trauma while it heals.

It is crucial to make sure the bandage never constricts the digital circulation, particularly the coronary circulation, while providing adequate protection to the germinal lamellar beds. One recommendation is a bandage that consists of a length of Combine dressing, cut into 4-inch–wide strips that are long enough to encircle the entire coronary band twice and held in place with 4-inch Elastikon. The Elastikon is applied over the Combine dressing and extended above (on the pastern) and below (on the hoof wall). Care is taken to make sure the Elastikon is adhered loosely to the pastern and hoof wall (Figure 19-12).

Bandaging for Acute Solar Prolapse

As in the case of coronary band rupture, bandaging the exposed solar corium provides a clean environment for healing, a means of keeping topical medications in place, and padding for the exposed sensitive structures. A length of 12-inch–wide Combine dressing can be used that is long enough to extend from the dorsal to the palmar/plantar aspect of the pastern, under the foot. First, a sterile nonstick (e.g., Telfa; Tyco Healthcare, Princeton, NJ) or gauze pad, with sterile antibiotic applied, is placed over the exposed tissue. The medicated pad is then covered with the Combine dressing and the entire digit is wrapped with an adhesive bandage, with care taken to avoid constriction of the digital circulation, particularly over the coronary band.

Hoof Socks for Cornification Phase

Once cornification of the damaged sole or coronary band has taken place, there may still be gaps of exposed tissue between the areas of new and old horn, particularly at the coronary band. An ointment based on organic lanolin can be applied over the new horn until the gaps between the tissue layers are filled in. At this point, the new tissue needs to be exposed to the air; the emollient ointment keeps the new tissue at the coronary band from drying out excessively.

An easy and inexpensive way to allow air to the new horn, while still providing some protection from the environment, is to take two athletic socks (men's extra-large), cut them in half lengthwise, and then sew the two socks together to form a single sock that is large enough to fit over the hoof and extend up over the stall bandage (Figure 19-13). The hoof sock is held in place by securing the top to the stall bandage using 1-inch wide adhesive tape (i.e., "white tape"). This protocol has the added advantage of keeping the stall wraps clean.

DECUBITAL ULCERS

Decubital ulcers are a fact of life when managing chronically recumbent horses, such as those with severe laminitis. The best way to treat these lesions is to get the horse standing as soon as possible. However, this is not always possible in horses with an unstable hoof capsule (e.g., acute cases of severe laminitis), particularly if the case is not amenable to transfixation pinning and hoof wall ablation (see Chapter 16).

Sweat, urine, feces, and prolonged compression under the weight of the body all predispose to decubital ulcer formation and also hamper treatment efforts. Environmental management is extremely important in minimizing the severity of these ulcers and in aiding healing. As mentioned earlier, the recumbent horse requires air conditioning in warm weather to prevent excessive sweating. Keeping a deep bed of clean, dry shavings and straw under the horse also is crucial.

Topical Treatments

The temporarily recumbent horse may develop shallow ulcers that involve the epidermis but do not penetrate the dermis. These lesions can be treated with any wound cream or aloe vera gel, as long as the chosen medication does not allow the bedding to adhere and further aggravate the skin.

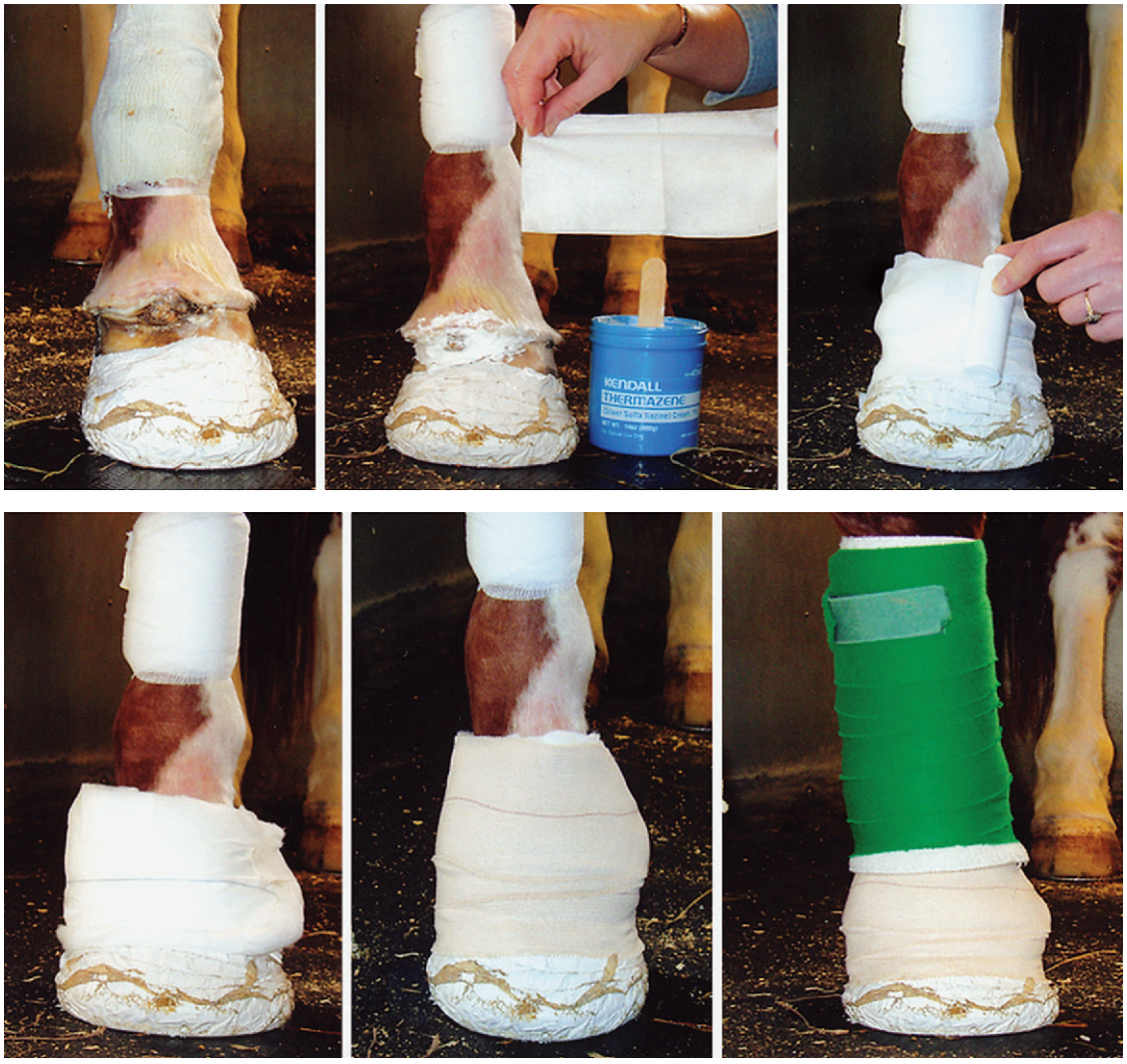

FIGURE 19-12 When bandaging the ruptured coronary band, it is essential that the digital vasculature not be constricted by the wrap.

With repeated irritation and persistent body pressure, these ulcers deepen to involve the dermis. If the horse remains in lateral recumbency, these pressure-induced ulcers deepen even further to involve and expose the underlying muscle or bone. Efforts to protect these areas with body stockings have been unsuccessful, as continual movement by the recumbent horse causes the dressings to slip. If the laterally recumbent horse switches from side to side (either voluntarily or with assistance and prompting), then the veterinarian has the opportunity to dress the ulcers on the "up" side. The ulcers should be dressed frequently during the day, as opportunities present themselves.

Equine amnion is a good dressing for decubital ulcers that are weeping serum. Antibiotic is applied before the wound is covered with amnion, which sticks to the exposed tissue and temporarily seals the wound. Alternatively, a very thin layer of cotton can be applied over the antibiotic-treated wound. Other topical preparations that are useful in these cases include silver sulphadiazine, milk of magnesia, hydrocortisone cream, pramoxin cream (a topical anesthetic that helps with the pain of deep dermal inflammation), and aloe vera gel.

Deep, Undermining Ulcers

In horses that develop deep ulcers that extend into the underlying tissue beyond the wound margins, undermining the skin, poultices of marigold and comfrey leaves packed into the defect can be very effective. These deep, undermining ulcers usually start as a firm mass, and then progress to become a fluctuant mass. Eventually, the overlying skin becomes necrotic and sloughs, leaving a deep ulcer that exposes the underlying muscle. This type of ulcer must be monitored very carefully because it can lead to septic myositis. These lesions can be controlled in the early stages (when still firm masses) with topical dimethyl sulfoxide (DMSO) gel, applied several times per day.

SEPTIC MYOSITIS

Septic myositis is a constant concern in the chronically recumbent horse. All horses with acute grade III or IV laminitis begin the recovery period in recumbency. However, most of these

FIGURE 19-13 A home-made hoof sock (made of two men's extra-large athletic socks, sewn together) protects coronary band ruptures and exposed sensitive laminae during the convalescent stage.

horses become more comfortable and begin standing and walking within a few weeks of proper digital manipulation and other therapy. Septic myositis becomes a concern in the horse that is still experiencing significant or worsening pain after a few weeks. The challenge is that if these horses stand, they will destroy their feet; and if they continue to lie down, they will destroy their muscles.

Septic myositis in this context can be defined as pressure necrosis involving large areas of muscle (particularly muscle overlying lateral bony prominences); colonization of the ischemic muscle by bacteria leads to liquefactive myonecrosis. Septic myositis can develop without disruption of the overlying skin (i.e., a closed lesion) or as a sequela to deep decubital ulceration.

These areas of compromised muscle are a perfect medium for bacterial growth. Organisms cultured from these lesions have included *Escherichia coli, Streptococcus* spp., *Staphylococcus* spp., *Klebsiella* spp., *Proteus* spp., *Pseudomonas* spp., and *Clostridium* spp. Broad-spectrum antibiotic therapy that includes anaerobic coverage is crucial to the survival of these horses. The parenteral (intravenous and/or intramuscular) route is preferred to the oral route.

These lesions are most commonly encountered on the side of the rib cage, the shoulder (over the greater tubercle of the humerus and/or the spine of the scapula), and the hip (over the greater trochanter of the femur and/or the tuber coxae; Figure 19-14). Closed lesions begin as a firm subcutaneous swelling that increases in size and softens to become a fluctuant mass. If left untreated, the ischemic tissue becomes septic and, if not treated aggressively, can lead to septicemia. Treatment of these lesions, once the overlying skin has sloughed, is discussed in the preceding section on decubital ulcers. Injection of antibiotics directly into the wound bed may also be of benefit. Frequent topical application of DMSO gel

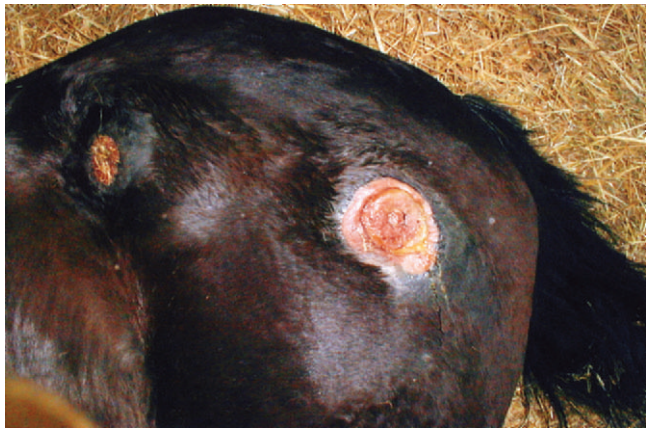

FIGURE 19-14 Decubital ulcers over tuber coxae predispose to septic myositis.

when the lesion is still just a firm mass may have helped prevent more serious necrosis in some cases.

MENTAL STIMULATION

Mental well-being is an often overlooked component of the healing process. In horses with severe laminitis, the healing process should be visualized as a triangle, with mental health, physical health, and the health of the foot composing the three sides. No one part can be neglected; all must be addressed, as complete healing will not occur otherwise.

Severe laminitis is a chronically and severely painful, debilitating disease process. The suffering these patients must endure can only be partially imagined. They suffer in silence, afraid of the outcome, and afraid of predation, as they have no defenses. It is the author's perception that these horses also suffer from a deep depression that transforms the psyche. They "disappear inside themselves," and there is great satisfaction for their caregivers when they finally return.

Recumbent Patients

During the recumbent phase, mental stimulation is best provided by frequent visits to the stall. Offering carrots, apples, melons, horse treats, peppermints, salt blocks, grass, herbs, and even grass roots with dirt clods attached helps stimulate the patient. Attending to the horse's bedding is another time in the daily routine in which interest is aroused.

Mental stimulation can also be achieved at transition points, such as when the recumbent horse begins to become ambulatory and switches from straw bedding to shavings. This minor alteration in environment can be a positive change for the horse, in much the same way that bed-ridden human patients feel better on having their sheets changed after breaking a fever or on moving from the intensive care unit to a private room.

Stall-Confined Ambulatory Patients

Once the horse becomes ambulatory but is still stall-bound for another 2 to 3 months, boredom can be relieved by providing hay ad libitum and stall toys (e.g., horse balls, traffic

FIGURE 19-15 Hanging empty juice bottles in the stall provides hours of entertainment for the stall-bound, recovering patient.

cones, hanging juice bottles). The recovering horse likes to explore and play by using its lips and muzzle. A number of plastic juice bottles have interesting hand grips and indentations that keep these inquisitive horses busy (Figure 19-15). When several of these bottles are hung in a line along the stall wall, horses have been observed playing them like a xylophone. Other horses take great pleasure in using their lips to unscrew the plastic caps and will spend hours in this diversion.

In addition, food treats are offered between meals. Another way to relieve boredom is to leave the stall door open for part of the day (with the horse's exit barred by a stall guard) so that the horse can lean out and see what is going on outside.

The severely laminitic horse may be stall bound for as long as 6 months, so the stall environment is their world. It can be just as frightening or as boring as a hospital bed is for a human patient, as the horse is at the mercy of the stall attendants, hospital staff, and veterinarian. Compassion, common sense, and a quiet and gentle demeanor will, with patience, bring these horses out of their pain and depression, making them whole mentally and physically once again.

CONCLUSION

In conclusion, the author would like to leave the reader with the words of an unknown veterinary author from the early 19th century:

> *The injury sustained by horses, called founder, is sometimes the effect of the cruelty of his master, and at other times brought on by injudicious treatment—but it is most frequently produced by carelessness, or a want of knowledge of the treatment necessary to those excellent animals on a journey.*
>
> *Although the horse is endowed with the strength and powers of a lion, yet he seldom exerts either to the prejudice of his master. On the contrary, he shares with him in his labours, and seems to participate with him in his pleasures. Generous and persevering, he gives up his whole powers to the service of his master—and though bold and intrepid, he represses the natural fire and vivacity of his temper, and not only yields to the hand, but seems to consult the inclination of his rider.*
>
> *But it must continue to be a matter of regret to every feeling mind that these excellent qualities should be so often shamefully abused in the most unnecessary exertions; and the honest labours of this noble animal, thrown away in the ungrateful task of accomplishing the purposes of unfeeling folly, or lavished in gratifying the expectations of an intemperate moment.*

20

DISEASES LEADING TO LAMINITIS AND THE MEDICAL MANAGEMENT OF THE LAMINITIC HORSE

ELEANOR KELLON

Laminitis can be initiated by a variety of toxic, pharmacologic or biologic, infectious, intestinal, or metabolic insults.

TOXIC

Laminitis can occur as a complication of a variety of plant toxicoses. Any poisonous plant that causes gastrointestinal damage or alters motility and function can induce laminitis, even when laminitis is not usually listed as a clinical sign of the poisoning. Similarly, plants that cause circulatory disturbances or hemolysis are also at times associated with laminitis.

As list of the most commonly cited plant-related causes of laminitis is given in Box 20-1. Black walnut is the best studied of the toxic inducers and has been proposed as a model for laminitis of varying origin. Laminitis can result simply through skin exposure in horses bedded on shavings containing black walnut. There is considerable evidence of circulatory disturbance[1-3] during the prodromal stages of laminitis induced by experimental administration of black walnut extract, followed shortly by an inflammatory component.[4] Adair et al.[1] noted significant decrease in laminar blood flow as early as 1 to 2 hours after administration, normalization for a subsequent 4 hours followed by persistent decrease after 6 hours. Eaton et al.[2] reported a drop in central venous pressure correlating in time with the initial decreased laminar blood flow, with increased capillary resistance to flow. Galey et al.[3] demonstrated increased sensitivity to epinephrine contracture in black walnut treated digital arteries.

With black walnut toxicity, the laminitis apparently results as a direct effect of the toxin juglone and other toxic principles. In other cases, however, the pathophysiology is not as clear and laminitis may be occurring as a secondary event following direct damage to the gut lining, damage to endothelial integrity (manifested clinically as edema) or hemolysis and hypoxia. In the case of white clover, carbohydrate overload has been suggested as the cause.

Selenium toxicosis can result in loss of laminar integrity and hoof wall separation to the point of hoof sloughing. Although similar clinically to laminitis of other origins, the cause is defective horn formation and subsequent mechanical failure. Histopathologic changes include parakeratotic cellular debris, separated by more normal hoof matrix, hyperplasia, acanthosis, parakeratosis, and disorganized germinal epithelium of varying severity in hoof epithelium.[6] Ingestion of mercury from a leg blister has been reported to cause laminitis,[7] although this was likely secondary to the extensive gastrointestinal damage caused rather than a direct mercury effect.

Treatment

Treatment of toxic plant–related laminitis primarily consists of removal of the exposure and supportive care. With selenium toxicity in pigs, arsenic in the form of arsanilic acid has been proposed as an antidote, but there are obvious drawbacks and dangers to replacing one source of toxicity with another. Dietary supplementation with vitamin E and methionine may assist with hepatic detoxification and elimination of accumulated selenium, and generous dietary levels of copper and sulfur suppress further intestinal absorption. (see Box 20-1)

IATROGENIC LAMINITIS

Administration of corticosteroids by any route may induce laminitis. The etiology of corticosteroid-induced laminitis has not been established and may be multifactorial. Corticosteroids induce insulin resistance, but the effect is short lived. Eyre et al.[8] documented marked potentiation of the constrictive response to epinephrine and other vasoconstrictive amines in digital veins and arteries incubated in the presence of corticosteroids. Bethamethasone effect was more pronounced than dexamethasone in that study.

Laminitis as a sequela to deworming has been anecdotally reported and is more likely to be a manifestation of a systemic inflammatory or allergic reaction to parasite antigens, rather than a direct drug effect.

Postimmunization laminitis has also been anecdotally reported, with the mechanism unclear. However, Wagner et al.[9] documented significant differences between normal and chronically laminitic horses in both immediate and delayed sensitivity response to 74 antigens injected intradermally. They conclude that the chronically laminitic horse may be at risk for exacerbation after antigenic challenges. Responses could theoretically be triggered by either antigens related to the infectious organism itself (e.g., *Streptococcus equi*), or to adjuvants used.

Box 20-1 Plants and Toxins Capable of Producing Laminitis

Black walnut *(Juglans nigra):* ingestion or exposure to shavings
Red oak *(Quercus rubor):* ingestion, acorns or buds
Hoary alyssum *(Berteroa incana):* ingestion, entire plant[5]
Black locust *(Robinia pseudoacacia):* ingestion, bark, leaves, sprouts
Avocado *(Persea):* ingestion, skins, leaves, pits
White clover *(Trifolium repens):* ingestion of large amounts of clover-predominant pasture
Selenium: ingestion, inadvertent oversupplementation or selenium-accumulating plants such as *Astragalus* spp. (Milkvetch), Prince's plume, Woody aster
Rattlesnake venom
Mercury: ingestion of mercury-containing blisters

Treatment

There is no specific treatment for iatrogenic laminitis beyond pain control and general supportive care. Pretreatment with flunixine meglumine or phenylbutazone may prevent or minimize laminitis in horses with a history of previous vaccine or dewormer reactions, or those that might reasonably be considered high risk.

INFECTIONS AND COLIC-RELATED LAMINITIS

Laminitis is well recognized as a possible sequela to systemic infection. Equine monocytic ehrlichiosis (Potomac Horse Fever), salmonellosis, or other illnesses caused by intestinal pathogens are most common, and laminitis is often a part of late purpura hemorrhagica complications of *S. equi* (strangles) infections. Laminitis in mares with retained placenta is also believed to be largely the result of proliferation of gram-negative bacteria within the degenerating placental tissues with subsequent endotoxemia. The mechanisms of laminitis in these instances have been incompletely described and likely involve a complex interplay of both direct effects of elaborated toxins and the body's response to them. Mallem et al.[10] documented a direct blocking effect of endotoxin on beta-3 adrenoreceptor-induced relaxation in digital veins, whereas Rodgerson et al.[11] showed that digital artery smooth muscle cells increase cyclooxygenase 2 production on exposure to endotoxin and that endotoxin triggers production of interleukin-1β and tumor necrosis factor alpha from monocytes, all of which are inflammatory mediators. Endotoxin also causes the release of platelet activation factor by macrophages, which inhibits the uptake of the vasoconstrictor 5-hydroxytryptamine by platelets,[12] resulting in spasm, and contributes to coagulopathy.[13]

Horses with infectious processes that directly involve the bowel, or serious colics with resultant acidosis in the bowel, may have laminitis as a result of the elaboration of vasoactive amines by intestinal micro-organisms or enhanced absorption of same through a compromised intestinal wall. As many as 15 different vasoactive amines have been identified in equine cecal contents.[14]

Treatment

Any horse with an intestinal disorder or infection, *S. equi* infection, or systemic signs of septicemia or endotoxemia must be considered at high risk for laminitis. Attendants of such horses should be carefully instructed to monitor the digital pulses and hoof temperature for clinical evidence of decreased perfusion (weak to nonpalpable pulses, cold feet) that are the prodrome to clinical laminitis. A prophylactic support protocol involving the application of Redden Ultimate Wedges (Nanric, Versailles, Ky.) has been adopted by North Carolina for all horses at high risk. Intravenous fluid support, appropriate antibiotics, monitoring of coagulation status with correction as indicated, and administration of nonsteroidal antiinflammatory drugs (NSAIDs) are all standard therapy. Combination therapy with flunixin meglumine and pentoxifylline was superior to either alone in a clinical model of endotoxemia.[15] Administration of an intravenous phospholipid emulsion is showing considerable promise investigationally.[16]

CARBOHYDRATE OVERLOAD

Both accidental grain overload in the "horse got into the feed room" scenario and experimental overload using soluble carbohydrate in the form of corn starch or chicory inulin can reliably induce laminitis in any horse. Although much is known about the pathophysiology of this condition, the search for a single trigger goes on and the mechanism may prove to be multifactorial. Most attention has been focused on the resultant disruptions of pH and microbial populations in the cecum and colon. There are many parallels with endotoxemia, and overproduction of vasoactive amines likely also contributes.[10-14,17,18]

Grass-induced laminitis may share similar mechanisms with grain overload, at least in horses with unlimited access to grasses high in soluble or complex sugar (e.g., fructans, levans; see Chapter 15). However, many horses and ponies prone to grass laminitis develop problems with even severely restricted periods of grazing that do not allow sufficient time for intake of an amount of grass necessary to produce significant disruption of the colonic environment. When animals develop laminitis with very limited grazing time, in particular if they fit the prototypical profile of the fat pony or horse prone to grass laminitis, it is worthwhile checking for insulin resistance as a predisposing factor.

HORMONE-INDUCED OR METABOLIC-INDUCED LAMINITIS

Laminitis has long been recognized as a sequela of pituitary pars intermedia dysfunction (PPID; Cushing disease) in older horses (Figure 20-1). Again, the pathophysiology is not clearly defined and may be multifactorial. Adrenal overstimulation via adrenocorticotropic hormone (ACTH) and other peptides produced by the tumor may result in laminitis risk by direct vascular mechanisms similar to those operating in iatrogenic cases involving corticosteroid use. Although there are some PPID cases that develop laminitis without concomitant significant elevations in blood glucose or insulin levels, this is the exception rather than the rule.

Cortisol is an insulin antagonist; it blocks the effects of insulin at the cellular level. Homeostatic mechanisms respond to the cellular insulin resistance by producing more insulin. If

FIGURE 20-1 An older pony with hirsutism and chronic laminitis.

sufficient insulin is being produced to overcome the block to insulin action and accomplish the job of getting glucose into the cells, blood insulin levels will be high but glucose concentration will remain in a normal range. As PPID advances, or if it is left untreated, this can eventually progress to frank diabetes with both elevated insulin and elevated glucose levels.

Insulin resistance (type II diabetes) in humans and other species is well documented to produce peripheral circulatory problems via both enhanced thrombotic tendencies[19] and spasm or impaired relaxation of peripheral vessels,[20] as well as a proinflammatory state.[21] Until quite recently, it was believed that insulin resistance and diabetes did not occur as a primary entity in the equine species and its presence was always an indicator of PPID. However, breed variability in insulin sensitivity has been known for at least 30 years and it is becoming increasingly obvious that a variety of metabolic syndromes unrelated to PPID may exist, each with insulin resistance at its core.

Investigation into the causes of insulin resistance in people and other species is a very active field of research, the scope of which is beyond this chapter. Contributing factors include genetic and racial predisposition, obesity, disruption of pituitary axes and abnormal feedback loops, aberrant peripheral cortisol metabolism, and nutritional deficiencies and imbalances, including iron overload.

When elevated insulin levels were found in younger animals or in the absence of laboratory confirmation of PPID by either ACTH or dexamethasone suppression testing, the tendency has been to label the condition *pre-Cushing* or *early Cushing* disease (Figure 20-2). More recently, interest has turned to investigating the possibility that insulin resistance arises as a direct consequence of obesity. A correlation between degree of obesity and insulin sensitivity or resistance has been noted,[22] but the premise that obesity per se is causal may not necessarily hold true. Cartmill et al.[23] found that insulin resistance correlated with high serum leptin levels in obese horses, rather than with the presence or severity of obesity. These studies looked at horses that were already obese, so whether the insulin resistance was a cause or effect is unknown, although Cartmill's findings fit very well with what is known about one type of genetic predisposition to insulin resistance in humans—leptin resistance.

Insulin resistance in horses appears to have similarities to a variety of different syndromes in humans, including the

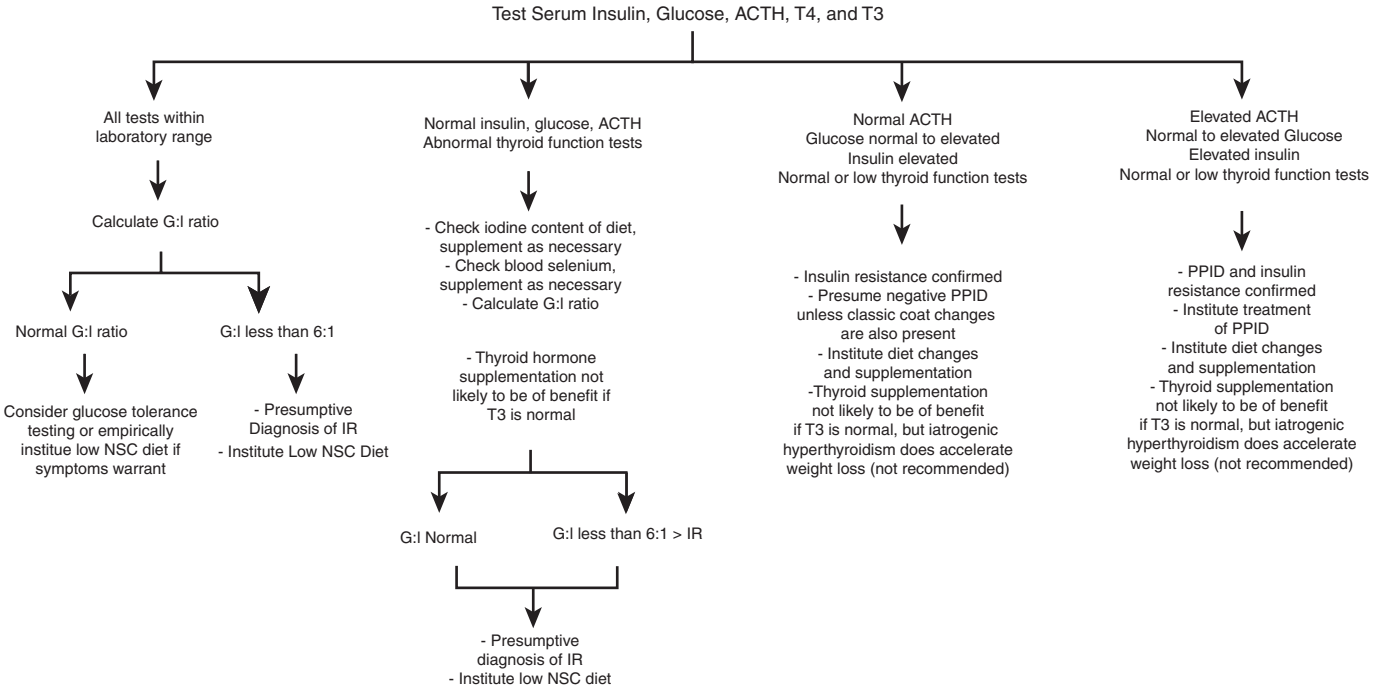

FIGURE 20-2 Algorithm for diagnosis of hormonal or metabolic-induced laminitis.

metabolic syndrome[24] and polycystic ovarian syndrome. However, understanding of insulin resistance in the horse is in its infancy, and there may be no reason to expect root causes are any less varied and complicated than in other species.

Diagnosis

These horses and ponies usually are examined because of laminitis, often recurrent or unrelenting. History and examination also typically reveal one or more of the following:

- Easy weight gain dating back to early adulthood
- Obesity disproportionate to energy or calorie content of the diet
- Lethargy, decreased performance
- Abnormal fat deposits along crest, withers, tailhead, or elsewhere
- Bulging fat deposits in the supraorbital fossa
- Abnormal cycling or fertility problems in mares of reproductive age
- Ravenous appetite
- Polydipsia/polyuria
- Mild unexplained elevations in serum creatine kinase level
- Mildly elevated liver enzyme levels, particularly γ-glutamyltransferase
- High normal to elevated triglycerides
- Elevated insulin level, usually with normal or high normal blood glucose
- Family or breed history of laminitis or easy weight gain, crestiness
- Seasonal laminitis related to grazing, even when sharply restricted
- Late summer (August) to fall seasonal laminitis flares, likely related to seasonal elevations in ACTH

Advanced cases involving horses on a diet inappropriately high in soluble carbohydrate may progress to extreme unexplained weight loss and muscle wasting, often misguidedly treated by increasing the grain portion of the diet, which only makes them worse.

Careful questioning often reveals that symptoms suggestive of low-level foot pain were present long before the acute severely painful episode. These include the following:

- Horse hesitates to move forward when standing still.
- Horse is reluctant to turn in a tight circle, even to turn neck to the side freely.
- Stride length, freedom of movement, and so on improve after a few minutes of moving around.
- Hoof growth rate tends to be slow.
- Horse is more comfortable on soft ground than hard.
- Horse is more comfortable going uphill than down.
- Muscles of the hindquarters are better developed than of the forequarters and chest.
- Muscles of the lower back and hindquarters feel tense/hard/bunched.
- Muscular tension is present in the shoulder area.
- Horse holds the head and neck rigidly when walking rather than the normal up and down movement (some horses with foot pain prefer to hold their head high, some try sticking their nose out in front of them more than usual).
- Horse rarely trots spontaneously.
- Horse has "stiff" hocks or stocking up behind (from weight shifting to the rear).
- Horse has subdued, "quiet" attitude, or irritability.

These symptoms are all familiar to veterinarians, but owners frequently do not make the connection with possible foot pain unless specifically asked.

Symptoms suggestive specifically of PPID include failure to shed normally, long or curly coat, and udder enlargement in mares, possibly with lactation. Weight loss and muscle wasting tend to be more common in horses with PPID, but advanced insulin resistance can also cause this, and horses that have been dealing with chronic pain for a prolonged period are also often cachectic.

Initial screening should include measuring levels of ACTH for PPID, thyroxine (T4) and triiodothyronine (T3), and insulin and glucose. In animals prone to hyperlipidemia (ponies, miniatures, donkeys), triglyceride and cholesterol screening is also advisable, particularly if the animal is off feed or has been having diet severely restricted. Thyroid testing, glucose and triglycerides/cholesterol may be available as part of a "Super Chem" panel from some laboratory tests and allows a check for elevated muscle and hepatic enzymes at little or no additional cost to the client. Mild elevations are often found with cases of severe insulin resistance, PPID associated or not.

Most laboratory norm have been established from sampling of horses with access to hay but not grain or other concentrated soluble carbohydrate sources for a minimum of 4 hours before testing. The suggested protocol is usually to withhold grain after the evening feed of the day prior to testing.

Interpretation

Adrenocorticotropic Hormone

Definitive diagnosis of PPID by laboratory parameters is not possible. A finding of complete failure to achieve results on dexamethasone suppression or of an ACTH level elevated to above 190 to 200 pg/mL are highly diagnostic, but usually results are equivocal. The recent finding that time of year can lead to spurious elevations in ACTH activity (false-positives in September compared to May and December) further complicates diagnostic testing.[25] The decision of whether to use pergolide is often made on clinical judgment. Advanced age and typical coat changes are the strongest indicators when laboratory results are not clear. Chronic laminitis and elevated insulin are not diagnostic of PPID.

Thyroid Function Tests

Considerable debate exists as to the validity of thyroid function tests, and the advisability of supplementation with thyroid hormone even when low values are found. Primary pathologic conditions of the thyroid gland are highly unlikely, and any abnormalities found are secondary to either specific hormonal aberrations or the euthyroid sick syndrome. However, supplementation, at least in the short term, can still be used. It is recommended to postpone supplementation until T3, the active form of the hormone, is low. An exception to this would be a horse with confirmed PPID with very low T4 levels. Thyroid-

stimulating hormone (TSH) is known to be stimulatory to pituitary adenomas. Avoiding high levels of TSH by maintaining T4 in a normal range may be beneficial. Once the PPID or insulin resistance is under control, the horse can be weaned off thyroid supplementation. When interpreting thyroid hormone test results, the veterinarian needs to remember that phenylbutazone significantly lowers T4 and T3 levels. T4 has a normal diurnal variation, with highest values in late afternoon/early evening and lowest in the morning, whereas T3 does not show any variations by time of day.

Insulin

Obvious hyperinsulinemia unrelated to feeding is present in the vast majority of horses with clinical evidence of insulin resistance by the time they are presented with laminitis. Horses very early in the course of disease, however, may have normal basal insulin and glucose levels and would require oral or intravenous glucose tolerance testing or other techniques such as euglycemic insulin clamp to detect the abnormality in glucose handling. The glucose-to-insulin (G:I) ratio, calculated from glucose in milligrams per decaliter and insulin in microunits per milliliter, is a readily available indicator that has been shown in humans with insulin resistance to be a valuable screening tool.[25] Although racial differences have been noted in humans, a cutoff for G:I ratio of between 4.5:1 and 6:1 is used, with values lower than this being highly suggestive of insulin resistance. Preliminary examination data suggests the same holds true for horses, and calculation of the G:I ratio will often show that this number is indeed low even if testing shows insulin and glucose values to be within the range of normal for the testing laboratory (E. Kellon, unpublished data). Also, animals that are clearly hyperinsulinemic on initial testing many times show a drop in insulin and glucose levels when dietary changes are made, while the G:I ratio remains low. Further study is needed, but the G:I ratio shows promise as an equine screening tool and is particularly appealing because no specialized testing or lengthy, multiple sample glucose tolerance testing is required.

Treatment

A variety of pharmacologic interventions are available in human medicine that have yet to be investigated in the horse, but the cornerstone of therapy for all species is appropriate diet and exercise. Medication alone cannot control insulin resistance, regardless of the cause.

Horses with insulin resistance and excess ACTH secretion related to PPID require medication to control hormonal output from the tumor. Pergolide is the current gold standard of therapy but also the most likely to be associated with side effects. Cyproheptadine and the herb Vitex agnus castus (a dopaminergic agent with known pharmacologic activity quite similar to pergolide) can be tried as first-line therapy with switch to pergolide always an option. Some animals respond favorably to medication alone, at least initially, but recurrent problems with laminitis relapse and poorly controlled insulin resistance are common when this is the sole therapy.

As in human insulin resistance, diet control is an essential part of treatment.[21] These horses and ponies do quite well on a diet with an extremely low glycemic index and strict limitation of soluble sugars and starches. Whole grains, senior or complete feeds containing grains or grain products, carrots, apples, and fresh grass must be eliminated. Beet pulp with no molasses added can be substituted as a feed and has a glycemic index in the horse of nearly zero, with the bulk of the calories being supplied in the form of the easily fermentable soluble fiber pectin. Shreds are preferable to pellets because molasses is commonly used as the binder in pellets and may not show up on the ingredients list. The nonstructural carbohydrate (NSC) content of plain beet pulp is typically as low as 4% to 5%; this number jumps to 10% or higher when molasses is added to the product. A mixture of 1 lb beet pulp with 2 oz rice bran or 3 oz ground stabilized flax has improved palatability, low glycemic index, and a fairly good major mineral balance. When the animal needs to lose weight, beet pulp feedings should be limited to a pound or less, presoaked dry weight, because of the higher caloric value (digestible energy [DE] of one pound of beet pulp equals 1.5 to 2.0 lb of hay). Conversely, animals in poor body condition can safely be fed increased amounts of beet pulp to encourage weight gain.

Grass hay should form the bulk of the diet. Because of the high sugar content of many hays, it is advisable to have it analyzed. Actively laminitic animals should be fed hay with a nonfiber carbohydrate (NFC) content no higher than 12%. Some may require an NSC content ceiling of 10% for good control. If low-NFC hay is not available, sugar content can be reduced by soaking before feeding, 30 minutes in hot water or 60 minutes in cold water. After soaking, a sample of hay should be dried and retested to ensure the NSC has been sufficiently lowered. Some insulin-resistant horses are sensitive to alfalfa hay, despite its typically low sugar and starch content. The reason for this is unclear. If unsure, it is best to avoid alfalfa. If the animal can tolerate it, alfalfa can be used for 10% to 20% of the hay ration.

It is a mistake to severely restrict calorie intake in an attempt to reduce weight. Insulin resistance often worsens when this is done. With a grass hay of average DE (0.65 to 0.75 kcal/lb), the horse can be fed at a rate of 1.5% to 2.0% of ideal body weight for a body condition score of 5. For an estimated target body weight of 1000 lbs, the horse should be fed 15 to 20 lbs of low-NSC hay per day. If beet pulp or beet pulp and rice bran is being fed, this should be substituted at a rate of 1 lb beet pulp per 1.5 lbs of hay. Clients should be carefully instructed to actually weigh all components of the diet. Animals should be weight taped every two weeks to follow progress, and amounts of feed adjusted up or down as necessary. If the animal is ambulatory and not stall confined, it is often possible to feed hay free choice once insulin resistance has been controlled, with no risk of excess consumption or weight gain.

Although control of the soluble carbohydrate intake is undeniably the cornerstone of diet control, careful attention to individual nutrients is also important. Deficiencies, excesses (e.g., iron), or imbalances of a variety of vitamins and minerals have been linked to insulin resistance in humans and experimental animals.[26-30] These include iron, magnesium, calcium, zinc, chromium, vitamin E, vitamin C, vitamin D, and essential fatty acids. Antioxidant nutrients have also been shown to help protect against vascular endothelial damage in cases of insulin resistance. Provision of adequate amounts of these key nutrients is beneficial, but care must also be taken to ensure that all minerals in the diet remain in correct propor-

tions to avoid both oversupplementation and undersupplementation of total amounts and induction of relative deficiencies by oversupplementation of individual nutrients.

The following guidelines are recommended:

- Intake of each individual mineral set at a minimum of 150% of current NRC minimums
- Ca:P:Mg between 1.5:1:1 to 2.0:1:1
- Cu:Z:Mn 1:2.5 to 3:3
- Cu:Fe maximum of 1:10
- Selenium total from all sources 1 mg/100 kg body weight
- Iodine total from all sources 1 mg/100 kg body weight
- Chromium 0.5 to 1.0 mg/100 kg body weight
- Vitamin E 400 IU/100 kg body weight
- Vitamin C 500 mg/100 kg body weight (hold for hays with iron content of over 150 ppm, or substitute Ester-C 50 mg/100 kg body weight)
- Lysine 1500 mg/100 kg body weight
- Methionine 500 mg/100 kg body weight
- Ground stabilized or freshly ground whole flaxseed 0.5 oz/100 kg body weight

High-fat diets worsen insulin resistance in other species, as well as in ponies. Horses may be less susceptible to this effect but until it is confirmed that increased fat intake does not pose a risk for horses with insulin resistance, it is best to avoid it.

High-protein diets are effective in other species but have yielded mixed results, or been detrimental, in insulin-resistant horses. Some amino acids are also potent triggers of insulin release and insulin is required for normal cellular uptake of amino acids. Adequate, but not excessive, protein intake is advisable. Protein requirements should be addressed in terms of grams of protein per day, not as a percentage of the diet. Protein intake is a function of both the percentage of protein in the diet and how many pounds per day are being consumed. Grass hays containing 7.5% protein and consumed at a rate of 2% body weight per day will meet crude protein requirements. At feeding rates of 1.5% of body weight per day, a 10% protein hay is needed. Because grass hays are typically low in lysine, this essential amino acid should be given as a supplement. Low-level methionine supplementation (2500 mg/day) to support good hoof quality is also helpful.

Other Supplements

Recent laboratory animal studies, as well as clinical trials in insulin-resistant humans, have documented a favorable effect of cinnamon[31,32] in both preventing and treating insulin resistance. Cinnamon is an insulin mimetic. Initial response in horses to 1 mg of cinnamon per pound of body weight, divided into two feedings, looks promising, but there may be a tendency toward blood glucose levels becoming slightly lower than the usual normal range (50s). Formal studies are needed.

Gynostemma pentaphyllum (Jiaogulan) is an oriental herb documented to support endothelial nitric oxide production while at the same time suppressing inducible and inflammatory nitric oxide pathways.[33,34] One hundred twenty-six horses, ponies, and donkeys with chronic foot pain related to laminitis of between 2 months' and 4 years' duration were studied. The average duration of chronic pain was 18 months. Gynostemma was started only when any underlying metabolic problems had been appropriately addressed and only if the animal was receiving proper foot care. Ninety-five percent of the animals were receiving constant or intermittent phenylbutazone, which was stopped because of the potential to interfere with nitric oxide generation. High-dose antioxidants, antiinflammatory herbal supplements, and other herbals with the potential to influence nitric oxide or inflammatory pathways (e.g., ginsengs, Hawthorne) were also stopped. Dosage started at 150 mg/500 lb twice daily, titrated up every 2 days to response or a maximum of 600 mg/500 lb twice daily.

Sixty-five of 128 horses showed return to soundness within 2 days to 2 weeks of starting Gynostemma. A period of abscessation preceded soundness in 47% of these horses. Forty-six of 128 showed improvement of 1 to 2 lameness grades within the same time period. Fifteen horses and ponies failed to respond or responded initially, only to show deteriorating soundness at a later interval. In 12 of these cases, worsening of the underlying medical condition (usually PPID), inability to trim or shoe the feet to a state that prevented further mechanical damage, or an already severely advanced demineralization of the coffin bone were present. In three animals, the reason for poor response was unknown (E. Kellon, unpublished data).

Box 20-2 Feed Terminology

NFC = nonfiber carbohydrates. Percentage of the hay or solid feed composed of sugars, starches, highly soluble fiber such as beta-glucan and pectin, or organic acids. (Latter two are low to nonexistent in grass hays.)
NSC = nonstructural carbohydrates. There is poor consistency between forage testing laboratories in exactly how this term is used. In some, it is interchangeable with NFC whereas in others it is used to express the starch/sugar component only. Laboratories that offer both NSC and NFC testing (e.g., Dairy One, Ithaca, New York) define NSC as the combination of sugar and starch.
DE = digestible energy, kcal/lb
Ppm = parts per million = mg/kg

LAMINITIS IN PREGNANCY

A small percentage of mares develop laminitis during middle to late pregnancy. This has usually been assumed to be related to the increased body weight. However, the hormonal changes of pregnancy can also result in insulin resistance.[35,36] If insulin resistance is found, institution of a diet with a low glycemic index with appropriate adjustments in calories, minerals, and protein intake can alleviate the problem (Boxes 20-2 and 20-3) (E. Kellon, unpublished data).

Case History 1

History: 15 YO grade pony gelding with a 3-year history of repeated bouts of laminitis. Not obese but did have abnormal fat deposits at tail base, cresty neck, fat bulging in the supraorbital fossa, PU/PD. Had not responded to thyroid supplementation (for low T4) and corrective shoeing over this 3-year period. Laminitis progressed from no rotation to 9 degrees. No other testing or management changes. Insulin then tested and found to be high, 156 μIU/mL (normal up to 30 for the testing lab),

Box 20-3 Recommended Hay Analysis Tests

When requesting hay analysis for an insulin-resistant horse or pony, the profile should include the following:
DE for horse (DE estimates for cattle do not apply)
Crude protein
NSC or NFC
Calcium
Phosphorus
Magnesium
Iron
Copper
Zinc
Manganese
Selenium

DE, Digestible energy; *NSC,* nonstructural carbohydrates; *NFC,* nonfiber carbohydrates.

prescribed pergolide for presumed PPID. Three months later, insulin had risen to 260 μIU/ml. Lameness persisted.

Diagnosis and treatment: I was consulted at this point, recommended that pergolide be stopped, and pony started on a low-NSC diet of beet pulp and soaked grass hay pending results of hay analysis. Retest 1 month later, off pergolide and thyroid supplement, showed T4 low but T3 normal, normal ACTH, insulin down to 55.27 μIU/mL, glucose 96 mg/dL, G:I ratio 1.7:1. Pony started on mineral supplement customized to match the hay analysis. One month later, insulin down to 16.38, glucose 80, G:I ratio improved to 4.9:1. Pony is sound barefoot.

Case History 2

History: 11 YO Saddlebred gelding, first episode of laminitis, originally misdiagnosed as being lameness related to ringbone. Body condition score 7, fatty crest, weight 1160 pounds, very swollen/fatty sheath over previous 6 years. Not being overfed. Diet at time of diagnosis was 1 lb/day of plain oats, 5 lb mixed grass/alfalfa hay, 3 to 4 lb grass hay. Found to have insulin of greater than 455 μIU/mL. Thyroid function tests normal. Original treatment was to restrict diet to limited amount of grass hay, Styrofoam to feet, high-dose phenylbutazone, acepromazine, chromium supplement.[37] Horse remained sore, unable to wean off phenylbutazone over subsequent 3 months, still very lame. No weight loss.

Diagnosis and treatment: Diet changed to 3 lb beet pulp/day, and hay quantity sufficient to maintain estimated ideal body weight of about 1000 to 1050 lb. Energy/calorie intake at this level of feeding was approximately 30% higher than it had been for the preceding 3 months. Started vitamin/mineral supplementation appropriate to the hay analysis. Weaned off phenylbutazone, supplemented with powdered Gynostemma pentaphyllum for circulatory support. Rapid improvement in soundness. 11 months later, weight down to 1020 pounds, insulin down to 85.7 μIU/mL. Current plan is to check ACTH for completeness, start formal exercise in hopes that will lower insulin further.

Case History 3

History: 10 YO Paso Fino gelding, 3-year history of chronic laminitis, multiple shoeing methods tried with little to no improvement. Body condition score 7, weight 1060 lbs, ravenous appetite, cresty neck, supraorbital fat bulges. Initial insulin 104 μIU/mL, glucose 113 mg/dL, G:I 1.08. Diet at this time was 1.5 lbs plain oats, 24/7 pasture. Initial treatment was to take the horse off pasture, grass hay only (limited to 10 lbs/day). Slight weight loss which leveled off. No improvement in foot comfort. Repeat labs 4 months later showed T3 normal, T4 low normal, insulin up to 375, glucose 113, G:I ratio 0.31:1.

Treatment: At this point changed diet to a commercial low NSC hay pellet with minerals to balance, 14 lb/day and 1 lb beet pulp. Six weeks later, insulin 25, glucose 92, G:I ratio 3.68:1. Sound for the first time in almost 4 years.

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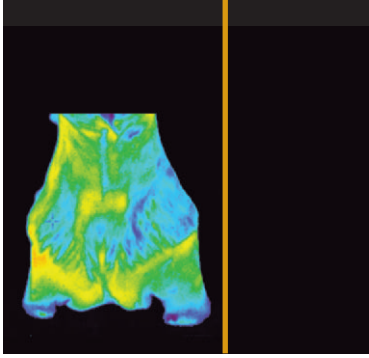


Section V
FARRIERY CONSIDERATIONS

21
BALANCING AND SHOEING THE EQUINE FOOT

DERIN FOOR

BALANCE

Balance is arguably the single most important factor in equine podiatry. It remains the most discussed issue among farriers and veterinarians worldwide. Achieving the seemingly impossible goal of balance takes years of practice, thousands of hooves, and consideration of many theories on how the equine foot can and should be balanced. Every year there are numerous articles, books, and Internet postings written about balancing a horse's foot; but how do the veterinarian and farrier know which methods will work and which ones will not?

The one point nearly every farrier will agree on is that the foot must be properly balanced before shoeing. Otherwise, one risks injury to the horse and breaks the first commandment of horse shoeing: "Do No Harm." To intentionally apply a shoe to an unbalanced foot not only risks injury to the horse, but it also jeopardizes the rider's safety and often has long-term consequences.

Horse owners rely on their farriers to provide them with proper care, good advice, and a willingness to work closely with the veterinary community when necessary. The costs involved with trimming and shoeing, generally speaking, are the largest part of horse ownership expenses. These expenses can be optimized with regular hoof care by a qualified professional. Maintenance is one of the key points to keeping a horse sound. Most horses in the United States are trimmed on a 5- to 8-week schedule. By addressing minor problems and fixing them before they become major ones, the farrier is able to maintain the health and well-being of the horse's feet and legs. Anything less often leads to unsound horses that become a burden to their owners, who end up frustrated with an animal that cannot be ridden or driven. This situation accounts for the very high turnover rate in the horse industry. So, how do equine practitioners keep these horses sound and productive? The answer is, through balance.

Diversity of Needs and Opinions

The horse's foot is an elastic, ever-changing capsule filled with sensitive structures, blood, and bone. It supports the large body mass through a series of checks and balances. It seems unlikely that such a small part of the total horse should have such a great impact on the well-being and longevity of this magnificent creature, but it does. It would seem logical that, for such an intricate part of the horse, specific guidelines for its care would exist, and perhaps that is where science is leading the practice.

One of the problems with formulating such guidelines is the diversity among horses and climates in which they live and work. Breed, age, discipline, and environment all play important roles in determining how to best trim and shoe a particular horse. Farriers need to be knowledgeable about the horses in their care. They must understand what the horse is used for, under what conditions it lives and works, and whether there are any diseases affecting the horse's feet. The farrier needs this information before trimming begins.

Farriers also need to understand the immediate as well as the long-term impact of trimming and shoeing. When routine trimming becomes just regular removal or "hacking off" of

some hoof wall, the horse is in trouble. It should be the goal of every hoof care provider to make a positive difference on every hoof of every horse. Anything less deprives the horse of the proper care that it deserves.

A larger problem than the diversity among horses is the tremendous diversity of opinion among farriers as to how a horse should be trimmed and balanced. If 50 farriers are asked to define balance, 50 different answers are given. If two of them say the same thing, then one likely trained the other. Although the responses will be similar on many accounts, most will differ in their specific methods of achieving balance.

The question then is how to get past this controversy and do what is right for the horse. Equine practitioners have to read, research, learn, and apply the knowledge that they feel is best for the horses in their care. The true leaders in this industry are the people who continue to do these things, who might be referred to as the *"What if" people.* These researchers understand that farriery cannot be thought of as an exact science, for all of the reasons previously mentioned. They know that, though the farrier works on the horse in a static environment, the reality is that the horse lives in a very dynamic world and needs to be trimmed accordingly. So, they keep asking themselves, "What if . . ."

TRIMMING

Equine podiatry is based on various theories and methods. Farriers, as well as veterinarians, tend to rely on a combination of methods as guidelines for their everyday practices. The following information discusses some of the theories and methods that are used worldwide to properly balance the equine foot. Some are widely accepted, whereas others have little if any scientific backing. These methods, which are given in no particular order, often share basic principles yet have distinct differences.

Shoeing applications are discussed later in this chapter, but it should be mentioned at this point that some of the theories concerning equine podiatry allow for the application of shoes when necessary, whereas others are strictly opposed to the idea. When the mindset becomes an either/or situation, all practitioners lose, and the horse is the biggest loser. Advocates of barefoot-only approaches are as detrimental to the horse population as are the farriers that insist every horse be shod.

Different Approaches

The following trimming approaches are in no way meant to be a complete discussion of the individual protocols. Further research on particular methods is encouraged.

Four-Point Trim

Ric Redden is one of the world's leading researchers in equine podiatry. He has spent many years operating the International Equine Podiatry Center in Versailles, Kentucky, and trying to perfect hoof balance. Thousands of horses, and almost every foot abnormality and pathologic condition known, have passed through his skilled hands. His research on hoof balance is based largely on studies of feral horses in the Midwest.

Redden introduced the four-point trim, in which the domestic horse's foot is trimmed to closely resemble the foot of the feral horse. In particular, the quarters are relieved (non–weight bearing) and the toe is rolled for easier breakover, basically leaving the horse bearing weight on the four pillars of the foot; hence the name *four-point trim*.

Natural Balance Trim

Gene Ovnicek, a Colorado farrier, has also spent many years studying feral horses and their feet. He has developed a trimming method known as *natural balance.* His theories extend to natural balance shoeing (NBS) and are incorporated into the Equine Digit Support System (EDSS), which focuses largely on laminitis and shoeing therapy for the foundered horse. Ovnicek's natural balance method uses landmarks on the solar surface of the foot to guide the trimming process. Emphasis is placed on preserving the sole callus, along with minimal frog trimming.

Duckett's Dot

Farrier David Duckett developed the system known as *Duckett's Dot.* His method for balancing the foot is based on the theory of finding the center of the individual foot, generally located at the widest part of the hoof and halfway across the frog. Once this point is located, the rest of the foot is centered around the designated midpoint for an even distribution of weight bearing.

Hoof Talk Natural Trim

Lyle Bergeleen of Mead, Washington promotes the Hoof Talk Natural Trim method for balancing a foot. This method uses specific measurements and takes into account the horse's weight, which is a factor not often calculated when trimming a horse's foot.

High Performance Barefoot Trim

K.C. LaPierre, a registered journeyman farrier of many years, now subscribes to a different approach to hoof balance. His "Shoeless Not Clueless" catch phrase is popular with some horse owners in the United States. LaPierre now contends that horses should remain barefoot, and he routinely gives clinics in which horse owners are taught to perform their own hoof trimming. His high performance trim (HPT) is taught at the School of Barefoot Equine Podiatry and is based on the suspension theory of hoof dynamics. The domesticated horse is the focus of his work, which emphasizes the relationship of a properly structured foot and a sound horse.

Strasser Barefoot Trim

German veterinarian Hiltrude Strasser advocates natural boarding, in which horses are kept in a way that mimics their natural environment, as opposed to conditions often found in boarding barns and stables. Her theory is that many of these horses will self-maintain their feet, given the proper environment. However, many horse owners find it difficult, if not impossible, to provide such an environment for their horses.

Strasser takes the position that shoes are never an option and if any of her Certified Hoofcare Specialists are found applying shoes, their certification is revoked. Her method requires that all horses must remain barefoot, even if that means the horse is in pain for a period of time. This makes it difficult for many farriers to consider implementing some of her teachings.

American Farriers Association Guidelines

The American Farriers Association (AFA) has a certification program in which the participants trim and shoe a horse according to certain guidelines consistent with what is considered to be traditional method. Traditional farriery is the most widely accepted method for balancing a horse's foot and is conservative in nature. Basically, the goal is to prepare the foot so that the hoof wall is level, and the pastern, shoulder, and hoof angles are all parallel. The sole and frog are pared conservatively and any flares on the outer wall are removed.

Basic Principles

This short list of different approaches to balancing the equine foot is by no means a comprehensive one. There are many more theories on this subject, but these are the ones that currently are most widely accepted and used. While the techniques vary, many of the principles of trimming have striking similarities. For example, a conservative approach is generally accepted as the best way to restore or maintain soundness. Over-trimming a foot often creates negative results and puts the horse at risk for further complications.

Routine Paring

The sole is the main defense against injury to the fragile coffin bone (third phalanx [P3]) and should be treated as such. Removal of the dead, flaky sole is generally accepted, but that is where the knifing needs to stop; further removal of live sole is contraindicated. The frog and bars should be treated in a similar manner, since they play an important role in the support and integrity of the hoof. Only the ragged, unstable portions should be removed so that the foot functions properly and does not trap debris.

Managing Flares and Crushed Heels

Flares of the hoof wall weaken the foot and tend to get worse over time unless they are addressed properly by the farrier. Flares predispose the foot to quarter cracks, a problem encountered when the foot is not properly balanced. A horse's foot will tend to flare out as the biomechanics change in association with the long toe/low heel (LT-LH) syndrome, poor trimming, or simply neglected feet.

The horn tubules are the tiny hairlike strands that make up the hard hoof wall. They are such a small part of the total hoof that they tend to get overlooked. However, it is important that these tiny structures remain straight so that the integrity of the foot is not compromised and thrown out of balance. Bent tubules are especially a problem in the horse with the LT-LH syndrome (Figure 21-1). Bent tubules are commonly seen as crushed and underrun heels. These horses are, by definition, not balanced and rarely improve without aggressive treatment by a skilled farrier. Attention to proper breakover is critical and shoes are often part of the correction process.

Coronary Band Contour

Another often overlooked part of the foot is the coronary band, the soft tissue just below the hairline responsible for producing the hoof wall. Generally speaking, when the foot is in balance, the coronary band is straight. Peaks and valleys in this soft tissue are indicative that the foot is overloaded in some areas and relatively underloaded in others. The LT-LH syndrome is the perfect example of this situation. The long toe delays breakover and forces the horse back on his heels, creating a jammed appearance to the coronary band directly above the heel region.

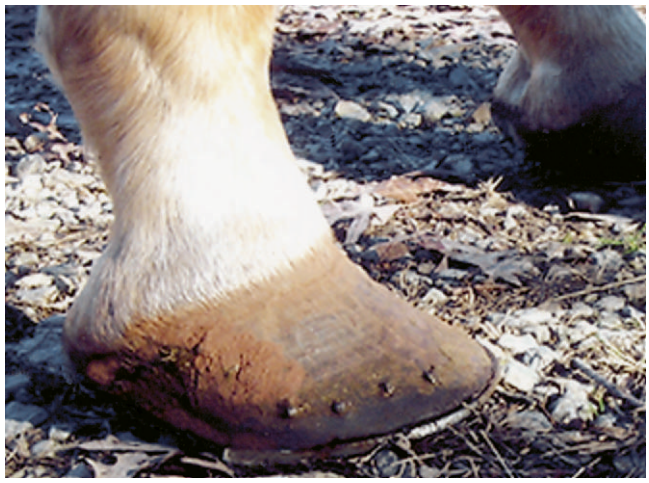


FIGURE 21-1 Example of long toe/low heel syndrome.

An interesting and seemingly little-known fact about the correction of such jamming is that, as the foot is brought back into balance, the soft tissues relax and actually move back into a more stable position over the course of 5 to 15 minutes. This phenomenon can be particularly frustrating to the farrier as he prepares the foot for a shoe, especially if the foot was grossly out of balance before trimming. The solution here is to properly level the foot and give it time to relax, if in fact the foot is going to do so. A hurried approach to nailing a shoe to a foot that is likely to change within a few minutes is less than useful. Doing so condemns the horse to an entire shoeing cycle with its feet still out of balance and the coronary band still out of alignment.

Checking Balance

Once the sole is properly pared, the frog cleaned up, the flares removed, the coronary band evaluated, and a particular trimming method used, the question is whether the foot is now balanced. Even though all of the major factors concerning hoof balance have been addressed, it is still necessary to evaluate whether bony column alignment has been achieved.

Properly taken radiographs are the only way to have conclusive evidence that a particular foot is in balance and the bony column is properly aligned (Figure 21-2). This is one area where it is imperative that a farrier–veterinarian team be able to work together for the benefit of the horse and its owner. (Radiography is discussed in Chapter 10.)

Bony column alignment can be sufficiently defined as having the first phalanx (P1), second phalanx (P2), and third phalanx (P3) properly oriented to one another and the hoof capsule situated in such a manner that load bearing is equal and there is no undue stress on any one area of the distal limb. Figure 21-3 illustrates a proper trim and shoeing. This photograph is of the same pair of feet shown in Figure 21-1, after the LT-LH deformity has been addressed and corrected.

External Landmarks

Few practitioners would disagree that to trim or shoe every foot on every horse with radiographic guidance, while ideal,

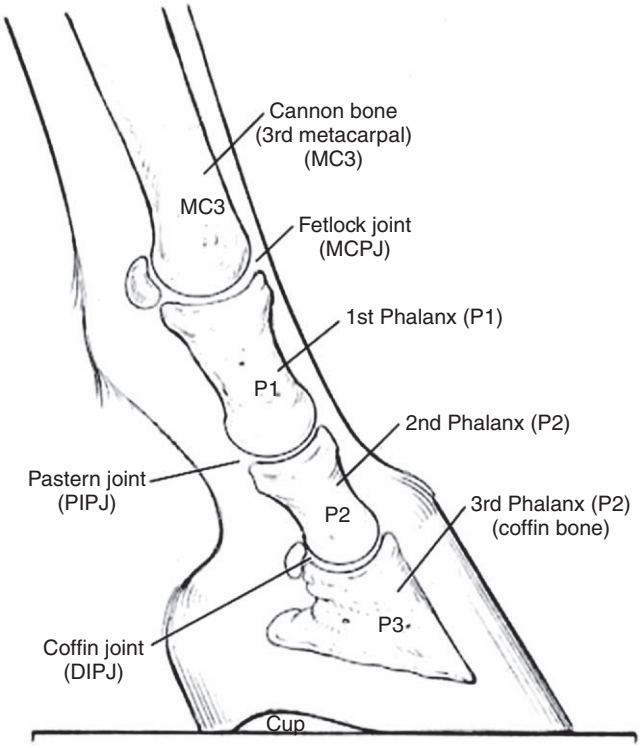


FIGURE 21-2 Proper alignment of digital column.

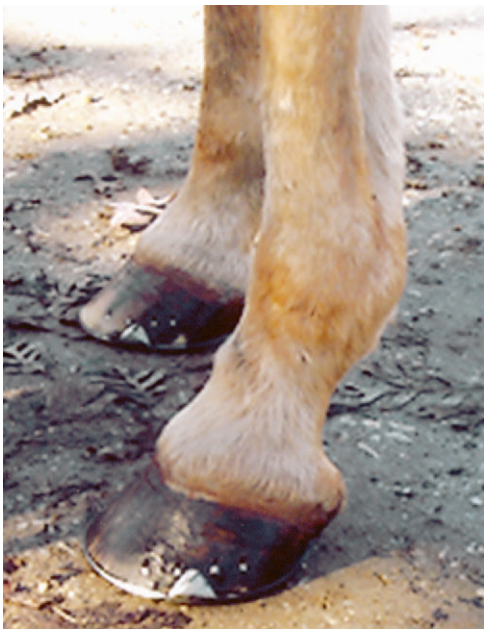


FIGURE 21-3 Properly shod foot.

is not practical. Farriers therefore need to use external landmarks to aid them in balancing the foot. In this regard, the pastern may well be the most useful area of consideration. When the angle of the pastern parallels the angle of the dorsal hoof wall and these two structures are in alignment with the shoulder angle, then the entire limb appears to be in harmony. If the pastern is broken back (fetlock lower than normal) or is broken forward (fetlock too far forward), then alignment is generally accepted to be in need of intervention. Therapeutic shoeing is often indicated in these situations.

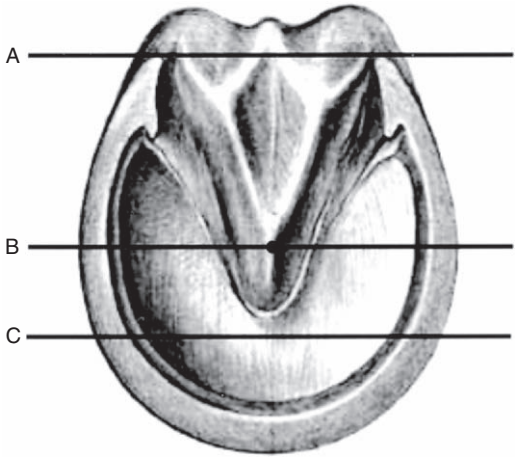


FIGURE 21-4 View of sole of foot. *A,* The widest part of the frog, where the heels should be set; *B,* the widest part of the foot called the quarters; *C,* the area of the sole where the tip of the coffin bone lies directly beneath. NOTE: The sole of the foot should be trimmed so that two thirds of the foot lies behind the apex of the frog and one third of the foot lies in front of the apex of the frog.

Dorsopalmar Balance. When evaluating balance of the foot itself, the farrier should attempt to have one third of the foot in front of the widest part of the foot, and two thirds of the foot behind the widest part (Figure 21-4). It is generally accepted that the heels need to be trimmed back to the widest part of the frog. This strategy gives the horse the proper amount of caudal support and helps keep the heels from becoming underrun. This may be easier said than done, especially given the wide range of hoof types. But, other than verifying balance with radiographs, this may well be the best way to achieve hoof balance from a dorsopalmar (DP) standpoint.

Frog Angle. Since radiographs are not generally used for everyday trimming and shoeing, a critical observation of the frog can be of tremendous value to the farrier by helping him or her develop a mental image of how P3 is situated inside the hoof capsule, and thus DP balance. Careful cleaning of the frog and a minimal amount of paring is all the farrier needs to do to make the following observations. (NOTE: Overparing will distort the frog and will negatively affect the farrier's ability to get a true read on this critical structure, and so generally it is not recommended.)

Observing the angle at which the frog is situated relative to the sole enables the farrier to determine the following:

If the point of the frog is deeply recessed into the surrounding sole and appears to be angling toward the coronary band at the front of the foot, then P3 is likely doing the same (Figure 21-5). In this situation, the foot is said to be *caudally rotated* (the rear potion of P3 is lower than the apex, when viewed from the side). Proper trimming of this foot would entail removing more toe than heel from the bottom of the foot to properly derotate the hoof capsule relative to P3. Radiographs would show an overabundance of hoof material forward of the point of breakover that consequently impedes proper breakover.

If the point of the frog appears to be diving downward out of the bottom of the foot, with the rear portion of the frog recessed up between the heels, then it can be assumed that the foot is cranially rotated and that P3 is pointing downward as well (Figure 21-6). Trimming this foot involves the removal

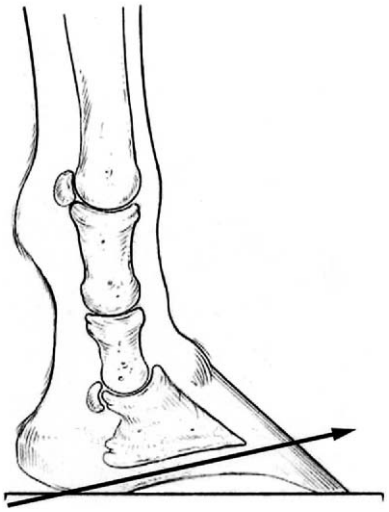


FIGURE 21-5 Example of caudal rotation of the coffin bone.

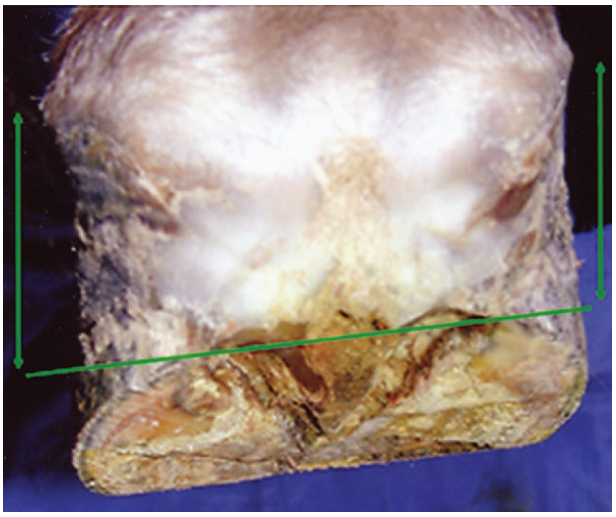


FIGURE 21-7 Example of mediolateral hoof imbalance.

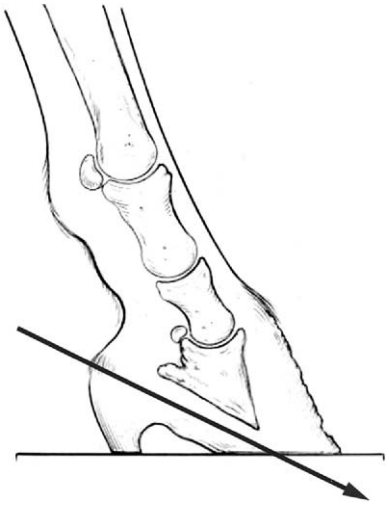


FIGURE 21-6 Example of cranial rotation of the coffin bone.

of more heel than toe to properly align the hoof capsule relative to P3.

In both situations, it is the palmar angle of P3 that the farrier should be focusing on. After derotation in a cranially rotated foot (due either to clubfoot syndrome or laminitis), the strain placed on the deep flexor tendon from lowering the heels must be addressed. Often the addition of either wedge pads or a wedge shoe or cuff is indicated. Not addressing this final step will cause tendon pull and misalignment and will negatively affect the performance of the horse.

Mediolateral Balance

Once DP balance has been achieved, the other major balance consideration is mediolateral (ML) balance. This part of balancing the foot is critical and ensures that load bearing is as uniform as possible. Overloading one side of the foot not only exerts too much pressure on the digital cushion and strains the ligaments in the foot, but it negatively affects the joints of the entire limb. An analogy would be if a person were to bend a finger sideways. The bone alignment would not only involve finger being angled to one side, but also the joint space would be narrower on the side of the finger that is in the direction of the bend, whereas the joint space on the side away from the bend would be wider. If this finger were required to bend frequently and be under load or tension for long periods of time, pain would start to develop.

Mediolateral balance is important because it means that P3 is in harmony with the hoof capsule as viewed from the front (or rear) of the foot. In a normal foot, ML balance begins with having the heel lengths the same on each side of the foot. When the foot is viewed from the heels to the toe, there is only one plane; however, it is possible to have a flat foot without having the foot in ML balance (Figure 21-7). A compact disc, for example, is a flat object. When tilted to one side, it remains a flat object, but now it is out of balance.

When ML balance is not attained, the horse's foot generally lands poorly. Ideally, the foot should land either flat or slightly heel first, but the medial and lateral heels need to contact the ground simultaneously, not one then the other. In cases in which the feet are severely out of balance, the horse will actually contact the ground with one side of the foot, and a flopping sound can be heard as the other side of the foot hits the ground. This problem is particularly noticeable when the horse is walked on concrete or asphalt.

It can be a real challenge for the farrier to bring this horse back into proper balance, especially if the horse has little foot with which to work. It can, and often should, take several shoeings to achieve proper ML balance in such a foot. The farrier is often able to use a medial or lateral wedge to artificially manipulate the hoof capsule orientation in severely affected feet. However, caution needs to be taken not to overcorrect the foot and cause misalignment in the opposite direction.

Regular Trimming Interval

It is important to restate here the need for evaluations at regular intervals of 5 to 8 weeks to maintain proper hoof conformation and balance. When the balance is evaluated regularly, small corrections can be made and major problems avoided. Hoof care is a maintenance issue for the majority of horses. Only a small percentage of them require continuous correction to balance a diseased or distorted foot. When hoof distor-

tion or disease occurs, a shorter trimming cycle often is indicated to ensure that the foot is returned quickly to a balanced state.

Tools and the Trained Eye

Farriers need tools, and there are some very useful tools on the market that make their jobs faster, safer, and more efficient. At the same time, however, the farrier must be able to evaluate a horse with little more than the naked eye. Farriers have been practicing hoof care since the horse was first domesticated centuries ago. Farriers and blacksmiths were responsible for keeping horses sound long before all of the modern farrier equipment came into play.

Hoof Gauge

One of the tools often found in a farrier's box is some type of hoof gauge. This gauge is used to measure hoof angles, and some of the more sophisticated ones check the mediolateral balance as well. The problem with this tool, along with many others, is the potential for misuse. A disfigured foot, as well as one that has been improperly trimmed, will give a false reading. Thus it is imperative that the farrier be able to judge the balance of the foot in relation to the limb using only his or her eyesight. The hoof gauge is a useful tool in the proper hands and little more than a crutch when used as the sole means of attaining balance.

Trimming the Foal's Hoof

Most farriers would agree that the proper time to begin trimming foals is somewhere around 6 to 10 weeks of age, depending on the foal's conformation, handling ability, and growth rate. Like any mature horse, foals have their own unique circumstances that determine how and when hoof care is initiated. These youngsters are born with a soft hoof wall so as not to damage the uterine lining of the mare. These feet remain soft for a few days and gradually harden as the foal becomes active.

Club Foot

Often the foal's feet develop a dished appearance; this defect needs to be addressed by the farrier, as it may be the beginning of club foot syndrome. Even with routine attention to their feet, some foals develop one or more club feet. Lack of exercise and improper diet are contributing factors. These babies need to constantly be on the move; it is what Mother Nature intended. What Mother Nature did not intend was for these young horses to be force fed so that they are full size by the time they are 2 years old. The common practice of overfeeding young horses so that they can compete in the show ring or on the racetrack has certainly predisposed many of them to having club feet (see Chapter 11).

Distortion of the hoof capsule results from abnormal forces on P3 via the deep flexor tendon. As the condition progresses, P3 is forced to rotate palmarly. This rotation pulls on the lamina and eventually causes a dished look to the dorsal hoof wall, and the toe is worn away from constant abrasion. Left untreated, this condition can lead to separation of the white line and laminitis if the condition progresses.

Early and vigorous intervention for these young feet is critical, as new hoof growth follows the shape of the existing foot. If the foot is in balance, it is likely that any new growth will follow suit and the foot will remain in balance, assuming there are no diseases interfering with normal, healthy hoof development.

FIGURE 21-8 Trimming the foal's hooves.

First Trims

The foal's first few trims are a critical time in his life. Not only do the feet require careful attention, but the foal's psyche is being stressed at this time. Nowhere in this baby's genetic makeup does it say that it should be willing to let a human handle its foot while it stands on the other three. A relaxed atmosphere, with its dam nearby, is where the foal's initial foot handling needs to take place (Figure 21-8). Daily training by the owner, in conjunction with patient trimming by the farrier, will result in a horse that is easily handled and routine hoof care that is safe for everyone involved, including the horse. Strict attention to diet and exercise also needs to be in place so that the foal can grow the healthy foot required to support its rapidly growing body. Assistance from an equine nutritionist or a veterinarian will ensure that the foal develops properly with few problems.

A conservative approach to trimming is paramount. Farriers need to be conscious of the fragility of foals' feet. The hoof capsule is the only thing between the sensitive structures inside the foot and the ground surface that the foal walks on. These feet are in a continuous state of development and are very vulnerable to overtrimming. While minor adjustments can be made, aggressive trimming is not advisable.

Most foals are active enough that there is little buildup of dead, flaky sole, and the farrier should make every attempt to leave as much sole intact as possible. It is far better that the farrier returns more often and trims only a little each time than to try to make major corrections to a foal's feet in one visit. Getting these babies started properly will have a long-term impact on the quality of their feet. On rare occasions on which major correction is needed (e.g., some cases of clubfoot), glue-on cuffs and shoes may be necessary (see Chapter 11).

SHOEING

Once balance has been achieved through trimming, the possibility of shoeing the horse enters into the equation. Not all horses need shoes, and some cannot remain sound

without them. If horse owners, farriers, and veterinarians can agree on that point, then a rational, educated discussion can follow. Those who are completely opposed to shoeing a horse need to focus on the following information to truly understand why it is often necessary. By the same token, those who feel that all horses must be shod to remain sound have missed the boat as well.

The question of whether to shoe a horse, and the rationale behind the decision, must be examined in terms of whether protection, support, traction, or gait alteration is the goal, or perhaps some combination of these. The intended purpose for this horse must be considered. In other words, what is the horse's job going to entail and how will the application of shoes help the horse to be better at what it does?

A term often used around the horse industry is *corrective shoeing*. The author would submit that there is simply correct shoeing and incorrect shoeing. Perhaps a better word to use in place of *corrective* would be *therapeutic*, which would direct discussion about the application of shoes in one of two directions: *corrective* for horses that simply are routinely shod and *therapeutic* for those in need of a more sophisticated shoeing package to improve the horse's mobility. Simply put, a proper job will fall into either one category or the other. It is important to state that a correct shoeing job is much more than simply applying shoes in a mindless fashion. Rather, the farrier should constantly strive to properly balance the foot and keep it healthy so that therapeutic shoeing does not become necessary.

Protection

To determine how a horse will be shod, the farrier needs to be familiar with the environment in which the horse lives and works. He needs to be aware of what is expected from each horse and any physical problems that might hinder the horse's performance. Understanding that every horse is different enables a knowledgeable farrier to shoe each horse to enhance the horse's natural abilities and reduce unnecessary physical stress on the horse at the same time. Protection of the hoof capsule can range from a temporary need, to the relatively simple shoeing, to the very complex shoeing package that not all farriers are able to provide.

Hoof Boots

For the short, infrequent trail ride, a pair of protective boots, such as Old Mac Boots or Easy Boots (Easy Care, Inc., Tucson, Ariz.), might provide sufficient protection. The feet must still be trimmed on a regular basis, and proper fit with this type of protection can be a problem, since the size of the foot varies based on how recently it was trimmed. Like any application, these devices need to be applied carefully and fit properly. Not doing so can injure the feet that the farrier is trying to protect.

The major disadvantage to using a boot (as opposed to a shoe) is the fact that boots are not customized for each horse. There are adjustment straps, but these boots are manufactured for a generic foot, not for each horse specifically as the shoes are that a farrier applies. Therefore the horse must accept where the breakover is and where the caudal support is and basically adapt as long as the boots are on its feet.

Boots have the added disadvantage of being an ideal place for water, rocks, and mud to collect. Any abrasion to the sensitive structures such as the heel bulbs can cause inflammation, abscessation, and abnormal heel growth. Therefore hoof boots should not be left on indefinitely and should be monitored diligently and daily. Boots that are left on for extended periods of time can also cause thrush, bruising, and white line disease. The debate remains whether horses wearing boots actually get the proper support and protection that they would otherwise get from being shod.

FIGURE 21-9 Types of impression material.

Simple Rim Shoe

A simple, light rim shoe is all many horses need to maintain hoof wall integrity, particularly when worked on rough ground. As protection requirements increase, generally so does the width of the shoe (i.e., the web). Some examples of a wide-webbed shoe are the St. Croix Eventer and Extra EZ (St. Croix Forge, Inc., Forest Lake, Minn.), the Kerkhart SX8 (Kerckhaert Horseshoes, Vogelwaarde, Netherlands), and the Natural Balance Shoe (Equine Digital Support System, Penrose, Col.). These shoes, when properly applied, all offer the normal foot adequate support and protection. Extremely flat-footed horses also benefit from this type of shoeing, as it provides just enough clearance between the foot and the ground to avoid sole bruising.

Pads and Packing Materials

Thin-soled horses often are shod with some type of pad between the foot and the shoe. Full pads provide protection to the entire bottom of the foot. It is necessary to apply packing material under the pads to prevent mud, rocks, and the like from getting under the pads. Most farriers have a preferred packing that works well for them and they often carry two or three types to use, depending on the circumstances. Some examples of these packing materials include Vettec's Equi-Pak (Vettec, Oxnard, Calif.), Forshner's hoof packing plus oakum (KM Elite Products, West Sussex, England), and the wide variety of sole support putties available, including Advance Cushion Support (Nanric, Lawrenceburg, Ky.), Provil Novo Soft Putty (Heraeus Kulzer, South Bend, Ind.), and impression material that is available in soft, regular, and firm (Figure 21-9).

Each form of hoof packing has unique qualities that make it the right choice for a particular horse in a given situation.

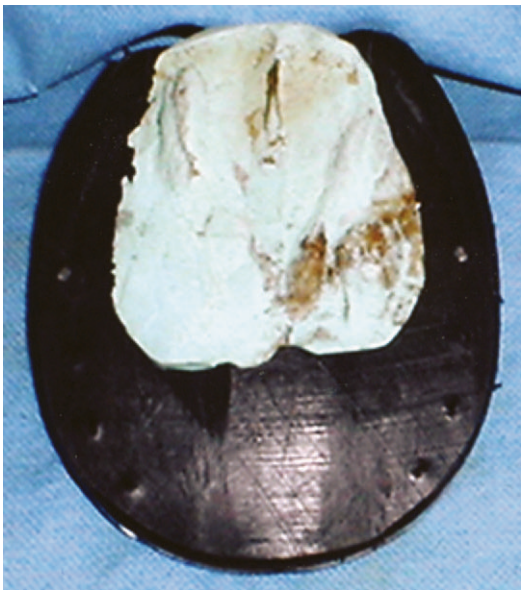


FIGURE 21-10 Example of wedge and impression material (solar view) removed from the horse's foot.

Some are easier to apply than others, and the costs vary greatly. Figure 21-10 shows a shoe/wedge pad combination that has been removed from the horse's foot. Visible is the putty that was applied from the point of the frog to the heels, leaving an impression of the solar surface of the foot in the area that is typically supported with the use of the impression materials.

Sometimes the farrier adds only a rim pad that does not cover the entire solar area of the foot yet provides the horse with a slight amount of cushion when needed. A snow pad is sometimes used where snow buildup in the feet is a problem. Snow pads are available in both the rim style as well as the full pad.

Hospital Plates

The most complex form of foot protection is the shoeing package that includes a hospital plate or hospital pad. These devices are used when treating foot disease that requires a protective covering for the foot yet allows periodic access to the solar surface of the foot. The owner or veterinarian can remove the plate, treat the foot, and reapply the plate without daily involvement of the farrier, who may not be available on a daily basis.

Support

Supporting a foot with a shoe has various meanings for different horses. For some it means providing caudal support that the bare foot may lack naturally. For others it is part of a more complex shoeing package that may involve pads and sole support. The farrier needs to keep in mind the incredible difference in the size of the equine foot relative to the size of the body it is supporting. Careless breeding programs often dictate how much artificial support will be necessary for the horse to remain sound throughout its entire life.

Additional support is often indicated when there has been a traumatic injury to the foot. A shoe can be amended to take the place of missing hoof wall so that the horse's gait is not affected. When an injury occurs, it is advisable to provide support to the opposite foot and leg, since it is now bearing more than its share of the load. Failure to do so can lead to laminitis and possibly permanent damage to the overloaded foot.

FIGURE 21-11 Dalric cuff.

Temporary Support

When a horse suffers an injury to his feet, temporary support can be the ideal option, especially in the acute stage of the insult. For example, when a horse is in the acute stages of laminitis, proper support and protection of the fragile foot is critical. However, the last thing the horse needs at this stage is additional concussion to the hoof capsule from attempting to nail on a shoe. In such cases, a Dalric cuff (Nanric, Lawrenceburg, Ky.) (Figure 21-11) or similar device can be taped or glued to the hoof, thereby providing the horse with protection that can be easily amended to meet the needs of the affected limb.

Daily attention and modification are often required when managing diseases of the horse's foot. The use of a temporary shoe makes it easier for the veterinarian and farrier to make adjustments in the treatment protocol; also, it is less stressful for the horse and is usually a more economical approach for the horse owner.

Traction

Terrain, usage, and rider ability are all factors that will determine how much, if any, extra traction is necessary when shoeing the horse. The typical rim shoe provides traction by holding dirt in the crease (or fuller) of the shoe. However, this small amount of traction may not be enough, depending on the ground conditions.

Studs and Caulks

Drive-in studs or screw-in caulks offer the horse more traction than a simple crease in the shoe. These devices can often be found on the shoes of fox hunters and other horses that compete on grass. The advantage to screw-in caulks is that the horse owner can remove them when the horse is not being ridden, thereby reducing any stress on the horse's legs that the extra traction might cause. Both the studs and the caulks come in a variety of sizes, depending on the individual needs of the horse and rider.

The other determination that the farrier must make is how many caulks or studs to use and where they will be located on the shoe to best help the horse. Typically, they are located in the heel region of the shoe; for more grip they can also be placed at the toe corners (also known as the toe pillars). The

farrier must install these traction devices in a balanced manner on the shoe so that the foot is not torqued sideways as it lands.

A more traditional type of caulk is one that is forged into the heel of the shoe, thereby providing a small amount of traction for the horse. An extreme example of this type of caulk can be found on the shoes of pulling (draft) horses, particularly those that compete in sled-pulling contests.

Borium

Another widely used traction device is the type that is welded onto the shoe with either an oxygen/acetylene torch or by heating in the forge. This material has several common names, including borium, Carbraze, and Drill-Tek (Hartwell Industries, Houston, Tex.). Regardless of the brand name, the material is applied in basically the same manner to provide the horse with a variable amount of traction. These products are packaged in rods and contain tungsten-carbide particles in a copper-nickel matrix.

The variability in the amount of traction provided is determined by two factors: the size of the tungsten-carbide particles, and the amount of material that is welded to the shoe. The particle sizes available are listed as small, medium, and large. Every effort should be made by the farrier to provide the horse with enough traction to avoid injury; but a conservative approach must be followed so as not to predispose the horse to problems or create an imbalance on the shoe itself.

The "bigger is better" idea does not apply here. In fact, adequate traction can be obtained with minimal application of one of these products. The heel region of the shoe is the most common area where the tungsten product is applied. Where more traction is needed, spots can be added to the front half of the shoe as well. Every horse requires its own unique application of this type of traction. Another option that works well for many horses is the use of borium-tipped nails. Results are often comparable to those achieved with welded-on traction, but the advantage is that the nails can be removed, if necessary, without removing the shoe itself.

A side benefit to using a welded-on product is that it will extend the useful life of the steel shoe to which it is applied. Carriage horses, buggy horses, and any other shod horse that must walk on asphalt can benefit from welded-on traction in most cases. In situations in which it is beneficial for the horse to have the toe of the shoe wear away over time to help with breakover, it would be contraindicated to weld any material forward of the breakover point.

Figure 21-12 shows a drive-in stud on one heel and borium on the other heel. Ordinarily, either one type of traction or the other is used on a particular pair of shoes. This photograph is simply for demonstration of the two types of traction device.

Toe Grabs

The racing industry, Thoroughbred racing in particular, uses a variety of shoes, many of which have toe grabs. These devices are designed to grip the ground surface, thereby allowing the horse maximum purchase and diminishing the slippage often observed when horses move at higher speeds. The use of toe grabs is a somewhat controversial topic among trainers, as some are convinced that toe grabs add undue stress to the lower limbs and are responsible for many of the career-threatening injuries that racing horses sustain each year. Toe grabs increase the risk for catastrophic breakdown injuries.

FIGURE 21-12 Example of borium (on the right) and a stud set in the heels on the left.

Gait Alteration

Gait alteration is a specialty for many farriers. Typically, the gait is affected by the application of heavy pads or weighted shoes. Horse breeds in which such gait alteration is routinely practiced include American Saddlebreds, Tennessee Walking Horses, Arabians, and several others. The application of this type of shoeing becomes a specialty for the farrier, owing to the criteria that must be followed for the horse to be shown according to the rules and regulations of the show ring.

A horse's movement can also be changed by the application of a lighter shoe, such as an aluminum shoe. This approach is commonly seen in Quarterhorses, in which the desired effect is a flatter knee motion and more of a sweeping look, as opposed to a bold, high step.

Selecting the Appropriate Shoe Type

When the shoeing is expected to last through a normal cycle, as is the case with most shod horses, it must then be determined what exactly will be applied to the foot in the way of a shoe. The question again is why this horse is being shod and what the ideal shoe is for the job that the horse is expected to do. The possibilities are almost endless. Many years ago, the most important consideration was the thickness of the steel bar stock from which the shoe would be forged. Today, farriers have the ability to choose shoes based on type of material, weight, and web width, not to mention the scores of modifications that can be added to virtually any shoe, including clips, enhanced breakover, and traction devices.

Material

Choosing the right material from which a shoe is made has several components. Does using a lighter material such as aluminum or titanium improve the way the horse moves? Will this horse benefit from the use of a wide-webbed shoe that offers a greater amount of support, versus a narrower rim-type shoe? Is steel necessary, since the horse will be worked in adverse conditions and needs the durability that these shoes

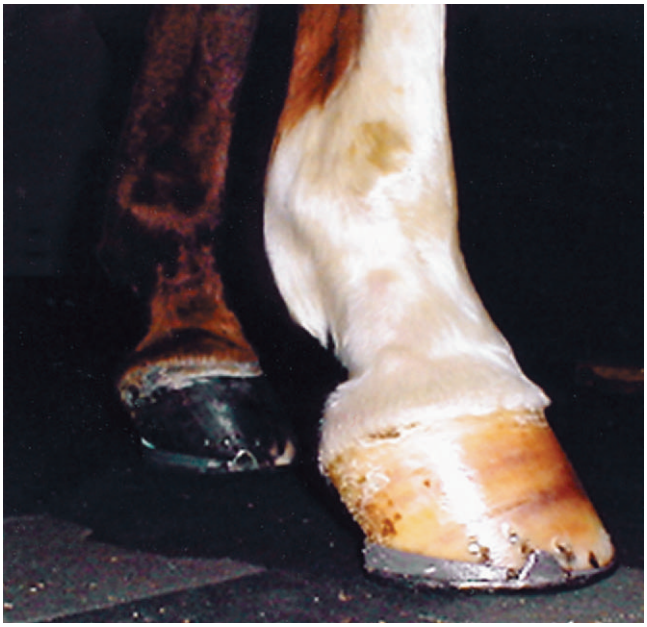


FIGURE 21-13 Example of hoof shod with a 5-degree wedge.

offer? Or is a plastic or rubber shoe a better alternative to accommodate therapeutic applications?

Selecting the right material is critical when choosing a shoe. For horses that tend to grow a long toe or when breakover issues arise, aluminum or plastic shoes work well, because as the foot grows forward, the shoe wears at the toe, thereby easing breakover and keeping the foot better balanced throughout the shoeing cycle. Another major consideration is the horse owner's pocketbook. Most people are not willing to spend extra money for higher priced shoes unless there is a justifiable reason to do so.

Heel Elevation

When it becomes necessary to elevate the heels on a horse to realign the bony column for proper balance, the farrier has several options. Wedge pads applied between the foot and the shoe (Figure 21-13) can provide the lift needed and, if used as a full pad, can also aid in protecting the sole and help keep impression material in place. These wedge pads, which are usually plastic, work well in many situations, but this package is often replaced with a wedge shoe if long-term elevation is required. Wedge shoes generally are made from aluminum, since the weight of the shoe is a consideration and often a limiting factor as the thickness of the shoe increases.

Rail Shoe. One of the newer concepts being used to elevate the heels is the use of rails that are applied to the ground surface of shoe. The advantage of this system is that the rails can be exchanged for larger or smaller rails, depending on the needs of the horse, without removing the shoe. Thus, application time and expense are reduced, not to mention the reduction in stress/damage to the hoof wall from frequent shoeing. A regular shoeing cycle is still needed, but intermittent adjustments can be easily made in between shoeings. The downside to the use of rails applied to the ground surface of the shoe is that, on soft ground, the wedge sinks and no longer provides the lift necessary to keep the tension off the deep flexor tendon.

FIGURE 21-14 Air-ride or rock-n-roll shoe and full rocker shoe.

Rock-n-Roll Shoe. Ric Redden has been using a system commonly known as the *rock-n-roll method*, whereby the ground surface of the shoe has both the toe and heel portion tapered, which allows the horse to find the breakover point that is most comfortable. The term *air-rail* refers to the appearance and function of the modified shoe using a flat shoe, as heel elevation and thus better alignment is achieved without anything physically raising the heels. A large majority of the horses that wear this type of shoe are being treated for laminitis, club foot, or other hoof diseases. There are now shoes available called *full rocker shoes* that allow a normal trim, with this shoe applied to achieve the same purpose for optimal alignment and breakover (Figure 21-14).

Shoe Placement

Maintaining balance should be the goal of the farrier when selecting shoes for the horse. Traditionally speaking, shoes are perimeter fit, meaning that the edge of the shoe is placed all the way to the edge of the trimmed hoof. In many cases, this approach is detrimental to the horse if the point of breakover needs to be brought back, closer to the point of the frog. Working from properly taken radiographs is the only way to be absolutely sure that breakover is positioned optimally. As it is unrealistic to think that this approach is possible with each shoeing, farriers must learn to use landmarks on the horses' feet to guide them in positioning the shoe correctly.

Shoe placement will vary from horse to horse, but there are general guidelines that should be followed. For proper support, the heels of the shoe need to, at the very least, cover the entire heel on each side. Ideally, the shoe would have about a dime's width of material past the hoof wall to allow for expansion and growth. The term *short shoeing* is used to describe a situation in which part of the heel is left uncovered by the shoe. This situation is extremely detrimental to the horse and will cause pain and eventually lead to the development of corns where the hoof wall becomes the bar of the foot (commonly referred to as the seat of the corn).

Short shoeing is often seen when an inexperienced farrier uses a shoe that is too small for the foot. It is seen frequently in horses that are notorious for pulling shoes. A much better option would be to have the farrier use the proper size shoe and box (i.e., taper) any part of the shoe that sticks out past the hoof wall. The boxing can be done with a hammer, rasp, or grinder. When the horse is provided with the proper amount of support, shoe pulling is kept to a minimum and lameness issues are avoided.

Optimal Breakover

The next factor to consider is the front portion of the foot. The farrier must determine where the breakover point needs to be

and how it will be worked into the overall shoeing scheme. About 99% of the time this is accomplished through simple observation. Whether the farrier is shoeing a horse that has been barefoot for a while or a horse that has been shod, the ability to observe the foot or the old shoe for wear patterns will greatly enhance the chances of putting the point of breakover where it needs to be for optimal performance.

Not only will a skilled farrier be able to place the breakover properly relative to P3, but he will also be aware of the direction of breakover and build that into the shoe as it is prepared for the hoof. In a perfect world, all horses would break over perfectly in the center of the toe. The reality is that a lot of them do not and should not be forced to do so. When careful consideration is given to the direction of breakover, a lot of fatigue factors can be avoided and the transition to a new set of shoes made easier.

Both the bare foot and the worn shoe have important information for the farrier—information that, if cast aside, would be a true waste of useful data. Providing proper breakover and direction of breakover is not overly difficult, it just takes some forethought and a little extra effort. These breakover features on the shoe can be applied using several different methods. One way is to forge the modifications while the shoe is hot. Common modifications to enhance breakover include a rolled toe, rockered toe, and a square toe. If working the shoe hot is not an option, then material can be removed from the shoe with either a rasp or a grinder. Results similar to the forged shoes can be attained; occasionally, both methods are employed on an individual shoe.

Placement. Placing the shoe on the foot for proper breakover varies greatly from horse to horse. Lateral radiographs enable the farrier to pinpoint the "perfect" shoe placement. Since that is often not an option, the farrier has to go back to the landmarks on the foot as a guide. Generally speaking, the point of breakover is approximately 1 to 1½ inches forward of the point of the trimmed frog. Obviously, the dimensions in a draft horse will be quite different from those in a miniature horse. This elusive point is where the area directly under the apex of P3 is leaving the ground as the horse starts to move forward. A look at a bruised sole will reveal where the crescent ring is on the sole relative to the trimmed frog. This bruising is indicative of the location of P3 (not to mention the fact that the balance of the hoof or its protection needs to be re-evaluated).

One of the methods of shoeing to maintain well-balanced feet is the EDSS. This type of shoeing applies the NBS principles and uses a shoe that, by its design, reduces breakover when properly applied (Figure 21-15). It should be mentioned that every type of shoe must be applied properly after a balanced trim. Anything less produces dismal results and leaves the horse owner and farrier feeling like the system itself has failed.

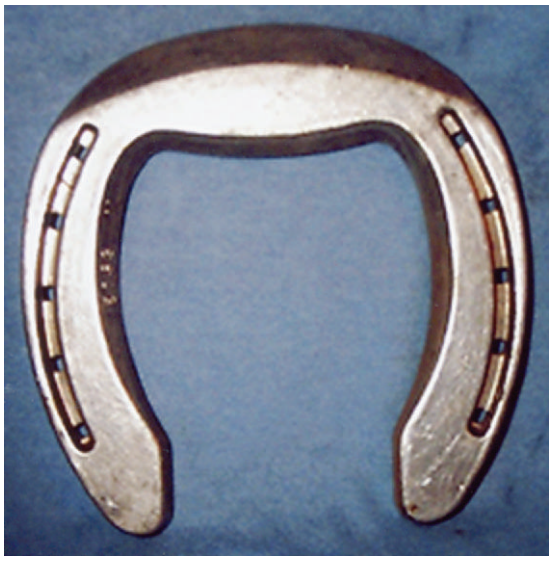


FIGURE 21-15 Example of Natural Balance shoe.

FIGURE 21-16 Shoe with front clips.

Clips and Other Modifications

Once the farrier has selected the proper shoe for the horse, a few final decisions must be made before the shoe is nailed or glued to the foot. If the horse has a good quality hoof wall and no gait faults, then perhaps the plain shoe can be applied at this juncture. If, however, the horse needs a shoe that is modified to ensure its stability, then the farrier must make those changes at this time. One of the most recognized modifications is the addition of clips to aid in the retention of the shoe for the full shoeing cycle.

Generally speaking, side clips (located between the second and third nail holes) are seen on front pattern shoes (Figure 21-16) and quarter clips (located between the first and second nail holes) on hind pattern shoes (Figure 21-17). These clips are designed to reduce the shear force placed on the nails each time the foot contacts the ground. If the clips are properly applied, it can be an invaluable addition to the shoe for both the horse and the farrier. Some farriers prefer to use a single toe clip in the center of the toe. This approach is often seen on horses shod in Europe and on many of the draft horses in the United States.

Clipped shoes can be ordered from the manufacturer or can be pulled by the farrier at the time of shoeing. Adding a clip to a shoe is simply a matter of heating the shoe enough to displace a bit of the metal from the outer rim of the shoe and

FIGURE 21-17 Shoe with hind clips.

forging it down to a V shape approximately as tall as the web of the shoe is wide. Clips can be produced using a variety of tools, and each farrier has his or her own style when it comes to this task. Regardless of how the clip is produced, it must then be properly fit to the angle of the hoof wall; hot-seating the clips into the wall produces the best results and has a better overall appearance.

Nails or Adhesive

Up to this point, the farrier still has the option of either nailing or gluing the shoes to the feet. The gluing of shoes is becoming a more common practice and is particularly beneficial when working on a horse with foot pain, especially those with laminitis. These horses simply cannot take the concussion produced by the nailing process, and farriers find it much less stressful for all involved to simply glue the shoes to the feet.

Before deciding that gluing is in fact the proper way to proceed, the horse's situation must be carefully considered. The majority of horses live outside, exposed to mud, rocks, sand, fences, and a whole host of other factors that might make it impractical to consider using an adhesive to apply the shoes rather than nails. If, however, the horse owner is willing and able to make the necessary provisions to ensure reasonable success, then the use of an adhesive can be an effective way to get the job done.

Adhesive Options

Three widely used adhesives are on the market today. Equilox adhesive (Figure 21-18; Equilox International, Pine Island, Minn.) is used for both hoof wall repair and as a way to glue on horse shoes. This product is a two-part system that, when combined, creates a substance that will harden to approximately the consistency of a horse's hoof wall and can then be rasped to attain the hoof shape that the farrier desires.

Vettec Hoofcare Products (Oxnard, Calif.) manufactures two products, both containing urethane components, that can be used to bond a shoe to the hoof when nailing is not an option. Adhere is the name of the product that was specifically produced for the application of glue-on shoes. Super

FIGURE 21-18 Equilox adhesive. *(Equilox International, Pine Island, Minn.)*

FIGURE 21-19 Super Fast adhesive. *(Vettec Hoofcare Products, Oxnard, Calif.)*

Fast (Figure 21-19; Vettec Hoofcare Products, Oxnard, Calif.) is a urethane product that has the unique capability of hardening very quickly (approximately 30 seconds) and was originally designed to be used for hoof wall repair. This product works well in some situations, but many farriers feel that the quick set time of the Super Fast product is a disadvantage, particularly when working alone and desiring the time necessary to make final adjustments to shoe placement. Both the Adhere and the Equilox products take several minutes to reach an initial hardness and considerably longer to reach a final cure.

Hoof Preparation

The application of a shoe using adhesives requires several additional provisions that are often not necessary when

nailing on shoes. The hoof wall and sole must be absolutely free of debris to ensure that the adhesive will have excellent contact where needed. Next, the shoe, which is typically aluminum to reduce the total amount of weight, must be free of debris and wiped with denatured alcohol to remove any oils that could impair the adhesive. At this point, the farrier should be wearing disposable gloves so that no contaminants or oils are transferred to the shoe or foot.

Horse Compliance

The final and perhaps the most critical component is patience on the part of both the horse and the farrier. As mentioned earlier, these adhesives need time to cure and must remain unloaded (i.e., non–weight bearing) for several minutes. This procedure can certainly test the patience of the horse and the physical ability of the farrier. In cases in which the horse is either in too much pain or simply too uncooperative to be still for the length of time needed, sedation administered by the veterinarian is likely the key to a successful shoeing.

There is little worse than having everything prepared and in place, and the shoe set where the farrier desires, only to have the horse jerk away and slam the foot on the ground before the glue has had time to set. When this situation occurs, the process must be completely restarted, resulting in frustration for the farrier and additional expense for the horse owner.

In this day of modern medicine, equine professionals need to be willing to employ the services of a qualified equine veterinarian when the task at hand is difficult and restraint of the animal is paramount. Twitches, hobbles, ropes, and other mechanisms designed to force the horse into submission do not work well in these situations, and the desired results are rarely achieved when the horse cannot or will not be cooperative. Horses that behave poorly are either apprehensive of pain or are in pain when attempting to flex, bend the leg, or stand for prolonged periods of time on a painful leg. These horses must be understood, the problem determined, and patience and shorter periods of time allowed for the foot to be off the ground.

Avoiding Sole Pressure

A critical step in the shoeing process, whether the shoes are applied with an adhesive or nails, is to make absolutely sure that there is no sole pressure as a result of the shoe application. It is very easy for adhesive material to seep onto the sole, even if the shoe has been properly seated out (beveled on the solar surface of the shoe from the nail holes inward toward the center of the foot). Sole pressure can also be encountered when nailing a shoe to the foot. The horses that are most likely to experience sole pressure by the shoe are those with a thin sole, flat foot, or 0-degree palmar angle and foundered horses in which P3 has rotated and is causing sole pressure. (Laminitis is discussed in Chapters 16 and 18.)

Nail Placement

When the shoe is applied in the traditional way, nail placement is critical. If the horse is low-nailed, the likelihood of that shoe staying in place through the shoeing cycle is greatly diminished. The other extreme can be even more devastating. When the nail fails to exit the wall at the proper height, a 'close' or 'hot' nail could be the result.

FIGURE 21-20 Keratex Hoof Putty. *(Keratex Hoofcare, Wiltshire, England.)*

A close nail refers to a nail that does not initially cause the horse any pain but within a few hours could be a problem. A close nail puts pressure on the sensitive laminae and eventually becomes a source of pain; it can lead to abscessation if not properly treated. A hot nail refers to a nail that has penetrated sensitive tissue, causing pain immediately and usually resulting in some bleeding; the likelihood of an abscess in this area is greater than with a close nail.

Every farrier has had the experience of close or hot nailing and the eventual abscess that ensues. Should the nail need to be pulled, injecting disinfectant into the hole may prevent an abscess from forming. Nail placement is a critical step in the shoeing process and should not be taken lightly. It varies from horse to horse, but generally speaking, the farrier has less than a dime's width margin of error, and often less in a diseased foot.

Clinching

When the farrier clinches the nail to secure it to the hoof wall, care needs to be taken not to crush the clinch deeply into the wall and thus create undue pressure on the sensitive laminae, especially when working with thin-walled hooves. Usually, an ugly clinch is just that: ugly. It does, however, create a place that horse owners could cut their hands when caring for the feet and basically leaves the feet looking neglected. Brittle walls or nails that are still rough to the touch once clinched can be covered with beeswax or disinfectant wax (Figure 21-20). This strategy will keep the clinches from deteriorating and will protect the owner's hands.

TEAM APPROACH

In a nutshell, when a horse has been trimmed for balance and has the right shoe for the job properly nailed and finished, then the farrier has done his or her part to ensure the soundness of that horse. A sound horse is every horseman's

dream, but it is obviously not that simple. To keep a horse sound and pain-free takes the cooperation of three major players: the horse owner, the farrier, and the veterinarian. If any one of these individuals is not a positive part of the equation in maintaining or achieving soundness in the horse, then they rapidly become the problem.

Veterinarian–Farrier Relationship

When everyday, routine work is being performed, the veterinarian and farrier rarely have to interact. It is when problems arise and horses are lame that there needs to be a good working relationship for the benefit of the horse and its owner. Each professional must stay up-to-date on the latest research and technology and be able to apply it in a variety of situations. Doing so ensures that there is no communication gap when situations arise, so problems can be addressed in a calm, timely manner and in a stress-free environment.

Initially, the two professionals often need to work together, side by side, to stabilize a horse with an acute problem. Over time, each then works independently, relying on the support of the other. The primary focus should not be on the individual's contribution but on the horse that each professional is being employed to work on.

Constructive criticism is something that should be openly discussed and accepted from the farrier, the horse owner, or the veterinarian, but it needs to remain just that: constructive. Badmouthing the other parties is unproductive and unprofessional. Every practitioner has a different idea about how a particular problem should be approached, but all need to accept the fact that there just might be a better way, a newer way, or a more efficient way.

A good veterinarian–farrier relationship is priceless. When a farrier needs a good set of radiographs from which to work, he needs to know he can get reliable, consistent pictures that will make his job more accurate and the horse more comfortable. If sedation is required, a veterinarian that is familiar with the horse will know what drugs will work best for that particular animal. Everyone, including the horse, is less likely to be injured in this scenario. Quite often, metabolic disturbances create problems for the horse where his feet are concerned, specifically when the problem is serious enough to induce laminitis. It is critical that the farrier–veterinarian team understand this fact and take measures to prevent the negative consequences, or at least keep damage and pain to a minimum.

When a veterinarian encounters a horse that is in need of hoof care, therapeutic or otherwise, he or she needs to know that there is someone in the area that is educated enough to handle the situation. With today's technology, farriers and veterinarians can communicate via the phone, e-mail, or fax machine and often work together on a horse without having to be present at the same time. Presumably, both professionals are interested in the well-being of the horse. Communicating with each other about the progress, setbacks, and unforeseen hurdles with a particular case helps to enhance the education of everyone involved and ensure that the horse can perform at an optimal level.

Horse Owner's Responsibilities and Requirements

Whereas there are few guarantees in life (and even fewer where horses are concerned), one thing is certain: horse owners must do their part on a daily basis to safeguard the well-being of their horses. They need to keep themselves educated, if for no other reason than to be able to ask the right questions. Once they have surrounded themselves with knowledgeable professionals, they need to use that knowledge on an on-going basis to provide their horses with an environment that is conducive to healthy living.

Every year the costs of maintaining a horse increase, so dedication on the part of the horse owner becomes that much more important. Today's educated horse owners realize that they need more than a veterinarian who stops by once a year to vaccinate their horses. They understand the difference between the farrier who nails on four shoes right out of the box in 30 minutes and the one who is able to keep their horse in balance, pain-free, and working well. They understand that cheaper is seldom better.

Whether the check is written to the farrier, the veterinarian, or the trainer, owners must understand that they are receiving more than four shoes, a few vaccinations, or a 1-hour lesson. They are reaping the benefits of years of practical education and insight, which is difficult to put a price on. Whether prompted or not, these professionals often offer advice, and it should be carefully considered. That said, the trainer should not be offering shoeing advice any more than the farrier should be suggesting a better way to ride a particular horse. When concerns about a particular topic surface, the horse owner should be referred to someone who specializes in that area.

CONCLUSION

When thinking about all of the intricacies involved in the quest to balance the horse, is the conclusion that this lofty goal primarily involves art or science? This author believes it takes both to be successful. The best farriers have enough artistic ability to balance and complement the scientific drive that makes them the leaders in their profession. They have that voice inside that constantly whispers, "What if . . .," and all for the well-being of their equine friends.

22

DISCIPLINE-SPECIFIC HOOF PREPARATION AND SHOEING

OLIN K. BALCH

In this chapter, discipline-specific hoof care is discussed by several well-respected farriers who are experts in their fields. These farriers discuss the specific aspects of hoof preparation and shoeing that are important in maintaining or enhancing the performance or functionality of the horse in the particular sport or discipline in which that horse is required to perform. For example, the optimal shoe type for the draft horse that is used to skid timber in the forest is quite different from that required by the Standardbred racehorse that is trotting a sub–2-minute mile. Nevertheless, as these authors demonstrate, there are more similarities than differences in hoof preparation, regardless of the horse's breed or use.

It was not the aim of this chapter to cover every possible equestrian pursuit, but rather to demonstrate, using specific examples, both the basic similarities in approach and the discipline-specific modifications that may be made to meet a particular need. To that end, trimming and shoeing are discussed for four very different types of horse: the Standardbred race horse, the draft horse, the eventer (which includes the disciplines of dressage and jumping), and the show hunter.

Within each discipline, farriers are challenged to identify and apply to the individual horse very specific and often subtle trimming and shoeing alterations to ensure the finest performance by that horse. Whether hoof care can actually increase a horse's performance beyond its innate ability is debatable. But in disciplines such as Standardbred racing, creating slightly longer hooves and adding weight to the shoes often is necessary to keep trotters from breaking into a gallop or simply not trotting as fast as they can.[1]

Few experienced veterinarians, farriers, or equestrians would dispute that inappropriate or sloppy hoof care hinders equine performance. Equine biomechanical studies have documented the effects of hoof preparation and shoeing on the kinematics of limb movement.[2,3] And epidemiologic studies have identified specific shoe types[4] and hoof preparation[5] as significant risk factors in catastrophic musculoskeletal breakdown in Thoroughbred racehorses. Thus even the small decisions the farrier makes in regard to hoof preparation or shoeing may have a great impact on the horse's health and performance.

TO SHOE OR NOT TO SHOE

As indicated by some of the other authors in this chapter, leaving the horse barefoot may either be alternated with episodes of shoeing or be a substitute for shoeing for some horses performing in some disciplines. Because this aspect of hoof care is a contentious issue, a discussion of whether and when to apply shoes is appropriate.

Brood Stock and Immature Horses

For brood stock and immature horses, the wear to the hoof capsule rarely exceeds normal hoof growth, so routine and timely trimming generally is the only hoof care necessary for these horses. (Young horses with specific problems, such as flexural limb deformities, may require some type of shoe or other device. However, this issue is beyond the scope of this chapter.)

If these horses have such fragile hoof capsules that they require shoeing in their normally undemanding home environment, then the home terrain should be evaluated. The suitability of these horses for the genetic pool should also be assessed. Until proven otherwise, the quality (i.e., robustness, growth, thickness) of the hoof capsule must be considered in large part to be genetically determined. Therefore the routine practice of shoeing brood stock and immature horses to be shown in halter classes should be discouraged and, arguably, prohibited by breed associations.[6]

Performance and Working Horses

For performance and working horses, the wear of the hoof capsule frequently exceeds normal hoof growth and necessitates foot protection, such as that provided by shoeing. For many short-footed horses (the typical Western pleasure or non–Standardbred performance horse) that are worked on abrasive or hard surfaces, historically the question of shoeing is not whether to shoe but when to shoe in the training or conditioning cycle. Sole soreness—an underappreciated and common cause of lameness—frequently is associated with conditioning programs and failure to shoe appropriately as the solar surface of the hoof capsule wears thin.[7]

Anatomic investigation of the horse's digit reveals the presence of an external hoof capsule surrounding the corium-covered living distal end of the limb. This hoof capsule is composed of insensitive (horny) epidermal tissue that arises from the germinal tissues adjacent to the corium (dermis), and consists of interlocking wall, frog, and sole plates. The epidermal covering protects the underlying dermis and its associated vascular and nervous tissue from trauma. As bruising of the corium and underlying tissue is one of the fundamental causes of lameness, proper care of the hoof capsule is important for maintaining soundness.

Goals of Trimming

Simply put, all hoof preparation consists of shortening, thinning, and flattening of this hoof capsule to some standard shape. Farriers, by training, excel in the manipulation and standardization of the hoof capsule. For short-footed horses,

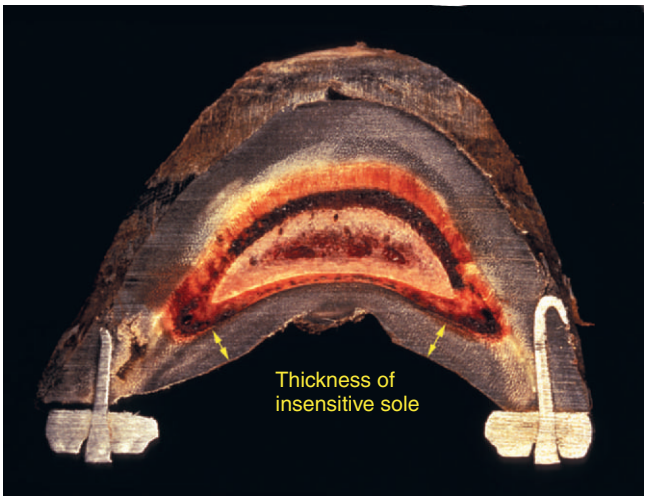


FIGURE 22-1 Frontal section of the hoof at the apex of the frog. The third phalanx (shown in cross-section in the center) is completely surrounded by the hoof capsule and associated soft tissues. Appropriate hoof preparation must leave adequate amounts of insensitive sole, bars, and frog to protect their associated coria and underlying tissues from bruising due to ground trauma. What the horse is asked to do and where it is asked to do it will determine how much solar horn is sufficient.

successful hoof trimming involves leaving the solar surface thick enough to protect the underlying corium (Figure 22-1), while still providing adequate ground contact and heel support in relationship to the axis of the limb. Trimming must also shorten the hoof adequately so that the ensuing normal hoof growth in the interval between trimmings does not create unfavorably long lever arms or biomechanically unstable supporting platforms that would promote clumsiness or lameness.

Performance or working horses can certainly be conditioned barefoot if attention is given to their footing and their hooves are periodically checked for excessive wear. Historically, when these horses started to demonstrate hoof soreness on hard or abrasive ground, they were shod with steel shoes attached with nails. However, conventional shoeing was necessary only if their handlers wanted to continue conditioning or training the horses. If these footsore horses were instead turned out in well-maintained runs or pastures, they generally displayed no lameness or only mild lameness that would quickly self-correct.

The author recently conducted a survey investigating the shoeing practices and preferences of endurance riders in the Pacific Northwest. Some interesting correspondence ensued, not all of which pertained to endurance horses. A barrel racer sent images of her horses who successfully competed barefoot in well-prepared arenas. She described using a rasp and a dremel drill weekly to shape the solar surface of her horses' hooves. When she chose to ride on rocky trails rather than in an arena, she simply applied Easyboots (Easycare, Tucson, Ariz.) for that occasion to prevent her horse from becoming foot sore.

One of the frustrating and unavoidable aspects of conventional farriery (in which most horses are shod every 6 to 8 weeks) is the inevitable increase in hoof length of 1/2 to 3/4 inch in the interval between shoeings. Presumably, this regular cycling of hoof length and thus hoof mass imposes an additional biomechanical burden on horses being asked to perform as athletes. By trimming her horses' hooves weekly, this owner may have been standardizing hoof length and mass and thus avoiding potential problems of increased lever arms and hoof mass that can interfere with performance.

Alternatives to Conventional Shoeing

Viable alternatives to steel shoes attached by nails for competitive or working horses have become more widely available. For example, horses competing in long-distance endurance events have successfully raced in Easyboots (O.K. Balch, unpublished data). Clever modifications to prevent dirt and debris from collecting between the boot and hoof can extend the length of time that Easyboots can be worn. Glue-on horseshoes (exemplified by the Sigafoos series; Sound Horse Technologies, Unionville, Pa.) have finally matured into farrier-friendly (i.e., easily and quickly applied), horse-friendly (i.e., secure and causing minimal or no damage to the hoof wall) hoof protection that does not use nails and is less restrictive to the normal expansion of the hoof capsule.

Shoeing for Specific Purposes

Whether nailed, buckled, or glued on, shoes and boots act as extensions of the hoof capsule to prevent excessive wear of the solar surface of the epidermal structures and excessive trauma to the underlying solar corium. Additionally, shoes can be used to either increase traction (e.g., jumping horses with caulked shoes) or decrease traction (e.g., reining horses with sliding plates), as need be. In unusual instances of hoof wall loss or abnormal limb conformation, shoes can also be used to extend the weight-bearing surface to normalize ground forces on the hoof or the limb.

IMPORTANCE OF A TEAM APPROACH

Appropriate care of the horse's hoof is one of the most controversial subjects in equine practice.[8] No doubt part of the vigorous debate stems from the unique circumstance that the hoof is the province of two very different and very differently trained groups of professionals: farriers and veterinarians. Theoretically, the dividing line separating professional responsibilities has been based on whether the tissue is insensitive (epidermis or horn) or sensitive (dermis). Historically, farriers have manipulated and modified the hoof capsule, whereas veterinarians have treated the underlying corium and associated living tissues. However, as understanding of the interrelationship and interdependence of the insensitive and sensitive tissues that collectively comprise the equine digit has expanded, the line defining the division of labor between the two professions has blurred and, in some instances, disappeared entirely.

Equine veterinarians are hindered by the absence of structured, in-depth training in manipulation of the hoof capsule within the formal veterinary curriculum. Even the most enthusiastic equine veterinarians cannot devote years to developing and honing the manual skills that characterize good farriery. Experienced farriers who specialize in the diseased equine digit often develop an intuitive and practical understanding of digital pathophysiology that facilitates innovative and successful mechanical treatment of many lameness problems. However, some diseases of the digit, laminitis being a prime

example, are best treated using a team approach with talented, enthusiastic members of both professions.

THE BAREFOOT DEBATE

Within the last decade, the traditional approach to hoof care (and disease) using conventionally trained farriers (and veterinarians) has been challenged by various barefoot proponents. The most prominent advocate of the barefoot approach is Hiltrud Strasser, a German veterinarian. Strasser has promoted a comprehensive horse care program that runs counter to many of the conventions of traditional domestic horse husbandry.[9]

To her credit, Strasser advocates a return to environments that are more in line with the horse's unique evolutionary development as herd animals that are constantly grazing and ambulating. She strongly advocates that horses' feet be simply trimmed and not shod. However, her iron-clad prohibition of shoeing has sorely perplexed traditional farrier organizations in Europe and the United States; official cautionary statements have even been issued.[6]

Owner Enthusiasm

Perhaps the most surprising aspect of this controversy is how quickly and enthusiastically the barefoot banner has been picked up by many members of the horse community. How deeply Strasser's ideas resonate is reflected by the number of websites that advocate equine barefootedness; poems have even been written celebrating equine barefootedness.[6] Likely part of the attractiveness of the barefoot movement is the self-empowerment that owners feel in providing their own hoof care rather than employing traditional farriers.

Strasser's most controversial premise—no shoes—has prompted other charismatic individuals (some of whom are disenchanted farriers) to develop similar but competing schools that offer training for owners who want to provide their own hoof care. Thus the Strasser approach is now one of several barefoot hoof care methods being promoted among horse owners. As it is perhaps the most widely disseminated, and arguably the most rigid in its requirements, the Strasser approach is the focus of this discussion.

Strasser Tenets

Strasser has published two small books[9,10] and a major treatise[11] explaining and promoting her ideas. While her advocates argue that Strasser's methods are successfully rehabilitating horses with navicular syndrome and laminitis,[12] there is a distinct absence of refereed publications documenting these successes. (The only article on her approach that has been published in an English-language, refereed veterinary journal was written by two experienced farriers who attended a presentation given by Strasser at a conference held at Tufts University titled *Hoofcare for the New Millennium: Exploring the Strasser Method*.[13])

Strasser rejects shoes for the following reasons, which, she argues, have negative consequences: (1) they protect the hoof wall from wear; (2) they cause vibration (800 Hz); (3) they impair hoof mechanisms; (4) they cause physical trauma to the corium and other tissues; (5) they add unnatural weight; (6) they alter natural breakover and movement; (7) they reduce nerve function; (8) they negatively alter traction; (9) they deform the hoof; (10) they cause nail problems; (11) they increase the risk and severity of injury; (12) they increase damage to properties and trails; (13) they prevent normal development of the horse's foot; and (14) they disrupt energy balance. Each of these points is explained in her books.

Examining the Historical Premise

Strasser's explanation of the widespread adoption of shoeing in Medieval Europe (in the face of her contention that shoes were really not necessary) is novel:

> *The results of historical research over the last few decades have confirmed that nailed-on horseshoes first came into use in the early Middle Ages. This means that the huge migrations of tribes and the endless cavalry warfare of early history took place by riders on unshod horses. By the time when castles were being constructed on hilltops in central Europe in the 6th Century, horses were required to live in small, enclosed spaces, standing in their own excrement. The hooves, lacking proper circulation to form good horn, and additionally weakened by ammonia, were no longer able to bear up to use on rocky terrain (in contrast to the hooves of those horses still kept in large open spaces). It was the horses of the vassals and common folk, still living more naturally, [that] did not have (or need) shoes. But, as is human nature, those things used by the 'rich and famous' quickly become desirable to the 'common man.'*[9]

Strasser's visions of endless cavalry warfare, successfully waged on barefoot horses, conflict with the historical observations that the military expeditions of Alexander the Great and Methridates were curtailed by sore-footed horses, however. In fact, it is recorded that the cavalry halted during campaigns to allow the horses "to restore the horn of their hooves."[14]

The authoritative book *The Medieval Horse and its Equipment*,[15] which is authorized by the Museum of London, states that horseshoes made their first English-literature appearance in the late 9th or the 10th century, along with concurrent references by Byzantine and Frankish authors. While unknown in Rome, the horseshoe might even have been used in Celtic Europe during the period of the Roman Empire.[15]

This explanation is consistent with Wolfgang Jochle's essay, which states that of the four waves of horseback invaders who sacked Medieval Europe from 5th to the 14th century, only the first, the Huns, used truly barefoot horses.[16] Jochle further states that the Magyars (10th century) and the Mongols (12th and 13th centuries) introduced the practice of shoeing horses to Christian Europe. Given Strasser's fundamental argument that horseshoes are without advantage, she has cast farriers with unprecedented success as marketeers and charlatans, to have thus fooled countless equestrians for centuries to invest nearly monthly in a product that is without value.

Beyond Healthy Discourse

This issue of barefootedness is quite polarizing among members of the lay horse community (O.K. Balch, unpublished

FIGURE 22-2 Solar view of the right forehoof of a barefoot horse just before the start of a 25-mile endurance race near Eagle, Idaho. This 6-year-old mare reportedly had never been shod.

FIGURE 22-3 Solar view of the foot shown in Figure 22-2 on the same day, but at the end of the 25-mile endurance race. The mare successfully completed the race and finished third out of 27 competitors. Careful examination of the hoof wall at the dorsomedial aspect reveals a small amount of outer wall broken away (*arrow*) that was not present at the pre-race check-in (see Figure 22-2).

data). Similar dissention can also be found in the veterinary community, as witnessed by the spirited discussion on the subject in the *Journal of Equine Veterinary Science* in 2003 and 2004. The most extreme sentiment expressed was that "Horseshoes kill horses. The shoe acts like a slow poison. . . All shoes are harmful to the health of the horses. There is no right way to do a wrong thing."[17]

The intensity of some of this discussion goes beyond healthy discourse on the best way to protect horse's hooves. The vehemence reflects a pervasive, underlying uneasiness with the conventional role of farriers as hoof care experts by some members of the equine community.

Middle Ground

Some notable human runners, for example South African Zola Budd and Ethiopian Abebe Bikila, have set records by training and competing barefoot. Likewise, properly conditioned and selected horses can successfully perform barefoot in certain activities (Figures 22-2 to 22-4). Certainly, more humans could go barefoot if they were willing to spend years conditioning their feet and choosing carefully where to ambulate. People wear shoes and horses are shod for the same basic reasons: shoes increase mobility and functionality.

Although there is good evidence that specific shoe types or adaptations (e.g., toe grabs) are significant risk factors for musculoskeletal injury in horses,[4] there is no scientific evidence that the routine use of appropriately fitted, nailed shoes correctly applied is detrimental. (Poorly fitted and designed shoes cause pain and lameness in people, and the same is true in horses; however, that fact is not in dispute.)

The design and material make-up of human sport shoes are constantly evolving, with the result that, arguably, athletic performance in certain disciplines has been enhanced. Critical evaluation of horseshoes (e.g., design, material construction, and effects on limb and hoof biomechanics) should be pursued, not to rationalize their prohibition or their use but to improve their functionality and efficacy. Development of new materials for shoes (e.g., plastics) and use of alternative attachment techniques (e.g., adhesives), as well as more sophisticated understanding of the biomechanics of the hoof and normal anticoncussion

FIGURE 22-4 Lateral view of the lateral surface of the same hoof shown in Figure 22-3. Wear and chipping (*arrow*) are evident on the distal portion of the lateral wall. This limited-distance race was conducted on footing that was ideal for barefooted horses (dirt trails almost completely free of rocks). Although this horse competed successfully at the 25-mile distance, a longer race or more abrasive trail might have resulted in a sore-footed horse. Note the apparent dorsally displaced coronet on the lateral side of the hoof. No lameness was observed and the horse had not been shod in several years.

mechanisms bode well for continued improvements in hoof care for equine athletes.

Ultimately, the choice to shoe or not to shoe depends on the hoof (and occasionally the limb) characteristics of the individual horse and the goals of the owner. Without a definitive reason for shoeing (e.g., necessary hoof protection, rider-desired alteration of traction, or biomechanical support to the hoof or limb), horses benefit from going barefoot. In fact, brood stock and immature horses should go barefoot

whenever possible. Robust hoof capsules, combined with judicious conditioning of the solar surfaces over months (and in some instances years) and owner selection of appropriate equine athletic activity and venue, can yield competitive barefoot horses.

And yet the example of Abebe Bikila offers a great lesson for hoof care providers of all backgrounds. This two-time consecutive Olympic Marathon champion first won barefoot and then, 4 years later, set a new world record while running in shoes. It is likely that truly elite performance in many equestrian disciplines will likewise require some form of hoof protection, whether nailed, glued, or buckled to the foot.

TRIMMING AND SHOEING THE SHOW HUNTER

Clark B. Beckstead

Show hunters are jumping horses that perform in a show ring. Not only are they judged on successful and timely completion of a course of jumps, show hunters are also judged on the quality of their movement. The judge watches how the horse moves, approaches the jumps, arranges its strides, and executes the jumps. Elite hunters have a fluid, minimally animated movement in which the foot is kept fairly low to the ground during foot flight. (Similar to an elite human runner wearing light running shoes and moving fluidly with minimal animation.) How the horse reaches, lands, and completes its breakover is important to the animal's success as a show hunter and is something that can be affected (either positively or negatively) by shoeing.

BASIC TRIMMING AND SHOEING

The basic principles of trimming and shoeing for show hunters are essentially the same as those recommended for any other type of horse. Shoeing for specific problems that are commonly found in show hunters is discussed in the next section.

Evaluating Movement Patterns

A good practice before shoeing the show hunter is to watch the horse move. The farrier should observe breakover, the flight (or path), and landing of each foot as the horse walks toward and away from him or her and note whether the horse breaks over straight, toes in, or toes out. Taking the time to identify the horse's pattern of movement will help ensure that the farrier does not alter the biomechanics of the limb in a potentially harmful way, such as torquing the knee. In a 1200- to 1400-lb horse, there is enormous load on the limbs and feet when the horse lands after a jump, so careful attention to hoof and limb balance is critical to ensuring optimal performance and career longevity. Watching the horse walk before any treatment or application to the feet will spotlight the horse's imperfections and idiosyncrasies and help guide trimming and shoeing decisions.

Aluminum or Steel

Aluminum shoes are popular among owners and trainers of show hunters, as they are lightweight and thus can substantially improve the movement of an individual horse. However, the particular needs of the individual horse should be considered when deciding between aluminum and steel shoes. Both types of shoe have their place in the hunter/jumper arena.

Compared with aluminum shoes, steel shoes tend to hold up better under the concussive forces these feet must endure, and they dissipate the force of impact better than aluminum. Aluminum shoes transmit more of the concussive force of impact to the hoof and thus to the rest of the limb. For this reason, aluminum shoes may not be the best choice in show hunters with poor-quality hoof walls or poor hoof conformation; thin, wide-web steel shoes may be better in these cases.

For the average show hunter with no significant problems, however, aluminum is lighter and easier on the horse. Adding a slight roll to the toe helps the horse move out easily, thereby allowing the horse to move with the low, fluid action that is considered ideal in this discipline. This action also is easier on the joints.

COMMON CHALLENGES AND PROBLEMS

The farrier faces several challenges when working on show hunters and several foot problems that are common in these horses. Perhaps the most prevalent challenge is the unnatural way in which these horses are kept.

Environmental Factors

While conditions vary from region to region, in general show hunters are kept in barn stalls (i.e., confined) and are overfed and bathed often. In other words, they live in conditions that are unnatural for a horse. This lifestyle is not in the best interests of the horse's feet, to say nothing of the rest of the horse. For example, the feet of many hunters are excessively moist as a consequence of frequent bathing.

Foot Quality and Load

Most show hunters are Thoroughbreds, so the various foot problems inherent to this breed are a common and often persistent challenge for the farrier. Some show hunters are former racehorses, and many of these horses arrive at the show barn with preexisting foot problems. The most common problem in these horses is the long toe/low heel (LT-LH) foot conformation, which is often called underrun or underslung heels. Mismatching of the pairs of front feet or hind feet also is common. In fact, many show hunters have the LT-LH conformation on one forefoot and a long toe and high heel (i.e., club foot) on the other forefoot. (NOTE: A clubfoot does not necessarily preclude a horse's use as a show hunter. When properly shod and conditioned, many of these horses can compete successfully.)

Another common problem is poor-quality hoof walls and thin soles. The horse's breed can contribute to this problem,

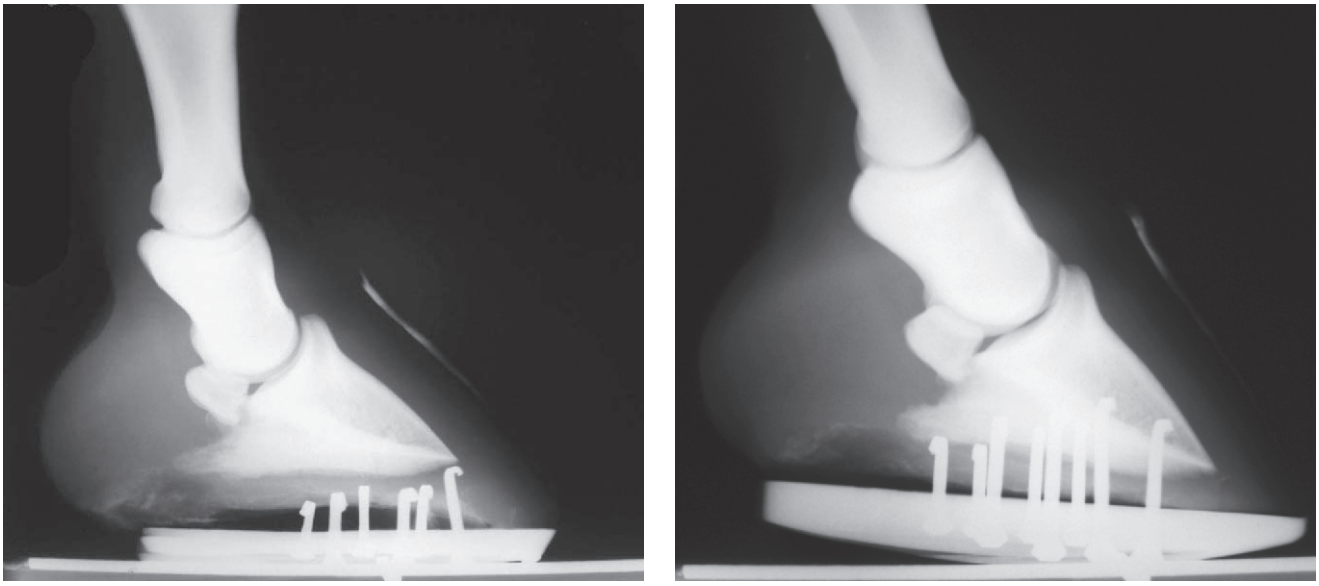


FIGURE 22-5 Difference in coffin bone angle before and after this shoe is applied.

but so can the repeated concussion a jumper's feet must endure during training and competition. Hoof conformation that is less than ideal is an important part of the equation, too. An abnormal hoof angle (whether too high or too low) is accompanied by abnormal forces on the sensitive tissues of the wall and sole, and thus decreased blood supply to these tissues, especially the solar corium (the germinal layer of the sole). This situation creates a destructive spiral of abnormal pressure, slowed horn growth, and thin walls and soles that cannot adequately protect the germinal tissues beneath. The all too common result is a tender-footed horse and chronic bruising of the solar corium. Overfeeding further adds to this problem by increasing the horse's body weight and thus the load on its feet.

MANAGING COMMON PROBLEMS

Mismatched and Clubbed Feet

Trimming and shoeing the show hunter with mismatched or clubbed feet basically involves attempting to match the hoof angles of the two feet as much as possible, assuming that the conformation of the distal limb is similar in both forelimbs. (Care should be taken not to so alter the mechanics of the limb that excessive torque is now placed on the knee or the joints of the lower limb when the horse moves.)

Depending on the individual horse's hoof conformation, this goal can be achieved by lowering the high heel, applying the shoe in a way that normalizes the position and loading of the coffin bone, and, if need be, rasping the hoof wall at the toe to eliminate any dishing. It is preferable to shoe the "better" foot first, then evaluate and shoe the clubfoot and attempt to match it as closely as possible to the other foot, while being careful not to cause lameness. To that end, the farrier should watch the horse walk away from and toward him or her to evaluate the horse's movement both before and after shoeing.

When trimming the heel on a severe clubfoot (grade 3 to 5 out of 5), the farrier should start at the heel and trim the wall at the heel and quarters to create a functional breakover point that is located directly below the tip of the coffin bone. The foot is then set down; if the horse is still fully loading the heels, some more heel can be trimmed off. The farrier should keep trimming gradually until both forefeet match in angle or until a piece of paper can be slipped under the "high" heels (indicating that the horse is no longer loading those heels).

A lateral radiograph of the foot is very helpful for making shoeing decisions. When no radiograph is available (which is the case most of the time), the farrier should set the foot down and visualize where the coffin bone should be relative to the hoof wall and sole. The farrier should imagine a straight line from the coronet (hairline) to the ground along the front of the hoof wall. (If the foot has a dished contour, the angle of the hoof wall should be followed just below the coronet, that is, above the dished wall. Any dishing at the toe will be rasped off later.) The point where the line meets the floor is where the tip of the shoe should be set. Once the shoe has been applied, the same guideline are used and any excess hoof wall at the toe is rasped to make the hoof angle align with the coffin bone and eliminate any dishing.

Using a Slightly Contoured Shoe

A method of shoeing the abnormal foot that is particularly useful is to use a shoe that has a slightly curved contour from heel to toe (i.e., the shoe slightly "cups" the foot). The hoof wall (but not the sole) is trimmed to match the subtle curve of the shoe, which involves slightly rolling the toe and the heel with a rasp. Good fit of this shoe requires that the finished shoe be carefully burned into the hoof wall.

When this shoe (which is flat in the center of the foot) is applied, the slightly curved shape allows the horse to restore a more normal coffin bone angle and to load the foot more normally and thus move better. Figure 22-5 shows the difference in coffin bone angle before and after this shoe is applied. This horse has the negative palmar angle (–7 degrees) that is typical of a horse with the LT-LH foot conformation, which this flat shoe is doing nothing to improve. The application of the

slightly contoured shoe changes the palmar angle of the coffin bone from negative to a neutral or slightly positive angle. This shoe also moves the functional breakover point back to a location just behind the tip of the coffin bone, thereby making it much easier for the horse to lift the heel and break over the toe.

This shoe also improves how the foot is loaded. Normally, a horse loads the heels first, then loads the rest of the foot, and finally breaks over in the center of the toe. The slightly contoured shape of this shoe facilitates this action, particularly in a horse with an abnormal hoof angle. The common adage, "shoe flat and level," may be inappropriate for an individual horse; even sound horses do not necessarily land flat when they walk.

This shoe further aids the horse by relieving concussion on the heels during landing. It can therefore prevent bruising in the heel area. And in horses with underrun/underslung heels, this shoeing strategy encourages heel and sole growth and makes the horse more comfortable, simply by restoring more normal hoof and coffin bone angles and mechanics. As a result, the horse moves better and so performs better.

Wedges

Heel wedges are commonly used to address the low-heel component of the LT-LH foot conformation. In the short term, wedge pads or wedge shoes can help, but in most cases they are not beneficial in the long term.

Thin Soles

Many show hunters have thin soles. The foot of the average-sized hunter should have a total sole depth of at least 22 mm. (Sole depth is measured on a lateral radiograph as the vertical distance between the underside of the coffin bone and the outer surface of the sole.) The germinal layer of the sole (the sensitive, inner layer that produces the insensitive, protective outer layer of horn) needs a certain amount of space between the coffin bone and the hard layer of horn; otherwise compression impedes its blood supply and sole growth is slowed.

In most Thoroughbreds, sole depth is 8 mm or less. These feet are particularly prone to sole bruising, which can occur simply from the weight of the horse directed down through the limb to the coffin bone. In other words, the horse does not have to step on a rock or be worked on hard, irregular ground to develop sole bruising when the sole is this thin.

A common recommendation in these horses is to shoe the horse with sole pads or sole packing material. This strategy can certainly protect the sensitive tissue of the sole from external insults; but unless the mechanics of the foot are appropriately addressed (e.g., the hoof angle is corrected), the sole will continue to be thin, tender, and prone to bruising.

Pads and Sole Packs

Neoprene or leather pads can help protect the sole, lessen concussion, and perhaps even encourage sole growth. In addition, packing is often placed between the pad and the sole. Many products are available for this purpose, including various combinations of tars, iodine, peat moss, and absorbent materials. These products are designed to medicate the foot and cushion it, absorb shock, and maintain the integrity of the sole. Pine tar and oakum are sometimes used to help keep dirt and other materials from getting between the pad and the sole, while gently medicating the foot.

A guiding principle, however, is to avoid using pads unless they are clearly necessary—for example, the horse has tender feet, refuses jumps, and responds positively to hoof testers. The other situation is when the veterinarian treating the horse requests them.

Common Farrier Errors

There are some preventable mistakes that farriers make in trimming or shoeing the show hunter. These mistakes bear mentioning here, because they can negatively affect the health of the hoof and the comfort and performance of the horse.

Excessive Rasping of the Toe

Farriers often excessively rasp the toe first and then apply the shoe. By doing so, it becomes impossible to determine where to place the front of the shoe for optimal breakover. The rationale often given for this trimming method is that it moves the breakover point back. However, rasping the toe from the outside does not create better breakover. In a healthy foot, functional breakover is located directly below the tip of the coffin bone, or just slightly in front of it. The foot should be trimmed with this point in mind.

The other problem with this approach is that excessive rasping of the toe (to the point of completely removing the stratum medium and exposing the white line) destroys the integrity of the wall in that area. Not only is the hoof capsule weakened, but some of the protection for the sensitive tissues within the hoof is removed. Such rasping or dressing of the toe is appropriate only when there is excessive thickness at the toe.

Excessive Trimming of the Sole

Another common error is trimming too much sole. Many show hunters (particularly the Thoroughbreds) already have soles that are too thin. Removing any of this protective layer further predisposes these horses to foot tenderness and sole bruising.

Shoeing too Tightly

Often the shoe is fitted to exactly match the dimensions of the hoof wall, rather than being set a little wider than the wall from the quarters back and so giving the heels room to expand when the horse lands and loads the foot. Shoeing too tightly can lead to bruising in the heel region as the hoof wall behind the quarters expands over the outer edge of the shoe during loading.

A good rule of thumb is that there should be at least a dime's width of shoe visible, extending beyond the sides of the hoof wall, from the quarter to the heel (a practice called *shoeing full*). This extra few millimeters of shoe metal allows for the necessary hoof wall expansion during motion. Naturally, care should be taken not to leave too much shoe extending beyond the wall on the inside (medial side) of the foot, as the horse is more likely to pull off the shoe in this situation.

Owner/Trainer Errors

Two common problems in the show world for which the horse owner or trainer bears the primary responsibility are a

shoeing interval that is too long and excessive use of hoof dressings.

Shoeing Interval

The recommended shoeing interval for most show hunters is 6 weeks, although 5 weeks is acceptable. Problems can arise if the shoeing interval exceeds 7 weeks. Even in horses with "perfect" foot conformation, excessive length of the hoof alters the biomechanics of the foot by increasing the lever arm (see Chapter 3). Thus owners and trainers should be encouraged to maintain the shoeing interval recommended for that particular horse.

Hoof Dressings

The physical appearance of the horse is important to owners in the show world. It is common practice for owners to apply hoof dressings and other topical treatments to their horses' hooves. However, frequent use can lead to build-up of chemicals, oil, and adhered dirt on the surface of the hoof, which can negatively affect the health and resilience of the hoof. Thus these products should be used very sparingly, if at all, and only on the advice and direction of the veterinarian or farrier. The best topical treatment for the hoof is fresh air.

A well-balanced foot and a properly applied shoe (whether aluminum or steel) aids in keeping the horse comfortable, healthy, and successful during its career as a show hunter. A happy foot is a happy horse is a happy horse owner.

TRIMMING AND SHOEING THE STANDARDBRED HORSE

Bruce Daniels

Although Standardbred horses are bred to a standard, they are divided into two classes, trotters and pacers, based on the gait at which they train and race. The gait at which an individual horse will race is determined by its breeding and conformation.

DIFFERENCES BETWEEN TROTTER AND PACERS

The trot is a diagonal, two-beat gait. The right foreleg and left hindleg move in near-unison, followed by the diagonal pair of left foreleg and right hindleg. Although the trot is referred to as a *two-beat gait,* there is a miniscule delay between the fall of the hindfoot and that of the diagonal front foot. It takes an experienced ear to detect this tiny delay, however. In both the trot and the pace, there is a moment of suspension between the strides when all four feet are off the ground. But unlike the pace, the horse's body moves very little from side to side during the trot, even at high speed. Thus the trot is an efficient method of travel. A horse can cover great distances with little fatigue at this gait.

The pace is a lateral, two-beat gait. The right foreleg and right hindleg move in near-unison, followed by the lateral pair of left foreleg and left hindleg. As with the trot, the hindfoot lands slightly before the paired forefoot. Listening to a pacer at speed, it sounds more like a "shush–shush" than a "clop–clop." Unlike with the trotter, there is considerable side-to-side motion of the body when a horse is pacing, which is why some people refer to pacers as *side wheelers.* Despite this lateral motion, pacers can travel at a faster speed than trotters.

Pacers usually wear hobbles (or hopples) which unite the legs on each side, thereby ensuring a lateral gait (i.e., pace rather than trot). Pacers that do not wear hobbles are referred to as *free legged*. These horses may be shod with a longer toe or with more weight in the shoe on the front feet.

PRINCIPLES OF TRIMMING AND SHOEING THE STANDARDBRED RACEHORSE

Trimming and shoeing the Standardbred horse is based on the same principles as those used for any other breed of horse. There are, however, three important areas that require special consideration when trimming and shoeing Standardbred racehorses: trimming for hoof balance, shoe weight, and the use of traction and breakover control.

Trimming for Hoof Balance at High Speed

The Standardbred racehorse is asked to go faster and farther at the same gait than are most horses under saddle. The way the Standardbred horse moves at high speed reflects this demand and must be considered when preparing and shoeing the hoof. It is an exercise in efficiency that the body hardly needs to move laterally to maintain balance when one or two feet are off the ground. But when moving at high speed, the stride both lengthens and widens. Also, with many really fast harness horses, the body lowers several inches when the horse "brushes" (i.e., sprints) for a short distance.

When trimming and shoeing the Standardbred racehorse, the goal is to optimize hoof placement at high speed, not necessarily at the walk. The objective is to trim the hoof so that it is placed properly when the horse is working at high speed. The hoof is placed properly when there is minimal deviation between where the hoof lands and where it is at the top of its arc of flight.

Shoe Weight

Also important in the Standardbred racehorse is to keep the shoes as light as possible. With the exception of weight needed to balance the gait, a heavy shoe is a handicap.

Judicious Use of Traction and Breakover Control

Another important difference between Standardbred shoeing and that of other breeds is the judicious use of traction and breakover control. The racetrack is both level and uniform in its entire surface, so traction devices and breakover control on Standardbred shoes are much more effective than they would be on horses in other disciplines and on other work surfaces. The harder the racetrack surface, the more effective traction devices become, sometimes even to the point of being damaging to the horse. Thus these devices must be used with discrimination and with consideration for the needs of the individual horse.

TRIMMING THE STANDARDBRED FOAL

Trimming the Standardbred foal should also follow the basic principles of good farriery. To the best of his or her ability, the farrier should keep a natural angle to the hoof and pastern: when viewed from the side, all three bones from the fetlock (ankle) to the ground form a straight line. Mediolateral balance is assessed while standing directly in front of the foal. From that aspect, the leg should stand in the center of the hoof.

Changes to the mediolateral balance should be made only when angular deviations are present; and even then, it is important to work up the leg with the time of growth plate closure in mind. Once the growth plates are closed, no attempt should be made to straighten the legs. Occasionally, deviations in structural alignment are hereditary, so it pays to know the conformation of the sire and dam when working on foals. Some bloodlines have an inherent broken-back hoof angle, and it may be futile and even harmful to try to change it in these horses.

ROUTINE SHOEING AND THE CORRECT USE OF SHOE WEIGHT

Trotters can start off being shod in front with a steel shoe made of 5/8-inch half-round steel. (A steel bar 5/8 inch in diameter and about 12 inches long, sliced lengthwise through its center and bent into the shape of a horseshoe, with three nail holes punched on each side, produces a half-round horseshoe.) The half-round shoe usually is flattened slightly for the first few shoeings to prevent fetlock damage in youngsters. The hindshoes are lighter and preferably made of flat steel, something like 9/16-inch by 3/16-inch in profile.

As the colt or filly gets stronger and more comfortable, flattening of the half-round shoe can be eliminated, and the flat hindshoe is changed to a full swage. The first pair of full swages usually are flattened slightly so that the change is not too radical. Thereafter, it is up to the trainer and farrier to choose what type of shoe is best for a particular horse. Horses that are breaking over too quickly may get a 1/4-inch by 5/8-inch shoe in front or any of the endless other combinations of front and hind shoes.

Pacers usually are started off with a very light 1/2-inch, half-round, slightly flattened shoe, like the trotter. The hindshoe may also be a light, flat shoe, or, on a youngster that does not show a lot of natural pace, it may be a heavier shoe, perhaps a 5/8-inch half-round. The 5/8-inch shoe can be reset until the weight lessens and the horse shows more pace.

Hoof Angle and Shoe Weight

Trainers of animated breeds such as Saddlebreds, Walking Horses, and Hackneys have long known that, on a conditioned horse, a heavier shoe will make the knee go higher and a longer hoof (or toe) will make the pastern flex farther, or extend its arc of motion. More simply put, weight gives knee and toe gives pastern. The result is a high stride that, although it is done rapidly, is not very energy efficient. The stride does not cover much ground and is definitely not the action wanted on a Standardbred racehorse. That is not to say that weight, judiciously used, cannot help a Standardbred racehorse.

The most efficient method of moving a hoof forward is a smooth motion, with minimal side-to-side motion. Too low a hoof angle is hard on the tendons, whereas too high a hoof angle is hard on the fetlocks. This observation has been made since horses were domesticated and has always been attributed to the hoof angle. In the author's opinion, it is the result of the point of breakover being too far forward on the hoof with the low angle. As a result, breakover is retarded, and the foot snaps almost vertically off the ground.

In the hoof with a high angle, the breakover is further back, and the foot is lifted off the ground in a less vertical flight path. So, although hoof angle is an important tool in achieving the desired arc of motion, it is really the point of breakover that has the most influence. Once the angle problem has been resolved, the height of the arc needs to be addressed. Here is where judicious use of weight comes in. It is important to remember, however, that with racehorses the weight involves ounces, not pounds.

SHOEING TO COMPENSATE FOR SPECIFIC LAMENESS PROBLEMS

When a horse's natural support is altered on one end (e.g., by adding weight to the front feet to enhance a trot or checking the head abnormally high), the horse is forced to compensate with the remaining support on the other end. Horses with gaits that have been animated (e.g., Walking Horses) lift their front feet much higher than normal and compensate by taking a longer stride, or walk, with their hind feet. This exaggerated gait causes stress on the hock joints and the supporting soft tissues. A similar problem can occur in Standardbred racehorses. Whatever the reason, compensation for loss of support in the front end causes the hindfeet to come further under the body. This additional effort eventually fatigues the hock joints and may result in hock damage.

Considering the Horse's Conformation

The horse's conformation must be considered when determining proper balance for the hindfeet. A wide-hipped horse that stands with a narrow base behind is most comfortable standing that way. Care must be taken to keep the leg and hoof in alignment; it may not be vertical when viewed from directly in front of or behind the horse, but it is in proper alignment when viewed from the direction the leg is pointing. There is a natural outward rotation or wringing of the hocks as the hoof on the base-narrow horse is unloaded, because the hock is not directly over the hoof. Cow-hocked, or base-wide horses, on the other hand, rotate their hocks inward.

The usual procedure to address this situation on the base-narrow horse is to slightly lower the inside hoof wall and build some lateral support into the shoe. The opposite is done for the base-wide horse. While this approach may alter the normal mediolateral balance, it may be necessary to remedy interference problems.

Caution must be taken in the use of traction or trailers that would completely stop the hock from rotating. To experience this painful effect, one need only place both of one's own feet

firmly on the ground and rotate the body hard to one side. The same effect can be felt on knee that a base-narrow or base-wide horse feels on its hock when the hoof is prevented from rotating on the ground.

Hock Problems

Hock problems show themselves to the farrier in two basic ways. The first indicator is the pattern of shoe wear. When a horse that has been wearing the hindshoes level from heel to toe begins to wear the toe excessively, it is an indication that the horse has been moving "strung out" or leaving the hocks behind in an effort to minimize extension. This action reduces the full stride of the hindlegs and prohibits the horse from moving to its potential speed.

This movement and wear pattern can be unilateral or bilateral. If it affects just one hock, then it most likely is compensation for a clinical condition or injury in the limb. When one hock is uncomfortable, the horse may take a shorter stride with that leg and place the opposite hoof further under the center of the body. As a result, the horse appears to move "dog fashion," and the front feet no longer come between the hindlegs. Instead, one will be outside the hindleg and the horse will be "on one line," which usually causes it to bear in or out from the rail.

A second indication of a hock problem is dragging of the toe on the distressed leg. Excessive toe wear is the result of the horse trying to minimize hock flexion and carrying the hoof in a low arc of flight. In most cases, the toe of the hindhoof is clean and shiny or even worn down. The worst-case scenario is to find the toe totally worn away, as though it was rasped straight down. There are several ways of handling these situations. First and foremost, however, it is important to get professional help from a knowledgeable veterinarian.

Basic Approach

In most cases, the basic shoeing approach is to increase the angle of both hindfeet and apply a squared-toe, flat shoe. The raised angle decreases the effort necessary to move the limb forward; the squared-toe shoe may prevent the toe from contacting the ground; and the flat shoe allows the foot to move freely on the ground, thus reducing torque on the hock. Raising the angle may reduce the length of stride and may slow the horse down, but a horse that is more comfortable will go faster than a horse that is in pain. In an extreme situation, in which the front of the hoof continually wears off from the toe dragging, an extreme rocker toe shoe, drawn thin and fit to the worn hoof, will save the remaining dorsal hoof wall.

Jacks

The wear pattern of horses with jacks (inflammation of the cunean tendon at the front of the hock) is different from that of a horse with curb (inflammation of the long plantar ligament or other soft-tissue injury at the back of the hock). While horses with curb show excessive wear at the toe, horses with jacks wear the inside branch of the shoe. Wearing of the inside branch is the result of the foot being deliberately placed wide to reduce the concussion on the affected area of the hock. This situation is definitely one in which, unless the foot is out of mediolateral balance, the farrier should do nothing. This injury is the veterinarian's domain and should be treated by him or her.

Stifle Problems

Asking an unconditioned horse to do too much can result in a stifle problem. It hurts to flex the stifle, so the horse swings the leg wider than normal and then brings it further under the body in an effort to minimize flexion of the joint. As a result, the shoe will be excessively worn on the toe and lateral branch. In many cases, the hindfoot of a pacer coming further under the center of the body will cause interference with the diagonal front foot (cross-firing). Many trainers want the farrier to lower the inside wall on the hindfoot to address the cross-firing; however, that only makes it harder for the horse. It is better to improve the horse's physical condition and work on getting the front feet off the ground sooner. In most cases, raising the angle of the hind feet by 2 or 3 degrees helps while the stifle recovers. Once the injury has resolved, the angle should then be returned to normal.

Splints

Most horses develop splints from overwork or having unbalanced feet. However, Standardbred racehorses may also develop splints from striking the inside of one foreleg with the opposite hoof. The resulting trauma can range from bruising beneath the periosteum (the bone's outer covering) or within the interosseus ligament (which anchors the splint bone to the cannon bone) to fracture of the splint bone. Broken splint bones can be very painful and most of these horses resist the concussion of having nails driven into their hooves.

If the hoof is not balanced properly, the hoof can be simply dressed to a position where both splints are equally loaded. If the horse is wearing a heavy shoe, then a lighter shoe is used to minimize concussion. If the injury is the result of interference, then a shin boot can help prevent further injury. Occasionally an injury in another leg causes a perfectly gaited horse to place a foot so that it is out of balance and thus pop a splint. It is rare, but it does happen.

SHOEING TO CORRECT INTERFERENCE

Interference is a common problem in trotters and pacers. The most common types of interference, and the general approach to their correction, are discussed here.

Knee Hitting

Knee hitting is probably the most common interference problem in Standardbreds. It occurs when the hoof of one front leg strikes the inside of the knee on the opposite leg as it passes in flight. There are many causes, but usually knee hitting is the result of poor conformation, unbalanced feet, improper conditioning, or an injury somewhere.

Horses that stand toed out are prone to hitting their knees, but horses that stand perfectly straight may also have interference if they have pastern deviations. Pastern deviation is

evident when the fetlock is flexed and the digit rotates inward. Although the leg moves forward in a straight line, the deviation will cause the foot to pass close to the opposite knee and possibly hit that knee.

Corrections

There are as many ways of "getting a horse off his knees" as there are trainers. The most common technique is to lighten the shoe and lower the outside of the offending foot. The lighter shoe will lower the arc of flight, and lowering the outside of the foot will place the foot further under the body and cause it to go wider at the peak of flight. (NOTE: The outside of the foot on the leg that is being hit should not be lowered; that would move the offending hoof out and place the victim back in harm's way.) If the width of the foot can be reduced safely, it may afford adequate clearance. Bar shoes may elevate the foot flight, so they may not be appropriate.

Horses that "paddle," or arc outward, can also hit their knees, if they paddle dramatically. The outward arc may come back in past the center line of the body and allow the foot to strike the opposite knee. The paddling must be addressed by either squaring the toe radically to the outside or even removing the toe of the shoe from the center to the outside first nail hole. Horses that toe out radically can be helped with a traction device on the outside corner of the shoe, either a crease or a drop of borium.

Cross-Firing

Cross-firing occurs in pacers when the inside toe of a hind-shoe on one side strikes the inside of the diagonal front hoof. The strike can injure the heel bulb, drive the heel of the shoe under, or even pull the shoe off. Tight hobbles, unbalanced feet, poor conditioning, and poor driving are the most common culprits. What many drivers do not realize is that when they turn a horse's head in a radical way, the feet no longer go where they were headed.

Corrections

Cross-firing is usually addressed by lowering the inside of the hind hoof and using a shoe that is set under in the area that is contacting the front foot. Shoes squared to quicken the breakover of the front foot also are used with good results. It should be kept in mind, however, that premature breakover can fatigue the shoulders and pasterns, particularly in youngsters.

Trailers will limit hock wringing and help direct the foot forward. Egg bars will prevent a hindfoot with a naturally low angle from sliding forward after descent and are used with some success. Side-weighted shoes, combined with a slightly lowered inside branch, can be used on horses that travel narrow behind.

What must be kept in mind is that if the horse never cross-fired before and is starting now, then something has changed. If the shoeing has not been changed, then something else is the problem, and a veterinarian should be called to examine the horse for lameness.

Scalping, Speedy Cutting, and Shin Hitting

These problems are basically different degrees of the same kind of interference: a front hoof and a hindhoof on the same side interfering. Owing to the differences in gait, these problems occur in trotters, not pacers. When a front foot fails to breakover fast enough or a hindfoot comes under too far, the front of the hindfoot strikes or even slides under the toe of the now-almost-vertical front shoe. It may just leave a small mark on the front of the hindhoof or it may wear a large pyramid right up the front, a condition called *scalping*. In an extreme situation, the injury extends up to the coronet.

When the horse widens its stride behind, the interference can move to the inside of the pastern and is called *speedy cutting*. Shin hitting and even hock hitting are more extreme variations of this problem. They are more common in Thoroughbred racehorses. For these injuries to occur, the horse is taking an extremely long stride behind, and if there is lameness that causes the front foot to travel in other than a straight line, it can interfere with the hock.

Corrections

Several possibilities for correction should be considered. The first is to determine on which end of the horse to work. If too many changes are made at the same time, then it is impossible to tell what is working and what is not. A horse with a low angle behind may get more leverage and cover more ground, but it may also be sliding under too far. This problem can be addressed with a higher angle, an egg-bar shoe, or heel caulks.

The reverse is true in the front end. Too high an angle in front will cause the horse to travel under itself and not give the hindfeet a place to go. Bad conformation (e.g., upright shoulders) may be the cause. If it is possible to lighten the front shoes and make breakover easier, then that should be done. (NOTE: Toe grabs, borium, and sharp swages on the front shoes only aggravate the situation.)

A third technique is to lower the inside of the offending front foot. That will cause the foot to be placed wider and be more under the horse in flight. Fitting the shoe tight on the outside (the branch that is striking the pastern) will also reduce the extent of injury.

SHOEING TO MANAGE QUARTER CRACKS AND OTHER HOOF WALL FRACTURES

Quarter cracks and other fractures of the hoof wall are very common in Standardbred racehorses. Quarter cracks originate at the coronary area, between the widest part of the hoof and the heel. A number of different factors can contribute to their development or persistence, including the following:

1. Extreme disparity in flexibility between the coronary area and the distal hoof wall, such as can occur in wet or very humid environments or with overuse of hoof softeners.
2. Excessive heel height, which limits hoof wall expansion distally and so allows the hoof wall to expand more at the top than at the bottom.
3. Improper mediolateral balance, which causes improper distribution of weight across the hoof capsule. On unbalanced hooves, the longest heel is the most restricted in expansion at the ground surface and therefore most prone to a quarter crack. This situation is a very common

cause of quarter cracks in Standardbred racehorses, when one side of the hoof has been lowered excessively to remedy an interference problem.

4. Clips located behind the widest part of the hoof. These clips restrict expansion of the hoof wall and may cause a quarter crack in working horses. (Heel clips should be used only for lay-up in horses with P3 wing fractures.)
5. A functionally inactive frog that fails to equalize the ratio of expansion of the distal wall with the coronary area. (Treatment involves lowering the heels to remove some of the restriction and regain frog pressure and/or shoeing with a full pad and impression material or hardened silicone packed into the frog area to rejuvenate and restore its weight-bearing function.)
6. Gluing on a shoe in such a way that heel expansion is restricted. The contemporary method of attaching shoes with glue has been a tremendous breakthrough; however, the technique of gluing the shoe completely to the heel restricts any heel expansion and is detrimental to the intended function of the healthy foot.

It has always been assumed that relieving shoe contact under a quarter crack works because it eliminates ground concussion. Beyond that, it reduces constriction at the heel, simply by reducing its mass. Also, the distal hoof wall is no longer restricted by its contact with the shoe and can flex more freely with the proximal wall.

Heels that have an oblique angle (i.e., underrun heels) tend to press down and under against the shoe instead of expanding outward; as a result, the horn tubules bend under the foot rather than spreading outward when loaded. In this situation, the expansion of the wall at the coronary band is far in excess of the expansion at the solar surface. These hooves benefit from support placed under the frog as well as from relieving shoe contact at the affected heel.

A brief comment about patches for treating quarter cracks: When the patch grows down more than halfway to the heel, it restricts expansion of the lower hoof wall and creates the same condition that led to the crack in the first place. Inflexible quarter crack patches restrict the normal hoof expansion. When this hoof is subjected to continuous concussion, it will feel hot to the touch and the horse may become lame.

Broken Bars

Broken bars are fractures of the caudal aspect of the bar, where it unites with the wall. Because flexibility of the hoof originates from this area, broken bars are a source of great discomfort for the horse. Causes include unbalanced feet, excessive softening of the wall, and lack of frog contact with the ground. Treatment involves balancing the feet, floating the affected heel, opening the fracture all the way to the bottom, treating the defect like any open wound, and applying a bar shoe that offers support but not pressure on the frog.

Sheared Heel

Sheared heel is not to be confused with a broken bar. Rather, it is a separation between the buttress of the hoof wall and the frog. Sheared heel may be caused by an extreme mediolateral imbalance. Wedged-heel shoes without frog support or a functionally inactive frog combined with a very soft hoof can also be detrimental. In this situation, the frog is driven down through the foot during loading, potentially causing sheared heel.

Sheared heel is the most painful of the hoof wall fractures and requires immediate attention and lay-up time. The area involved must be opened up and medicated. The heel that has been driven up should be floated by at least 1/4 inch so that all shoe-hoof contact is removed, allowing the heel to lower to its natural level. The hoof must be stabilized with a bar shoe that has a frog plate that just barely touches the frog; this device keeps the frog from descending further.

Heel pain from any cause encourages the horse to bear more weight on the toe. As a consequence, it may result in soreness from the shoulders all the way down the back of the limb to the navicular area.

Coronary Injuries

These painful injuries occasionally occur when a hindfoot cross-fires high and slices the coronary band of the diagonal front foot. After routine wound care, a 1/4-inch groove should be made in the wall immediately below the injury. This simple action almost always relieves the pain. Furthermore, if it is not done, the new hoof wall growing down has nowhere to go and will "shelve" or grow over the old wall.

SHOEING FOR INJURIES: HELPING THE HORSE WITHOUT INJURING THE FARRIER

Shoeing lame horses is an art unto itself, requiring patience and compassion. A horse with a stifle lameness, for example, will resent having its hindleg jerked high and pulled way back. It also hurts the horse to have the leg swung wide from the nailing position to the clinching position. Occasionally, just placing the hoof on the arch of the farrier's instep is as high as the horse is willing to go. The farrier should find out where the horse is comfortable and try to stay there. A farrier–horse fight is easy to get into, and the farrier will pay for it even if he wins.

The same is true when working on horses with hock or knee problems. The farrier should feel them out and find their most comfortable place. On hindfeet, the most comfortable position for the horse usually is directly under the hip and no higher than the other hock.

Horses suffering from ringbone or sidebone appreciate the hoof being held while it is being nailed, rather than the pastern being held and the horse feeling the concussion through the entire digit. When the injury is in the foot, such as a fractured coffin bone, perhaps nailing should be avoided entirely and the support glued on rather than nailed. If there is no way to avoid nailing, it would be wise to wax the nails to reduce the resistance as they penetrate the wall.

With minimal handling, the Standardbred is a docile horse. With an affection for horses and a little judgment, the farrier will not be injured when working around them.

SHOEING THE THREE-DAY EVENT HORSE

Todd Meister

The most basic principles of shoeing are the same for all disciplines: protecting the hoof from excessive wear and providing additional traction. However, as the stresses on the horse's body and limbs change with its work, so does its shoeing needs. Shoeing the event horse is difficult for two reasons: (1) the horse must perform well at multiple disciplines, and (2) the horse must often perform over different climates and terrains with little or no acclimatization period (e.g., going from cool, wet England to hot, dry, eastern United States). The majority of the stresses and injuries occur during the speed and endurance phase. It is this phase and the fitness program leading up to it that cause most of the soundness issues with these horses. Issues related to the foot can be as simple as a lost shoe or decreasing hoof quality due to heavy work, or as complicated as a navicular flare-up or soft tissue injury higher in the limb.

An event-horse farrier has done his job if the horses under his charge need minor or no adjustments during the time leading up to the competition and the competition itself. However, this is not always possible.

FACTORS AFFECTING SHOEING SPECIFIC TO THE EVENT HORSE

1. Hoof quality: With the level of work these horses are expected to perform, hoof quality is an important factor in avoiding problems at the upper levels. This will be controlled by the individual horse's genetics, footing where the horse is housed and trained, nutrition, rider/groom care, and farrier decisions.
2. Hoof capsule shape and conformation: Hoof quality and hoof capsule shape are the primary determining factors in the decisions that a farrier/veterinarian team will have to make regarding shoeing decisions for an individual. These factors determine specifics regarding trimming. These trimming decisions are often the most important decisions; they form a foundation upon which to build a healthy foot capable of withstanding the rigors of competition.

SPECIFIC TRIMMING PARAMETERS FOR EVENT HORSES

Periodic radiographs of the feet can be very useful for the farrier. Annual lateral radiographs of the foot should be adequate unless significant pathologic processes are present. In the absence of a significant pathologic condition, a knowledgeable horseshoer can estimate quite accurately where the third phalanx (P3) is. If uncertainty exists regarding any of the trimming or shoeing parameters set forth, radiographs taken at three key times can help with decisions: (1) just before the horse is due to be shod to help guide trimming; (2) just after the horse is trimmed to clarify whether trimming goals have been achieved; and (3) just after the shoe is applied to determine appropriateness of fit. The following trimming guidelines are for event horses shod at 4- to 5-week intervals and worked hard enough to slough extra sole:

Vertical depth and sole callus: The foot will need sufficient vertical depth (the distance from the ground surface of the foot to the palmar aspect of P3) and sole callus to withstand the pounding of galloping on hard ground and damage associated with potential shoe loss. Use of the hoof knife should be kept to a minimum, using it as not much more than a scraper to check reference points on the foot. Live sole should not be trimmed into more than 1/4 inch at the toe. The farrier should strive to maximize healthy frog and sole mass by strategically trimming sole and frog only when the horse indicates it will be sloughing these structures in the near future.

Hoof proportions: This includes toe length and angle, heel length and angle, orientation of the hoof capsule in relation to the coffin bone, and orientation of the hoof capsule in relation to the digital cushion. Some general trimming guidelines to encourage good proportions are to have one third of the foot in front of the position where the live frog ties into live sole (true point of the frog) and two thirds of the foot behind this spot. This may not be attainable via trimming while still maintaining proper mass, so shoe fit may need to be manipulated. A general guideline for how far to trim the heels is to trim to the point where the bar has been entered into about 1/4 inch. This should bring the heels back to the widest point of the frog.

Using these guidelines will determine the plane of the foot as defined by the ground contact surface of the hoof wall. If the quarters do not become level (in the same plane as the adjacent walls of the toe and heels), that is fine. Mass and proportions are what is most important.

Typical toe lengths are between 3 1/4 and 3 3/4 inches and hoof angle will be determined by the individual horse's conformation. Optimally, the dorsum of the pastern should be parallel to the dorsal hoof wall; hoof angle is difficult to accurately measure because it is so affected by shoe modifications and fit (set toes and rolled toes).

Anteroposterior balance: It is proven that speed and fatigue are components of soft tissue injury. It is advisable to be more aggressive with anteroposterior balance with event horses than with horses of other disciplines, erring on the side of faster breakover and shorter toe length. General guidelines are the one third to two thirds relationship with the point of the frog mentioned earlier and to have breakover near the leading edge of the coffin bone while maintaining proper mass. The hoof angle (as defined by a point from the coronet in the center of the hoof to the point of breakover on the bottom of the shoe) should be parallel the pastern axis (Figure 22-6).

Mediolateral balance: The farrier should focus on the loading phase of the stride in most instances as opposed to the landing phase. The loading phase is that portion of the stride where the horse is bearing weight on the limb as the body passes over the relatively fixed hoof. General guidelines include centering the frog, leveling the coronary band, and maintaining proper depth of sole throughout the hoof capsule. Depending on lower limb conformation, this can be difficult (Figure 22-7).

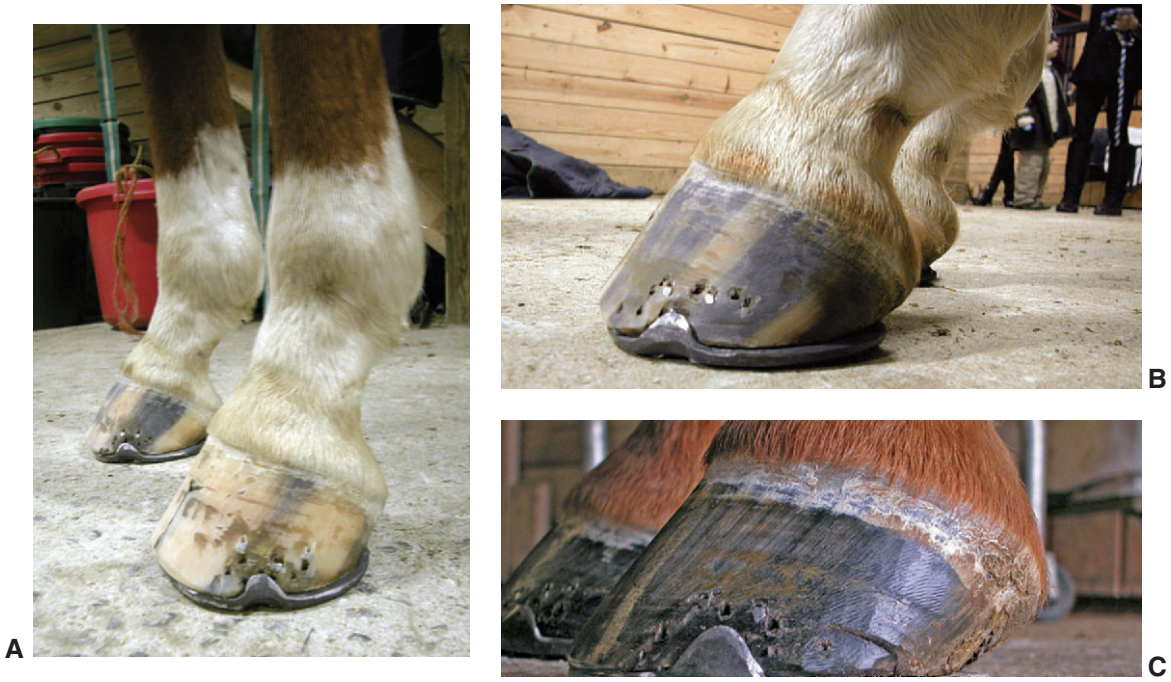


FIGURE 22-6 **A,** Anteroposterior balance. Note that even though this horse has long upright pasterns, its hoof and pastern axis are parallel. The breakover point of the shoe is well behind the dorsal hoof wall and this dorsal hoof wall, which is important for the integrity of the hoof capsule, is not removed. The toe is beveled at a 45-degree angle but not dubbed away. What is taken from the front of the hoof capsule is replaced at the rear by the caudal support of the bar shoe. **B,** This is the same horse wearing bar shoes because of an undiagnosed problem in the caudal portion of her feet. **C,** Bar shoes can be appropriate for event horses. This is a CCI two-star horse that wears the shoes year-round. The fit is supportive but not overly generous.

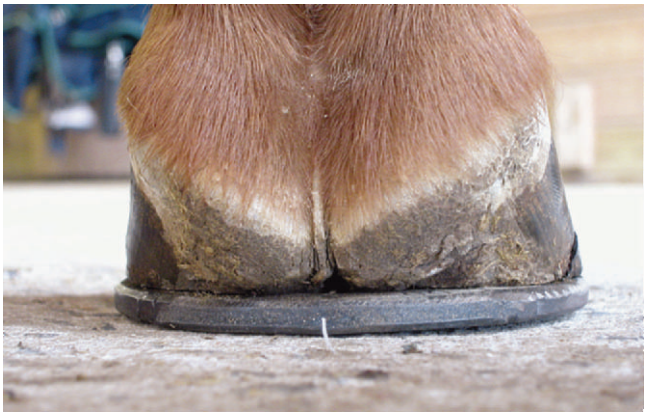


FIGURE 22-7 Caudal view of the same horse from Figure 22-6, *C*, illustrating mediolateral support without excessive fullness.

SHOE FIT

Shoe fit is an extension of trimming parameters and can be used to enhance and attain those parameters that may not be reachable by trimming alone. Included in shoe fit are considerations for the bearing surface of the shoe to the foot and how decisions are made. It is important that the hoof wall not be the only structure bearing weight, including on the shoe. Traditionally farriers are taught that the hoof wall is the only structure that is to touch the shoe. In shoeing horses with adequate hoof mass, this rule is unnecessary. (NOTE: Not only is it unnecessary, but it is also not best in all but a small percentage of cases.) A good proportion of horses have significant solar contact with the shoe. If sufficient solar mass is present, there is no need to use a knife to relieve the sole or a grinder to render the shoe concave. In some pathologic cases (P3 rotation, solar bruising, and extreme wall loss) it may be necessary to make the solar surface of the shoe concave.

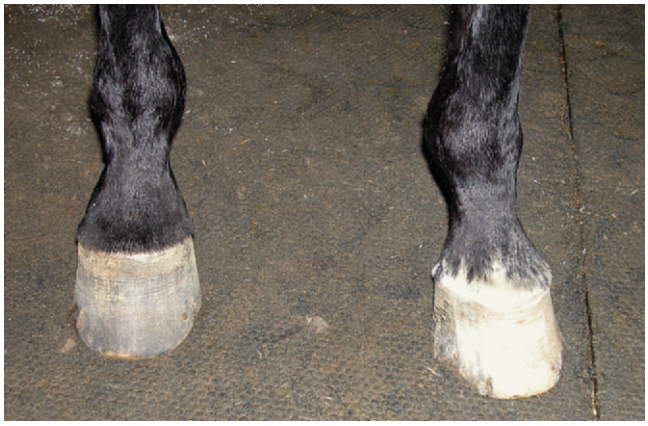
Event horses tend to be fitted with shoes with less width and length than horses in other disciplines, although the horse must be well balanced, well supported, and comfortable. Foot pain is an underrated contributor to less than optimal performance and soft tissue injury higher in the limb.

The length and intensity of the speed and endurance phase make shoe weight a small consideration (too light a shoe will flex and spring, creating greater problems than too heavy a shoe). A medium-weight steel shoe that provides support and rigidity without excessive weight is preferred ($^5/_{16} \times {}^3/_4$-inch stock for horses normally shod with keg shoes sized "00-1," and $^3/_8 \times {}^7/_8$ inch for "2-4").

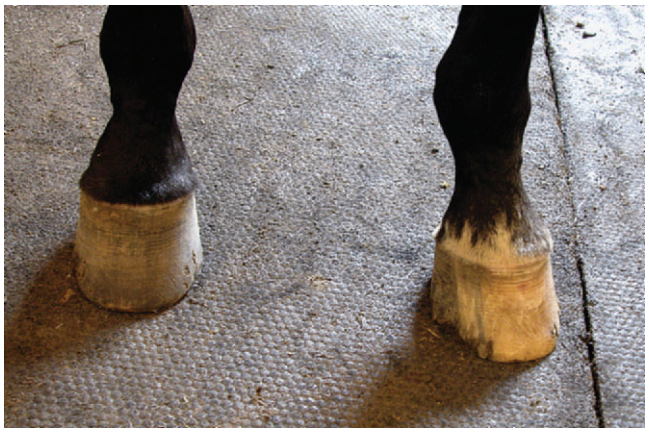
The shoeing interval should be arranged to maximize hoof capsule mass. Four to five weeks is optimal depending on individual hoof quality and conformation, work load, and timing of the competition.

BAREFOOT PERIOD

Three-day eventing is the only Federation Equestrian International (FEI) discipline in which the horses are likely to have a regular letdown period and corresponding barefoot period. If managed well, the barefoot period can be a great help in giving the horse proper foot proportion and mass. During this barefoot period, it is important to trim the horse regularly (5 weeks) and choose the time of year carefully (spring or fall in Northeast when the ground is



A



B

FIGURE 22-8 **A,** Both front feet with a different mass. **B,** The horse has two relatively similar feet (e.g., width, toe length, etc.)

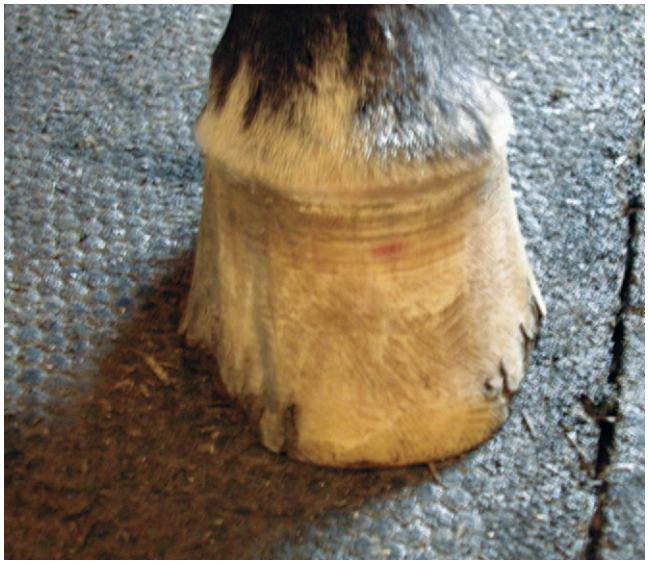


FIGURE 22-9 Close-up of left foot with poor hoof mass.

FIGURE 22-10 Close-up of right foot with good hoof mass.

A

B

FIGURE 22-11 Left foot bottom with poor hoof mass. Note lack of wall thickness and health, lack of solar depth (see ridge of burn mark in sole), and lack of integrity of the bars, especially when compared with Figure 22-12, *B*.

FIGURE 22-12 Right foot bottom with good hoof mass. Compare with Figure 22-11, *B*.

soft). Most importantly, a barefoot period is only useful if it is of sufficient length (8 to 10 weeks)—the longer the better. As the horse comes back into work, shoes should be applied before the feet show significant wear to preserve the mass created during the controlled barefoot period (Figures 22-8 to 22-12).

YEARLY SHOEING SCHEDULE FOR THE ELITE EVENT HORSE

The following section is an example of a competition season including a 3-day event from the eyes of the horseshoer. An upper level event horse will come back into training after the post-fall season letdown between December 15 and January 1. The 3-day competition the horse will be aimed for takes place approximately the third week of April. The ideal shoeing dates would be the 10th to the 15th of each month, January through April. Shoeing dates are determined by working back from the competition, so the horse is shod about 10 days before the start of the event.

Preseason

December 15

At this shoeing, the horse usually gets front shoes only. The feet will be short, with weight borne primarily by the sole and frog. During the next few shoeings, the farrier should be aware that he is transferring additional weight bearing to the hoof wall. The farrier may choose, depending on the health of the hoof capsule, to use a sole support material to take some of the load off of the hoof wall (Figure 22-13). Also, at this time, special attention must be paid the to balance of the feet. The horses have had an opportunity to correct any errors in trimming that occurred or were compounded during the last season. The horse is providing a template that must be interpreted.

January 15

At this shoeing, the horse receives hindshoes as well as stud holes. They will have little growth due to the hoof capsule adjusting to the recent barefoot period. The horse's work will be getting progressively more intensive.

Stud holes are an absolutely necessary equipment for the intermediate or advanced level event horses competitions classified as CCI two-star to four-star. (NOTE: Two-star to four-star are level classification defined by the FEI, including speed, jump height, and number of jumping efforts at each level.) Stud hole placement, stud hole number, and stud choice are critical. Stud hole placement should be about halfway between the widest part of the foot and the end of the shoe (Figure 22-14). There should be two stud holes per foot. A third stud hole is unnecessary with event horses and would constitute excessive studding in almost all conditions. Stud choice varies with conditions. Common mistakes include overstudding on hard ground, resulting in sore feet and or soft tissue strain in the lower limb, and, more common recently, understudding on soft ground, resulting in upper soft tissue injuries or potentially catastrophic falls into unyielding fences.

Drilling and tapping of the shoes by the farrier will occur using a $^{5}/_{16}$-inch or P-sized drill bit (or an appropriately sized stud punch) and a $^{3}/_{8}$-inch × 16-tpi tap in the United States (Figure 22-15). Sizes are different in other parts of the world where metric systems are used. This can be important at an international competition.

February 15

Usually by now, soundness issues specific to each horse are beginning to surface. It is the job of the farrier to notice subtle changes in the horse's comfort level. Multiple opportunities to do this exist, including the following:

1. How does the horse look while walking to the cross ties?
2. Are there any changes in the feel or appearance of the horse's limbs?

FIGURE 22-13 Use of sole support material. To precisely load portions of the foot, it is often necessary to use two people. In this instance, pet screen is being used to help hold the Equipak in throughout the entire shoeing. A rim pad can also be used.

FIGURE 22-14 Ground surface of spare shoes showing marks on the outside indicating right or left and stud holes. The spare shoes should be as precisely fit as if they were going to be nailed on the horse.

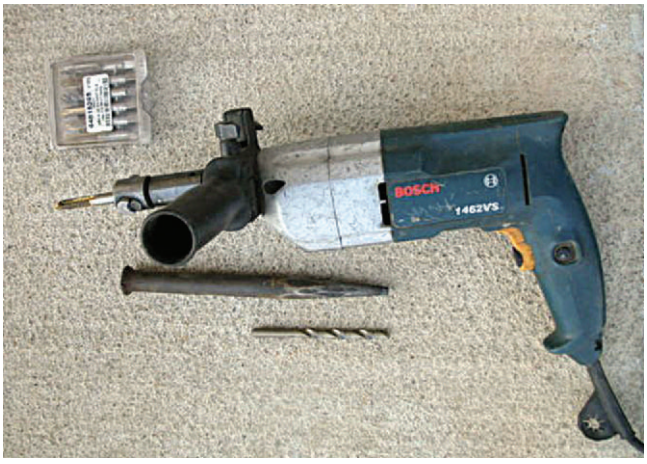


FIGURE 22-15 Implements for drilling and tapping shoes: 3/8-inch × 16-tpi taps, power tapper, stud punch, and 5/16-inch or P-sized drill bit.

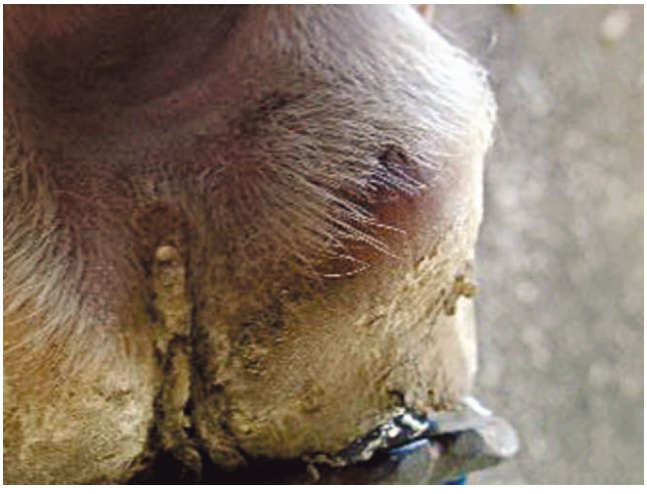


FIGURE 22-16 Fresh heel bulb bruise. This could be the result of interference and may or may not cause lameness. It is easy to see due to unpigmented hoof.

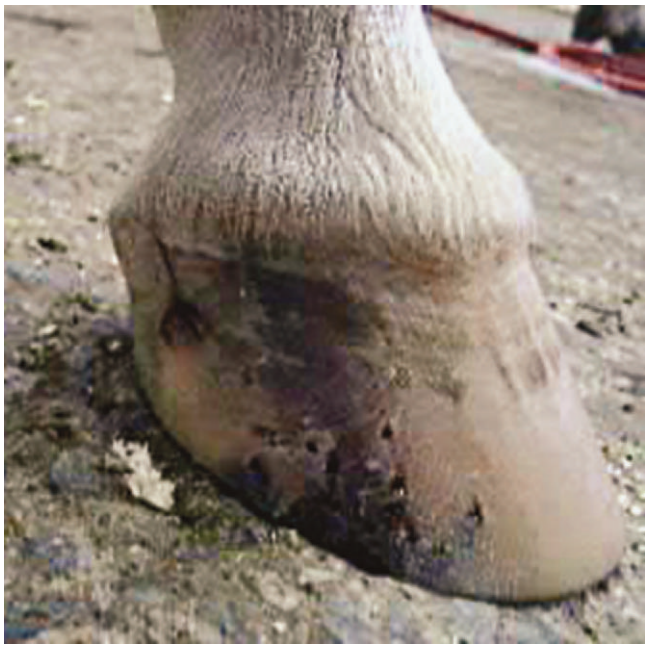


FIGURE 22-17 Distortion of the hoof capsule and correspondent bruising. Warning signs should be noticed long before the horse was presented like this, with a quarter crack and significant bruising indicating laminar pain. Significant shoeing changes will have to be implemented for this horse to be able to continue to compete.

FIGURE 22-18 Decreased hoof quality is an issue during the late summer in the northeast region. When preparing for the fall event season, this could be a common sequel to the type of fitness work required of an upper-level event horse. A team effort is required, along with significant management changes, for the quality of this horse's foot to improve (e.g., change in turn-out, change in surface worked on, decrease in bathing, and increase in topical moisture control products).

3. How is the horse's comfort as the farrier is working on it?
4. Are there any obvious warning signs on visual examination the hoof capsules, such as significant fresh bruising, distortion of the hoof capsule, or dramatic decrease in hoof quality (Figures 22-16, 22-17, 22-18)?

The farrier is entrusted to help evaluate these horses during their 3-day event, particularly before the veterinary inspection (trot-up). Each shoeing represents an opportunity to get a solid baseline assessment of what is normal for this horse when it is sound and comfortable. Farriers should make a habit doing a hoof tester examination and watching the horse trot on a hard surface in a straight line. If a problem is suspected, the horse can be watched longeing in both directions on hard and soft footing.

March 15

At the time of this shoeing, the horse's work will be dramatically increasing. They will be making the transition from competing in preparatory horse trials to gaining fitness for their 3-day event. Both trot sets and especially gallops will be increasing in duration and intensity. As the work load increases, so does the stress on the hoof. It is important to anticipate potential foot problems and make necessary adjustments to prevent them from becoming serious. If the farrier feel a horse needs something different, now is the time to make those changes, not the next shoeing, which will be 7 to 14 days before the competition.

This shoeing sets the farrier up for the last shoeing before the competition. It is especially important to carefully manage the feet so they are in optimal shape for the 3-day competition. Careful thought must be given to all aspects of trimming and shoeing. The goal is optimal sole protection, excellent quality frog, and optimum hoof wall length, shape, and mass. The farrier is striving for maximum hoof capsule mass with appropriate mechanics.

April 15

Ideally, the farrier will have planned the entire shoeing schedule backward from the 3-day competition. This shoeing date should be 7 to 14 days before trot-up. Unlike other FEI disciplines, there are two veterinary inspections, one at the beginning of competition (Wednesday) and one at the beginning of the show jumping day (Sunday). To complete the competition, the horse must trot sound at both inspections, with the second one occurring 15 to 24 hours after the completion of the speed and endurance phase, which will be about 20 miles and vary in speed from a walk to 690 m/min and cover approximately 40 to 50 fences. Under FEI rules, horses may receive no medication, so the farrier is an important resource if foot and limb pain becomes an issue.

The horse works up to this competition all winter and spring; at the upper levels, there are only a handful of competitions per year. No matter how confident or skilled the farrier is, the horse should not be shod within 72 hours of the first veterinary inspection.

It is especially important to repeat the examination process at the last shoeing. At the conclusion of this shoeing, the farrier should watch the horse trot in a straight line to ensure that he is comfortable with the horse's soundness level.

The Competition

If at the time of competition, nothing but checking and watching the horse jog are needed, then the farrier has done their job. However, this is not always possible.

If possible, both the farrier and the veterinarian should watch the horse jog on the same surface as the one used for

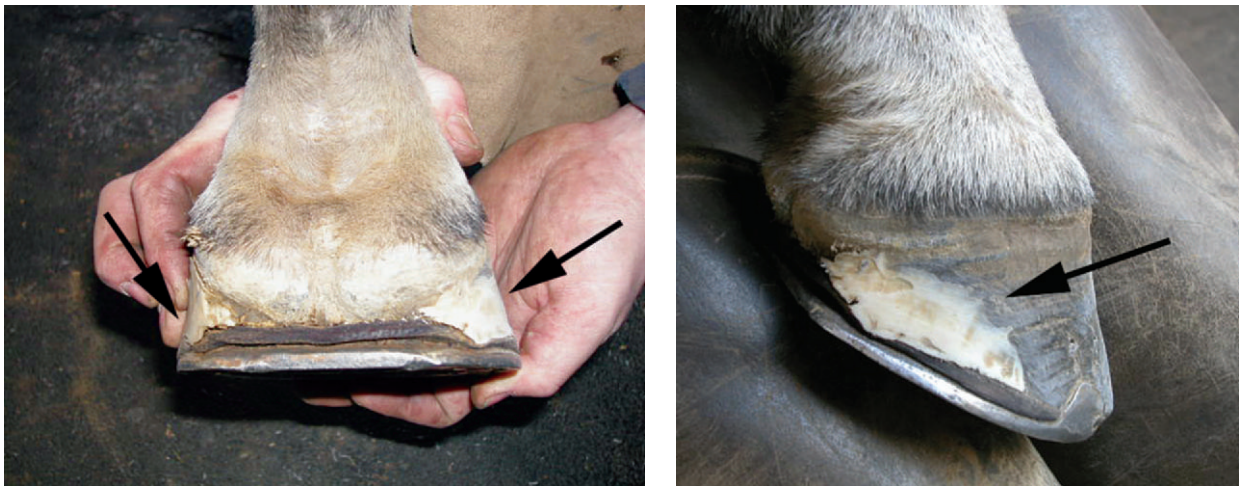


FIGURE 22-19 **A,** Use of Equibond to protect the heels of shoes during a cross country event. Note that there is no glue extending up to the hairline or around the bulbs to the rear. **B,** Lateral view.

trot-up the Tuesday before trot-up. The farrier is potentially in real trouble if adjustments need to be made to get through the first trot-up. Both the veterinarian and farrier should be involved to maximize success and so injuries secondary to foot pain can be considered and avoided. Dressage will be on Thursday or Friday. Many riders do not like to run cross country with the horse in bar shoes, because of the increased chance of shoe loss with some horses. These changes should be made on Friday evening or Saturday morning after dressage. At this time, depending on footing and the horse's needs, sole support material adjustments should be made. For example, if the footing is very soft, sole support is removed for better traction, or if the footing is rocky, sole support is added for protection. Equibond can be used to cover the shoe at the heels behind the widest point of the foot to prevent shoe loss (Figure 22-19). Spare shoes should also be checked if this has not already been done. A proper set of spare shoes should be well fitted, new, boxed slightly heavier than the set on the horse in case a great deal of hoof is lost with the shoe, and lightly concaved (Figure 22-20). Over-concaving will make last-minute adjustment more difficult.

Shoes can be replaced on the horse during the cross country event at three different places: the authorized assistance area at the end of B (steeplechase), the C holding box at the beginning of C, and the 10-minute box at the end of C. It is best to just tape up the foot at the end of steeplechase, since the horses are still fractious and the clock is running. The farrier will have several minutes to get shoes back on the horses in either 10-minute box (beginning or end of C).

The steeplechase and the cross country events (B and D) are the most common places for shoes to be lost. There is no rush to replace shoes lost on phase D. The next thing the horse must do is trot-up on Sunday morning. Problems that can occur when a horse loses a shoe, such as stepping on a clip or a nail are usually much more apparent after the horse has had 1 to 3 hours to cool down and get rid of adrenaline after the cross country event.

The three most common foot problems that can occur during the cross country event are grabs, heel soreness, and sole soreness. The best time to evaluate the horses is several hours after completion of the event and then again Saturday evening and very early Sunday morning. The soreness of

FIGURE 22-20 Foot surface of set of spare shoes demonstrating light concaving and heavier than usual boxing.

horses can change dramatically during this 12- to 16-hour period. During these evaluations, the extra knowledge the farrier gained over the last few shoeings by examining the horse and watching it go will be invaluable. No medications can be given during this period, so shoeing changes and peripheral care (e.g., icing, laser and ultrasound therapy, massage) are the only solutions for foot and lower limb problems.

Veterinarians and farriers must work together to isolate lameness associated with the foot. Hoof testers and a methodical examination and gait evaluation process will be necessary because no local anesthesia can be performed. Shoeing skill will be at a premium, as specific hoof mechanics must be just right to allow these sore-footed horses to trot sound. Unloading and corresponding loading of specific areas of the hoof capsule may be necessary to eliminate hoof pain (Figure 22-21). The farrier must be well versed in the use of sole support materials and the heart-bar shoe. Heart-bar shoes are par-

ticularly useful because of the additional stability they provide when unweighting a portion of the foot (Figure 22-22).

The period before the trot-up is one of the most demanding and stimulating situations a horseshoer can encounter. Problem-solving, creativity, and intuition are important ingredients to success.

The farrier should follow his horses to trot-up to ensure things go smoothly. A set of hand tools, hoof testers, sole support materials (for loading), and a saw blade (for unloading) should be at hand to make last-minute adjustments. The ground jury or official veterinarian may want to question the farrier if the horse is held. An added bonus of attending trot-up is the extra experience that is gained by seeing the standards set by the ground jury. This will help the farrier make important judgments concerning what is sound enough to pass evaluation by the ground jury. When to stop working on a horse is an important consideration in the work of the farrier.

FIGURE 22-21 Use of heart bar and Equipak to load and unload particular parts of the foot.

The next phase of the competition is show jumping. Often the comfort level of the horses will determine how well they jump. Work performed on Sunday morning can be factor in the competition results. At the stadium, all that will be needed is a set of hand tools in case of shoe loss during warm-up.

CONCLUSION

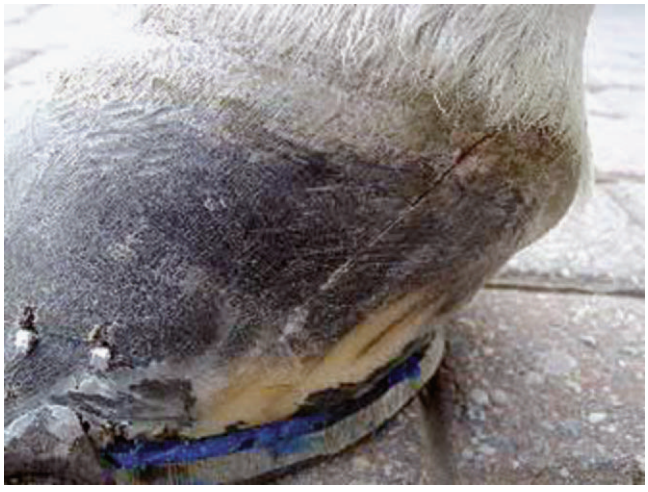
Eventing is an exciting discipline for the horseshoer. There is more opportunity for involvement and impact than in many of the other disciplines, especially during the competition. The upper-level three-star and four-star competitions are especially demanding because of the high levels of work that are required of the horse before and during the competition. This makes soundness issues much more likely to occur and recur. Appropriate decisions made before and during the competition by the farrier and veterinarian can positively influence the outcome.

The sport of eventing is in flux right now with the new shortened CCI format in effect for the first time at the 2004 Olympics in Athens. This style of competition does not have roads and track or steeplechase, phases A through C. This has changed the type of horse that competitors are choosing, with much more emphasis being placed on show jumping and dressage. Trot-up will probably be less of a problem. These eventing changes will be evolving over the next few years and may alter the role of the farrier in the care of the elite event horse.

SHOEING THE FOREST AND PULLING HORSE

Michael Wildenstein

The anatomy of the heavy horse hoof is the same as that of light horses. The difference is what is above the hooves. The size and weight of the draft horse will have negative effects



A

B

FIGURE 22-22 **A,** Use of bar shoe with Equipak and Play-Doh (to keep the Equipak out from underneath the wall/heel) to unload a specific quadrant of the foot. These techniques might be necessary before the second veterinary inspection on Sunday morning. **B,** A heart-bar shoe, which is particularly useful when attempting to unweight specific sore portions of the foot. When using this type of shoe to unweight parts of the foot, full and even contact of the frog plate with the frog is necessary.

on the hooves. It is important to have the working horse trimmed and reshod every 6 weeks.

TRIMMING

First, the farrier finds the parameter at the point of the frog. This parameter is found by trimming at the point of frog until a separation of the insensitive frog and insensitive sole is no longer visible. Next, the farrier finds the parameter at the collateral sulcus. This parameter is defined by finding the junction of the insensitive frog and the wall at the widest point of the collateral sulcus. The frog is trimmed to its natural symmetrical shape with a hoof knife, as the frog is the center of the hoof and is used as a guide for trimming and shoeing. With a hoof knife, the central sulcus is trimmed so that dirt and debris will not accumulate in this area. The bars of the hoof will need to be trimmed if they are folded over and enclosing dirt and debris. Rough exfoliating sole can be removed with a hoof knife. Dirt and debris are removed in the area of the white line, which is the junction of the wall and insensitive sole. Next, the farrier trims the excess hoof wall with nippers and uses a rasp to flatten the bottom of the hoof to a plane surface. Uniform wall thickness should be achieved.

Draft horses do not stand well with one hoof off the ground for prolonged periods of time. When working on a heavy horse, moving from one hoof to another provides the horse a break. The farrier should work on diagonals, especially when shoeing. The task should be completed on one diagonal before starting on another.

All horses need to be trained at an early age to stand for the farrier. Working on an adult heavy horse that has not been trained is very dangerous. The use of a hoof jack to help hold the limb will ease the task. Working with or around large animals can injure or kill those attempting this task, so care must be taken.

Shoes are applied for several reasons:

1. The hooves are wearing faster than they are growing, so there is a need for protection.
2. Traction is needed to provide the horse confidence and stability to perform a particular task.
3. The shoes are applied to remedy a disease or pathologic condition.
4. The gait or way of moving is altered with shoes to prevent interference or to animate the movement for aesthetic purposes.
5. Shoes may be applied because of a fad or custom.

Regardless of the need for shoes, trimming and regular hoof maintenance is important for the well-being of the horse. Shoes are applied by a qualified farrier by first trimming the hooves and then shaping the shoe to fit the hoof. The type of shoe will depend on environmental conditions type of work expected from the horse, and the quality of the hooves. The type of shoe for the horse working in the forest will depend mostly on the season. The following shoes are used on the author's horses for work in the woods in the northeastern United States. The shoe types that work well in this area may not work well in other places.

- In the spring, a shoe with toe and heel calks, as the mud demands traction for the horse.
- In the summer, a flat plate with drive-in studs and a leather pad to protect the sole and frog.
- In the fall, when the mud is seldom so deep as in the spring, the flat plates with a drive-in stud but without the pad. Despite the type of packing used between hoof and pad, mud seems to work its way under the pad, creating an environment conducive to decay. During the mud season shoes are lost. By reducing the baggage on the hoof, the placement of the shoes on the hooves can be maintained.
- In the winter, traction is a priority. Large removable studs are used to facilitate changing traction with the ever changing weather. Mustad No-Snow Pads (O. Mustad and Son, Aubum, NY) are used to keep snow from building up on the hoof.

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23
PREVENTIVE FOOT CARE PROGRAMS

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This chapter presents three individual approaches to developing a preventive foot care program (PFCP) for a particular practice or farm. The common components of each program are (1) detailed assessment, tailored for the needs of the practice or farm population; (2) good record keeping; (3) regular reassessment; and (4) prompt action when a potentially detrimental situation is identified or something has changed for the worse since the previous evaluation.

Before discussing each of these programs, it is worth pointing out that the concept of preventive foot care is much broader than the careful recording of certain measurements by a veterinarian or farrier. It encompasses good environmental management, good nutrition, and common sense. Keeping the barn, paths, and paddocks free of nails and other objects that could damage the foot; providing a safe, clean, level, well-lit workplace for routine shoeing, examination, and treatment of the horse's feet; and always being safety conscious when working with and around horses are every bit as important as measuring hoof angles and recording data from radiographs.

In each of the following sections, a team approach is emphasized. The team includes, at a minimum, the horse's owner (or owner's agent), the farrier, and the veterinarian. Each one is responsible for their part in preventing problems, both during a single visit and over the long term. The owner's or agent's role, for example, includes following the farrier's recommendations regarding shoeing interval, one simple yet very important aspect of preventive foot care. An irregular shoeing interval on its own can lead to lameness, especially if the interval between shoeings or trimmings is allowed to extend for several days or more beyond that recommended by the farrier. This situation can also lead to problems if the farrier feels pressured to remove a lot of horn at one time to compensate, thus leaving the sensitive tissues of the horse's foot with too little protection.

Another simple yet often overlooked aspect of preventing foot problems is to avoid hard work, especially tight circles (like longeing), or work on hard ground immediately after a horse is shod. The contact surfaces of the foot are softer after the hardest horn is trimmed away and so are potentially easier to bruise. The angles in the lower limb joints may be slightly altered because of the trimming, so giving the horse a few nonactive hours to adjust immediately after trimming or shoeing is wise in terms of preserving the joints. This can be very important to an older horse whose joints are beginning develop arthritis or to a very young horse whose joints have not become fully conditioned. It may also be helpful to restrict pasture turnout for 12 to 24 hours to avoid galloping and spinning around, especially if the footing is dry, firm, and a bit rocky or irregular. Finally, most prudent of all is to schedule shoeing far enough in advance of an important competition or sale to allow for any minor pain adjustments to occur so the horse is in peak condition.

In addition to lameness caused by trimming too much horn, by close nails, or by bent nails, the farrier (and owner) must also be aware that horses with chronic joint pain (osteoarthritis, in particular) may be more stiff or lame for a few days after shoeing, simply from having had the affected joints flexed or extended beyond their comfortable limits or from having to stand on the lame limb while the opposite foot was trimmed. It would be the owner's or agent's responsibility, as the person in charge of the horse, to let the farrier know beforehand that the horse has chronic joint problems. The farrier can then make positional adjustments accordingly to minimize the horse's discomfort during trimming and shoeing.

Another factor that can cause stiffness or lameness for a couple of days after trimming and shoeing, of which many farriers may be unaware, is vaccination. It is not uncommon for horse owners to have several routine health care procedures performed at one time, such as vaccination, deworming, and trimming and shoeing. It is also relatively common for horses to be a little muscle-sore for a few days after intramuscular injection with vaccines. Unless this fact is taken into consideration, the farrier is likely to be blamed if the horse is moving a little stiffly or is lame after this preventive health care blitz. In rare circumstances, stiff gait from systemic problems such as "tying up" or pleuropneumonia coincidentally originating at the same time as trimming and shoeing can mimic close trimming. So stiffness of a gait after trimming and shoeing is not always the farrier's fault. Any horse that is not sound in 2 days after trimming should have a thorough veterinary examination to determine the specific cause of the stiffness.

These various scenarios illustrate that veterinarians and farriers must extend their concept of preventive foot care to include the everyday care of the horse and to everything they do to and with the horse and should encourage their clients to think along the same lines. The following discussions present a more structured programmatic approach to preventive foot care for veterinary practices, farriery businesses, and even for breeding or training facilities.

VALUE OF A PREVENTIVE FOOT CARE PROGRAM

A preventive foot care program is of value to a veterinary practice or farriery business for several reasons:

- It identifies problems that may otherwise have been missed until they become more serious.

- It allows ongoing monitoring of an individual horse.
- It provides data on the population of horses regularly seen in the practice or business.
- It enhances wellness programs and client education.
- It has economic value for all participants.

Early Identification and Intervention

Most people involved with horses believe in the axiom, "no hoof, no horse," although it is not put into practice very much in the breeding or purchasing of horses or even in courses and research in educational institutions. A program that increases awareness of the horse's foot would have value. For example, a study reviewed 1852 pictures of stallions from eight different annual breed magazines and found that in only 241 pictures (13%) were the horse's feet visible. In 39%, the stallions were standing in grass. In one magazine, some of the horse's feet appeared to be air-brushed in.[1] The authors were not suggesting that equine foot conformation is completely inherited but were suggesting that mare owners might not be seeing the complete picture.

On a case-by-case basis, the greatest value of a single PFCP evaluation is the identification of a particular problem, or the propensity for that problem, that may otherwise have gone unnoticed until it evolved into a serious issue. Analysis of data from 50 horses that were enrolled in a model PFCP found that 66% of the horses had a problem that was unknown or not being addressed by the owner, farrier, or veterinarian.[2] Some of these problems were relatively minor, such as the horse being slightly overweight or having inadequate heel support. However, other problems were potentially more significant, such as suspensory problems, thin soles, ringbone, and coffin bone rotation.

Each of the 50 horses in this study were considered sound by their owners and were in work at the time of the evaluation. By identifying these particular problems, the owner, farrier, and veterinarian were then in a position to initiate a plan that would hopefully protect the horse from future aggravation or advancement of these problems. The initial evaluation also served as a baseline, or reference point, for monitoring progress over time.

Laminitis is a potentially devastating disease, so any steps taken toward its prevention are well worth the effort. Even a single, mild episode of laminitis can have permanent consequences. And not only does that bout increase the potential for laminitis to recur, but subsequent bouts of laminitis can cause cumulative damage. Thus, one particularly important service the PFCP can provide an individual horse and owner is to identify subtle physical or radiographic indicators of a prior episode of laminitis. That horse is then identified as being at increased risk for laminitis, and the veterinarian or farrier can educate the owner on risk factors and management strategies aimed at reducing the risk for future bouts of laminitis.[3]

Data Collection and Record Keeping

Collecting and recording consistent information on a particular horse is perhaps the most important component of any PFCP. The data have immediate value, as they assist in determining the horse's current needs. When the assessment is repeated at a later time, it yields even more valuable data, as comparisons can be made between the current and previous assessments, which assists the veterinarian and farrier in making both immediate decisions and long-range plans for that horse. When results from several horses are compared, these data also help document trends that may be occurring in a particular veterinary practice or farriery business.

Data collection and analysis has an even broader value: it allows the veterinarian or farrier to document specific cases or types of foot problems for publication in veterinary or hoof care journals. Much of what is accepted as true regarding hoof problems and the value of specific shoeing strategies is anecdotal, that is, experience based, at best. Organized and purposeful collection and recording of data from a large number of horses over time will greatly enhance our understanding of the genesis and progression of various foot problems and thus the management and perhaps even the prevention of these problems. In medical circles, this approach is called *evidence based*. Although the evidence-based approach is not flawless and has its limitations, it can help advance our knowledge and debunk some commonly held yet poorly founded beliefs in both professions.

Practice Enhancement

In addition to being of value to individual horses and their owners, a PFCP can add considerable value to a veterinary practice or farriery business.

Veterinarians

In a veterinary practice, a PFCP could be a stand-alone program or be integrated into an existing preventive medicine program that might include wellness, vaccination, and dentistry. It could also be incorporated into a mare/stallion reproductive health care program, and even into a foal preventive medicine and management program.

This tailored PFCP could then help to enhance the growth of a practice. It could be part of an internship program in a practice, or be an excellent way of introducing a new associate to the practice clientele. A PFCP could also be used to expand technician responsibilities, especially in those states where the Veterinary Practice Act allows indirect supervision for veterinary technicians. In fact, such a program could lead to the development of an entire podiatry special interest section of a practice.

If organized appropriately, a PFCP could even help in transitioning a veterinary practice as it grows. For example, if a one-veterinarian practice had enough work for one and a half veterinarians, but economics did not allow the hiring of a full-time associate, starting or upgrading a PFCP might provide sufficient income to hire a second full-time veterinarian. If a veterinary practice were looking to up-grade its imaging equipment, developing an organized, wisely marketed PFCP could be helpful (A.R. Clark, personal communication, 2004).

Farriers

Being involved in a PFCP, whether self-initiated or part of a veterinarian's program, encourages the farrier to keep good records of each visit. It is a great tool for teaching apprentice farriers specific aspects of hoof care and the importance of keeping detailed records. Such a program also provides the farrier with a valuable tool for evaluating new horses, educating clients, monitoring a horse's progress, and assessing specific trimming or shoeing techniques. Finally, such a program

would enhance the relationship of the owner and veterinarian with that farrier practice to give better, educated help to the horses in their care.

Clients

By instituting a PFCP, the income of the veterinarian and farrier are enhanced in a way that also provides financial value to the horse owner, a "win-win-win" situation. The most obvious financial value to the owner is in identifying existing or potential problems and addressing them before they have a costly effect on the horse.

Perhaps less obvious, but no less important, is the educational value for the horse owner. From the first evaluation, the client will learn new things about the horse's feet, and about foot care in general, which will have both immediate and long-term benefits. Not only will the daily care of the horse be improved, but the client will be better able to identify any future problems early enough for appropriate intervention to avoid any serious consequences.

A third potential value to the client is the detailed historical information this program provides, should the horse develop a serious medical problem that requires referral to a specialty hospital or podiatry clinic. When serious foot problems develop, having detailed baseline information is a tremendous asset in case management and in formulating a prognosis.

DEVELOPING THE PROGRAM

A PFCP can be developed in many different ways. There is no specifically accepted preventive foot care program, only measurement suggestions and models. When determining which indices to include, the principal goals for the program in the particular practice or business should first be defined. This can be used as the guiding principle to decide what data should be collected, what conditions identified or studied, what service provided, and what questions answered. The practitioner should also consider what methods of assessment are at his or her disposal. For example, a veterinarian may include radiography or thermography, whereas a farrier may use physical measurements and photography. A computer program is available to farriers and veterinarians to assist in data collection.[4]

In the PFCP these authors developed, three general aspects for each horse are assessed:

- Body condition and body type
- Foot and lower limb conformation
- Specific hoof measurements

The following descriptions are not intended to be comprehensive in scope or detail. They do not represent a complete list of measurements that can be used in an individual PFCP, but rather they serve to provide an example of how such a program can be devised and implemented. The primary goal of the program is to bring to awareness any change in the horse's foot that may have detrimental implications. Regardless of which indices are measured and what else the evaluation entails, keeping detailed records (including specific measurements, photographs, radiographic findings, and the dates of the evaluations) is critical to the success of the program.

Body Condition

In human medicine, the patient's weight is an important indicator of health and is a standard part of the medical evaluation. This numeric medical indicator has steered diagnostic testing and guided treatment plans for generations in human medicine. The effect of increased weight has been examined over the long term in paired littermates in dogs. The prevalence and severity of osteoarthritis in several joints was less in dogs with long-term reduced food intake as compared with their mates fed ad libitum.[5] Although body weight is an important index in most branches of veterinary medicine, precise weighing of horses has not achieved by the vast majority of horse owners and veterinarians. Visual assessment of body condition or tape measuring is often used in its place. So having some way to make owners aware of their horse's weight has the potential of increasing the long-term health of the horse.

Body Condition Score

The body condition score (BCS) system is based on the visual assessment of regional fat accumulation on the horse's body. As Table 23-1 depicts, the BCS system for horses assigns a visual score between 1 (emaciated) and 9 (obese). Regardless of the horse's age, breed, gender, height, body type, and body weight, a BCS of between 4 and 5 is considered ideal; a BCS of 6 or more indicates that the horse is overweight. Potentially BCSs of 1 and of 9 are equally unhealthy for a horse and can be equally life threatening. This system can easily be learned by any horse owner, farrier, or veterinarian and the horse's BCS recorded regularly (e.g., quarterly or semiannually) for monitoring.

As a general guide, it takes a 20% reduction in total dietary intake (grain, hay, and pasture) and about 2 weeks to decrease a horse's BCS by one grade (e.g., from 7 to 6). Of course, regular exercise also is important in any weight reduction effort and is an essential component of any preventive health care program. A definition of regular exercise could be some form of structured exercise (e.g., driving, ground exercises, riding, longeing) at least 5 days a week, never missing more than 2 days in a row. In horses or ponies with very high or very low BCS, weight loss or weight gain should be done more slowly to prevent metabolic crises.

Weight Tape

A weight tape is often used by horse owners to estimate the horse's body weight. However, this method is quite inaccurate, and it does not factor in the horse's body type or amount of fat deposition. A possibly more accurate method for using a weight tape would be to record the girth circumference, the body length from the point of the shoulder to the tuber ischium, and the height at the withers. The weight could then be estimated by the following formula:

$$\text{estimated weight (kg)} = [\text{girth (cm)}]^2 \times \text{length (cm)} \div 11{,}877$$

The body mass index could be calculated as[6]:

$$\text{estimated weight (kg)} \div [\text{height (m)}]^2$$

To be as accurate as possible, the same weight tape or tape measuring technique should be used by the same person each

Table 23-1 Body Condition Scoring System for Horses*

Rights were not granted to include this table in electronic media.
Please refer to the printed publication.

From King C, Mansmann RA: *Preventing laminitis in horses,* Cary, NC, 2000, Paper Horse.
BCS, Body condition score.
*Regardless of the horse's age, gender, breed, height, and body weight, a BCS of 4 or 5 is ideal. Horses with a BCS of 6 or greater are overweight.

time. For veterinary practices or farms that want to accurately weigh horses, livestock scales should be used.

Importance of Evaluating Body Condition

The importance of assessing and documenting the horse's BCS lies in the fact that overweight horses are at increased risk for laminitis. Being overweight also worsens the debility and hampers the management of other orthopedic conditions, including foot problems. Putting a number to a horse's body condition enables the veterinarian or farrier to emphasize a horse's risk for these problems, impress upon the owner the importance of weight reduction for that horse, help the owner set goals for weight reduction, and also monitor their progress.

There are certain physical indicators that a horse is at increased risk for laminitis. One is a body type often described by horse owners as an "easy keeper" or a "good doer." These are the horses that have cresty necks and excess fat deposits at the base of the tail and over the shoulders, and perhaps in other locations (e.g., in the sheath in geldings, and just in front of the udder in mares). These horses gain weight easily, and they lose weight slowly or not at all with mere dietary restriction. This body type is seen in all breeds, but it is more common in some, such as Morgans and ponies. These horses may fit into a syndrome currently being investigated as equine metabolic syndrome.[7,8]

Another telltale physical picture of increased laminitis risk is the older horse that has pituitary gland dysfunction (equine Cushing disease).[9] It is common for these horses to have hair coat abnormalities. Initially, the horse may simply fail to shed its winter coat in the spring; in more advanced cases, the horse may have an abnormally long, wavy, or curly hair coat. These horses frequently have other physical signs of equine Cushing disease (see Chapter XXX). Not only are horses with this condition at increased risk for laminitis, but laminitis can be very difficult to manage in these animals unless appropriate medication is given to control the abnormal pituitary gland output. These horses can also be at risk for slow healing or recurrent foot abscesses. Thus farriers need to be aware of the existence of this condition and its common manifestations and need to urge veterinary intervention if the owner has not already sought veterinary advice.

Example

Many people consider Thoroughbreds to be "hard keepers," in other words, it is difficult for them to gain or maintain weight. However, Thoroughbreds, as with any other breed, can become overweight on a high-carbohydrate diet and thus be at increased risk for laminitis. In fact, an overweight Thoroughbred may be at even higher risk for laminitis, as so many Thoroughbreds have relatively poor-quality feet. The thin-soled foot conformation so commonly found in this breed may put these horses at greater risk than horses with thicker soles.

Unfortunately, it is fairly common for older Thoroughbred stallions to be euthanized because of laminitis, whatever the inciting cause. Typically, these horses are fed high-carbohydrate diets (e,g, grain, lush pasture, the best hay), even though they are no longer in race training; as a result, many are overweight.

One example relates to a valuable Thoroughbred stallion in just such a condition—overweight and suffering from repeated bouts of laminitis in May. As a routine, the farm manager weighed his horses monthly and so had detailed records of the stallion's body weight for the past few years. A review of those records showed a cyclic increase in body weight from 1260 lb in January to 1330 lb in May, when the stallion had episodes of laminitis.

The amount of grain and hay fed to the horse had not increased during that time, but the amount and quality of the grass in the horse's paddock had dramatically increased during the spring. After reviewing the weight records, the conclusion was drawn that this stallion's ideal body weight

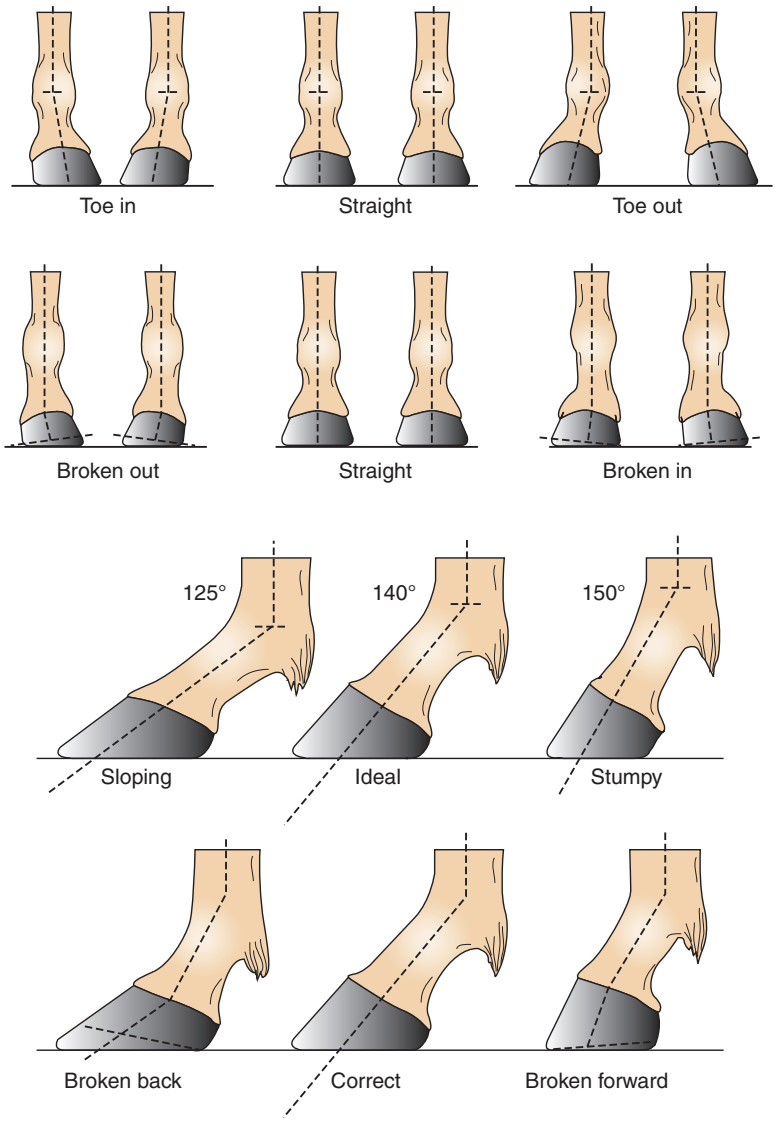

FIGURE 23-1 Several lower limb positional abnormalities, compared with normal conformation, with their accepted nomenclature. *(Modified from Butler D:* The Principles of Horseshoeing II, *LaPorte, Col, Butler Publishing.)*

seemed to be around 1250 lb. By simply keeping the stallion's weight at approximately 1250 lb through dietary management, further bouts of laminitis were prevented.

In some human patients with severe heart disease and other serious health conditions, a change in body weight of as little as 1% (1.5 lb for a 150-lb patient) can precipitate decompensation.[10] If the same is true in horses, then monitoring body weight closely and making management changes accordingly may have significant preventive benefits in high-risk horses or, for that matter, any horse.

Foot and Lower Limb Conformation

When considering conformation in any horse, the examiner should generally look for symmetry. Ideally symmetry means that the left foreleg is a mirror image of the right foreleg, as are the hindlegs. The normal horse may have a bit less bearing surface medially (slightly straighter wall) than laterally, so not be perfectly symmetrical medial to lateral on each leg.[11]

Evaluating and recording the horse's lower limb conformation in some way has value in a PFCP, as it indicates how the horse probably loads the foot (Figure 23-1). Photographs can be very useful in recording and monitoring a horse's foot conformation, especially when the photographs are taken in a consistent manner, with the camera placed in the same position each time. There are many different types of abnormal foot conformations. Figure 23-2 models several foot abnormalities with their accepted nomenclature. Another way to describe and record hoof conformation is to make a full-scale model of the horse's foot. Although time-consuming to prepare, the resulting life-size, three-dimensional model of the hoof is a durable, anatomically precise, and detailed material record of the foot from a particular point in time that no two-dimensional representation or written description can match.[12]

Recording conformational information allows the veterinarian or farrier to monitor changes in hoof conformation over time. A common example is the Thoroughbred that has a propensity for developing the long toe/low heel foot confor-

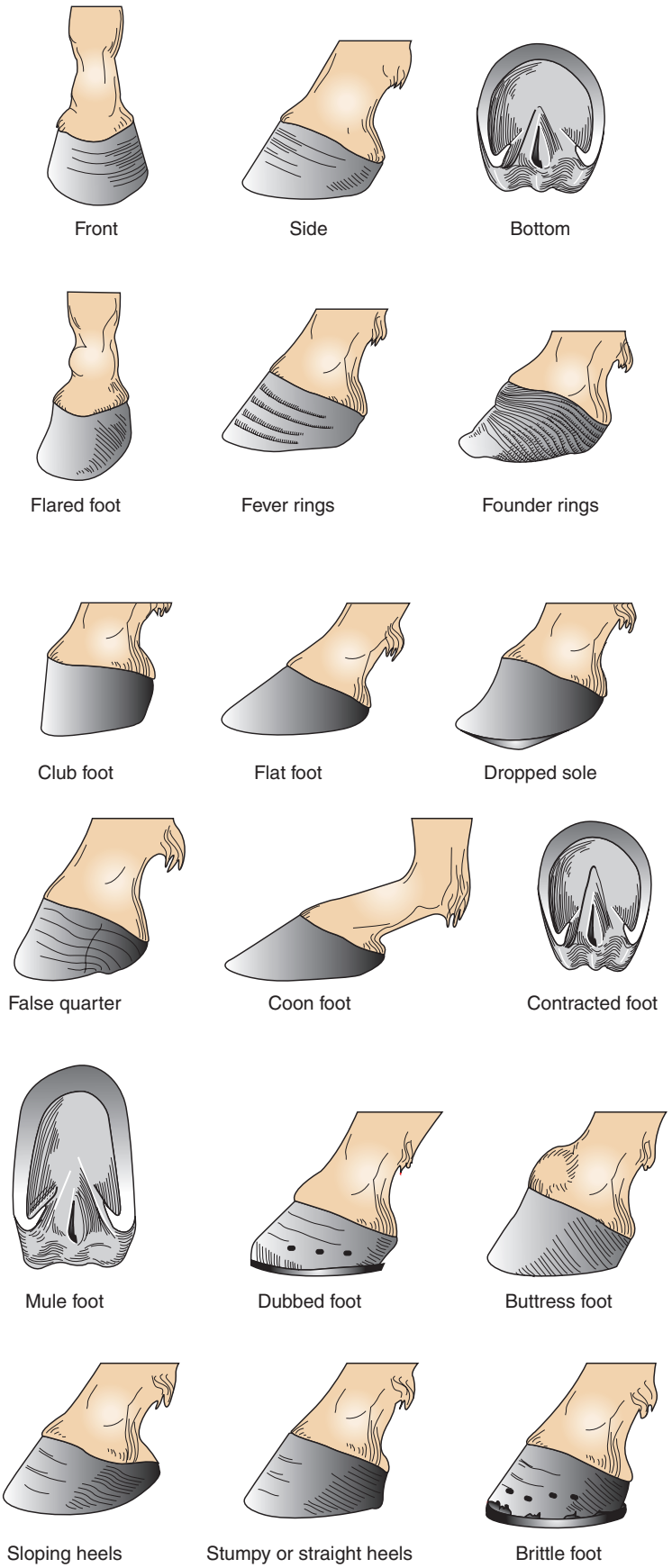

FIGURE 23-2 Several foot conformation abnormalities with their accepted nomenclature as compared to ideal. *(Modified from Butler D:* The Principles of Horseshoeing II, *LaPorte, Col, Butler Publishing.)*

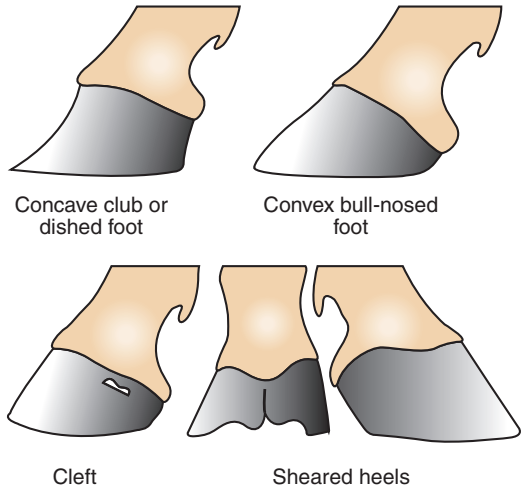

FIGURE 23-2, cont'd

mation. Another example is the horse that has a vertical medial wall that has the propensity to tuck under or become "broken-in" (i.e., the bearing surface of the medial wall is located axial to the coronary band above it).

As the horse ages, any conformational defects could become progressively more exaggerated for many reasons if not diligently addressed. These conformational distortions may worsen only slightly from one shoeing to the next, so changes may not be very apparent unless careful records are kept and data from previous evaluations are compared with current findings.

Several factors, most of which are preventable, can contribute to the worsening of these conformational problems:

- Environment, especially that which causes repeated wetting and drying of the feet
- Irregular shoeing intervals
- Shoe loss, with delay in protection of the bare foot
- Musculoskeletal pain that causes compensatory changes in load of the limbs
- Improper trimming or shoeing

Examples

Following are two common examples of how a conformational problem could slowly worsen over time, owing to a combination of inherent predisposition and human mismanagement. The first situation involves a horse that is in the process of developing a longer toe, a flatter sole, and a lower heel.[13] The horse may be a bit overweight, increasing downward forces into the hoof capsule. The horse may be kept in too moist an environment, softening and weakening the hoof decreasing support, increasing separations and flattening the sole. As the sole flattens, blood flow to the solar corium may be compromised, so sole growth is slowed; thus this horse develops not only a flatter sole but a thinner sole as well. As a result, the horse is prone to sole bruising.

Of course, nothing occurs in isolation, so at the same time as the sole is flattening, hoof mass is decreasing, particularly in the heel area. With such a flat, thin sole and now lowering heels, the farrier is careful to avoid trimming too much off the underside of the foot, and the toe then elongates. This situation could occur in the shod or barefoot horse. In the shod horse,

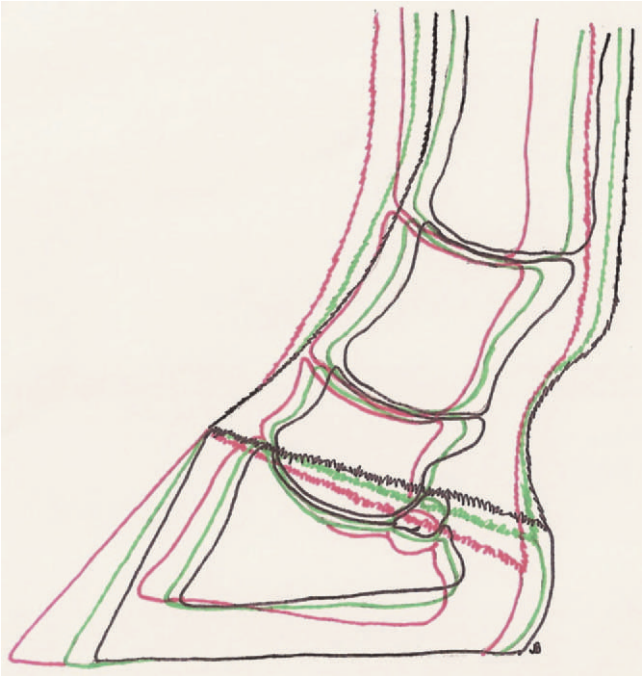

FIGURE 23-3 The black lines represent normal foot anatomy with a positive palmar angle. As time progresses under various adverse conditions, such as poor trimming and environmental management, the loss of posterior hoof mass occurs with decreasing palmar angle and increasing broken back hoof-pastern axis (*green* to *red*).

however, the farrier may apply perimeter shoeing, which involves placing the shoe to support the entire perimeter of the hoof. Perimeter shoeing works well for normally conformed feet but may not adequately support the bony column with various abnormal foot conformations. Thus in this flattening sole, lowering heel situation, shoeing the entire circumference of this hoof will lead to the shoe creeping forward in successive shoeings. At the same time, the bearing surface of the hoof wall at the heels "runs" further under the foot than is ideal for optimal hoof loading, which weakens the wall and overloads the underlying soft tissues at the heels. This horse may have adequate heel support but often does not. The use of shoes with premade quarter clips may also contribute to this situation, as the clips may not allow the shoe to be placed far enough back under the foot to give adequate heel support.

In this situation, it is not uncommon for the hoof angle to progressively decrease by approximately $^1/_2$ to 1 degree per year. At the same time, the loss of hoof mass at the heel (e.g., in the digital cushion) results in a lower palmar angle (the angle of the palmar margin of the coffin bone relative to the ground surface), which increases tension in the deep flexor tendon and diminishes the digital cushion (Figure 23-3).

This worsening of an already poor foot conformation exposes the sensitive tissues in the heel area, including the navicular bone and its supporting structures, to increased concussion and overload during weight bearing. It also alters the biomechanics of the limb, requiring more leverage during breakover and lift-off. All of these factors can result in chronic heel pain (Figure 23-4).

The second situation involves a horse with a broken-in foot conformation, in which the hoof wall on one side of the

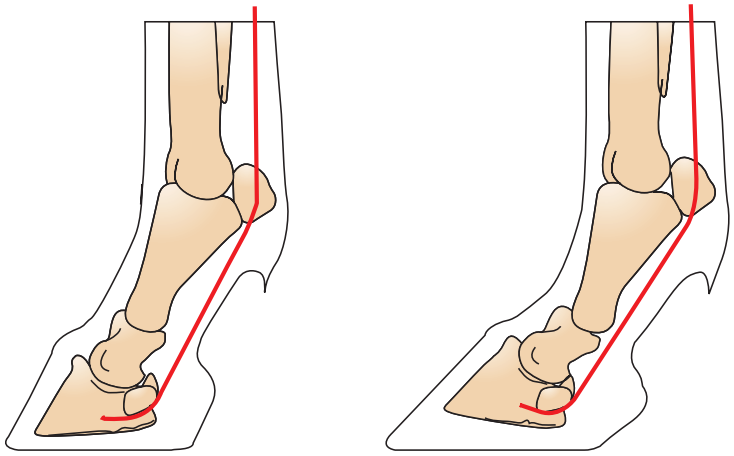

FIGURE 23-4 Factors such as increasing toe length, decreasing heel height, and increased stretching of the deep digital flexor tendon *(red line)* result in chronic heel pain.

foot (typically the medial side) becomes more upright than normal. If not appropriately addressed, this problem can progress to the point at which the wall on that side tucks under, such that the bearing surface is actually located more axially than the coronary band above it (Figure 23-5). (In other words, rather than coning out from top to bottom, the hoof wall cones in at that quarter or heel.) Perimeter shoeing rather than placing the shoe properly under the bony column of the horse, then, will aggravate this conformational foot condition just like it did in the long toe/low heel previous example. This abnormal conformation becomes self-perpetuating as vertical load during weight bearing encourages the tilted wall to be broken-in further.

In both of these worsening foot conformation situations, sooner or later (depending on what else is going on in that foot and limb), the horse will become lame. Rehabilitation for these feet is long and difficult and often only partially successful. In both of these situations, a preventive foot care program would have identified the foot conformation problem and monitoring may have helped in the earlier intervention to prevent continuing damage to the underlying anatomy. By using a combination of recorded measurements, written descriptions, and photographs, the conformation of the foot can be monitored as appropriate intervention hopefully moves the situation in a positive direction.

RADIOGRAPHY

Radiography of the feet is an important component of a PFCP. At a minimum, a lateral radiograph of each forefoot should be considered, but a lateral and dorsopalmar podiatry film of each forefoot and hindfoot would make a more complete preventive program. When the radiographs are taken with the shoes on, a pictorial record is created. In addition, specific measurements can be made from the films for current or future reference.

Radiography of the equine foot is discussed in Chapter 10. The section on radiographic views that are of particular interest to farriers details the techniques for taking routine lateral and dorsopalmar views of the feet. The lateral view, with the beam centered approximately 1/2 inch (1 cm) above the bearing surface of the wall, is a standard part of this author's PFCP evaluation.

To ensure reliable, repeatable films, there is great value in having a positioning system for both front feet and hindfeet that allows the horse to stand on wooden blocks of appropriate height (with both feet on blocks at the same time) while allowing the cassette to be supported against the foot, perpendicular to the beam, without anyone holding it. This author has two systems (Figures 23-6 and 23-7). Both the front feet blocks and hindfeet tray can be used for true lateral and dorsopalmar views to be taken. It is much easier to direct a horse's front feet onto a block with precut slots for various foot radiographic views. The ideal hindfoot system would allow the horse to walk up on it and comfortably stand anywhere on the "blocks." The hindfoot system is composed of a 24 × 30-inch tray of 1 1/2-inch square wooden dowels (designed by R.F. Redden). Each dowel can be taken out one-by-one to create a slot to hold the cassette anywhere in the tray. With the removal of different dowels, the cassette can be positioned such that it lies parallel or perpendicular to the sagittal plane of the foot, thus allowing a true lateral or dorsopalmar projection to be made regardless of the individual horse's conformation or stance. Once the horse stands the hindfeet on the tray, blocks are put under the front feet to even the horse's stance. This tray-and-block system is stable and robust enough to support the horse's weight.

A radiopaque marker such as soldering wire of known length to delineate the dorsal hoof wall can be easily taped onto and conformed to the shape of the anterior hoof wall. If the exact length of the wire is known, the amount of magnification present can then be calculated. It can also help with measurements if used in any computerized program. Barium paste also can be used to outline the anterior hoof wall. Consistently determining the skin-coronet junction for the placement of the wire or paste is important.

Radiographic measurement norms have not been clearly established and obviously would depend on consistent radiographic technique and specifically what each researcher or clinician used as a technique. The authors use a 60-cm focal to film distance with the beam 1 cm above the hoof shoe contact on a line midway between the anterior and posterior border of the

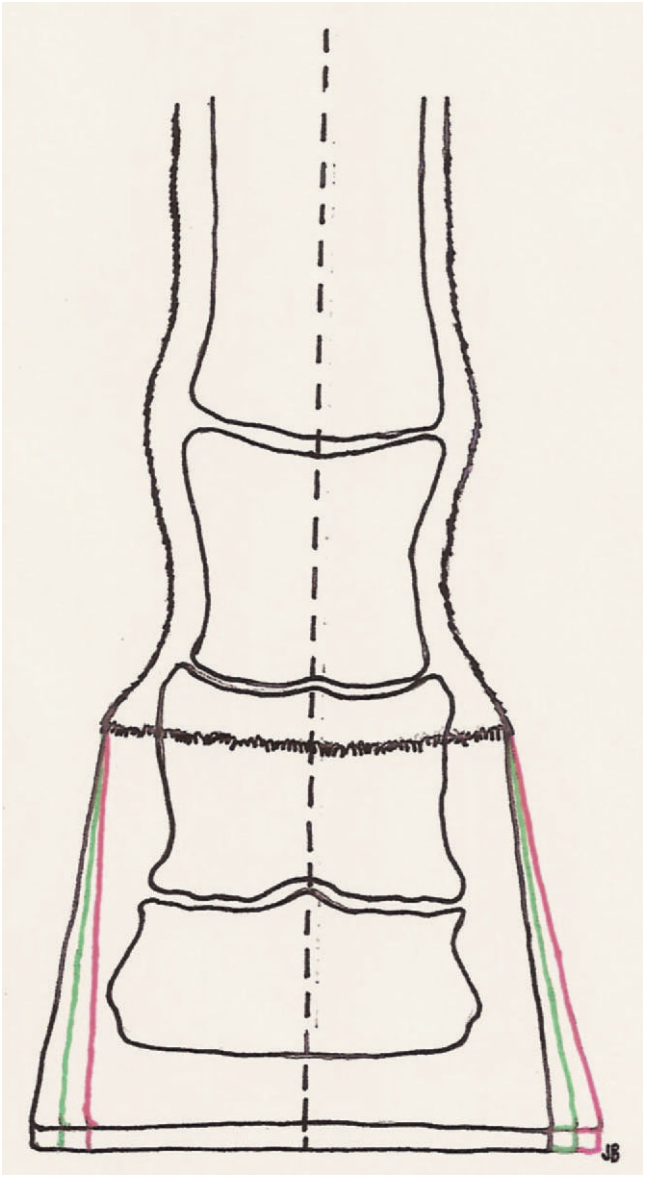

FIGURE 23-5 As foot conformation is incorrectly trimmed "supporting" a broken-in or broken-out foot conformation, the medial-to-lateral imbalance continues to increase (*green* to *red*).

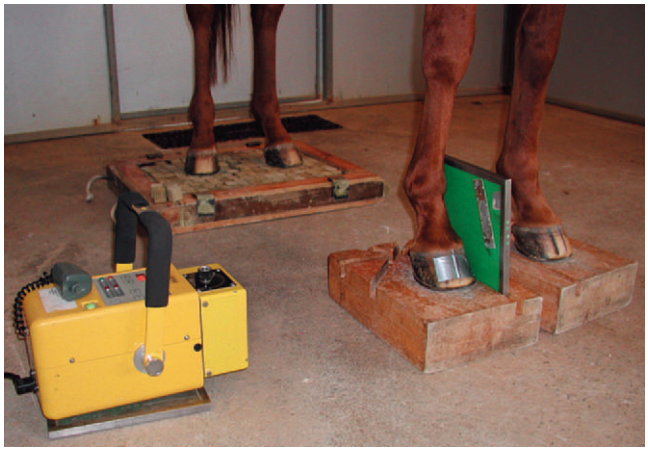

FIGURE 23-6 Front foot blocks that allow for both front feet to be off the ground equally. The block is high enough so the central beam of the x-ray machine points about ½ inch (1 cm) above the hoof-bearing surface or hoof-shoe contact. Slots can be cut in the block to reduce handling of cassettes, thus reducing technician exposure. If the blocks are too high, the x-ray machine can be raised appropriately for the proper central beam height.

FIGURE 23-7 A tray with removable dowels created to the height allowing for the x-ray machine's central beam to strike the foot ½ inch (1 cm) above the hoof's bearing surface. In many instances of hind foot radiography, this type of tray is easier to use than blocks. The tray is slid under the horse, between the front and hind feet. The horse is then "asked" to step up on the tray. The horse can stand comfortably on the tray without needing to have both feet precisely positioned. The cassette can be placed at any perpendicular position by removing the appropriate dowels. The radiograph is then taken with the vertical beam positioned from a line drawn down from the middle of the coronet. This tray can be used for horses with sore front feet, making it easier for them to step up onto the tray rather than having each limb picked up and placed on a block.

coronet. Currently this author uses the combination of measurements delineated on this schematic taken from two authors' radiographic foot evaluations (Figure 23-8).[14,15] A review of 110 preventive foot evaluations demonstrated that these measurements seem to have some validity (R.A. Mansmann, unpublished data, 2005).

HOOF MEASUREMENTS

There are many ways in which to measure a horse's foot. The practitioner should choose the indices that make the most sense within the context of the particular PFCP and that he or she is likely to continue using. A key factor in the success of any preventive program is simplicity and the ease with which data are collected and recorded. If data collection or recording is made too tedious, the program will fail. New measurements can always be added to the program.

Some simple measurements that can be taken directly from the foot include the following:

- Hoof angle—angle of the dorsal hoof wall relative to the ground surface
- Hoof width—measured across the widest part of the foot; foot tracings with foot standing on paper can be added to this measurement.

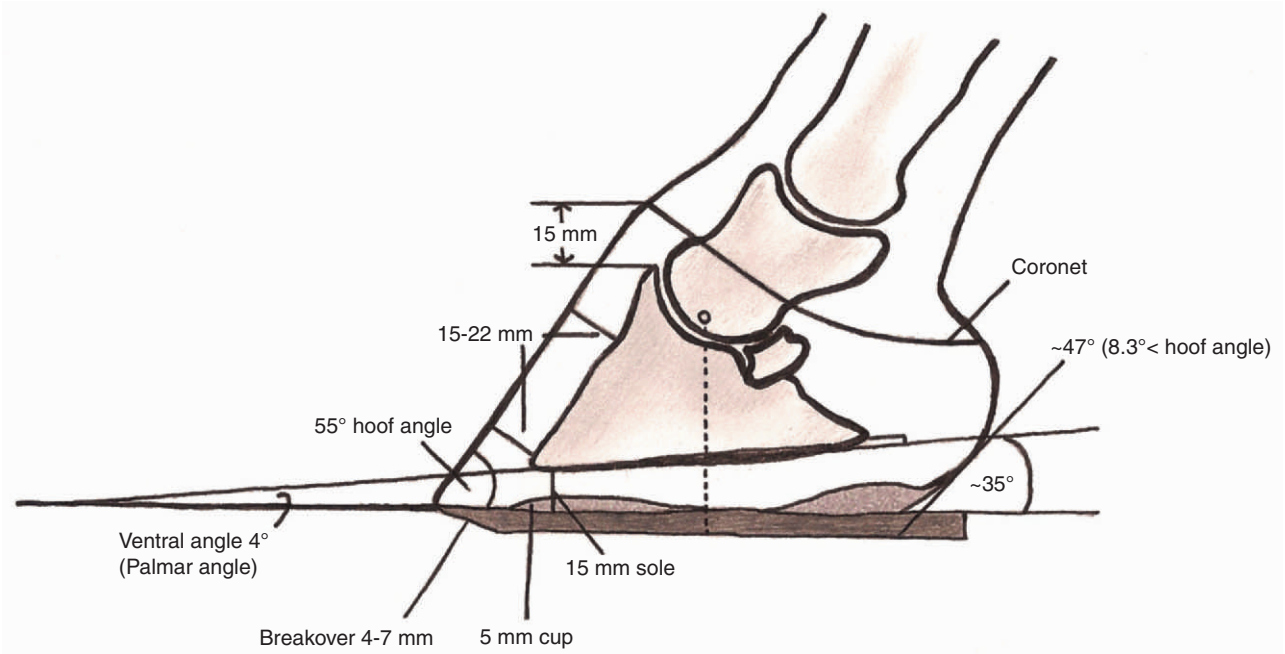

FIGURE 23-8 These measurements taken from a 60-cm focal distance with the medial side of the hoof touching the cassette can be used as working "normals" for a healthy horse's front feet. Some measurements, such as the H-L zone (15 to 22 mm), vary according to the size of the horse. Appropriate breakover in front of the tip of P3 (4 to 7 mm) varies with the size of the horse.

- Toe length—height of the dorsal hoof wall, from coronet to ground surface

Measurement of hoof angle requires a hoof gauge; the other two measurements require just a ruler. Other simple measurements, such as coronary circumference, may be of use in a particular practice population. Regardless of which measurements are routinely made, it is important that each measurement is performed by the same individual, using the same instrument each time. That way, operator variability (i.e., margin of error) is kept as small as possible.

Using Hoof Angles

Hoof gauges have their own controversies but can have value when used in a consistent manner.[17] The measurement can be verified by comparing it to the lateral radiograph. For example, lower hoof angles generally have lower palmar or plantar coffin bone angles. High hoof angles can be lowered by trimming the heels. Low hoof angles are very difficult to raise.

One way in which hoof angle measurement is helpful is to determine the optimal shoeing interval for a particular horse. This approach is especially useful in horses with mismatched hoof angles. An example is a horse that has a narrower, upright foot on the left side. Immediately after being shod, the hoof angles on the left and right feet are the same or only a degree or two different. The angles are still similar 2 weeks after shoeing; but by 4 weeks after shoeing, the angle on the narrower foot (the left forefoot) increases, and by 6 weeks after shoeing there is at least a 4-degree difference between the two feet. This information suggests that a shoeing interval of 4 to 6 weeks would be appropriate for this horse.

Another example of how hoof angles can be used in a PFCP relates to change causing lameness. A particular horse was followed through the program for 2 years, during which time he consistently had normal foot conformation and hoof angles of 53 degrees bilaterally. The rider and horse went to boarding school. The horse did fine for about 3 months but then started having lameness problems. After multiple veterinary visits, diagnoses, and treatments, the horse was returned home, having been chronically lame for about 3 months.

On subsequent lameness examination, the horse was sensitive to hoof tester pressure in the heels, and low palmar digital nerve blocks abolished the lameness. Both hoof angles were now 49 degrees. The original farrier took over the care of the horse's feet and worked at restoring the horse's hoof angles to their original state. The horse became sound with no other treatment.

Hoof Width

Whereas the widest part of the hoof is more difficult to deliberately change, it is easily measured and monitored. Over time, it can provide important information to both veterinarian and farrier. Simply by regularly recording hoof width, narrowing or widening of the foot over time may be noticed before a change in hoof shape or angle is obvious to the eye, particularly if the change in hoof shape is gradual (and thus easily overlooked by someone who sees the horse frequently). Any alteration in hoof width over time is an indication to look closely for the reason and decide whether it indicates a positive or negative change. Hoof tracings can also be an adjunct to measuring the widest part of the foot.

Toe Length

Toe length measurement is a measurement common to most farriers. Like all measurements, it needs to be done the same

Table 23-2 Estimated Healthy Body Weight for Foot Size

Hoof Wall Circumference (inches)	Maximum Body Weight (pounds)
13	1050
13.5	1130
14	1220
14.5	1300

way each time to be valuable. The anteriormost coronet band is palpated for the proximal point and the bearing surface of the hoof (or shoe) is the distal point. Some farriers use a caliper to make this measurement and then apply it to a ruler to determine the number.

Coronary Circumference

The coronary circumference is a simple measurement based on work by Dr. Tracy Turner, who evaluated hoof size (specifically, the circumference of the proximal hoof wall) in relation to body weight as a predictor of foot soundness in performance horses.[18] This study found that 78 lb/in^2 is the maximum body weight to hoof area ratio for a healthy performance horse. If the ratio is greater than 78 lb/in^2, then the horse is heavier than it should be for the size of its feet.

Although hoof size cannot be increased to any great degree, bodyweight can be decreased in horses whose weight to hoof area ratio is greater than 78 lb/in^2. This formula also enables the examiner to estimate the upper limit of "healthy" body weight for a particular horse and make management recommendations accordingly.[2]

The method is simple. The distance around the entire hoof wall just below the coronary band is measured. This measurement is the circumference (C). For the average-sized horse, C is usually in the range of 13 to 14 inches. To calculate the maximum healthy bodyweight for that horse, C is multiplied by itself to get C^2, and then that number is inserted into the following equation:

$$\frac{78 \times C^2}{12.56} = \text{body weight (lb)}$$

For example, if C is 13 inches, the horse should weigh less than 1050 pounds. Table 23-2 provides an easy reference for average-sized horses.

Heel to Toe Measurement, Determining Breakover

Another simple measuring system assumes the breakover should be equidistant from the balance point of the foot to the heel. The center is determined by the following process:

- Place an index finger on the anterior coronet and thumb on the frog 90 degrees from where the foot stands on the ground
- Make a mark
- Find the point of the junction of the frog with heel bulbs
- Measure
- Then reverse ruler and mark the toe

This is roughly where the breakover should be.[19]

This measurement can be used in evaluating foot conformation related to a *breakover score* based on the average linear growth of the hoof of 2.5 mm/week. By subtracting the heel-to–balance point distance from the toe-to–balance point distance and then divide by 2.5, one should arrive at the number of weeks from shoeing. Thus after 6 weeks from shoeing, if the heel portion measured 55 mm, the toe portion measurement would be 70 mm. (Thus 2.5 is divided into 15, which equals 6). This foot might be considered balanced anteriorly to posteriorly for that point in the shoeing cycle. However, if the horse is shod for only 1 week and the heel measurement is 55 and the toe measurement is 70 mm, then dividing 15 by 2.5 means the foot (the breakover) is the equivalent of being 6 weeks from shoeing, not 1, and thus the toe was left too long.

APPLICATION AND ADAPTATION

By using any imaging methods or measurements in a consistent fashion, one can develop a program that yields useful information for making hoof care decisions, accumulating data on a specific problem or population of horses, and even for augmenting prepurchase or lameness examinations. An important component of any preventive program is to periodically evaluate its effectiveness. By examining the data, the program can be tested, challenged, and adapted as needed.

A COMPUTERIZED PREVENTIVE HOOF CARE PROGRAM

David W. Jensen

GOAL OF THE PROGRAM

The primary goal of an effective PHCP is to encourage a meaningful and interactive dialogue among the team of individuals who are primarily responsible for the care and maintenance of the horse's feet: the owner or agent, the farrier, and the veterinarian. The main function of this dialogue is to focus attention on the horse's foot using an accurate, understandable, and repeatable description of both the exterior of the hoof and the skeletal structures within the equine digit. The effective PHCP program provides team members with a common language for describing and discussing the hoof (i.e., universal terminology) and detailed measurements and images that can be used immediately and archived for future reference.

The initial phase of the program is focused on creating a complete description of the digit as it currently exists. The information obtained during evaluation of the hoof is computerized using commercially available software (Metron-PX; Eponatech, Creston, Calif.). This software program has been shown to provide precise, accurate, and repeatable measurements of the equine digit.

The initial evaluation then serves as a framework for communication among the team members; recommendations are agreed upon and then implemented by the farrier. Routine maintenance checks encourage careful attention to detail in maintaining the agreed-upon changes. Finally, a gait

and performance evaluation is made to determine whether the goals that have been met have actually produced the desired effects on the horse's health, comfort, and athletic ability. Although the most common uses of this program may be to develop a database or help eliminate lameness, a more altruistic goal would be to use the program to acquire maximal performance. This goal possibly could be achieved by accurate record keeping of consistent measurements with team member review after important training sessions or competitions.

TEAM MEMBERS

The members of an effective PHCP variably include the veterinarian, the farrier, the horse owner or trainer/agent, and perhaps the rider.

Veterinarian

The veterinarian generally takes the lead role in organizing the hoof care team, gathering accurate data and images, and implementing the action plan and follow-up evaluation. As team leader, the veterinarian's duties are to be both the team coordinator and the evidence-based medical advisor. The veterinarian's observations on gait evaluation, medical knowledge, and ability to provide radiographic images are his or her unique contributions to the team. In addition to knowledge of pharmacology, the veterinarian also provides supplemental medical and surgical treatments, as necessary.

Farrier

The farrier, acting in concert with the veterinarian and the owner, must translate specific two-dimensional data about the hoof into an appropriate trimming and shoeing strategy. In addition to the commonly recognized skills of the farrier, he or she must also bring to the team the invaluable art of molding a three-dimensional dynamic structure—the hoof. A farrier's ability to visualize the foot as a three-dimensional object, while holding in his mind an understanding of the inner anatomy, is essential. He must then have the artistic ability to sculpt and support the desired form from the foot that is presented to him.

Trainer, Rider, Owner

The trainer, rider, or owner (if they are not the same individual) must be able to provide accurate feedback and interpretation of the results. Their evaluation is essential in determining which changes to maintain, which should be modified, and how aggressively the program should be implemented. In addition, their knowledge of the horse's training schedule and competition dates often dictates trimming and shoeing intervals.

IMPLEMENTATION

For effective implementation of a PHCP, annual or perhaps semiannual evaluations are necessary. More frequent evaluations may be indicated when specific problems are being addressed or higher level performance is expected. These evaluations should be timed to work with the horse's competition schedule to maximize the effectiveness of the program.

Two levels of evaluation are offered in this author's practice:

- Basic evaluation, which consists of a single lateral radiograph of each foot
- Complete evaluation, which includes lateral and dorsopalmar radiographic projections and three digital photographs of each foot; the digital photographic study includes lateral, dorsopalmar, and solar surface views.

With both levels, the images (photographs and/or radiographs) are evaluated using a computerized program. The radiographs are placed on a light box and photographed using a digital camera; the images are then imported into the computerized program. The software makes it easy to evaluate the images, as it directs the operator to pick key points and then computes and reports the parameters of the operator's choice. A recent software upgrade now provides for a composite overlay image that merges the lateral radiograph with the corresponding lateral radiograph. The images are accurately sized and key points identified to allow precise alignment of the two images. These images are extremely valuable in owner education. A written report can be printed for use by the PHCP team members.

Ideally, the evaluation is performed before the horse's feet are trimmed and shod; recommendations are made and implemented, and then a post-shoeing evaluation is performed. If the client elects just a single evaluation, then this author's preference is to perform it immediately after the horse is trimmed and shod. The PHCP program this author uses is primarily intended to evaluate the shod athlete. However, the evaluation can be performed on the unshod foot. In fact, the published normal ranges used by the software manufacturer are based on measurements of the unshod foot.

Equipment

Digital Camera

Digital cameras are best for this program, as they provide an instant image that can be quickly imported into the computer software. A camera with the added feature of a lens housing that swivels independently from the viewfinder allows the operator to remain upright, rather than having to lie flat on the ground when framing ground-surface views of the foot. Cameras with a 1150 × 800-pixel resolution are acceptable for this application. A conventional camera could be used, but importing the images into the software is cumbersome, as it requires digitally scanning and perhaps manipulating each photograph.

X-Ray Equipment

No special radiographic equipment is necessary. The x-ray machine, film, and cassettes used routinely in most equine practices are suitable for the radiographic applications. Digital radiography will easily integrate into the software system.

Computer and Printer

The software runs on any IBM-compatible personal computer that uses the Windows 95 operating system or a later version

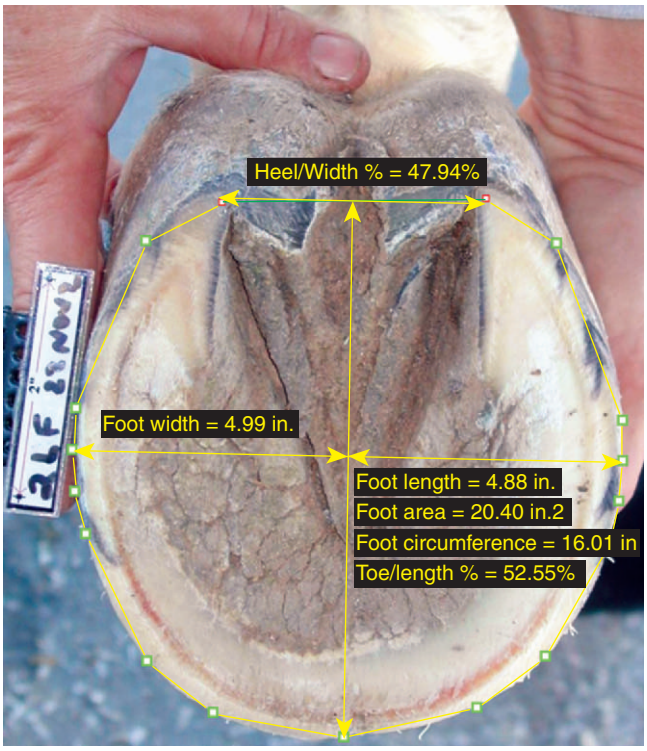

FIGURE 23-9 The erasable marker is always placed laterally (to left of the solar view of this hoof). It provides identifying names and dates, and provides the scale that translates to the radiograph or photograph. This figure also demonstrates some of the measurements that can be acquired and then printed for reporting or record keeping.

of Windows (Microsoft Corporation, Seattle, Wash.). A color printer adds greatly to the quality of the reports and is a relatively inexpensive investment. A modem and a high-speed Internet connection are helpful in some instances, but not necessary to produce the hoof care evaluation and a high-quality, full-color report.

Wooden Blocks and Scaled Markers

To produce consistent radiographs, it is essential to use a pair of wooden blocks to elevate the horse's feet a set distance from the ground. The same wooden blocks are also used for obtaining photographs of the feet. These blocks can be made or purchased but should have small lead "b-b" pellets or lead rods implanted into the surface of each block at regular distances to act as a simple scaling system for the radiograph once it is imported into the software.

Using an erasable, write-on label mounted on the front and sides of the block, which includes a scaled marker, is also helpful. Clip-on scaled markers (Eponatech, Creston, Calif.) provide an accurate scaling system when held in the field of interest in the photograph. This item could also be handmade (Figure 23-9).

Radiographic Techniques

The radiographic technique should be consistent and repeatable. All evaluation systems are most accurate when a consistent film to focal distance (FFD) and object to film distance (OFD) is maintained from one study to the next.

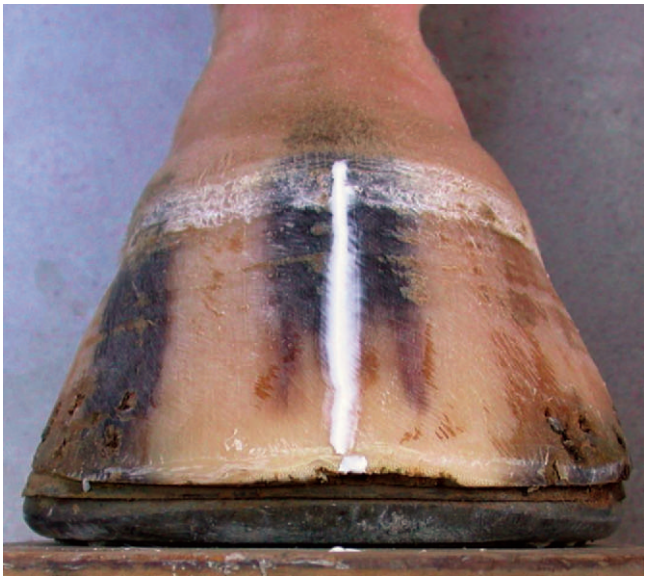

FIGURE 23-10 Barium paste striped down the anterior of the hoof in preparation for a lateral radiograph.

A wooden positioning block is placed under each foot so that symmetry of posture and consistency of radiographic projection are maintained throughout the radiographic study. If the commercially available blocks with built-in lead scale are not used, a radiopaque object of known length can be taped to the cassette.

The horizontal beam is positioned so that it is centered at the ground-bearing surface of the hoof. For the lateral projection, a radiopaque marker is placed on the dorsal hoof capsule to delineate the hoof wall and identify the coronary band (Figure 23-10). For the dorsopalmar projection, both the medial and lateral hoof walls and the coronary band on each side need to be delineated (Figure 23-11).

A single lateral radiograph of each foot is the minimum requirement for an effective PHCP. An additional dorsopalmar view is strongly recommended. If a comprehensive evaluation is desired, three digital photographs are also obtained: a lateral and a dorsopalmar view, each taken at ground level so as to mimic the respective radiographic projection, and a photograph of the solar surface of the foot, taken at a 90-degree angle to the weight-bearing surface.

Program in Action

In this author's solo, mobile equine practice, the implementation of these goals frequently involves working with more than one farrier and may incorporate individuals with other areas of expertise. The following are three examples of how a PHC program could be implemented involving several professionals.

Optimizing Performance and Career Longevity

In the first example, a horse had no detectable hoof or lameness issues. The owner's intent was to monitor the health of this horse's feet to maximize his performance and career longevity as an eventer. She was also very interested in the recent advances in glue-on plastic shoes for performance horses.

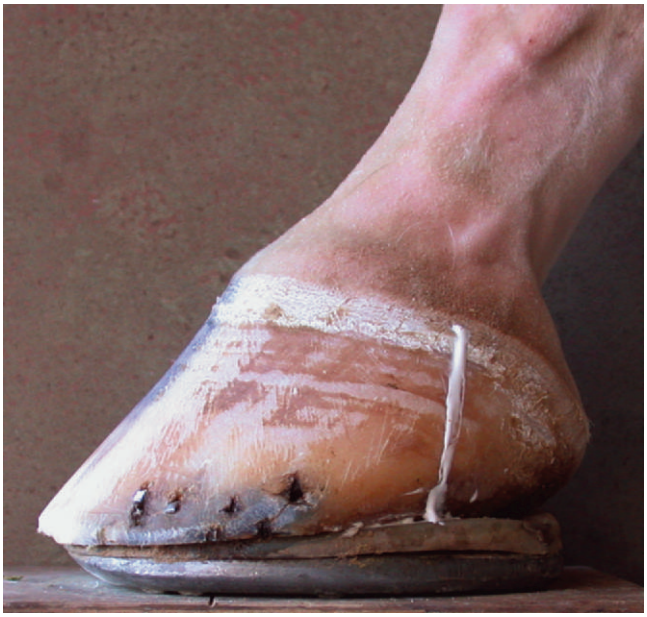

FIGURE 23-11 Barium placed at the widest part of the foot (on both sides) in preparation for the dorsopalmar radiograph. It is critical for the technician to be consistent with the identification of the coronary band in order to make proper and repeatable measurements.

The veterinarian and leader of the team made an appointment with the primary farrier, and a meeting with a consulting farrier familiar with the use and application of glue-on shoes was organized. The group also enlisted the expertise of the manufacturer of the plastic glue-on shoes. A computerized evaluation of the feet was obtained before the meeting. With the written report as the focus, the goals and concerns of the owner were clearly articulated and addressed, which resulted in an action plan that was immediately implemented by the assembled team of experts. The horse was re-evaluated on a semiannual basis.

Support During Recovery

In the second example, a horse recently underwent surgical neurectomy as treatment for chronic, bilateral foot lameness. The owner's goal was to provide the best possible support for the feet, with the eventual hope of producing a rideable horse. In this case, a group meeting involving the owner's primary farrier, the attending veterinary surgeon, and a consulting farrier was arranged. A complete computerized hoof evaluation was performed before the meeting and copies of the report were made available to all parties.

During the group meeting, detailed information and history findings were exchanged among the team members, which provided valuable insights. Evidence of early degenerative joint disease of the distal interphalangeal joint was discovered. This diagnosis was essential to an accurate long-term prognosis. A farriery plan was agreed upon and immediately implemented. Follow-up included subsequent lameness evaluation by the veterinary surgeon and quarterly monitoring of hoof health.

Early Intervention

In the third example, a horse was being leased by an aspiring young horsewoman who was actively competing in the sport of eventing. The rider had noticed intermittent lameness and thought there was progressive contracture of the heels in one foot. A hoof abscess, thought to have been brought on by earlier sole bruising, had been plaguing this mare for several weeks and had caused her withdrawal from a planned event. With the pressure of a heavy competition schedule, this rider was anxious to resolve these problems and questioned the shoeing techniques of her current farrier.

In this situation, the rider, the owner, the original farrier, and a consulting farrier where brought together for a group meeting. A computerized hoof evaluation was performed prior to the meeting. After consultation with the plastic glue-on shoe manufacturer, an action plan was made and then implemented the same day, using new shoeing technology. The mare was sound within 2 days with no other medical treatment. The rider was able to immediately resume training and placed second in a 3-day preliminary competition 2 weeks after shoeing. The rider elected to compete with the plastic glue-on shoes that were used during the dressage phase of the competition. The shoes were replaced with traditional metal shoes during the cross country and jumping phases. The horse continues to perform well with no lameness. Over the following 8 months, the horse competed successfully at an intermediate level and continued to improve with no evidence of foot pain.

KEYS TO SUCCESS

Open-Minded Team Members

When initiating a PHC program, the initial team must be chosen carefully. As with all new concepts, there will be those individuals who will actively embrace change and those who will dismiss change out of hand. It is the innovators who seek out new information who should be recruited initially. This quality is of paramount importance in the farrier. As the program builds, the more reluctant farriers, owners, and riders can then be persuaded to investigate and consider the program.

Time Management

Ideally, the individual responsible for entering the data into the software should be trained under the direction of the examining veterinarian to save veterinary time. With experience, the data-entry process can be performed in 30 minutes or less. A sole proprietor with limited staff can consider teaming up with a veterinary colleague who is already using the software. Internet accessibility allows for the transfer of the images for evaluation at a remote site. An e-mailed report could then be received and printed.

Responsible Use of the Information

The use of the computerized hoof evaluation system as a critique or "report card" of a farrier's work can be both divisive and inaccurate. In this author's opinion, the scoring system available with this software is only helpful in evaluating the horse's inherent conformation and has limited correlation with proper trimming and shoeing techniques. Fortunately, one of the strengths of this software program is

that it allows the user to select which features to use and which to ignore.

The hoof measurements available to the operator are numerous. It is best to choose only those measurements that are most meaningful to the practitioner's needs, rather than indiscriminately including every measurement available from the software. Selectivity is easily accomplished by clicking on the specific criteria that are desired for inclusion in the report.

QUALITY FOOT PROGRAM AT PIN OAK STUD

Clifford P. Barry

This is the story of how one farm improved the quality of its horses' feet through a carefully constructed and diligently applied program of hoof care that begins even before the foal is born.

ORIGINS

I am the general manager of Pin Oak Stud, a Thoroughbred breeding farm in Versailles, Kentucky. The farm consists of more than 900 acres of farmland, about 160 horses of various ages and stages (i.e., mares and foals, yearlings, racehorses, and breeding stallions), and a staff of about 45 people. I arrived here in the spring of 1988 with an extensive background in Thoroughbred breeding and racing and what I thought was a very good understanding of equine hoof care. But, as it turns out, little did I know…

In 1995 I took over as general manager when then-manager Joe Osborne returned home to Ireland. I had worked as Joe's assistant for several years and had learned a lot from him, as Joe Osborne was the person most instrumental in setting up the Quality Foot Program at Pin Oak. When I took the reins, I very much wanted to continue the legacy of being a better caretaker of the feet of these animals that give us so much pleasure.

Pin Oak's Success

The main impetus for change came from the owner of Pin Oak, Josephine Abercrombie. Over the past 50 years of breeding and racing Thoroughbreds, Ms. Abercrombie has guided Pin Oak to become one of the leading Thoroughbred breeding farms in Kentucky. She has bred and raced numerous Grade 1 winners, including champion Laugh and Be Merry; Horse of the Year winners Peaks and Valleys, Changeintheweather, Missed the Storm, Confessional, and See How She Runs; and multiple-graded stakes winners Broken Vow, Hasten to Add (champion), African Dancer, Green Means Go, Look Daggers, and Bedanken, just to name a few.

Pin Oak has also received several major awards, including the 1990 Eclipse Award for Champion Turf Female, 1995 Sovereign Awards for Canadian Horse of the Year, Champion Two-Year-Old Colt, and Champion Grass and Older Horse. A huge triumph for the farm came when the Thoroughbred Owners and Breeders Association recognized Pin Oak as the state and national breeder of the year in 1995, a once-in-a-lifetime achievement in this very competitive business.

Since the beginning, Ms. Abercrombie's objective has always been to breed top racehorses. She strives to maintain a band of quality broodmares, mate them to suitable stallions, and hopefully produce some top contenders. Pin Oak has also been competitive in the stallion market, currently standing Broken Vow (multiple-graded stakes winner), Maria's Mon (champion two-year-old colt, sire of 2001 Kentucky Derby winner Monarchos), Peaks and Valleys (Canadian Horse of the Year and Champion Three-Year-Old Colt), Sky Classic (four-time champion, including an Eclipse Award), and newly retired Changeintheweather (Grade 1 winner, standing his first season in 2004).

Why a Quality Foot Program?

With such a lineup of winners, why would Pin Oak need to implement any sort of program? Well, it is simple: foot-related soundness problems were costing us too much time and money and limiting the potential of too many good horses.

Our sport, once known as the *sport of kings*, has become a serious business for the vast majority of its players. Millions of dollars are invested daily in the Thoroughbred industry, whether it is in breeding, racing, or selling horses. To protect their investment, investors and owners need to have a healthy product to market or compete. Because healthy feet are such a large component of a horse's overall health and performance, a program that produces horses with good-quality feet is a necessity in this highly competitive field. In other words, a Quality Foot Program is the starting gate for achieving your goals as an investor/owner.

Getting straight to the point, our farm owner demanded a change. We had been working on a number of improvements over the years, but Ms. Abercrombie, a very astute horsewoman who is also knowledgeable of the Thoroughbred breeding and racing business, believes that one can always do better. So, we began paying even closer attention to the health and welfare of our horses, both on the farm and at the racetrack. Using common sense, we initiated a Quality Foot Program.

Common Problems on the Farm

Coffin bone fractures in foals were one of the common problems we were experiencing. Affected foals would be lame and would require stall rest for 30 to 60 days, which is less than ideal during this vital time in a young foal's life. Also, while many foals would heal and show no further signs of lameness, some foals remained mildly lame for several months; a few even developed persistent foot problems. Before our program began, the incidence of these fractures was one in four foals. Now it is fewer than one in 20 foals.

We also found that we were frequently having to use hoof extensions for developmental problems in our foals. While hoof extensions can be very helpful, excessive use can deform the feet and set up the foal for problems (e.g., sore heels, quarter cracks, very unbalanced feet) in the future. With our program, the need for these interventions has decreased, as has the length of time we leave extensions on a foal's feet.

Another common problem we were experiencing was broodmares being footsore for several days after being trimmed. Whether or not the farrier was simply trimming too

much in one visit, this common outcome was unacceptable to us and required correction.

Common Problems at the Racetrack

The long toe/low heel foot conformation is commonplace at racetracks; in fact, it seems to be the "in" thing with many trainers and farriers. It was once thought that this foot shape increased a horse's stride length, but that has since been proven incorrect. With this syndrome, the shoe is set a little further forward each time, and before you know it the heels are low and the toe is way out in front. We have experienced various racing injuries related to this conformation, including quarter cracks, bowed tendons, fetlock chips, and sore backs. Not only do these injuries sideline a horse at the peak of his racing career, but some horses never return to their full potential.

At one time, 25% of our horses had diverse feet problems. Looking at the big picture, one can see where Ms. Abercrombie was coming from when she demanded a change. The expense of treatment, coupled with lower-than-expected earnings, prompted careful examination of what we were doing and where change was needed. Our Thoroughbred operation could not continue in this manner and expect to compete with the best in our business.

CHANGE AND HOW IT STARTED

Change is never easy; at times, we are very hesitant to make the leap. But change was a necessity for Pin Oak, so we set out to establish a Quality Foot Program, under the direction of Ms. Abercrombie and the guidance of Ric Redden, who was brought in as a consultant to our management team to help us move in the right direction.

Self-Education

One of the first things we did was create a comprehensive education program for our assistant managers and staff. This program included (1) a series of lectures that addressed our particular foot problems; (2) hiring a qualified farrier who could also continue to teach our staff; (3) weekly discussion groups; (4) sharing up-to-date literature on foot problems and proper treatment with our barn staff; and (5) allowing our staff to accompany the farrier on rounds, to attend foot-related seminars, and to visit a local horseshoeing school.

An education is something that we get everyday, especially in the horse business. Whether farm managers or caretakers, we have to develop an eye for what we want. At Pin Oak, we had regular lectures so that our staff could learn to identify foot problems, learn their causes, and plan rehabilitation. We wanted them to know how to prevent an injury by providing proper foot care on a daily basis. To assist the learning process, we included visual materials from slide shows to handouts, and being able to have lectures during a luncheon provided by the farm was very popular with the staff.

In seeking to educate ourselves, we found that there is no shortage of books on this topic. The ones we found most helpful, and which we highly recommend, are *The Horse's Foot* by Christoper C. Pollitt, *The Equine Foot* by Fran Jurga, *Conformation and Anatomy* by Equine Research, and *Equine Lameness* by Equine Research.

Our assistant managers directed this new approach on foot care toward our barn staff members, as they are the ones responsible for the day-to-day care of the horses. With proper instruction, the barn staff members were able to identify and report any problems as soon as they arose and also assist with specific treatment. Although this education process was easier said than done, perseverance has paid off.

Open Communication

We visited with several farriers and discussed certain problems in an effort to guide our team forward. We also visited several trainers and conveyed our thoughts on hoof care and preventing hoof problems. We observed the horses under their care and discussed shoeing techniques and what we were trying to achieve with our Quality Foot Program. It was essential that we incorporate our trainers into the program, so we conveyed the message that foot care within our racing program was being given a new priority and that the initial work was to start right here at the farm. These horses were to leave the farm for the trainer's barn with their feet in a fit condition to sustain the rigors of the racetrack.

Thereafter, we paid regular visits to the trainers at the various racetracks where our horses trained and raced to ensure that our foot program continued while the horses were away from the farm. This part was not so simple. It took a bit of time to make headway with some trainers, given that a trainer, like any athletic coach, is inclined to use his or her own approach to get the best performance from an individual. Here again, we found open communication to be the best way to start. We expressed our concerns and addressed the situation whenever a horse at the track became lame or was not performing to its potential. By asking questions and making suggestions, we found that the good trainers listened and took our thoughts into consideration in an effort to reach our mutual goal—a successful racehorse.

The Right Farrier

To tie all the loose ends together, we had to hire a farrier. Choosing a farrier who would fit our needs took some time. The key was to find someone who was willing to work with our program and with our veterinarian. Teamwork is essential if one is to maintain a successful healthy-foot program; all egos must be put aside and everyone must work together.

CHALLENGES

Perhaps the biggest challenge we faced in implementing our program was the need to stay focused on our goal of producing quality feet that could stand up to the demands we placed on them at the farm and at the racetrack. Part of meeting that goal was to examine the reasons when things did not go according to plan. Tempting though it is to point the finger at someone or something else, it is important to accept responsibility for our shortcomings and also to acknowledge that certain things are beyond our control. For example, the farm manager has to deal with such diverse factors as the weather, farm terrain, soil types, and turnout schedules. Trainers not only have to factor in the weather but

Horse	Blacksmith	Date	Barn	2-Week Trim	X-Rays
	Carpenter	03/12/03	Yearling No. 1	✓	n/a
	Comments: Small crack in left front; clubby right front. Trimmed all fours; dressed back crack in left front. May need shoes next time.				

	Angles	LF	48°	RF	60°	LH	47°	RH	47°

FIGURE 23-12 Sample farrier's report for a yearling.

also how the current weather conditions may affect the training surface.

Another factor that is somewhat beyond our control is genetics. Certain bloodlines are well known within our business for producing poor-quality feet. So, closer attention should be paid when planning broodmare matings. When you think about it, it is amazing that the equine hoof can carry a 1200-lb animal galloping at speeds of up to 35 miles per hour. Imagine the forces that the hoof must withstand each and every stride. It should be obvious that if there is a flaw in the hoof, it will become apparent very quickly under such intense loads. It is no wonder these flight animals do not want to run when their feet hurt. It does not matter where we live in the world, we all encounter the same types of problems, just to different degrees. Our job as caretakers is to minimize those problems as much as we possibly can.

In addition to a commitment to education, the goals required of our management team also included setting and maintaining a high standard and working hard to meet it. Again, all that is easier said than done. Of utmost importance was to establish an excellent line of communication with our farrier, as a good relationship between caretaker and farrier makes it a lot easier to discuss what needs to be done. It also took the employment of a good staff of people, as they would be the ones who would be working with the horses on a daily basis. Our motto became, "Take care of the little problems and the big ones will take care of themselves."

FOOT CARE FROM DAY ONE

Our program actually begins before the foal is born, with the careful selection of breeding stock and meticulous care of our pregnant broodmares. We start trimming foals at about 2 weeks of age, mostly just touching up around the edges with a rasp. Thereafter, our foals are trimmed once a month, unless a particular foal requires further attention, in which case we recheck that foal every 2 weeks. (The rate of hoof growth in a foal is approximately 15 mm per month, much faster than that of an older horse; hence the short interval between trimming.) The trimming schedule for any foal depends on the foal's conformation, particularly its axial alignment.

When a foal is born, its foot is very much underdeveloped. So for the first couple of weeks, we simply teach the foal to have its feet picked out every day, which allows us to check each foot and to teach the foal to hold up each foot so that the farrier's job is made easier. Also during this early stage, foals are taught to walk in a straight line on a flat surface so that we can watch their walk for any conformational problems. Most importantly, we keep detailed written records with both farrier and management comments. We sometimes find it valuable to keep photos on file for certain foals. Figure 23-12 shows a sample farrier's report for a yearling at Pin Oak.

BROODMARES

Our mares are brought up to their barns daily to have their feet washed and picked out thoroughly. The mares are trimmed monthly, with 2-week checks as needed. A full report is detailed on each mare during a farrier's visit. Our guiding philosophy is that mares with healthy feet are more comfortable and feel more at ease and less stressed than mares with unhealthy feet. We believe that having healthy feet helps the mare to conceive and hold her pregnancy late in gestation, when she may weigh in excess of 1200 lb. In our experience, mares with chronic foot problems seem prone to having barren years.

We have also tried to minimize the number of shoes on our mares; our preference is no shoes. The weaning process from shoes to bare feet can take some time and care, but in the long run, we have achieved much better and stronger feet for all our hard work. Our shoe-weaning process generally takes 4 to 6 weeks. When first turning out a mare without shoes, we may use hoof packs and paint her feet with a hoof hardener each day. We may also put the mare in a small paddock and on a 2-week trim schedule, just to keep on top of any chips or small cracks.

One thing we feel is very important is to take a set of lateral radiographs on mares that have just retired from racing and have returned home to the farm. If anything unusual or in need of closer attention appears, we take lateral radiographs on a more regular basis, depending on the particular concern at the time. If nothing else, these films are a good point of reference should problems arise with that mare in the future.

RACEHORSES AND BREEDING STALLIONS

Our yearlings, racehorses, and stallions all have a similar routine: they are in a barn daily for up to 8 hours at a time, depending on the season. This routine gives their feet a chance to rest from the hard ground (whether it be the height of a dry summer or the frozen ground of winter). It also gives us a chance to take care of their feet properly.

With our yearlings, we try to keep our corrections to a minimum. As part of our program, most of our young horses, from yearlings through to the early stage of the 2-year-old year, train and gallop barefoot. We believe that this approach gives them a much stronger foot when they go into training in the late spring of their 2-year-old year. The first time that they may get shod is just before they leave for the training center.

Our racehorses are stabled around a number of different racetracks, with several different trainers, so we need to have frequent communication with our trainers regarding the horses' training schedules and their foot care. We also ask for detailed farrier reports from the trainers on each horse, on a regular basis.

SUMMARY

The Quality Foot Program at Pin Oak did not happen overnight. It has taken several years to evolve to where we are now. We were persistent in making it better, but a lot of things had to fall into place as well, including a good nutritional program, good pasture management, and a good support staff getting the necessary work done. All these little extras took time. Without the hard work and dedication of our people, our program probably would not have gotten too far. We have all seen how far we have come with our foot program and, truthfully speaking, we could never go back to where we were. That old saying "no hoof, no horse" has never meant so much to us at Pin Oak.

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IMPORTANCE OF A TEAM APPROACH TO EQUINE PODIATRY

RICHARD A. MANSMANN and KURT E. vom ORDE

Over the past decade, much lip service has been paid to the veterinarian–farrier relationship, with mutual respect touted on both sides as the guiding tenet. However, if one were to conduct an informal survey of either group, the prevailing sentiment would likely be that veterinarian–farrier relations still have a way to go. And even when a veterinarian and a farrier enjoy a very good working relationship, these professionals are not the only individuals responsible for restoring or preserving hoof health. It is the individual, or individuals, responsible for the day-to-day management of the horse who ultimately have the greatest impact on hoof health. Without the active cooperation of the owner or rider, trainer, and farm or barn manager (who may or may not be the same individual), the very best efforts of the veterinarian and the farrier may be thwarted.

The best results generally are obtained with a team approach to equine hoof care, incorporating all of the individuals responsible in some way for the horse's care and management: veterinarian, farrier, owner or rider, and trainer or manager—the VFOT team. This team approach is particularly important, and in many cases essential, for a good outcome in horses with existing hoof disorders, those with a particular propensity for hoof disease, and those performing athletic activities that are physically demanding or that require great precision.

The majority of healthy, well-conformed, well-conditioned horses do not need the combined efforts of a veterinarian and a farrier to maintain optimal hoof health and function. Most of these horses simply need regular attention by a knowledgeable, skilled farrier to meet the demands of their work. However, if the work demands for that horse increase or if the horse is beginning with or develops a problem that involves the feet in some way, then it often becomes important for the veterinarian, the farrier, the owner, and the trainer to work together to optimize hoof structure and function. Obviously, the sooner the team approach is instituted, the better it is for the horse and often less expensive for the owner. The team approach can also be important in preventing problems, if in no other way than by identifying the potential for a particular problem to develop in an individual horse or noticing the early signs of a problem, and taking appropriate steps to prevent or resolve the problem before it causes lasting damage.

HOW HAS THE HUMAN MULTIDISCIPLINARY HEALTH TEAM EVOLVED?

Human medical specialties developed in the early 1900s, and soon these specializing physicians needed lines of communication between themselves and the primary care physicians. In the 1930s, nurses began advocating for a team approach to medical care in hospital settings, primarily to organize the increasing numbers of health professionals, especially in areas of home care, mental health, and rehabilitation. In the 1960s, the federal government gave impetus to the "team concept," supporting care to poor and underserved geographic areas. In the 1970s and 1980s, training programs in an interdisciplinary team approach to care were started. Grants were given to teach collaboration and teamwork to medical and health professionals. In the 1990s, team approaches began to include educating patients on healthy lifestyles and disease prevention.[1]

WHAT CONSTITUTES A TEAM?

The concept of a team is not so much about the individual members but about the process: individuals gathered together and working toward a common end. Thus a team constitutes these elements:

- A group of two or more people
- A shared sense of purpose among the members
- Interactions between members that make them able to accomplish more than each could accomplish working individually toward the shared purpose

Each of these factors is necessary for an interdisciplinary team to function effectively.[2]

BENEFITS OF THE TEAM APPROACH

There are numerous benefits of a team approach to equine foot problems and hoof health. First and foremost, an effective VFOT team most benefits the horse, which, in fact, is the shared purpose of this particular team. Benefits can be long-ranging, from enhancing the resolution of existing problems or identifying and addressing new problems early enough for treatment to be successful, to preventing certain problems altogether and maximizing comfort, locomotive efficiency, performance, and thus career longevity.

An effective VFOT team also benefits the individual members. The owner and trainer are benefited because this approach maximizes the horse's performance and career longevity, minimizes down time, and limits the overall cost of hoof problems (e.g., professional fees, medications, lost training days or missed competitions, permanent loss of use). The trial-and-error approach that often is used when one individual works in isolation can prove to be very costly in the long run. As the old saying goes, "Two heads are better than one"; the

best approach may be to activate the VFOT team early, even if it seems to be overkill at the outset.

The VFOT team approach also provides unique educational opportunities for each of the members. In addition, it can allow the veterinarian and the farrier to provide a higher standard of patient care than either professional can manage alone, and it can provide a mutually satisfying working relationship and added income.

ROLES OF THE INDIVIDUAL TEAM MEMBERS

> *It is the veterinarian's job to understand the medical and biological functions of the foot. It is the farrier's job to manage the foot mechanically. It is the owner's job to know when to ask for help. No one person has all the answers; no doubt all three have questions for the others. Only by working together can the three-member team help the horse.*[3]

The roles and contributions of the individual team members vary somewhat with the situation. For example, in horses with hoof disease, the veterinarian assumes the role of team leader, whereas in horses with healthy feet, the farrier is primarily responsible for making and executing hoof care decisions, and the veterinarian may be only peripherally involved. The roles of each team member in these two general situations—routine hoof care (i.e., horses with healthy feet) and hoof problems—are elaborated further in the following sections.

Veterinarian

Routine Hoof Care

The veterinarian has two basic roles in horses with healthy feet. The first is as an educator, answering questions and filling in gaps in the knowledge base of the other team members, particularly the individuals responsible for the day-to-day care of the horse. The second, and perhaps more important, role of the veterinarian in this situation is to take a proactive approach to preventive hoof care and be the medical leader in problem cases.

Various models for preventive hoof care programs have been devised. The underlying theme of all these programs is identification of potential problems before they cause functional limitation or structural damage, or, at the very least, early identification and prompt treatment of existing or developing problems. Preventive programs and their values have been discussed in detail in a previous chapter.

Hoof Problems

In horses with existing hoof problems, the veterinarian generally assumes the role of team leader. In most states in the United States, the state Veterinary Practice Act dictates that the veterinarian is the diagnostician, as making a medical diagnosis is considered an act of veterinary medicine. The observations of the farrier can be very important in reaching a diagnosis, but by law the farrier is not permitted to make a medical diagnosis. The requirements of the Veterinary Practice Act also mean that the veterinarian is the primary record keeper for the case, as up-to-date medical records must be kept for each patient. Farriers at this time are under no legal obligation to keep detailed records, although it is wise for them to do so.

The veterinarian also is the team member who formulates and implements the therapeutic plan via Practice Plan requirement, although, again, the farrier's input often is very important in both the planning and the implementation phases of treatment. The veterinarian, too, is responsible for monitoring the response to therapy (and keeping appropriate records), and adjusting the treatment plan accordingly.

Other Responsibilities

Another important responsibility of the veterinarian as team leader is to effectively communicate with the other team members and foster an atmosphere of mutual respect, open communication, and shared purpose. In no situation is this more important than when interacting with the farrier member of the team.[4] Veterinarian–farrier communications can be divided into several categories, from ideal to unsatisfactory:

- Ideal—Both professionals meet in person and together examine the horse or the radiographs (or other diagnostic information), formulate a therapeutic plan, and, if possible, implement the plan (e.g., the veterinarian is present when the farrier trims or shoes the horse).
- Adequate in most cases—When the veterinarian and farrier cannot get together because of time or geographical constraints, they talk by phone at least once to discuss their findings and formulate a plan, and they continue to communicate via phone, e-mail, or mail, sharing copies of photographs, radiographs, and written reports as necessary.
- Adequate in some cases—The veterinarian leaves or sends a written prescription for the farrier to implement as he or she deems appropriate, with an invitation to call for clarification or discussion if necessary.
- Unsatisfactory—The veterinarian leaves written or verbal specific instructions for the farrier with the owner or trainer, disregarding the farrier's opinion or experience with the horse.

No matter how experienced the veterinarian is with equine podiatry, it is important to treat the farrier with respect. The veterinarian must make it clear, in word or action, that he or she understands the farrier's role in contributing to and implementing the shoeing strategy, rather than treating the farrier merely as a technician or a "carpenter." For example, instead of directing the farrier to apply an egg-bar shoe, a more appropriate approach would be for the veterinarian to ask the farrier, "What do you think about using an egg-bar shoe in this case?"

Unless there are clear indications that the farrier is not committed to cooperation or is not sufficiently experienced, it generally is wise for the veterinarian to err on the side of deference and allow the farrier to make shoeing decisions based on his or her experience with similar problems or with that particular horse. Provided the specific goals of therapy are understood by all team members and there is clear and open communication between veterinarian and farrier, this approach usually yields the most satisfactory results.

Continuing education is also an important responsibility of the veterinarian. A certain amount of continuing

education is required annually by most state veterinary boards, although there is no requirement for continuing education to include equine podiatry. Fortunately, numerous continuing education opportunities are now available for veterinarians wishing to get or stay up-to-date, follow specific interests, and develop new skills in the field of equine podiatry.

From time to time, the veterinarian may also find himself or herself cast in the role of mentor to a young farrier or counselor to a distraught client. One's personal philosophy, interest, and experience should be the guide when placed by others in any of these roles.

Farrier

Routine Hoof Care

In most cases, the farrier can be considered the team leader when it comes to making and executing hoof care decisions in horses with healthy feet. By virtue of the frequency with which the farrier sees the horse and his or her familiarity with the facility, the farrier is also in an ideal position to assume a primary role in preventive hoof care. Simply by raising a red flag concerning the propensity for a particular problem to develop (e.g., identifying an overweight, inactive horse as a laminitis risk), a potentially dire situation may be avoided. Although the farrier must be careful about making statements that could be construed as a diagnosis, it is appropriate for the farrier to point out to a client the potential for certain medical conditions to develop. Thus the farrier may be considered the front-line defense against many preventable conditions.

Hoof Problems

The farrier can be an important contributor to both the diagnostic process and the treatment plan by providing observations regarding hoof growth, hoof and shoe wear, structural and functional abnormalities, recent changes, shoeing history, and so forth. This fact alone is a good reason for farriers to keep detailed written records of their observations and activities at each shoeing.

A knowledgeable and experienced farrier can be considered the deputy team leader when it comes to the therapeutic plan. Not only can the farrier provide valuable input in the planning phase, he or she is the primary means by which the chosen trimming and shoeing strategy is implemented. The farrier's observations may also be a vital component of assessing the response to therapy and determining whether to stay the course or make changes to the plan.

Other Responsibilities

As mentioned in the previous section on the veterinarian's role, by law the farrier is not permitted to make a medical diagnosis nor prescribe treatment for an animal. In effect, the various state Veterinary Practice Acts dictate when a farrier should involve a veterinarian in a particular case. Whenever a farrier feels he or she is being asked to make a medical diagnosis or recommend treatment for a particular animal, it is wise to advise the client to consult a veterinarian, even if the farrier has some definite ideas on what is wrong with the horse and what should be done about it. The veterinarian may defer to the farrier on how to manage the case, but the farrier will have fulfilled his or her obligations under the law as well moral obligations to the client and the patient by involving the veterinarian in the diagnostic and therapeutic process.

Continuing education should also be considered an important responsibility of the farrier, even though it is not legally required. There are many and diverse opportunities for farriers to expand their knowledge base and skill level in equine hoof care, including some unique educational experiences in which the veterinarian and the farrier learn alongside each another.

Owner/Manager

Routine Hoof Care

The owner's or manager's primary role is to handle daily hoof care (inspect and clean the horse's feet every day), ensure an optimal environment for hoof health (not too wet, not too dry; remove urine and manure daily), and carefully follow the farrier's instructions for hoof care and shoeing interval. In addition, it is useful for the daily caretaker (whether owner or manager) to keep good records regarding health events (e.g., preventive care, illnesses and injuries), diet, training and competition schedule, and any other events that can affect hoof health. It is also the caretaker's responsibility to contact the veterinarian, and possibly the farrier simultaneously, immediately whenever a problem with the horse's feet is first noticed.

Hoof Problems

Owners and managers should recognize that if their horse is in pain (any lameness or reluctance to move), and even if they suspect a foot problem, their state's Practice Act law prohibits a farrier from making a diagnosis or administering drugs. It is not fair for an owner or manager to put any farrier in a compromising position. Too frequently, what an owner or farrier thought was a primary foot problem was actually an early case of pleuropneumonia, intramuscular injection reaction, or some more important primary problem that needed addressing along with the secondary early laminitis. Delays in these cases can cause very serious financial and health consequences, including death. If the owner has any doubts, having both professionals present initially may well be the best and quickest solution to the problem.

When problems arise, it is the owner's or manager's responsibility to provide thorough and accurate information to the veterinarian (hence the importance of being observant and keeping good records). This role is just as important in evaluating the response to therapy as it is in establishing a diagnosis. It is also the caretaker's responsibility to carry out the veterinarian's and the farrier's instructions for daily care and follow-up visits. Thus, as mentioned earlier, the caretaker's role may ultimately be the most pivotal in the success or failure of the treatment plan.

Other Responsibilities

The owner or manager should get involved in the horse's hoof care plan, whether routine trimming and shoeing or the management of a problem, by listening attentively, asking questions, and reading any materials recommended by other team members. When conducting one's own research, the caretaker would be well advised to question the source of the

information and, if possible, discuss it with the rest of the team for accuracy and relevance.

Trainer

Routine Hoof Care

Unless the trainer is also the manager (i.e., the daily caretaker), his or her primary role in routine hoof care is to reinforce the farrier's recommendations. The trainer may also have some input into the type of shoe used, based on the horse's specific use or training level. In this regard, the farrier and the trainer work together, with the owner's approval. The veterinarian may also be involved in these types of decisions, depending on his or her experience with the particular horse or the sport or activity in which the horse is engaged.

Professional trainers serve their clients, the horses they train, and ultimately themselves by making hoof health a priority. "No hoof, no horse" comes home to roost for the trainer when the horse is sidelined by a foot problem. The trainer usually sees the horse more frequently than the farrier, so he or she is in an even better position than the farrier to be proactive regarding preventive hoof care. Although a farrier may be more attuned to noticing the potential for a particular problem to develop in an individual horse, the trainer may be the first to notice the development of a foot problem. Thus it may be the trainer who first alerts the owner or the farrier to a foot problem and who activates the VFOT team.

Hoof Problems

When dealing with an existing or developing foot problem, the trainer's responsibilities are to provide accurate information, offer suggestions or highlight potential problems with the execution of the proposed plan (based on his or her particular knowledge of the horse and its management), encourage the owner or manager to carry out the veterinarian's and the farrier's instructions, and provide feedback on response to therapy.

Other Responsibilities

The trainer is expected to be more knowledgeable than the owner or rider, so trainers should consider it a professional responsibility to increase their awareness and knowledge about the horse's feet in health and disease states. Educational avenues for trainers include veterinary and farrier discussions, reputable books and articles by knowledgeable authors, and seminars and trade shows.

EXPERIENCE OF THE TEAM MEMBERS

The level of experience of the individual members is perhaps the single most limiting factor in the success of the VFOT team approach. The more each team member understands about the structure and function of the horse's foot (and other aspects of horse health and performance), the sooner it is that problems are identified, the more effective the team's interactions are, and the better the outcome is.

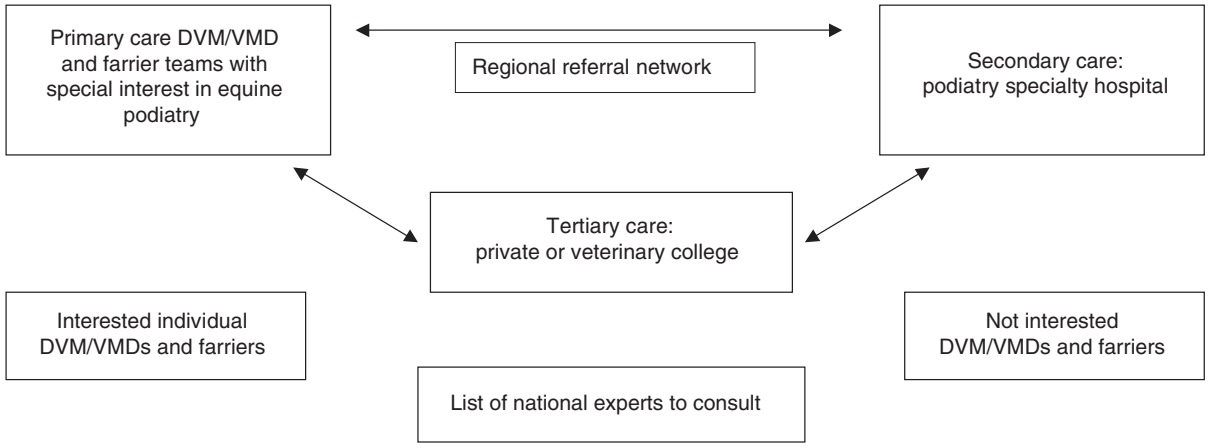
The following quotation comes from a very successful practicing farrier who is a graduate veterinarian. (Many equine podiatrists are practicing veterinarians who were farriers.)

> *There is a primary difference between most vets and farriers—the way their brains work. Most farriers I know are very creative and obviously strong on the right side of their brains. Most vets are more analytical and strong on the left side of their brains. To further add to this fundamental difference, we have significant differences in experience and training. A vet tends to see a horse one to four times a year, and a farrier tends to see the same horse eight to 12 times a year. A vet gets extensive schooling and is taught a problem solving method, while a farrier must search out a learning opportunity and rely on gut instinct to develop problem solving skills.*
>
> *(T. Meister, personal communication, 2005).*

The experience level of the veterinarian can range from that of a new graduate with limited knowledge of the horse's foot, to a veterinarian who specializes in treating foot problems (an equine podiatrist, several of whom also are experienced farriers). Most veterinarians have 4 years of college schooling and then 4 years of veterinary college. These 38 colleges are related to land grant or private large universities and are accredited by the American Veterinary Medical Association. Successful completion of the 4-year course work is required for the veterinary graduate to sit for a national licensure examination. After successful passage of that examination, the veterinarian then is qualified to take the licensure examination for each state in which he or she wishes to practice. Most states require a fixed number of continuing education credits per year for the veterinarian to maintain a license within that state. These continuing education credits can be in the area of equine podiatry. But during the 4 years of veterinary college, there is very limited direct teaching about the equine foot. Currently there may be opportunities for veterinary students to work with on-staff farriers in a few colleges of veterinary medicine or organized elective courses that have an equine podiatry emphasis that might include working with area farriers. Those veterinarians interested in equine podiatry may have gained hands on experience on their own, gone to farrier school while in veterinary college, or been farriers before going to veterinary college.

The experience level of the farrier can range from that of an individual who attended a brief course on horseshoeing, to a farrier who specializes in hoof problems and who works closely with a veterinary facility at which the primary focus is equine sports medicine or equine podiatry. In 2005 there were about 65 farrier schools in North America; 11 of these are associated with a university or community college. The rest are proprietary private schools, including 19 that are run by one person. These educational opportunities range from 1 week to 6 months and there is no oversight as to quality of education. Currently there is no licensure for American farriers, but there are advanced certification levels in various associations after successful completion of testing by the farrier. Recently there has been an attempt by the American Farrier's Association to begin studying farrier education and potential licensure.[5]

The experience and education level of the owner, rider, manager, or trainer generally is dictated by the number of years the individual has been actively involved in the horse industry, the degree of exposure to high-quality veterinary



Mission: This regional network allows for the total care of the horse's feet: treatment, showing, advanced critical care, and imaging.

Goal:

- To develop a plan that gives interested owners, veterinarians, and farriers options to explore
- To reduce the hopelessness to owner, veterinarian, or farrier when another veterinarian and/or farrier says, "Since we see no solution, there is no hope for you and your horse"
- To increase awareness to veterinarians, farriers, and owners that experienced second opinions and referrals are available
- To increase awareness to some regions (or parts of regions) in the United States that other regions have these systems in place
- Set up retrospective and prospective joint studies

Marketing:

- Websites of various teams
- Horse magazines

FIGURE 24-1 Regional equine podiatry referral system.

and farriery care during that time, openness and attention to continuing education, and the level of performance required of the horse. In general, the more experienced the horseperson is and the greater the performance demands on the horse are, the more attuned the individual is to the importance of hoof health and good shoeing and the value of the team approach to restoring or maintaining optimal health and performance.

To a large extent, the VFOT team is only as strong as its weakest link. When there is great disparity in knowledge and experience among the team members, there is the potential for ineffective communication, misunderstandings, frustration, and possibly even treatment failure that would otherwise have been prevented. In this situation, careful communication and close monitoring by the more experienced team members are critical for successful management of the horse's problem.

Effective communication, mutual respect, and a clear understanding of each member's role also help to avoid ego-driven conflicts and thus maximize the team's effectiveness. When conflicts do arise, a quiet reminder of the shared purpose—the benefit of the horse—usually is enough to get the team back on track.

Referral

From time to time, the care required by the horse may be beyond the scope of the particular veterinarian or farrier. In such a situation, it is appropriate, and perhaps even an ethical obligation, for the veterinarian or farrier to refer the case to an individual who is more experienced in managing those types of cases. That does not mean, however, that the VFOT team is disbanded; in fact, team interaction and communication becomes even more important, as another member has temporarily been added. Regional referral systems are loosely in place at this time but could take on a more formalized structure (Figure 24-1). For the good of the horse, it is important for the referring team members to put ego aside and approach the referral process as a valuable educational opportunity, in addition to an important step in restoring hoof health and function. Sooner or later, the case will be returned to the core VFOT team for on-going care. Thus frequent communication among team members, including the specialist to whom the horse was referred, is important throughout and after the referral process.

ETHICAL CONSIDERATIONS

The professional and personal ethics of each team member must be acknowledged and respected for the VFOT team to work effectively. Ethical considerations include the welfare of the horse (all team members), professional codes of conduct (spelled out for veterinarians, left up to the individual for other team members), confidentiality of client/patient information (a legal and ethical requirement for veterinarians, a good idea for farriers and trainers), and compliance with competition rules regarding medication and shoeing.

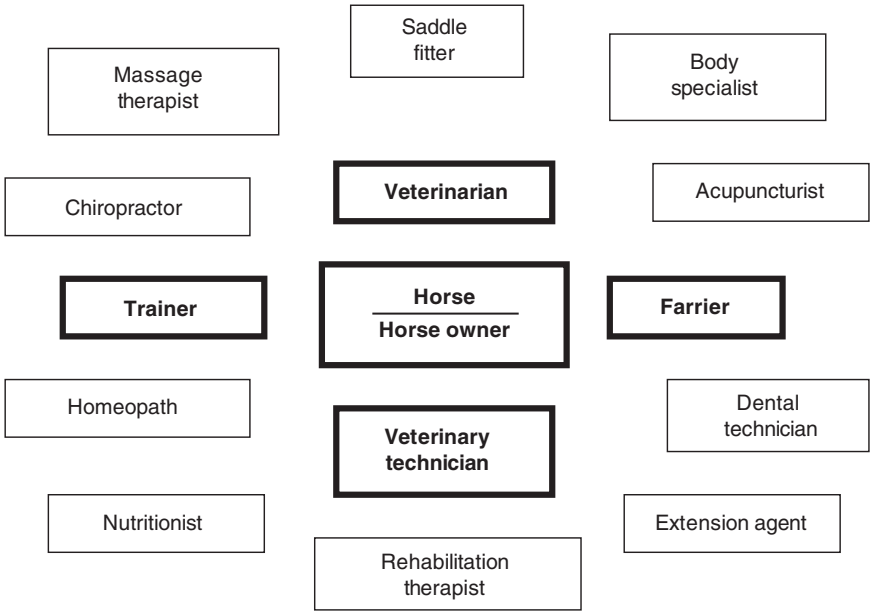


FIGURE 24-2 As the equine health care delivery system is developed, these potential members surrounding the core caregivers *(boldface)* could all play significant roles in a horse's health management. Hopefully this entire system would work in harmony with good patient-information flow, such as in human medicine, for the ultimate benefit of the horse. Ideally each of these caregiver groups would gain their own education system, professional societies, and licensure systems so each member of the care giving team, especially the owner, will know exactly what the specific paraprofessional can contribute to the horse's health.

THE FUTURE

As combined understanding of equine podiatry grows, and as more horse owners, riders, managers, and trainers see the value of the team approach, the VFOT team will become more commonplace and perhaps even the standard of care in some situations (e.g., laminitis, digital fractures, and other serious foot problems). For certain problems, the core team of veterinarian, farrier, owner, and trainer may be expanded to include an equine rehabilitation therapist, equine nutritionist, or other equine health care specialist (e.g., veterinary acupuncturist, veterinary chiropractor, veterinary homeopath). In such situations, the principles of effective teamwork outlined in this chapter become even more important, but the core primary caregivers should remain in place and be apprised of all additional health care given the horse (Figure 24-2).

Equine Podiatry Practices

An exciting trend in equine practice is the development of veterinary facilities dedicated primarily or exclusively to equine podiatry. In addition to numerous private practices that have taken this step, veterinary schools now examining how they can better include basic and advanced farriery services for both in-patient and out-patient care. These schools are not only providing a higher standard of therapeutic and preventive care for their patients; the faculty and staff are also teaching the next generation of veterinarians the value of working closely with farriers and respect for these essential members of the equine community.

Evidence-Based Approach

A trend that has been much slower to catch on but that is no less important is the evidence-based approach. In the medical context, an evidence-based approach involves critically evaluating all available data to determine which diagnostic or therapeutic approach is most appropriate in a particular individual, based on available proof of its value or efficacy. The core of an evidence-based approach to any problem is good data collection. If the art and science of equine podiatry is to progress beyond a handful of research studies and a tradition of trial-and-error, strongly held opinions, and blindly followed beliefs, equine practitioners must get into the habit of recording their observations and then objectively analyzing what they have collected.

More advanced methods of measurement and monitoring the horse's foot are used by equine podiatrists. For example, multiple measurements may be made from a single lateral radiograph of the horse's foot.[6-8] A computer program (Metron-PX; Eponatech, Creston, Calif) has recently become available for developing standardized measurements from photographs or radiographs of the foot.[9] This software makes systematic data collection, storage, and retrieval for analysis easy.

National Awareness Campaigns

In the future, national awareness campaigns aimed at equine hoof care could be embraced by equine and agricultural media outlets, product manufacturers, and cooperative extension services to help make prevention a more common thought for horse owners. For example, April 2005 was designated National Laminitis Prevention Month by *Equus* magazine,[10] with the goal to develop an increase of owner awareness of this devastating but largely preventable disease before the onset of spring.

Another nationwide campaign that would have a great impact on equine health, including hoof health, is one that teaches horse owners the body condition scoring system for evaluating their horses. Simply by alerting owners to the health risks of allowing their horses to become or remain overweight, various medical problems, including laminitis, could be avoided. Other national, regional, and breed- or activity-specific awareness campaigns may also be of value in preventing equine hoof disorders.

In conclusion, the whole purpose of the team approach to equine podiatry, and to developments at every level from owner education to the research laboratory, are aimed at restoring and preserving the health and welfare of the horse. Provided all participants keep this shared purpose in mind, the future of equine podiatry cannot be anything but bright.

EVIDENCE-BASED MEDICINE

Malcolm C. Roberts

Evidence-based medicine (EBM) can advance the practice of equine podiatry: objective clinical evidence is needed. Evidence-based medicine can be defined as the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients.[11,12]

The principles of this rapidly developing discipline in human medicine have been adopted by an increasing number of clinical teachers in colleges of veterinary medicine and by many clinicians in practice.[12,13] The intent is to enhance and elevate clinical decision making above that limited to the standard of opinion (often dogma) that relies on clinical impression and clinical experience.

The process of EBM is as follows:

- Define the question
- Find the evidence
- Critically appraise the evidence for validity (closeness to the truth) and usefulness (clinical applicability)
- Integrate the appraisal with clinical experience for the case in question
- Evaluate the decision in the light of the patient's response

Background knowledge is familiar to equine practitioners. Evidence-based medicine promotes use of foreground knowledge to augment the background information. This concept is not so familiar, although it is used throughout the practitioner's daily activities. It relates to seeking an answer to a question posed for a specific issue that could involve therapy, diagnosis, causation, or prognosis.

A well-formulated question has several key elements:

- What is the problem?
- What is the intervention?
- What are the alternatives?
- What are the outcomes?

This concept is pivotal to understanding the value of EBM.

In practice, clinical decision making and advice rely on clinical experience, collegial expert opinions, knowledge of the pathophysiology, common sense, community standards, published articles, and many other sources. The practice of EBM uses the same sources for clinical advice after passing them through a filter of "on what evidence is the advice based?"

Quality of Evidence

The quality of evidence varies from that obtained from at least one properly randomized controlled trial (best), through observational studies (cross-sectional, case-control, cohort), to the opinions of respected authorities based on clinical experience, descriptive studies, case series, and case reports (least).

Case series (retrospective assembly of selected cases without controls) and case reports, however, can yield interesting information on unique, novel conditions that tend to be rare. On the positive side, their value lies in raising questions and generating hypotheses that potentially can be tested experimentally or through a prospective, population-based study. Such an approach can lead to providing evidence where none existed before. Unfortunately, findings from case series or case reports frequently are considered as "the evidence" and are cited in a review or book chapter. Thereafter the speculation and inferences of the original author become treated as fact and are perpetuated.

This is not to imply that clinical experiences that have contributed much to advance clinical veterinary medicine should be ignored, but rather that such experiences should be overlaid with the best evidence. This will enhance the quality of service to clients who are increasingly better informed and seek guidance on the cost-effectiveness of an intervention or the likelihood of the horse's return to a certain level of activity, that is, measures of outcome that have credible estimates, rather than guesstimates. The equine professional should ask himself or herself, is this evidence available, attainable, valid and applicable to this situation?

Randomized controlled trials (RCTs) provide the highest level in the hierarchy of evidence. These are widely used in human medicine but are expensive to conduct and require large numbers of subjects in the treatment and control arms and a sufficient timeframe to evaluate differences from the "gold standard." An increasing number of randomized controlled trials are being conducted in some small animal specialties, especially for oncology, dermatology, and cardiology therapy studies. They are rare in equine medicine. Recently, however, the efficacy of tiludronate in the treatment of navicular disease in lame horses has been assessed in a double-blind, placebo-controlled clinical trial.[14] Despite design limitations, this was a major accomplishment.

Properly designed and conducted observational studies offer the greatest opportunity to generate evidence for many questions encountered in veterinary medicine. These can be retrospective case-control studies, prospective cohort or longitudinal studies, and prospective case-control studies. A retrospective case-control study starts with the disease (cases) and a similar group without the disease (controls) and works back through medical records or questionnaires to identify risk factors. The odds ratio (OR) reports an estimate of the difference between those subjects with and those without the disease for each factor or multiple factors. In a cohort study, a population is followed forward in time until disease occurs, and exposure differences are examined between those subjects with and those without the disease. This provides an estimate of the risk of getting the disease, the risk ratio (RR), and a measure of incidence. The challenge is to engage in observational studies in particular prospective studies for clinically relevant problems using specific protocols, the power afforded through advances in computer connectivity, secure data transfer, and centralized collation and analysis.

Searching for Evidence

In the field of equine podiatry, except for the immediacy of a decision made in the case of an acute condition, the majority of problems require a well thought out plan of action. There is

time to locate evidence to substantiate or consider the most appropriate therapy or intervention. This requires searching the literature, not of office books and journals, but on-line. In this age of lifelong and distance learning, equine practitioners should have Internet access to PubMed.[15] Search results can be overwhelming. The Clinical Queries option (PubMed) will produce more sensitive and specific searches for therapy, diagnosis, etiology, or prognosis. Discriminating searches using specific key words will yield a list of articles that can be further refined by scanning the most appropriate abstracts. The abstract should provide a sense of the study and outcome without having to read the full paper, which is unlikely to be available on-line. If necessary, in some instances a copy of a specific article may be available for a fee.

Descriptive articles or case series should not be dismissed just because they do not meet the rigorous standards of randomized controlled trials or observational studies. They can and do provide useful information but are not valid to determine causation or outcome. The compromise is to merge clinical experience of similar cases with whatever published information is available, recognizing that this may be relatively weak evidence. Time is of the essence in a busy practice. Finding time for literature searches requires a change of mindset, but this is essential if the best service is to be provided. The results of the effort may be more profitable than calling to consult a colleague who may also be unaware of the current best evidence. The evidence can then be shared.

All on-line literature searches are not equal. PubMed cites articles from many but not all peer-reviewed veterinary and comparative medicine journals. Relatively few are in languages other than English, particularly if the journal does not include an English abstract. Negative studies are more difficult to get published. Material presented at a conference or workshop and included in Proceedings will not be accessed, such as a double-blind study evaluating polysulfated glycosaminoglycan for the treatment of navicular disease,[16] unless subsequently published in a peer-reviewed journal. Information is available, however, that could be useful evidence in addressing a question. Other veterinary information sources are available on the Internet, some are subscription based, such as the Commonwealth Agricultural Bureau and CABdirect,[17] or offer a diagnostic support system, such as Consultant.[18] Farriery journal articles are unlikely to be included in any of the above systems.

The foregoing provides a somewhat grainy snapshot rather than a digital image of where the profession is now. The intent of this introduction to EBM is to illustrate what is possible for groups of clinicians and of clinicians and farriers to contribute to and participate in the generation of evidence that addresses important questions arising in the course of managing or preventing foot problems in the horse.

Generation of Evidence

There is probably no consensus definition of equine podiatry; for example, it encompasses the study of the foot and associated structures, foot-related problems, and their local and systemic treatment and management. The range of problems may be as great or greater than in other body system in veterinary medicine and reflects the variable nature of the feet both within the individual horse and between horses and the overarching influence of the remainder of the complex musculoskeletal system.

The key to increasing the quality and body of evidence pertaining to issues in podiatry is through observational studies. Whether prospective or retrospective, the crucial first step is the study design. This includes case definition, inclusion and exclusion criteria, sample size and power determinations, what is to be observed or measured directly or indirectly (markers), timeframe, and outcome determinants. There must be consistency and uniformity. The data and findings must have internal (repeatability) and external validity; that is, they must be generalizable to the population at large, at least in the study area, if not on a more national scale. Objectives are best achieved through multilocation collaboration of practitioners and farriers with colleagues at veterinary schools, universities, or government agencies who have epidemiologic and statistical expertise to ensure valid study design, conduct, and data analysis. All participants can become familiar with the concepts and principles of epidemiology and EBM[12] and commit to following established protocols. Retrospective case-control studies based on medical records without a priori study design are rife with missing data from inconsistent, nonuniform entries. Each missing data point has the potential of reducing group sizes to a level that the statistical power is too low to detect any group difference even when they may exist.

Apart from many retrospective case-control studies in the literature, several prospective case-control studies have been conducted on equine problems, in particular colic,[19] diarrhea,[20] and laminitis.[21-23] Most of these involved tertiary referral centers, predominantly university clinics, some single-center and some multicenter, whereas others involved practitioners. Investigators at the Texas Veterinary Medical Center have been the leaders in establishing partnerships with practitioners. The prospective case-control study design may prove the most acceptable to organize and manage. A longitudinal study of colic incidence and risk factors on horse farms in Virginia demonstrated the quality of a cohort study, although it was exceedingly time and labor intensive for the investigators.[24]

Laminitis

There is abundant literature on equine laminitis, in keeping with the diversity of opinion and theories on causation, pathophysiologic mechanisms, therapy, and management, including foot trimming and shoeing at different stages of the disease. Much controversy, contradiction, and inconsistency exist. Information comes from descriptive clinical observations, from in vivo or in vitro experimental studies of disease induction by various methods, or from highly variable naturally occurring disease. Despite this complexity, some evidence is available if the right questions are asked and appropriate key words are entered in the search.

Example of the Need for Evidence

A 7-year old Quarterhorse mare, 3 days after abdominal surgery, exhibits early signs of bilateral forelimb laminitis. Pentoxifylline and acepromazine are frequently administered at this stage in such acute conditions, together with application of heel and frog pressure. The mare was receiving flunixin meglumine at 0.5 mg/kg every 12 hours. Intravenous fluids have been discontinued.

- What are the benefits of using one or both of these drugs in the acute phase?
- Will the outcome be affected?
- What is the optimal dose, frequency, and duration of administration?
- Are there side effects (does the harm outweigh the benefits)?
- PubMed search results
  - Horse pentoxifylline: 21 citations, general
  - Horse pentoxifylline laminitis: one citation; accessing "related links" expands the list to 237, primarily nonspecific
  - Horse acepromazine: 142 citations (horse acepromazine acetylpromazine, 115 citations), general
  - Horse acepromazine (acetylpromazine or both) laminitis: two citations; "related links" to one reference expand list to 237 and links to the other reference expand list to 256; majority are the same and are nonspecific.

Intravenous acepromazine (0.066 mg/kg IV) used as a positive control in a study of oral isoxsuprine and pentoxifylline (4.4 mg/kg q8 hours, 10 days) on digital and laminar blood flow in healthy horses, caused an increased blood flow to the digit.[25] The authors inferred that potentially it would have a greater effect on improving digital blood flow than oral isoxsuprine or pentoxifylline when treating ischemic conditions of the equine foot.

Pentoxifylline was assessed in two endotoxemia induction models in horses. In one, a flunixin meglumine (1.1 mg/kg) and pentoxifylline (8 mg/kg IV) combination may help offset deleterious hemodynamic effects of endotoxin more effectively than either flunixin or pentoxifylline alone.[26] Moreover, when given as a bolus (7.5 mg/kg IV) followed by infusion (3 mg/kg/h) over 3 hours, pentoxifylline showed limited beneficial effects after in vivo challenge exposure with endotoxin.[27] In another study, bolus pentoxifylline (7.5 mg/kg IV) followed by infusion (1.5 mg/kg/h) for 3 hours, increased 6-keto-prostaglandin F1 alpha and significantly suppressed ex vivo endotoxin-induced tumor necrosis factor activity in horses during the period of infusion.[28]

No descriptive or experimental study was cited in peer-reviewed articles describing the use of either one or both drugs in laminitic horses. In the absence of such evidence, the adoption of drug usage in acute laminitis cases appears to have arisen by extrapolating data from in vivo healthy animal studies or from in vitro studies with tissue other than from the laminae or with peripheral blood cells. Experimental findings could indicate that acepromazine would be preferable to pentoxifylline, although pentoxifylline in the presence of flunixin meglumine could be beneficial in endotoxin-associated post-abdominal surgery laminitis. There is a high risk of systemic endotoxemia after intestinal surgery, particularly associated with manipulation and resection. The inference is that pentoxifylline could be useful, but at what stage after surgery should it be given, at what dose rate, and for how long? The practitioner should ask himself or herself at this point whether the foreground questions been answered. The answer is that not one of them has been addressed. A further question is, is this evidence? These drugs are still widely used in acute laminitis therapy. Finally, is there room for improvement?

In this situation, at least a retrospective study or assessment could be conducted at a tertiary center to see whether use of these drugs can be teased from the rest of the pharmacological input for acute laminitis. This may shed light on potential dose rate, frequency, and duration. If clinicians believe that the question of whether these drugs are crucial in early acute laminitis takes precedence over questions related to other adjunctive treatment and management procedures, then a prospective study should be designed and conducted to assess each vasoactive agents in early acute laminitis. Subjects at several centers could be enrolled immediately after surgery in a longitudinal design or after exhibiting initial signs of disease in a matched case-control study. The protocol would include standardized drug dose, frequency, duration, inclusion and exclusion criteria, clinical and physiological parameters to record, permitted drug therapy and management practices, determinants of outcome, and other factors.

Studies of these drugs in cases of acute laminitis may be ongoing and incomplete with preliminary data already presented at a meeting. However, currently there is no definitive evidence to support or refute their administration. This is one example of the lack of clinical evidence for much of what is practiced not only in podiatry but also for most other aspects of equine medicine.

Conclusion

Fortunately, progress is being made on all fronts in veterinary medicine. Professionals committed to advancing the practice of equine podiatry have the responsibility to take the lead and to find the time and money to conduct valid studies. They have the unique opportunity to cooperate and address questions of clinical relevance. This will involve acceptance of the majority view in following protocols, rather than the pursuit of one's individual treatment and management options. Outcomes can be measured from many perspectives of benefit and cost-effectiveness. These will differ depending on the type of work the horse is engaged in. Whatever the podiatry problem and the specific question posed, if the outcome reveals that more horses return to a functional level of activity and remain free of lameness for longer periods of time, then there are benefits for all. This evidence can be used.

The practice of evidence-based medicine is here and now. It is not an esoteric premise or philosophy having no practical or realistic clinical merit that can thus be ignored. Management of the many varied problems encountered in the horse's foot should be addressed by assessing therapeutic and procedure interventions in an objective manner. Clinical evidence will enhance the practice of equine podiatry. It is a powerful tool.

FARRIER EDUCATION

Michael Wildenstein

Horseshoeing is an art and a skill, but most of all it is a profession. The horse-owning public expects competence of farriers, and farriers must educate and comport themselves accordingly. The importance of proper hoof care and good farriery skills has been acknowledged by eminent authors of veterinary and farriery texts for at least the past 100 years (Boxes 24-1 to 24-6). Current society does not depend on the horse as it has in times past, but the importance of professional

Box 24-1

Rights were not granted to include this box in electronic media.
Please refer to the printed publication.

From Roberge D: *The foot of the horse,* Liverpool, 1984, William R. Jenkins.

Box 24-2

Rights were not granted to include this box in electronic media.
Please refer to the printed publication.

From Gamgee J: *A treatise on horseshoeing and lameness,* London, 1871, Longmans Green and Co.

Box 24-3

"The injury to feet resulting from improper shoeing may not be apparent at once. It may be, and often is, of a slow and gradual nature, and not credited to its true cause until the horse is rendered an incurable cripple."

From Hunting W: *The art of horseshoeing, a manual for farriers,* London, 1895, H & W Brown.

Box 24-4

Rights were not granted to include this box in electronic media.
Please refer to the printed publication.

From Holmes CM: *The principles and practice of horseshoeing,* Leeds, 1949, Farriers Journal.

hoof care is of no less value. Proper management, veterinary care, and hoof care must be handled by educated individuals who are trained at accredited schools and are willing to meet the standards set by their respective professional organizations. Horse owners should be as comfortable with their farriers' qualifications as they are with those of the other professionals consulted in the well being of their horses.

The care and maintenance of the horse's hoof is a physically demanding profession that requires years of practice and education to perform skillfully. Good horsemanship skills are a prerequisite for learning to care for the horse's hoof. A knowledge of the anatomy and diseases of the equine foot and the ability to communicate with both the horse owner and the veterinarian are also very important. In fact, the ability to read a radiograph is just as important as learning to forge and to use adhesives to attach a shoe to a hoof.

Farrier Schools

Before the decline of the horse population after World War II, most farriers learned the trade by apprenticing with an experienced farrier. Even today, there are many farriers who learned their craft primarily from other farriers or from working in a family farrier practice. During the 1950s and 1960s, more schools began offering training for would-be farriers. The horse population has continued to grow since the 1960s, as have educational opportunities for aspiring farriers. Today, most farriers learn the rudiments of their profession by attending a farrier school. More than 50 private schools offer farrier training, and several colleges offer courses in hoof care (although courses vary widely in length and quality among schools and programs).

Setting Standards

Unfortunately, despite the abundance of farrier schools and other educational opportunities, there are still no universal minimum standards for farriers. Those who trim or apply shoes to horses' hooves have a dynamic, life-long effect on the welfare of the horse. All farriers should all be required to meet a minimum standard of knowledge and competence so that no harm is done to the horse. Being required to pass a standardized examination in the practical and theoretical aspects of hoof care and trimming would raise the standard of farriery and prevent unqualified persons from performing this important task. To that end, all hoof care and horseshoeing courses should provide a sufficient level of training to enable the graduates to pass such an examination.

The American Farriers Association (AFA) currently sets such a standard and offers certification examinations at three different levels (see below), on a voluntary basis. Anyone who cares for horses' hooves should undertake at least basic AFA certification. Veterinarians, breed associations, and the horse-owning public are voicing concerns about the quality of work performed on horses' hooves. Times are changing, and farriers and hoof care practitioners need to be more professional as a group and meet the concerns of the client for the sake of the horse.

Professional Organizations

The Worshipful Company of Farriers

In England, The Worshipful Company of Farriers (WCF) has been in existence since 1356. A great deal of work by the WCF led to the passing of the Farriers Registration Act in 1975, the goals of which are summarized in Box 24-6. The WCF has the general function of securing adequate standards of competence and conduct among persons engaged in the shoeing of horses. The WCF also endeavors to promote, encourage, and advance the art and science of farriery and education in connection with the art and science of farriery.

To be a registered farrier in England, a candidate must complete 4 years of training that includes an apprenticeship with schooling and successful completion of a comprehensive examination. The WCF has three levels of examination. Successful completion of the first level, Diploma, is required for one to shoe horses. The second level is Associate, and the highest level is Fellow of The Worshipful Company of Farriers (FWCF).

Box 24-5

The scope of horseshoeing is by no means narrow and insignificant as it may appear, and since a knowledge of the anatomy and physiology of the horse's body in general, and of instruction, with demonstrations upon dissected material and upon living horses, but also an abundance of daily work at the forge and on the floor in shoeing of horses is needed. Therefore a course of four to six weeks is not sufficient.

From Lungwitz A, Adams JW: *A textbook of horseshoeing,* Philadelphia, 1897, JB Lippincott.

Box 24-6

Rights were not granted to include this box in electronic media.
Please refer to the printed publication.

From Scott AGW: *The company, the craft, and the city,* London, 1990, The Worshipful Company of Farriers.

Other Farrier Associations

The European Farriers Association is working to establish acceptable standards for all farriers, which, at this time, vary from one country to another. Walt Taylor started the American Farriers Association in the United States in 1971 and has since helped raise the standards of farriery in many parts of the world. As he brought farriers together in the United States, Taylor also brought farriers together in Europe to meet the great challenge of creating a single standard for acceptable hoof care.

The AFA has three levels of certification: basic, tradesman, and Certified Journeyman Farrier (CJF). Every farrier and any other hoof care professional should obtain at least the basic AFA certification and continue to undertake further training and other educational opportunities throughout their career. Horseshoeing has a great future, and thanks to the work of Walt Taylor and the AFA, farriers have an opportunity to advance the profession, for the good of the horse.

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