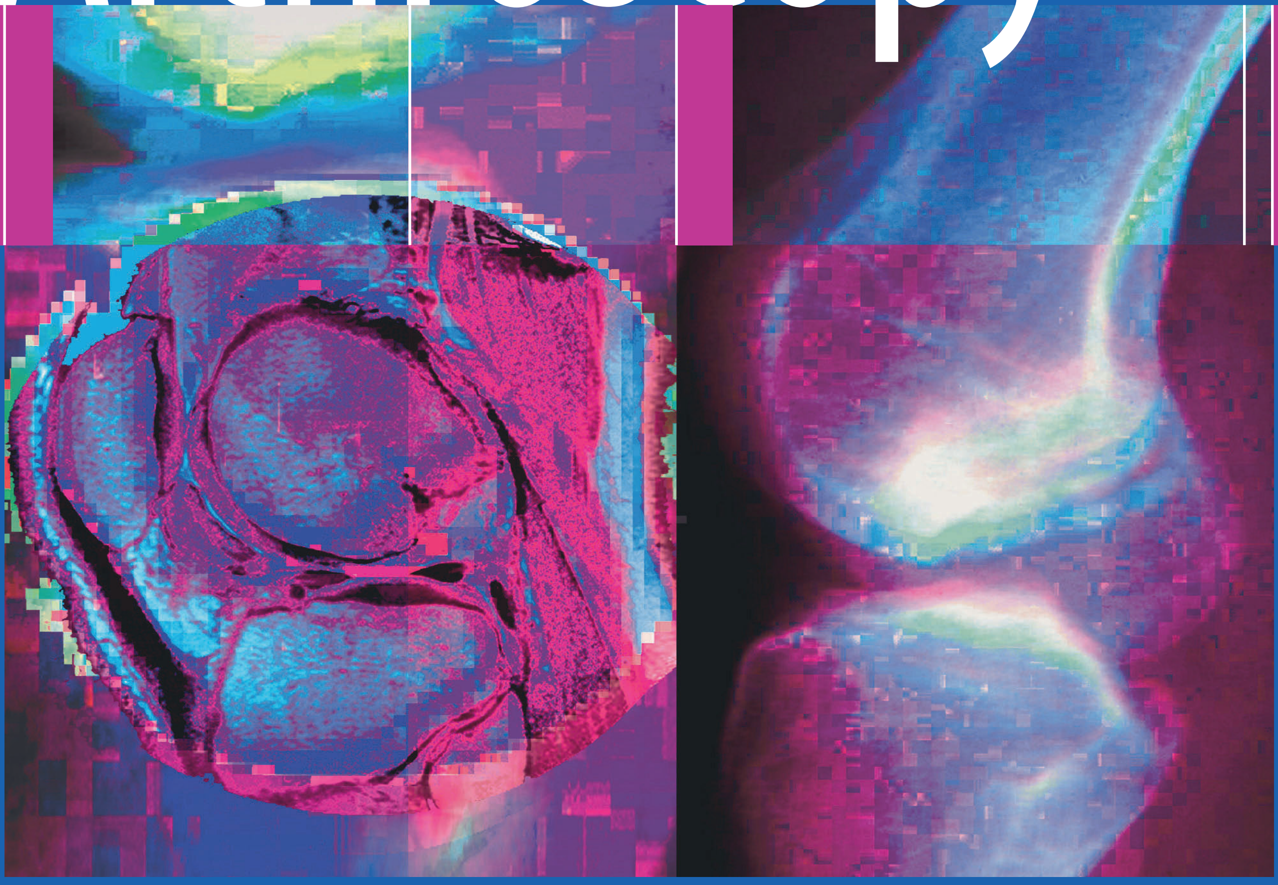
Brian P. McKeon
James V. Bono
John C. Richmond *Editors*

Knee Arthroscopy



Foreword by James P. Tasto

Springer

Knee Arthroscopy

Brian P. McKeon · James V. Bono ·
John C. Richmond
Editors

Knee Arthroscopy

Foreword by James P. Tasto

Springer

Editors
Brian P. McKeon
Assistant Clinical Professor
Department of Orthopaedics
Tufts University School of Medicine
Boston, MA 02111, USA
Department of Orthopaedic Surgery
New England Baptist Hospital
Boston, MA 02120, USA
Boston Sports and Shoulder Center
Chestnut Hill, MA 02467, USA

John C. Richmond
Chairman, Orthopaedic Surgery
New England Baptist Hospital
Boston, MA 02120, USA
Professor, Orthopaedic Surgery
Tufts University School of Medicine
Boston, MA 02111, USA

James V. Bono
Clinical Professor of Orthopaedic Surgery
Tufts University School of Medicine
Boston, MA 02111, USA
Department of Orthopaedic Surgery
New England Baptist Hospital
Boston, MA 02120, USA

ISBN 978-0-387-89503-1
e-ISBN 978-0-387-89504-8
DOI 10.1007/978-0-387-89504-8

Library of Congress Control Number: 2009920355

Printed on acid-free paper

springer.com

To my precious daughters, Molly and Nell, whose energy and passion for life are contagious.

To my loving wife, Heidi: You are my true guiding light and soul mate.

Finally, to my parents, Robert and Diane: Your dedication and sacrifices allowed me to explore all opportunities.

BPM

To my wife and best friend, Meg, for her unconditional love and support and for the everlasting joy she has brought to my life.

To our blessed little family: Andrew, Olivia, Caroline, and Thomas.

To my father, for the opportunities he has given me.

JVB

To all of my patients: Entrusting me with your care has permitted me to learn and teach the next generation of arthroscopists.

JCR

Foreword

With this book, *Knee Arthroscopy*, Dr. Brian McKeon, Dr. James Bono, and Dr. John Richmond and their selected contributors have carefully amassed a compendium of chapters well suited for the general orthopaedist as well as for the accomplished knee surgeon. Authoring and editing a textbook as well as contributing chapters reflect an unselfish dedication to the education of others. The inclusion of selected case reports in the chapters adds a unique feature that brings the reader back to the more pragmatic aspects of patient care.

The authors and editors have combined a careful and unbiased review of the literature on each subject with a synopsis of current thinking and suggested pathways for the reader. Comprehensive references and illustrations complement the text and add to the clarity of the topics.

The selection of topics represents a comprehensive and complete array of almost every knee condition from simple to complex. The reader is able to use selective components of the review to tailor his or her treatment regiments to a vast array of clinical conditions.

This book will be a stable and enduring reference for years to come.

University of California, San Diego, California — James P. Tasto, MD

Preface

Knee Arthroscopy is intended to be a clinical text for the arthroscopic management of knee disorders and to be used as a practical reference for all health care professionals engaged in the treatment of the knee. Each chapter includes a discussion of relevant anatomy, indications, step-by-step descriptions of surgical techniques, rehabilitation, complications, clinical pearls, as well as case reports.

Knee Arthroscopy begins with a description of normal knee anatomy and traditional arthroscopic techniques. Separate chapters on meniscectomy, meniscal repair, and meniscal transplantation follow. Ligament reconstruction – including anterior cruciate ligament, posterior cruciate ligament, and posterolateral corner reconstruction – is described in detail. Individual chapters address the arthroscopic management of patellofemoral disorders, cartilage repair, arthrofibrosis and synovial lesions, knee fractures, articular cartilage injuries, and degenerative joint disease.

We are grateful to have received the support of so many recognized master surgeons who have contributed to the text, and we are honored to be able to present their combined experience in the ensuing pages.

Boston, Massachusetts — Brian P. McKeon
Boston, Massachusetts — James V. Bono
Boston, Massachusetts — John C. Richmond

Contents

Contributors

Michael J. Angel, MD., Orthopaedic Sports Fellow, Kerlan Jobe Orthopaedic Clinic, Los Angeles, California

James V. Bono, MD., Clinical Professor of Orthopaedic Surgery, Tufts University School of Medicine, New England Baptist Hospital, Boston, Massachusetts

Craig Edson, MS, PT, ATC, Department of Orthopaedic Surgery, Geisinger Clinic, Danville, Pennsylvania

Iván Encalada-Diaz, MD., Orthopaedic Surgeon, Assistant Clinical Professor of Orthopaedics, Department of Orthopaedics, National Autonomous University of Mexico (UNAM), National Institute for Rehabilitation, Mexico City, Mexico

Gregory C. Fanelli, MD., Department of Orthopaedic Surgery, Geisinger Clinic, Danville, Pennsylvania

John P. Fulkerson, MD., Clinical Professor of Orthopaedic Surgery, Sports Medicine Fellowship Director, University of Connecticut Medical School, Farmington, Connecticut

Robert Hunter, MD., Professor of Clinical Orthopaedics, Department of Orthopaedic Surgery, University of Arizona, Tucson, Arizona

Darren L. Johnson, MD., Professor and Chairman, Department of Orthopaedic Surgery, University of Kentucky, Lexington, Kentucky

David M. Junkin, Jr., MD., Orthopaedic Specialty Center, Willow Grove, Pennsylvania

Laurie M. Katz, MD., Department of Orthopaedics, New England Baptist Hospital, Boston, MA

Jordan Kerker, MD., Resident Physician, Department of Orthopaedic Surgery, Long Island Jewish Medical Center, New Hyde Park, New York

Brian P. McKeon, MD., Assistant Clinical Professor, Department of Orthopaedics, Tufts University School of Medicine, New England Baptist Hospital, Boston, Massachusetts; Boston Sports and Shoulder Center, Chestnut Hill, Massachusetts

S.L. Mortimer, MD., Clinical Instructor, Arizona Institute for Sports Medicine, Department of Orthopaedic Surgery, University of Arizona, Tucson, Arizona

Jayesh K. Patel, MD., Resident, Orthopaedic Surgery, Department of Orthopaedic Surgery and Sports Medicine, University of Kentucky, Lexington, Kentucky

Kristin N. Reinheimer, PAC, Department of Orthopaedic Surgery, Geisinger Clinic, Danville, Pennsylvania

John C. Richmond, MD., Chairman, Orthopaedic Surgery, New England Baptist Hospital; Professor, Orthopaedic Surgery, Tufts University School of Medicine, Boston, Massachusetts

Glen Ross, MD., Assistant Professor of Surgery, Department of Orthopaedics, Tufts School of Medicine, New England Baptist Hospital, Boston, Massachusetts

Anthony Schena, MD., Department of Orthopaedics, New England Baptist Hospital, Boston, Massachusetts

Nicholas Sgaglione, MD., Program Director and Associate Chairman, Department of Orthopaedic Surgery, Long Island Jewish Medical Center, New Hyde Park, New York

Jeffrey T. Spang, MD., Assistant Professor, Department of Orthopaedic Surgery, University of North Carolina School of Medicine, Chapel Hill, North Carolina

Mark E. Steiner, MD., Section Chief, Orthopaedic Sports Medicine, Department of Orthopaedic Surgery, New England Baptist Hospital, Harvard Medical School, Tufts Medical School, Boston, Massachusetts

Carl T. Talmo, MD., Instructor in Orthopaedic Surgery, Department of Orthopaedic Surgery, Tufts University School of Medicine, New England Baptist Hospital, Boston, Massachusetts

Daniel J. Tomaszewski, MD., Department of Orthopaedic Surgery, Geisinger Clinic, Danville, Pennsylvania

Paul P. Weitzel, MD., Department of Orthopaedics, New England Baptist Hospital, Boston, Massachusetts

Knee Arthroscopy: Technique and Normal Anatomy

Anthony Schena and Glen Ross

Introduction

Arthroscopy of the knee is the most common orthopaedic procedure performed in the United States [1–6]. Given the frequency of this procedure, it is hard to fathom that knee arthroscopy did not enter into the mainstream of orthopaedic surgery until the 1970s. In 1969, Masaki Wantanabe and colleagues published the *Atlas of Arthroscopy* [7]. In their seminal publication, they described the results of their first arthroscopic procedure, the removal of an intraarticular pigmented villonodular synovitis (PVNS), and provided pictures of their first arthroscopic meniscectomy. Jackson and Abe followed up on the work of their Japanese peers with the publication of their arthroscopic technique and outcomes in 1972 [8]. Arthroscopy of the knee became an accepted practice in the 1970s. Several forward-thinking orthopaedists contributed to the early evolution of knee arthroscopy, developing the techniques and tools that are still used today [9–12]. Over the past 40 years, knee arthroscopy has evolved from a rudimentary diagnostic tool to a state-of-the-art system of fiber optics and precision equipment. Knee arthroscopy has become a standard part of orthopaedics. It is the foundation for procedures ranging from the simple meniscectomy, to the multiligamentous knee injury, to cartilage restoration. First, the techniques of knee arthroscopy and the anatomy of the knee will be examined.

Anatomy

External Anatomy

Most knees have palpable bony prominences that can be used to determine the topography of the knee (Fig. 1). The patella, patella tendon, and the medial and lateral joint line are usually accessible. The medial and lateral condyles are also useful for identifying and mapping out the knee

Fig. 1 Anterior knee anatomy and portal placement

Intraarticular Anatomy

Familiarity with the basic anatomy of the knee is essential for knee arthroscopy and treatment of knee pathology. In the patellofemoral joint, the patella should sit within the natural groove of the trochlea. There is a prominent medial and lateral facet. The inferior pole is generally nonarticulating. The patella should track well through the trochlea when brought through a range of motion. There should be no evidence of an overriding plica on the medial side of the normal knee.

In the medial compartment, the "C" medial meniscus is firmly attached to the joint capsule by the meniscotibial (coronary) ligament. The mid aspect of the meniscus is directly attached to the deepest fibers of the medial collateral ligament. The width of the meniscus from the capsule to the inner aspect is approximately 9–10 mm.

A. Schena (✉)
Department of Orthopaedics, New England Baptist Hospital, Boston, Massachusetts

B.P. McKeon et al. (eds.), *Knee Arthroscopy*, DOI 10.1007/978-0-387-89504-8_1,

The average thickness of the meniscus is 3–5 mm. The medial meniscus will bear 40% to 50% of the joint force in extension and up to 85% to 90% of the force in flexion [13]. In general, there should be less than 5 mm of translation of the intact meniscus [14]. Vedi et al. have demonstrated that the anterior horn of the medial meniscus moves 7.1 mm, the posterior horn moves 3.9 mm, and that there is 3.6 mm of mediolateral radial deviation [14]. They postulated from this that the posterior horn of the meniscus has less excursion and is thus prone to injury.

The medial femoral condyle has a notch anteriorly for the meniscus to settle into when the knee in is extension. The condyle has a curved shape to allow for medial tibial plateau external rotation in full extension: the screw-home mechanism.

In the intracondylar notch, the origin of the posterior cruciate ligament (PCL) should be noted just off the cartilaginous margin of the medial femoral condyle. The insertion of the anterior cruciate ligament (ACL) rests in line with the anterior horn of the lateral meniscus. The notch should easily accommodate both cruciate ligaments and is usually obscured by the ligamentum mucosum. The meniscofemoral ligaments, when present, should also be identified and inspected. Seventy percent of knees will have either the anterior (Humphrey) or posterior (Wrisberg) ligaments; 6% will have both [15]. The ligament of Humphrey is approximately one third the diameter of the PCL. It is attached to the posterior horn of the lateral meniscus, runs anterior to the PCL, and inserts on the distal edge of the femoral attachment of the PCL. The ligament of Wrisberg is larger, approximately one half the diameter of the PCL, and runs from the posterior horn of the lateral meniscus to the medial femoral condyle [16, 17].

In the lateral compartment, the elongated C-shaped lateral meniscus is firmly attached to the capsule by the coronary ligament, except along the hiatus for the popliteus tendon. The posterior horn of the meniscus has attachments to one or both of the meniscofemoral ligaments when present. The lateral meniscus can have up to 11 mm of excursion. The anterior horn has 9.5 mm of excursion, the posterior horn 5.6 mm of excursion, and there is 3.7 mm of radial displacement [14]. It is slightly larger than the medial meniscus, with a width of 10–12 mm and a thickness of 4–5 mm [13]. As noted on the medial side, the lateral meniscus will bear up to 90% of the joint forces in flexion and from 50% to 70% of the force in extension [13, 18].

As noted in the medial compartment, there is a notch along the lateral femoral condyle to accommodate the lateral meniscus when the knee is in extension. There is also a sulcus for the popliteus tendon. This intraarticular aspect of the sulcus creates a gap in the attachment of the meniscus to the tibia.

Preoperative Preparation

Most knee arthroscopies are carried out in conjunction with another procedure. In these instances, the primary procedure, such as an ACL reconstruction, will need to be prepared for. All necessary x-rays, magnetic resonance imaging (MRI) scans, and other ancillary studies should be present and displayed for easy access during the procedure.

Techniques

Operating Room Setup

The room is set up for maximum efficiency and ease (Figs. 2 and 3). Anesthesia is located at the head of the bed. The monitor and stack setup is placed on the contralateral side of the patient. A Mayo stand can be brought in from the contralateral side once the patient is prepared and draped to provide a resting place for shavers, probes, and biters. The surgical scrub technician is set up at the foot of the bed, usually on the contralateral side. Suction canisters and fluid can be set up on the ipsilateral side of the bed. Prior to the start of the surgery, the monitor should be on, the fluid line flushed, the suction set up, and the power to the shaver on. The camera should be set for the proper number of pictures, and any recording device, such as a CD-ROM device, should be in place. Be sure that the surgical staff has allowed proper time between sterilization of the equipment and the start of surgery to avoid fogging of the lens.

Anesthesia

The majority of our knee arthroscopies are performed under general anesthesia with the use of an laryngeal mask airway (LMA). This allows for the maximum amount of patient relaxation and access to the compartments. Prior to the surgery, the patient receives 650 mg acetaminophen and, if able, 400 mg celecoxib. Preoperative celecoxib has reduced the need for perioperative narcotics and the incidence of adverse opioid effects in the ambulatory knee surgery patient [19]. Once in the operative suite, general anesthesia is induced and the LMA is placed. The portal sites are then preinjected with 10 mL of 1% lidocaine with epinephrine (1:200,000). Local anesthetic injection limits the somatosensory afferent signals created by the surgical incision, thus decreasing the postoperative pain and opioid need [20]. At this point, the patient is positioned for the surgery. There are other reports in the literature focusing on the perioperative

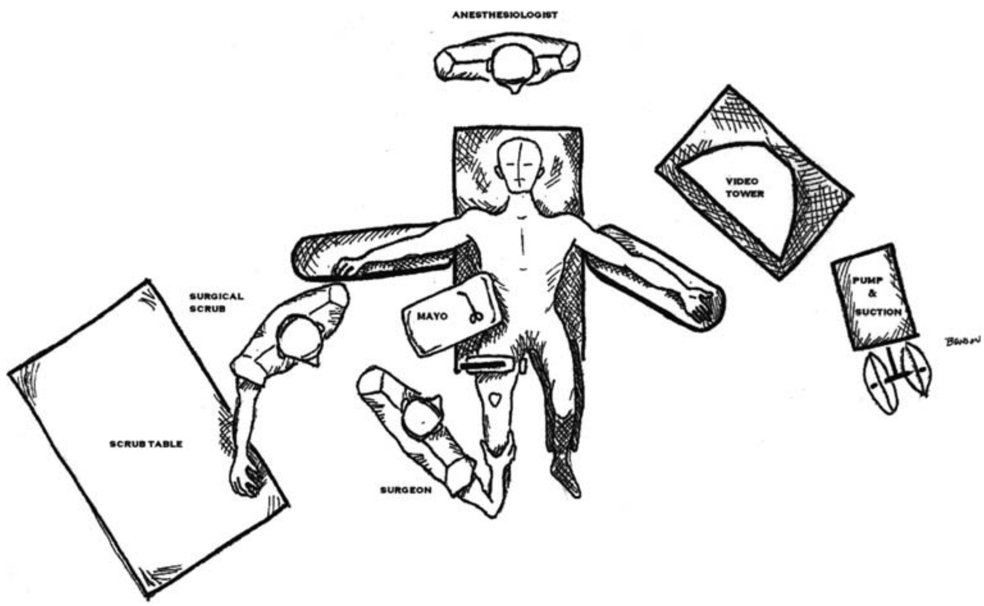


Fig. 2 Operating room setup. (Courtesy of Emily Benson, MD, Ventura County Medical Center, Ventura, CA.)

Fig. 3 Arthroscopic instruments

are injected with 1% lidocaine with epinephrine (1:200,000). The patient's well leg is placed in a padded leg holder. This leg is wrapped in an elastic bandage to limit blood pooling in the nonoperative limb. The operative extremity is placed in a commercially available leg holder (Fig. 4). If a post is going to be used for the arthroscopy, it should be set up to ensure that it will be at the right level. A tourniquet, if desired, should be applied prior to placing the leg in the leg holder. To provide maximum mechanical advantage, the leg holder should be placed as close to the knee joint as possible. However, if the surgery will involve procedures beyond a simple meniscectomy, such as an ACL reconstruction or meniscal repair, great care should be taken to provide enough room for the passage of surgical instruments around all aspects of the knee. In these instances, a hand breadth of distance is

pain. Miskulin and Maldini reported the usefulness of local anesthesia and intravenous diclofenac [21], and others have reported less favorable results with use of local anesthesia only [22–24]. In those patients undergoing simple knee arthroscopy, Denti et al. reported the success of preoperative morphine as a means of reducing the noxious stimuli created by surgery and thus reducing the opioid need [20].

Patient Setup

Prior to surgery, the patient is seen in the preoperative holding area, and the correct extremity is identified and marked. In the operative suite, the patient is positioned supine on the table. After general anesthesia is induced, the portal sites

Fig. 4 Leg holders for operative and nonoperative legs

Fig. 5 Patient positioning

needed between the superior aspect of the patella and the leg holder. Once the leg is in the leg holder, the bed is placed into a reflexed position to relax the hamstrings and the lower back, and the foot of the table is dropped (Fig. 5). The knee is then prepared and draped. Draping involves use of an adhesive, impervious U-drape, a standard knee arthroscopy drape, impervious stockinet, and a Coban wrap, 3M (St. Paul, MN)

Portals (Fig. 6)

Anterolateral Portal

The anterolateral portal is the primary viewing portal for knee arthroscopy. With the knee flexed to 90 degrees, the inferior pole of the patella, the lateral border of the patella, and the lateral joint line are palpated. An incision is made with a no. 11 blade approximately 1 cm above the joint line and in line with the lateral border of the patella. A vertical incision angled toward the intracondylar notch is used unless a horizontal portal is preferred. In either case, great care is taken to protect the meniscus and intraarticular structures. Once the joint capsule has been incised, a blunt trochar is advanced into the notch. The knee is then brought into extension while gently advancing the trochar into the suprapatellar pouch. The trochar is removed, and the 30-degree camera is placed into the knee.

Prior to establishing the anteromedial portal, a preliminary inspection can begin. The patella and trochlea are both inspected for cartilage wear or damage. The medial and lateral gutters are inspected for loose bodies and impinging osteophytes. The medial-sided synovium is inspected for a large, engaging plica (Figs. 7 and 8). Once this initial inspection is complete, the arthroscope is passed along the medial femoral condyle and into the medial compartment.

Anteromedial Portal

The anteromedial portal is created with the knee at 30 degrees of flexion. The 30-degree scope needs to be rotated to obtain an unobstructed view of the anterior aspect of the medial meniscus and anterior capsule. The soft spot just medial to the medial border of the patella tendon is palpated. An 18-gauge spinal needle is used to find the most appropriate spot for this portal. Medial meniscal pathology requires a portal that will allow unencumbered access to the posterior horn. Lateral meniscal pathology requires clearance of the tibial spines to gain access to the lateral compartment. The 18-gauge spinal needle is very helpful in establishing this portal. The portal is created using the no. 11 blade

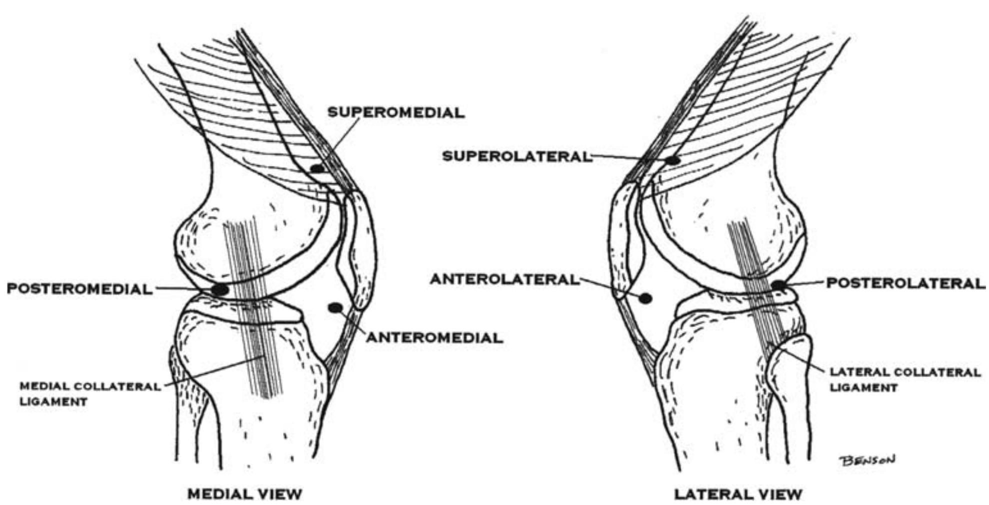


Fig. 6 Portal placement for knee arthroscopy. (Courtesy of Emily Benson, MD, Ventura County Medical Center, Ventura, CA.)

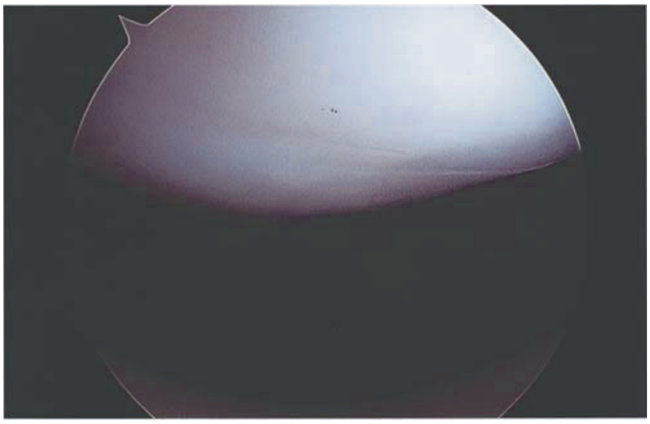


Fig. 7 Arthroscopic view of the patellofemoral joint

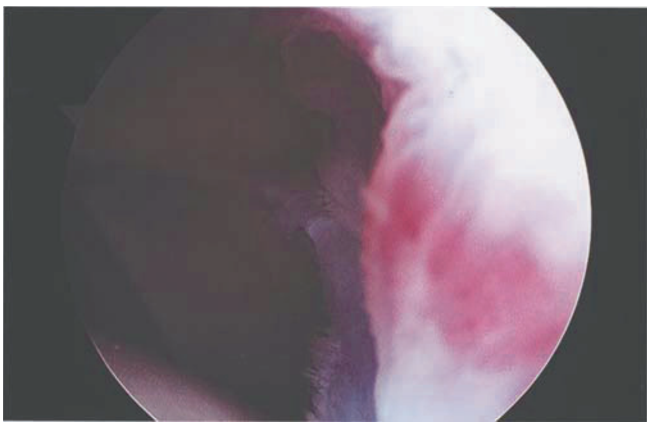


Fig. 8 Medial plica

placed parallel to the tibial plateau. Once the capsule has been penetrated, the blunt trochar can be used to establish the portal.

With the establishment of the anteromedial and lateral portals, diagnostic arthroscopy commences. The probe is brought into the medial compartment. The leg is slightly flexed (10–30 degrees), and a valgus force is placed on the knee joint. The tibia should be externally rotated. The entire medial meniscus is probed. If there is difficulty getting to the posterior horn of the meniscus, the knee can be gently brought into extension, with great care taken to avoid scuffing the condyle with the camera. This usually opens up the posterior aspect of the joint and allows for probing of the horn and posterior meniscus. If there is difficulty assessing the posterior horn of the meniscus, then a posteromedial portal can be established or a 70-degree camera can be used. After thorough inspection of the posterior meniscus, the remainder of the meniscus is inspected. Once the meniscus has been probed, the medial femoral condyle and tibial plateau is inspected. The condyle is viewed as the knee is brought through a full range of motion. Once the inspection of the medial compartment is complete, the arthroscope is brought into the intracondylar notch (Figs. 9 and 10).

In the notch, the ACL, PCL, meniscofemoral ligaments, and ligamentum mucosum are identified and probed. The morphology of the notch, its depth and width are noted, especially in the case of a ligamentous injury. The ligamentum mucosum is noted running from the superior aspect of the notch to the fat pad. It may be excised if it impedes thorough evaluation of the ACL, PCL, or other intraarticular structures. Often, the arthroscope must be passed up and over the ligamentum to establish a view of the ACL. An intraoperative Lachman test at 30 degrees can be carried out if ACL pathology is questioned. In cases involving an old ACL injury or loss of extension after ACL reconstruction, the footprint of the ACL should be inspected for a remnant of the ACL (Cyclops lesion). Once these structures are inspected, the probe should be placed along the lateral side of the ACL, and the knee should be brought into a varus position or a figure-four position if a post is being used (Fig. 11).

In the figure-four/varus position, the lateral compartment is inspected. A varus force of the leg just above the knee

Fig. 9 Normal medial meniscus

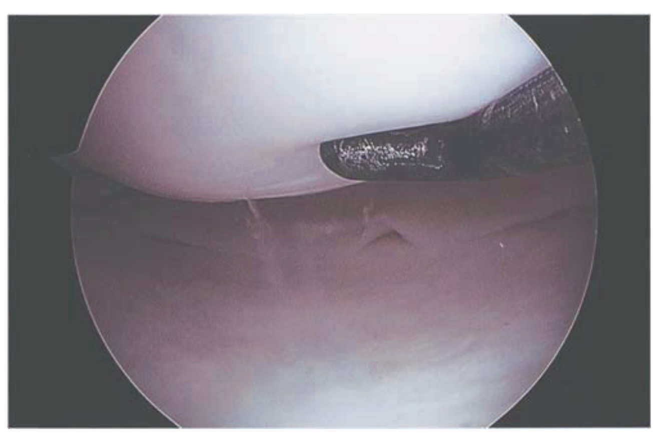


Fig. 10 Medial femoral condyle

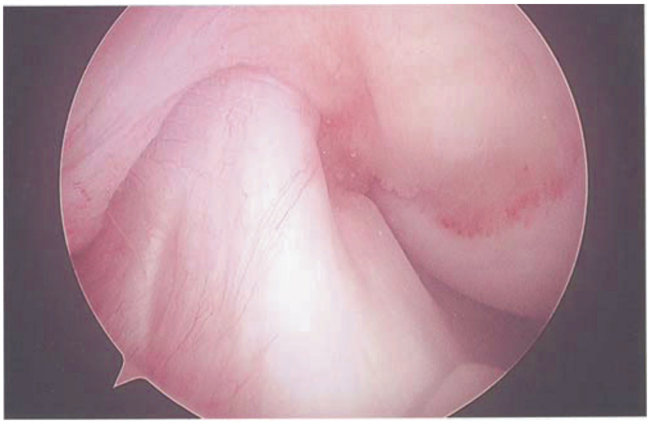


Fig. 11 Anterior cruciate ligament

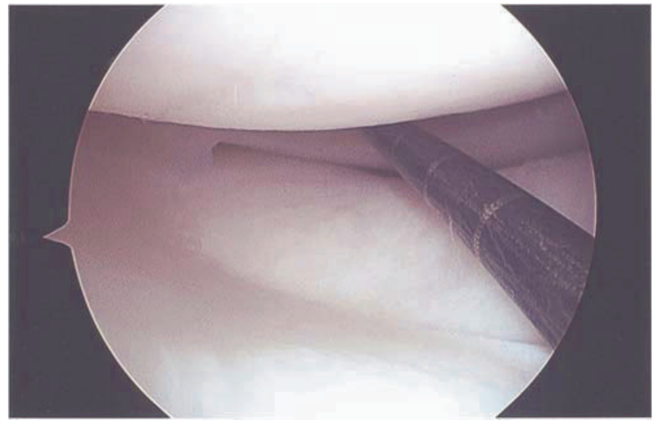


Fig. 13 Lateral femoral condyle

can open the lateral compartment. As on the medial side, the probe is used to inspect and test the lateral meniscus. The lateral meniscus is usually easier to inspect than is the medial meniscus. If the anterior horn is unable to be fully inspected, whether due to the fat pad, ligamentum mucosum, or portal placement, the arthroscope can be switched to the medial portal and directed toward the anterior horn. When inspecting the posterior meniscus, the popliteal tendon should be noted and the popliteal hiatus/sulcus probed. Remember that the lateral meniscus may have as much as twice the excursion as the medial meniscus and that the popliteal hiatus is a normal-occurring interruption in the meniscocapsular ligament. As with the medial compartment, the lateral femoral condyle is inspected through the entire range of motion for any chondral injury. The lateral tibial plateau is examined as well (Figs. 12, 13, and 14).

Once the lateral compartment has been inspected, the arthroscope is brought back into the suprapatellar pouch. The undersurface of the patella and the trochlea can now be probed and inspected for any cartilage injury. In those instances of patellofemoral disease, the exact tracking of the patella may be of primary interest. At this point, the superomedial portal as described by Fulkerson can be made [25].

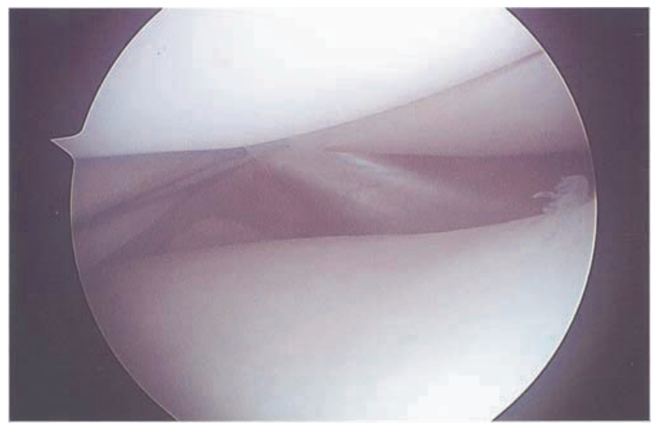


Fig. 14 Popliteus

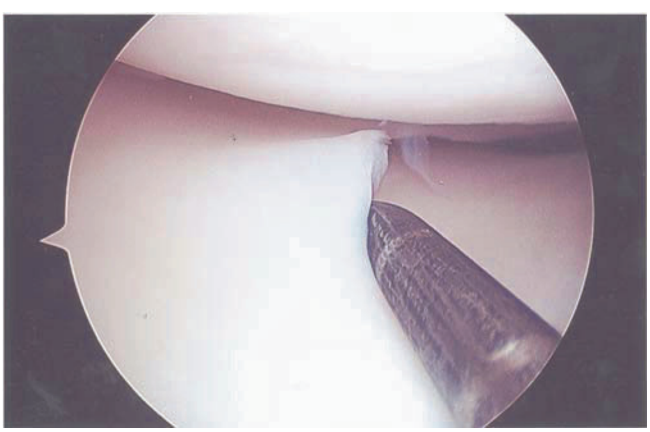


Fig. 12 Lateral meniscus

Superomedial Portal

This portal is created with the knee in extension and the camera in the anterolateral portal. An area 2–3 cm proximal to the superior pole of the patella and approximately 1 cm medial to the midline is identified first by palpation and then with the 18-gauge spinal needle. The skin is incised with a no. 11 blade. The portal is created under direct visualization with a mosquito hemostat or blunt trochar. A switching stick is then passed into the suprapatellar pouch and the sheath placed over it. From this vantage point, the articular surfaces and the tracking of the patella can be visualized with ease.

Having completed the examination and inspection of the menisci, the articular surfaces, and the ligaments, one can move to inspection of the posteromedial and posterolateral compartments. The ability to examine these compartments is essential for the arthroscopist, especially when there is a need to establish a posterior portal or retrieve a loose body. In the ligament-intact knee, the arthroscope needs to be passed by the ACL and PCL to gain access to the posterior compartments. With the scope in the anterolateral compartment and the knee flexed to 90 degrees, a switching stick can be passed through the medial portal between the medial femoral condyle and the PCL. The switching stick should be eased into position. Once in the posteromedial compartment, the sheath for the arthroscope can be passed over the switching stick and the arthroscope delivered into the posteromedial compartment.

Posteromedial Portal

The posteromedial portal should be established approximately 1 cm posterior to the medial femoral condyle and 1 cm proximal to the joint line. The knee itself should be flexed to 90 degrees, abducted, and externally rotated. Generally, the position can be palpated and then identified with the 18-gauge spinal needle. Once the spinal needle is in the proper position, the skin is incised with the no. 11 blade. A mosquito hemostat or blunt trochar is then used to bluntly create the portal. When performing a PCL reconstruction, it is helpful to place a cannula in this portal. With the arthroscope coming in through the posteromedial portal, the posterior horn of the medial meniscus, the posterior medial femoral condyle, and the synovial lining of the posteromedial compartment may all be inspected. A probe can be brought through the anterolateral portal, between the PCL and condyle, to help with the inspection of this area of the knee.

Once finished with the posteromedial compartment, the camera is withdrawn into the intracondylar notch. At this point, it may be possible to pass the arthroscope between the ACL and lateral femoral condyle into the posterolateral compartment. The knee should be held at 90 degrees of flexion. Often, the switching stick needs to be used to enter this compartment. It may be necessary to switch the scope back to the anterolateral portal prior to entering this space and passing the switching stick from the anteromedial portal into the posterolateral compartment. Once in the posterolateral compartment, a visual inspection of the posterior horn of the lateral meniscus, the meniscofemoral ligaments, and synovial folds can be carried out. With the camera facing the lateral condyle, it may be advanced toward the popliteal hiatus. With the knee flexed to 70 degrees and a valgus force applied, it is possible to trace the popliteus into the hiatus and view the femoral insertion of the tendon. Often, the space is too tight to view the tendon it its entirety and a posterolateral portal is needed.

Posterolateral Portal

As with the posteromedial side, the site for the posterolateral portal can be palpated prior to its creation. The knee is held at 90 degrees of flexion. The site for the portal is approximately 1 cm posterior to the lateral femoral condyle and 1 cm proximal to the joint line. The surgeon should be aware of the location of the biceps femoris and the common peroneal nerve when making this portal. As with the medial side, once the site is determined, an 18-gauge spinal needle is used to mark the portal and the skin incision is made. The portal is created bluntly using a mosquito or blunt trochar. As noted above, the arthroscope can be passed along the posterior aspect of the condyle and meniscus to the popliteal hiatus. Again, a probe may be brought in from the anteromedial portal and used to aid in the inspection of this compartment.

Accessory Anterior Medial and Lateral Portals

Accessory anterior portals may be needed, depending on the pathology encountered. The accessory medial and lateral portals are created under direct visualization. The accessory medial portal is more medial and inferior to the standard portal, whereas the accessory lateral portal is more lateral and inferior to the standard portal. The 18-gauge spinal needle is used to identify the proper track for the portal. It is essential to visualize the needle as it enters the joint to ensure that the portal will clear the menisci and articular cartilage. Once the proper track has been identified, the skin is incised with a no. 11 blade and the portal made with the blunt trochar. If necessary, a transpatellar portal can be made in similar fashion.

Once the case is finished, the knee is copiously irrigated through the arthroscope. A mechanical shaver, if used, can function as the outflow to remove any debris. The portal can be closed with simple nylon sutures or Steri-Strips, 3M (St. Paul, MN). The patient is placed in a dry, sterile compression dressing, extubated by anesthesia, and brought to the recovery room.

Rehabilitation

Rehabilitation will depend upon the pathology encountered during the case. Specific rehabilitation protocols will be outlined in later chapters. For the basic knee arthroscopy, the patient usually remains on crutches for 1–3 days. Early, active range of motion, quadriceps sets, and ankle pumps are all allowed immediately. Within the first week, the patient starts with a stationary bike and low-demand activities. Physical therapy may be employed to help mobilize the fluid around the knee and improve range of motion. In weeks 2–4, a return to normal, daily activities and low-impact activity is encouraged. After week 4, the patient may increase his or her activity, adding light jogging and other moderate-impact activity. Return to sports will hinge on the pathology dealt with at the time of surgery and the preparedness of the joint for the specific sport.

Complications

Although knee arthroscopy is a relatively straightforward procedure, it is still associated with surgical complications. In 1988, Small presented a study composed of complications gathered from the Arthroscopy Association of North America. In this study, 8,741 knee arthroscopies were reported. Of these cases, the overall complication rate was 1.68% [5, 6]. The list of complications included postoperative hemarthrosis (65% of all complications), infection, deep venous thrombosis, complex regional pain syndrome, iatrogenic injury, neurologic injury, and anesthesia complication [26]. Loss of motion and instrument breakage also have been reported [27–30]. Compartment syndrome has also been reported it the literature [31]. As in all instances with compartment syndrome, a high level of suspicion is necessary to make the diagnosis. Careful monitoring of the pump pressure and procedure time is necessary to prevent this relatively rare complication.

In 2006, Reigstad and Grinsgaard reported the complications for simple knee arthroscopy performed from 1999 through 2001 [32]: 876 arthroscopies on 785 patients were reviewed with 98% follow-up. The overall complication rate was 5%. Reigstad and Grinsgaard broke down these complications into two groups: those with therapeutic consequences (0.68%) and those without. The complications without significant therapeutic consequences consisted of preoperative bradycardia, asthmatic events, subcutaneous infusion of intravenous anesthetics, instrument breakage, conversion to arthrotomy, hemarthrosis, portal bleeding, temporary sensory loss, and postoperative pain. Significant complications consisted of two superficial infections, one venous thromboembolic event, and one return to surgery for scar tissue.

Deep Venous Thrombosis

In 2005, Ilahi et al. published a meta-analysis looking at the rate of deep venous thrombosis (DVT) after knee arthroscopy [33]. Ilahi et al. limited this analysis to those studies that were prospective, had no antithrombotic prophylaxis, had performed universal screening with an ultrasound or contrast venous venography, and were limited to unilateral knee arthroscopy with the exclusion of ligament or open surgery. The conclusion of this study was that unprophylaxed patients undergoing a routine knee arthroscopy have a 9.9% risk for a DVT and a 2.1% risk of a proximal DVT. In 2001, Delis et al. reported that patients were at a greater risk for DVT if they had two or more of the following risk factors: age > 65 years, obesity, smoking, female hormone replacement, venous insufficiency, prior history of DVT [34]. Others have published reports on the rate and the treatment of DVT in the ambulatory knee arthroscopy patient. Hoppener et al. performed a prospective cohort study on 335 patients undergoing knee arthroscopy [35]. Nineteen (6%) patients demonstrated a DVT by complete compression ultrasonography, with two symptomatic patients. One patient developed a nonfatal pulmonary embolism. Based on this data, Hoppener et al. did not recommend routine prophylaxis against DVT for the ambulatory knee arthroscopy patient. Michot et al. performed a similar-sized study looking at the effectiveness of treating patients with prophylaxis [36]. Michot et al. performed a prospective, single-blind study with 130 randomized patients in which the study group received low-molecular-weight heparin given perioperatively and then for 4 weeks and the control group received no prophylaxis. All were screened with compression ultrasonography. In the study group, 1 of 66 (1.5%) patients developed a DVT, whereas 10 of 64 (15.6%) patients in the control group had a positive DVT.

Currently, the authors do not use antithrombotic therapy for straightforward knee arthroscopy unless the patient has risk factors that increase the rate of DVT. These patients are evaluated individually and treated with either low-molecular-weight heparin for 2 weeks or aspirin and T.E.D, Covidien (Mansfield, Ma) stockings, depending on what the preoperative risk factors are.

Clinical Pearls/Summary

Currently, diagnostic knee arthroscopy is generally used as the starting point for more specialized surgery of the knee. The authors of the chapters that follow have included many tips and pearls that will aid in the treatment of various injuries to the knee. It is critical to plan for each aspect of your case. If you plan to perform other procedures beyond the diagnostic knee arthroscopy, be sure to set up your room and your patient appropriately. Preoperatively, make sure that the patient's nonoperative side is well protected and well padded. Be sure to place some reflex in the table to take the pressure off of the patient's low back and hip flexors. Be sure that the tourniquet cuff and the surgical leg holder are positioned appropriately. If either of these is too distal, the surgeon's ability to treat intraarticular pathology may be compromised. Check to make sure that the video screen is in the proper ocation and can be comfortably viewed from the foot of the bed. When performing knee arthroscopy without an assist, it is helpful to have the inflow connected to the lateral port and the outflow connected to the medial port. This allows the surgeon to drain the knee without crossing his hand over the arthroscope. During the case, a step stool under the surgeon's foot can aide the surgeon with balancing the surgical leg. At the end of the procedure, review the pictures obtained to be sure that you have captured the appropriate images before the arthroscope is taken off the field and contaminated.

Case Report

Case 1

Chief Complaint and Patient History: A 23-year-old man presented with a 4-month history of knee swelling and loss of motion. There was no history of trauma to the knee. His attention was drawn to his knee after having some difficulty during his regular running routine.

Physical Exam: On examination, he was found to have fullness over the medial aspect of his knee. This fullness was medial to the patella tendon and over the medial femoral condyle. His range of motion was intact, although he experienced tightness when he reached terminal flexion. There was no instability. His patella tracked well but rested in a slightly lateralized position when compared with that of the opposite knee.

Imaging: Radiographs of the knee were normal. An MRI scan was obtained, and it demonstrated what appeared to be an intraarticular mass (Fig. 15A).

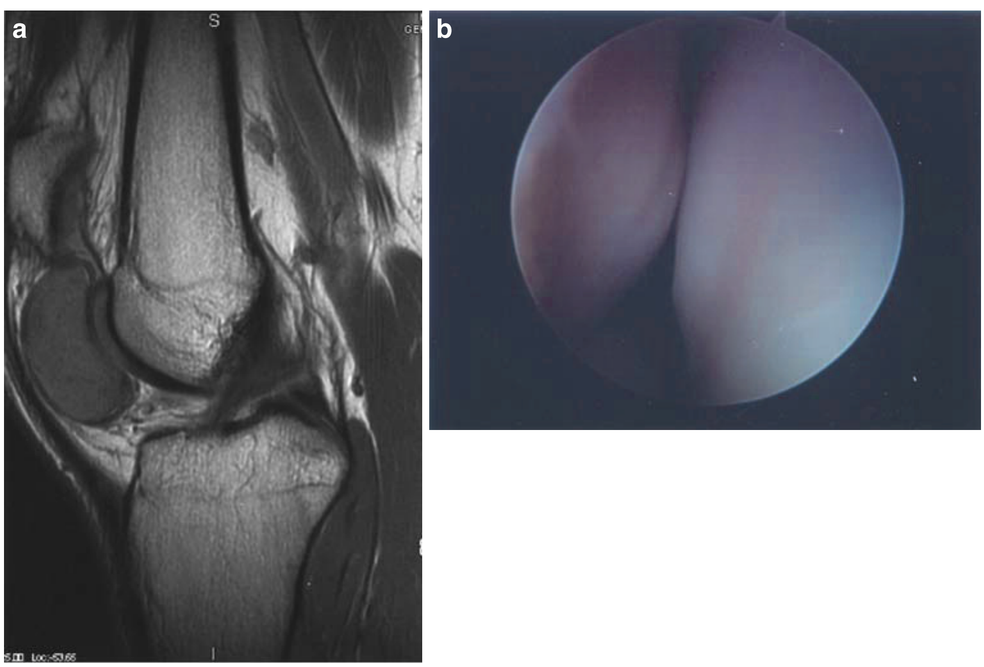


Fig. 15 Case 1. (**A**) MRI scan of giant cell tumor anterior to the medial compartment. (**B**) Arthroscopic view of extraarticular mass pushing against medial femoral condyle

Surgery/Treatment: The patient elected to proceed with a diagnostic arthroscopy and potential open excision of the mass. Knee arthroscopy revealed normal intraarticular anatomy (Fig. 15B). There was no evidence of an intraarticular mass or loose body. An incision was made using the medial portal, and a large, extraarticular mass was excised. Pathology study confirmed that the mass was a giant cell tumor of the patella tendon with negative margins. The patient returned to all activity after a short rehabilitation period.

References

1. Garrett WE, Swiontkowski MF, Weinstein JN, Callaghan J, Rosier RN, Berry DJ, Harrast J, Derosa GP. American Board of Orthopaedic Surgery Practice of the Orthopaedic Surgeon: Part II, Certification Examination Case Mix. *J Bone Joint Surg Am* 2006;88:660–667.
2. Kim TK, Savino RM, McFarland EG, Cosgarea AJ. Neurovascular complications of the knee. *Am J Sports Med* 2002;30:619–629.
3. McGinty JB, Johnson LL, Jackson RW, McBryde AM, Goodfellow JW. Uses and abuses of arthroscopy: a symposium. *J Bone Joint Surg Am* 1992;74:1563–1577.
4. Rodeo SA, Forester RA, Weiland AS. Neurologic complications due to arthroscopy. [Current Concepts Review]. *J Bone Joint Surg Am* 1993;75:917–926.
5. Small NC. Complications in arthroscopic surgery of the knee and shoulder. *Orthopedics* 1993;16:985–988.
6. Small NC. Complications in arthroscopic surgery performed by experienced arthroscopists. *Arthroscopy* 1988;4:215–221.
7. Wantanabe M, Takeda S, Ikeuchi H. *Atlas of Arthroscopy.* 2nd ed. Tokyo: Igaku Shoin Ltd., 1969.
8. Jackson RW, Abe I. The role of arthroscopy in the management of disorders of the knee. *J Bone Joint Surg Br* 1972;57:310.
9. Johnson LL. *Diagnostic and Surgical Arthroscopy: The Knee and Other Joints*. St. Louis, MO: CV Mosby, 1981.
10. Metcalf RW. An arthroscopic procedure for lateral release of the subluxing or dislocating patella. *Clin Orthop* 1982;667:9.
11. McGinty JB (ed.). *Operative Arthroscopy.* New York: Raven Press, 1996.
12. McGinty JB, Freedman PA. Arthroscopy of the knee. *Clin Orthop* 1976;121:173.
13. Gries P, Bandana D, Holstrom M, Burks RT. Meniscal injury: I. Basic science and evaluation. *J Am Acad Orthop Surg* 2002;10:168–176.
14. Vedi V, Spouse E, Williams A, Tennant JJ, Hunt D, Gedroyc W. Meniscal movement: an in-vivo study using dynamic MRI. *J Bone Joint Surg Br* 1999;81:37–41.
15. Cupte CM, Bull AM, Thomas RD, Amis AA. A review of the function and biomechanics of the meniscofemoral ligaments. *Arthroscopy* 2003;19:161–171.
16. Kusayama T, Harner CD, Carlin GJ, Xerogeanes JW, Smith BA. Anatomical and biomechanical characteristics of human meniscofemoral ligaments. *Knee Surg Sports Traumatol Arthrosc* 1994;2:234–237.
17. Wan AC, Felle P. The meniscofemoral ligaments. *Clin Anat* 1995;8:323–326.
18. Fitzgibbons RE, Shelbourne KD. Aggressive non-treatment of lateral meniscal tears seen during anterior cruciate ligament reconstruction. *Am J Sports Med* 1995;23:156–159.
19. Ekman EF, Wahba M, Ancona F. Analgesic efficacy of perioperative celecoxib in ambulatory knee surgery. *Arthroscopy* 2006;22:635–642.
20. Denti M, Randelli P, Bigoni M, Vitale G, Marino R, Fraschini N. Pre- and postoperative intra-articular analgesia for arthroscopic surgery of the knee and arthroscopic assisted anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc* 1997;5:206–212.
21. Miskulin M, Maldini B. Outpatient arthroscopic knee surgery under multimodal analgesic regiments. *Arthroscopy* 2006;22: 978–983.
22. Munk S, Pederson K. Knee arthroscopy under local anesthesia with or without anesthesiologic assistance. *Ugeskr Laeger* 1994;156:313–316.
23. Shapiro MS, Safran MR, Crockett H, Finerman GAM. Local anesthesia for knee arthroscopy: efficacy and cost benefits. *Am J Sports Med* 1996;23:50–53.
24. Jacobson E, Forssblad M, Rosenberg J, Westman L, Weidenhielm L. Can local anesthesia be recommended for routine use in elective knee arthroscopy? A comparison between local, spinal and general anesthesia. *Arthroscopy* 2000;16:183–190.
25. Fulkerson JP. Anteromedial tibial tubercle transfer. In Jackson DW (ed.), *Reconstructive Knee Surgery.* 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2008.
26. Deutch A, Wyzykowski RJ, Victoroff BN. Evaluation of the anatomy of the common peroneal nerve. Defining nerve at risk in arthroscopically assisted lateral meniscus repair. *Am J Sports Med* 1999;27:10–15.
27. Irrang JJ, Harner CD. Loss of motion following knee ligament reconstruction. *Sports Med* 1995;19:150.
28. Milankov M, Savic D, Miljkovic N. Broken blade in the knee: a complication of arthroscopic menisectomy. *Arthroscopy* 2002;18:E4.
29. Oldenburg M, Mueller RT. Intra-articular foreign body after arthroscopy. *Arthroscopy* 2003;19:1012–1014.
30. In Y, Bank WJ, Park JB. Detachment of the tip of a motorized shaver within the knee joint: a complication of arthroscopic surgery. *Arthroscopy* 2003;19:257.
31. Kaper BP, Carr CF, Shirreffs TG. Surgery of the knee: a report of two cases of compartment syndrome managed non-operatively. *Am J Sports Med* 1997;25:123–125.
32. Reigstad O, Grinsgaard C. Complications in knee arthroscopy. *Knee Surg Sports Traumatol Arthrosc* 2006;14:473–477.
33. Ilahi O, Reddy J, Ahmad I. Deep venous thrombosis after knee arthroscopy. *Arthroscopy* 2005;21:727–730.
34. Delis KT, Hunt N, Strachan RK, Nicolaides AM. Incidence, natural history and risk factors of deep venous thrombosis in elective knee arthroscopy. *Throm Haemost* 2001;86:817–821.
35. Hoppener M, Rettema HB, Henry CP, Verhegen C, Büller HR. Low incidence of deep venous thrombosis after knee arthroscopy without thromboprophylaxis. *Acta Orthop* 2006;77: 767–771.
36. Michot M, Conen D, Holt D, Erni D, Zumstein M, Ruflin G, Renner N. Prevention of deep venous thrombosis in ambulatory arthroscopic knee surgery. *Arthroscopy* 2002;18: 257–263.

Partial Meniscectomy

Laurie M. Katz and Paul P. Weitzel

Introduction

In 1897, the meniscus was described by Sutton as a functionless remnant of a leg muscle [1]. By 1936, King [2, 3] recognized that the meniscus actually plays a role in joint stability, lubrication, congruity, and chondroprotection. His early studies on canines were among the first to document degenerative changes in postmeniscectomy knees, as well as the limited healing potential of meniscus tears. Radiographic evidence of the consequences of total meniscectomy was presented by Fairbank [4] in 1948, when he described ridge formation, joint space narrowing, and flattening of the femoral condyles in patients who had undergone this procedure. We have since continued to improve our understanding of the biomechanical functions of the meniscus, as well as the long-term consequences of its removal. Treatment of meniscus pathology has therefore evolved to include repair when feasible or, if not feasible, a limited partial meniscectomy.

The incidence of acute symptomatic meniscus tears has been reported to be between 60 and 70 cases per 100,000 people [5–7]. It is therefore not surprising that arthroscopic surgery for their treatment has become one of the most common orthopaedic surgeries performed in the United States. In some centers, arthroscopic treatment for meniscus pathology constitutes 10% to 20% of all surgeries [8]. As shown by Laprade et al. [9] asymptomatic meniscus pathology also can occur. They performed a magnetic resonance imaging (MRI) evaluation of 54 painless knees and found a 5.6% prevalence of meniscal tears.

The cause of meniscus tears varies by age. A meniscus tear in the younger, athletic population is more likely due to trauma, whereas in the older population it is more likely due to degeneration. A cadaver study by Noble and Hamblen [10] found the prevalence of degenerative horizontal cleavage tears to be 60% in specimens with an average age of 65 years. Meniscus tears also show a male predominance, with a male:female ratio of at least 2.5:1 [11].

Meniscus tears do not always occur in isolation. A study by Poehling et al. [11] found that one third of patients with an anterior cruciate ligament (ACL) injury had a meniscus tear as well. In the subset of patients with an acute ACL tear, the lateral meniscus is more commonly injured. This is likely due to anterolateral rotatory translation of the tibia at the time of injury, which puts the lateral meniscus at risk. In contrast, medial meniscus tears are more common in patients with chronic ACL tears. This may be attributed to the function of the medial meniscus as a secondary restraint to anterior-posterior translation of the tibia. Increased stress is therefore placed on the medial meniscus in an ACL-deficient knee [12]. Other injuries associated with meniscus tears include tibial plateau fractures and femoral shaft fractures [13, 14].

Anatomy

Gross Anatomy

The menisci are two semicircular, fibrocartilaginous disks found between the femoral condyles and the tibial plateau (Fig. 1). The medial meniscus is crescent-shaped and approximately 3.5 cm in length. It can be divided into a posterior horn, a central body, and an anterior horn [15]. The posterior horn attaches to the tibia just anterior to the insertion site of the posterior cruciate ligament (PCL). The anterior horn can have a variable site of attachment, into either soft tissue or bone, but a firm bony attachment to the flat intercondylar region of the tibial plateau is most common (Fig. 2) [16]. The periphery of the medial meniscus is attached to the joint capsule and deep medial collateral ligament by means of the short coronary ligaments.

The lateral meniscus is more circular in shape, and, compared with the medial meniscus, it covers a larger surface

L.M. Katz (✉)
Department of Orthopaedics New England Baptist Hospital Boston Massachusetts

B.P. McKeon et al. (eds.), *Knee Arthroscopy*, DOI 10.1007/978-0-387-89504-8_2,

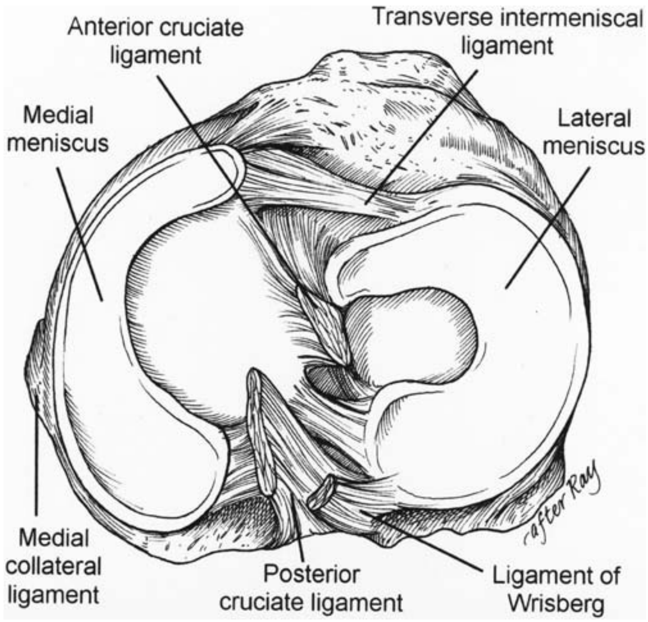


Fig. 1 Anatomy of the menisci as viewed from above. (From Pagnani MJ, Warren RF, Arnoczky SP, Wickiewicz TL. Anatomy of the knee. In Nicholas JA, Hershman E (eds.), *The Lower Extremity and Spine in Sports Medicine*, 2nd ed. St. Louis: Mosby, 1995, pp. 581–614. Copyright © 1995 by Elsevier. Reprinted with permission.)

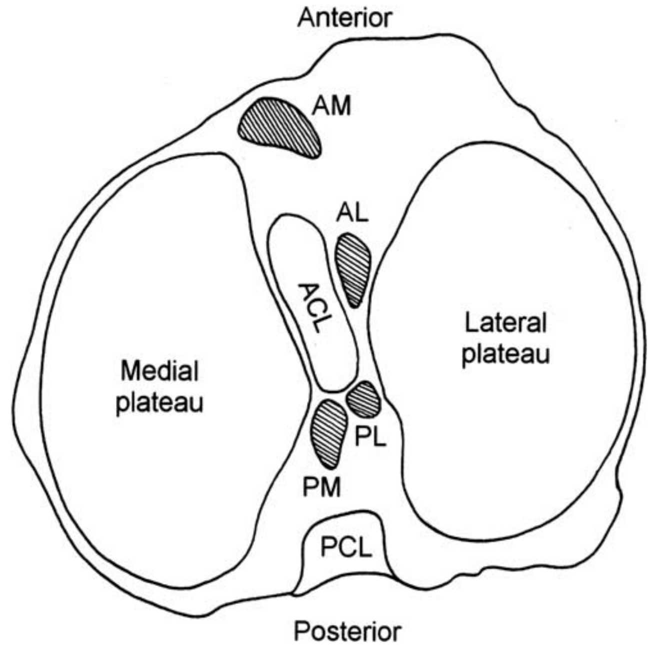


Fig. 2 Meniscus horn insertion sites as viewed from above. ACL, anterior cruciate ligament; AL, anterior horn lateral meniscus; AM, anterior horn medial meniscus; PCL, posterior cruciate ligament; PL, posterior horn lateral meniscus; PM, posterior horn medial meniscus. (From Johnson DL, Swenson TM, Livesay GA, Aizawa H, Fu FH, Harner CD. Insertion site anatomy of the human menisci: gross, arthroscopic and topographical anatomy as a basis for meniscal transplantation. *Arthroscopy* 1995;11:386–394. Copyright © 1995 by the Arthroscopy Association of America. Reprinted with permission from Elsevier.)

area of the tibial plateau. The anterior horn inserts on the tibia in front of the intercondylar eminence, just posterior and lateral to the ACL insertion. The posterior horn attaches to the tibia in between the insertion sites of the PCL and the posterior horn of the medial meniscus (Fig. 2). In some individuals, fibers from the posterior horn of the lateral meniscus extend to insert on the lateral aspect of the medial femoral condyle in the intercondylar notch. The fibers that pass anterior to the PCL are referred to as the ligament of Humphrey, whereas those that pass posterior to the PCL are referred to as the ligament of Wrisberg.

Studies using polarized light and electron microscopy, as well as x-ray diffraction, have detailed the ultrastructure of the menisci. The majority of the collagen fibers are arranged circumferentially. This allows the creation of hoop stresses in an effort to resist compressive loads. Midsubstance and some surface fibers are arranged in a radial fashion, acting as tie fibers to resist longitudinal splitting. Several of these radial fibers assume a woven pattern near the surface of the meniscus, which may help distribute sheer stress [17, 18].

The discoid lateral meniscus is a variant that warrants discussion. The true incidence of this variant is unknown, although it is cited to be approximately 4% to 5%, with a higher incidence in Asian populations [19, 20]. The discoid lateral meniscus has been classified by Watanabe et al. [21], who described three types based on arthroscopic appearance. Discoid lateral menisci with stable tibial attachments are classified as complete (type I) or incomplete (type II). Type I discoid lateral menisci cover the entire lateral tibial plateau, whereas type II covers more than a normal meniscus but not the entire lateral plateau. Type III, the Wrisberg type, lacks posterior capsular attachments with the exception of the meniscofemoral ligament of Wrisberg.

Vascular Anatomy

The blood supply to the meniscus originates from the superior and inferior branches of the medial and lateral genicular arteries. These vessels form a perimeniscal capillary plexus within the synovial and capsular tissues of the knee joint. This plexus supplies the peripheral 10% to 30% of the medial meniscus and 10% to 25% of the lateral meniscus. The remainder of the meniscus, as well as the area of the popliteal hiatus of the lateral meniscus, is avascular [22].

The vascular anatomy of the meniscus is a key factor in its ability to heal tears, as it determines the availability of inflammatory mediators and cellular elements [22]. The vascular periphery of the meniscus is referred to as the red-red zone. Tears in this area have a high likelihood of healing due to the abundant blood supply. The avascular part of the meniscus is referred to as the white-white zone. Tears in this area have a much lower probability of healing, due to a lack of blood supply. Tears that occur between these two areas are said to occur in the red-white zone. The outcome of tears that occur in the red-white zone is less predictable [23].

Neuroanatomy

Neuroanatomy studies of the human meniscus have shown that the anterior and posterior horns are richly innervated, whereas only the peripheral one third to two thirds of the body is innervated [24–27]. This neural distribution, which is similar to that of the blood supply, has led to the description of a vasomotor function. But this nerve distribution is not exclusively perivascular, indicating other roles for the nerve endings, including proprioception and sensory feedback [24, 25]. Both immunohistologic and clinical studies have confirmed this theory, including a study by Dye et al. [28], where neurosensory mapping of the knee was performed. Upon probing, the central rim of the meniscus gave a nonpainful awareness, whereas the more peripheral tissue, capsular margin, and anterior and posterior horns caused a slight to moderate discomfort. Additional studies are needed to further describe the proprioceptive and sensory feedback roles of the menisci.

Biomechanics

The menisci serve many important biomechanical functions for the knee joint. They contribute to load transmission, joint stability, and proprioception. They also serve to decrease contact stresses and increase contact area and congruity of the knee. With ambulation, the forces across the knee joint average between three and six times body weight. The actual amount of force that is transmitted through the menisci varies based on the degree of knee flexion. Ahmed and Burke [29] showed that at full extension, 50% of the forces across the knee are borne by the menisci. At 90 degrees of flexion, this figure increases to 85%.

The biomechanical changes across the knee joint when meniscal pathology is present have been investigated and reported in the literature. Radin et al. [30] found that a longitudinal tear in the red-white zone resulted in no increase in the magnitude of stress across the joint. Partial medial meniscectomy resulted in a modest increase, and total medial meniscectomy resulted in a three- to fivefold increase. The distribution area of the stress was adequately maintained until the entire meniscus was removed. In 1986, Baratz et al. [31] showed a minimal change in contact area and an approximately 16% increase in contact stresses when a peripheral meniscus tear was present.

Quantitative results after partial and total medial meniscectomy have also been reported. Partial medial meniscectomy decreases contact area by 10% to 20% and increases contact stresses by 40% to 70%. Complete meniscectomy decreases contact area by 40% to 75% and increases contact stresses by 136% to 236% [29, 31–33]. In addition, the shock-absorbing capacity of the knee has been found to decrease by 20% after total meniscectomy [34]. In general, the biomechanical changes are less severe when a partial meniscectomy is performed compared with those of a total meniscectomy [29, 32, 33, 35]. At a minimum, the rim of the meniscus should be preserved to allow maintenance of hoop stresses [36, 37].

The abnormally high stresses seen after a meniscectomy eventually translate into clinical changes. Fairbank [38] was the first to describe the radiographic changes seen after meniscectomy. These changes were confirmed by Roos et al. [39] in a follow-up study of 107 post–total meniscectomy patients using age-matched controls. The relative risk for advanced radiographic degenerative changes in the patients who had undergone a total meniscectomy was significant. Radiographic changes also have been noted after partial meniscectomy but are less severe compared with those of total or subtotal meniscectomy [40, 41]. Cartilage changes after meniscectomy have been quantified as well. Cicuttini et al. [42] reported a 6.9% per year increase in cartilage loss in patients who underwent partial meniscectomy compared with that in control knees.

Tear Anatomy

Meniscal tears are most commonly classified according to tear pattern (Fig. 3). Tears can be described as vertical longitudinal, oblique, complex, radial, or horizontal [43, 44]. Metcalf [43] reported his experience with meniscal tears and found that 80% of tears are either vertical longitudinal, or oblique. The medial meniscus was involved in 69% of cases, the lateral meniscus in 24%, and both menisci in 7%. The most common location for tears was in the posterior one half of the meniscus. There was an increased frequency of degenerative tears with advancing age.

Metcalf and Greis and colleagues [43–45] have further described the characteristics of different tear patterns. Vertical longitudinal tears, which most commonly occur in younger patients, are usually seen in the posterior horn.

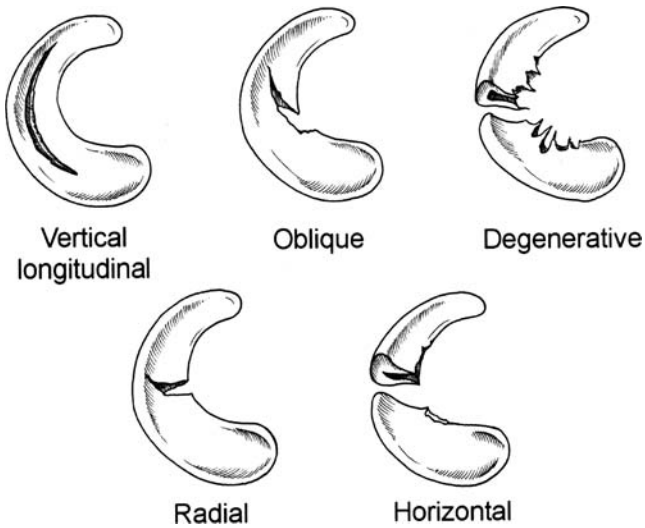


Fig. 3 Classification of meniscal tears. (From Ciccotti MG, Shields CL Jr, El Attrache NS. Meniscectomy. In Fu FH (ed.), *Knee Surgery*. Baltimore: Williams & Wilkins, 1994, pp. 591–613. Reprinted with permission of Wolters Kluwer.)

If the tear propagates into the anterior horn, it creates a bucket-handle tear. Unstable bucket-handle tears frequently cause mechanical symptoms. If the central portion of a bucket-handle tear displaces into the joint, a locked knee may result. Vertical longitudinal tears are associated with ACL injuries. They are also more common in the medial meniscus, likely due to its sturdy attachments to the tibial plateau, capsule, and deep medial collateral ligament [44].

Oblique tears are sometimes referred to as flap or parrot-beak tears. They are most commonly found at the junction of the posterior and middle third of the meniscus and cause mechanical symptoms when the torn edge catches between the femur and tibia [44]. Complex tears are associated with older patients and are commonly found in knees with degenerative changes. They occur mainly in the posterior horn and midportion of the body. The complex free fragments can lead to mechanical symptoms [45].

Radial tears are commonly located at the junction of the posterior and middle thirds of the medial meniscus, in the middle third of the medial meniscus, or near the posterior attachment of the lateral meniscus. If these tears propagate through the periphery of the meniscus, they cause a loss of hoop stresses and therefore a loss of the ability for meniscal load transmission [44]. Radial tears of the lateral meniscus are common in acute ACL injuries [12].

Horizontal cleavage tears begin near the inner edge of the meniscus and propagate in a horizontal plane toward the periphery. Their incidence increases with age, and they are associated with meniscal cysts [44].

Indications

History

When evaluating a patient with a suspected meniscus tear, begin with a thorough history. Important information to obtain includes the mechanism of injury, symptoms, and exacerbating factors. Up to 95% of patients will recall a specific injury [46, 47]. One particular exception to this is in the degenerative knee, where symptoms may be insidious in onset. The mechanism of injury resulting in a meniscus tear is often a twisting injury, with the knee flexed and the foot planted. Alternatively, a hyperflexion episode may be recalled [45].

Symptoms include pain, swelling, loss of motion, and mechanical symptoms such as catching or clicking. With an isolated meniscus tear, swelling may be delayed a few days, in contrast with tears that occur in the setting of an ACL injury, where swelling is usually immediate [48]. Patients who have sustained a displaced bucket-handle tear may complain of locking, with an inability to extend their knees [47].

Physical Exam

Examination of the knee should begin with an inspection of the injured side compared with the uninjured side. Look for evidence of an effusion and quadriceps atrophy. Next, evaluate the range of motion of the knee to determine if there is a mechanical block to flexion or extension. Palpation of the medial and lateral joint lines can assess for tenderness as well as swelling associated with a meniscal cyst. A ligament exam should be performed to evaluate the integrity of the collateral and cruciate ligaments. Finally, specialized tests designed to elicit symptoms of a torn meniscus may be performed. Aside from joint-line palpation, described maneuvers include the squat test [48], the McMurray test, the Apley grinding test, and others. Joint-line tenderness appears to be the best clinical indicator of a meniscal tear, with sensitivity reported to be as high as 75% to 92% [49–53]. Sensitivity decreases when an acute ACL tear is present [54].

Diagnostic Studies

Plain radiographs should be obtained, consisting of a posteroanterior 45-degree flexion weight-bearing view of both knees [55], a lateral view, and a sunrise view. These allow evaluation of the joint spaces as well as the bony structure. MRI is also valuable in the workup of a meniscus tear.

Accuracy of MRI in the diagnosis of a meniscus tear is reported in the literature as being between 68% and 100% [56, 57].

Surgical Decision Making

Treatment for a meniscus tear is undertaken in patients whose symptoms interfere with their activities of daily living, ability to participate in athletics, or ability to work. In the active individual with an acute injury, mechanical symptoms, and no evidence of degenerative changes, operative intervention is recommended. Older individuals with a mildly degenerative knee who can recall an acute event associated with the onset of symptoms and have focal pain as well as mechanical symptoms are also candidates for early surgical intervention.

Nonoperative treatment, including activity modification and physical therapy, is an option that should at least be discussed with patients with meniscal tears. An initial course of nonoperative treatment is usually recommended for an older individual with an insidious onset of symptoms in a degenerative knee without mechanical symptoms. We have found that this patient population has a significant likelihood of improvement with quadriceps strengthening exercises so as to make nonoperative treatment a worthwhile recommendation [58].

Surgery

Once a meniscus tear is confirmed at arthroscopy, the appropriate treatment must be determined. A meniscus tear may be treated by meniscectomy, repair, synovial abrasion/trephination, or observation. Overly aggressive removal of meniscus tissue can have deleterious effects on the knee. Therefore, it is beneficial to preserve as much meniscus as possible.

When evaluating a meniscus tear, meniscal stability should be determined. Tears are considered stable if they are partial thickness, less than 1 cm in length, or if the central portion of a vertical longitudinal tear cannot be displaced more than 3 mm from the intact peripheral rim [59, 60]. Stable tears may respond to conservative treatment as well as to abrasion or trephination, and these options should be considered [45]. The ability to repair the tear must also be evaluated. Tears are generally believed to be irreparable if they are complete tears with an oblique, radial, horizontal cleavage, or complex degenerative pattern, or if they occur in the white-white zone of the meniscus [60]. These irreparable tears are the ones suited for partial meniscectomy. Of note: The indications for meniscus repair are occasionally extended to the white-white zone in patients undergoing concomitant ACL reconstruction or in young patients with the use of an exogenous fibrin clot to enhance healing capability [61, 62].

Occasionally, a meniscus tear is noted incidentally during arthroscopy. There are instances where these tears may be left untreated. Radin et al. [30] suggested that nondisplaced, nonpainful tears of the meniscus should not be an indication for meniscectomy, as retention of a torn meniscus did not have a deleterious effect on the magnitude or distribution of the stresses in the knee. In addition, Weiss et al. [59] found that 17 of 26 stable vertical longitudinal tears in the peripheral portion of the meniscus had healed at a second-look arthroscopy. It was recommended that these tears be left alone unless they are the only abnormality found and can explain the patient's symptoms. Shelbourne et al. [63] also have noted that lateral meniscus tears that are entirely posterior to the popliteus hiatus are not usually symptomatic. It was recommended that these tears be treated in situ with abrasion and trephination. In contrast, an incidental tear that is clearly unstable should be treated with either repair or partial meniscectomy as indicated. This can occur in a patient whose exam may have been limited due to a ligamentous injury.

Technique

Knee arthroscopy with partial meniscectomy can be performed under general or regional anesthesia or alternatively using local anesthesia combined with monitored anesthesia care (MAC). Perioperative antibiotics are administered, consisting of cefazolin or, in the case of a documented penicillin or cephalosporin allergy, clindamycin or vancomycin. Once the patient is anesthetized, the knee is injected with 30 to 60 mL of 1% lidocaine with epinephrine 1:100,000. This is believed to assist with both postoperative pain control and hemostasis during the procedure.

The patient is placed supine on the operating room table. A tourniquet is applied to the thigh, although we generally do not inflate unless excessive bleeding is encountered. A thigh holder or lateral post is used to allow for controlled varus and valgus stress. The patient may be positioned so that the leg can be draped off the side of the bed if using a lateral post or the end of the bed can be lowered when using a thigh holder.

With the knee flexed to approximately 70 degrees, the lateral portal is made first. We use a vertical incision for this portal. This orientation allows the portal to be close to the patellar tendon without placing the fibers at risk during the incision. When the medial portal is created, a horizontal incision is used to minimize the risk of injury to the infrapatellar branch of the saphenous nerve and the medial meniscus. If the suspected tear is in the medial meniscus, making the

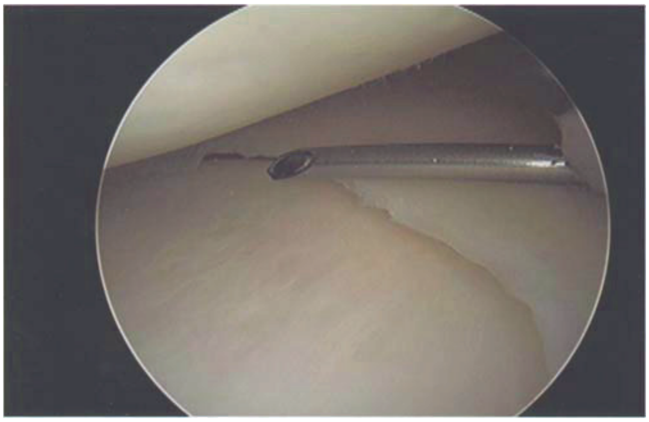


Fig. 4 Spinal needle to place low medial portal to facilitate medial meniscectomy

medial incision lower and more medial can improve access to the tear (Fig. 4). Access to lateral meniscal tears is facilitated by making the medial incision higher and closer to the patellar tendon. This position helps to maneuver instruments over the tibial eminence.

While viewing both the medial and lateral compartments, the menisci should be thoroughly evaluated for evidence of a tear. An angled probe is used to examine the entire undersurface and oversurface of both menisci to avoid missing a flipped fragment. Evaluate the stability of the rim by gently attempting to displace the meniscus into the notch with the use of the probe.

Once a tear is identified and determined to be irreparable, a partial meniscectomy is performed. Metcalf [43] described basic principles to adhere to when performing this procedure:

1. Remove all mobile fragments.
2. Do not leave sudden changes in rim contour.
3. Do not try to obtain a perfectly smooth rim as some remodeling may occur [64].
4. Use the probe often to reevaluate the tear.
5. Protect the meniscus-capsular junction to avoid the loss of hoop stresses.
6. Use both manual and motorized instruments to maximize efficiency.
7. When uncertain if an area should be resected, err on the side of leaving more meniscus intact rather than compromising biomechanical properties.

In addition to these general principles, there are techniques that can be used to address the specific tear configurations. A vertical longitudinal tear that is limited to the posterior portion of the meniscus can be resected in a piecemeal fashion. Resection of the meniscus tissue that is central to the tear is initiated with a manual cutting instrument. These instruments come in a variety of designs – including up, down, left, and right cutters – to accommodate different angles. A motorized shaver is then used to resect any remaining frayed edges. The area that has been resected should be contoured to the remaining meniscus to avoid any sharp edges.

When the vertical longitudinal tear propagates into the anterior horn, a bucket-handle tear is created (Fig. 5). A displaced bucket-handle tear should first be reduced to improve visualization. The anterior horn attachment of the torn meniscus is then partially transected using a manual cutter until it is connected by a wisp of tissue. This wisp of tissue prevents the torn fragment from flipping into the posterior compartment during resection of the posterior horn. During this step, an accessory portal may be used to place tension on the anterior horn. Next, the posterior horn attachment of the torn meniscus is resected. Attention is then turned back to the anterior horn. The remaining fibers can either be transected with a manual cutter or be detached by grasping the torn fragment and twisting it until the fibers separate. The fragment is then pulled out of the joint through the portal, and a motorized shaver is used to contour the resected edges.

Oblique tears result in torn fragments that require resection. The torn fragments are removed with either a manual cutter or a motorized shaver. The resection is complete when there are no sudden changes in the contour of the meniscus. Oblique tears may result in a large flap that can flip underneath the meniscus and easily be missed. Be suspicious of this type of tear if the inner edge of the meniscus looks blunt rather than having the usual tapered appearance (Fig. 6). A probe can be used to bring this flap into view (Fig. 7), and the resection should proceed as described.

Complex tears often involve multiple tissue planes and should be thoroughly probed to define their extent [65]. When these tears occur in a younger individual, careful consideration should be given to their capacity for repair, as resection often removes a large amount of tissue. When

Fig. 5 Bucket-handle tear flipped into notch.

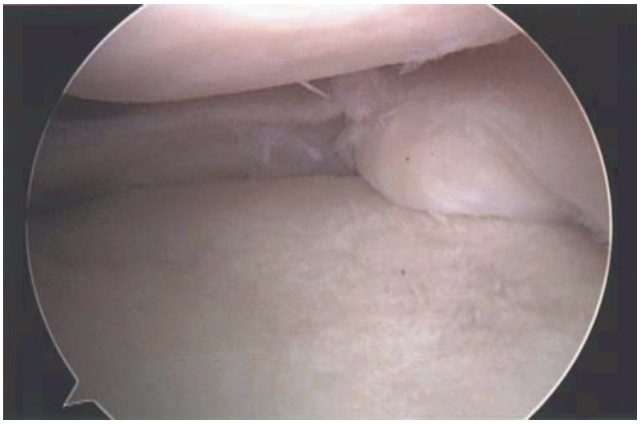


Fig. 6 An oblique tear with a blunted inner edge suggesting a flap tear that is flipped underneath.

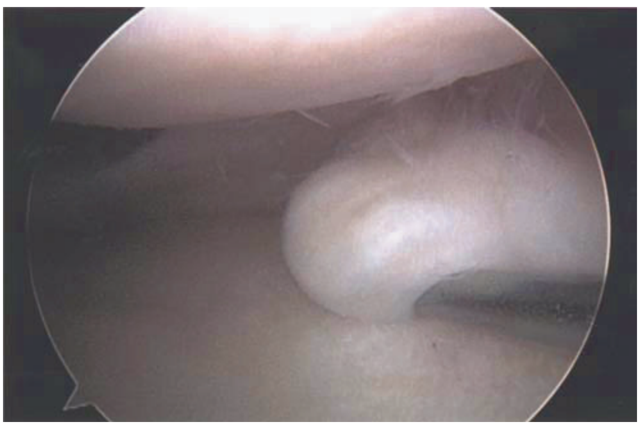


Fig. 7 The flap tear brought out from under the meniscus using an angled probe. This illustrates the extent of the tear that may be hidden when first viewed.

deemed irreparable or when occurring in the degenerative knee, complex tears should be treated with partial meniscectomy. A manual cutter can be used to remove any large fragments, and a motorized shaver can be used for contouring. A motorized shaver works well for these tears because the tissue is often friable. Be cautious to leave the rim intact to preserve meniscal function.

Radial tears are treated by resecting the corner edges with a manual cutter. The edges should be resected back to the depth of the tear. A motorized shaver is then used to contour the resected area so that a smooth transition remains.

The horizontal tear involves two leaves of tissue created by a cleavage plane. When the cleavage plane is shallow, both leaves may be resected back to a stable edge. When the cleavage plane extends deep toward the capsule, the two leaves should be evaluated to determine which one appears more stable. The more stable leaf should be preserved, and the less stable leaf should be resected. If both leaves are unstable, both should be resected back toward the rim to prevent the persistence of symptoms.

An asymptomatic discoid meniscus that is an incidental finding should be left alone. When the discoid meniscus has a symptomatic tear or is unstable, partial meniscectomy should be performed [66]. These resections are challenging because of the increased thickness and size of the meniscus [67]. Ikeuchi [67] described his technique for treating tears of the discoid meniscus. For L-shaped tears or flap tears, a grasper was used through an accessory far-lateral portal to tension the flap anteriorly. A cutting instrument was then used to resect the torn fragment. Bucket-handle tears of the discoid meniscus are resected using a technique similar to that described above for this tear pattern. For unstable discoid menisci without a tear, the goal of treatment is to resect the central portion until the remaining rim is the width of a normal meniscus. This can be performed in a piecemeal fashion or in one piece with an arthroscopic knife [65].

Outcome Studies

Functional outcomes after partial versus total meniscectomy have been evaluated [41, 68–71]. Hede et al. [70], in a prospective randomized comparison, described the long-term outcome of patients undergoing partial versus total meniscectomy. Knee function was found to be inversely related to the extent of tissue resection. This correlation has been supported by Englund et al. [71] and Northmore-Ball et al. [69].

Several studies have looked specifically at the short-term and long-term clinical and functional outcomes after partial meniscectomy [58, 72–86]. Osti et al. [76] showed an 85% excellent or good result and 98% return to full sports activities in 41 athletes with partial lateral meniscectomies at a 3-year average follow-up. Schimmer et al. [80] reported an excellent or good result in 91.7% of patients at an average of 4 years after partial meniscectomy; 77% returned to their baseline sports activities. Unfortunately, at a 12-year follow-up, only 78.1% maintained excellent or good results. Similarly, Jaureguito et al. [77] noted 92% excellent or good results 5 months to 2 years after partial lateral meniscectomy, with 85% of patients returning to their baseline level of activity. By 8 years, only 62% maintained excellent or good functional results, and only 48% were able to continue at their baseline level of activity. Long-term studies [74, 75, 77–86] have shown excellent or good results in 58% to 96% of patients at an average follow-up of 7–14.7 years. Overall, studies confirm that partial meniscectomy is beneficial to patients, with some deterioration in functional outcome noted over time.

The presence or absence of preexisting degenerative changes is a factor to consider when evaluating a patient for a partial meniscectomy. Several authors have demonstrated an inferior functional outcome in patients who had preexisting degenerative changes at the time of partial meniscectomy [78, 80, 81]. Schimmer et al. [80] reported that the factor with the highest impact on long-term results was damage to the articular cartilage. At a 12-year follow-up, 94.8% of patients with a history of an isolated meniscal tear and no articular damage at the time of surgery rated their results as excellent or good compared with only 62% of patients with articular damage present. It should be noted, though, that at the initial 4-year follow-up, 90% of the patients with articular damage described excellent to good results. Barrett et al. [81], Matsusue and Thomson [78], and Bonamo et al. [73] also reported less favorable results in patients with grade III or IV articular changes yet believed that the increased satisfaction rate and the ability for some patients to resume athletic activities warranted the procedure in this population. This is in contrast with the findings of Herrlin et al. [58], who found no difference in outcome in degenerative meniscus tears treated with arthroscopy and rehabilitation versus rehabilitation alone. It is the authors' opinion that partial meniscectomy is indicated in a knee with degenerative changes when the patient has had an acute onset of symptoms, has focal pain, and has mechanical symptoms that are consistent with meniscal pathology.

Inferior results have been documented after lateral meniscectomy compared with those after medial meniscectomy [87–89]. McNicholas et al. [87] conducted a 30-year prospective, longitudinal review of 95 adolescents who underwent a total meniscectomy. After medial meniscectomy, 80% of the patients had good or excellent results long-term, whereas only 47% of the patients had similar results after lateral total meniscectomy. Lateral meniscectomy may also to lead to an increased risk of radiographic degeneration [88, 89]. These correlations may be explained by the finding that the lateral meniscus carries a higher percentage of the load transmitted through its compartment than does the medial meniscus [90].

The consequences of meniscectomy may be exacerbated in patients with ACL insufficiency [46, 79, 91–93]. Burks et al. [79] showed that, after 14.7 years, patients who underwent partial meniscectomy in an ACL-deficient knee had significantly worse satisfaction scores and advanced radiographic changes compared with those of patients who underwent a partial meniscectomy with an intact ACL. Sherman et al. [92] looked at ACL-deficient knees with and without meniscectomy. Inferior radiographic scores were seen in the group that underwent meniscectomy. A combination of increased contact stresses from the meniscectomy and increased instability from the ligament injury has been offered as an explanation of these findings [91].

Factors such as age [46, 73, 74, 79, 82, 83, 88, 94, 95], gender [46, 73, 74, 79, 82, 83, 88, 94–96], and weight [73, 85] have also been evaluated in patients who have undergone meniscectomy. Unfortunately, there have been conflicting findings in the literature regarding the association of these factors with outcome measures.

Rehabilitation

Patients are allowed to bear weight as tolerated immediately after partial meniscectomy. Crutches are usually required for 2–5 days, until the patient is able to fully weight-bear without discomfort. Patients are instructed on performing quadriceps strengthening and range of motion exercises without restrictions. The majority of patients are able to perform these exercises on their own and do not require formal physical therapy. Patients are allowed to return to full athletic activities when their quadriceps muscle tone returns and they have full painless range of motion. This varies but usually averages 4 to 6 weeks postoperatively and slightly longer in the setting of degenerative changes.

Complications

Complications related to arthroscopic partial meniscectomy can be broken down into those related to knee arthroscopy in general and those specifically associated with partial meniscectomy. Small [97] reported on the complications of 21 experienced arthroscopists over a 19-month period. The complication rates for arthroscopic partial medial and lateral meniscectomies were 1.78% and 1.48%, respectively.

Aside from the general complications of knee arthroscopy, which are covered elsewhere in this text, partial meniscectomy can be complicated by instrument failure, knee ligament injury, neurovascular injury, iatrogenic damage to articular cartilage [65], and persistent pain [45]. In a review by the Arthroscopy Association of North America [98], instrument failure represented 18% of all arthroscopic complications. Small, in his later study, found instrument failure to represent only 2.9% of all arthroscopic complications [97]. This decreased incidence of failure may be due to enhanced design and quality control, as well as to an improvement in surgeon skill [99].

Knee ligament injury during partial meniscectomy usually involves the medial collateral ligament [97]. This infrequent injury may occur when excessive valgus force is placed on the knee in an attempt to gain better access to the medial compartment. Neurovascular injury, also an infrequent complication, can occur from penetration of the posterior capsule with sharp instruments [98]. Iatrogenic damage to articular cartilage may take place during forceful insertion of instruments, which can gouge the articular surface. Levering on the articular cartilage to access a difficult area of the meniscus also may lead to injury.

Persistent pain after partial meniscectomy may occur because of incomplete resection of the tear or from coexistent knee pathology. When resecting a tear, in an attempt to preserve the maximal amount of menisci, it is possible that torn remnants may be left behind. This complication can be avoided if the surgeon observes the previously noted principles described by Metcalf to ensure proper treatment of the tear. Pain due to coexistent knee pathology is often associated with degenerative changes. Patients with degenerative changes of the knee who undergo partial meniscectomy have inferior results to those of patients who do not have degenerative changes [78, 80, 81]. They also may continue to have mechanical symptoms from an unstable articular cartilage lesion rather than from the meniscus tear [45]. Having the patient focus on quadriceps strengthening can reduce his or her symptoms.

Clinical Pearls

1. Once anesthesia is induced, inject the knee with 1% lidocaine with epinephrine 1:100,000. This will provide preoperative analgesia as well as help with hemostasis during the procedure. This practice has eliminated the authors' use of a tourniquet during isolated partial meniscectomy and therefore avoids the risk of denervation and decreased functional capacity [100]. A tourniquet should still be in place should unexpected bleeding be encountered.
2. For easier excursion of instruments, change the location of the medial portal based on the expected pathology. For medial meniscal tears, make the medial portal lower and more medial. For lateral meniscal tears, make the medial portal higher and closer to the patellar tendon.
3. For resection of a bucket-handle or discoid meniscus, consider using an accessory portal to provide traction on the fragment while it is excised.
4. Always probe along the entire superior and inferior surfaces of the meniscus to ensure that there are no missed vertical longitudinal tears or oblique flap tears that have flipped underneath.
5. If there is difficulty viewing or resecting a section of the meniscus, consider switching the camera and working portal to obtain a different angle. This is especially helpful for tears of the anterior horn and body of the medial meniscus. Using the instrumentation from the lateral portal allows more efficient access to contouring these tears.

Summary

Meniscus tears can result in pain as well as mechanical symptoms. Through a careful history and physical exam, an accurate diagnosis can be obtained. When the diagnosis is less clear, supplemental diagnostic tests, such as MRI, are available. Surgical intervention is warranted for symptomatic tears that are either not responsive or not conducive to conservative treatment. Partial meniscectomy is the treatment of choice when repair is not feasible. Given the biomechanical consequences of removing meniscus tissue, the goal should be to obtain a stable rim, resecting only the necessary fragments.

Case Reports

Case 1

Chief Complaint and Patient History: A 43-year-old man was playing tennis and developed acute knee pain and decreased range of motion.

Physical Exam: On examination, medial joint-line pain, stable ligament exam, and medial joint-line tenderness were noted. Range of motion was 15 degrees (extension) to 90 degrees (flexion).

Imaging: Radiographs: negative. MRI scan showed buckle-handle medial meniscal tear.

Surgery/Treatment: At arthroscopy, a nonreparable medial meniscal tear was noted (Fig. 8A). Anterior horn was released nearly completely, posterior horn was released, anterior horn was completed, and fragment was removed (Fig. 8B–D).

Discussion: To avoid the bucket-handle fragment flipping into the posterior recess, the anterior horn is not released until after the posterior horn is released. Leaving the anterior horn attached by a small amount allows tension on the meniscus while releasing the posterior horn.

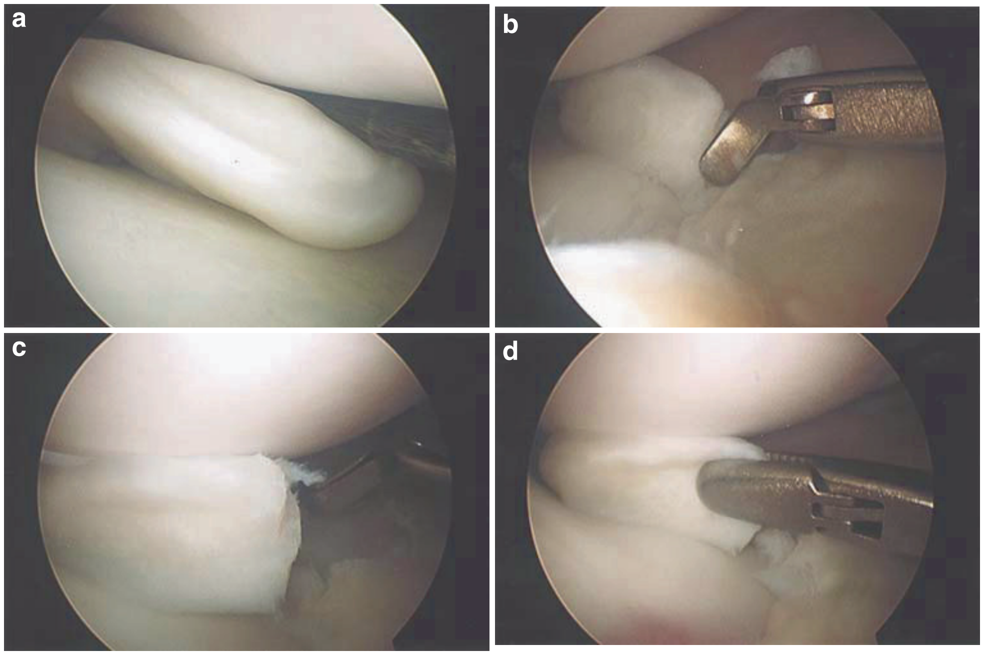


Fig. 8 Case 1. (**A**) Unstable nonreparable bucket-handle tear. (**B**) Anterior horn resected leaving a wisp of tissue to maintain tension for posterior resection and to avoid fragment from flipping into posterior knee. (**C**) Anterior horn resection completed after posterior horn released. (**D**) Removing bucket-handle tear.

Case 2

Chief Complaint and Patient History: A 54-year-old woman who teaches spin and yoga twists her knee and experiences knee pain. She has intermittent locking and pain.

Physical Exam: On examination, medial joint-line tenderness, stable ligament exam, and pain and popping with McMurray testing are noted. Range of motion was 0 degrees (extension) to 120 degrees (flexion).

Imaging: Radiographs show mild degenerative changes. MRI scan shows medial meniscal tear.

Surgery/Treatment: At arthroscopy, a medial meniscal tear was noted with flipped fragment (Fig. 9A, B). Medial meniscectomy is performed.

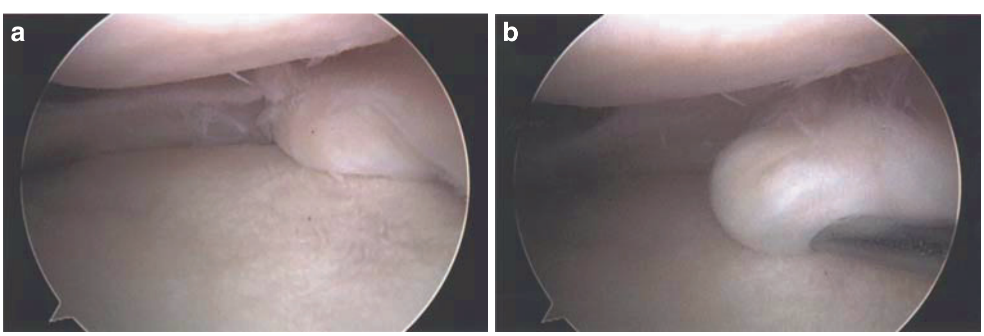


Fig. 9 Case 2. (**A**) Rolled meniscal fragment with smooth edge. (**B**) Fragment reduced from underneath to reveal large tear.

Discussion: Flipped fragments at first glance can appear like normal anatomy. A rolled meniscal edge indicates abnormal anatomy. Always probe the inferior and superior surfaces of the meniscus in both compartments and after meniscectomy to ensure no missed fragment.

References

1. Sutton JB. *Ligaments: Their Nature and Morphology*. London: MK Lewis, 1897.
2. King D. The healing of semilunar cartilages. *J Bone Joint Surg* 1936;18:333–342.
3. King D. The function of semilunar cartilages. *J Bone Joint Surg* 1936;18:1069–1076.
4. Fairbank TJ. Knee joint changes after meniscectomy. *J Bone Joint Surg Br* 1948;30:664–670.
5. Baker BE, Peckham AC, Pupparo F, Sanborn JC. Review of meniscal injury and associated sports. *Am J Sports Med* 1985;13:1–4.
6. Hede A, Jensen DB, Blyme P, Sonne-Holm S. Epidemiology of meniscal lesions in the knee: 1,215 open operations in Copenhagen 1982–84. *Acta Orthop Scand* 1990;6:435–437.
7. Nielsen AB, Yde J. Epidemiology of acute knee injuries: a prospective hospital investigation. *J Traum* 1991;31:1644–1648.
8. Renstrom P, Johnson RJ. Anatomy and biomechanics of the menisci. *Clin Sports Med* 1990;9:523–533.
9. LaPrade RF, Burnett QM II, Veenstra MA, Hodgman CG. The prevalence of abnormal magnetic resonance imaging findings in asymptomatic knees: with correlation of magnetic resonance imaging to arthroscopic findings in symptomatic knees. *Am J Sports Med* 1994;22:739–745.
10. Noble J, Hamblen DL. The pathology of the degenerative meniscus lesion. *J Bone Joint Surg Br* 1975;57:180–186.
11. Poehling GG, Ruch DS, Chabon SJ. The landscape of meniscal injuries. *Clin Sports Med* 1990;9:539–549.
12. Duncan JB, Hunter R, Purnell M, Freeman J. Meniscal injuries associated with acute anterior cruciate ligament tears in alpine skiers. *Am J Sports Med* 1995;23:170–172.
13. Vangsness CT, DeCampos J, Marritt PO, Wiss DA. Meniscal injury associated with femoral shaft fractures. *J Bone Joint Surg Br* 1993;75:207–209.
14. Vangsness CT, Ghaderi B, Hohl M, Morre TM. Arthroscopy of meniscal injuries with tibial plateau fractures. *J Bone Joint Surg Br* 1994;76:488–490.
15. O'Connor BL. The histological structure of dog knee menisci with comments on its possible significance. *Am J Anat* 1976;147:407–417.
16. Berlet GC, Fowler PJ. The anterior horn of the medial meniscus: an anatomical study of its insertion. *Am J Sports Med* 1998;26:540–543.
17. Bullough PG, Munuera L, Murphy J, Weinstein AM. The strength of the menisci of the knee as it relates to their fine structure. *J Bone Joint Surg Br* 1970;52:564–570.
18. Aspden RM, Yarker YE, Hukins WL. Collagen orientations in the meniscus of the knee joint. *J Anat* 1985;140:371–380.
19. Jordan MR. Lateral meniscal variants: evaluation and treatment. *J Am Acad Orthop Surg* 1996;4:191–200.
20. Ikeuchi H. The early days of arthroscopic surgery in Japan. *Arthroscopy* 1988;4:222–225.
21. Watanabe M, Takeda S, Ikeuchi H. *Atlas of Arthroscopy*, 3rd ed. Tokyo: Igaku-Shoin, 1979.
22. Arnoczky SP, Warren RF. Microvasculature of the human meniscus. *Am J Sports Med* 1982;10:90–95.
23. DeHaven KE, Arnoczky SP. Meniscal repair: part I: basic science, indications for repair, and open repair. *J Bone Joint Surg Am* 1994;76:140–152.
24. Wilson AS, Legg PG, McNeur JC. Studies on the innervation of the medial meniscus in the human knee joint. *Anat Rec* 1969;165:485–492.
25. Day B, MacKenzie WG, Shim SS, Leung G. The vascular and nerve supply of the human meniscus. *Arthroscopy* 1985;1:58–62.
26. Zimny ML, Albright DJ, Dabezies E. Mechanoreceptors in the human medial meniscus. *Acta Anat* 1988;133:35–40.
27. Mine T, Kimura M, Sakka A, Kawai S. Innervation of nociceptors in the menisci of the knee joint: an immunohistochemical study. *Arch Orthop Trauma Surg* 2000;120: 201–204.
28. Dye SF, Vaupel GL, Dye CC. Conscious neurosensory mapping of the internal structures of the human knee without intraarticular anesthesia. *Am J Sports Med* 1998;26: 773–777.
29. Ahmed AM, Burke DL. In-vitro measurement of static pressure distribution in synovial joints-part I: tibial surface of the knee. *J Biomech Eng* 1983;105:216–225.
30. Radin EL, de Lamotte F, Maquet P. Role of the menisci in the distribution of stress in the knee. *Clin Orthop Relat Res* 1984;185:290–294.
31. Baratz ME, Fu FH, Mengato R. Meniscal tears: the effect of meniscectomy and of repair on the intraarticular contact areas and stress in the human knee. A preliminary report. *Am J Sports Med* 1986;14:270–275.
32. Ihn JC, Kim SJ, Park IH. In vitro study of contact area and pressure distribution in the human knee after partial and total meniscectomy. *Int Orthop* 1993;17:214–218.
33. Lee SJ, Aadalen KJ, Malaviya P, Lorenz EP, Hayden JK, Farr J, Kang RW, Cole BJ. Tibiofemoral contact mechanics after serial medial meniscectomies in the human cadaveric knee. *Am J Sports Med* 2006;34:1334–1344.
34. Voloshin AS, Wosk J. Shock absorption of meniscectomized and painful knees: a comparative in vivo study. *J Biomed Eng* 1983;5:157–161.
35. Burke DL, Ahmed AM, Miller J. A biomechanical study of partial and total medial meniscectomy of the knee. *Trans Orthop Res Soc* 1978;3:91.
36. Seedhom BB, Hargreaves DJ. Transmission of the load in the knee joint with special reference to the role of the menisci: part II: experimental results, discussion and conclusions. *Eng Med* 1979;8:220–228.
37. Hede A, Larsen E, Sandberg H. Partial versus total meniscectomy a prospective, randomised study with long-term follow-up. *J Bone Joint Surg Br* 1992;74:118–121.
38. Fairbank TJ. Knee joint changes after meniscectomy. *J Bone Joint Surg Br* 1948;30:664–670.
39. Roos H. Lauren M, Adalberth T, Roos EM, Jonsson K, Lohmander LS. Knee osteoarthntis after meniscectomy: prevalence of radiographic changes after twenty-one years, compared with matched controls. *Arthritis Rheum* 1998;41: 687–693.
40. Rockborn P, Gillquist J. Long-term results after arthroscopic meniscectomy: the role of preexisting cartilage fibrillation in a 13 year follow-up of 60 patients. *Int J Sports Med* 1996;17: 608–613.
41. Andersson-Molina H, Karlsson H, Rockborn P. Arthroscopic partial and total meniscectomy: a long term follow-up study with matched controls. *Arthroscopy* 2002;18:183–189.
42. Cicuttini FM, Forbes A, Yuanyuan W, Rush G, Stuckey SL. Rate of knee cartilage loss after partial meniscectomy. *J Rheumatol* 2002;29:1954–1956.
43. Metcalf RW. Arthroscopic meniscal surgery. In McGinty JB (ed.), *Operative Arthroscopy.* New York: Raven Press, 1991, pp. 203–236.
44. Greis PE, Bardana DD, Holmstrom MC, Burks RT. Meniscal injury: I. Basic science and evaluation. *J Am Acad Orthop Surg* 2002;10:168–176.
45. Greis PE, Burks RT. Arthroscopic meniscectomy. In McGinty JB (ed.), *Operative Arthroscopy*, 3rd ed. Philadelphia: Lippincott William & Wilkins, 2003, pp. 218–232.

46. Johnson RJ, Kettelkamp DB, Clark W, Leaverton P. Factors affecting late results after meniscectomy. *J Bone Joint Surg Am* 1974;56:719–729.
47. Shakespeare DT, Rigby HS. The bucket-handle tear of the meniscus. A clinical and arthrographic study. *J Bone Joint Surg Br* 1983;65:383–387.
48. Urquhart MW, O'Leary JA, Giffin JR, Fu FH. Meniscal injuries: 1. Meniscal injuries in the adult. In DeLee J (ed.), *DeLee and Drez's Orthopaedic Sports Medicine: Principles and Practice*, 2nd ed. Philadelphia: Saunders, 2003, pp. 87–96.
49. Steinbruck K, Wiehmann JC. Examination of the knee joint: the value of clinical findings in arthroscopic control. *Z Orthop Ihre Grenzgeb* 1988;126:289–295.
50. Scholten RJ, Deville WL, Opstelten W, Bijl D, van der Plas CG, Bouter LM. The accuracy of physical diagnostic tests for assessing meniscal lesions of the knee: a meta analysis. *J Fam Pract* 2001;50:938–944.
51. Eren OT. The accuracy of joint line tenderness by physical examination in the diagnosis of meniscal tears. *Arthroscopy* 2003;19:850–854.
52. Karachalios T, Hautes M, Zibis AH, Zachos V, Karantanas AH, Malizos KN. Diagnositc accuracy of a new clinical test (the thessaly test) for early detection of meniscal tears. *J Bone Joint Surg Am* 2005;87:955–962.
53. Wadey VMR, Mohtadi NGH, Bray RC, Frank CB. Positive predictive value of maximal posterior joint-line tenderness in diagnosing meniscal pathology: a pilot study. *Can J Surg* 2007;50: 96–100.
54. Shelbourne KD, Martini DJ, McCarroll JR, VanMeter CD. Correlation of joint line tenderness and meniscal lesions in patients with acute anterior cruciate ligament tears. *Am J Sports Med* 1995;23:166–169.
55. Rosenberg TD, Paulos LE, Parker RD, Coward DB, Scott SM. The forty-five-degree posteroanterior flexion weight-bearing radiograph of the knee. *J Bone Joint Surg Am* 1988;70: 751–759.
56. Ryzewicz M, Peterson B, Siparsky PN, Bartz RL. The diagnosis of meniscus tears. *Clin Orthop Relat Res* 2007;455:123–133.
57. Weinstabl R, Muellner T, Vècsei V, Kainberger F, Kramer M. Economic considerations for the diagnosis and therapy of meniscal lesions: can magnetic resonance imaging help reduce the expense? *World J Surg* 1997;21:363–368.
58. Herlin S, Hållander M, Wange P, Weidenhielm L, Werner S. Arthroscopic or conservative treatment of degenerative medial meniscal tears: a prospective randomized trial. *Knee Surg Sports Traumatol Arthrosc* 2007;15:393–401.
59. Weiss CB, Lundberg M, Hamberg P, DeHaven KE, Gillquist J. Non-operative treatment of meniscal tears. *J Bone Joint Surg Am* 1989;71:811–822.
60. Klimkiewicz JJ, Shaffer B. Meniscal surgery 2002 update: indications and techniques for resection, repair, regeneration, and replacement. *Arthroscopy* 2002;18:14–25.
61. Cannon WD, Vittori JM. The incidence of healing in arthroscopic meniscal repairs in anterior cruciate ligament-reconstructed knees versus stable knees. *Am J Sports Med* 1992;20:176–181.
62. Hashimoto J, Kurosaka M, Yoshiya S, Hirohata K. Meniscal repair using fibrin sealant and endothelial cell growth factor. *Am J Sports Med* 1992;20:537–541.
63. Shelbourne KD, Patel DV, Adsit WS, Porter DA. Rehabilitation after meniscal repair. *Clin Sports Med* 1996;15:595–612.
64. Arnoczky SP, Warren RF, Kaplan N. Meniscal remodeling following partial meniscectomy: an experimental study in the dog. *Arthroscopy* 1985;1:247–252.
65. Ciccotti MG, Shields CL Jr, El Attrache NS. Meniscectomy. In Fu FH (ed.), *Knee Surgery*. Baltimore: Williams & Wilkins, 1994, 591–613.
66. Albertsson M, Gillquist J. Discoid lateral meniscus: a report of 29 cases. *Arthroscopy* 1988;4:211–214.
67. Ikeuchi H. Arthroscopic treatment of the discoid lateral meniscus. *Clin Orthop Relat Res* 1982;167:19–28.
68. Gillquist J, Hamberg P, Lysholm J. Endoscopic partial meniscectomy and total meniscectomy: a comparative study with a short term follow up. *Acta Orthop Scand* 1982;53:975–979.
69. Northmore-Ball MD, Dandy DJ, Jackson RW. Arthroscopic, open partial, and total meniscectomy: a comparative study. *J Bone Joint Surg Br* 1983;65:400–404.
70. Hede A, Larsen E, Sandberg H. The long term outcome of open total and partial meniscectomy related to the quantity and site of the meniscus removed. *Int Orthop* 1992;16:122–125.
71. Englund M, Roos EM, Roos HP, Lohmander LS. Patient-relevant outcomes fourteen years after meniscectomy: influence of type of meniscal tear and size of resection. *Rheumatology* 2001;40: 631–639.
72. Covall DJ, Wasilewski SA. Roentgenographic changes after arthroscopic meniscectomy: five-year follow-up in patients more than 45 years old. *Arthroscopy* 1992;8:242–246.
73. Bonamo JJ, Kessler KJ, Noah J. Arthroscopic meniscectomy in patients over the age of 40. *Am J Sports Med* 1992;20:422–429.
74. Faunø P, Nielsen AB. Arthroscopic partial meniscectomy: a long-term follow-up. *Arthroscopy* 1992;8:345–349.
75. Bolano LE, Grana WA. Isolated arthroscopic partial meniscectomy: functional radiographic evaluation at five years. *Am J Sports Med* 1993;21:432–437.
76. Osti L, Liu SH, Raskin A, Merlo F, Bocchi L. Partial lateral meniscectomy in athletes. *Arthroscopy* 1994;10:424–430.
77. Jaureguito JW, Elliot JS, Lietner T, Dixon LB, Reider B. The effects of arthroscopic partial lateral meniscectomy in an otherwise normal knee: a retrospective review of functional, clinical, and radiographic results. *Arthroscopy* 1995;11:29–36.
78. Matsusue Y, Thomson NL. Arthroscopic partial medial meniscectomy in patients over 40 years old: a 5- to 11-year follow-up study. *Arthroscopy* 1996;12:39–44.
79. Burks RT, Metcalf MH, Metcalf RW. Fifteen-year follow-up of arthroscopic partial meniscectomy. *Arthroscopy* 1997;13: 673–679.
80. Schimmer RC, Brülhart KB, Duff C. Glinz W. Arthroscopic partial meniscectomy: a 12-year follow-up and two-step evaluation of the long-term course. *Arthroscopy* 1998;14: 136–142.
81. Barrett GR, Treacy SH, Ruff CG. The effect of partial lateral meniscectomy in patients $\geq$ 60 years. *Orthopedics* 1998;21: 251–257.
82. Krüger-Franke M, Siebert CH, Kugler A, Trouillier HH, Rosemeyer B. Late results after arthroscopic partial medial meniscectomy. *Knee Surg Sports Traumatol Arthrosc* 1999;7: 81–84.
83. Higuchi H, Kimura M, Shirakura K, Terauchi M, Takagishi K. Factors affecting long-term results after arthroscopic partial meniscectomy. *Clin Orthop* 2000;377:161–168.
84. Hoser C, Fink C, Brown C, Reichkendler M, Hackl W, Bartlett J. Long-term results of arthroscopic partial lateral meniscectomy in knees without associated damage. *J Bone Joint Surg Br* 2001;83:513–516.
85. Scheller G, Sobau C, Bulow JU. Arthroscopic partial lateral meniscectomy in an otherwise normal knee: clinical, functional, and radiographic results of a long-term follow-up study. *Arthroscopy* 2001;17:946–952.
86. Bonneux I, Vandekerckhove B. Arthroscopic partial lateral meniscectomy long-term results in athletes. *Acta Orthop Belg* 2002;68:356–361.
87. McNicholas MJ, Rowley DI, McGurty D, Adalberth T, Abdon P, Lindstrand A, Lohmander L. Total meniscectomy in adoles-

cence: a thirty-year follow-up. *J Bone Joint Surg Br* 2000;82: 217–221.

88. Allen PR, Denham RA, Swan AV. Late degenerative changes after meniscectomy: factors affecting the knee after operation. *J Bone Joint Surg Br* 1984;66:666–671.
89. Jorgensen U, Sonne-Holm S, Lauridsen F, Rosenklint A. Long-term follow-up of meniscectomy in athletes: a prospective longitudinal study *J Bone Joint Surg Br* 1987;69:80–83.
90. Seedhom BB, Dowson D, Wright V. Proceedings: functions of the menisci: a preliminary study. *Ann Rheum Dis* 1974;33:111.
91. McDermott ID, Amis AA. The consequences of meniscectomy. *J Bone Joint Surg Br* 2006;88:1549–1556.
92. Sherman MF, Warren RF, Marshall JL, Savatsky GJ. A clinical and radiographical analysis of 127 anterior cruciate insufficient knees. *Clin Orthop* 1988;227:229–237.
93. Sommerlath K. The importance of the meniscus in unstable knees: a comparative study. *Am J Sports Med* 1989;17: 773–777.
94. Chatain F, Robinson AHN, Adeleine P, Chambat P, Neyret P. The natural history of the knee following arthroscopic medial meniscectomy. *Knee Surg Sports Traumatol Arthrosc* 2001;9:15–18.
95. Roos EM, Östenberg A, Roos H, Ekdahl C, Lohmander LS. Long-term outcome of meniscectomy: symptoms, function, and performance tests in patients with or without radiographic osteoarthritis compared to matched controls. *Osteoarthr Cartil* 2001;9:316–324.
96. Tapper EM, Hoover NW. Late results after meniscectomy. *J Bone Joint Surg Am* 1969;51:517–526.
97. Small N. Complications in arthroscopic surgery performed by experienced arthroscopists. *Arthroscopy* 1988;4:215–221.
98. Small NC. Complications in arthroscopy: the knee and other joints. *Arthroscopy* 1986;2:253–258.
99. Small NC. Complications in arthroscopic meniscal surgery. *Clin Sports Med* 1990;9:609–617.
100. Dobner JJ, Nitz AJ. Postmeninscectomy tourniquet palsy and functional sequelae. *Am J Sports Med* 1982;10:211–214.

Meniscus Repair and Future Directions

Michael J. Angel, Jordan Kerker, and Nicholas Sgaglione

Introduction

Meniscal tear pathology is one of the most common orthopaedic diagnoses in the United States, with knee arthroscopy being the most common orthopaedic procedure performed by orthopaedic surgeons. Approximately 800,000 meniscectomies and 100,000 meniscal repairs are performed each year [1]. Whereas many patients seek treatment for a torn meniscus and large subsets of those patients may undergo some form of meniscectomy, the prediction of which patient will go on to develop postmeniscectomy osteoarthritis remains unclear.

It has been argued that restoration of the meniscus by performing repair instead of meniscectomy can restore joint biomechanics and theoretically reduce the progression of chondrosis and arthrosis. In 1975, Walker and Erkman found that with loads of up to 150 kg, the lateral meniscus transfers most of the weight in that compartment, while the medial meniscus shares approximately 50% of the load with the articulating surfaces of the tibiofemoral joint [2]. Whereas partial meniscectomy and total meniscectomy both have been shown to increase the contact stresses exerted on the articular cartilage, resulting in its degeneration and ultimately in osteoarthritis, a partial meniscectomy has been shown to improve prognosis and decrease chondral wear compared with total meniscectomy [3]. After partial meniscectomy, femoral-tibial contact areas decrease by approximately 10%, with peak local contact stresses (PLCSs) increasing by approximately 65%. After total meniscectomy, contact areas decrease approximately 75%, and PLCS increases approximately 235% [3]. PLCSs and contact areas have been reported as the same when meniscus repair is performed, indicating that preservation of the joint by repairing the meniscus may reduce the risk of developing osteoarthritis.

M.J. Angel (✉)
Orthopaedic Sports Fellow, Kerlan Jobe Orthopaedic Clinic, Los Angeles, California

Clinical evaluation of meniscal pathology begins with the history, including the location of pain, recent trauma, prior injuries/surgery, as well as symptoms of recurrent effusions, antalgia, or instability (which may indicate associated ligamentous pathology). Specific mechanical symptoms such as locking, catching, or loss of motion should be noted. Meniscus tears can be described as traumatic or degenerative. An acute traumatic tear will typically present in a young patient (average age of 25 years) who reports a recent injury to the knee resulting in immediate pain and typically swelling. A commonly described injury pattern results from compression and rotation as the knee is brought from a flexed to an extended position. A chronic tear is typically seen in older patients (average age of 50 years) who deny any recent trauma but report an insidious increase in pain over time. Factors such as patient age, functional demands, activity level, occupation, goals, expectations, and other associated medical problems should be considered and will help determine nonsurgical versus surgical treatment and resection versus repair.

Meniscus tear evaluation and physical examination begins with inspection to determine whether the patient has an effusion of the knee or muscular atrophy, and observations of the gait may define antalgia. Localized swelling may indicate the presence of a meniscal cyst, which occurs due to degenerative horizontal tears, found more commonly on the lateral side. These patients often have focal point tenderness over the joint line. A knee with a "locked" or limited range of motion often indicates a displaced bucket-handle tear. Pain on axial compression-rotation testing may be associated with a meniscus tear. Some of the most commonly used tests include the McMurray test, the Apley compression test, the Childress test, and the Steinman test. The McMurray test is performed with the patient supine and the hip and knee flexed, while applying a valgus force and externally rotating the tibia while extending the knee. An audible or palpable pop or snap indicates a medial meniscal tear. The lateral meniscus may be assessed with a varus and internally rotating force. The Apley compression test is peformed with the patient in the prone position and the knee flexed to 90

B.P. McKeon et al. (eds.), *Knee Arthroscopy*, DOI 10.1007/978-0-387-89504-8_3,

degrees. While maintaining an axial force, internal and external rotation of the tibia is performed; pain indicates a likely meniscus tear. The Childress test has the patient peform a "duckwalk" maneuver. Pain or limitation of motion is associated with meniscal pathology. The Steinman test is performed first with the patient supine and the knee and hip flexed to 90 degrees. The examiner applies a quick and forceful compression of the knee with internal and external rotation. Pain in the lateral compartment with internal rotation indicates a meniscus tear, as does pain in the medial compartment with external rotation.

A complete physical exam of the knee should be performed, including evaluation for associated pathology such as chondral defects and ligamentous pathology. An assessment of cruciate ligament pathology includes a history of hearing a "pop" at the time of injury with acute swelling, a positive Lachman test, and anterior/posterior drawer or pivot shift. Combined ligament pathology with a posterolateral corner injury would be evident by asymmetry on the dial external rotation test. Collateral ligament injuries may be assessed by palpation over the anatomic origins and insertions and with abnormal widening with varus/valgus stresses at 30 degrees and at full extension.

Anatomy

The knee menisci are crescent-shaped in the transaxial plane and triangular in cross section. The medial meniscus is C-shaped and covers approximately 64% of the tibial plateau. Its width varies more compared with that of the lateral meniscus from anterior to posterior, with an average of 10 mm (Fig. 1). The lateral meniscus is more circular in shape, and it covers approximately 84% of the tibial plateau. It has an average width of 12–13 mm and is more constant in size between the anterior and posterior horns.

The medial meniscus and lateral meniscus are fibrocartilaginous structures made up of water (70% to 75%) and collagen (20% to 25%), with the remainder consisting of fibrochondrocytes, glycosaminoglycans, and other adhesion molecules. Ninety percent of the collagen is type I, with the remainder being types II, III, V, and VI. On an ultrastructural level, the surface collagen fibers are arranged in a circumferential pattern in the peripheral one-third, whereas the inner two-thirds consist of radially directed tie fibers. The deep fibers in transition are randomly oriented. The circumferential fibers resist compressive forces, whereas the radially directed fibers resist tensile forces (Fig. 2).

The menisci function to deepen the articular surface of the tibial plateau, providing shock absorption, and compensate for gross incongruity between the articulating surfaces, acting as joint stabilizers. They enhance joint lubrication and synovial fluid diffusion, providing nutrition for articular cartilage [4]. The vascular supply originates from the perimeniscal capillary plexus supplied by the medial and lateral inferior and superior geniculate arteries. The plexus penetrates the meniscus peripherally and its abundance decreases as it crosses centrally. The vascular anatomic distribution gives rise to the clinical labeling of the *red-red, red-white*, and *white-white* zones [5] (Fig. 3). In addition, vascular distribution has been shown to decrease with age [6]. At

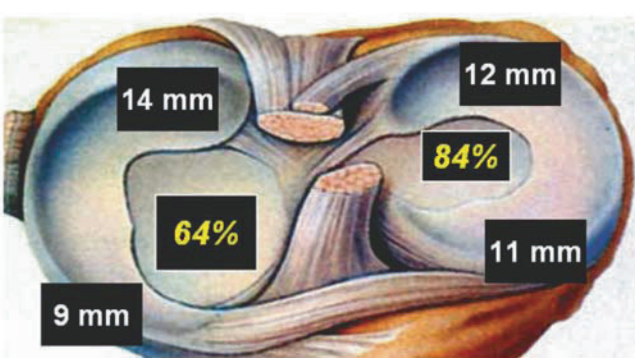


Fig. 1 Shape of the medial and lateral meniscus

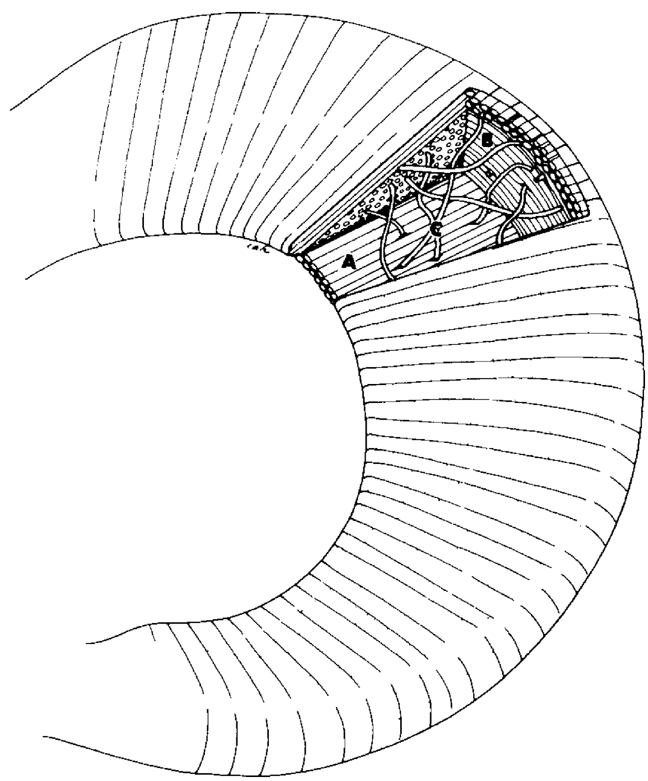


Fig. 2 Ultrastructure of the meniscus: *A*, radially directed fibers; *B*, circumferential fibers; *C*, randomly oriented fibers. (From Shahriaree H. *O'Connor's Textbook of Arthroscopic Surgery.* Copyright © Lippincott Williams & Wilkins, Philadelphia, 1984. Reprinted with permission.)

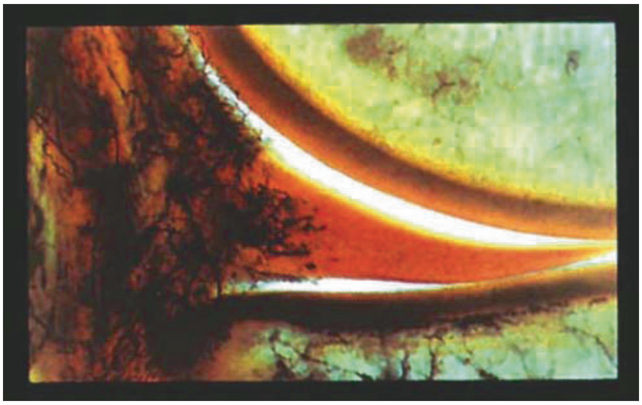


Fig. 3 Vascular penetration of the meniscus. (From Arnoczky SP, Warren RF. Microvasculature of the human meniscus. *Am J Sports Med* 1982;10:90–95. Copyright © 1982 American Orthopaedic Society for Sports Medicine. Reprinted with permission of SAGE Publications, Inc.)

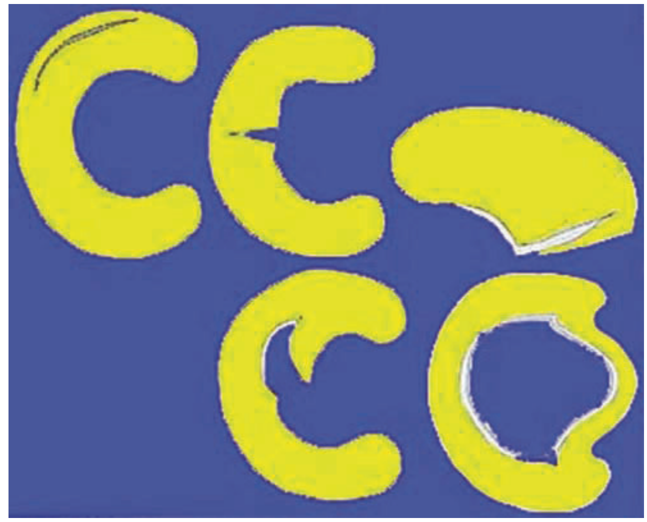


Fig. 4 Meniscus tear patterns. (From Douglas J, Sgaglione NA. Meniscal injuries. In Schepsis A, Busconi B (eds.), *Orthopaedic Surgery Essentials: Sports Medicine*. Copyright © Lippincott Williams & Wilkins, Philadelphia, 2006. Reprinted with permission.)

birth, the entire meniscus is vascularized, whereas only one third remains vascularized in the second decade of life. This decreases to one quarter by the fifth decade [6]. The menisci have been shown to contain free nerve endings and corpuscular mechanoreceptors potentially contributing to nociceptive and proprioceptive function in the knee [4].

Pathology

Meniscal tears may be classified according to anatomic zone vascularity, as discussed above, or by tear pattern. Tear patterns are described as horizontal, radial, longitudinal, bucket-handle, oblique, or complex [7] (Fig. 4).

Preoperative radiographic evaluation includes plain radiographs and also may include magnetic resonance imaging. Plain radiographs should be taken to evaluate for bony pathology, full-length standing mechanical axis alignment, evidence of arthritis, chondrocalcinosis, or findings consistent with associated acute or chronic injuries such as osteochondritis dissecans lesion, osteochondral fracture, or ligamentous injuries. Four views are optimally obtained: a 45-degree posteroanterior flexion weight-bearing view, a true lateral view, a notch view, and a patella skyline view. Radiographs help better define the extent of pathology, particularly in the case in which osteoarthritis correlates with the presence of a meniscus tear.

Magnetic resonance imaging (MRI) using specific sequencing (spin-echo T1-weighted, spin-echo fat-saturated T2-weighted, proton density, and T2-weighted fast low-angle shot images) may not be necessary to diagnose a meniscus tear; however, it is more useful as a confirmatory test and is valuable in the evaluation of associated injuries and concomitant pathology when a meniscus tear is suspected. The sensitivity of MRI for meniscus tears is reported to be as high as 96%, with a specificity of 97% [8]. The MRI classification typically used for meniscus tears is

Grade 1: Small focal area of increased signal, not extending to the joint surface.

Grade 2: Linear area of increased signal, not extending to the joint surface.

Grade 3: Linear area of increased signal, extending to the joint surface [9].

The use of MRI may accurately identify a bucket-handle meniscus tear. On a sagittal T1-weighted MRI scan of the knee through the intercondylar notch, a fragment of torn meniscus that appears as a low-signal-intensity, longitudinally oriented band lying beneath and parallel to the posterior cruciate ligament (PCL) creates a double cruciate configuration. This is referred to as the *double PCL sign* (Fig. 5).

Indications and Techniques

Nonoperative Treatment

Clinical assessment of meniscus tears includes nonoperative and operative treatment options. Nonoperative treatment includes physical therapy, bracing, rest, activity modification, analgesics, and inflammation reduction measures such as icing, nonsteroidal antiinflammatory medications, and, occasionally, corticosteroid injections. Nonoperative treatment is usually instituted and followed for approximately 6

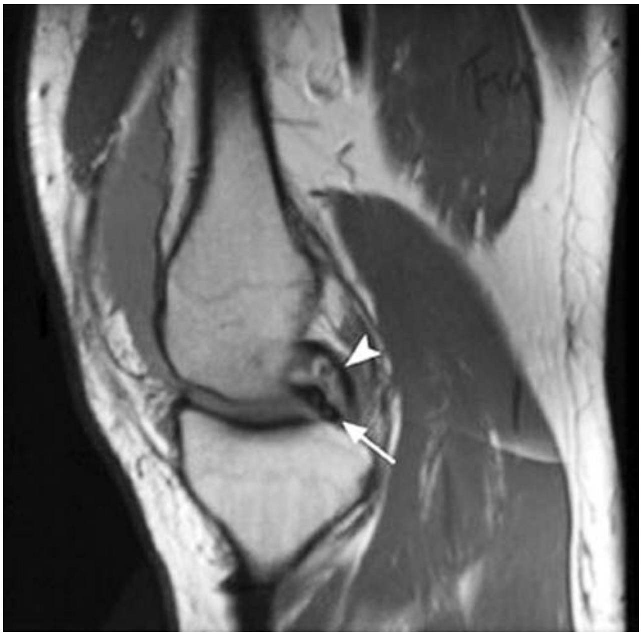


Fig. 5 Double PCL sign on T1 sagittal MRI indicating a bucket-handle meniscus tear (arrow refers to a fragment of torn meniscus, arrow-head refers to posterior cruciate ligament(PCL))

Fig. 6 Unstable meniscus with the “ability to roll.”

Table 1 Tear patterns and their potential to be repaired

Tear pattern	Repair potential
Horizontal tears	Irreparable
Longitudinal tears	Repairable
Radial (transverse) tears	Potentially repairable
Bucket-handle tears	Repairable
Oblique (flap or parrot beak) tears	Irreparable
Complex (degenerative) tears	Irreparable

weeks, and typically patients return to full activities within 3 months. If the patient does not improve, then surgery must be considered. The orthopaedist may indicate a patient for surgery more promptly when they exhibit recurrent effusions (consistent with hypertrophic synovitis) or painful mechanical symptoms, including locking and catching, which are less likely to resolve nonoperatively or may indicate a repairable bucket-handle tear.

Surgical Treatment

The indications for meniscus repair have been expanding as a better understanding of pathophysiology, repair-site healing, and biomechanical behavior and performance of various repair techniques and devices is developed. Most commonly, surgical treatment is indicated for continued pain and symptoms refractory to nonoperative treatment. More acute indications include (1) persistent mechanical symptoms of the knee joint; or (2) a younger, more active patient with a history and exam highly suspicious for an acute and possibly repairable meniscus tear.

The goal of arthroscopic surgery for meniscal tears is to provide pain relief through tear resection or repair while preserving as much of the meniscus as possible. The decision to resect versus repair is dependent upon the stability of the tear. An unstable tear will be easily mobilized, displaces at least 7 mm, and has the “ability to roll” (Fig. 6). This should be addressed surgically, whereas a stable tear may often be left untouched. Decision making in meniscus repair is based on the tear pattern, location, stability, chronicity, and associated pathology, in addition to a patient’s age, activity level, compliance, goals, and expectations (Table 1). Clinical assessment and judgment are essential: vertical longitudinal tears located in the red-red and red-white vascularized zones of the meniscal periphery are anatomically optimal for repair, whereas asymptomatic tears that are not clinically correlative, particularly in patients over the age of 60 years, with associated articular cartilage degenerative arthritis are clearly not repaired.

Surgical Technique

The patient is placed supine on the operating room table, and the lower extremity is stabilized using either a leg holder or a lateral post. The knee joint and portals may be injected under sterile conditions with local anesthesia prior to formal preparation and draping of the patient. The joint is distended with approximately 30 mL of 0.5% Marcaine/1% (Bupi vacaine Hcl, AstraZeneca, London, UK) lidocaine with epinephrine mixture, and the planned portal sites are similarly injected with 5–10 mL of the same mixture. A tourniquet may be applied on the upper thigh and may be used as needed.

Fig. 7 Standard and accessory arthroscopic portal sites

The standard portal sites are used whether a repair or meniscectomy is planned. They may include an outflow portal in the superomedial or lateral positions and inferolateral and inferomedial portals for the arthroscope and "working" instruments. Accessory portals may be used but are not typically created until determined necessary intraoperatively. They include the superolateral, posteromedial, posterolateral, midpatella, and central portals (Fig. 7).

A precise evaluation of the tear must be performed prior to repair. If the tear is deemed irrepairable, a partial meniscectomy is performed. Resection is performed with meniscal baskets or biters and motorized shavers. All efforts should be made to preserve as much meniscus as possible. Mobile, unstable meniscus fragments should be resected, leaving a smooth contoured transition. The meniscosynovial junction is preserved because of the importance of circumferential collagen fibers that contribute to "hoop stress" dissipation.

If meniscus repair is carried out, "preparation" of the meniscus tear must be performed with stimulation of the tear edges and meniscal periphery to increase vascularity to the repair site. Rasping is performed with an arthroscopic shaver or proprietary low-profile meniscal rasp. If trephination is performed, a long, 18-gauge needle may be used either percutaneously or through an arthroscopic portal. Care should be taken to trephinate the peripheral meniscal tissue in a manner that is perpendicular to the circumferential fibers (to avoid inducing punctures that may act as stress risers). Trephination is performed until blood flow is achieved. This may best be appreciated with the arthroscopic flow turned off.

Meniscus repair may be performed open, arthroscopically assisted, or all arthroscopically. The open meniscus repair is rarely performed since the development of arthroscopic techniques. The specific techniques include inside-out, outside-in, and all arthroscopic, as well as hybridized methods. Determining which method to use takes into account the tear pattern, location, surgeon experience, and preference (Table 2).

Inside-Out Technique

Inside-out meniscus repair is best performed on tears of the posterior horn, middle third, peripheral capsule, or on bucket-handle type tears. This is an arthroscopically assisted procedure performed by passing sutures through needle cannulas inserted through the portals, exiting via a strategically placed posterolateral or posteromedial accessory incision (Fig. 8 A–C). The incision is made prior to the passage of the sutures to safely capture the suture needles as they exit the knee capsule. In this manner, the neurovascular structures are protected.

The knee should be positioned dependent upon the compartment in which the repair is being performed. For passage of a needle through the medial compartment, the knee will be placed in 20–30 degrees of flexion to avoid teth-

Table 2 Repair techniques and their general indications

Repair technique	Indications
Outside-in technique	Anterior horn tears
	Middle-third tears
	Radial tears
Inside-out technique	Posterior horn tears
	Middle-third tears
	Bucket-handle tears
	Peripheral capsular tears
	Meniscal allograft
All-arthroscopic fixation technique (first-generation devices)	Posterior horn tears
	Vertical, longitudinal tears
	Tears with >2- to 3-mm rim width
All-arthroscopic suture fixation technique (second-generation devices)	Posterior horn tears
	Middle-third tears
	Bucket-handle tears
	Radial tears

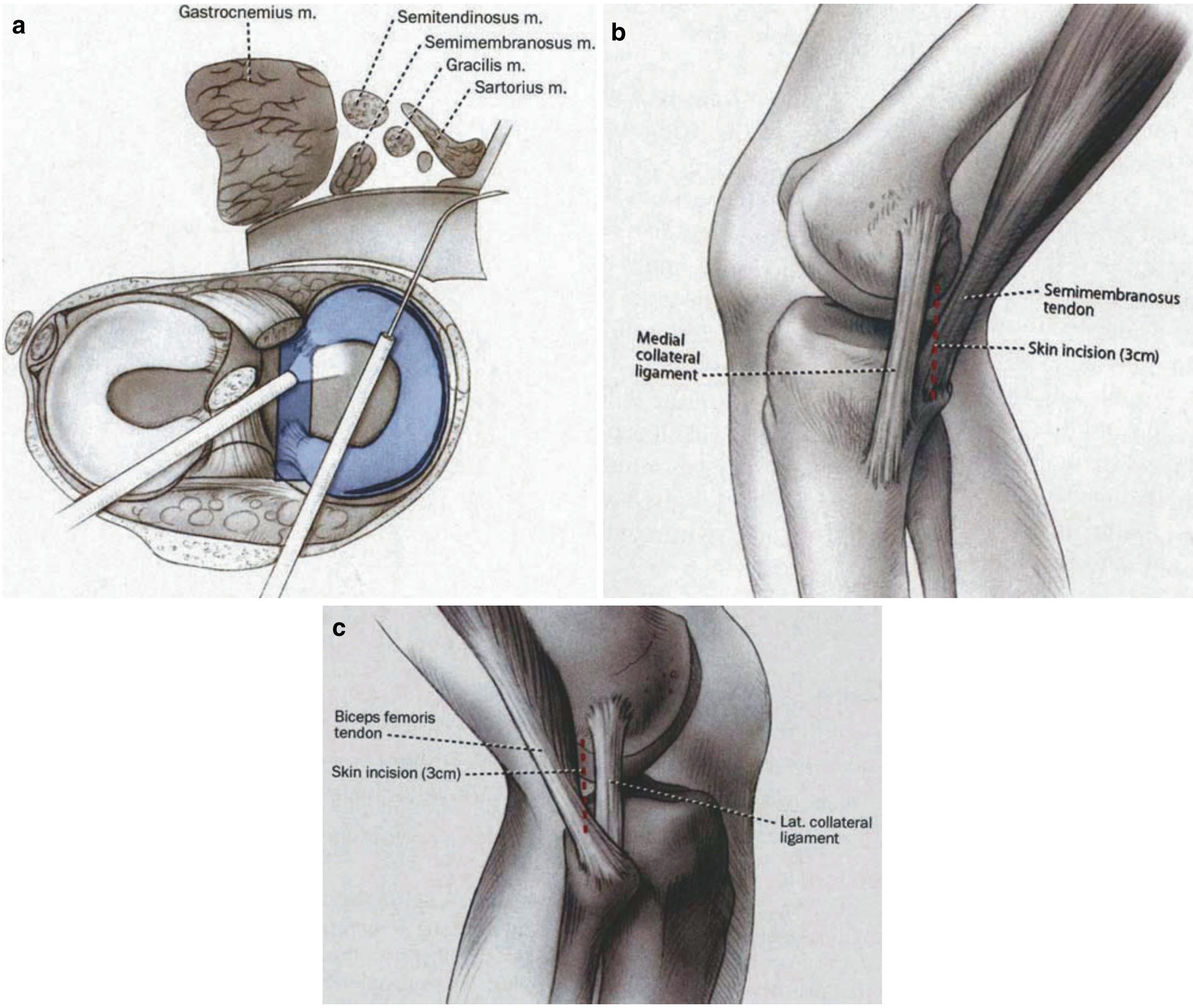


Fig. 8 (**A, B, C**) Inside-out meniscus repair. (From Noyes FR, Barber-Westin SD, Rankin M. Meniscal transplantation in symptomatic patients less than fifty years old. *J Bone Joint Surg Am* 2005;87(Suppl 1, Pt 2):149–165. Reprinted with permission from The Journal of Bone and Joint Surgery, Inc.)

ering the capsule, and the lateral compartment is positioned in 90 degrees of flexion to allow the peroneal nerve, popliteus, and lateral inferior geniculate artery to fall posteriorly.

For repair of a medial meniscus tear, a 4- to 6-cm posteromedial incision is made just posterior to the medial collateral ligament, extending approximately one third above and two thirds below the joint line. Dissection is continued anterior to the sartorius and semimembranosus musculature. The saphenous nerve remains protected just posterior to the sartorius muscle. Flexion of the knee joint also helps prevent the saphenous nerve from moving anteriorly. The deep dissection is carried to the medial head of the gastrocnemius, where the interval between the gastrocnemius (retracted posteriorly) and posteromedial capsule may be developed.

In lateral meniscus repair, a 4- to 6-cm incision is made just posterior to the lateral collateral ligament, extending one third above and two thirds below the joint line. The superficial dissection is carried between the posterior aspect of the iliotibial band (retracted anteriorly) and anterior to the biceps tendon complex (retracted posteriorly). The deep dissection is carried anterior to the lateral head of the gastrocnemius, retracting it posteriorly to protect the peroneal nerve and expose the posterolateral capsule.

Upon exposure of the capsule from either the medial or lateral side, a popliteal retractor is placed against the capsule to visualize and deflect the exiting suture needles. A single-lumen or double-lumen cannula is passed through the contralateral arthroscopic portals to the site of the tear. Long, flexible, single-loaded or double-loaded 2-0 or 0 nonabsorbable suture needles are then passed through the cannula, piercing the meniscus above and below the tear site and creating verticle mattress sutures. The needles are captured one at a time by an assistant who is retracting on the capsule. Care is taken not to pull either suture all the way through until both needles are passed. This will prevent the needles from shredding the sutures. The sutures are then tensioned and tied to the capsule while viewing the repair arthroscopically. Vertical mattress sutures should be placed every 4–5 mm until stability of the tear is satisfactory.

Outside-In Technique

The outside-in technique is best performed on tears of the anterior and middle-third of the meniscus as well as on radial-type tears. It is performed by passing multiple, long, 18-gauge spinal needles percutaneously from outside of the knee to inside the knee joint, then shuttling sutures through the needles to be retrieved and tied to the meniscus surface (Fig. 9A, B). Proprietary curved needles with wire-loop retrievers (or looped 3-0 suture) may be used to facilitate the outside-in method. The needle should enter the joint through the periphery to achieve vertical or horizontal mattress suture configuration. The needles should be spaced approximately 3–5 mm apart with the use of an absorbable monofilament suture. After tensioning of the mattress suture, a 3- to 5-mm skin incision is made near the suture strands and blunt dissection carried down to the capsule with a hemostat. A probe may be used to retrieve the sutures and tie them down to the capsule under direct visualization, taking care to avoid incarceration of any neurovascular structures.

All-Inside Fixator Technique

The all-inside fixator devices are considered the first-generation all-arthroscopic meniscus repair devices. Meniscal repair fixators are commonly based on a reverse-barbed design (e.g., Meniscus Arrow and Biostinger, ConMed Linvatec, Largo, FL; Dart, Arthrex, Naples, FL) and are composed of various bioabsorbable copolymers such as poly-L-lactic acid and poly-D-lactic acid (Fig. 10).

Fixators are best used in vertical tears in the red-white zone of the posterior horn. After identification of the tear site, an accurate measurement of the size of the meniscus is performed with an arthroscopic measuring device. The fixators must be delivered and inserted perpendicular to the tear and parallel to the tibial surface. If they are inserted obliquely, the fixation construct strength may be compromised, resulting in loosening or migration of the device [10]. The fixators should be placed at approximately 3- to 5-mm intervals, and care must be taken to implant the fixator so that it is seated flush or countersunk to the meniscus surface while spanning the tear equally on both sides to appropriately compress the tear (Fig. 11A, B).

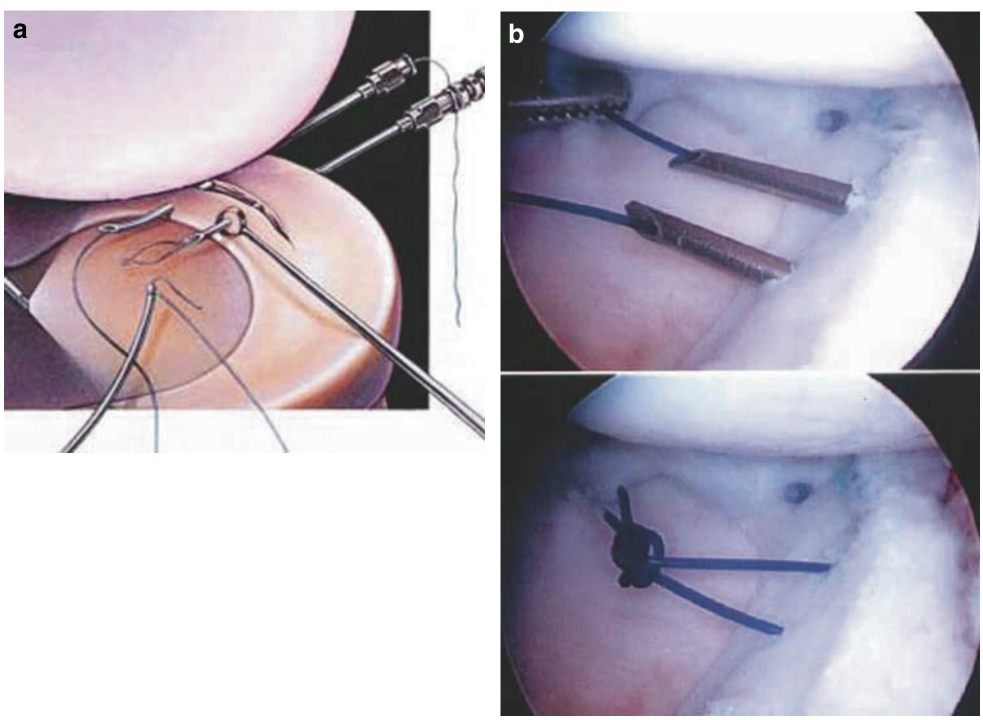


Fig. 9 (**A, B**) Outside-in meniscus repair. (From Sgaglione NA. Meniscus repair: update on new techniques. *Techniques in Knee Surgery* 2002;1(2):113–127. Reprinted with permission of Lippincott Williams & Wilkins.)

Fig. 10 Meniscal fixator devices (first-generation fixators). (From Sgaglione NA. Meniscus repair: update on new techniques. *Techniques in Knee Surgery*, 2002;1(2):113–127. Reprinted with permission of Lippincott Williams & Wilkins and of ConMed Linvatec, Largo, FL.)

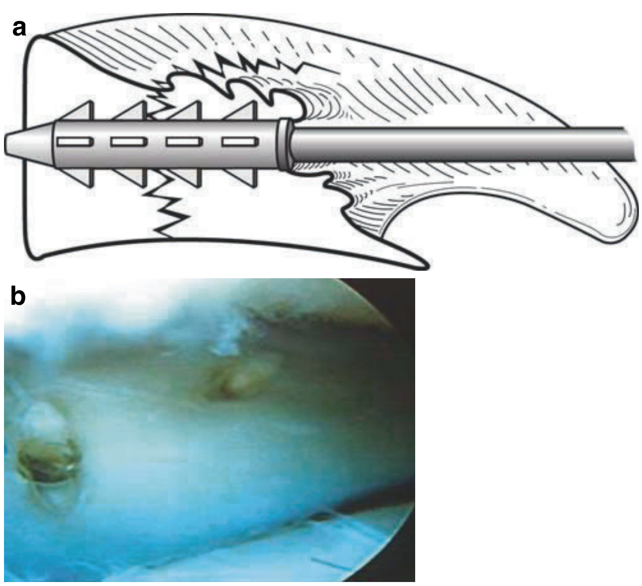


Fig. 11 (**A, B**) Meniscal fixator device placed in appropriate position. (From Sgaglione NA. Meniscus repair: update on new techniques. *Techniques in Knee Surgery* 2002;1(2):113–127. Reprinted with permission of Lippincott Williams & Wilkins.)

Recently, multiple studies have been published that criticize the use of these devices, citing high failure rates and complications. Kurzweil et al. reported a failure of meniscus healing of 28%, with significant complications such as implant loosening and breakage, chondral damage, and significant postoperative joint-line pain [11]. In addition, Gifstad et al. reported a reoperation rate of 41% with use of the Biofix Arrow (Bionx Implants Ltd, Tampere, Finland) [12]. Other reports also have been published regarding failures. Lee and Diduch reported a significant deterioration in success rate from 90% at 2 years to 70% at 6 years [13]. Most recently, Seibold et al. reported a high failure rate after following 113 repairs an average of 6 years postoperatively [14]. They found a failure in 28% of patients, who had to undergo revision and meniscectomy. Approximately 80% of failures occurred in the first 3 years, suggesting failure of the implants.

All-Inside Suture Fixation Technique

The second-generation all-inside meniscus repair also is performed without making an accessory incision; it incorporates all-arthroscopic techniques and hybrid bioabsorbable fixator/anchor and suture constructs. These devices are based on the arthroscopic delivery of extended resorptive or nonabsorbable braided polyester sutures across the tear site using pretied, sliding knot configurations that allow for cinching of the interposed and interconnected sutures between two anchoring implants. There are currently two proprietary designs available (e.g., FasT-Fix, Smith & Nephew, Andover, MA; Meniscal Viper, Arthrex, Naples, FL) (Fig. 12A, B).

The FasT-Fix delivery surgical technique begins with preparation of the tear site. This may be performed with use of meniscal shavers or rasps. The passage of a fixator device through the contralateral working portal requires some preparation before reducing and repairing the tear. The portal may need to be enlarged either by extending the portal incision or with use of a large blunt dilator. The surgeon may then choose a straight or curved needle delivery system. Use of a curved fixator device will improve the potential of obtaining a vertical mattress suture configuration compared with that for use of a straight needle. Needle sheaths and nondisposable metallic portal skids also may be used to facilitate arthroscopic entrance and delivery of the suture fixators. The instrument is then inserted from the contralateral working portal through a disposable split-sheath (provided with the instrumentation) that prevents the delivery system from getting caught on loose tissue.

After the device has been placed in the appropriate contralateral compartment, the sheath is removed, exposing the needle-delivery system. At this point, the tear is reduced with use of the suture-fixator. An outside-in traction suture that

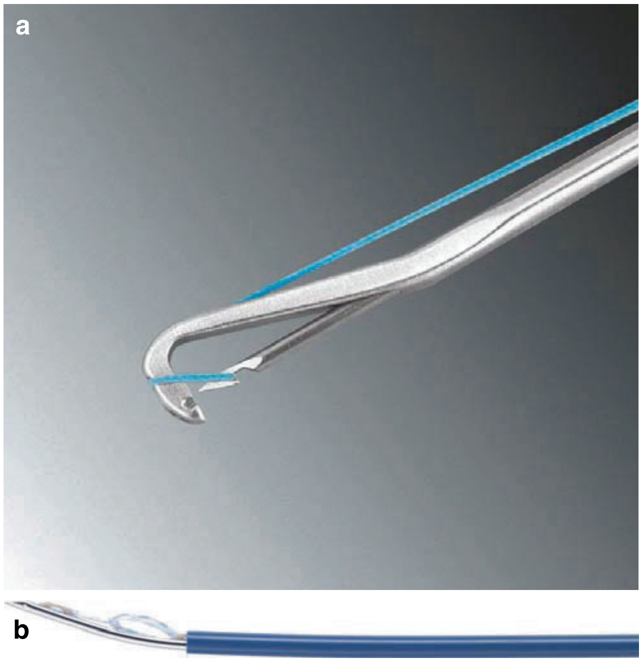


Fig. 12 Second-generation meniscal suture devices: (**A**) Meniscal Viper (reprinted with permission of Arthrex, Naples, FL); (**B**) Meniscal FasT-Fix (reprinted with permission of Smith & Nephew, Andover, MA).

functions as a provisional reducing stitch also may be used to hold the reduction until the sutures can be placed. This is especially useful in displaced bucket-handle tears.

When introducing the fixator through the meniscus, hold the instrument "like a dart" (Fig. 13). This provides tactile feedback, control, and reduces the risk of bending the needle. The repair is initiated from the center and continued to the periphery. This avoids gapping, ruffling, and "dog-ears." When inserting the suture through the meniscus, the optimal placement is perpendicular to the tear and parallel to the tibial plateau. The first anchor should be placed in the superior and posterior position of the tear. Both femoral and tibial surface side sutures should be placed to optimize the suture repair strength (double vertical mattress suture) [10]. The placement of the anchor is complete when it has penetrated the meniscus periphery and rests in the meniscosynovial junction. If the anchor has not deployed after placement of the first anchor, it is likely the device has not been placed deep enough. After placement of the first anchor, the FasT-Fix device is withdrawn to the meniscus edge while maintaining the device in full view. The second anchor is prepared in the instrument by advancing the trigger until the anchor is completely in the deployed position. The second anchor is placed inferior and anterior across the tear to create a vertical mattress. If the second anchor does not deploy, most often the anchor has not been advanced to the tip of the delivery needle.

Fig. 13 Placement of the suture fixator device

After placement of the anchors, the instrument is removed while holding tension on the pretied suture knot and suture strand that exit the working portal. The knot pusher may be used to slide and manually assist in cinching down the knot. If the knot does not cinch smoothly, it usually requires a more forceful pull, which may be facilitated by wrapping the suture around a finger like a pulley and placing steady traction while stabilizing the hand against the front of the knee. However, it is important to avoid overtightening as this may pucker the repair. Each suture should be placed approximately 4–5 mm apart until the repair appears stable.

Repair Augmentation and Future Directions

Recently, there has been increased support for use of biologic augmentation of repair methods. In cases of isolated (no concomitant anterior cruciate ligament reconstruction [ACLR]) meniscus repair in which concerns exist regarding healing (e.g., avascular tears, complex tears, or bucket-handle tears associated with a deformed tissue edge), then augmentation techniques to enhance vascularization and improve the biological healing response should be considered. It is well established that meniscus repair performed at the time ACLR is performed is associated with a higher healing rate than that of isolated repair [15]. The ability to capture an optimal healing environment that may be predicated upon introducing blood (or marrow) elements into the joint from the drilled tunnels (as well as from the protection afforded by the immediate postoperative protection phase) should be considered as a mechanism to enhance meniscal healing. Methods may include use of a fibrin clot or platelet-rich fibrin matrix placed into the site of repair [16, 17].

Various methods for preparation and insertion of a fibrin clot have been reported, and clinical evidence has documented increased healing rates in isolated tears and avascular regions treated with fibrin clot [16, 17]. The authors' preferred technique used in cases of isolated repair has been to obtain 30–50 mL of blood from the patient′s intravenous site and then sterilely transfer it to a glass container and stir it with a sintered glass rod. After formation of the clot, the rest of the blood is decanted off and the clot is blotted dry. Upon completion of the repair and with the arthroscopic fluid turned down, the clot is inserted under arthroscopic visualization using a grasper within a 5-mm-diameter cannula with its diaphragm removed and placed across the portal. The clot is then inserted under the repair site adjacent to the tibial surface of the tear. No specific sutures are used to anchor the clot.

More recently, much attention has been given to refined techniques that produce a more concentrated and volume-stable fibrin matrix that is rich in trapped platelets and associated growth factors (platelet-derived growth factor, transforming growth factor beta, epidermal growth factor, fibroblast growth factor, vascular endothelial growth factor, and endothelial cell growth factor). The platelet-rich fibrin matrix technique is performed by obtaining a smaller sample of autologous blood intraoperatively (approximately 10 mL) and placing it in a centrifuge for approximately 15 minutes; then, 6 minutes after centrifugation is completed, the fibrin matrix is retrieved and placed arthroscopically into the repair site in a similar fashion to that of the fibrin clot insertion method. Proprietary systems are currently available (Cascade Autologous Platelet System; MTF, Edison, NJ) to perform this method.

Rehabilitation

The postoperative rehabilitation program must be individualized to the patient's goals, abilities, and progression while remaining directed toward treating the patient's pathology. Most commonly, the protocol is directed toward treatment of isolated meniscus repair or repair in conjunction with an associated ACLR. Postoperatively, the patient is immediately placed in a brace or knee immobilizer and provided crutch training to facilitate ambulation and transfers. The brace or immobilizer remains until the patient exhibits adequate muscle function (typically 3 to 4 weeks). When repair is performed in conjunction with ACLR, partial weight-bearing is allowed in extension initially on crutches for up to 4 weeks and then advanced as comfort allows. When an isolated meniscus repair is performed, the patient also is kept to partial weight-bearing to protect the repair. Full weight-bearing is encouraged when antalgia and effusion subside and quadriceps firing is adequate. Range of motion from 0 to 90 degrees is begun immediately on postoperative day 1. Progression of motion, particularly in terminal flexion, is encouraged after 4 weeks, depending upon the repair site, size, geometry, and strength. Rehabilitation is prescribed on an individual basis: for example, a large bucket-handle tear extending through to the posterior horns and considered an "at-risk" repair is progressed more slowly over the first 2 months from the standpoint of terminal flexion, loading, and squats beyond 90 degrees.

A functional rehabilitation protocol is followed, progressing each patient depending upon comfort with range of motion, restoration of strength, and ultimately the ability to perform agility and functional drills. Running begins at approximately 8–10 weeks, with cutting activities at approximately 10–12 weeks, then incorporating sport-specific drills. Return to sports is usually at 4–6 months, when appropriate functional goals are reached and the patient no longer has point tenderness over the repair site.

Complications

The overall incidence of complications from arthroscopic meniscus surgery is 0.56% to 8.2% [18]. Meniscus repair surgery has a higher complication rate than that of meniscus resection, with reports as high as 18%. This is due to the significant manipulation of the soft tissues as well as surgical dissection in open repairs [19].

The most commonly reported complications include infection, deep vein thrombosis (DVT), vascular injury, and neurologic injury. The rate of infection is 0.23% to 0.42%, with increasing incidence associated with extended operating time, extended tourniquet time, performance of multiple concurrent procedures, and a history of prior surgeries [20]. There are also published reports of increased incidence of infection associated with intraarticular corticosteroid injections intraoperatively [21]. Currently, however, there is no clear consensus on use of prophylactic perioperative antibiotics. When an infection has been diagnosed after a repair, it is appropriate to leave the implant/sutures in place; however, there is a higher associated failure rate. Other complications include vascular injury and DVT. The incidence of DVT ranges from 1.2% to 4.9% after arthroscopic knee surgery [22]. The overall incidence of vascular complications is 0.54% to 1.0%, with complications including popliteal artery injury, pseudoaneurysm, and arteriovenous fistulae [23]. While repairing the meniscus, the surgeon pierces the meniscosynovial junction, which puts these structures at risk.

Neurologic complications include direct or indirect nerve injury, including complex regional pain syndrome. The most common complications associated with the inside-out and outside-in techniques are traumatic neuropathy to the saphenous or peroneal nerves. The overall incidence of this is reportedly 0.06% to 2.0% [24]. Medial meniscus repairs using an inside-out or outside-in technique are more likely to result in saphenous neuropathy or neuropraxia, with reports of up to 43% of cases [25].

The all-arthroscopic implant fixators have been reported to be associated with pull-out and pull-through device failure, migration and breakage, cystic hematoma, foreign body reaction, transient soft tissue inflammation, and chondral injury [26–29]. The issue of chondral abrasion secondary to meniscal implant fixators is particularly worrisome and has prompted a trend toward use of lower profile devices as well as newer hybridized suture-based systems [11].

Results

Outcomes after meniscal repair have been reported by DeHaven et al. with 100% retrieval of 33 cases (average age, 18.9 years; average follow-up, 10.9-years [range, 10.1–13 years]) treated with open meniscal repair. They noted a 79% long-term survival rate [30]. Early reports of results after the inside-out repair technique are provided by Scott et al. in 260 repairs performed in 240 patients (average age, 22 years) at an average follow-up of almost 2 years. Concomitant anterior cruciate ligament (ACL) reconstruction was performed in 80% of cases. The results indicated that 62% of repairs were healed at arthroscopic second-look or at arthrogram compared with 17% incompletely healed and 21% not healed. Of note, based on clinical and subjective evaluation, 92% of cases were stable and 80% returned to active sports [31]. In a comprehensive review of 117 consecutive inside-out repairs by Cannon and Vittori, 90 cases were reported: 68 repairs with concomitant ACLR and 22 isolated repairs [15]. Overall rate of clinical success was 82%, with 93% of the ACL-associated cases successful compared with 50% of the isolated cases. Outside-in technique results have been published by Rodeo, who found that in 90 patients (average age, 25 years; average follow-up, 46 months [range, 36–89 months]), overall 87% had a successful outcome [32]. Failure was noted in 38% of the unstable knees, 15% of the stable knees, and 5% of the ACL-reconstructed knees. The more recently introduced meniscal repair fixators have been reported on, although long-term published data remain limited. Jones and coauthors reported on a retrospective series of 38 patients undergoing meniscal repair with the Meniscal Arrow at 29.7-month follow-up. In 21 cases in which concomitant ACLR was performed, no clinical failures (defined as reoperation) were noted, and in 17 isolated repair cases, success was noted in 93%. The authors noted, however, that there was a 31.6% incidence of transient local soft tissue inflammation related to device migration, length, prominence, and possible reaction to the resorbable materials [26]. Sgaglione reported a consecutive series of 109 meniscal repairs performed using an all-arthroscopic hybrid technique using the Meniscal Arrow and the T-Fix system. The study group was followed prospectively, with an average age of 28 years (range, 15–49 years) and an average follow-up of 3.2 years (range, 2–4.4 years). The Meniscal Arrow was used exclusively for the repair in 55% of cases, and Arrows and the T-Fix were used in 45% of cases. An associated ACLR was performed in 72 (60%) cases, and an isolated repair was performed in 37 (40%) cases. All isolated repairs were treated with an autologous fibrin clot technique. All patients received uniform postoperative care, with 4 weeks of bracing in extension beginning with immediate range of motion 0–90 degrees, increasing beyond that at 4 weeks and partial weight-bearing on crutches for 4 weeks. No difference was noted at outcome between the Arrow-alone group and the hybrid group. The overall failure rate, defined as the need to return for meniscal surgery, was 5.5%, with the isolated repair cases noted to have a failure rate of 10.8% (four failures), whereas in the ACL-reconstructed cases there was a failure rate of 2.7% (two cases) [33].

Clinical Pearls/Summary

Meniscal repair in select active individuals with repairable meniscal tears should be performed whenever indications are met and appropriate patient counseling regarding outcomes is addressed. As the techniques and devices continue to improve, the decision to select one technique or techniques over another should ultimately be based on the sound evaluation of and experience associated with a particular device and its safety and potential efficacy. All repair methods are associated with their own particular learning curves. Morbidity and technical pitfalls will be reduced by addressing the specific learning curve issues, instituting surgical technique pearls, and adhering to precise indications when using a specific repair device. In general, meniscal fixators and implants should be used for vertical, longitudinal red-white tears that are not peripheral detachments and are associated with at least a 2- to 3-mm rim width in order to provide optimal barb-tissue contact. Newer all-arthroscopic suture-based systems as well as inside-out or outside-in sutures may be best used for repair of more complex tear patterns or less vascular tears with less optimal tissue viability or with significant deformity or deformation as seen in large displaced bucket-handle tears. In addition, in cases of peripheral capsular detachment of the meniscus or in repairing meniscal allografts, sutures should be used. On the horizon, augmentation of biologic healing through the introduction of growth factors using autologous platelet-rich concentrates remains a promising and evolving clinical method.

Case Reports

Case 1

Chief Complaint and Patient History: A 27-year-old woman who is a competitive basketball player injured her knee during a game while landing in a "funny position." She felt immediate pain and reports hearing a "pop." The knee swelled up, and she currently reports difficulty ambulating because of pain and the knee giving out. She further describes an episode of knee "locking" that resolved on its own. She reports undergoing an ACLR using bone–patellar tendon–bone autograft 2 years ago.

Physical Exam: She walks with an antalgic gait. The knee has a mild effusion and diffuse tenderness to palpation, with maximum tenderness over the medial joint line. Her range of motion is 15–60 degrees.

Imaging: X-rays: negative. MRI scan shows a bucket-handle tear of the medial meniscus. The ACL appears intact.

Surgery/Treatment: The patient underwent a medial meniscus repair (Fig. 14A–E) using six all-arthroscopic suture fixators placed in vertical mattress fashion. Postoperatively, the patient was made non-weight-bearing, with continuous passive motion begun immediately. The patient healed well, advanced to strengthening and plyometrics by 10 weeks postoperatively, and returned to sports at 6 months.

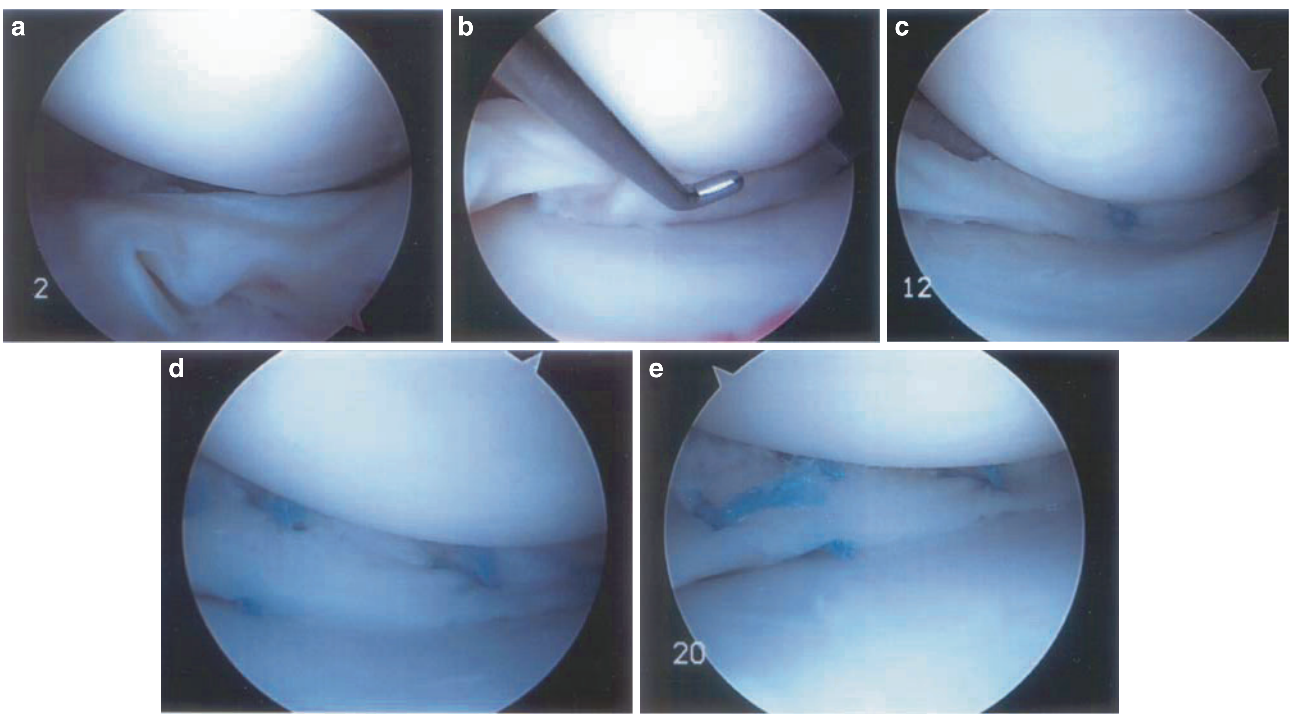


Fig. 14 Case 1. (**A**) Bucket-handle meniscus tear. (**B**) The reduction of the meniscus is performed with an arthroscopic probe. (**C**) The first arthroscopic knot is placed with an all-inside technique in the posterior aspect of the reduced meniscus tear. (**D, E**) The remainder of the knots are placed by sequentially reducing the remainder of the tear

Discussion: The decision to repair the meniscus was crucial to the maintenance of the knee joint biomechanics in a young athlete with many years of use remaining. Use of an all-arthroscopic technique was offered because of the patient's commitment to a stringent rehabilitation and desire to return to play in a shorter period of time. She maintained that commitment postoperatively, healed well without complication, and returned to play at 6 months.

Case 2

Chief Complaint and Patient History: A 42-year-old athletically active man complains of instability and associated pain. He reports a history of undergoing an ACLR with bone–patella tendon–bone allograft 17 years ago. Approximately 3 years ago, the patient began experiencing pain and episodic subluxation.

Physical Exam: The patient exhibits a normal gait. He has a moderate joint effusion with a range of motion of 0–125 degrees. He has tenderness to palpation on the medial joint line. The patient appears to have ACL laxity on exam, however has an appreciable end point.

Imaging: X-rays were normal with minimal medial compartment narrowing. MRI scan reveals a medial meniscus tear with the ACL attenuated. The hardware (screw) is intact with the femoral fixation in an anterior position.

Surgery/Treatment: Arthroscopic evaluation noted the ACL was partially torn (Fig. 15A) and attenuated. The patient underwent a revision ACLR with a bone–patella tendon–bone allograft with a medial meniscus repair (Fig. 15B, C). The ACL was fixed with two bioabsorbable interference screws, and the medial meniscus was repaired using three all-arthroscopic suture fixators. The patient remained partially weight-bearing after the surgery, with immediate passive range of motion. He was advanced to running at approximately 5 months, with return to full activities shortly thereafter.

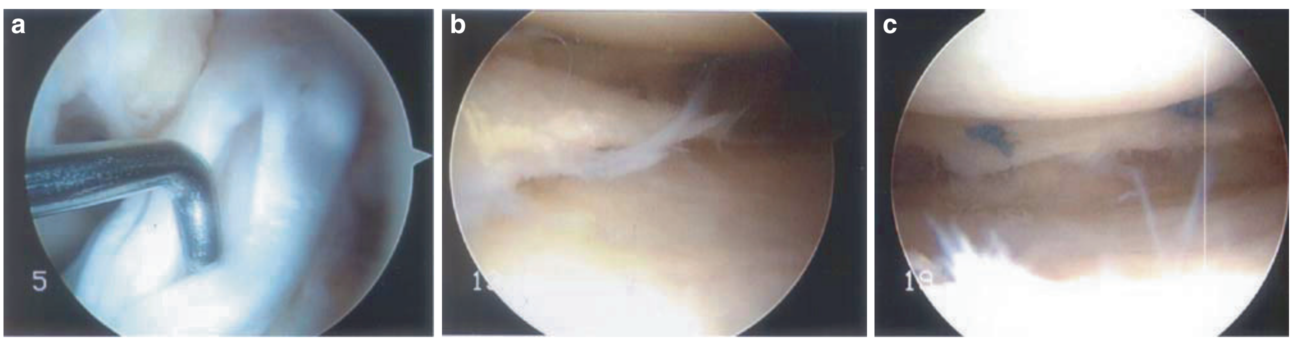


Fig. 15 Case 2. Meniscus repair with ACL revision. (**A**) Longitudinal partial tear of the ACL. (**B**) Radial tear of the medial meniscus. (**C**) All-inside repair of the medial meniscus

Discussion: The decision to perform a revision ACLR in addition to the meniscus repair was made because of the patient's age, activity level, and desire to avoid joint replacement surgery. Use of all-arthroscopic meniscus repair was chosen because the hemarthrosis created from the bone tunnel revision provided the patient the bioactive factors known to improve healing. It was believed that, in this setting, an all-inside technique offered the patient an excellent chance of healing in the face of ACLR.

Case 3

Chief Complaint and Patient History: The patient is a 28-year-old active man who presents with right knee pain and a localized mass that he has had for approximately 3 years. He denies any trauma to the knee and reports increasing pain with activities. He denies mechanical locking or symptoms of giving way.

Physical Exam: The patient is a young athletic man with a normal gait. He has full range of motion with no evidence of effusion, crepitus, or mechanical clicking/locking. He has lateral joint-line tenderness with a 3-cm mass centered over the joint line. There is no evidence of ligamentous pathology.

Imaging: X-rays show no evidence of arthritic changes or etiology of the mass. An MRI scan reveals a large horizontal tear of the body of the lateral meniscus with an associated parameniscal cyst.

Surgery/Treatment: Arthroscopic evaluation noted a horizontal meniscus tear. (Fig. 16A, B) The patient underwent a right knee arthroscopy with a partial meniscectomy of the irreparable portion with repair of the peripheral meniscus using the all-arthroscopic suture fixators (Fig. 16C, D).

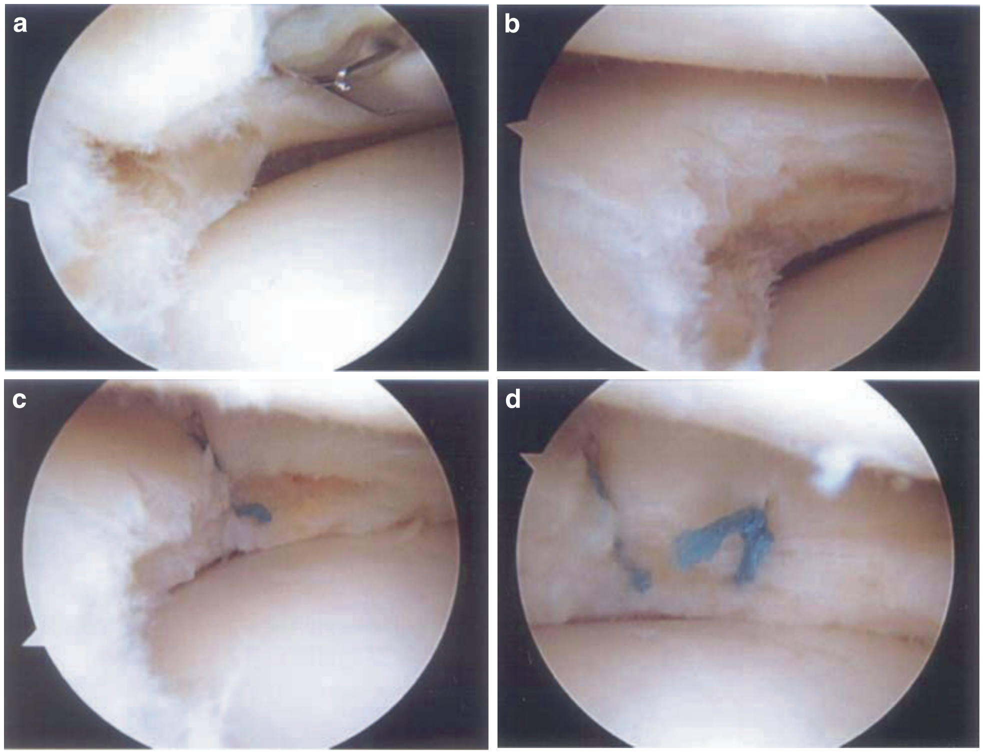


Fig. 16 Case 3. Partial meniscectomy and repair around horizontal tear with parameniscal cyst. (**A**) Horizontal meniscus tear, the source of the parameniscal cyst. (**B**) A partial meniscectomy is performed to debride the leaves of the tear back to a stable point. (**C, D**) Vertical mattress sutures were placed using an all-inside technique to close down the periphery of the tear and prevent the cyst from reforming

Discussion: The decision to perform a meniscus repair was made intraoperatively. Upon inspection of the meniscus tear, the patient was noted to have a large horizontal tear that extended from the white-white zone of the meniscus to the entrance of the parameniscal cyst at the meniscosynovial junction. A partial meniscectomy was performed upon the central portion of the meniscus as this was determined to be unstable. The peripheral aspect of the tear was repaired with two vertical horizontal mattress sutures using the all-arthroscopic suture fixator technique. It was believed that a repair of this small portion of the periphery was worth attempting in order to maintain the meniscus hoop stresses and obstruct the entrance to the parameniscal cyst. Given the patient's young age and activity level, he was willing to undergo the repair in an effort to prevent the almost certain arthritic changes that would result with a subtotal meniscectomy.

Case 4

Chief Complaint and Patient History: The patient is a 17-year-old male soccer player who reports sustaining a twisting injury to his knee a week ago. He reports swelling and pain at the time of injury. The day prior to his doctor's visit, the patient reports locking of the knee after bending down to pick something up. He is able to bear weight but has significant pain with flexion and extension. He denies the knee giving way.

Physical Exam: The patient ambulates with a mild antalgic gait and sits with the knee in approximately 20 degrees of flexion. He has a moderate-size effusion. The patient's range of motion is limited to approximately 60 degrees of flexion with resistance to full extension secondary to pain and a palpable mechanical block. He has tenderness to palpation along the medial joint line. Apley compression is positive. Lachman test and anterior and posterior drawer tests are negative.

Imaging: X-rays are normal. MRI scan shows a bucket-handle tear of the body of the medial meniscus.

Surgery/Treatment: The patient underwent arthroscopic reduction of the meniscus tear with subsequent repair of the meniscus. The meniscus was repaired in inside-out fashion with a medial accessory incision.

Discussion: The decision to perform an inside-out meniscus repair on this patient was chosen to give the patient the best opportunity to heal. Given his young age, a large (4 cm) isolated bucket-handle meniscus tear must be accurately reduced and repaired with the strongest fixation method available. After reduction and trephination of the tear site, vertical mattress sutures were placed through arthroscopic cannula from the anterolateral portal (Fig. 17A, B). The patient remained non-weight-bearing for 4 weeks. Advancement of motion and strength were allowed at 6 weeks. At 3 months, the patient was performing aerobic and sport-specific training. He returned to soccer at approximately 6 months.

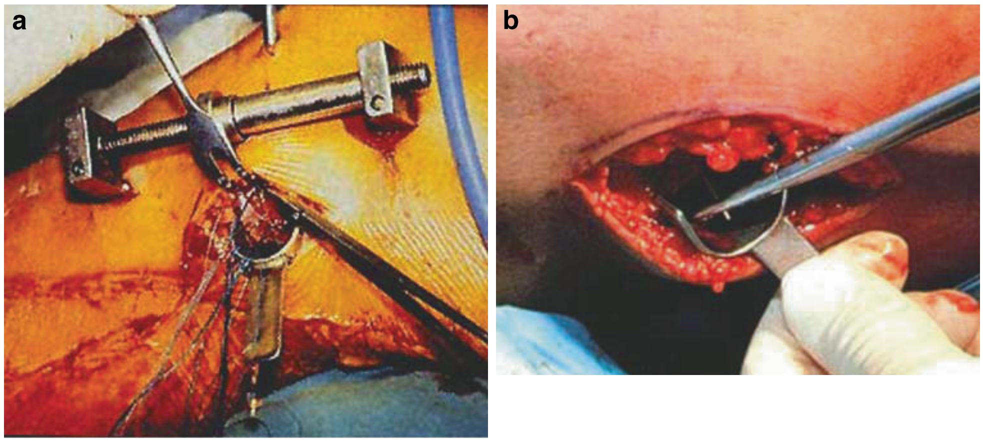


Fig. 17 Case 4. Posteromedial accessory incision and suture passage for inside-out meniscus repair. (**A**) Medial incision used to perform an inside-out meniscus repair. A small speculum was used to protect the neurovascular structures as an assistant catches the needles as they pass out of the capsule. (**B**) Multiple vertical mattress sutures are passed and tied down to the capsule sequentially

Case 5

Chief Complaint and Patient History: The patient is a 34-year-old man with a history of knee pain for approximately 1 month duration. He reports sustaining an injury to his knee while playing tennis. He complains of pain with ambulation and persistent swelling of the knee. He denies any episodes of locking or giving way.

Physical Exam: The patient is an athletic man who ambulates with mild antalgia. He has a mild palpable effusion and maintains full range of motion. He has tenderness to palpation over the lateral joint line, but no tenderness to palpation medially. Lachman test and anterior and posterior drawer tests are negative.

Imaging: X-rays are normal. MRI scan shows no evidence of pathology in the posterior horns or body of the meniscus. There are "signal changes" in the midanterior horn of the lateral meniscus.

Surgery/Treatment: The patient underwent a diagnostic arthroscopy after failing nonoperative treatment. Arthroscopic exam showed a 3-cm longitudinal tear in the anterior horn of the lateral meniscus. The patient underwent repair of the tear with hybrid fixation including an all-arthroscopic fixator and outside-in mattress suture (Fig. 18A–C).

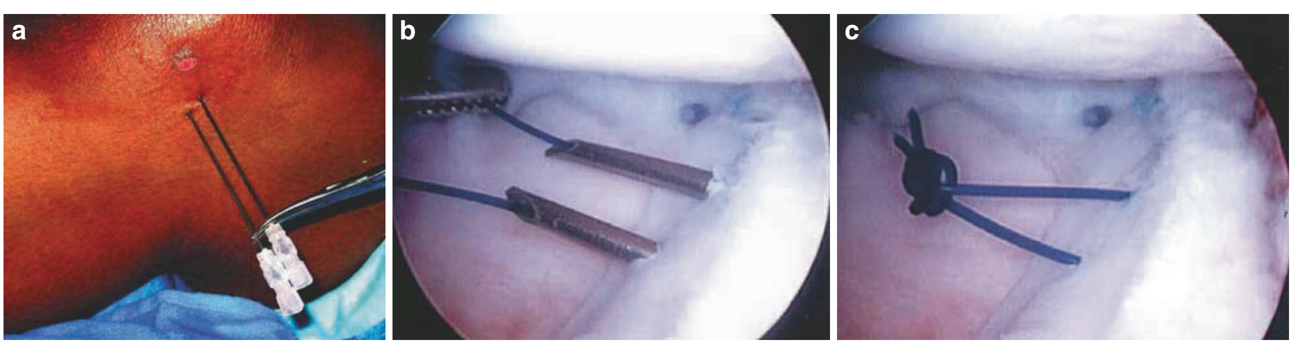


Fig. 18 Case 5. Outside-in meniscus repair and percutaneous needle placement for outside-in meniscus repair. (**A**) Intraoperative photo of two large-bore spinal needles with use of the outside-in meniscus repair technique. (**B**) The spinal needles as they traverse the meniscus tear. Two sutures are then shuttled through the needles and both retrieved out of one arthroscopic portal. (**C**) The sutures are tied to each other, and the knot is passed back through the portal and reduced to the meniscus tear

Discussion: The patient was initially treated conservatively, given no definite source of his pain. Although use of MRI has greatly increased the sensitivity and specificity of diagnosis of meniscus tears, reading of anterior horn tears continues to be user dependent and imprecise. At times, arthroscopic examination is performed for diagnostic purposes. The patient sustained an anterior horn tear that extended into the body of the meniscus. Given the difficulty in reducing and repairing these tears all-arthroscopically, an outside-in repair was performed anteriorly to augment the arthroscopic fixator used in the body of the tear. The proximity of the tear anteriorly is located far from neurovascular structures that are typically of concern with posterior or body-type tears; therefore, it was thought safe to perform an outside-in repair with percutaneous techniques.

References

1. Garrett WE, Swiontkowski MF, Weinstein JN, Callaghan J, Rosier RN, Berry DJ, Harrast J, Derosa GP. American board of orthopaedic surgery practice of the orthopaedic surgeon: Part II, certification examination case mix. *J Bone Joint Surg Am* 2006;88:660–667.
2. Walker PS, Erkman MJ. The role of the menisci in force transmission across the knee. *Clin Orthop* 1975;109:184–192.
3. Baratz M, Fu F, Mengato R. Meniscal tears: the effect of meniscectomy and of repair on intraarticular contact areas and stress in the human knee. A preliminary report. *Am J Sports Med* 1986;14: 270–275.
4. Renstrom P, Johnson RJ. Anatomy and biomechanics of the menisci. *Clin Sports Med* 1990;9:523–538.
5. Arnoczky SP, Warren RF. The microvasculature of the meniscus and its response to injury: an experimental study in the dog. *Am J Sports Med* 1983;11:131–141.
6. Petersen W, Tillman B. Age related blood and lymph supply of the knee menisci. A cadaver study. *Acta Orthop Scand* 1995;66: 308–312.
7. Cooper DE, Arnoczky SP, Warren RF. Arthroscopic meniscal repair. *Clin Sports Med* 1990;9:589–607.
8. Magee T, Williams W. 3.0 T MRI of meniscal tears. *Am J Radiol* 2006;187:371–375.
9. Thaete FL, Britton CA. Magnetic resonance imaging. In *Knee Surgery,* Fu FH, Harner CD, Vince KG, Miller MD (eds.) Vol. 1. Philadelphia: Williams & Wilkins, 1994, pp. 325–352.
10. Borden P, Nyland J, Caborn D, Pienkowski D. Biomechanical comparison of the fast-fix meniscal repair suture system with vertical mattress sutures and meniscus arrows. *Am J Sports Med* 2003;31:374–378.
11. Kurzweil P, Tifford C, Ignacio E. Unsatisfactory clinical results of meniscal repair using the meniscus arrow. *Arthroscopy* 2005;21:905.e1–905.e7.
12. Gifstad T, Grøntvedt T, Drogset J. Meniscal repair with biofix arrows: results after 4.7 years follow-up. *Am J Sports Med* 2007;35:71–74.
13. Lee G, Diduch D. Deteriorating outcomes after meniscal repair using the meniscus arrow in knees undergoing concurrent anterior cruciate ligament reconstruction increased failure rate with long-term follow-up. *Am J Sports Med* 2005;33:1138–1141.
14. Seibold R, Dehler C, Boes L, Ellerman A. Arthroscopic all-inside repair using the meniscus arrow: Long-term clinical follow-up of 113 patients. *Arthroscopy* 2007;23:394–399.
15. Cannon W, Vittori J. The incidence of healing in an arthroscopic meniscal repairs in ACL-reconstructed knees versus stable knees. *Am J Sports Med* 1992;20:176–181.
16. Henning C, Lynch M, Yearout K, Verquist S, Stallbaumer R, Decker K. Arthroscopic meniscal repair using an exogenous fibrin clot. *Clin Orthop* 1990;252:64–72.
17. Henning C, Yearout K, Vequist S, Stallbaumer R, Decker K. Use of the fascia sheath coverage and exogenous fibrin clot in the treatment of complex meniscal tears. *Am J Sports Med* 1991;19: 626–631.
18. Small N. Complications in arthroscopic surgery performed by experienced arthroscopists. *Arthroscopy* 1988;4:215–221.
19. Sherman O, Fox J, Snyder S, Del Pizzo W, Friedman M, Ferkel R. Arthroscopy—"No problem surgery." Analysis of complications in 2640 cases. *J Bone Joint Surg Am* 1986;68: 256–265.
20. Armstrong R, Bolding F, Joseph R. Septic arthritis following arthroscopy, clinical syndromes and analysis of risk factors. *Arthroscopy* 1992;8:213–223.
21. Montgomery S, Campbell J. Septic arthritis following arthroscopy and intraarticular steroids. *J Bone Joint Surg Am* 1989;71: 540–544.
22. Jauregito J, Greenwald A, Wilcox J, Paulos L, Rosenberg T. The incidence of deep vein thrombosis after arthroscopic knee surgery. *Am J Sports Med* 1999;27:707–710.
23. Kim T, Savino R, McFarland E, Cosgarea A. Neurovascular complications of knee arthroscopy. *Am J Sports Med* 2002;30: 619–629.
24. Rodeo S, Forster R, Weiland A. Neurological complications due to arthroscopy. *J Bone Joint Surg Am* 1993;75:917–926.
25. Stone R, Miller G. A technique of arthroscopic suture of a torn meniscus. *Arthroscopy* 1985;1:226–232.
26. Jones H, Lemos M, Wilk R, Smiley R, Gutierrez R, Schepsis A. Two-year follow-up of meniscal repair using a bioabsorbable arrow. *Arthroscopy* 2002;18:64–69.
27. Anderson K, Marx R, Hannafin J, Warren R. Chondral injury following meniscal repair with a biodegradable implant. *Arthroscopy* 2000;16:749–753.
28. Ross G, Grabill J, McDevitt E. Chondral injury after meniscal repair with bioabsorbable arrows. *Arthroscopy* 2000;16:754–756.
29. Sgaglione N. Complications of meniscus surgery. *Sports Med Arthrosc Rev* 2004;12:148–159.
30. DeHaven K, Lohrer W, Lovelock J. Long term results of open meniscal repair. *Am J Sports Med* 1995;23:524–530.
31. Scott G, Jolly B, Henning C. Combined posterior incision and arthroscopic intra-articular repair of the meniscus. *J Bone Joint Surg Am* 1986;68:847–861.
32. Rodeo S. Arthroscopic meniscal repair with use of the outside-to-inside technique. *J Bone Joint Surg Am* 2000;82:127–141.
33. Sgaglione NA. Meniscus repair: update on new techniques. *Tech Knee Surg* 2002;1:113–127.

Meniscal Allograft Transplantation

David M. Junkin, Jr., Jayesh K. Patel, and Darren L. Johnson

Introduction

Not all meniscal tears are reparable, resulting in partial or complete meniscectomy as the course of treatment. With the incidence of meniscectomy approximately 61 per 100,000 annually [1], the treatment for the meniscectomized patient has long been the dilemma of the treating physician, particularly in the young patient (less than 50 years old). The association of advanced progression of arthritic changes in the meniscectomized knee is well known. In 1948, Fairbanks described the radiographic changes after meniscectomy [2]. Numerous studies since then have correlated the observed radiologic changes with tibiofemoral arthritis. These changes, however, poorly predict clinical symptoms and pain.

Baratz et al. [3] in 1986, using a human cadaver model, studied the effects of meniscectomy on the contact areas of the knee. With loss of the medial meniscus, an approximately 75% decrease in contact area and 235% increase in the peak contact pressures were described. This increased stress upon the articular cartilage is associated with biochemical changes of the proteoglycan matrix and an increase in hydration [4]. Animal models also have demonstrated macroscopic and microscopic changes to the articular surface after meniscectomy [4, 5].

The surgical management of meniscal injuries has evolved over the past few decades as the understanding of the importance of the menisci has become more apparent. The preservation of the menisci has been the main goal in therapy for meniscal injuries. On occasion, a total meniscectomy is the only course of treatment due to an irreparable injury. The subsequent gonarthrosis and pain leave few surgical options. Unicondylar or total joint arthroplasty is a feasible option in the older, low-demand population. The limited longevity of joint replacements is not ideal in the younger, more active individual who may require multiple revision arthroplasties throughout his or her lifetime. Meniscal allograft transplantation is a viable option for the restoration of normal knee mechanics and preservation of the native joint in patients deemed too young for surgical arthroplasty, as well as for improvements in laxity in the grossly unstable knee.

D.M. Junkin (✉)
Orthopaedic Specialty Center, Willow Grove, Pennsylvania

History

Milachowski et al. reported the first meniscal allograft transplantation in 1984 and subsequent follow-up results in 1989 [6]. Later, Garrett and Stevenson [7] described an open technique with use of a parapatellar arthrotomy and an ipsilateral collateral ligament takedown with a bony block from the femoral origin. Early results were promising. The advancement in arthroscopic techniques and technology has allowed the development of less invasive surgery, decreasing morbidity and facilitating rehabilitation. Less invasive surgical techniques were described by Whipple [8] and Wirth and Kohn [9]. Both used a 12-mm arthrotomy and bone plug fixation. Arthroscopy has become the procedure of choice due to the decreased morbidity of less soft tissue dissection. The surgical technique for meniscal allograft transplantation, however, continues to be refined.

Anatomy

The menisci are C-shaped fibrocartilaginous structures. Sixty percent to 70% of the dry weight [10] of the meniscus is type I collagen arranged in circumferential and radial bundles. This arrangement disperses compressive forces and resists shear forces, respectively [11]. Water accounts for approximately 70% of the menisci, and this composition provides further resistance to compressive loads [10]. Supplied by the superior and inferior medial and lateral genicular arteries, a perimeniscal capillary plexus provides the peripheral 10% to

B.P. McKeon et al. (eds.), *Knee Arthroscopy*, DOI 10.1007/978-0-387-89504-8_4,

30% of the adult meniscus with its blood supply [12, 13]. The remaining central portion of the meniscus obtains its nutrients via diffusion [14]. This avascular zone accounts for the poor healing potential of the meniscal tissue.

The medial meniscus is oval shaped, covering approximately 30% of the medial tibial plateau, and the semicircular lateral meniscus covers a larger portion of the tibial articular surface, roughly 50% [15]. Centrally, the medial meniscus attaches to the fibers of the deep medial collateral ligament and to the tibia via the meniscotibial ligament. Posterior attachment to the capsule is shared with fibers of the semimembranous tendon, causing the posterior horn of medial meniscus to retract with knee flexion [16]. This firm attachment about its periphery allows only an average of 5.2 mm of excursion [17], likely accounting for the increased susceptibility to injury. The lateral meniscus has no attachment to the fibular collateral ligament, allowing greater mobility of up to 11 mm [18].

An understanding of the meniscal insertion sites or footprints is critical for anatomic placement of the meniscal allograft. A cadaveric study by Johnson et al. [18] identified the bony and arthroscopic landmarks of the meniscal insertion sites (Fig. 1A–D). The anterior horn of the medial meniscus inserts an average of 7 mm anterior to the anterior cruciate ligament (ACL) in line with the tibial eminence. The surface area of the anterior horn insertion site approximates

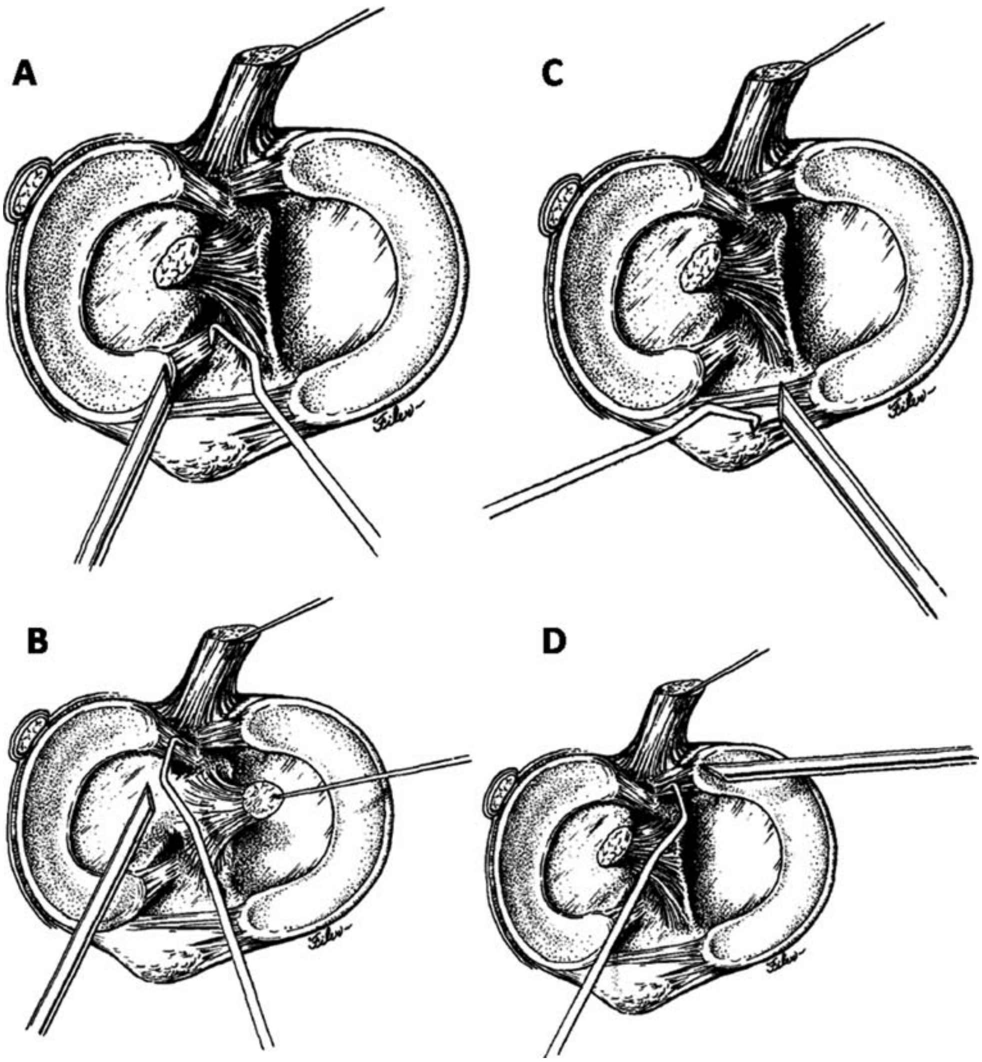


Fig. 1 Arthroscopic visualization and arthroscopic drill guide placement for a lateral meniscus: For both the (**A**) anterior and (**B**) posterior horns, the arthroscope is placed in the anterolateral portal with the drill guide used in the anteromedial portal. Arthroscopic visualization and drill guide placement for a medial meniscus: (**C**) For the anterior horn, the arthroscope is placed in the anteromedial portal with the arthroscopic drill guide used in the anterolateral portal. (**D**) For the posterior horn, the arthroscope is placed in the posteromedial portal with the arthroscopic drill guide again placed in the anterolateral portal. (From Johnson DL, Swenson TM, Livesay GA, Aizawa H, Fu FH, Harner CD. Insertion-site anatomy of the human menisci: gross, arthroscopic, and topographical anatomy as a basis for meniscal transplantation. *Arthroscopy* 1995;11(4):386–394. Copyright © 1995 Arthroscopy Association of North America. Reprinted with permission of Elsevier.)

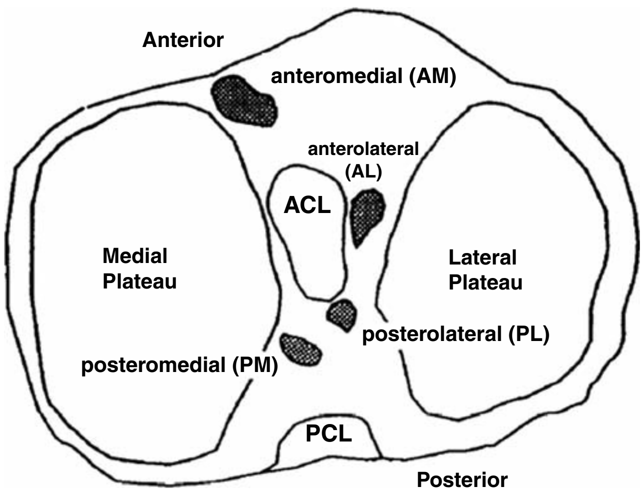


Fig. 2 Axial view of a right tibial plateau, showing the meniscal horn insertions. Note the intimate association of the lateral meniscal horn insertions with that of the ACL and also their close proximity to one another. (From Johnson DL, Swenson TM, Livesay GA, Aizawa H, Fu FH, Harner CD. Insertion-site anatomy of the human menisci: gross, arthroscopic, and topographical anatomy as a basis for meniscal transplantation. *Arthroscopy*1995;11(4):386–394. Copyright © 1995 Arthroscopy Association of North America. Reprinted with permission of Elsevier.)

61 mm^2 but is difficult to visualize arthroscopically without debridement of the patellar fat pad, as this bony insertion lies at the junction of the medial tibial plateau and the anterior tibia. The intermeniscal ligament attaches to the posterior half of the anterior horn. The posterior horn insertion site is on the downslope of the posterior intercondylar fossa, lying directly anterior to the posterior cruciate ligament (PCL) tibial insertion. The anterior horn of the lateral meniscus is laterally adjacent to the ACL tibial footprint and inserts directly anterior to the lateral tibial spine. The posterior horn attachment is only 6–10 mm posterior to the anterior horn, inserting directly posterior to the lateral tibial spine and anterior to the insertion of the medial meniscus posterior horn (Fig. 2).

Arthroscopic localization of the meniscal insertion sites is critical for anatomic placement of the meniscal allograft. Landmarks for the visualization of the meniscal footprints are summarized in Table 1. A modified notchplasty as discussed later will aid in the crucial localization of the posterior meniscal insertion sites without the need for a posterior arthroscopic portal.

Function

The primary function of the menisci is load sharing, with secondary functions in shock absorption and stability. The triangular or wedge shape of the meniscus, seen on cross section, improves knee congruency and increases contact area of the tibiofemoral joints. A study by Seedhom [19] has shown that the medial meniscus transmits up to 50% of the compressive load in the medial compartment and that the lateral meniscus transmits up to 70% of the compressive load in the lateral compartment. The circumferential and radial collagen fibers allow the menisci to absorb axial force as the fibers elongate and push toward the periphery, converting the load into tensile strain. As these hoop stresses are removed, the meniscus can therefore resume its original shape [15]. Loss of meniscal tissue after partial or complete meniscectomy has been reported to significantly increase peak stresses and decrease contact area, as discussed earlier in the study by Baratz et al. [3].

The viscoelastic properties of the menisci provide for a secondary function of shock absorption. Voloshin and Wosk [20] reported a decrease in the shock absorption capabilities of the knee by 20% after meniscectomy.

Table 1 Arthroscopic portals and landmarks for each meniscal horn bony insertion site

	Portal for arthroscope	Portal for endoscopic guide	Arthroscopic landmarks
Anterior horn medial meniscus	Anteromedial	Anterolateral	1. Anterior border ACL tibial insertion 2. Articular margin of anteromedial tibial plateau 3. Anterior intercondylar fossa
Posterior horn medial meniscus	Posteromedial	Anterolateral	1. PCL 2. Medial tibial spine 3. Articular margin posteromedial tibial plateau
Anterior horn lateral meniscus	Anterolateral	Anteromedial	1. A half of ACL tibial insertion 2. Lateral tibial spine 3. Articular margin on anterolateral tibial plateau
Posterior horn lateral meniscus	Anterolateral	Anteromedial	1. Posterior border ACL tibial insertion 2. Lateral tibial spine 3. Articular margin on posterolateral tibial plateau

Source: From Johnson DL, Swenson TM, Livesay GA, Aizawa H, Fu FH, Harner CD. Insertion-site anatomy of the human menisci: gross, arthroscopic, and topographical anatomy as a basis for meniscal transplantation. Arthroscopy 1995;11(4):386–394. Copyright © 1995 Arthroscopy Society of North America. Reprinted with permission of Elsevier.

In the ACL-deficient knee, the medial meniscus acts as secondary stabilizer of anteroposterior translation. Anterior tibial translation is increased 58% to 90% after complete medial meniscectomy in an ACL-deficient knee [12], with the posterior horn especially important in contributing to joint stability [21]. Similarly, the lateral meniscus provides posterolateral rotatory stability by increasing the concavity of the tibial articulation with the lateral femoral condyle.

Surgical Indications

As noted in a review of the literature by Matava [22], many authors are in agreement regarding the surgical indications for meniscal allograft transplantation: "This procedure is indicated for the active, physiologically young patient (generally less than 50 years of age) who has undergone either a complete or near-complete meniscectomy and has pain in the involved compartment prior to the development of moderate to severe arthrosis" [22]. Radiographic measurements have been defined to determine the degree of arthritis. Joint space narrowing of less than 2–3 mm has become the agreed standard. Rosenberg et al. [23] described such measurement with a weight-bearing posteroanterior radiograph in 45 degrees of knee flexion. Others use an anteroposterior weight-bearing radiograph in full knee extension to determine any joint narrowing [24, 25]. Magnetic resonance imaging (MRI) has also been used to evaluate the articular surface [26], but its use is limited among authors. However, the presence of bipolar edema on the MRI scan will suggest bony involvement and advanced articular surface wear.

Serious articular disease, whether determined by Fairbanks [2] changes on radiographs or by advanced Outerbridge [27] stages (III or IV) by arthroscopic evaluation, are agreed by many as exclusion criteria for allograft meniscal transplant. By far, joint space narrowing, osteophyte formation, and flattening of the femoral condyle as evidenced by radiographic evaluation are the most common contraindication for performing this procedure [28]. Obesity is a concern, though no literature to support its use as an exclusion measure exists.

Knee stability is critical for an optimal environment for allograft survival. Any instability of the knee or malalignment is a contraindication for meniscal transplant as an isolated procedure. Any instability or lower extremity malalignment that can be addressed concurrently should not preclude one from performing a meniscal replacement. Meniscal transplantation combined with realignment osteotomies and/or ACL reconstruction has been reported in the literature with successful outcomes. Failure to assess both malalignment or ligamentous instability has resulted in early failure of meniscal replacement surgery [7, 29].

Systemic inflammatory conditions such rheumatoid arthritis or a history of knee infection also are exclusion criteria. Soft tissue disease and lack of full or normal knee range of motion may alter normal knee kinematics. This alteration may not provide a suitable environment for allograft survival. The history of an intraarticular infection of the knee may have a theorized increased risk of recurrent infection after the implantation of allograft material. As such, many recommend the avoidance of transplantation of foreign material into a site of previous infection.

Patient Evaluation

A thorough history and physical examination are essential for determining the appropriate patient for a meniscal transplant. A history of the initial injury and any prior additional surgical procedures must be known and discussed with the patient. Along with a progressive worsening of joint-line pain after a near complete or complete meniscectomy, the patient must have joint-line tenderness isolated only to the involved compartment. The knee range of motion is to be assessed and must be within normal limits. Any malalignment or ligamentous laxity should be determined. Appropriate radiographic studies should be performed to grade any possible arthritic change, joint space narrowing, or lower-extremity deformity. Preoperative planning is critical for survival of the allograft meniscus and a successful surgical outcome.

If the status of the articular cartilage and the degree of meniscal resection is unknown, a diagnostic arthroscopy may be warranted. This provides the advantage for determining the necessity of any additional procedures that may be indicated and direct assessment of the condition of the joint surface. If simultaneous osteochondral allograft transplant or autologous chondrocyte implantation is a possibility, a diagnostic arthroscopy is strongly recommended for preoperative planning, particularly if the patient has not had surgical intervention for more than 1 year, as the current status of the (osteo)chondral lesion may be unclear.

The patient must have a complete understanding of the expectations and goals of meniscal transplant surgery. One must understand this procedure is primarily for pain relief and hopefully for slowing of the arthritic progression in the involved compartment. Return to athletics is discouraged for the longevity of the graft.

Graft Preservation

Preservation of the allograft meniscal tissue can be carried out via multiple methods, including fresh, fresh-frozen, cryopreserved, and freeze-dried. Many prefer fresh-frozen or cryopreserved grafts, as they have had the highest success rates with no or minimal biomechanical degradation [22]. However, grafts preserved by any of the above methods have been shown to successfully heal to the host and function to varying degrees [30–34]. Disease transmission is of great concern. The Centers for Disease Control and Prevention estimates the incidence of disease transmission of all the approximated 900,000 allografts implanted per year to be 0.0004%.

Fresh grafts have the highest cell viability and survivability after transplant but may have the greatest risk of disease transmission. The grafts are kept at 4°C in sterile lactated Ringer's solution up to 7 days after harvest, after which fibrochondrocyte viability is no longer maintained. Such a restricted time frame limits fresh graft availability. Thorough serologic evaluation of the allograft and size matching typically takes greater than 7 days. Secondary sterilization techniques destroy any viable cells, thus eliminating any potential advantage of implanting living meniscal fibrochondrocytes. Jackson et al. [35], through DNA typing in a goat model, however, demonstrated donor DNA was replaced with host DNA at 4 weeks after transplantation. DeBeer et al. [36] showed similar results of 95% of donor cells replaced by host cells in human meniscus 1 year after transplant. Therefore, the benefit of cell viability of the donor allograft remains unclear.

Fresh-frozen grafts have increased in use and demand. Stored at –80°C, the process is less expensive and relatively simple. The freezing process, however, destroys viable donor cells and results in denaturation of histocompatibility antigens [37]. Such changes may reduce the risk of disease transmission and immunologic host response. The collage framework, however, is maintained [38], and repopulation of the meniscus periphery with host cells has been shown [39, 40].

Cryopreservation maintains donor cell viability [41], but the number of viable cells decreases with storage time [39]. The benefit of cryopreservation may not be worth the additional cost in comparison with fresh-frozen grafts, as both have been implanted with similar results [42].

Freeze-drying (lyophilization), like fresh-freezing, denatures histocompatibility antigens and destroys viable donor cells [37]. Graft shrinkage after transplantation, synovitis, and recurrent effusions have all occurred with freeze-dried meniscal allografts [6, 7, 43]. Therefore, grafts preserved by such methods have been largely abandoned and are not currently recommended.

The cost-benefit ratio of cryopreserved and fresh-frozen grafts is unclear at present. No literature currently supports the additional expense of cryopreservation at this time. The role of viable chondrocytes within the meniscal allograft is unknown. Clinical findings and animal models discourage use of freeze-dried allografts because of changes observed after transplant.

Sizing

Accurate sizing of the meniscal allograft is critical for incorporation and function. The allograft should be sized to be within 5% of the native meniscus [44]. Multiple imaging modalities can be employed for sizing including MRI, computed tomography (CT), and plain radiography [25, 44–47]. Consistent underestimates of the meniscus size and recipient site has been shown with both CT and MRI [44].

Standard anteroposterior and lateral radiographs can accurately and inexpensively estimate meniscal size [46, 47]. Pollard et al. [46] described an effective and inexpensive technique using standard anteroposterior and lateral radiographs. On the anteroposterior radiograph, the coronal width of the meniscus is measured, and on the lateral radiograph, the sagittal length is calculated. Correction for magnification is carried out with use of markers placed on the skin of the proximal leg. After correction for magnification errors, the length is multiplied by 0.8 or 0.7 for the medial versus lateral meniscus, respectively. A size match of 95% is achieved with this technique (Fig. 3). In addition, radiographs are sent to the tissue bank to compare with donor radiographs. Coronal and sagittal dimensions easily can be measured and estimated within an error of $\pm 3\%$ [47].

Surgical Technique

The surgical goal of meniscal allograft transplantation is to restore the normal relationship of the meniscofemoral and meniscotibial articulations by placing an allograft in an anatomic position. Both open and arthroscopic techniques have been described. Current trends are toward arthroscopically assisted techniques to reduce surgical morbidity [6, 48–56].

The allograft may be anchored one of two ways: either by a bone bridge in slot technique or by separate bone plugs. Secure horn fixation is necessary to prevent meniscal extrusion during weight-bearing. Bony fixation of the meniscal allograft has shown to have superior surface contact mechanics compared with those of soft-tissue fixation [57–60]. Either by using a bone bridge or bone plugs, the anterior and posterior horns of the meniscus must be placed in an anatomic position. The smaller distance of ≤ 1 cm between the anterior and posterior horns of the lateral meniscus does not allow for separate bone plug fixation due to the risk of tunnel communication compromising graft fixation

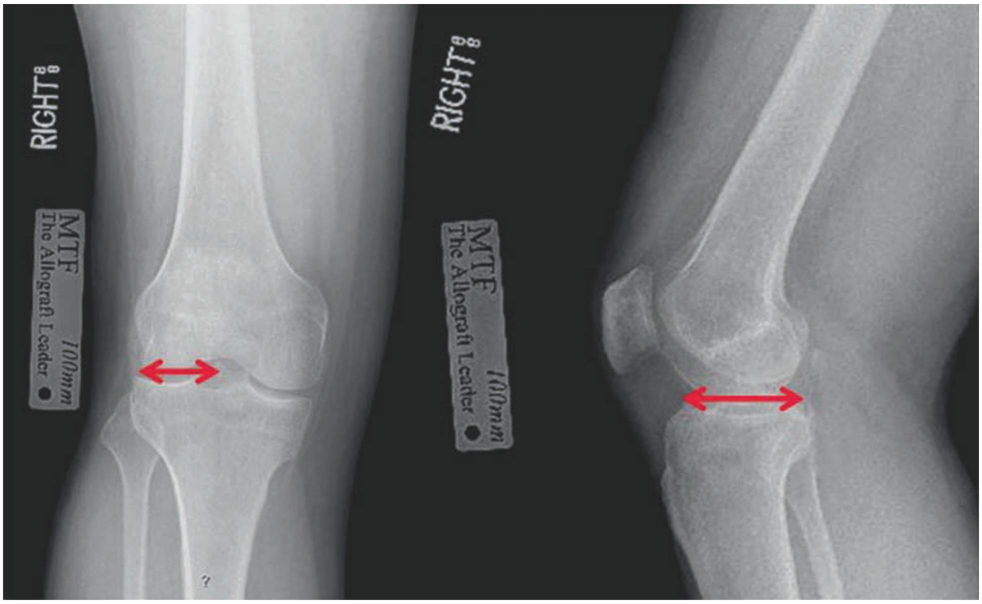


Fig. 3 Sizing using standard anteroposterior and lateral radiographs. Size markers (used with permission of the Musculoskeletal Transplant Foundation, Edison, NJ) are used to correct for magnification. The meniscal width is measured on the anteroposterior radiograph from the peak of the tibial eminence to the ipsilateral border of the tibial plateau. The length is the distance from the anterior to posterior borders of the tibial plateau (arrows depict areas of measurement for allograft)

and anatomic placement [16]. The advantage of the bone bridge is a fixed distance between the anterior and posterior horns, maintaining the native horn insertion distance of the allograft meniscus. Many argue easier insertion of the allograft [52]. The use of separate bone plugs medially, however, allows for anatomic placement of the medial meniscal horn insertion sites. Tunnel communication should not occur, as the distance between the anterior and posterior footprints is much greater than that of the lateral meniscus. Anatomic placement of the meniscal footprints should restore normal hoop stresses within the transplanted meniscus during weight-bearing. Improper placement leading to increased tensile stresses within the meniscus may be a cause of clinical failure. It is highly recommended to use a bone plug technique for the medial meniscal grafts.

After the induction of general anesthesia and administration of prophylactic intravenous antibiotics, the patient is placed in a supine position. An examination under anesthesia is performed to assess stability and document range of motion. A proximal thigh holder with tourniquet is positioned proximally enough to allow exposure as one would use for a typical posterolateral or posteromedial inside-out meniscal repair.

A diagnostic arthroscopy is performed using the standard parapatellar portals. The condition of the remaining meniscus and articular surfaces is documented. The integrity of the intraarticular structures, especially the ACL, is assessed. The allograft is then thawed and reconstituted in antibiotic solution as per protocol. The residual meniscus is debrided to a 1- to 2-mm peripheral rim (Fig. 4). Rasping of the synovium on the upper and lower borders of the remnant promotes an aggressive vascular response, providing a vascular source at the meniscocapsular junction for graft healing and incorporation. Remnants of the anterior and posterior horns are left to aid in localizing the position for the slot or bone tunnels. A modified notchplasty is then performed to allow for improved visualization and ease of graft passage. This is done cautiously while protecting the cruciate ligaments.

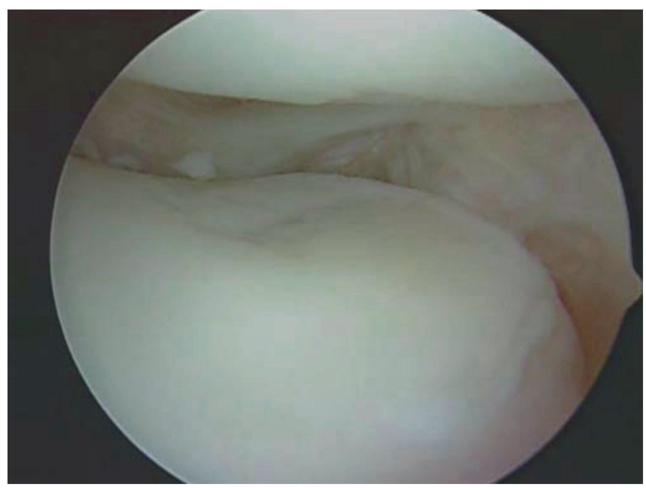


Fig. 4 Arthroscopic view of the lateral compartment after meniscal debridement to less than 2 mm peripheral rim

Bone Bridge or Slot Technique for Lateral Meniscal Reconstruction

After the preparation of the meniscal rim, a modified notchplasty is performed under the ACL on the femur; this allows one to visualize the posterior insertion site of the lateral meniscus, as the footprint is posterior to the lateral tibial spine. An 18-gauge needle is placed percutaneously to align with the anterior and posterior horns of the meniscus and to identify the location for the meniscal slot. A vertical arthrotomy, appropriately 3 cm in length, is made in line with the anterior and posterior meniscal horns. Electrocautery is used to mark a line between the centers of the meniscal horn footprints (Fig. 5). A superficial reference slot is then made using a 4-mm burr along the line previously marked. The slot should parallel the sagittal slope of the tibial plateau and approximate the width of the burr (Fig. 6). A drill guide (Meniscal Transplant Set; Stryker Endoscopy, San Jose, CA) is placed in the slot and hooked onto the posterior tibial cortex to measure the length of the slot (Fig. 7). A guide wire is then drilled through the guide handle parallel to the tibial slope to the posterior tibial cortex but not through it (Fig. 8). This can be done under fluoroscopy to aid in placement. The guide handle is removed, and an 8-mm cannulated reamer is advanced over the guide wire to the appropriate depth, leaving a shelf of posterior tibial cortex (Fig. 9). The remaining roof of the reamed slot is removed with a rongeur or pituitary rongeur. A box cutter, size 8 × 10 mm, is used to convert the rounded slot to a box-shaped trough. The edges are then smoothed with a rasp (Fig. 10A–D).

A 3-cm posterolateral incision is made between the iliotibial band and the biceps femoris superficially. Deep dissection is carried out through the interval of the fibular collateral ligament and lateral gastrocnemius tendon, exposing the posterolateral capsule for an inside-out meniscal repair and graft passage.

Fig. 5 The line made with the electrocautery device between the anterior and posterior horn footprints for the slot positioning. (Courtesy of Stryker Endoscopy, San Jose, CA.)

Fig. 6 A 4-mm burr is used to make a reference slot in line with the anterior and posterior horns, parallel to the sagittal slope of the tibial plateau and a width no greater than that of the burr. (Courtesy of Stryker Endoscopy, San Jose, CA.)

Allograft Preparation

The meniscal allograft is sent from the tissue bank with meniscal tissue attached to the hemiplateau of the tibia. All nonmeniscal soft tissue should be removed. The bone bridge is cut with an oscillating saw to a width of 7–8 mm and a height of 10 mm, as shown in Figs. 11 and 12. To allow for ease of passage of the graft into the tibial bone slot, the bone bridge width should be undersized by 1 mm. For posterior seating of the graft, the posterior bony wall of the bone bridge should be flush with the posterior aspect of the posterior meniscal horn (Fig. 13). The sizing is checked by passing the bone bridge through a calibrated trough (Fig. 14). A no. 0 horizontal mattress traction suture is placed at the junction of the middle and posterior thirds of the meniscal allograft.

Allograft Insertion

Using a zone-specific meniscal repair cannula, a meniscal stitching needle (Smith & Nephew, Inc., Andover, MA) is inserted toward the posterior corner of the recipient compartment. The free ends of the allograft no. 0 traction suture are placed through the eyelet of the meniscal stitching needle. The needle is then advanced and retrieved with the

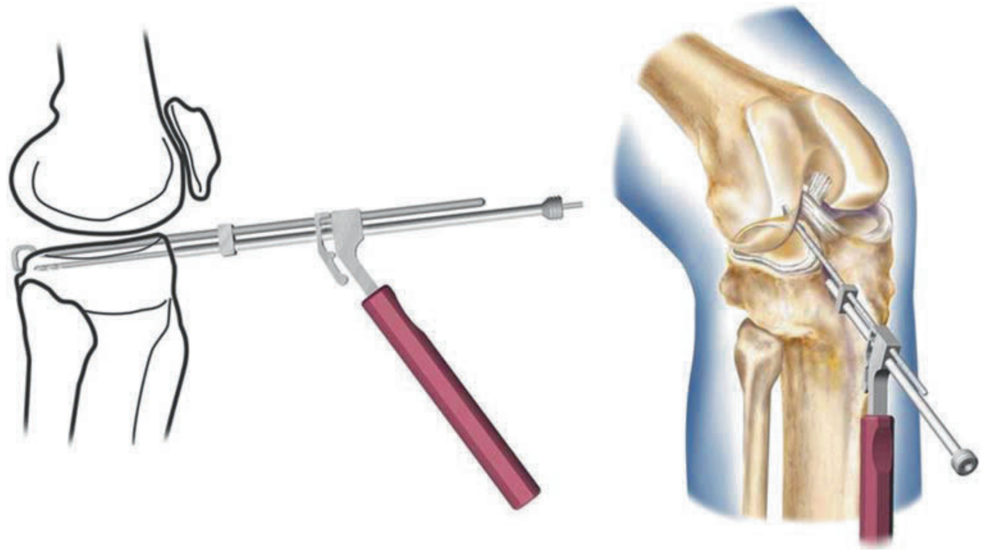


Fig. 7 Stryker guide placed within the reference slot and hooked onto the posterior tibial plateau. The drill guide is in place to measure the length of the tibial slot. (Courtesy of Stryker Endoscopy, San Jose, CA.)

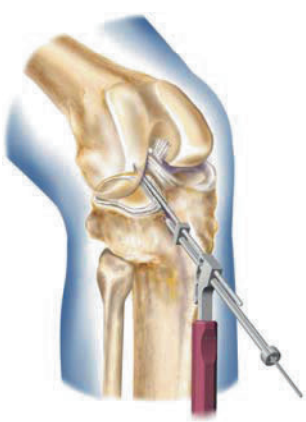


Fig. 8 Guide pin placed through the guide handle; take care to drill to the posterior tibial cortex but not through it. (Courtesy of Stryker Endoscopy, San Jose, CA.)

ends of the traction suture through the posterior incision made earlier for the meniscal repair. The allograft is inserted through the parapatellar arthrotomy and aligned with the tibial slot. Positioning is accomplished by pulling on the traction suture and positioning of the knee in the figure-of-four position (Fig. 15). Varus stress, as well as careful pressure anteriorly on the graft, will facilitate passage of the meniscus.

After proper seating of the bone bridge, a guide wire is inserted between the central wall of the slot and bone bridge. A cannulated tap is inserted over the guide wire to create a pilot hole for an interference screw. A periosteal elevator can be used to hold the bone bridge in place while the tap is advanced. Either a 7 × 28 mm or an 8 × 28 mm interference screw is inserted to secure the bone bridge (Fig. 16). Arthroscopic inspection is used to confirm the placement and sizing of the meniscus. The remainder of the meniscal tissue is secured with a standard inside-out meniscal repair technique. Working from posterior to anterior, 8–10 vertical mattress 2-0 nonabsorbable sutures are placed. To reduce the risk of neurovascular injury, an all-inside meniscal repair system may be used to secure the most posterior extent of the meniscal allograft. Final arthroscopic inspection and gentle probing is used to confirm placement and fixation of the transplanted meniscus (Fig. 17).

Bone Plug Technique for Medial Meniscal Reconstruction

Allograft Preparation

As mentioned earlier, the allograft is received from the tissue bank as a hemiplateau. All nonmeniscal soft tissue is sharply removed. The posterior horn is prepared first. This may be done with a rongeur, burr, or bone coring system (Smith & Nephew, Inc.). If using a coring drill, a hole angled at 60 degrees relative to the posterior horn origin and centered within the meniscal footprint is made with a 2.5-mm drill (Fig. 18). To minimize the risk of avulsion of meniscal tissue, the hole must be centered within the posterior horn insertion site. A collared pin is inserted into the inferior opening of

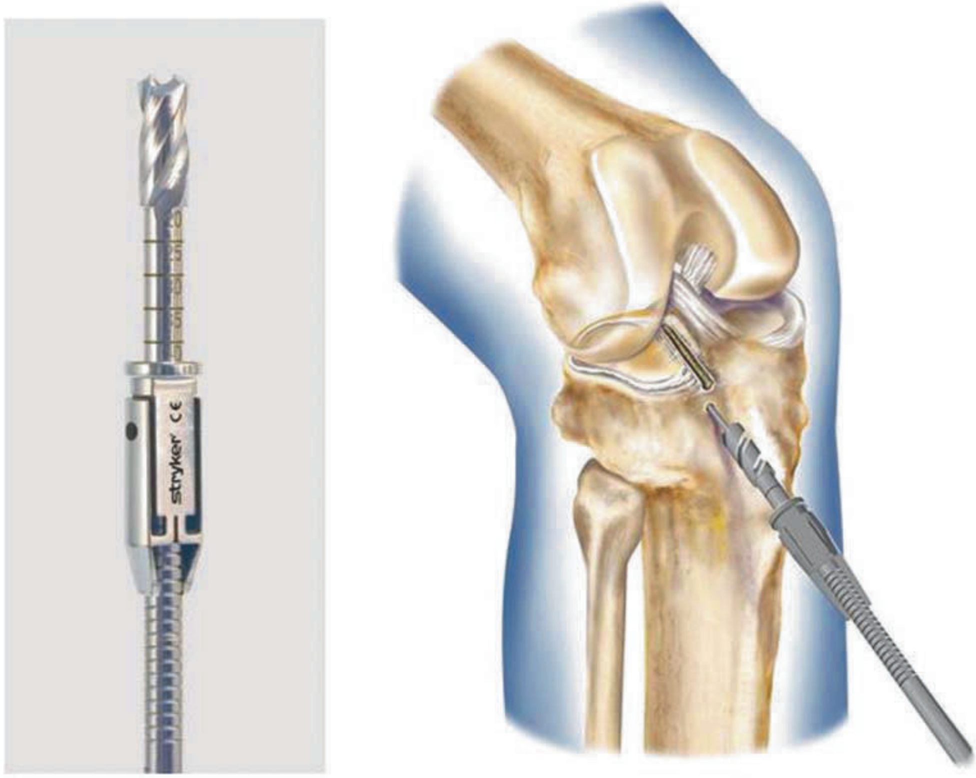


Fig. 9 An 8-mm cannulated reamer is advanced over the guide to the measure depth. (Courtesy of Stryker Endoscopy, San Jose, CA.)

the hole until the collar abuts the bone. A 7- or 8-mm coring reamer is passed over the collared pin, creating a bone plug with an intact meniscal origin. The plug length is then trimmed to approximately 8 mm (Fig. 19A–D). The anterior horn plug is then prepared in a similar fashion, but the initial drill hole is angled 90 degrees to the meniscal footprint, and a 7- or 8-mm coring reamer is used, as the anterior plug will be secured in a press-fit fashion.

Tunnel Preparation

Using the separate bone plug technique, the plugs are placed into separately prepared bone tunnels within the tibia. An ACL guide is used. The posterior tunnel is prepared first, with the ACL guide tip set into the middle of the posterior horn footprint and positioned so the tibial tunnel entrance is lateral to Gerdy's tubercle. It is important to have performed a modified notchplasty to fully visualize the posterior meniscal footprint for anatomic placement of the posterior tunnel. The guide is typically set to 60 degrees, which allows the guide to be held perpendicular to the tibial plateau (Fig. 21A, B). A guide pin is advanced through the guide until the tip is intraarticular. Positioning is confirmed with arthroscopic visualization. A reamer sized 1 mm greater than the posterior bone plug is then drilled over the guide pin to create the tibial tunnel. The differences in diameter of the tunnel versus the posterior plug allow for easier graft passage but do not lead to plug toggle and potential loss of fixation. A curette can be used to prevent advancement of the guide pin while reaming. A shaver and rasp are used to remove any debris at the tunnel opening.

The anteromedial portal is extended inferiorly to create a 3- to 4-cm arthrotomy for graft passage and anterior tibial tunnel preparation. The tip of the ACL guide is used to mark the anatomic center of the anterior horn footprint through the arthrotomy. The anterior tunnel is then drilled to the width of the anterior meniscal bone plug. The ACL guide is set to 40 degrees and positioned so that the tunnel entrance is on the lateral tibial metaphysis medial to Gerdy's tubercle. The anterior and posterior tunnels are positioned such that the tibial openings are separated by 1 cm and surround Gerdy's tubercle (Fig. 22A).

A 3-cm posteromedial incision is made for the inside-out meniscal repair technique and graft passage. The interval between the medial collateral ligament and the posterior oblique ligament is developed. Care is taken to protect the infrapatellar branch of the saphenous nerve in the proximal extent of the incision. The posteromedial capsule is identified through this interval.

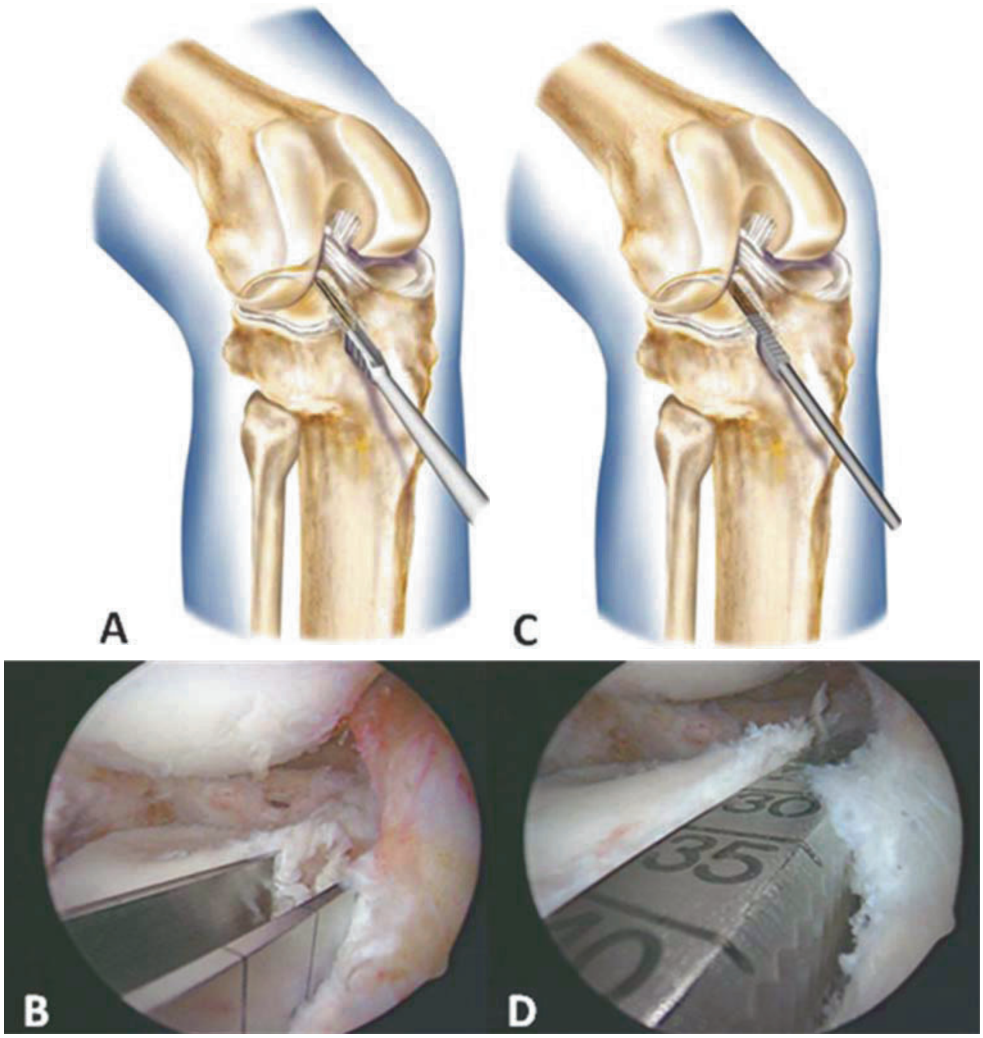


Fig. 10 (**A**) Box cutter is used to convert the rounded slot to a box-shaped trough. (**B**) Arthroscopic view of the box cutter in place. (**C**) Rasp is used to smooth the edges of the slot. (**D**) Arthroscopic view of an 8-mm rasp in the bone trough. (A and C courtesy of Stryker Endoscopy, San Jose, CA.)

Allograft Insertion

A wire loop or suture retrieval device is passed retrograde into the anterior and posterior tunnels and retrieved through the anteromedial arthrotomy. The bone plug sutures are passed through the tunnels with the wire loop and retrieved at the anterior entrance of the tunnels at the tibial cortex. The traction suture is passed through the posteromedial capsule and retrieved through the posteromedial incision using meniscal repair cannulas and meniscal needles in a similar fashion as described with the bone bridge technique for the lateral meniscus. A valgus stress is applied to the knee to open the medial compartment as the graft is delivered into the knee by traction on both the posterior bone plug suture and traction suture. Passage is facilitated by maintaining the valgus stress and placing the knee at approximately 30 degrees of flexion. Once the posterior bone plug is positioned, the knee is cycled multiple times to properly seat the posterior horn.

A no. 2 Ultrabraid (Smith & Nephew, Inc.) is passed in a figure-of-eight fashion through the meniscal soft tissue insertion, leaving two long ends of the suture that are then passed using a Keith needle through the bone hole (Fig. 20). These sutures will be used for traction and fixation of the allograft. A no. 0 horizontal mattress traction suture is placed at the junction of the middle and posterior thirds of the meniscal allograft for insertion.

The anterior plug is passed into the anterior tunnel in a similar fashion. The sutures of the anterior and posterior bone plugs are then tied to one another over an anterior tibial bone bridge, securing the allograft insertion sites (Fig. 22B). The intermeniscal ligament is sutured to the graft for additional

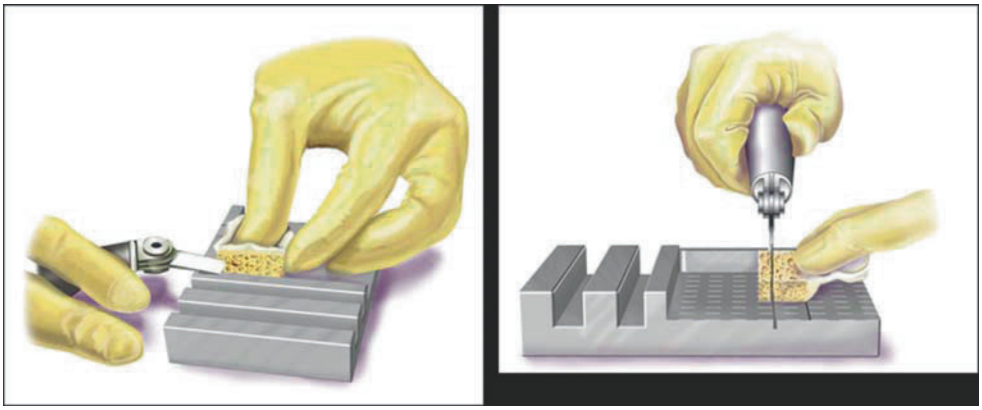


Fig. 11 Allograft bone block being cut to appropriate size. (Courtesy of Stryker Endoscopy, San Jose, CA.)

Fig. 12 Bone bridge after being cut to size. (Courtesy of Stryker Endoscopy, San Jose, CA.)

Fig. 13 Lateral meniscal allograft with bone bridge cut to size and traction suture placed at the junction of the posterior and middle thirds.

fixation. The remainder of the meniscal tissue is secured with a standard inside-out meniscal repair technique using 8–10 vertical mattress 2-0 nonabsorbable sutures as described earlier.

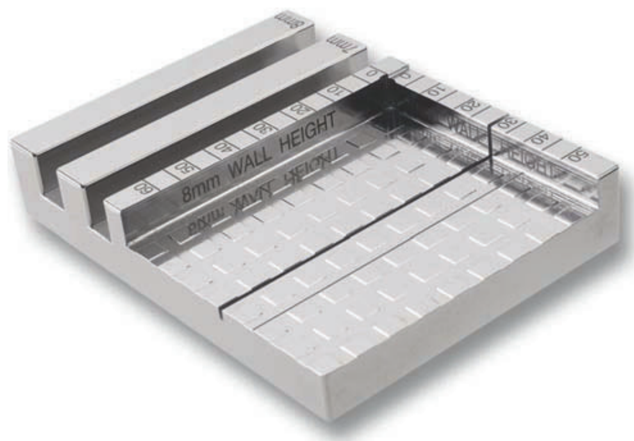


Fig. 14 Calibrated trough and measuring plate for allograft preparation. (Courtesy of Stryker Endoscopy, San Jose, CA.)

Fig. 15 Meniscus insertion. (Courtesy of Stryker Endoscopy, San Jose, CA.)

Fig. 16 Allograft meniscus secured in place. (Courtesy of Stryker Endoscopy, San Jose, CA.)

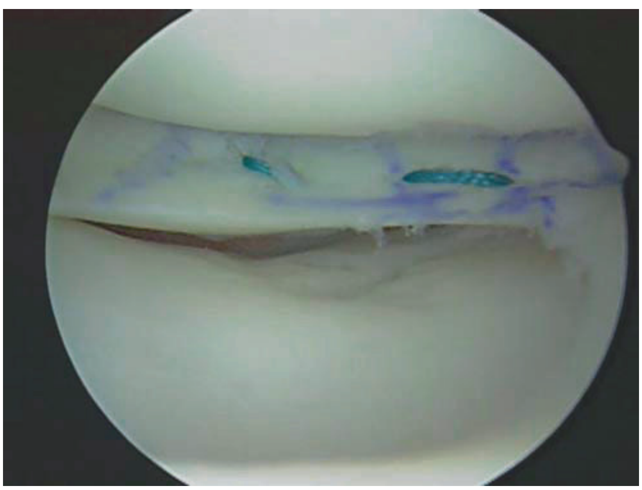


Fig. 17 Final arthroscopic view of the transplanted meniscal allograft.

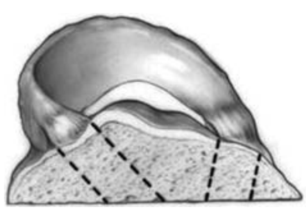


Fig. 18 Proper angulation for the bone plugs. (Courtesy of Cryolife, Inc., Kennesaw, GA.)

Advanced Techniques

Corrective Osteotomy

If varus or valgus malalignment coexists, the recipient compartment may be under more than physiologic compression, which can lead to allograft failure. Realignment procedures must be performed concurrently [61]. Varus alignment may be present with medial meniscus deficiency. In such a situation, a proximal tibial osteotomy should be performed as an adjunct procedure. However, unlike high tibial osteotomies performed for medial compartment arthritis, the goal is to restore alignment to just beyond neutral. An opening medial osteotomy (the authors' preferred method) or closing lateral osteotomy can be performed at the surgeon's discretion. Fluoroscopy will aid in avoidance of the tibial trough or tunnels.

A distal femoral osteotomy may be performed to correct valgus alignment. Numerous techniques have been described and performed with success. An opening wedge osteotomy is the authors' choice. Regardless of whether varus or valgus correction is performed, care must be taken not toINTbreak; overcorrect.

Ligament Reconstruction

Meniscus allograft transplantation and simultaneous ACL reconstruction have been performed successfully and have proved to be beneficial [62–65]. The biomechanical relationship of the medial meniscus and an intact ACL is well known. The medial meniscus is a secondary stabilizer to anteroposterior stress and therefore critical for a successful ACL reconstruction [66, 67]. An intact ACL in turn protects the articular cartilage and menisci [68, 69].

With the bone bridge in slot technique, the tunnel entrance should be placed more distally on the tibia, allowing a longer tunnel with a more rounded articular opening [28, 56]. The femoral tunnel can then be drilled in traditional fashion. The meniscal slot is then prepared as described. Some confluence of the slot and tibial tunnel will occur but will not be problematic [63]. If a bone plug technique is used, the tibial tunnel for the ACL reconstruction is positioned more medially to avoid communication with the tunnel for the posterior horn bone plug. The ACL reconstruction is then performed in the usual fashion.

After the meniscal slot is completed, the ACL graft is passed in traditional fashion and secured within the femur. The soft tissue portion of the ACL graft can then be displaced manually to allow passage of the meniscal graft. Once the meniscus is properly positioned, the ACL graft is tensioned and secured distally. The interference screw for meniscal fixation is placed, and the meniscus is repaired as described earlier.

Alternatively, the meniscal transplant can be performed and all steps completed first as described [28]. The tibial tunnel is then positioned more medially and distally, reducing confluence of the slot and tunnel. As stated previously, the intersection of the slot and tibial tunnel will not be problematic [63]. The ACL reconstruction can then be completed

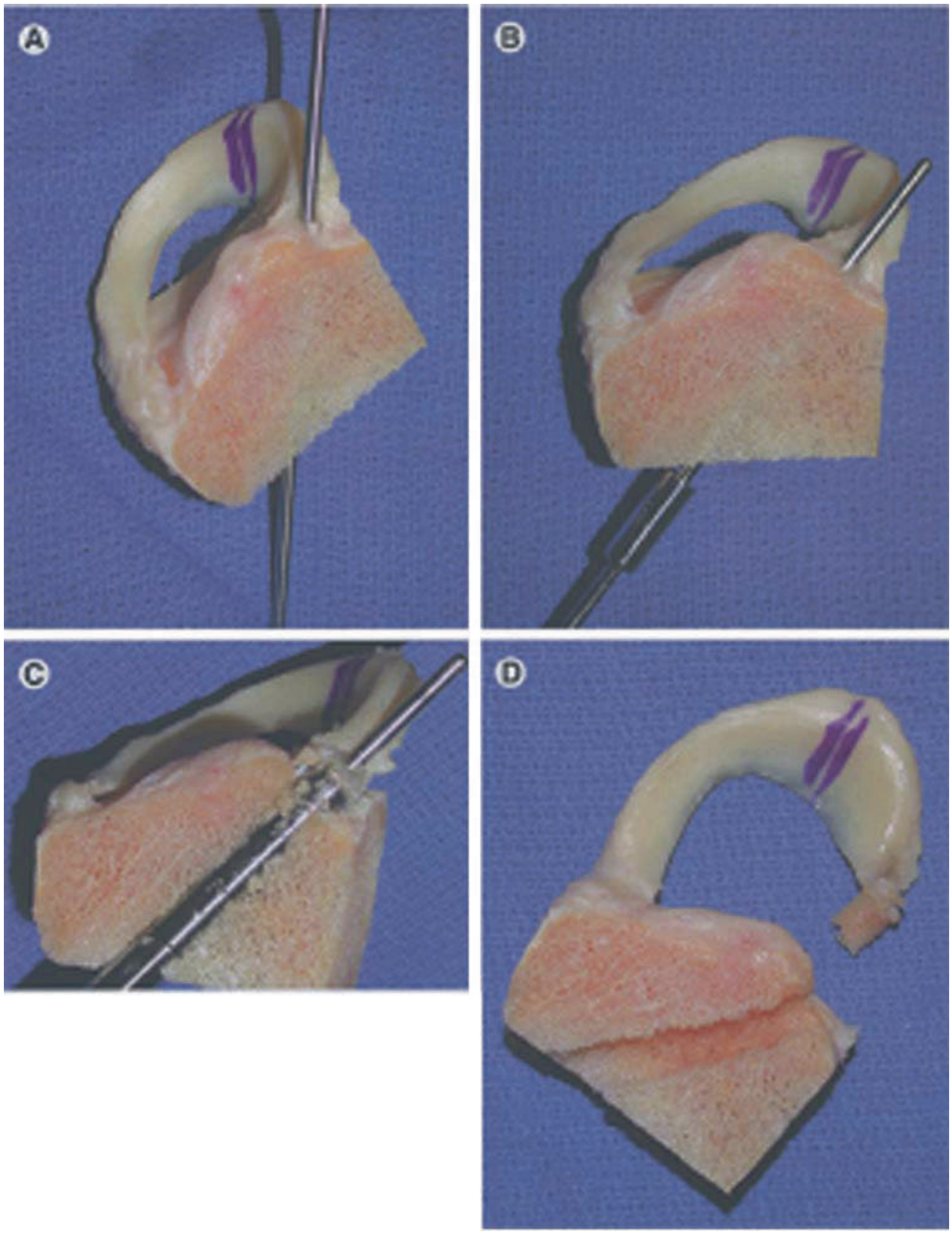


Fig. 19 Meniscal preparation. (**A**) A 2.5-mm twist drill at 60 degrees to the posterior horn insertion site. (**B**) Collared pin in place. (**C**) An 8-mm cannulated coring reamer capturing the majority of the meniscal insertion site. (**D**) Final 8 mm × 8 mm posterior horn bone plug meniscal construct. (From Fox JA, Lee SJ, Cole BJ. Bone plug technique for meniscal transplantation. *Oper Tech Sports Med* 2003;11:161–169. Copyright © 2003. Reprinted with permission of Elsevier.)

in traditional fashion. A hamstring graft is recommended, allowing for a smaller tunnel diameter, facilitating graft passage.

As important as the medial meniscus is to anteroposterior stability in the ACL-deficient knee, the lateral meniscus provides secondary restraint to posterolateral rotation. A posterolateral corner injury combined with a deficient lateral meniscus must be treated as a combined procedure. Failure to address the deficient lateral meniscus concurrently with a posterolateral reconstruction/repair will not restore adequate posterolateral stability, leading to continued rotatory instability and potential future articular destruction and wear.

Cartilage Restoration

Simultaneous cartilage restoration procedures such as osteochondral allograft transplantation and autologous chondrocyte implantation have been performed with success [70,

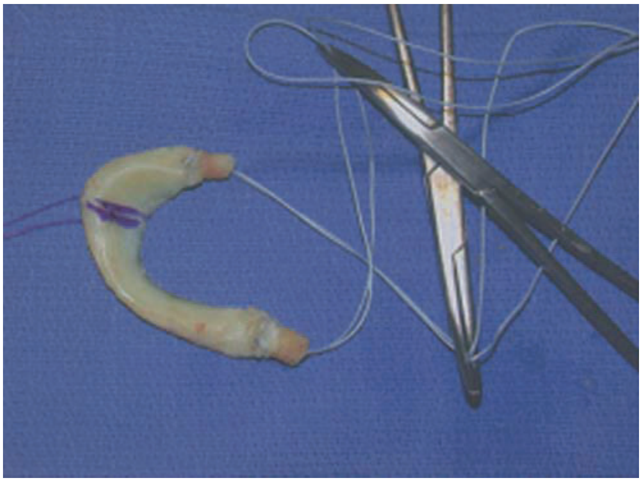


Fig. 20 Fully prepared meniscus with monofilament traction suture in place at the junction of the middle and posterior thirds of the meniscus (*blue mark*). (From Fox JA, Lee SJ, Cole BJ. Bone plug technique for meniscal transplantation. *Oper Tech Sports Med* 2003;11:161–169. Copyright © 2003. Reprinted with permission of Elsevier.)

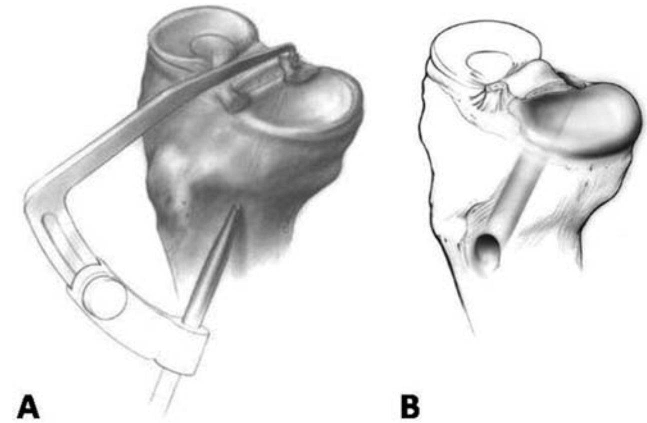


Fig. 21 (**A**) ACL guide set to 60 degrees and guide tip placed at the anatomic center of the posterior footprint of the medial meniscus. (**B**) Tunnel drilled for the posterior bone plug. (All images courtesy of Cryolife, Inc., Kennesaw, GA.)

71]. Careful preoperative planning is critical for positioning of incisions and the order of surgical steps. It is recommended that chondral procedures be performed after the meniscal transplant is completed to avoid inadvertent damage to the periosteal patch or osteochondral graft with the meniscal instrumentation [72].

Rehabilitation

Postoperatively, patients are placed in a hinged knee brace locked in extension and allowed to partially weight-bear (approximately 50% body weight). Patients are instructed to immediately start straight leg raises, and quadriceps and hamstring isometric exercises can be started as pain tolerates. Weight-bearing as tolerated with crutch use is begun at 2 weeks as well as range of motion from 0 to 90 degrees of flexion. Closed chain exercises also are begun 2 weeks after surgery. The patient may discontinue crutches at 6 weeks postoperatively if gait is normal. Any rotational exercises for the first 8 weeks are avoided due to shear stress to the graft. After 8 weeks, the patient should be full weight-bearing, the brace is discontinued, and full active range of motion exercises are initiated. Use of a stationary bike is encouraged. Proprioceptive training also is started at 8 weeks. Sport-specific exercises and jogging may be started at 12 weeks postoperatively. The authors' preferred postoperative rehabilitation protocol is summarized in Table 2.

Currently, there are no standardized rehabilitation protocols that exist for patients undergoing meniscal allograft transplantation. There have been numerous studies published

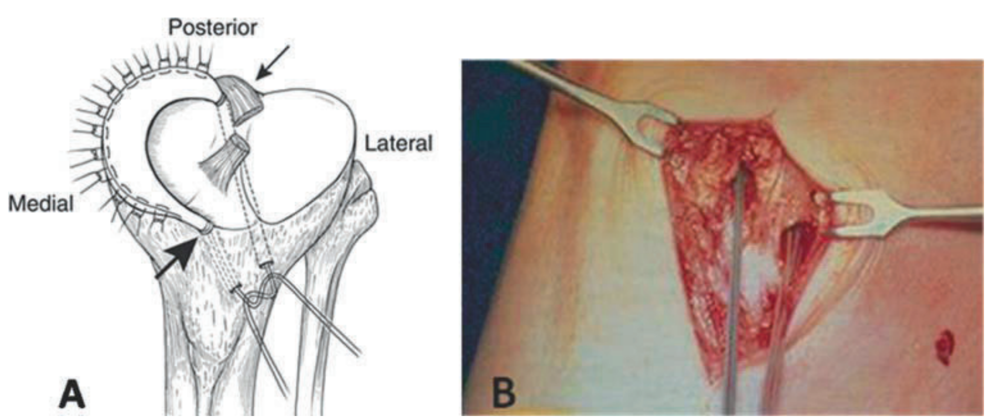


Fig. 22 (**A**) Medial meniscal allograft transplantation with anterior (*large arrow*) and posterior (*small arrow*) bone plugs through transosseous tunnels. (From Sekiya JK, Ellingson CI. Meniscal allograft transplantation. *J Am Acad Orthop Surg*2006;14(3):164–174. Copyright © 2006 American Academy of Orthopaedic Surgeons. Reprinted with permission.) (**B**) Clinical photograph showing the exiting traction sutures that are then tied over a cortical bone bridge for fixation.

Table 2 Rehabilitation protocol

Time frame	Weight-bearing, range of motion	Brace use	Therapeutic elements
0–2 weeks	Partial weight-bearing (50%) *No tibial rotation*	Full-time, locked in extension	Quadriceps sets, straight-leg raises, hamstring isometrics, patellar mobilization
2–8 weeks	Advance weight-bearing to that tolerated with crutches Discontinue crutches at 6 weeks if normal gait pattern *No weight-bearing with >90 degrees of knee flexion* *No tibial rotation*	Brace locked 0–90 degrees Discontinue if gait normal at 6 weeks	Closed chain exercises, terminal extension
8–12 weeks	Full weight-bearing Full active range of motion	Discontinued	Begin stationary bike Hamstring strengthening, lunges with flexion restricted to 90 degrees of flexion, proprioception exercises
12–16 weeks	Full weight-bearing Full active and passive range of motion	Discontinued	Plyometrics, jogging, advance to sport-specific exercises

on meniscal allograft transplantation, but none have focused on rehabilitation. Many authors recommend use of continuous passive motion machines for immediate postoperative range of motion [6, 29, 62, 64, 73]. Most authors recommend full weight-bearing by 6 weeks, with immediate to early range of motion. The timing for full weight-bearing is still controversial, and more long-term studies are needed to address this issue. It is recommended that flexion be limited to 90 degrees for the first 4–6 weeks to prevent shearing and rotational forces on the transplanted meniscus. The ability to return to running and other sporting activities appears around 6–9 months, with no consensus on exact timing. The ability to return to preinjury sporting activities seems to be limited, but there has not been enough long-term follow-up to confirm this idea. It seems most authors limit athletic activity to light sports.

The postoperative protocols in all the studies reviewed by Matava [22] were contingent upon any concurrent procedures performed. After combined cruciate ligament reconstruction and meniscal transplant, many authors restricted the rehabilitation to their preferred protocols for the ligament reconstruction. Similarly, restrictive weight-bearing is dictated by a concurrent realignment osteotomy procedure.

Complications

The complications after meniscal allograft transplantation were reviewed in an analysis by Matava [22]. The most common complication in the 547 patients in the review was graft tearing, resulting in 45 tears, or 8.2%. In a series by Stollsteimer et al. [54] and Graf et al. [65], this resulted in reoperation in 26% and 25% of patients with tears, respectively. In both series, treatment was partial meniscectomy or repair, which effectively eliminated mechanical symptoms and relieved pain.

There have been no reports of viral infection, namely HIV or hepatitis transmission, in the literature. Bacterial infection after meniscal transplant has been reported in three studies ranging from 3% to 4.5% [6, 49, 54]. However, it is unclear whether these infections resulted from the surgical procedure or the transplantation of a contaminated graft, as bacteriologic data were not provided.

Three studies [6, 24, 54] reported postoperative immunologic responses but no follow-up. There are no reports of neurovascular injury in the literature. Three case series [6, 24, 74] have reported loss of motion after transplantation, resulting in five closed manipulations under anesthesia [24, 74]. Additional complications have been reported attributable to concurrent procedures at the time of the meniscal transplant, such as nonunion of osteotomy sites, revision ACL reconstruction, and hardware failure. Minor complications such as suture granulomas also have been reported.

Clinical Pearls

Meniscal allograft transplantation is a technically challenging procedure. Proper patient selection will increase the likelihood of success. The patient must have thorough understanding of the surgical and rehabilitative expectations. Serial physical examinations of the knee and lower extremity will aid detection of any malalignment and ligamentous instability that must be addressed simultaneously.

Anatomic placement of the allograft and appropriate sizing are essential to restore near-normal function in the involved compartment. Failure to place the allograft in the anatomic position may not restore the normal contact area and stresses across the tibiofemoral articulation. Failure to place the bone plugs in the normal meniscal insertion site may not restore normal hoop stresses within the meniscus and may increase the risk of injury to the implanted meniscus.

To aid in visualization and graft passage, a modified femoral notchplasty is necessary, particularly for medial meniscal transplants. Debridement of the anterior fat pad to fully visualize the anterior insertion site of the meniscus is required. Poor visualization may lead to poor placement of the meniscal allograft and ultimately to failure of the allograft meniscus. Two surgical assistants may be necessary, as one assistant may aid the surgeon in positioning of the meniscus while the second assistant remains responsible for holding the extremity and providing the necessary knee flexion and varus or valgus stress.

Repair sutures should be placed in a vertical or oblique direction, not horizontally. Vertical and oblique sutures have been demonstrated to have greater pull-out strength [75]. Suture placement also is very important. Divergent suture placement will aid in proper seating of the meniscal graft in an anatomic location. This also may restore the ability to resist hoop stresses, as any folds or wavy portions of the meniscus will be prevented. Avoid placing the repair sutures in the central or middle thirds of the meniscal allograft as this may weaken the meniscus and not provide stable fixation, which may ultimately lead to meniscal tears.

Summary

Pain-free activities of daily living are the goal of meniscal replacement surgery. Currently, the degree of athletic participation has not been established, though most authors recommend the avoidance of strenuous activities or competitive sports. Biomechanics and healing of the allograft after meniscal transplantation as well as the progression of weight-bearing and its influence on graft success have yet to be determined. Until such determination, high-level activities and sports are discouraged.

In 15 studies reviewed by Matava [22], most patients after transplantation still experienced intermittent swelling and pain to a varying degree. However, the success rate as judged by subjective measures (the Tegner and Lysholm [76] scales, International Knee Documentation Committee [IKDC] score, Cincinnati Knee-Rating System, and the Knee Outcome Survey) is more than 60%. In a review of 100 patients by Verdonk et al. [77], 70% of patients reported a good to excellent result at 10 years after transplant. Cole et al. [71] and Sekiya et al. [78] in 2006 reported a success rate of approximately 90% at 2 years and 96% at 3.3 years, respectively, using the IKDC examination score. Earlier cohorts by Sekiya et al. [64], Cameron and Saha [49], and van Arkel and de Boer [29] showed a success rate of 86%, 87%, and 87%, respectively.

Successful outcomes have been achieved when meniscal allograft transplantation was performed concurrently with ligament reconstructions, realignment osteotomies, and cartilage restoration procedures. Studies by Noyes and Stabler [79], Ryu et al. [74], and Yoldas et al. [62] of meniscal allograft transplantation combined with ACL reconstruction showed results similar to those of an isolated meniscal transplant. Verdonk et al. [80] recently have showed greater clinical improvement in patients who underwent medial meniscal allograft transplantation in combination with high tibial osteotomies than in patients who underwent medial meniscal transplant in isolation. When in combination with osteochondral graft transplantation, Zukor et al. [70] reported a 79% success rate.

Retrospective studies have demonstrated the efficacy of meniscal allograft transplantation in relieving pain and improving function [81, 82]. Yet, it remains unclear whether meniscal allograft transplantation provides protection and preservation of the articular cartilage. Long-term prospective studies are needed to answer such questions.

Case Reports

Case 1

Chief Complaint and Patient History: A 36-year-old man who is a laborer presented with right knee pain and instability. With weight-bearing, the patient reported a sense of knee hyperextension and pain. The pain was localized to the posterolateral aspect of the knee. He reported that the pain and instability had been progressive since undergoing a knee arthroscopy 10 months earlier for a lateral meniscal tear that required a complete lateral meniscectomy. The patient denied effusions or mechanical symptoms. He denied any preinjury knee symptoms or complaints of instability. The patient was referred to the authors' institution for further evaluation.

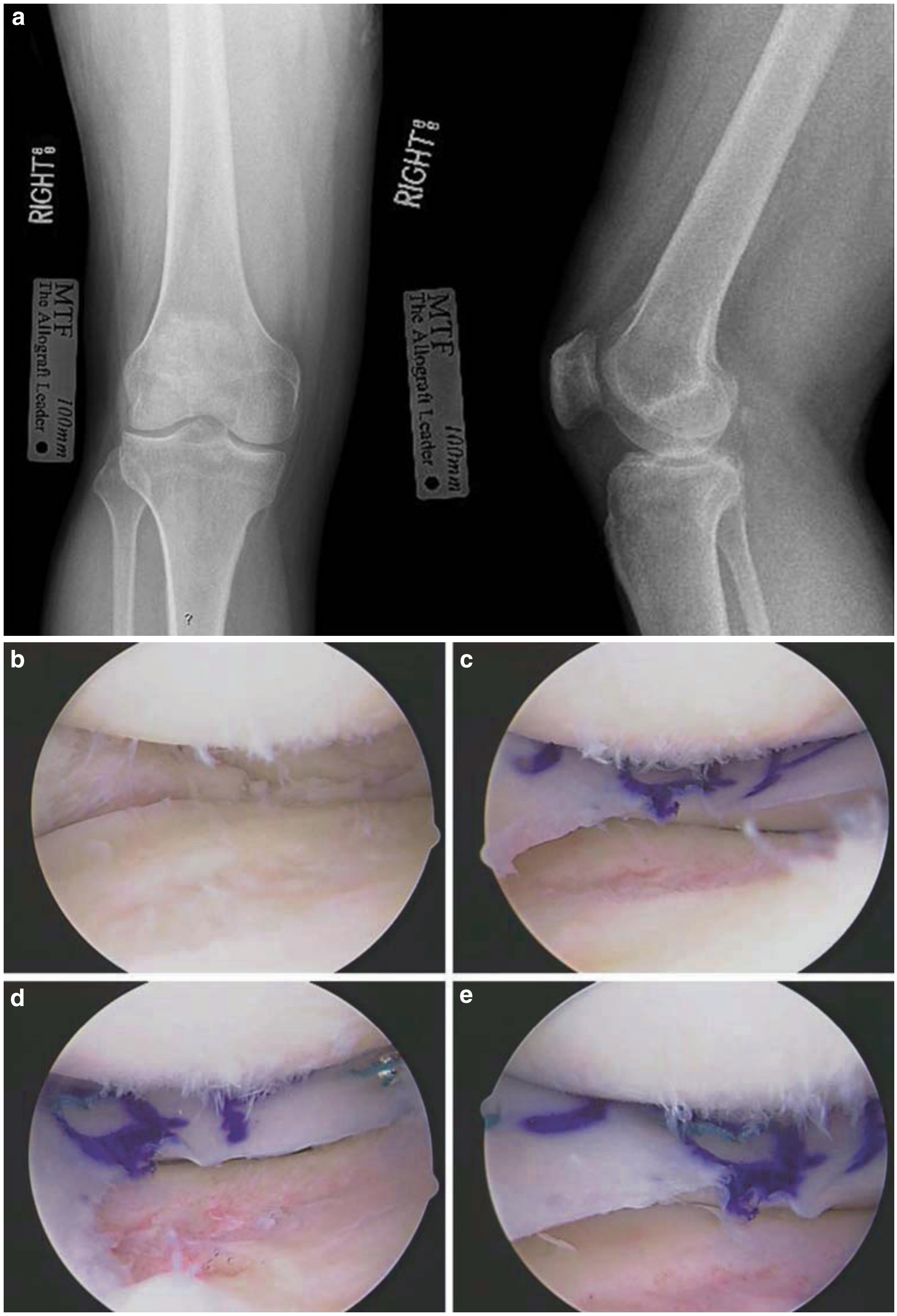


Fig. 23 Case 1. (**A**) Standard anteroposterior and lateral radiographs demonstrating minimal arthritic changes. Sizing markers (used with permission of the Musculoskeletal Transplant Foundation, Edison, NJ) are present for meniscal allograft measurements. (**B**) Intraoperative arthroscopic picture of a lateral meniscal transplantation after debridement of the remaining native meniscus back to rim of approximately 2 mm. (**C**) Intraoperative arthroscopic picture of a lateral meniscal transplantation after placement of the allograft into the lateral compartment. (**D, E**) Final views after meniscal repair. (**F**) Anteroposterior and lateral radiographs obtained 9 weeks postoperatively showing proper placement of the lateral bone bridge.

Fig. 23 (continued)

Physical Exam: On physical examination, the right lower extremity had no deformity or clinical malalignment. The knee had no effusion and a full range of motion. The Lachman test and the anterior and posterior drawer tests were negative. There was a grade II varus laxity at 30 degrees of knee flexion and no valgus laxity. On the prone dial test, there was an increased external rotation at 30 degrees of knee flexion compared with that of the uninjured left knee. There was tenderness to palpation of the lateral joint line.

Imaging: Standard weight-bearing anteroposterior and lateral radiographs revealed minimal lateral space narrowing (Fig. 23A). Review of the previous operative records and intraoperative arthroscopic pictures showed the popliteus tendon to be intact and a near-complete lateral meniscectomy. The MRI scan was reviewed and showed the lateral collateral ligament and popliteofibular ligament to be intact.

Surgery/Treatment: The surgical plan included lateral meniscal allograft transplantation as well as possible posterolateral corner reconstruction versus augmentation if required. The physical examination and history suggested insufficiency of the posterolateral corner structures; however, the previous arthroscopy, MRI, and lack of a new injury were to the contrary. The functional insufficiency of the lateral meniscus and loss as a secondary restraint may have been the cause of the physical findings of the dial test and recurrent sense of hyperextension instability. A complete absence of the lateral meniscus may present as an unstable knee, as the lateral meniscus is an important structural restraint for posterolateral rotatory instability.

Examination under anesthesia demonstrated no ligamentous laxity and a normal range of motion. A diagnostic arthroscopy confirmed the near-complete absence of the lateral meniscus, the presence of an intact popliteus tendon, and lack of a drive-through sign laterally. The patient underwent a lateral meniscal allograft transplantation using a bone bridge technique without complication (Fig. 23B–E). The patient was admitted overnight for pain control. Postoperatively, the patient was instructed to remain partially weight-bearing and was placed in a brace locked in full extension. The postoperative rehabilitation protocol outlined previously was initiated.

At follow-up 1 and 2 weeks postoperatively, the patient's pain had continued to diminish. By 5 weeks after surgery, the patient was ambulating without crutches, and the brace was discontinued at 9 weeks. Stationary bike and elliptical trainer exercises were then begun. Radiographs obtained 9 weeks postoperatively showed proper placement of the lateral bone bridge (Fig. 23F). At 14 weeks after surgery, the patient reported decreased lateral-sided pain and no instability. He was cleared to return to work with restrictions.

Discussion: The examination under anesthesia and diagnostic arthroscopy confirmed the lack of injury or insufficiency of the posterolateral corner of the knee. It is critical to address any ligamentous laxity simultaneously. If this patient proved to have any deficiency of the lateral structures of the knee, reconstruction or augmentation would have been necessary to increase the likelihood of a successful meniscal transplantation. Such a case illustrates the importance of a well-documented examination and the importance of preoperative planning.A surgeon performing a meniscal allograft transplantation must be prepared to address any additional pathology suspected from the physical examination and diagnostic imaging.

Case 2

Chief Complaint and Patient History: A 37-year-old man who is a laborer was referred to the authors' institution for the evaluation of worsening knee pain. He had undergone a partial medial meniscectomy for a complex tear of the left medial meniscus approximately 3 years earlier. A second debridement of the left medial meniscus was performed less than 1 year later for a recurrent meniscal tear. Since the time of the second surgery, the patient complained of worsening knee pain and intermittent effusions. The use of a cane and an off-loading brace provided only minimal relief of his knee discomfort. Additional nonoperative therapies had failed to provide symptomatic relief.

Physical Exam: On physical examination, the left lower extremity was without clinical malalignment. Tenderness to palpation was localized to the medial joint line. The knee was without an effusion, and the active and passive range of motion was normal.

Imaging: Plain weight-bearing radiographs showed minimal joint space narrowing (Fig. 24A). By MRI, a small remaining remnant of the medial meniscus and no evidence of articular lesions were confirmed (Fig. 24B).

Surgery/Treatment: The patient agreed to a meniscal allograft transplantation. Diagnostic arthroscopy at the time of the meniscal transplant showed grade III changes to the medial tibial articular surface posteriorly. The meniscal replacement was performed without complications (Fig. 24C). At 1 week after the transplant, the patient's pain was diminishing.

By 9 weeks after the transplant, the patient was ambulating with an unloader brace, had a minimal effusion, and had a passive range of motion of 0–100 degrees. At 3 months, the patient reported no complaints, the brace was discontinued, and the range of motion was 0–120 degrees. Weight-bearing radiographs performed at the 3-month follow-up showed a slight varus alignment of the left knee not previously present (Fig. 24D–F). The patient agreed to a diagnostic arthroscopy and a proximal tibial osteotomy to prevent further varus angulation and "protection" of the allograft meniscus.

Fourteen weeks after the meniscal transplant, a diagnostic arthroscopy and a closing wedge high tibial osteotomy (HTO) were performed. The allograft meniscus was secure without defect, and synovial ingrowth at the meniscal rim was present (Fig. 24G). The articular changes of the medial compartment had not progressed; however, grade III and IV Outerbridge changes were present within the patellofemoral articulation. The HTO was completed without complication. The patient was discharged home the next day and allowed to weight-bear as tolerated with a hinged knee brace locked in extension.

One week after the HTO, range of motion exercises were begun. By 5 weeks postoperatively (19 weeks after the meniscal transplant), the patient was meeting all goals set by the rehabilitation protocol. At the 6-month follow-up after the HTO, the patient had a nonantalgic gait, improved strength, and full range of motion. He was cleared to return to work.

One year after the meniscal transplant, the patient complained of occasional pain localized in the anterior knee that worsened with deep knee flexion. No joint-line tenderness was present on examination. At follow-up 2 years after the meniscal transplant, the patient reported 100% improvement in symptoms (Fig. 24H).

Five years after the meniscal allograft transplantation, the patient underwent removal of the proximal tibial hardware secondary to hardware irritation. A diagnostic arthroscopy was performed simultaneously, demonstrating an intact medial meniscus and grade III changes to the medial femoral and tibial joint surfaces; weight-bearing radiographs were taken 6 years after meniscal allograft transplantation (Fig. 24I, J). Nine years after the meniscal transplant, the patient is ambulating without a limp, has no joint-line tenderness, but has the occasional anterior knee pain. This is attributable to the patellofemoral arthrosis and employment as a laborer, which requires him to be on his feet all day.

Discussion: In hindsight, a simultaneous proximal tibial osteotomy should have been performed, potentially decreasing the surgical morbidity and recovery period. The recent report by Verdonk et al. [80] supports this point, as they reported greater clinical improvement in patients who underwent simultaneous medial meniscal allograft transplantation and high tibial osteotomy in comparison with that of patients who had an isolated medial meniscal allograft transplantation. The second- and third-look arthroscopies provide support for the theoretical benefit of meniscal transplant as evidenced by of lack of progression of the medial compartment articular changes.

Fig. 24 Case 2. (**A**) Preoperative weight-bearing radiographs showing minimal joint-line narrowing (Anterior–Posterior, AP). (**B**) Preoperative MRI scan showing only a remnant of the medial meniscus as indicated by the *arrows*. (**C**) Arthroscopic views of the transplanted meniscal allograft in the medial compartment. (**D**) Preoperative standing anteroposterior radiograph demonstrating the varus alignment and medial joint space narrowing after the meniscal transplant. (**E, F**) Radiographs after the valgus producing osteotomy to unload the medial compartment (Tunnel AP view, Flexion Lateral view). (**G**) Arthroscopic images taken 3 months later at the time of the high tibial osteotomy showing the synovial ingrowth and incorporation of the meniscal allograft. (**H**) Follow-up MRI scan performed 28 months after meniscal transplantation indicating the intact allograft. (**I**) Diagnostic arthroscopy performed 5 years after the transplantation showing an intact meniscus. (**J**) Weight-bearing radiographs 6 years after meniscal allograft transplantation (AP and Lateral views).

Fig. 24 (continued)

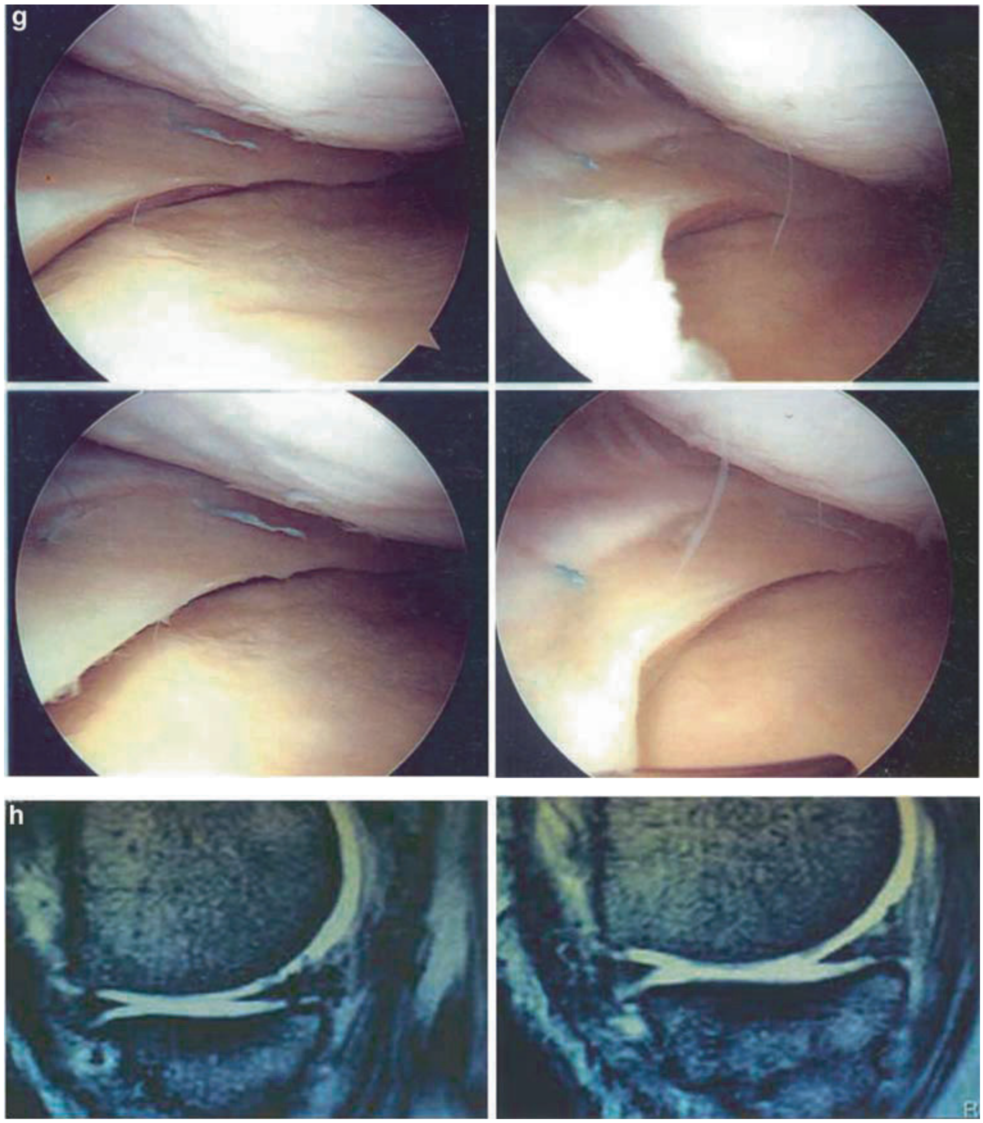


Fig. 24 (continued)

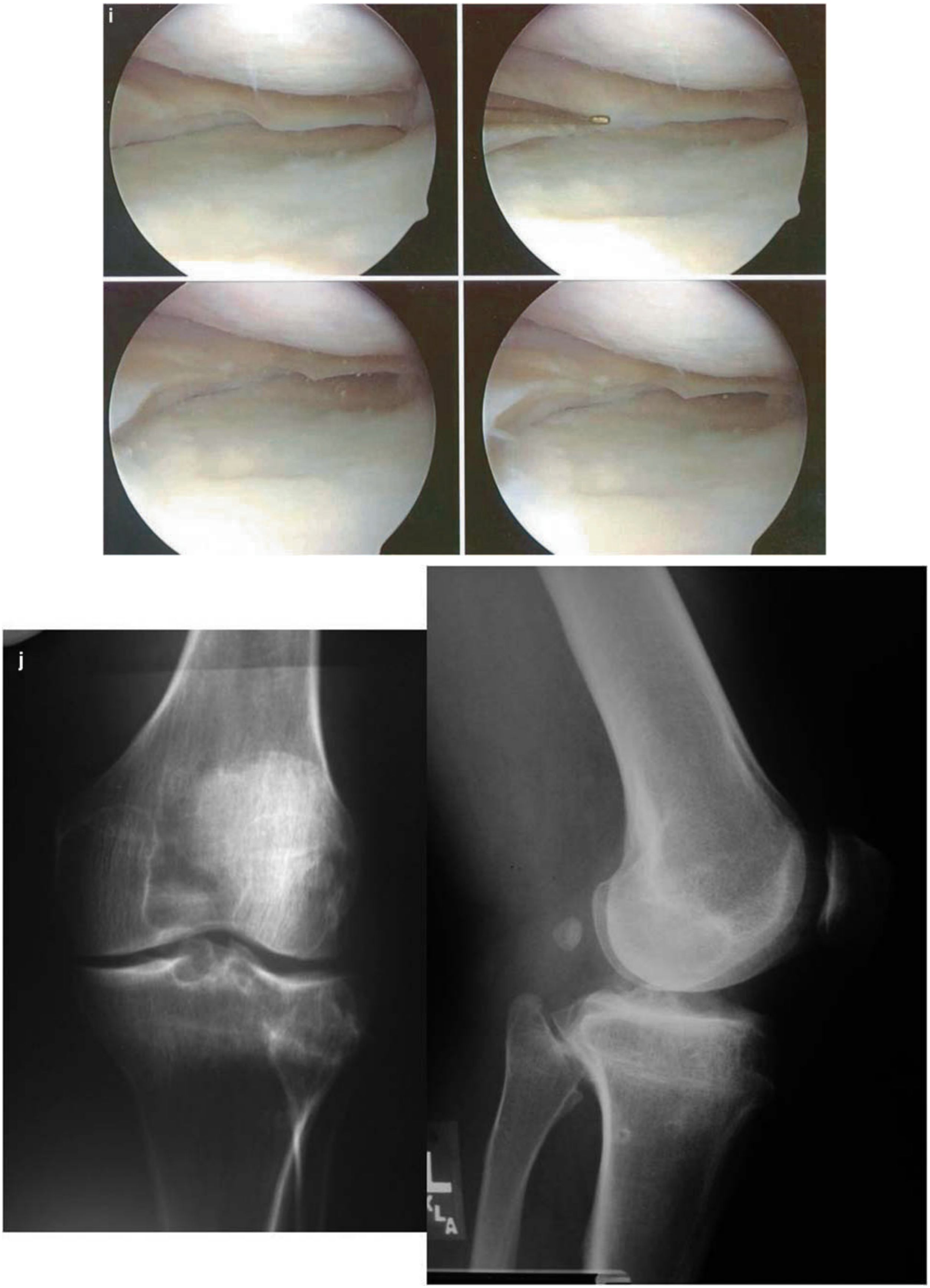


Fig. 24 (continued)

References

1. Baker PE, Peckman AC, Pupparo F, Sanborn JC. Review of meniscal injury and associated sports. *Am J Sports Med* 1985;13: 1–4.
2. Fairbanks TJ. Knee changes after meniscectomy. *J Bone Joint Surg Br* 1948;30:664–670.
3. Baratz ME, Fu FH, Mengato R. Meniscal tears: the effect of meniscectomy and of repair on intraarticular contact areas and stress in the human knee. *Am J Sports Med*1986;14:270–274.
4. Lanzer WL, Komenda G. Changes in articular cartilage after meniscectomy. *Clin Orthop*1990;252:41–48.
5. Korkala O, Karaharju E, Gronblad M, Aalto K. Articular cartilage after meniscectomy: rabbit knees studied with the scanning electron microscope. *Acta Orthop Scand*1984;55:273–277.
6. Milachowski KA, Weismeier K, Wirth CJ. Homologous meniscal transplantation: experimental and clinical results. *Int Orthop*1989;13:1–11.
7. Garrett JC, Stevenson FS. Meniscal transplantation in the human knee: a preliminary report. *Arthroscopy* 1991;7:57.
8. Whipple T. Least invasive insertion technique for meniscus transplantation. Presented at the Meniscal Transplant Study Group; February 15, 1997; San Francisco, CA.
9. Wirth CJ, Kohn D. Meniscal transplantation and replacement. In Fu FH, Harner CD, Vince KG (eds.), *Knee Surgery*. Baltimore: Williams & Wilkins, 1994, pp. 631–641.
10. McDevitt CA, Webber RJ. The ultrastructure and biochemistry of meniscal cartilage. *Clin Orthop Relat Res* 1990;252:8–18.
11. Beaupre A, Choukroun R, Guidouin R, Garneau R, Gerardin H, Cardou A. Knee menisci: correlation between microstructure and biomechanics. *Clin Orthop Relat Res*1986;208:72–75.
12. Greis PE, Bardana DD, Holmstrom MC, Burks RT. Meniscal injury: I. Basic science and evaluation. *J Am Acad Orthop Surg*2002;10:168–176.
13. Arnoczky SP, Warren RF. Microvasculature of the human meniscus. *Am J Sports Med*1982;10:90–95.
14. Kuhn JE, Wojtys EM. Allograft meniscal transplantation. *Clin Sports Med*1996;15:537–556.
15. Johnson DL, Bealle D. Meniscal allograft transplantation. *Clin Sports Med* 1999;18:93–108.
16. Hughston JC, Eilers AF. The role of the posterior oblique ligament in repairs of acute medial collateral ligament tears of the knee. *J Bone Joint Surg Am*1997;55:923.
17. Thompson WO, Thaete FL, Fu FH, Dye SF. Tibial meniscal dynamics using three-dimensional reconstruction of magnetic resonance images. *Am J Sports Med*1991;19:210–216.
18. Johnson DL, Swenson TM, Livesay GA, Aizawa H, Fu FH, Harner CD. Insertion-site anatomy of the human menisci: gross, arthroscopic, and topographical anatomy as a basis for meniscal transplantation. *Arthroscopy* 1995;11:386–394.
19. Seedhom BB. Transmission of the load in the knee joint with special reference to therole of the menisci: anatomy, analysis, and apparatus. *Sports Med Arthrosc Rev* 1996;8:207–219.
20. Voloshin AS, Wosk J. Shock absorption of meniscectomized and painful knees: a comparative in-vivo study. *J Biomed Eng*1983;5:157–161.
21. Levy IM, Torzilli PA,Warren RF. The effect of medial meniscectomy on anterior posterior motion of the knee. *J Bone Joint Surg Am*1982;64:883–887.
22. Matava MJ. Meniscal allograft transplantation: a systemic review. *Clin Orthop Relat Res*2007;455:142–157.
23. Rosenberg T, Paulos L, Parker R, Coward D, Scott S. The forty-five degree posteroanterior flexion weight-bearing radiograph of the knee. *J Bone Joint Surg Am* 1988;70: 1479–1483.
24. Noyes F, Barber-Westin S, Rankin M. Meniscal transplantation in symptomatic patients less than fifty years old. *J Bone Joint Surg Am* 2004;86:1392–1404.
25. Rath E, Richmond J, Yassir W, Albright J, Gundogan F. Meniscal allograft transplantation: two- to eight-year results. *Am J Sports Med* 2001;29:410–414.
26. Potter HG, Linklater JM, Allen AA, Hannafin JA, Haas SB. Magnetic resonance imaging of articular cartilage in the knee. An evaluation with use of fast-spin-echo imaging. *J Bone Joint Surg Am*1998;80:1276–1284.
27. Outerbridge RE. The etiology of chondromalacia patellae. *J Bone Joint Surg Br* 1961;43:752–757.
28. Alford W, Cole BJ. The indications and technique for meniscal transplant. *Orthop Clin North Am* 2005;36:469–484.
29. van Arkel E, de Boer H. Human meniscal transplantation: preliminary results at 2 to 5 year follow-up. *J Bone Joint Surg Br* 1995;77:589–595.
30. Cummins JF, Mansour JN, Howe Z, Allan DG. Meniscal transplantation and degenerative articular change: an experimental study in the rabbit. *Arthroscopy*1997;13:485–491.
31. Jackson DW, McDevitt CA, Simon TM, Arnoczky SP, Atwell EA, Silvino NJ. Meniscal transplantation using fresh and cryopreserved allografts. An experimental study in goats. *Am J Sports Med*1992;20:644–656.
32. Milachowski KA, Weismeier K, Erhardt W, Remberger K. Transplantation of the meniscus: an experimental study in sheep. *Sportverletzung Sportschaden*1987;1:20–24.
33. Mikic ZD, Brankov MZ, Tubic MV, Lazetic AB. Allograft meniscus transplantation in the dog. *Acta Orthop Scand*1993;64: 329–332.
34. Jackson DW, Whelan J, Simon TM. Cell survival after transplantation of fresh meniscal allograft. DNA probe analysis in a goat model. *Am J Sports Med*1993;21:540–550.
35. Jackson DW, Whelan J, Simon TM. Cell survival after transplantation of fresh meniscal allograft. DNA probe analysis in a goat model. *Am J Sports Med*1993;21:540–550.
36. DeBeer P, Decorte R, Delvaux S, Bellemans J. DNA analysis of a transplanted cryopreserved meniscal allograft. *Arthroscopy*2000;16:71–75.
37. Brown KL, Cruess RL. Bone and cartilage transplantation in orthopaedic surgery. A review. *J Bone Joint Surg Am*1982;64: 270–279.
38. Graham WC, Smith DA, Mcguire MP. The use of frozen stored tendons for grafting: an experimental study. *J Bone Joint Surg Am* 1955;37:624.
39. Arnoczky SP, McDevitt CA, Schmidt MB, Mow VC, Warren RF. The effect of cryopreservation in canine menisci: a biochemical morphologic and biochemical evaluation. *J Orthop Res*1988;6: 1–12.
40. Amoczky SP, DiCarlo EF, O'Brien SJ, Warren RF. Cellular repopulation of deep-frozen meniscal autografts: an experimental study in the dog. *Arthroscopy* 1992;8:428–436.
41. Kuhn JE, Wojtys EM. Allograft meniscal transplantation. *Clin Sports Med*1996;15:537–556.
42. Fabbriciani C, Lucania L, Milano G, Schiavone Panni A, Evangelisti M. Meniscal allografts: cryopreservation vs deep-frozen technique. An experimental study in goats. *Knee Surg Sports Traumatol Arthrosc*1997;5:124–134.
43. Siegel MG, Roberts CS. Meniscal allografts. *Clin Sports Med*1993;12:59–80.
44. Garrett JC. Meniscal transplantation. In Aichroth PC, Canon WD, Patel DV (eds.), *Knee Surgery: Current Practice*. New York: Raven, 1992, pp. 95–103.
45. Veltri DM, Warren RG, Wickiewicz TL, O'Brien SJ. Current status of allograft meniscal transplantation. *Clin Orthop Relat Res* 1994;303:44–55.

46. Pollard M, Kang Q, Berg E. Radiographic sizing for meniscal transplantation. *Arthroscopy* 1995;11:684–687.
47. L'Insalata JC, Klatt B, Caravaggio H, Fu FH, Harner CD. Accuracy and reliability of office techniques for meniscal allograft sizing. Presented at the Annual Meeting of the Meniscal Transplantation Study Group; February 24, 1996; Atlanta, GA.
48. Garrett JC. Meniscal transplantation: review of forty three cases with two-to-seven year follow up. *Sports Med Arthrosc Rev* 1993;1:164–167.
49. Cameron JC, Saha S. Meniscal allograft transplantation for unicompartmental arthritis of the knee. *Clin Orthop* 1997;337: 164–171.
50. Carter TR. Meniscal allograft transplantation. *Sports Med Arthrosc Rev* 1999;7:51–62.
51. Cole BJ, Fox JA, Lee SJ, Farr J. Bone bridge in slot technique for meniscal transplantation. *Oper Tech Sports Med* 2003;11: 144–155.
52. Farr J, Meneghini RM, Cole BJ. Allograft interference screw fixation in meniscus transplantation. *Arthroscopy* 2004;20: 322–327.
53. Fox JA, Lee SJ, Cole BJ. Bone plug technique for meniscal transplantation. *Oper Tech Sports Med* 2003;11:161–169.
54. Stollsteimer GT, Shelton WR, Dukes A, Bomboy L. Meniscal allograft transplantation: a 1- to 5-year follow-up of 22 patients. *Arthroscopy* 2000;16:343–347.
55. Ellington CI, Sekiya JK, Harner CD. Meniscal allograft transplantation. In El Attrache NS, Harner CD, Mirzayan R, Sekiya, JK. (eds.), *Surgical Techniques in Sports Medicine*, Philadelphia: Lippincott Williams & Wilkins, 2007, pp. 477–482.
56. Kang RW, Lattermann C, Cole BJ. Allograft meniscal transplantation: background, indications, techniques, and outcomes. *J Knee Surg* 2006;19:220–230.
57. Alhalki MM, Howell SM, Hull ML. How three methods for fixing a medial meniscal allograft affect tibial contact mechanics. *Am J Sports Med*1999;27:320–328.
58. Alhalki MM, Hull ML, Howell SM. Contact mechanics of the medial tibial plateau after implantation of a medial meniscal allograft. *Am J Sports Med*2000;28:370–376.
59. Chen MI, Branch TP, Hutton WC. Is it important to secure the horns during lateral meniscal transplantation? A cadaveric study. *Arthroscopy*1996;12:174–181.
60. Paletta GA, Manning T, Snell E, Parker R, Bergfeld J. The effect of allograft meniscal replacement on intraarticular contact area and pressures in the human knee: a biomechanical study. *Am J Sports Med*1997;25:692–698.
61. Ghazavi MT, Pritzker KP, Davis AM, Gross AE. Fresh osteochondral allografts for post-traumatic osteochondral defects of the knee. *J Bone Joint Surg Br* 1997;79:1008–1013.
62. Yoldas EA, Sekiya JK, Irrang, JJ, Fu FH, Harner CD. Arthroscopically assisted meniscal allograft transplantation with and without combined anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc*2003;11:173–182.
63. Cole BJ, Carter TR, Rodeo SA. Allograft meniscal transplantation: background, techniques, and results. *Instr Course Lect* 2003;52:383–396.
64. Sekiya J, Giffin J, Irrgang J, Fu F, Harner C. Clinical outcome after combined meniscal allograft transplantation and anterior cruciate ligament reconstruction. *Am J Sports Med* 2003;31:896–906.
65. Graf K, Sekiya J, Wojtys E. Long-term results after combined medial meniscal allograft transplantation and anterior cruciate ligament reconstruction: minimum 8.5-year follow-up study. *Arthroscopy*. 2004;20:129–140.
66. Levy IM, Torzilli PA, Warren RF. The effect of medial meniscectomy on anterior-posterior motion of the knee. *J Bone Joint Surg Am* 1982;64:883–888.
67. Shelbourne KD, Gray T. Results of anterior cruciate ligament reconstruction based on meniscus and articular cartilage status at the time of surgery. Five to fifteen-year evaluations. *Am J Sports Med* 2000;28:446–452.
68. Barrack RL, Bruckner JD, Kneisl J, Inman W, Alexander A. Outcome of nonoperatively treated complete tears of the anterior cruciate ligament in active young adults. *Clin Orthop Relat Res* 1990;259:192–199.
69. Bonamo JJ, Fay C, Firestone T. The conservative treatment of the anterior cruciate deficient knee. *Am J Sports Med*1990;18: 618–623.
70. Zukor DJ, Cameron JC, Brooks PJ, et al. The fate of human meniscal allografts. In Ewing JW (ed.), *Articular Cartilage and Knee Joint Function: Basic Science and Arthroscopy.*New York: Raven Press, 1990, p. 147.
71. Cole BJ, Dennis MG, Lee S, Nho S, Kalsi RS, Hayden JK, Verma NN. Prospective evaluation of allograft meniscus transplantation: minimum 2-year follow-up. *Am J Sports Med* 2006;13:1–9.
72. Albrecht-Olsen P, Lind T, Kristensen G, Falkenberg B. Failure strength of a new meniscus arrow repair technique: biomechanical comparison with horizontal suture. *Arthroscopy*1997;13:183–187.
73. Wirth C, Peters G, Milachowski K, Weismeier K, Kohn D. Long-term results of mensical allograft transplantation. *Am J Sports Med* 2002;30:174–181.
74. Ryu R, Dunbar W, Morse G. Meniscal allograft replacement: a 1-year to 6-year experience. *Arthroscopy*. 2002;18:989–994.
75. Kocabey Y, Taser O, Nyland J, Doral MN, Demirhan M, Caborn DN, Sarban S. Pullout strength of meniscal repair after cyclic loading: comparison of vertical, horizontal, and oblique suture techniques. *Knee Surg Sports Traumatol Arthrosc* 2006;14:998–1003.
76. Tegner Y, Lysholm J. Rating systems in the evaluation of knee ligament injuries. *Clin Orthop Relat Res* 1985;198:43–49.
77. Verdonk PC, Demurie A, Almqvist KF, Veys EM, Verbruggen G, Verdonk R. Transplantation of viable meniscal allograft. Survivorship analysis and clinical outcome of one hundred cases. *J Bone Joint Surg Am*2005;87:715–724.
78. Sekiya JK, West RV, Groff YJ, Irrgang JJ, Fu FH, Harner CD. Clinical outcomes following isolated lateral meniscal allograft transplantation. *Arthroscopy*2006;18:771–780.
79. Noyes F, Stabler C. A system for grading articular cartilage lesions at arthroscopy. *Am J Sports Med* 1989;17:505–513.
80. Verdonk PC, Verstraete KL, Almqvist KF, De Cuyper K, Veys EM, Verbruggen G, Verdonk R. Meniscal allograft transplantation: long-term clinical results with radiological and magnetic resonance imaging correlations. *Knee Surg Sports Traumatol Arthrosc* 2006;14:694–706.
81. Shelton WR. Meniscus allograft transplantation. *Oper Tech Sports Med* 2007;15:81–85.
82. Hommen JP, Applegate GR, Del Pizzo W. Meniscus allograft transplantation: ten-year results of cryopreserved allografts. *Arthroscopy*2007;23:388–393.

Arthroscopic Treatment of the Osteoarthritic Knee

Carl T. Talmo and James V. Bono

Introduction

The knee is the most commonly affected weight-bearing joint by osteoarthritis, accounting for approximately 1 million surgical procedures yearly and for the majority of nonsteroidal anti-inflammatory drugs purchased in the United States. More than 20 million people in the United States are afflicted with early osteoarthritis, and it is the leading cause of limitation of activities of daily living and second only to cardiac disease in causing loss of time from vocational activities and work disability [1, 2]. In addition, an increased life expectancy and increased awareness of the benefits of physical fitness and participation in sports activities has resulted in a larger population with chronic articular cartilage injuries and early degenerative disease and higher expectations with respect to activity levels and recreational activities [3].

These active individuals are frequently resistant to and dissatisfied with total knee arthroplasty as a long-term solution to their symptoms. In addition, survival rates for total knee arthroplasty in very active individuals under the age of 55 years have historically been unsatisfactory [4, 5]. Many of these patients may be candidates for osteotomy; however, frequently these patients are also candidates for arthroscopic debridement for symptomatic relief of pain and mechanical symptoms without the risks and recovery associated with osteotomy.

The etiology of osteoarthritis of the knee stems from a myriad of causes, including traumatic, genetic, iatrogenic, as well as idiopathic. Discerning the causative factors in any given case may contribute to the prognosis after arthroscopic debridement [6–8]. In general, exacerbation of pain and mechanical symptoms associated with injury to an osteoarthritic joint will be associated with a better prognosis after arthroscopic treatment than will other causes [9–11].

The pathology of osteoarthritis consists of a predictable sequence of loss of articular cartilage accompanied by changes in its cellular and acellular composition, ineffective repair processes, and remodeling of subchondral and juxtaarticular bone. In addition, there is also thickening of the joint capsule, inflammation in the synovium, and bone cyst formation. The pain and symptoms associated with these changes are frequently unpredictable as is the rate of progression, which can complicate the outcome of arthroscopic debridement for this disease [10, 12].

Numerous theories have been proposed to account for the pain generators in the osteoarthritic joint and how these might account for symptomatic improvement after direct treatment. Various contributing factors include electromechanical influences on chondrocyte activity, changes in humoral, synovial, and chemical factors within the joint, immune response to proteoglycan and collagen breakdown products within the synovium, altered joint mechanics and irritation of unprotected subchondral bone, meniscal pain, and the presence of inflammatory mediators and degradative enzymes within synovial fluid. Synovial fluid in the osteoarthritic knee contains disproportionate concentrations of catabolized matrix proteins, interleukins, collagenases, metalloproteinases, and numerous other enzymes [13]. The presence of this altered biochemical milieu has inspired many researchers to investigate the therapeutic effects of arthroscopic lavage and its potential for symptomatic improvement. However, the possibility of altering or delaying the natural history of the disease in this manner seems unlikely and remains controversial [6, 14].

History

Reports of arthroscopic treatment of the arthritic knee originate in the 1920s when Bircher reported on beneficial effects of diagnostic arthroscopy [15]. Burman et al. reported on the use of arthroscopic lavage of the knee in 10 patients with osteoarthritis in the 1930s, reporting significant improvement

C.T. Talmo (✉)
Department of Orthopaedic Surgery, Tufts University School of Medicine, New England Baptist Hospital, Boston, Massachusetts

B.P. McKeon et al. (eds.), *Knee Arthroscopy*, DOI 10.1007/978-0-387-89504-8_5,

in all patients [16]. Watanabe et al. later championed the use of arthroscopic lavage in reducing symptoms of osteoarthritis in the 1950s [17].

Following on the experimentation of Magnusun and Pridie in open debridement of the knee, a number of investigators began exploring arthroscopic debridement and marrow stimulation techniques in the treatment of osteoarthritic knee in the 1970s after numerous technologic advancements in arthroscopic equipment. The use of arthroscopy as a diagnostic tool in the assessment of the extent of cartilage injury became widespread at this time, aiding in predicting one's candidacy for osteotomy or prosthetic replacement [11, 18, 19].

In the past decade, a growing body of research has been devoted to further defining the most appropriate indications for arthroscopic interventions for osteoarthritis in large part due to economic pressures and the fear that arthroscopy is overused with little real benefit to a large subset of this population [9, 12, 20, 21].

Indications

Although it continues to be mired in controversy, the arthroscope remains a useful tool in the surgeon's arsenal for the treatment of the degenerative knee. Diagnostic arthroscopy is frequently helpful in defining the extent of degenerative changes in the younger patient with precocious arthritis or when there is suspicion of multicompartment disease. Planning of subsequent treatment from cartilage grafting to unicompartmental or total joint arthroplasty is efficiently accomplished in this manner. Concomitant cartilage or synovial biopsy also can be performed for subsequent diagnostic and therapeutic interventions as well. Though frequently elusive and frequently requiring an associated arthrotomy, loose bodies associated with pain or mechanical symptoms may be removed arthroscopically.

A number of studies have demonstrated discrepancies between x-ray findings and arthroscopic evaluation in the diagnosis of osteoarthritis. A significant number of patients with debilitating knee pain (up to 33%) and preoperative x-rays demonstrating joint space narrowing have been demonstrated to have normal joint surfaces upon arthroscopic evaluation [22]. Lysholm et al., on the other hand, found that only patients with Outerbridge IV [23] changes at arthroscopy had preoperative radiographs consistent with osteoarthritis [18].

A number of retrospective studies have shown significant benefit after arthroscopic treatment of unicompartmental osteoarthritis associated with mild degenerative changes, normal alignment, and unstable meniscal tears [6–8, 24, 25]. Other studies have demonstrated poorer results in the setting of malalignment associated with osteoarthritis [9, 24, 26]. Varus malalignment may impart a worse prognosis than that of increased valgus [24]. Other risk factors associated with a poor prognosis include severe or tricompartmental disease and calcium pyrophosphate deposition [7, 13].

Critics of arthroscopic debridement would argue how many patients go on to further surgery and total knee arthroplasty after arthroscopy and that theoretically some patients might be made worse by removal of functional meniscal tissue and cartilage. Indeed, partial meniscectomy may increase the force transmitted across the articular surfaces of the tibiofemoral joint by as much as 45% [27]. However, other studies have failed to demonstrate a negative impact of arthroscopy over time. In a retrospective study, Pearse and Craig demonstrated that meniscal debridement did not hasten the progression of osteoarthritis to joint arthroplasty over lavage alone [28].

The simple presence of a meniscal tear in the osteoarthritic knee should not be used alone as an indication for arthroscopic intervention. Magnetic resonance imaging (MRI) studies have demonstrated a 91% prevalence of meniscal tears in knees with osteoarthritis compared with 76% in a population of asymptomatic subjects. Furthermore, it has been postulated that the torn meniscus is an infrequent source of pain in the osteoarthritic knee [29]. However, there is excellent evidence that traumatic tears in the osteoarthritic knee associated with mechanical symptoms and appropriate physical exam findings indicate a good candidate for arthroscopic treatment [9, 12, 26, 30].

Authors' Preferred Surgical Technique

After the induction of general or spinal anesthesia in the supine position, a tourniquet and leg holder are applied at approximately the middle of the thigh. If the leg holder is applied too distally, it may interfere with use of superior portals when necessary, and, if applied too proximally, it will prevent the appropriate counterforce when manipulating the leg into varus or valgus for visualization of the medial or lateral compartment. This is particularly important in a degenerative knee where stiffness, joint space narrowing, and capsular contraction may make visualization and instrumentation of the compartments more challenging, requiring a better mechanical advantage upon the extremity.

Prior to draping, the knee is sterile injected with 30 mL 0.5% Marcaine (Bupivacaine Hcl, AstraZeneca, London, UK) with epinephrine for hemostasis and anesthesia. Use of this technique has made the need for insufflation of the tourniquet uncommon. The anterolateral portal is established just lateral to the patella tendon in the soft-spot at approximately the inferior pole of the patella. A vertical or oblique incision is preferred, in the event that capsular

Table 1 Outerbridge grading of chondral surface lesions

Grade I: Softening and swelling of cartilage (Fig. 1)
Grade II: Fragmentation and fissuring, less than 0.5-inch-diameter lesion (Fig. 2)
Grade III: Fragmentation and fissuring, greater than 0.5-inch-diameter lesion (Fig. 3)
Grade IV: Erosion of cartilage down to exposed subchondral bone (Fig. 4)

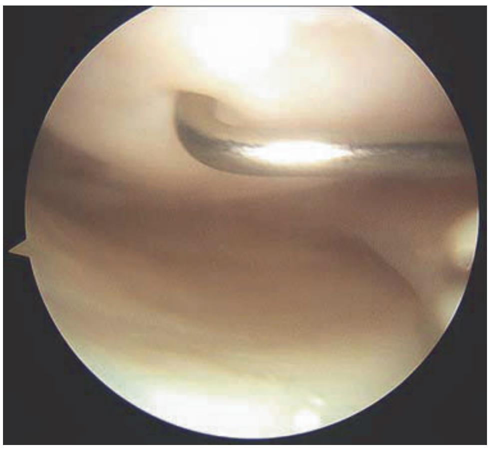


Fig. 1 Outerbridge grade I lesion

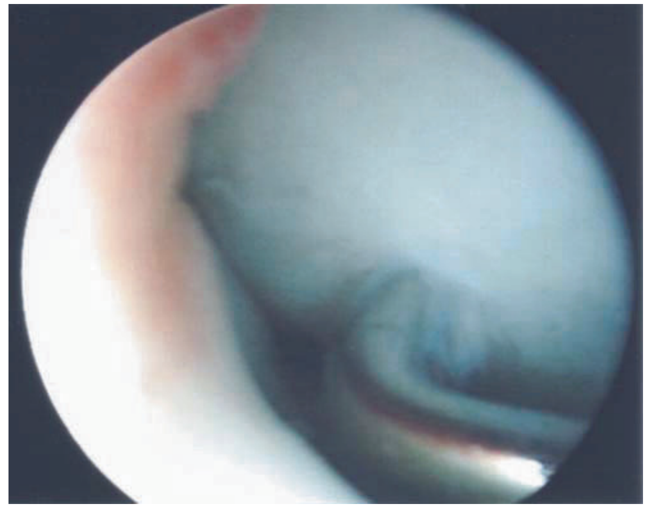


Fig. 2 Outerbridge grade II lesion of the lateral femoral condyle. Note probe used to estimate size of lesion

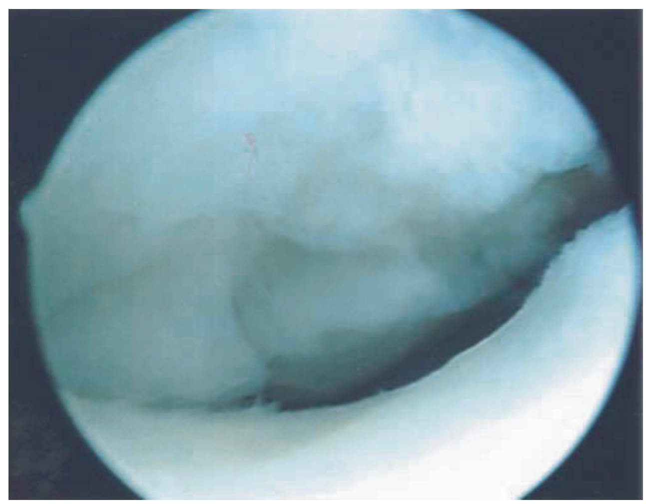


Fig. 3 Outerbridge grade III lesion of the patella

contracture or osteophytes in the intercondylar notch restrict the mobility of the arthroscope, such that it can be raised or lowered slightly to avoid these obstructions. The incision is carried through skin and capsule angled slightly into the intercondylar notch. The blunt arthroscopic cannula is inserted initially into the notch area with the knee flexed to avoid injury to articular cartilage and then redirected into the suprapatellar pouch as the knee is carried into full extension.

Routine diagnostic arthroscopy is then carried out. Visualization of the suprapatellar pouch may reveal synovitis common in degenerative conditions and cartilaginous loose bodies. Routine synovectomy in this area is not typically carried out. The facets of the patella and trochlea are visualized, and grading of cartilage injury is noted using the Outerbridge classification (Table 1; Figs. 1–4).

The arthroscope is then directed laterally over the trochlea ridge and to the lateral gutter. Care must be taken in translating the arthroscope over the lateral edge of the trochlea as a prominent osteophyte may be present in this area and the scope may have to be levered to prevent cartilage damage of fracture of osteophyte, potentially contributing to postoperative pain. The scope is directed down the lateral gutter while slightly retracting the arthroscope in order to avoid the lateral synovial fold and then advanced into the area of the popliteal hiatus, a common hiding place for degenerative loose bodies. While in the lateral gutter, femoral osteophytes may be visualized as well as the peripheral aspect of the mobile lateral meniscus (Fig. 5). Peripheral lateral meniscal tears or subluxated flaps of torn meniscus may be visualized in this area along with synovitis adjacent to painful tears.

If the trochlea cannot be gently negotiated, the scope should be redirected carefully down the middle of the trochlea while visualizing the articular cartilage into the medial compartment or intercondylar notch area.

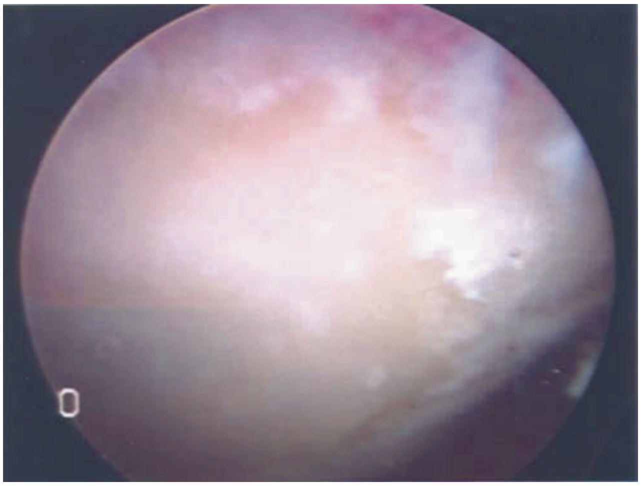


Fig. 4 Outerbridge grade IV lesion of the medial femoral condyle

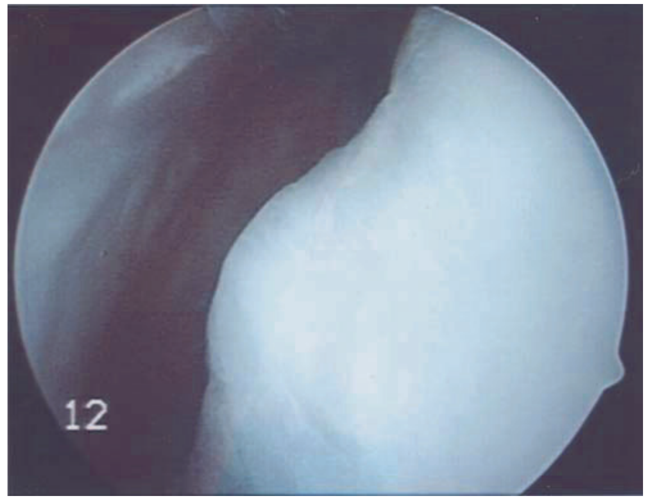


Fig. 6 Tracing the arthroscope down the medial femoral condyle demonstrates intact articular cartilage and medial osteophyte formation

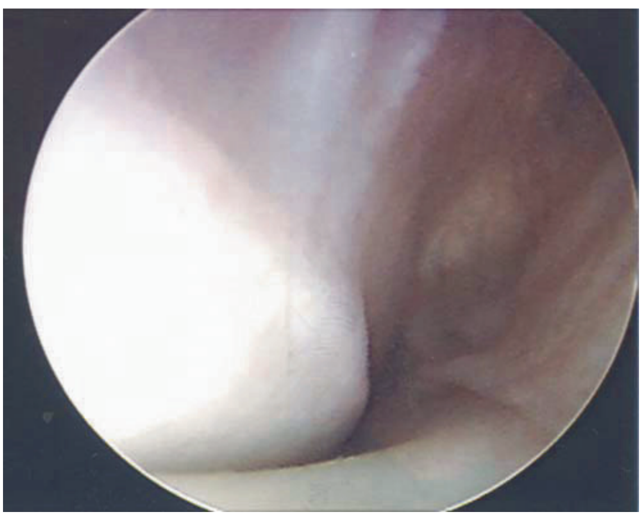


Fig. 5 Visualization of lateral gutter demonstrates intact peripheral rim of the lateral meniscus and popliteus hiatus in the background. Note early osteophyte formation on the lateral femoral condyle

The scope is then redirected into the suprapatellar pouch and across to the medial gutter. The articular cartilage of the medial femoral condyle is carefully traced and visualized as the scope is directed into the medial compartment (Fig. 6). A gentle valgus stress is applied to the knee in midflexion for visualization of the medial compartment. The anteromedial portal is then established for insertion of a probe.

The authors' technique for establishing the anteromedial portal allows for minimal violation of the joint capsule and minimizes the potential for iatrogenic articular cartilage injury. A 17-gauge spinal needle is placed through the skin anteromedially and into the medial compartment of the knee under direct vision. Advancing a small-bore hypodermic needle while instilling local anesthetic helps determine the proper orientation prior to inserting the spinal needle. A skin incision but no formal arthrotomy is made. The stylet from the spinal needle is removed, and a flexible wire is passed through the needle and into the joint. Once proper positioning of the needle and guide wire have been confirmed, the needle is removed and a cannulated switching stick is placed over the guide wire to provide gentle dilation of the needle arthrotomy (Fig. 7). The switching stick may then be removed and exchanged for a small cannula, which is passed over the guide wire. The guide wire can then be removed, and arthroscopic probe is introduced through the cannula (Fig. 8).

A shaver or arthroscopic punch also can be inserted atraumatically using this technique (Figs. 9 and 10). After removal of the switching stick, the inner shaving portion of an arthroscopic shaver is disengaged from its outer barrel. The outer barrel is then placed over the guide wire and the guide wire removed. The inner shaver is then reassembled with the barrel, and arthroscopic debridement is commenced. A similar technique of cannulation using the outer barrel of an arthroscopic shaver has been described by Shen and Meislin for use in hip arthroscopy [31].

The medial meniscus is probed on its superior and inferior surface, and tears are identified. Frequently adjacent chondral injury/degeneration is also present. Horizontal cleavage and complex tears are common in the degenerative knee.

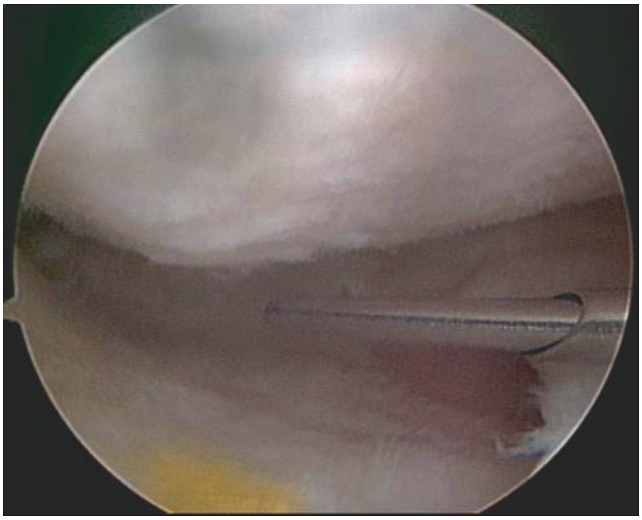


Fig. 7 Serial dilation of medial portal is initiated over a cannulated switching stick

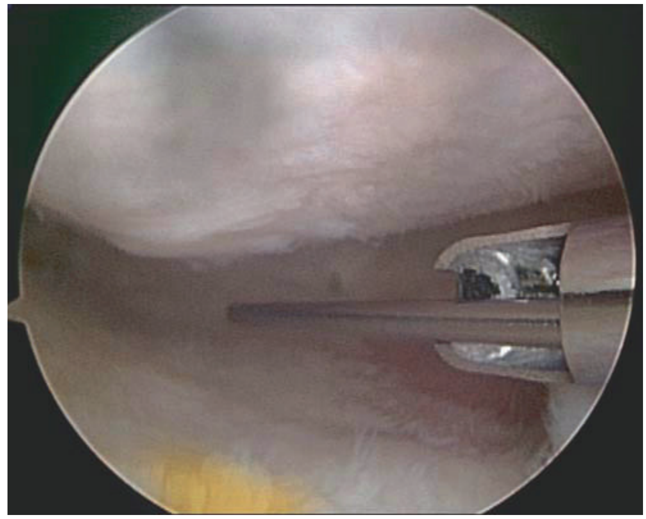


Fig. 9 Arthroscopic shaver is atraumatically introduced over switching stick

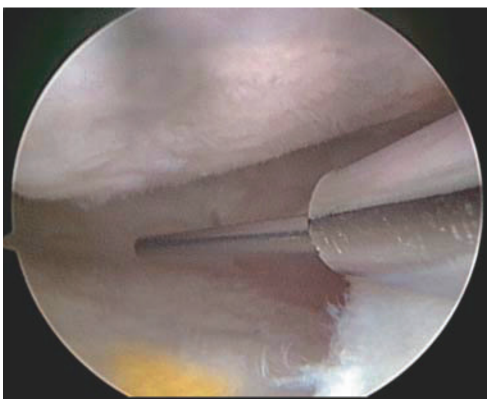


Fig. 8 Larger arthroscopic cannula is placed over switching stick

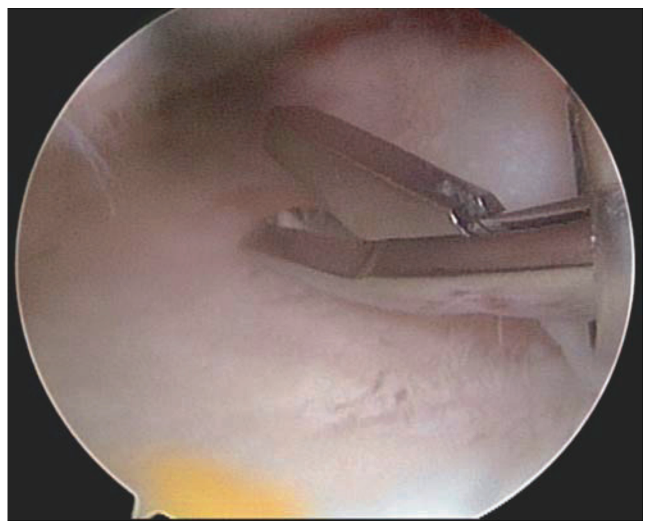


Fig. 10 Arthroscopic punch is inserted atraumatically through large medial cannula

The minimum resection of meniscus that results in a stable peripheral rim should be resected (Fig. 11A–C). The authors' preference is to use a 4.5mm full radius shaver, as the degenerative meniscus is often firm and rubbery and resistant to debridement with less aggressive instruments. Arthroscopic punches are useful for initiating the resection at the apex of the tear followed by the shaver. Horizontal tears are approached by debriding the more unstable flap of meniscus, frequently the inferior aspect, with preservation of the remaining tissue if stable (Fig. 12A–C). Occasionally, synovitis may be detected at the menisco-synovial junction associated with a tear (Fig. 13A, B). Gentle synovectomy may be performed (Fig. 13C); however, care should be taken, as overaggressive synovial debridement may lead to a painful postoperative hemarthrosis. Care is taken to pass a probe under the meniscus in the vicinity of the tear and over the edge of the tibia, as a flap of torn tissue may sublux medial to the joint, resulting in failure of the procedure if left undetected. Unstable flaps of articular cartilage may be the source of mechanical symptoms and should be debrided back to a stable rim to prevent further propagation. Only the minimum amount of articular cartilage is resected.

The intercondylar notch is then inspected, and the anterior cruciate ligament (ACL) is probed. If a loose body is suspected or if there is interest in decompression of an arthritic popliteal cyst, the arthroscope can be directed under the superior aspect of the posterior cruciate ligament (PCL) just

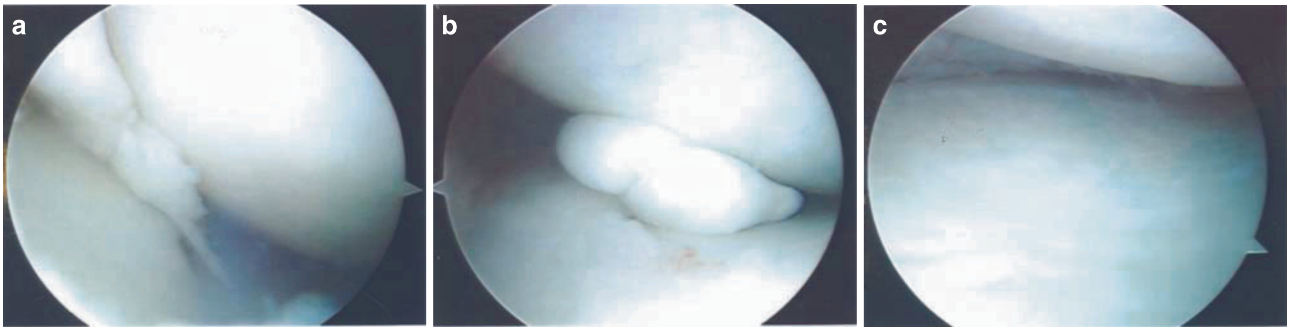


Fig. 11 (**A, B**) Unstable tear of the medial meniscus associated with Outerbridge grade II changes of the medial femoral condyle. (**C**) Image taken after debridement of unstable meniscal fragments to a stable rim of the medial meniscus

lateral to the medial femoral condyle and with modest force directed into the posterior aspect of the knee. Frequently, the arthroscope must be replaced with the tapered blunt stylus to pass into this area.

The arthroscope is then directed lateral to the lateral tibial spine, and the limb is taken into a figure-of-four position so that the lateral compartment may be visualized. Preservation of as much healthy lateral meniscus as possible is paramount to the maintenance of normal knee kinematics. Visualization of the anterior horn of the lateral meniscus may be challenging from the anterolateral portal, and, if a tear is suspected in this area, visualization may be improved from the medial portal.

Finally, attention is returned to the patellofemoral joint, where debridement of unstable flaps of cartilage may improve mechanical symptoms.

Rehabilitation

Patients are mobilized weight-bearing as tolerated immediately with crutches to wean as tolerated, and active range of motion exercises are begun upon discharge. Patients are instructed in quadriceps strengthening exercises to be performed daily. Deep venous thrombosis (DVT) prophylaxis includes aspirin daily, and T.E.D Covidien (Mansfield, Ma). stockings are encouraged for 1 month after the procedure. Patients are seen at 2–3 weeks postoperatively to review arthroscopy findings and discuss prognosis. Patients with extensor lag, quadriceps atrophy, or limited range of motion are referred for formal outpatient physical therapy. Patients should be counseled that maximal improvement in symptoms may not be experienced until 3–4 months postoperatively.

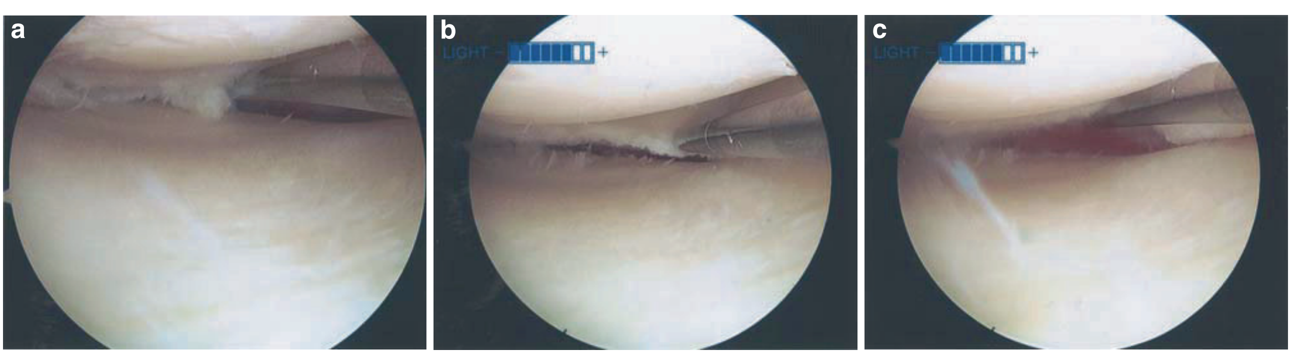


Fig. 12 (**A**) Degenerative horizontal cleavage tear of the posterior horn of the medial meniscus associated with Outerbridge grade II changes of the medial femoral condyle and grade III changes of the tibial plateau. After debridement of the unstable inferior flap of the tear, the (**B**) superior and (**C**) inferior surfaces of the meniscus are probed and inspected to ensure that debridement is complete

Complications

Complications after arthroscopic knee surgery are fortunately rare and include hemarthrosis, infection, thromboembolic disease, nerve injury, reflex sympathetic dystrophy, osteonecrosis, and ligament injury [32, 33]. The most common complication in the degenerative knee is that of recurrent pain and swelling secondary to underlying osteoarthritis with eventual progression to and the need for total knee replacement. Based on the available literature, this can be anticipated in at least 10% of this population by 2 years postoperatively [9, 12, 20, 21, 26, 30]. Though uncommon, spontaneous osteonecrosis can be a quite disturbing outcome after this procedure, with severe unrelenting pain and a decline in function that is frequently worse than the patient's preoperative symptoms. Because of the potential for this and other negative outcomes, it is important to counsel patients that, whereas the procedure is low risk, there is a small chance of worsening symptoms after the procedure. In addition, pain that does not improve in 6 weeks warrants repeat MRI scan to rule out post-arthroscopy osteonecrosis, which may benefit from a period of protected weight-bearing [33].

There is significant concern for thromboembolic disease in this frequently older population, many with concurrent underlying venous insufficiency. Fortunately, the incidence of symptomatic DVT and pulmonary embolism (PE) remains quite rare. Investigations into the rate of symptomatic DVT after arthroscopic procedures have demonstrated a rate of approximately 0.5%. Studies using ultrasonography or other imaging modalities at a set interval postoperatively indicate a rate of up to 17%, most of which are asymptomatic and localized to the calf [34].

Results

The results of arthroscopic debridement for internal derangements of the knee in the setting of osteoarthritis have been mixed but mostly favorable at short-term and intermediate-term follow-up in the literature. Harwin reviewed the results of arthroscopic debridement in 204 knees with osteoarthritis. At an average of 7.4 years follow-up, he found significantly better results in patients with a more normal mechanical alignment, including satisfactory results in 84% of patients with normal alignment. Older age and previous surgery were also risk factors for a poorer outcome [26]. In a retrospective review of 36 patients undergoing arthroscopic debridement, Fond et al. demonstrated improvements in pain and function (by hospital for special surgery (HSP), New York, N.Y. knee scores) in 88% at 2 years and in 69% at 5 years. Risk factors for failure of arthroscopic debridement included a greater preoperative flexion contracture and a lower preoperative HSS score [30]. Whereas a number of retrospective studies like these have demonstrated significant benefit for arthroscopic debridement of the osteoarthritic knee, most are lacking validated outcomes scores for pain and function.

Other intermediate-term studies have demonstrated poorer results for arthroscopic debridement in osteoarthritis. Dervin et al. prospectively evaluated 126 patients undergoing arthroscopic meniscal and chondral debridement using Western Ontario and McMaster Osteoarthritis Index (WOMAC), The SF-36 is a multi-purpose, short-form health survey with only 36 questions (SF-36) and SF-36: 44% were rated as having had a clinically important reduction in pain, by the WOMAC pain scale, at 2 years. Physicians were poor at predicting which patients would improve. Three variables were significantly associated with improvement after arthroscopic

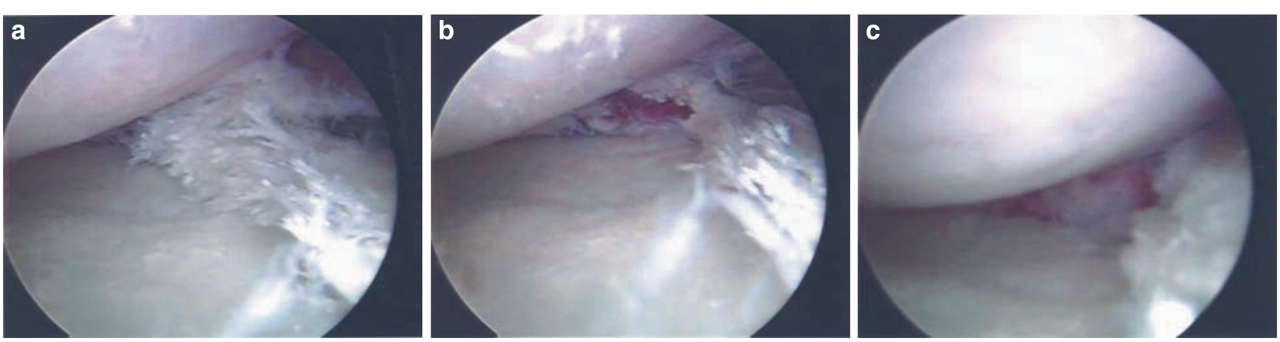


Fig. 13 (**A**) Degenerative tear of medial meniscus associated with chondrocalcinosis and Outerbridge grade III changes of the medial femoral condyle and medial plateau. (**B**) Note inflamed synovium at the posterior apex of the tear. (**C**) After debridement of meniscus and synovium, patient had improvement in pain and mechanical symptoms for more than a year postoperatively

debridement: the presence of medial joint-line tenderness, a positive Steinman test, and the presence of an unstable meniscal tear at arthroscopy [12]. Other longitudinal studies of administrative data sets have demonstrated that 18.4% of 6,212 Canadian patients undergoing arthroscopic debridement underwent total knee replacement (TKR) within 3 years [21]. In a prospective randomized study of arthroscopic debridement in a population of veterans with osteoarthritis, Moseley et al. demonstrated no benefit to debridement over placebo surgery [20]. These results have remained controversial as patients were not excluded based on previously known risk factors for poor outcome such as malalignment and significant joint contracture.

More recently, Aaron et al. reviewed the results of arthroscopic debridement in a consecutive group of 110 patients with osteoarthritis at a mean 34 months postoperatively. They found that 90% of patients with mild arthritis, normal alignment, and joint space >3 mm had significant improvement in Knee Society pain scores when compared with patients who had evidence of severe arthritis, malalignment, and a joint space <2 mm (25% improved). The severity of the articular lesion as graded intraoperatively was also predictive of outcome [9].

In the authors' experience, arthroscopic debridement and lavage provide short- to intermediate-term symptomatic improvement to the majority of patients with pain and mechanical symptoms associated with meniscal tear in the setting of mild osteoarthritis. The results are generally better in younger patients with normal alignment, a recent history of trauma or injury, and a shorter duration of symptoms. Absolute contraindications include severe or multicompartmental disease, malalignment of more than 3–5 degrees from the mechanical axis, the absence of mechanical symptoms or joint-line tenderness, and significant joint contracture or stiffness.

Clinical Pearls/Summary

Use of arthroscopy in the management of the osteoarthritic knee remains controversial. Careful patient selection is paramount to good results and improvement in patient satisfaction. In the appropriate patient with osteoarthritis, arthroscopy can result in sustained relief of pain and improvement in mechanical symptoms and activity levels. Risk factors for poor results include severe disease, contractures, malalignment, and the absence of mechanical symptoms, a history of injury, or joint-line tenderness.

Operative technique should emphasize avoiding any further injury to articular surfaces, which may hasten the progression of disease. Unstable meniscal tears and loose flaps of cartilage are debrided; however, aggressive chondroplasty and use of marrow stimulation techniques should be avoided in patients with significant osteoarthritis. Particular attention is paid to subluxated flaps of meniscal tissue, which can be a source of persistent pain if undetected. The role of joint lavage is unknown but seems unsupported by the current body of literature.

Case Report

Case 1

Chief Complaint and Patient History: A 55-year-old active woman with a history of mild osteoarthritis of both knees presented 8 weeks after a mild twisting injury to the left knee, complaining of progressive medial-sided knee pain associated with episodes of swelling, buckling, and occasional locking sensation.

Physical Exam: Physical exam demonstrated a trace effusion, medial joint-line tenderness, and pain with deep flexion. Lachman test and ligamentous examination was negative.

Imaging: Radiographs (Fig. 14A) reveal mild medial compartment narrowing on both knees, which was worse on the right than on the left. MRI scans (Fig. 14B, C) show degenerative changes and chondromalacia present in the medial compartment associated with degeneration and tear of the medial meniscus.

Surgery/Treatment: Arthroscopy was performed, initially demonstrating Outerbridge grade III changes in the medial compartment (Fig. 14D). Probing of the medial meniscus demonstrated subluxation of torn fragments of the medial meniscus, which were freed with the probe and debrided (Fig. 14E, F). Probing of the tibial surface and meniscus demon1strates Outerbridge grade IV lesion under the stable rim of the medial meniscus (Fig. 14G). After arthroscopy, the patient had complete relief of mechanical symptoms and dramatic improvement of her pain and remains satisfied 2 years after the procedure.

Fig. 14 Case 1. (**A**) Anteroposterior radiographs of the knees demonstrate mild medial compartment narrowing of both knees, with minimal degenerative changes. (**B, C**) MRI scans shows horizontal cleavage tear of the medial meniscus associated with articular cartilage degeneration and reactive bone edema. (**D**) Initial arthroscopic view of the medial compartment shows Outerbridge grade III fibrillation of articular cartilage of the femur and tibia. Torn meniscus is initially not apparent. (**E**) Probing of articular side of the medial meniscus reveals subluxated flap of torn meniscus. (**F, G**) Resection of unstable flaps of the medial meniscus back to stable rim reveals Outerbridge grade IV changes on the tibial plateau

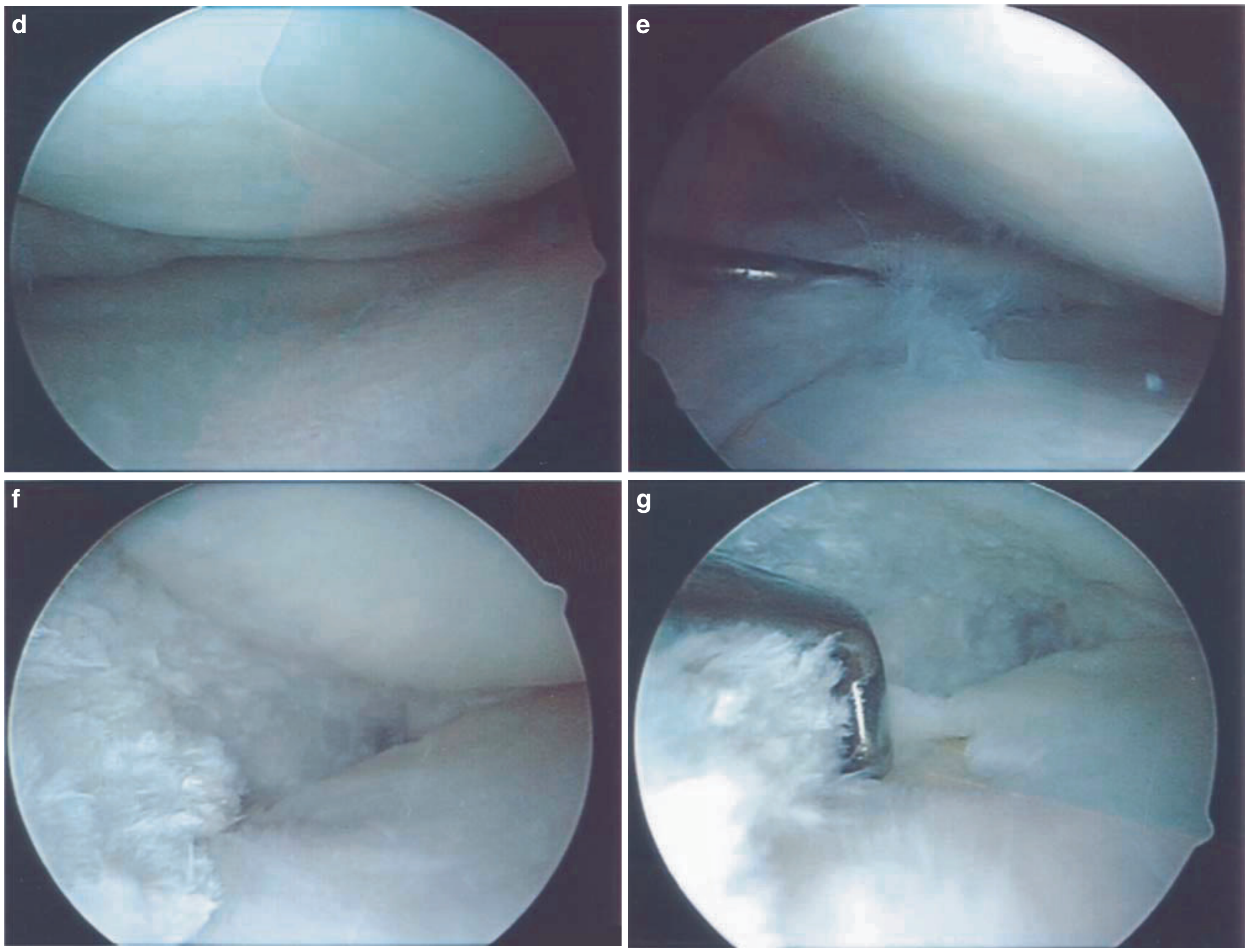


Fig. 14 (continued)

References

1. CDC. Arthritis prevalence and activity limitations – United States 1990. *MMWR Morb Mortal Wkly Rep* 1994;43:433–438.
2. From the Centers for Disease Control and Prevention. Arthritis prevalence and activity limitations – United States 1990. *JAMA* 1994;272:346–347.
3. Pate RR, Pratt M, Blair SH, Haskell WL, Macera CA, Bouchard C, et al. Physical activity and public health: a recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *JAMA* 1995;273:402–407.
4. Vazquez-Vela Johnson G, Worland RL, Keenan J, Norambuena N. Patient demographics as a predictor of the ten-year survival rate in primary total knee replacement. *J Bone Joint Surg Br* 2003;84: 52–56.
5. Gioe TJ, Novak C, Sinner P, Ma W, Mehle S. Knee arthroplasty in the young patient: survival in a community registry. *Clin Orthop* 2007;464:83–87.
6. Casscells SW. What, if any, are the indications for arthroscopic debridement of the osteoarthritic knee? *Arthroscopy* 1990;6: 169–170.
7. Ogilvie-Harris DJ, Fitsialos DP. Arthroscopic management of the degenerative knee. *Arthroscopy* 1991;7:151–157.
8. Rand JA. Role of arthroscopy in osteoarthritis of the knee. *Arthroscopy* 1991;7:358–363.
9. Aaron RK, Skolnick AH, Reinert SE, McK. Ciombor D. Arthroscopic debridement for osteoarthritis of the knee. *J Bone Joint Surg Am* 2006;88:936–943.
10. Iorio R, Healy WL. Unicompartmental arthritis of the knee. *J Bone Joint Surg Am* 2003;85:1351–1364.
11. Jackson RW, Abe I. The role of arthroscopy in the management of disorders of the knee. An analysis of 200 consecutive cases. *J Bone Joint Surg Am* 1972;54:310–322.
12. Dervin GF, Stiell IG, Rody K, Grabowski J. Effect of arthroscopic debridement for osteoarthritis of the knee on health-related quality of life. *J Bone Joint Surg Am*2003;85:10–19.
13. Edelson R, Burks RT, Bloebaum RD. Short-term effects of knee washout for osteoarthritis. *Am J Sports Med* 1995;23: 345–349.
14. Salisbury RB, Nottage WM, Gardner V. The effect of alignment on results in arthroscopic debridement of the degenerative knee. *Clin Orthop* 1985;198:268–272.

15. Bircher E. Die Arthroendoskopie. *Zentralbl Chir* 1921;48: 1460–1461.
16. Burman MS, Finkelstein H, Mayer L. Arthroscopy of the knee joint. *J Bone Joint Surg* 1934;16:25.
17. Watanabe M, Takeda S, Ikeuchi H. *Atlas of Arthroscopy.* Tokyo: Igaku Shoin, 1957.
18. Lysholm J, Hamberg P, Gilquist J. The correlation between osteoarthritis as seen on radiographs and arthroscopy. *Arthroscopy* 1987;3:161–168.
19. Jackson RW. The role of arthroscopy in the management of disorders of the knee. *Clin Orthop* 1974;101:28–35.
20. Moseley JB, O'Malley K, Petersen NJ, Menke TJ, Brody BA, Kuykendall DH, et al. A controlled trial of arthroscopic surgery for osteoarthritis of the knee. *N Engl J Med* 2002;327:81–88.
21. Wai EK, Kreder HJ, Williams JI. Arthroscopic debridement of the knee for osteoarthritis in patients fifty years of age or older, utilization and outcomes in the province of Ontario. *J Bone Joint Surg Am* 2002;84:17–22.
22. Fife RS, Brandt KD, Braunstein EM, Katz BP, Shelbourne KD, Kalasinski LA, Ryan S. Relationship between arthroscopic evidence of cartilage damage and radiographic evidence of joint space narrowing in early osteoarthritis of the knee. *Arthritis Rheum* 1991;34:377–382.
23. Brandt KD, Fife RS, Braunstein EM, Katz B. Radiographic grading of the severity of knee osteoarthritis. *Arthritis Rheum* 1991;34:1381–1386.
24. Sprague NF III. Arthroscopic debridement for degenerative knee joint disease. *Clin Orthop* 1981;160:118–123.
25. Bert JM, Maschka K. The arthroscopic treatment of unicompartmental gonarthrosis: a five-year follow-up study of abrasion arthroplasty plus arthroscopic debridement and arthroscopic debridement alone. *Arthroscopy* 1989;5:25–32.
26. Harwin SF. Arthroscopic debridement for osteoarthritis of the knee: predictors of patient satisfaction. *Arthroscopy* 1999;15: 142–146.
27. Jackson RW, Rouse DW. The results of partial arthroscopic menisectomy in patients over 40 years of age. *J Bone Joint Surg Br* 1982;64:481–485.
28. Pearse EO, Craig DM. Partial menisectomy in the presence of severe osteoarthritis does not hasten the symptomatic progression of osteoarthritis. *Arthroscopy*2003;19: 963–968.
29. Bhattacharyya T, Gale D, Dewire P, Totterman S, Gale ME, McLaughlin S, Einhorn TA, Felson DT. The clinical importance of meniscal tears demonstrated by magnetic resonance imaging in osteoarthritis of the knee. *J Bone Joint Surg Am* 2003;85:4–9.
30. Fond J, Rodin D, Ahmad S, Nirschl RP. Arthroscopic debridement for the treatment of osteoarthritis of the knee: 2- and 5-year results. *Arthroscopy*2002;18:829–834.
31. Shen M, Meislin R. Technical note: a new method to utilize a motorized shaver in hip arthroscopy without use of a cannula. *Arthroscopy* 2005;21:895.e1–895.e3.
32. DeLee JC. Complications of arthroscopy and arthroscopic surgery: results of a national survey. *Arthroscopy*1985;1:214–220.
33. Pape D, Seil R, Anagnostakos K, Kohn D. Postarthroscopic osteonecrosis of the knee. *Arthroscopy* 2007;23:428–438.
34. Ramos J, Perrotta C, Badariotti G, Berenstein G. Interventions for preventin venous thromboembolism in adults undergoing knee arthrosocopy. *Cochrane Database Syst Rev* 2007;18(2):CD005259.

Current Concepts in Articular Cartilage

Brian P. McKeon

Introduction

The treatment of articular cartilage (AC) lesions remains controversial. Studies have documented a significant incidence of these defects [1–3]; however, progression of chondral defects and necessity of treatment are widely debated. Certainly, symptomatic, full-thickness AC lesions are challenging to treat. Additionally, there is very little evidence-based medicine that argues for one treatment method over another. However, over the past decade, there has been a tremendous surge of interest in AC. The International Cartilage Repair Society (ICRS) began in 1997 and has provided a forum to allow clinicians and basic scientists to share expertise and develop ground-breaking concepts. In the past decade, our basic science knowledge has improved exponentially, thus laying the foundation for strategic clinical concepts. With the addition of biological scaffolds and gene therapy techniques, the future holds many promises for patients with a symptomatic AC defect.

Since Hunter realized more than 250 years ago that AC is a “troublesome thing and once destroyed, it is not repaired” [4], surgeons have sought for methods to restore or regenerate AC, more properly termed hyaline AC. It is well established that AC has no blood supply and thus cannot “heal” itself. This very simple and basic principle is the sole reason that true hyaline AC has not been replicated. Hunter was correct: once AC is lost, it is gone forever. Partial-thickness chondral defects are generally not symptomatic [5]; however, there is concern for lesion propagation. Full-thickness chondral defects, however, can be symptomatic, and there is evidence that these defects do further degenerate [6, 7]. The purpose of this chapter is to review the basic science of AC and to discuss various treatment options for chondral defects in the knee.

Basic Science

Matrix Composition

Hyaline AC is described as a biphasic material composed of a solid matrix and a water fluid phase. The major structural components of AC are water, collagen, proteoglycan, and chondrocytes. AC has no vascular, neural, or lymph supply and, hence, the inability to mount an immune or vascular response. Water constitutes approximately 70% to 80% of the total volume of AC. Collagen accounts for 10% to 20% of the total weight of AC, and type II collagen is the principle form in the matrix. In fact, the presence of type II collagen distinguishes hyaline AC from the various repair tissues that contain mostly type I collagen. Collagen fibrils are the predominant matrix component that resists tensile forces and forms a large mesh that serves to secure the large proteoglycan aggregate molecules (Fig. 1) [8].

Proteoglycans account for 4% to 7% of the total weight of AC. Aggrecan is the principle proteoglycan, which is composed of hydrophilic carboxyl and sulfate chains that project off a protein core. The protein core is attached to a hyaluronic acid chain that forms the “backbone” of the huge aggrecan molecule (Fig. 2). This construct produces a matrix-fixed negative charge that attracts the positive charge of water. The high proteoglycan affinity for water trapped in the mesh of collagen produces a swollen tissue that can support significant loads [9].

The only cell type in AC is the chondrocyte, which accounts for less than 3% of the total volume of AC. The chondrocyte is responsible for matrix production and functions differently depending on what zone it is located in [10]. Because of the dense matrix, there is little cell-to-cell contact; however, the flow of synovial fluid throughout

B.P. McKeon (✉)
Department of Orthopaedics, Tufts University School of Medicine, New England Baptist Hospital, Boston, Massachusetts; Boston Sports and Shoulder Center, Chestnut Hill Massachusetts

B.P. McKeon et al. (eds.), *Knee Arthroscopy*, DOI 10.1007/978-0-387-89504-8_6,

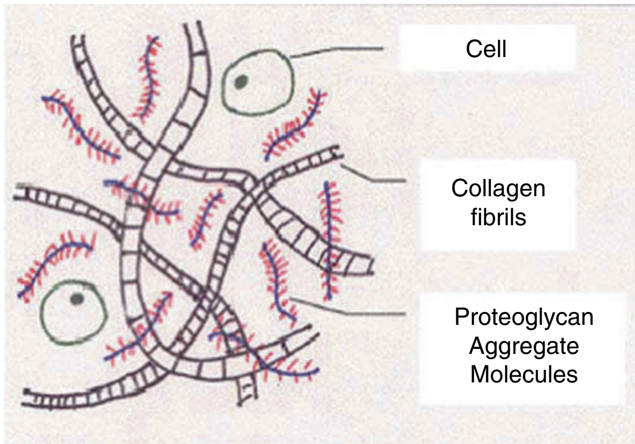


Fig. 1 Basic cartilage meshwork configuration

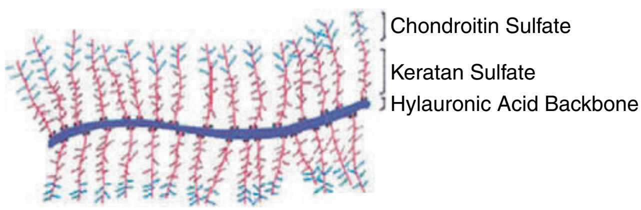


Fig. 2 The huge aggrecan molecule that has abundant negatively charged sulfate molecules that attract positively charged water molecules. This affinity for water combined with a tight meshwork provides the "charged" hyperhydrated sponge described in the text

the matrix in response to joint load provides a chemical and mechanical environment to which the chondrocyte responds. In fact, chondrocyte survival depends upon this mechanoelectrical matrix environment, which stimulates growth factor production, paracrine effects, and electrostatic forces [11–14]. Hyaline AC cannot survive without viable chondrocytes.

AC Structure

Adult AC is 2–4 mm in thickness and is organized into three zonal layers supported by a fourth calcified basement membrane layer (Fig. 3). The superficial, transitional, and deep zones feature unique chemical and biomechanical features that allow AC to sustain 1–4 megapascals (MPa) of force up to 2 million times per year [14].

The superficial zone is covered by the lamina splendens, which is composed of a thin surface of collagen fibers that help to protect the deeper zones from shear forces. The chondrocytes are flattened with high collagen and water content in this zone, whereas the proteoglycan content is low. The superficial shear forces are encountered by the transitional or middle zone, which prepares the deeper zone for compressive forces. The chondrocytes are spherical in appearance, and the collagen is obliquely oriented in a crossing pattern. There is less water but greater proteoglycan in this layer. The deep zone consists of round chondrocytes in columns supported by vertical collagen bundles designed to resist the compressive loads present in the deeper layers of AC. Lastly, the calcified zone acts as a biological glue, attaching the collagen-rich deep zone to the subchondral bone. The tidemark is between the deep zone and the calcified layer and is designed to support deep shear forces with collagen fibers arranged parallel to the AC surface.

Biomechanics

AC exhibits a viscoelastic property that allows the tissue to respond to stress based on the rate of loading. For instance, while walking, AC responds in a more viscous manner; however, while running, AC can respond in a more protective elastic manner. The cartilage matrix is hyperhydrated and essentially acts like a mechanical sponge that allows for absorbing tremendous compressive forces [14]. The huge proteoglycan macromolecules confer a high fixed-negative charge, and, therefore, AC has a very high affinity for water [15]. The collagen fibrils secure the large proteoglycan molecules in a tightly woven arcade that then "traps" water. This intact macromolecular arrangement with high affinity for water is why intact AC can sustain tremendous compressive loads. The fixed-negative charge not only affects the mechanical behavior of AC but also the electrical environment by "charging" the tissue. A charged tissue always supports a larger load than does the same uncharged tissue [16]. Hence, the dynamic compressive loading of AC generates a mechanoelectrical environment that influences cartilage biosynthesis and repair and modulates chondrocyte activity [17]. This knowledge allows the clinician to understand the significant biomechanical issues with partial-thickness AC lesions and to consider a breakdown in the collagen/proteoglycan matrix interaction and, hence, the loss of ability to contain water effectively. This structural loss of integrity is likely the reason for further deterioration and lesion propagation.

Incidence/Natural History

In a retrospective review of 31,516 arthroscopies, Curl et al. found that 19,827 (63%) had a chondral defect [18].

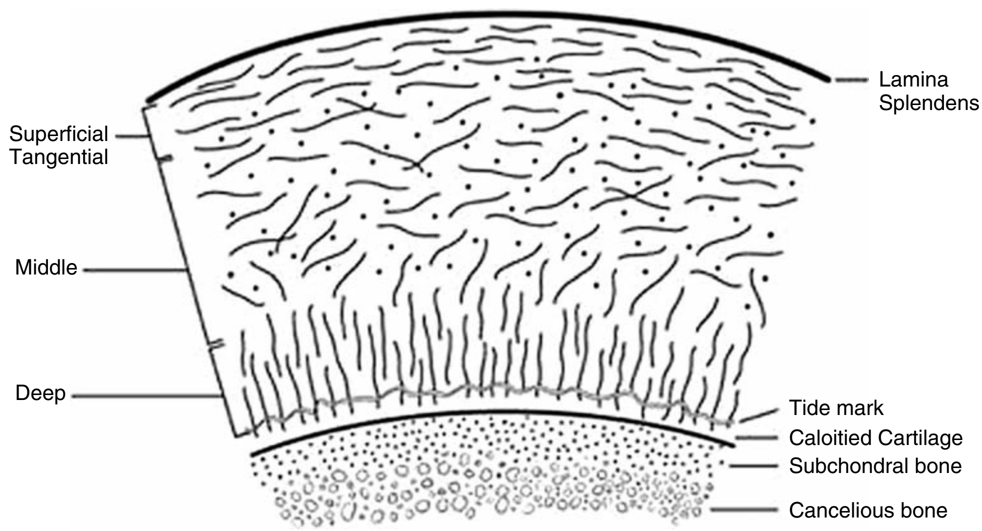


Fig. 3 The zonal layers of articular cartilage. (From Alford JW, Cole BJ. Cartilage restoration. *Am J Sports Med* 2005;33:295. Copyright © 2005 American Orthopaedic Society for Sports Medicine. Reprinted by permission of SAGE Publications, Inc.)

Full-thickness articular lesions were noted in 5,988 (19%) patients. Aroen et al. reported 11% full-thickness defects in a review of 993 arthroscopies [19]. Villalobos et al. documented a 61.4% prevalence of articular damage in a review of 1,309 arthroscopies. The mean age of patients was 37.3 years, and 73.8% of the lesions presented as ICRS grade 3 or 4 [3].

Although AC lesions are quite prevalent, the documentation of lesion progression is limited. Moreover, there is no evidence to support treating asymptomatic lesions, regardless of the size. However, Sahlstrom et al. [20] documented 100% progression of radiographic osteoarthritis once Ahlback stage II (>50% joint space narrowing) was reached. Earlier stages, including Ahlback 0 (sclerosis and preserved joint space) and Ahlback stage I (50% joint space narrowing), showed 57% and 61% progression, respectively.

Messner and Maletius [21] followed 28 patients with isolated unicompartmental lesions after surgical debridement for 14 years. Interestingly, despite most patients demonstrating progressive radiographic abnormalities, 22 of the 28 had good or excellent results at long-term follow-up. In a 33-year follow-up of untreated knee osteochondritis dissecans, Linden reported that only 2 of 23 (9%) patients with open growth plates at the time of diagnosis had radiographic progression, whereas 81% of mature patients revealed progression at last follow-up [22]. It appears that the larger the defect and the more advanced the degenerative changes are, the greater the likelihood of progression [23, 24]. Additionally, ligamentous or meniscal injury can accelerate and magnify the progression of chondral damage [25, 26].

Classification of AC Lesions

Thorough documentation and grading of chondral lesions are necessary for treating patients with AC defects. The simplest scale was described by Outerbridge [27] in 1960 from direct observation of damaged patellas during arthrotomy (Fig. 4A–D). Although widely accepted, the Outerbridge grading system has size, depth, and lesion locale descriptive limitations. Other classification systems have been proposed [28, 29]; however, the ICRS grading system recognizes the importance of subchondral osseous involvement and is the most comprehensive (Fig. 5) [30].

Imaging

Standard radiographs including weight-bearing long alignment films are essential when evaluating a patient with a symptomatic chondral defect. Current magnetic resonance imaging (MRI) techniques have a high sensitivity for cruciate and meniscal injury; however, there are limitations evaluating AC. Partial-thickness lesions are particularly difficult to detect [31–33]. Potter et al. [33] reported an 87% sensitivity and a 94% specificity in detection of AC chondral defects using a specialized proton-density-weighted, fast-spin-echo sequence. These findings were confirmed by arthroscopy; however, the surgeons had access to the MRI reports at the time of surgery. Table 1 depicts the ICRS-recommended MRI protocols, which emphasize fast-spin-echo sequences [33].

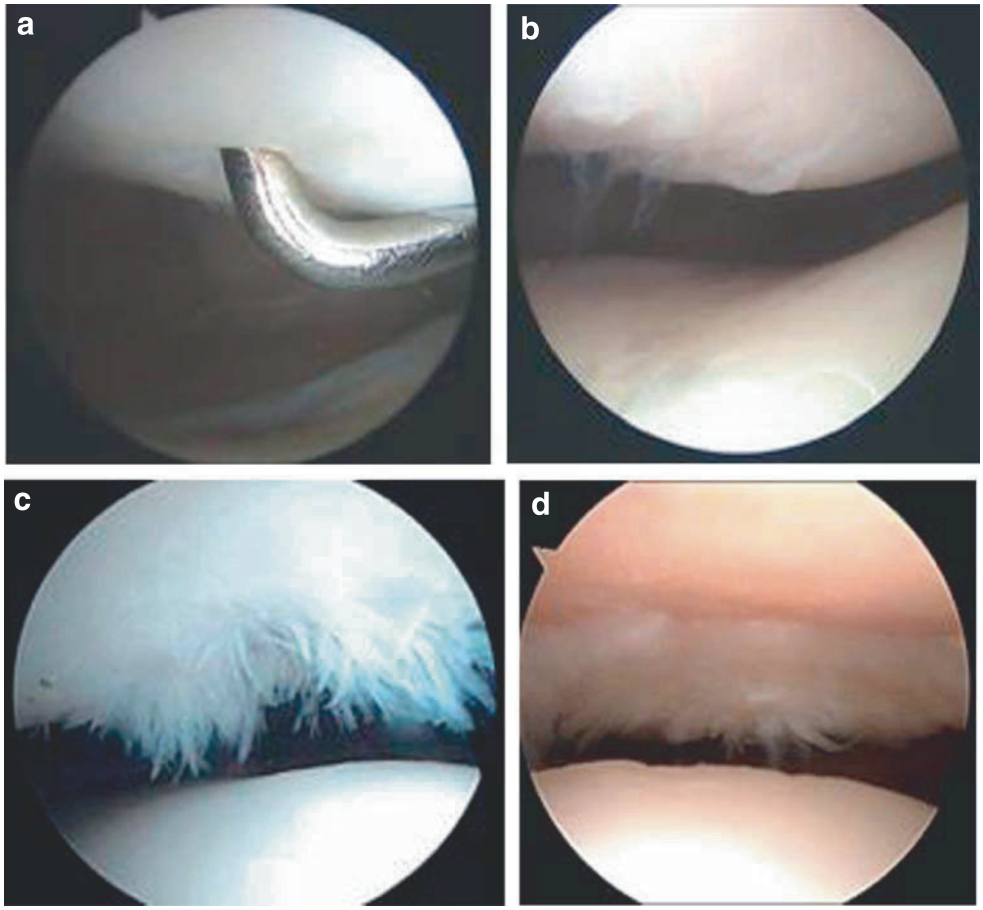


Fig. 4 Arthroscopic representation of the Outerbridge classification defined by four grades: (**A**) grade I, intact soft cartilage; (**B**) grade II, partial-thickness lesion <1.5 cm; (**C**) grade III, partial-thickness lesion >1.5 cm; (**D**) grade IV, exposed bone

Conventional MRI is insensitive in detection of early chondral changes [31–34]. The increase in water and loss of matrix equilibrium, for instance, are not necessarily detected. The concept of molecular imaging in which a contrast agent diffuses into the AC and allows for calculation of the fixed-charge density (by calculating macromolecular quantity) [35] has gained increased interest. This particular technique, called delayed gadolinium-enhanced magnetic resonance imaging of articular cartilage (dGEMRIC), is a validated technique to calculate the quantity of AC proteoglycan content and, hence, fixed-charge density. This technique may be particularly useful in evaluating and following various cartilage repair techniques, efficacy of therapeutic approaches such as chondrosupplements, and the impact of exercise and injury [36, 37]. For instance, serial evaluations of autologous chondrocyte transplanted defects demonstrated increased and normalized proteoglycan content and, hence, one can infer a functioning tissue-engineered chondrocyte [38].

Patient Evaluation with Symptomatic AC Defect

Initial consultation of a patient with a suspected or confirmed AC defect is the most important portion of the treatment cycle. Inquiring about the "chief complaint" and completing a thorough history and physical can provide subtle information such as true time frame of symptoms, a forgotten injury, or even an old radiograph that was taken years prior. Initial evaluation of a workers' compensation patient can be difficult, particularly assessing appropriate causality to the work injury. Focusing on the clinical presentation and allowing the patient to describe his or her complaints can give the clinician a real sense of the patient's disability. All prior surgeries should be discussed, and review of old records including operative reports, photographs, and even video clips is invaluable. Often, a simple arthroscopic photograph can summarize years of pain and multiple surgeries (Fig. 6).

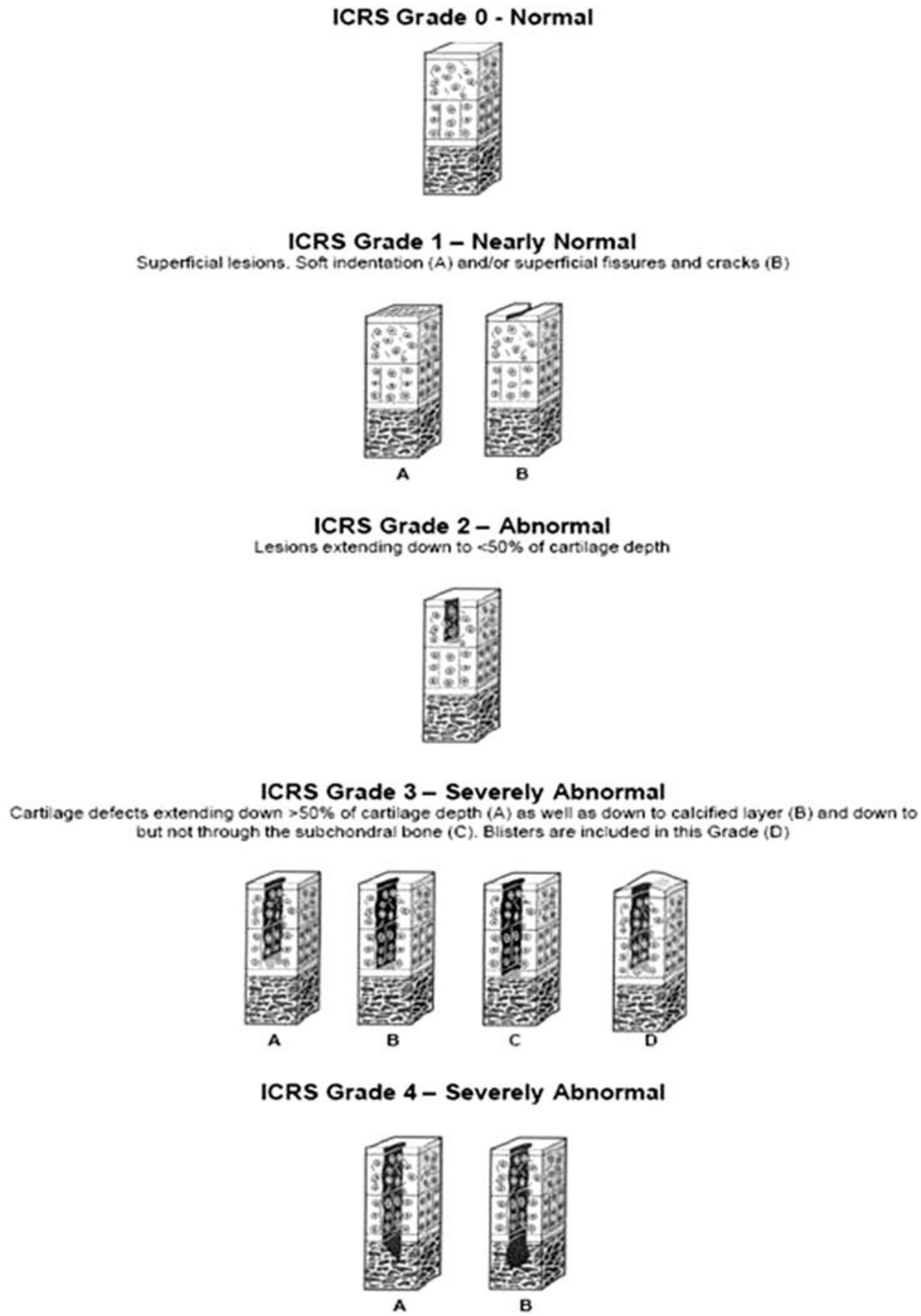


Fig. 5 ICRS grading system for chondral defects. (Reprinted with kind authorization of the International Cartilage Repair Society [ICRS]. Copyright © ICRS.)

The physical can often be quite normal and may only be related to weight-bearing activity. Examination for effusion, muscular atrophy, multiplanar stability, crepitus, alignment, and precise region of pain is mandatory. Standard radiographs including a weight-bearing posterior-anterior view at 45 degrees can be helpful to evaluate the region where condylar defects most commonly occur [39]. Hip-knee-ankle weight-bearing radiographs followed by precise measurement of the mechanical axis must be performed if alignment is a concern on initial evaluation. MRI with cartilage specific sequences [40] is necessary, and these scans should be compared with old images if possible. MRI can provide location of defect(s) and extent of bone involvement. Regardless of the AC restorative procedure used, it simply will not endure without intact ligaments and supporting meniscus (Fig. 7). Cruciate and collateral ligament reconstructive

Table 1 ICRS recommended MRI protocol for articular cartilage

Fast Spin Echo (FSE)
Proton-density-weighted FSE with fat saturation
TR: 3,000 (adjust to number of slices)
TE: 22–30 eff
ETL: 5–8
FOV: 14 cm
Slice thickness/gap: 3.5/0.5 mm
Matrix: 512 × 256
NEX: 2
Frequency direction: S/I coronal, A/P sagittal, A/P axial
Proton-density-weighted FSE without fat saturation
TR: 4,000
TE: 43
ETL: 7
FOV: 14 cm
Slice thickness/gap: 3/0 mm
Matrix: 512 × 256
NEX: 2
Frequency direction: S/I coronal, A/P sagittal, A/P axial
T2-weighted FSE with or without fat suppression
TR: 4,500
TE: 80
ETL: 8
FOV: 8 cm
Slice thickness/gap: 3.0/0 mm
Matrix 512 × 256
NEX: 2
Frequency direction: S/I coronal, A/P sagittal, A/P axial
T1-Weighted Gradient Echo
Gradient echo with fat suppression
TR:40–60
TE: 5
Flip angle: 40
FOV: 14 cm
Matrix: 256 × 160
Slab: 96 cm
Partitions: 64
NEX: 1
Gradient echo with water excitation
TR: 30
TE: 10
Flip angle: 30
FOV: 14
Slab: 96 cm
Matrix: 256 × 160
NEX: 1
Partitions: 64

Source: Reprinted with permission from the International Cartilage Repair Society (ICRS).
TR, repetition time; TE, echo time; ETL, echo train length; FOV, field of view; NEX, number of excitations eff, effective; S/I, superior inferior; A/P, anterior posterior.

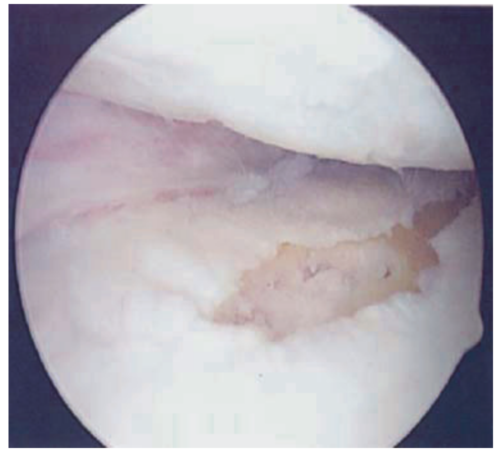


Fig. 6 Patient presented to the author's clinic complaining of an unstable, painful knee. Exam and photos from a recent surgery confirmed a varus anterior cruciate ligament (ACL)-deficient knee with no medial meniscus. Patient was postoperative microfracture with immediate WB. Instability, mechanical alignment, absent meniscus, and rehabilitation are important factors that must be addressed for successful AC resurfacing

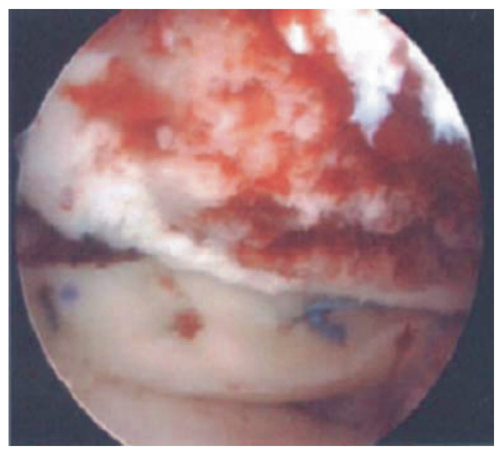


Fig. 7 This patient presented to the author's clinic status postarthroscopy with subtotal medial meniscectomy and microfracture of a large femoral chondral defect. The patient underwent a revision microfracture of a large defect combined with meniscal allograft transplant. The repair tissue has a much greater opportunity to mature with meniscal support

surgeries are relatively straightforward. Meniscal transplantation can be chondroprotective, and improved techniques have simplified this procedure [41]. Realignment surgery can be staged or be performed at the time of AC resurfacing. In general, AC-resurfacing patients do not have arthritic knees; hence, no exact guidelines have been agreed upon for ideal femoral-tibial postoperative alignment. However, most agree

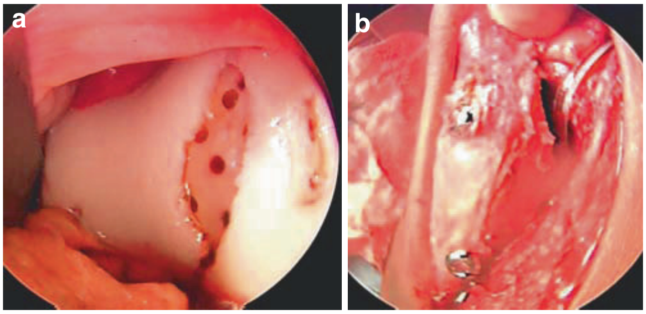


Fig. 8 (**A, B**) Anteromedialization osteotomy of the tibial tubercle will unload a lateral trochlear defect, which, in this case, was treated with microfracture

that any significant malalignment should be treated with an unloading osteotomy [42–44]. The author believes that the postoperative mechanical axis should measure at least through the opposite joint downsloping tibial spine.Patella-femoral malalignment should be corrected with a tibial tubercle osteotomy to redistribute forces across the patellofemoral joint as well as to unload resurfaced patella or trochlear chondral defects (Fig. 8A, B) [45–47].

Cartilage Restoration Treatment Options

Arthroscopic Lavage and Debridement

More than 60 years ago, Magnusson [48] described the benefits of knee joint debridement to alleviate arthritic symptoms. With the advent of arthroscopy, Jackson et al. [49] became a proponent of arthroscopic debridement for the treatment of a symptomatic arthritic knee. The concept entails removing impinging osteophytes, inflamed synovium, and loose chondral debris. Hubbard [50] reported a 5-year prospective follow-up on 76 knees treated with arthroscopic debridement. Two groups were randomized and treated with either debridement or lavage. At 1 year, the debridement arm showed 80% pain-free compared with the lavage arm with 20% relief. More importantly, at 5 years the results deteriorated. Other clinical trials have shown both debridement [51, 52] and lavage [53] result in significant improvements in knee symptoms when compared with results in nonoperative controls. However, no long-term benefit can be expected from arthroscopic debridement and lavage, specifically when there are no localized mechanical symptoms [54]. A painful arthritic knee with a clear history of mechanical complaints including locking or catching and persistent synovial effusions has the indications for arthroscopic debridement, chondroplasty, and synovectomy of the arthritic knee.

Surgical Technique

Standard arthroscopic setup and technique are used (see Chapter 1). Marcaine (Bupivacaine HCL, AstraZeneca (London, UK)) with epinephrine is injected prior to tourniquet elevation. The arthritic knee is often stiff,

and, therefore, local anesthesia with sedation is not recommended. Maximum relaxation with general or spinal anesthesia allows for more control and easier access. Gravity or more preferably a pump is used for inflow established through an initial anterolateral portal. A large superolateral or superomedial outflow is helpful for irrigation of debris. Liberal use of accessory portals allows access to tight areas and removal of loose bodies. A variety of shavers, burrs, and radiofrequency (RF) wands is available to ensure access to all compartments.

All three compartments are systematically reviewed and treated. Unstable chondral flaps and impinging synovium are addressed with a shaver. RF treatment of articular cartilage is not recommended. Only osteophytes that contribute to loss of motion or impingement of soft tissue should be resected. Excessive removal of osteophytes and diffuse abrasion arthroplasty can contribute to postoperative hematoma and a painful, prolonged recovery. Unstable degenerative meniscus tears should be resected conservatively. Meniscal tissue preservation is of utmost importance while treating the arthritic knee. The knee is irrigated and reinjected with Marcaine with epinephrine, and portals are closed. A compressive wrap is applied and tourniquet is deflated.

Postoperative Care/Rehabilitation

Initially, partial weight-bearing (PWB) is recommended with progression to full weight-bearing (FWB) as tolerated. Aggressive cryotherapy and supervised physical therapy are initiated usually by postoperative day 3. Swelling reduction, muscle activation, and gait training are the initial goals, followed by progressive resistive exercises. Overzealous rehabilitation can precipitate a significant postoperative flare in this patient population. Therefore, communication with the rehabilitation team is critical. Providing surgical pictures, a video, and an operative report allows the therapist to structure a specific program for the patient. Occasionally a postoperative cortisone injection can help a persistent synovitis.

Case Report

Case 1: Lavage/Debridement

Chief Complaint and Patient History: The patient is a 45-year-old man who is an office worker and who presents for medial pain and swelling of his left knee. He underwent arthroscopy 8 years prior for a “cartilage” problem. The joint occasionally swells, especially with athletics. Over the past several months, his pain has increased, affecting activities of daily living. He received a cortisone injection from his primary-care physician 3 months ago with little relief.

Physical Exam: The patient is 6′0″ tall and weighs 250 lb. There is minor crepitus with passive flexion and extension and a small effusion. He has normal alignment and range of motion. Ligaments are stable, and he has equivocal meniscal signs. Deep anteromedial discomfort is evident with palpation of the hyperflexed knee.

Imaging: See Fig. 9A, B.

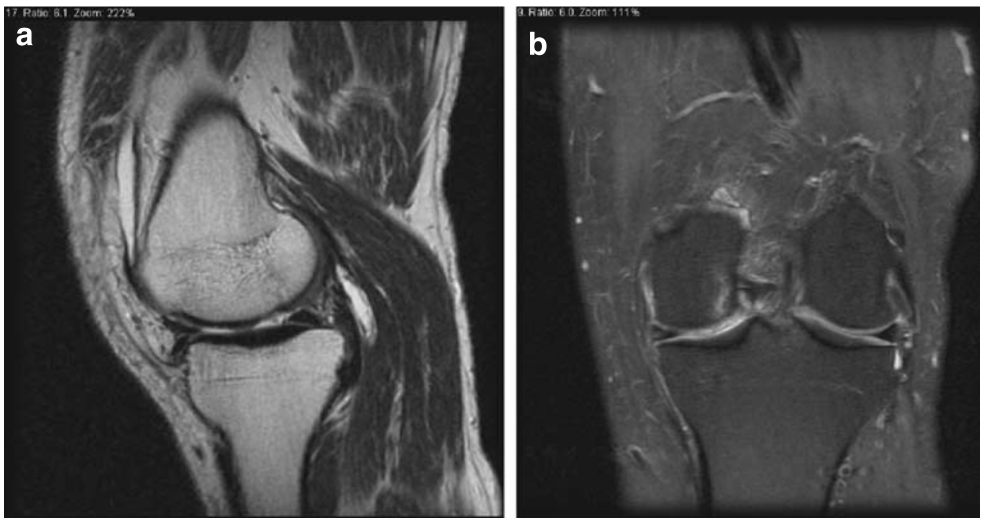


Fig. 9 (**A, B**) MRI scans demonstrates cartilage defect of medial femoral condyle (MFC) with associated bony edema. A small medial meniscal tear may be present

Surgery/Treatment: The patient underwent arthroscopic partial meniscectomy, with chondroplasty of chondral ICRS grade 3C defect (Fig. 10A, B).

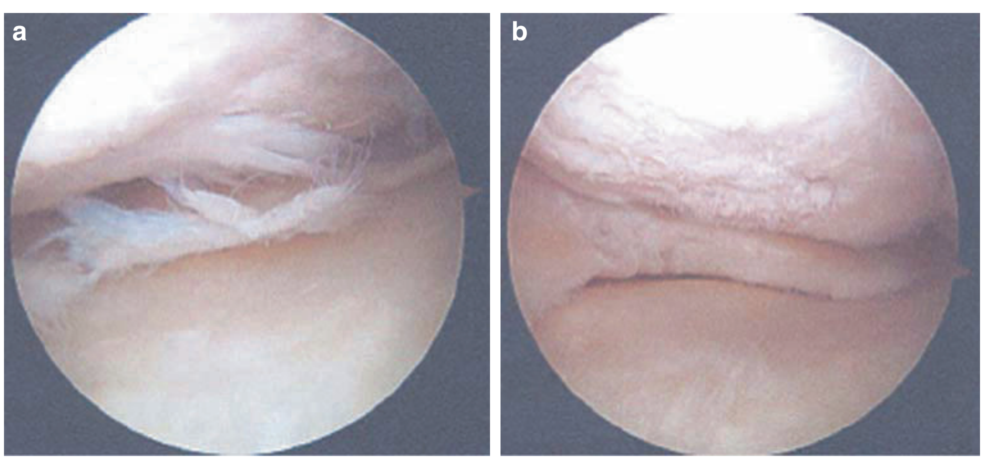


Fig. 10 (**A, B**) Arthroscopic views demonstrate cartilage wear of medial femoral condyle. A chondroplasty was performed with partial medial meniscectomy

Postoperative Care/Follow-Up: The patient progressed well with physical therapy. He returned to work on postoperative day 3 to a desk position. Remaining postoperative care was relatively benign; however, patient continued to complain of medial pain with impact activities. Weight loss and strengthening was recommended. Patient was well versed in the surgical treatment of cartilage injuries. We discussed arthroscopic microfracture and associated rehabilitation. The patient found the rehabilitation for this procedure to be too demanding and requested not to have this procedure.

Marrow Stimulation

The concept of penetrating the subchondral bone to allow for release of blood, growth factors, and mesenchymal cells into the chondral defect was popularized by Pirdie more than 50 years ago [55]. The "super clot" generated consists of a mixed fibrous repair with type I collagen that matures over a 12- to 16-month time frame [56]. This so-called repair cartilage does not have the structural, physiochemical, or biochemical properties of hyaline AC with a type II collagen framework (Fig. 11A, B) [57, 58]. Abrasion arthroplasty, subchondral drilling, and microfracture are the three described techniques used to penetrate the subchondral bone. Abrasion arthroplasty uses a motorized burr to remove 1–3 mm of subchondral bone. Excessive trauma to the underlying bone and thermal necrosis can potentially be more destructive than helpful [59]. Resecting a consistent amount of bone can be challenging, and the author does not recommend this technique. A motorized smooth wire is used to perform subchondral drilling. Because of poor access and thermal necrosis concerns, drilling is no longer recommended as well. The microfracture technique developed by Steadman et al. [60] is the current preferred marrow-stimulation method. Arthroscopic tapered awls perforate the subchondral bone (Fig. 12).

Steadman et al. reported microfracture outcomes with an average 11-year follow-up [61]. Eight percent of patients reported continued improvement at 7 years. Patients younger than 35 years had improved Tenger, Western Ontario and McMaster Universities Osteoarthritis Index, Lysholm, and Short Form-36 scores compared with those of the older cohorts in this series. Mithoefer et al. [62] recently reported a prospective evaluation of 32 athletes treated with microfracture. The mean age was 38 years, and the patients had regularly participated in high-impact, pivoting sports prior to articular cartilage injury. At 2-year minimum follow-up, 21 (66%) patients reported good to excellent results on the Brittberg scale. They concluded that lesions under 200 mm^2, preoperative symptoms of less than 1 year, and an age of under 40 years are the ideal indications for the microfracture technique. Kreuz et al. [63] reported similar conclusions in a recent series and concluded that results of microfracture are age-dependent. In this prospective evaluation of 85 patients, the ICRS score significantly deteriorated in patients >40 years of age by between 18 and 36 months. Based on the literature and the author's own personal experience, the current indications for microfracture are younger active patients with smaller (<2 cm^2) chondral defects.

Surgical Technique

Microfracture is very technique-dependent, and the success is dependent on the amount of fibrocartilaginous fill of the chondral defect. Under tourniquet control, a standard diagnostic arthroscopy is performed. All articular surfaces are inspected and probed after other knee pathology is addressed, including meniscal and ligament procedures. Defining the extent of the lesion and debriding to a stable border is the most important step in the technique. A ring curette (Fig. 13) is an excellent instrument with which to remove unstable flaps and sculpt vertical walls on the border of the lesion. Next, it is critical to remove the calcified cartilage layer at the base of the lesion. It has been shown in animal models that, with removal of the calcified layer, the percentage of defect fill significantly increases (Fig. 14) [64]. This layer consists of a heavy deposition of apatites and normally serves as a barrier to cellular migration and nutrient flow [65]. Therefore, it is theorized that the "superclot" will adhere to the base of the defect more predictably after removal of this barrier.

Next, the microfracture awls are used to penetrate the subchondral bone starting at the periphery (Fig. 15). The holes should be spaced at least 2- to 3-mm apart to avoid connecting them. Loose debris is treated with a motorized shaver, and the joint is thoroughly irrigated. The tourniquet is deflated, and pump pressure is turned down to confirm subchondral bleeding (Fig. 16). The knee is injected with plain Marcaine, and wounds are closed followed by application of a compressive wrap and hinged brace.

Postoperative Care/Rehabilitation

Compliance with a structured rehabilitation program is just as important as the surgery itself. A preoperative discussion is wise, if not mandatory, regarding the postoperative plan with the patient. This is particularly important when possibly treating occult lesions. A dedicated time frame of protected

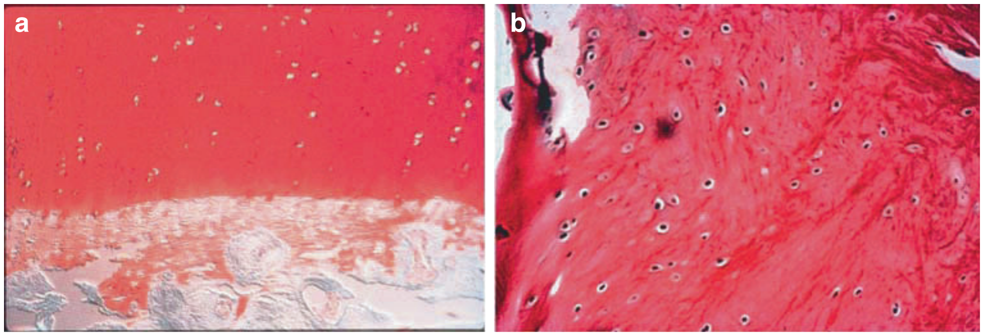


Fig. 11 The differences in cellular population and lack of zonal organization are apparent in this histologic comparison of (**A**) normal hyaline cartilage versus (**B**) fibrous-based cartilage

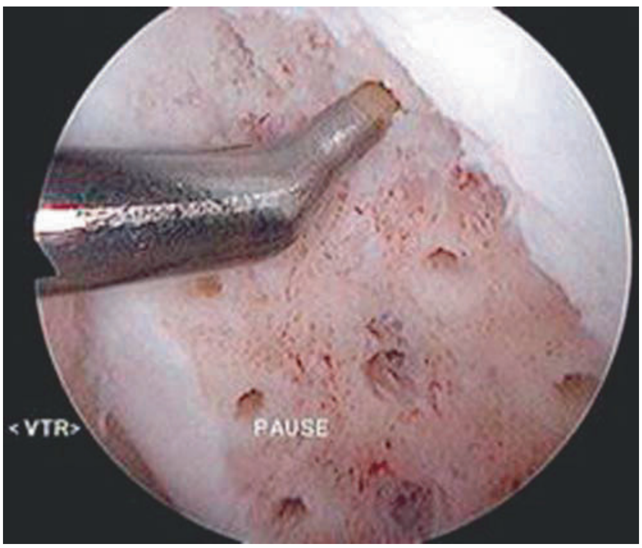


Fig. 12 An arthroscopic awl is used to perforate the subchondral bone during a microfracture procedure

Fig. 13 A ring curette is used to remove unstable flaps and establish stable borders

weight-bearing combined with continuous passive motion (CPM) demands a compliant patient.

In general, the patient remains touch-down weight-bearing (TDWB) for 6 weeks. For uncontained defects, 8 weeks is recommended. However, for trochlear, patella, and very posterior condylar lesions, a patient can be FWB with the extremity locked in extension for ambulation. If the lesion is not loaded while weight-bearing, then allowing the patient to fully weight-bear to avoid atrophy is very advantageous for the entire muscular sleeve and skeleton.

The use of CPM has been shown to improve the quality of repair tissue after microfracture [66, 67]. CPM provides nutrition and accelerates cellular differentiation of the marrow mesenchymal cells into matrix-producing fibrochondrocytes [68]. Essentially, the better philosophy is to recommend a minimum of at least 8 hours per day. Separating this into shorter intervals can be helpful for compliance, or, alternatively, this can take place in the evening or at night for patients who work or attend school.

Fig. 14 Calcified cartilage layer (white tissue covering subchondral plate) must be removed prior to microfracture

Fig. 15 A completed microfractured lesion demonstrates multiple perforations of the subchondral plate that started at the periphery of the defect

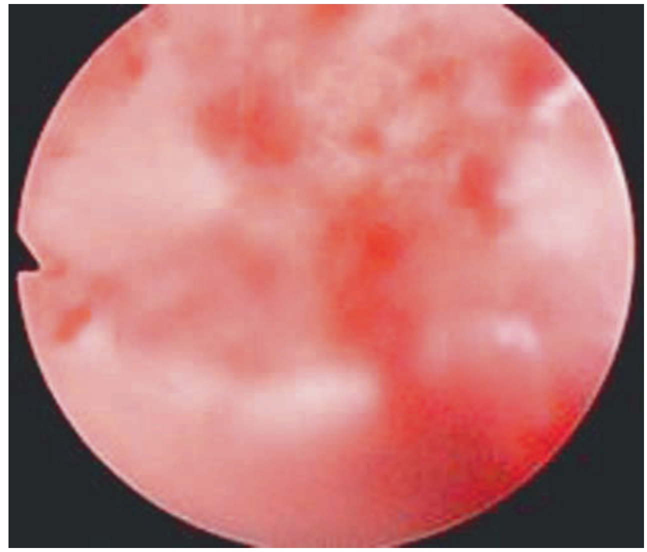


Fig. 16 Release of tourniquet should reveal subchondral bleeding via microfracture perforations

Formal physiotherapy is recommended to assist in weight-bearing compliance and to initiate swelling control and protected strengthening. Wilk et al. [69] have published a detailed postoperative program that is recommended. Return to pivoting and impact sports can often be a difficult decision, particularly with a high-level or professional athlete. Gill et al. [70] recommend return to cutting sports at 4 months. Often, a high-resolution MRI [40] at 4–6 months can help with return-to-play decisions. Assessing degree of lesion fill and subchondral plate reactivity can help estimate amount of maturation. Maximum tissue integration likely does not occur for several months, with complete maturation not achieved until 1 year. Certainly, a knee with no swelling combined with normal muscular activation and strength patterns is mandatory for return to sports. The lesion size and location and the exact demands on the knee also play a role in the decision factor. For instance, a soccer goalie may return quicker than will a center. In summary, the combination of time from surgery, lesion locale and size, strength of extremity, and the particular demands on the knee should decide return to activity.

Case Report

Case 2: Microfracture

Chief Complaint and Patient History: The patient is an 18-year-old woman with persistent left knee pain after drilling of an osteochondritis dissecans (OCD) lesion 2 years earlier. In review of the prior surgical notes, a soft area of cartilage was discovered in the region of the posterior lateral femoral condyle (LFC), and drilling with an 0.064 inch wire was conducted. The patient has been experiencing worsening left knee pain and failure to return to athletics since undergoing this procedure. All discomfort is laterally based. Moderate joint effusion is reported when physical exercise/sports are attempted. The patient experiences pain in the knee up to a week after participation in this type of activity.

Physical Exam: The patient is 5′3″ tall, weighs 120 lb, and has a thin build. There is no knee effusion. There is minor synovial thickening with no instability or meniscal signs and grossly normal alignment with full range of motion. There is minor discomfort with palpation of the LFC in the deep-flexed position.

Imaging: See Fig. 17

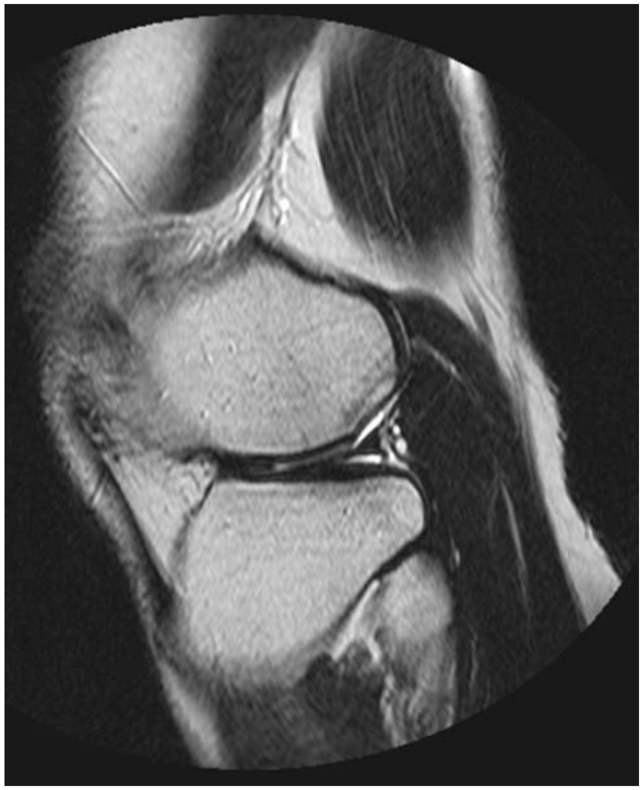


Fig. 17 Sagittal MRI scan demonstrates an 18-mm osteochondral defect

Surgery/Treatment: The patient underwent an arthroscopic microfracture procedure for the LFC OCD. On first inspection, the tissue at the region of the defect appeared fibrous and delaminated. This tissue was debrided using a ring curette and shaving instrument until stable borders were achieved. The final defect measured 22 × 20 × 4 mm (Fig. 18A–C).

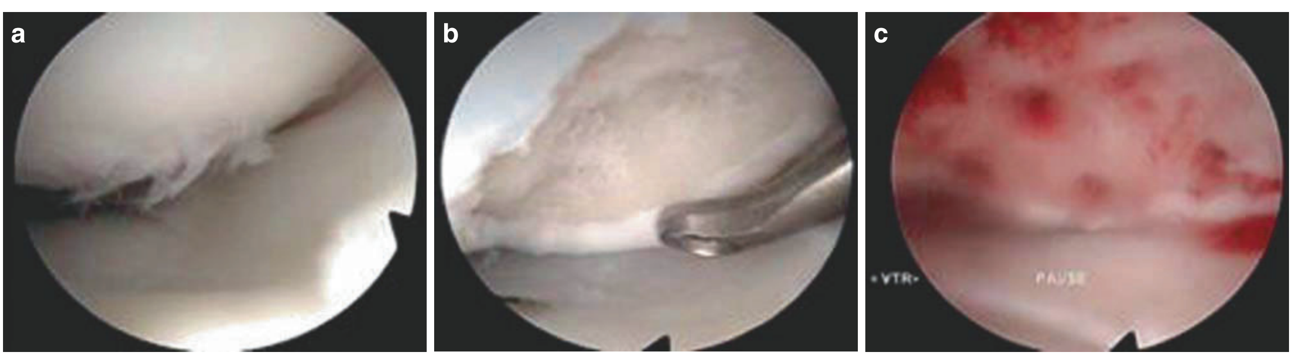


Fig. 18 (**A–C**) Arthroscopic views of microfracture surgery

Postoperative Course/Follow-Up: The patient was placed in a hinged knee brace with TDWB restriction. A CPM machine was used for 6 hours per day for 6 weeks. Physical therapy was initiated immediately postoperatively. Biking and closed chain activity were initiated at 4 weeks; swimming and open chain activity were initiated at 4 months. At her 10-month follow-up appointment, the patient reported no knee pain or swelling, and physical examination was benign. A new MRI scan was reviewed at this time (Fig. 19). At that time, full unrestricted activity was permitted.

Fig. 19 Sagittal postoperative MRI scan 10 months after microfracture with satisfactory fibrocartilaginous fill of defect

Osteoarticular Grafting

To date, osteochondral grafting (OCG), which includes both autogenous and allogenic sources, is the only cartilage restoration procedure that produces true hyaline AC. The objective is to take a normal, minimally loaded osteoarticular composite from the knee (or donor allograft) and transfer it to a chondral defect. This concept is not new; in fact, Judet [71] in 1908 reported pain relief associated with transplanting osteochondral fragments after trauma. Several animal models have demonstrated consistent survival and integration of the transplanted grafts [72, 73]. OCG techniques, specifically osteochondral allograft, have the longest published follow-up compared with those of other AC resurfacing techniques [74–76]. There are several systems available to perform osteochondral grafting. The term *mosaicplasty* is reserved to describe use of multiple smaller-diameter grafts. Mechanical studies have shown superior fixation of larger traditional osteochondral grafts (Fig. 20) versus smaller mosaic multiple grafts [77]. Mosaicplasty, however, offers more surface area for osseous integration and potentially improved contouring [72, 78, 79].

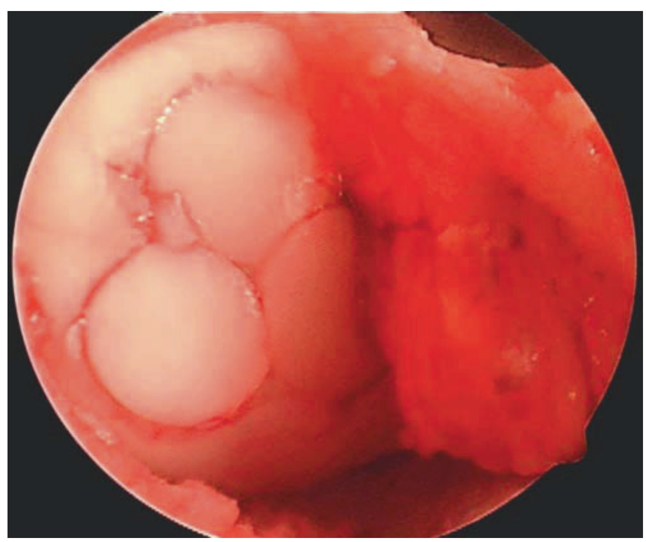


Fig. 20 Intraoperative example of three 10-mm osteochondral grafts. Because of the larger graft size, the inferior graft (*lower left*) was placed slightly proud posteriorly in order to be congruent in the more superior weight-bearing portion of this lateral femoral condyle defect. Mosaicplasty, with smaller osteochondral grafts, could potentially offer improved contouring

Performing either technique of osteochondral grafting, whether it be moasicplasty with smaller plugs or the larger-diameter grafts, is technically demanding. Properly positioned grafts consistently integrate; however, mechanical studies prove that graft-length mismatch is prone to failure. Proud grafts undergo excessive motion and fissuring [80], and depressed, poorly fitted grafts are unloaded and degenerate [80, 81]. In summary, regardless of the OCG technique, a perpendicular press-fit–placed graft that is level with surrounding AC will produce an optimal result [82].

Autograft

The active patient with a symptomatic condylar defect less than 2.5 cm is the ideal candidate for an osteochondral autograft. Limited graft availability and potential donor-site complications restrict the autograft OCG indications to smaller defects. Proponents of allogenic OCG note that cell-based therapies produce inferior repair tissue that deteriorates over time. In a randomized, prospective trial comparing osteochondral autograft and microfracture, Gudas et al. [78] recently demonstrated that, at an average of 37.1 months follow-up, the OCG shows significant clinical results over those of microfracture in an athletic population. Fifty-seven athletes with a mean age of 24.1 years were treated with either microfracture or the mosaicplasty technique. The average size in both treatment groups was roughly 2.8 cm^2, and, at follow-up, the OCG group, according the modified hospital for special surgery (HSS) and ICRS scores, showed a 96% good or excellent result compared with 52% after the microfracture procedure. Others [79, 83] have shown similar results and cite the repair tissue is true hyaline cartilage with healthy subchondral bone.

In summary, the advantages of a single-stage procedure with a relative low cost make the autograft technique attractive. However, limitations of this surgery include donor-site morbidity, limited graft availability [77, 84, 85], and difficulty in re-creating the normal articular contour. Even a slightly prominent graft is exposed to significant shear [86] and chondrocyte death at the edges of the plug [87].

Surgical Technique of Autograft OCG

There are several commercially available systems to perform OCG. Based on stability concerns and degree of lesion fill with smaller plug sizes, the author uses the Osteochondral Autologous Transplantation System (OATS; Arthrex Inc., Naples, FL). Users of the Mosaicplasty (Acufex Microsurgical, Inc., Mansfield, MA) and Consistent Osteochondral Repair (COR) systems (Mitek Inc., Norwood, MA) note benefits of easier contouring from smaller-diameter plugs and shallower harvest depth, respectively.

AC Lesion Evaluation and Preparation

Standard arthroscopic setup is used, with a leg holder and tourniquet positioned very high on the thigh. It is critical that the knee can be ranged through a maximum arc of motion. Arthroscopy is performed, and the AC lesion is identified and measured with sizing tamps. In general, single-plug lesions are performed arthroscopically as others have described [88], whereas multiple-plug lesions are completed through a small arthrotomy.

Donor Graft Harvest

After estimating the sizes and amount of grafts required, the superior lateral trochlea is the initial donor site used, followed by medial margin of trochlea and intercondylar notch. The appropriate “donor” labeled harvester is placed in a perpendicular fashion over the desired AC donor site. If multiple plugs are required, it is beneficial to gently score the chondral surface to avoid converging donor grafts (Fig. 21A, B). With the OATS system, the donor tube is driven to a depth

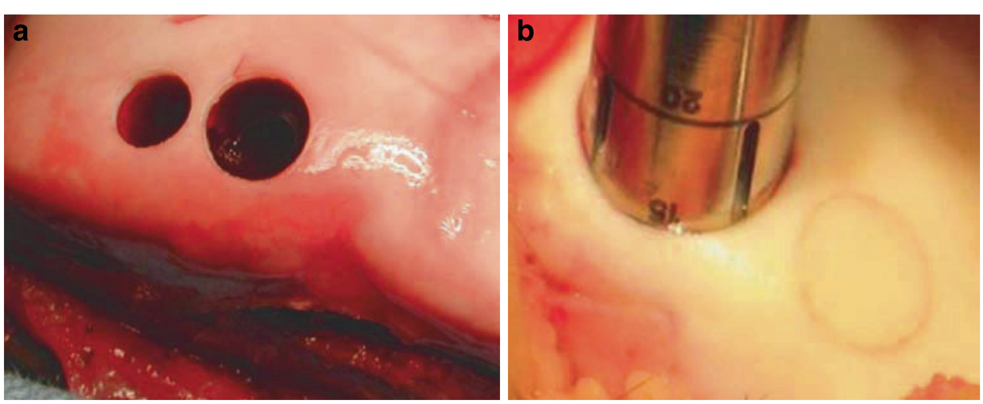


Fig. 21 (**A, B**) Intraoperative views depicting appropriate spacing of donor osteochondral graft sites to avoid converging and harvesting incomplete subchondral bone

of 15 mm. It is mandatory to avoid toggling the system while inserting the donor tube. Once a 15-mm depth is confirmed, a forceful axial load and rotational force is required to release the harvested plug. Complete osteochondral graft harvest is confirmed and placed on a back table. Final measurements and graft obliquity are noted to help plan for recipient-site preparation. A measuring rod inserted in the donor site can confirm exact depth and obliquity of graft. Each graft is harvested and then placed in a successive fashion.

Recipient-Site Preparation

In the case of multiple plugs, a miniarthrotomy is completed. Angled knee and Hohmann retractors are helpful once the capsulotomy is completed. To create the recipient socket, complete visualization is required to ensure a perpendicular approach. The recipient labeled tube is driven to approximately 2 mm less than the donor graft length and easily removed with an axial load and rotational maneuver. With younger and firmer bone, a 1-mm discrepancy is recommended. Matching the graft obliquity resulting from the donor harvest can be estimated using the lasered numeral system located on the tip of the tube (Fig. 22). An equal-diameter measuring rod is inserted into the recipient site and then tamped to match the exact length of the donor graft. This ensures an exact donor-recipient match.

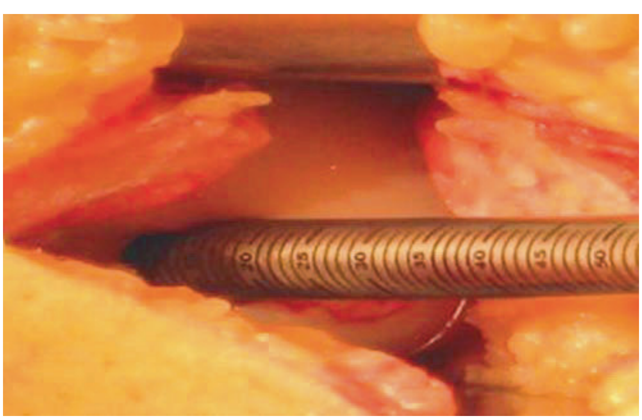


Fig. 22 Measuring device placed at donor site for osteochondral harvest using the OATS system. The laser line system is used to measure the correct depth. (Courtesy of Arthrex, Inc., Naples, FL.)

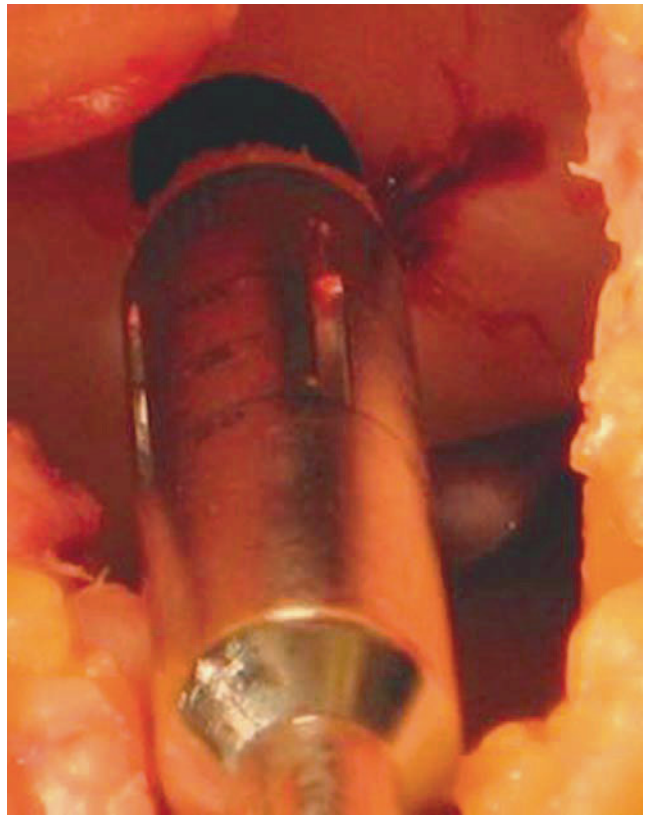


Fig. 23 Intraoperative view depicting insertion of a donor osteochondral graft into the recipient socket. The graft should be left a few millimeters proud and completed with an oversized tamp under direct observation

Graft Insertion

The donor plug is gently tapped, leaving the graft a couple of millimeters proud to assist in guiding the construct into the recipient socket (Fig. 23). The graft should be inserted with very soft mallet strikes to avoid AC injury [89]. A tamp allows better visualization and should be used to complete the transfer (Fig. 24). It is preferable to leave the high portion of an oblique graft level with the surrounding AC. The steps are repeated as necessary to fill the defect. Awkward or uncovered areas can be microfractured.

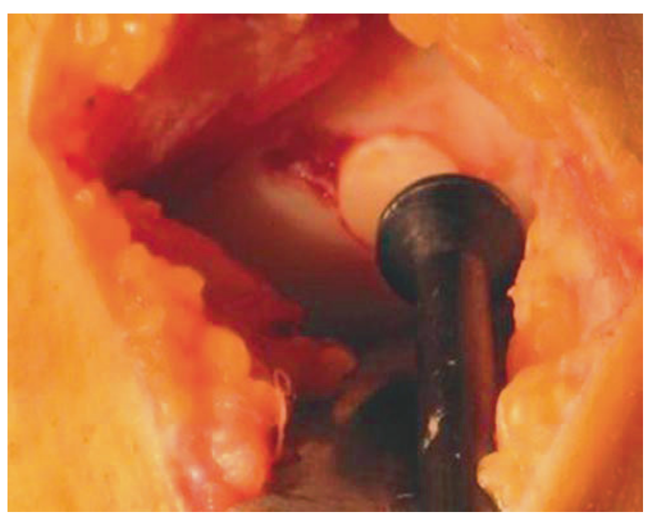


Fig. 24 A tamp is used to complete the insertion of the plug and secure proper placement

Postoperative Care/Rehabilitation

Similar to other AC resurfacing procedures, the postoperative program must be individualized [90]. The size and location of the repair dictate progression of weight-bearing status and degree of strengthening. In general, single-plug OCG is TDWB for 2–3 weeks, followed by progressive FWB to tolerance. Multiple-plug OCG remains TDWB for 4 weeks, with progression to FWB until 6 weeks. In either case, unassisted FWB is not permitted until normal gait patterns are achieved. CPM is recommended for up to 6 hours a day for at least 3 weeks. Low-resistance stationary cycling is allowed the first week. "Rocking" the pedals back and forth until a full rotation is achieved can be an early rehabilitation goal for patients. Immediate pelvic and upper-body strengthening is permitted. An upper-body ergonometer (UBE) is recommended for the dedicated athlete to maintain conditioning. Extremity isometric strengthening in arcs of motion that avoid loading the graft can start to tolerance the first week. After suture removal, a water-based program, if available, is very beneficial. At 6 weeks, closed chain strengthening and treadmill walking are encouraged. Accelerated progression to sports-specific and functional training begins around 10–12 weeks and beyond, depending upon how the joint is reacting to increased demands. Patellofemoral OCG generally has a longer return to sports and, like other restorative procedures, has less success in returning the patient to sports participation.

Allograft

Of all the AC restoration options, allograft OCG has the longest clinical follow-up in the literature. Recently, Maury et al. [91] demonstrated chondrocyte viability after 25 years postimplantation. Similar to autogenous OCG, the allograft composite also contains viable and functioning chondrocytes to support the matrix [92]. Early reports confirmed success with fresh osteochondral allografts [93, 94]; however, they are no longer available in the United States. "Fresh stored" osteochondral allografts are now the preferred source because of safety and medicolegal issues. The success of osteochondral grafting directly relates to chondrocyte viability [92, 95]. However, the goal of maximizing chondrocyte viability is challenged by the risk of disease transmission and pathogen contamination. Current testing guidelines set by the American Association of Tissue Banks generally allow an osteochondral allograft to be available for approximately 30 days [96]. Recent literature supports use of hypothermically "fresh-stored" allografts as a source for AC resurfacing [97, 98] Davidson et al. reported on 2-year outcomes of 10 patients who received massive hypothermically stored osteoarticular allografts for femoral condyle defects. At a mean 40 months postimplantation, second-look arthroscopy with biopsy confirmed the graft and native cartilage cellular density and viability as similar. All clinical measures revealed statistical improvement at follow-up.

A prior failed restorative procedure, or a large chondral defect (greater than 2.5 cm^2), particularly with significant bone loss, is the ideal candidate for osteochondral allograft transplantation. Tibial-sided lesions may benefit from this option as well.

Surgical Technique of Allograft Osteoarticular Transplantation

Generally, all osteochondral allograft techniques use a matched graft. However, smaller grafts (<2 cm) may be amenable to grafting from an unmatched condyle. Similar to meniscal allograft transplantation, a matched allograft is selected based on sizing using standard radiographs with a radiolucent marker. The allograft can be implanted in a few different ways. A sculpted free-hand shell graft is one option that is particularly useful on the tibia because of instrumentation access (Fig. 25). A bulk graft for massive defects including near-complete trochlear or condylar replacement is another useful method. Both the free-hand and matched grafts generally require supplemental internal fixation. Finally, the most common technique is a dowel-

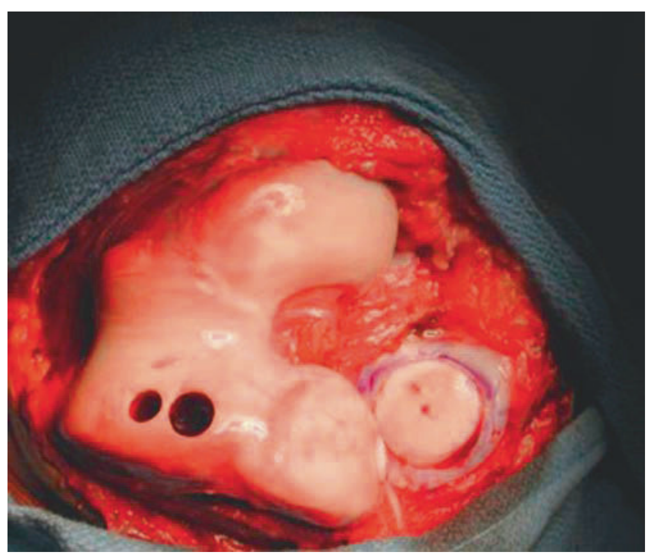


Fig. 25 Intraoperative view depicting the placement of a matched, sculpted, free-hand shell allograft used for a large tibial plateau defect. The lateral meniscus was released at the capsular junction and later repaired. Also note an osteochondral autograft was used for a "kissing" femoral condyle defect

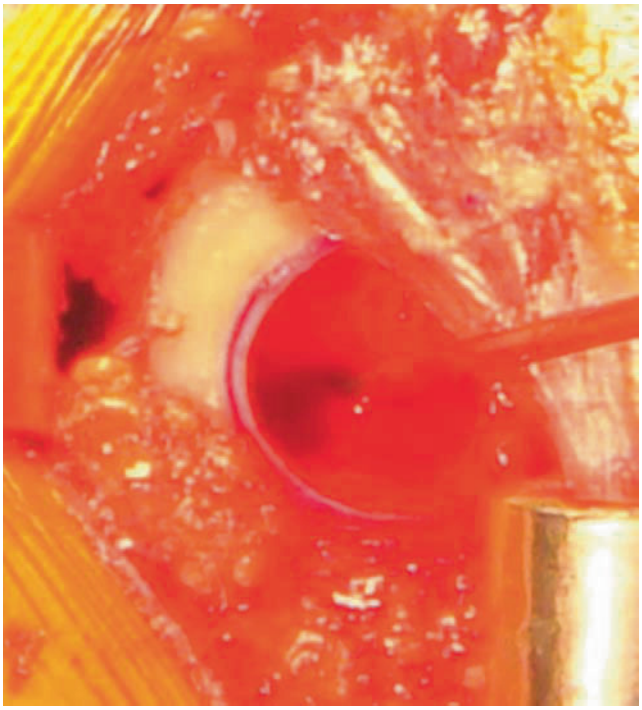


Fig. 26 Intraoperative view showing guide pin placed in the center of the host defect and reamed to a depth of 8 mm with an appropriately sized instrument

shaped press-fit graft technique that is performed with precise instrumentation from Arthrex, Inc. (Naples, FL).

The dowel technique (or "super" OATS) can insert allograft dowels of up to 35 mm. Always inspect the graft prior to anesthetizing the patient. The proper side and acceptable tissue quality should be noted. After initial arthroscopic evaluation, a small arthrotomy can be used to perform the transplant.

The entire borders of the lesion are identified, and a guide wire is drilled in a perpendicular manner through the center of the defect. The appropriate sizing dowel is selected, and the recipient site is then reamed to a depth of healthy bone, which is generally 6–10 mm (Fig. 26). Deeper grafts will have a greater press-fit. When completed, the depth of all four quadrants is measured, and a sterile pen is used to mark the 12 o'clock position.

The 12 o'clock position of the delivered graft is then marked and secured in the work station (Fig. 27). The appropriate-size harvester is then drilled through the entire graft. All four quadrants of the graft are then measured and trimmed to the corresponding depth of the recipient site. Graft-holding forceps are available to stabilize the graft while contouring; this is followed by pulse lavage to reduce the quantity of marrow elements. The graft is then press-fit initially by hand and seated flush with gentle taps with an oversize tamp (Fig. 28). Any evidence of graft instability warrants supplementary fixation with either pins or screws. The author's preference is metal screws. The knee is then cycled to check for stability and conformity.

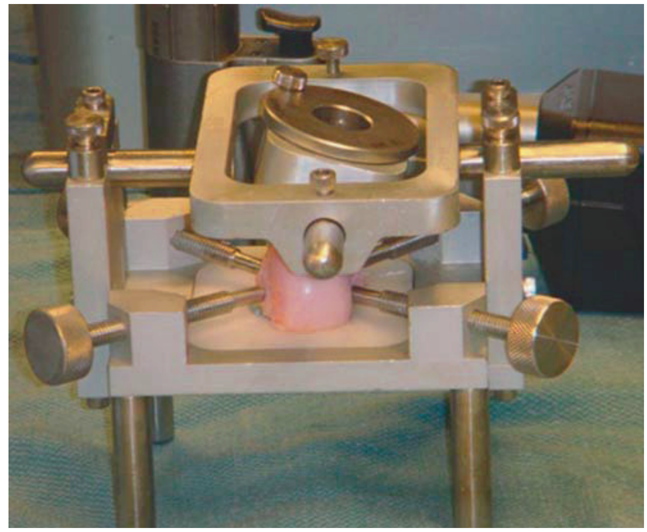


Fig. 27 The matched hemi-condyle allograft is secured in the work station. Note the saw guide is positioned in a manner perpendicular to the graft. (Courtesy of Arthrex, Inc., Naples, FL.)

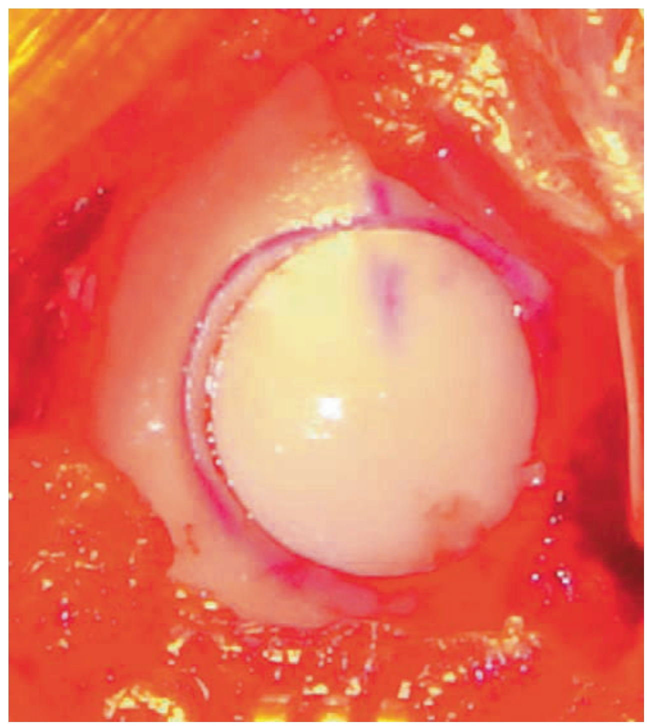


Fig. 28 The osteochondral allograft is seated flush with surrounding native cartilage

Postoperative Care/Rehabilitation

TDWB is recommended for a minimum of 8 weeks to avoid collapse while graft incorporation occurs. CPM is used up to 8 hours a day for the first 4 weeks, and then daily low-resistance stationary biking is recommended. A simple home program of quadriceps sets and heel slides is started immediately, followed by supervised physiotherapy, including a water-based program, when the wound is stable. Depending upon the size of the graft, return to sports may be up to 1 year away. Large bulk allograft recipients should permanently avoid impact activities.

Case Report

Case 3: Osteochondral Allograft

Chief Complaint and Patient History: The patient is a 33-year-old woman who is 1 year status post–autologous chondrocyte transplantation (ACT) surgery for a medial femoral condyle OCD. She presents with persistent knee pain with swelling and catching sensation. The patient underwent two cartilage procedures prior to this, including microfracture, all of which failed to provide symptom relief. She states that the ACT surgery improved symptoms of pain and swelling for about 6 weeks, but both symptoms have returned. The knee catches and has locked on numerous occasions.

Physical Exam: The patient is 5′7″ tall, weighs 135 lb, and is athletic appearing. She has normal range of motion, ligamentous exam, and alignment (confirmed by long-standing radiographs). There is a small effusion with no obvious meniscal signs and very positive discomfort with medial condylar palpation with associated painful crepitus.

Imaging: See Fig. 29.

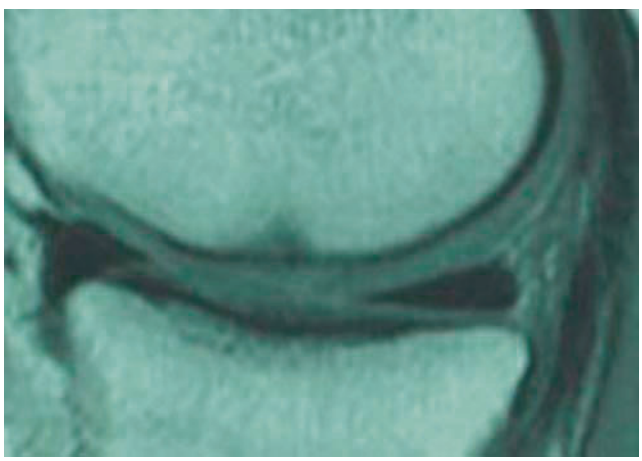


Fig. 29 MRI scan demonstrates region of chondral defect, with moderate reactive marrow edema

Surgery/Treatment: The patient underwent arthroscopy for assessment of autologous chondrocyte implantation (ACI) cartilage and evaluation for an impinging intraarticular osteophyte. A well-attached and smooth chondral graft was observed (Fig. 30A, B).

After arthroscopy, the patient underwent 3 months of physical therapy followed by a course of viscosupplementation. This provided only minimal relief of her medial- based pain with activities of daily living. A repeat MRI was ordered.

Imaging: See Fig. 31A, B.

Surgery/Treatment: The patient underwent placement of a matched osteochondral allograft to the medial femoral condyle via a small arthrotomy. A press-fit dowel technique was used (Fig. 32A, B).

Postoperative Care/Follow-Up: The patient was maintained TDWB for 8 weeks. Traditional physical therapy (PT) and CPM were initiated during the first postoperative week. The patient is currently 6 months postoperative and pain-free with activities of daily living. Light sports participation will be permitted at the 1-year mark when repeat MRI is planned.

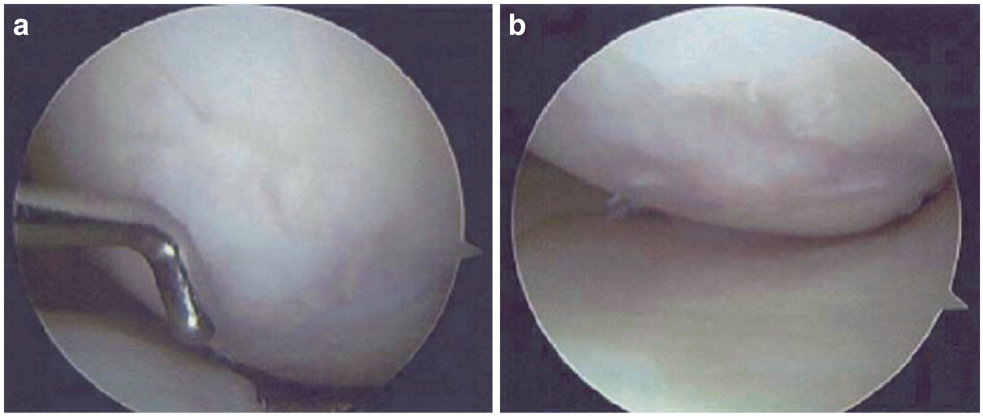


Fig. 30 (**A**, **B**) Arthroscopy demonstrates a well-integrated chondral graft with minor fraying

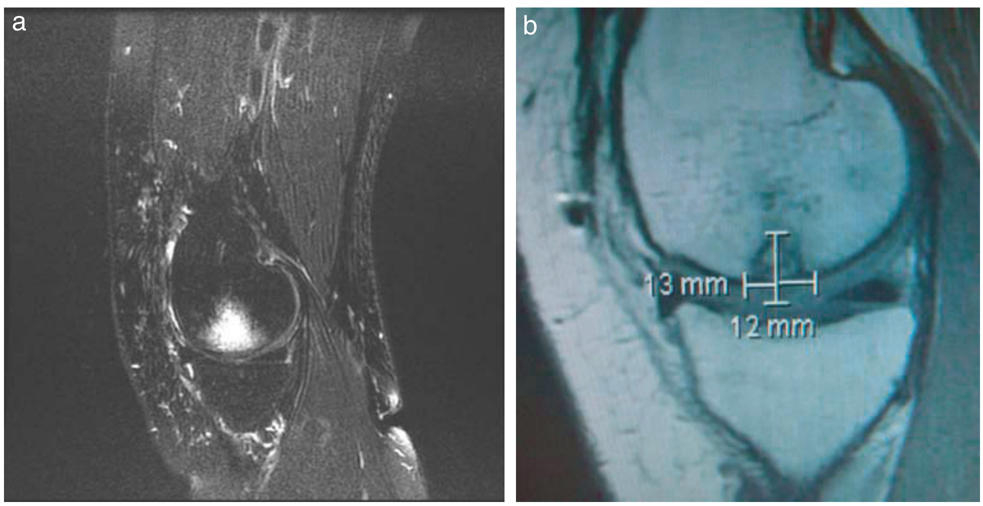


Fig. 31 (**A**, **B**) MRI scans demonstrate an unstable chondral defect, with significant regional bony effusion

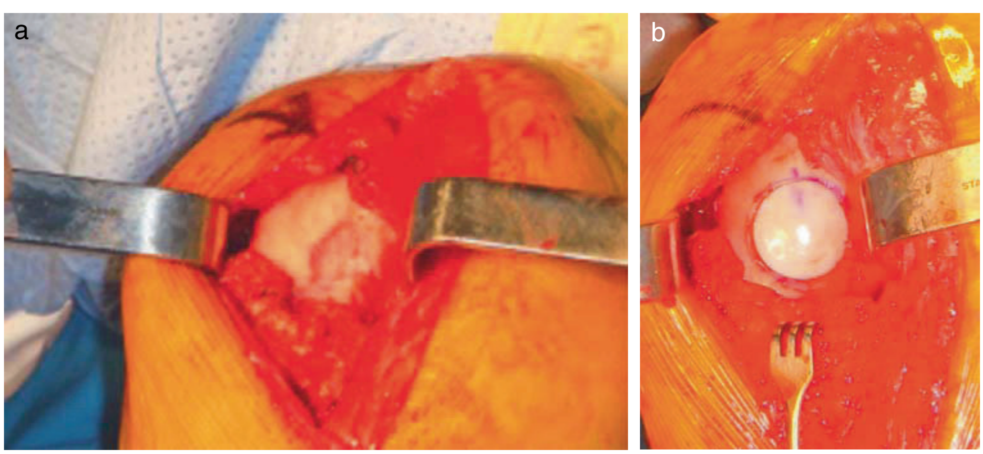


Fig. 32 Chondral defect (**A**) after debridement and (**B**) after OATS allograft. (Courtesy of Arthrex, Inc., Naples, FL.)

Autologous Chondrocyte Transplantation

First clinically reported in 1994 [99], this controversial articular resurfacing method is the first and only technique that uses true tissue engineering. The procedure requires two surgeries. During the first, the articular lesion is evaluated and normal host cartilage is harvested, followed by cell culturing. The second procedure involves transplanting the cells under a harvested periosteal patch through an arthrotomy. The goal is to achieve a more durable "hyaline-like" repair tissue that resembles hyaline AC. Autologous chondrocyte transplantation (ACT) human biopsy studies, however, demonstrate a 10% to 67% presence of hyaline repair tissue. Studies support that transplanted chondrocyte repair tissue is biomechanically superior to traditional fibrocartilage-based procedures [100, 101]. Knutsen et al. [102] recently reported a randomized trial comparing ACT and microfracture at 5 years. They found no clinical difference in clinical scores; however, the authors did observe that the patients with better-quality repair tissue had no late failures comparing the 2- and 5-year results. Others have confirmed the relationship of clinical success directly with the presence of hyaline-like repair tissue [103, 104]. Several authors have documented clinical success with ACT [105–107]. One recent series demonstrated excellent results for the treatment of trochlear defects [108], and three reports have shown success in returning athletes to competition with ACT [109–111]. It seems that the closer the repair tissue resembles normal hyaline structure, the better the biomechanical resilience and clinical success results will be.

Surgical Technique

Defect Evaluation and Chondral Biopsy

First-stage arthroscopy is performed, and, if chondral defect is amenable to ACT, then a cartilage specimen is harvested. Three acceptable nonarticulating sites are the superolateral or superomedial trochlear ridge and the intercondylar notch. A ring curette or gauge is used to harvest enough AC around 5 × 10 mm (equivalent to three Tic-Tac candies). In the United States, all specimens are shipped in a sterile container to Genzyme Biosurgery (Cambridge, MA) for cell culturing. A minimum of 30 days is required to prepare the cells. Patients are sent to physical therapy for an aggressive preoperative strengthening and conditioning program in preparation for the second surgery while protecting the defect.

Arthrotomy and Defect Preparation

Patients are anesthetized supine, and a tourniquet and small foot rest are positioned. Antibiotics are administered. A minimum lateral or medial parapatellar arthrotomy is performed. Without compromising exposure, efforts should be made to avoid dislocating the patella, which may improve quadriceps muscle recovery. The author prefers to sublux the patella versus eversion and dislocation. A bent Hohmann retractor placed in the notch can improve exposure. The defect is radically debrided back to healthy vertical borders. Internal osteophytes, often from prior microfracture, are tamped or gently curetted to avoid destabilizing the subchondral bone (Fig. 33). Hemostasis is very important to ensure better-quality repair tissue. The defect is measured with a flexible ruler or templated with either sterile paper from surgical gloves packaging or aluminum from suture packaging. The defect is then packed with thrombin- and epinephrine-soaked patties.

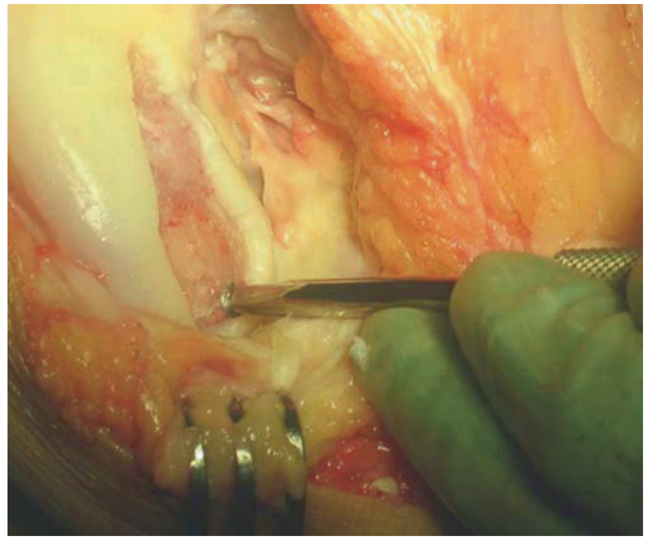


Fig. 33 After debridement of the lesion to stable AC margins, the subchondral internal osteophytes can be gently tamped to even the base of the defect

Periosteal Harvest

The tourniquet is deflated to allow for maximum defect hemostasis, and the periosteal patch is obtained. A separate incision at the proximal tibia is the preferred site for periosteal harvest. In patients greater than 40 years of age or if the tibial periosteal tissue is inadequate, the distal femoral condyle can be a reliable source. Graft overgrowth is a known complication and is related to periosteal thickness; therefore, the thinnest periosteal patch is the goal. Subsynovial dissection and removal of all excess fascial layers is critical to help avoid this adverse event. Always harvest 2 mm extra periosteum to allow for shrinkage while securing the patch. Using two hands, a small elevator is used to gently elevate the periosteum (Fig. 34). The harvested patch

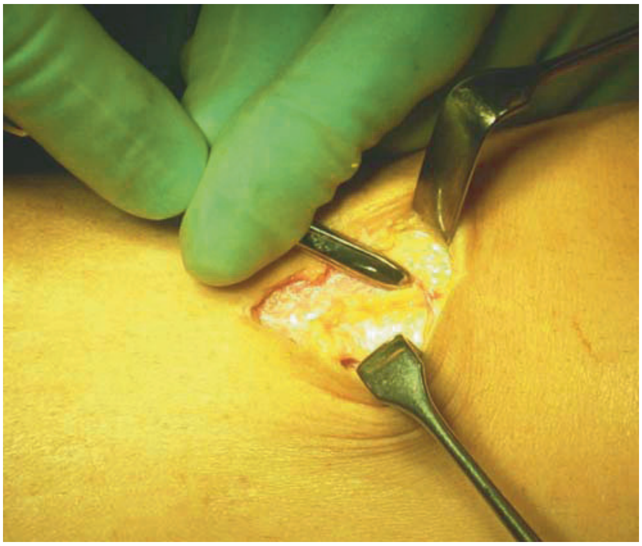


Fig. 34 "Two-hand" technique for elevating the periosteum from the proximal tibia

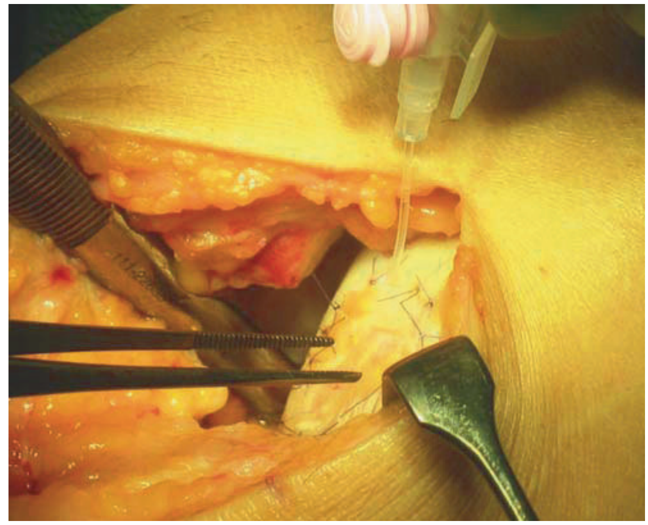


Fig. 35 The autologous chondrocyte cells are injected underneath a water-tight periosteal patch

is checked for perforations and placed in a sterile soaked sponge.

Periosteal Patch Suturing

The patch is brought to the defect and sewn with dyed 6-0 Vicryl swedged on a P-1 needle (Ethicon, Johnson & Johnson, New Brunswick, NJ). Removing half of the suture and soaking it in sterile mineral oil to minimize trauma to the patch is helpful. The cambium (bone side) of the patch is secured facing the defect with interrupted and simple sutures. The completed patch should be watertight with the suture knots on the side of the periosteum. A superior stitch is passed but not tied to allow for an angiocatheter to fill the chamber. Additional sutures are placed to secure the water-tight chamber, which is then sealed with TISSEEL fibrin sealant (Baxter Healthcare Corporation, Deerfield, IL).

Transplanting Chondrocytes

The cultured cells are then carefully aspirated with a sterile 18-gauge angiocatheter. The cells are resuspended a few times to dissolve the solid pellet and distribute the cells evenly in the syringe. The volume of the cell aspirate is typically around 0.2–0.4 cm^3. The general rule is to request one vial per defect transplanted. The suspension is then slowly injected into the bottom of the chamber and filled while withdrawing the catheter (Fig. 35). The final stitch is tied and layered with TISSEEL sealant. Careful irrigation follows, and documenting the range of motion that corresponds with tibia-graft (in the case of femoral graft) contact is helpful information for the physiotherapist and follow-up exams.

Postoperative Care/Rehabilitation

Like all AC restoration procedures, ACT is extremely individualized and must be customized for each patient. Regular communication between physician, patient, and therapist is critical to support the transplant maturation process. Trochlear and patella lesions are weight bearing as tolarated (WBAT) with brace locked in extension for 6 weeks. Tibial and condylar lesions are TDWB for 6 weeks with a hinged brace for protection. All patients initiate CPM and quadriceps sets postoperative day 1. A formal physiotherapy program starts at the end of the first week, focusing on swelling control and range of motion. By 8 weeks, all braces and ambulatory aids are discontinued while the progressive closed chain strengthening program is under way [90]. Return to impact activities and running sports is permitted from 4 to 12 months, depending on many variables including the size and location of the defect. The actual sport or activity must be considered, with cutting sports likely the last to be recommended. MRI and biopsy studies show continued graft maturation and better-quality tissue up to 18 months [49, 112]. Hence, remaining active with progressive joint loading appears to enhance repair and produce better results [109, 111].

Case Report

Case 4: Autologous Chondrocyte Transplantation

Chief Complaint and Patient History: The patient is a 27-year-old elementary school teacher who presents with right knee pain and swelling. Initial injury more than 3 years ago was a contact injury while playing competitive soccer. Initial recovery from this event was relatively benign, but the patient was left with a chronic joint effusion and residual right knee pain. The patient underwent an osteochondral autograft procedure with one 8-cm plug 3 years earlier. Surgery failed to relieve her symptoms of pain and swelling.

Physical Exam: The patient is 5′6″ tall, weighs 145 lb, and has an antalgic gait pattern favoring her left knee. The right knee has a small effusion with positive crepitus. There are no meniscal or ligamentous signs. There is no ipsilateral hip or ankle pain, and normal alignment is present.

Imaging: See Fig. 36A, B.

Surgery/Treatment: The patient underwent a right knee arthroscopy with chondroplasty of medial femoral condyle (MFC) and cartilage biopsy (Fig. 37A–C). A single osteochondral plug was observed in the MFC with no integration at its borders. The defect appeared to have propagated and now measures 30 mm × 20 mm. Cartilage was harvested via the superolateral trochlear groove for future ACT surgery.

Postoperative Care: The patient was placed WBAT, and physical therapy was initiated. As expected, the patient continued to experience knee joint discomfort and swelling.

Surgery/Treatment: The patient underwent ACT of the medial femoral condylar defect approximately 2 months after her biopsy (Fig. 38A, B).

Postoperative Care: The patient maintained TDWB for 6 weeks, and CPM was used throughout this period. She continued to progress well relatively pain-free. At the 6-month mark, the patient started to experience progressive medial knee pain and increased crepitus. The patient denied new injury and joint locking. A cycle of viscosupplementation was performed, and physiotherapy was continued.

Imaging: Six-month follow-up (Fig. 39).

Further Treatment: The viscosupplementation provided moderate pain relief. However, the patient started to experience moderate catching sensation with active knee movement.

Imaging: Nine-month follow-up (Fig. 40).

Surgery/Treatment: The patient underwent right knee arthroscopy with chondroplasty of MFC (Fig. 41A, B). Ninety percent incorporation of the graft was appreciated. The remaining superior 10% existed as a flap of cartilage at the anterior leading edge of the cartilage graft. This region was debrided, and a chondroplasty was performed. The minimally overgrown graft noted below the defect was left alone.

Postoperative Course/Follow-Up: The patient's mechanical symptoms were markedly improved. No further treatment was provided.

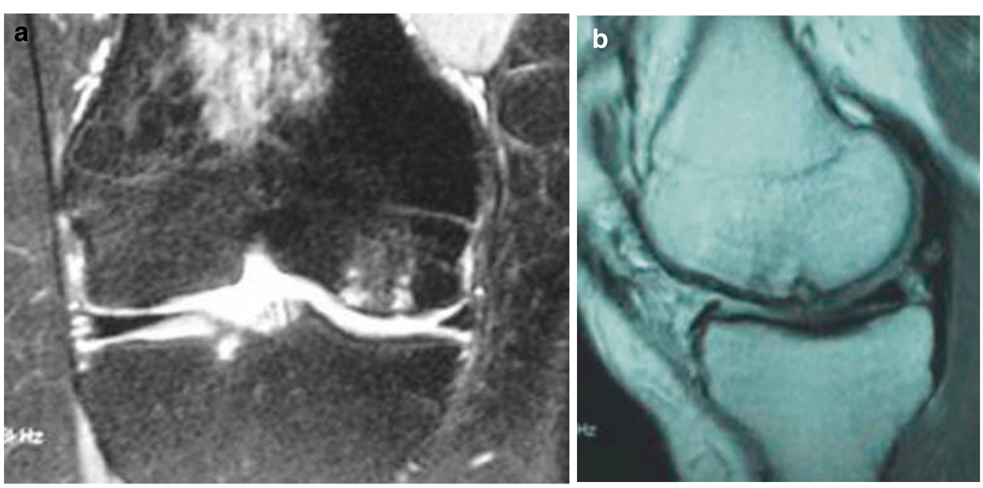


Fig. 36 (**A, B**) Coronal and sagittal MRI scans demonstrate bone edema at the site of osteochondral transplant, which is now devoid of cartilage

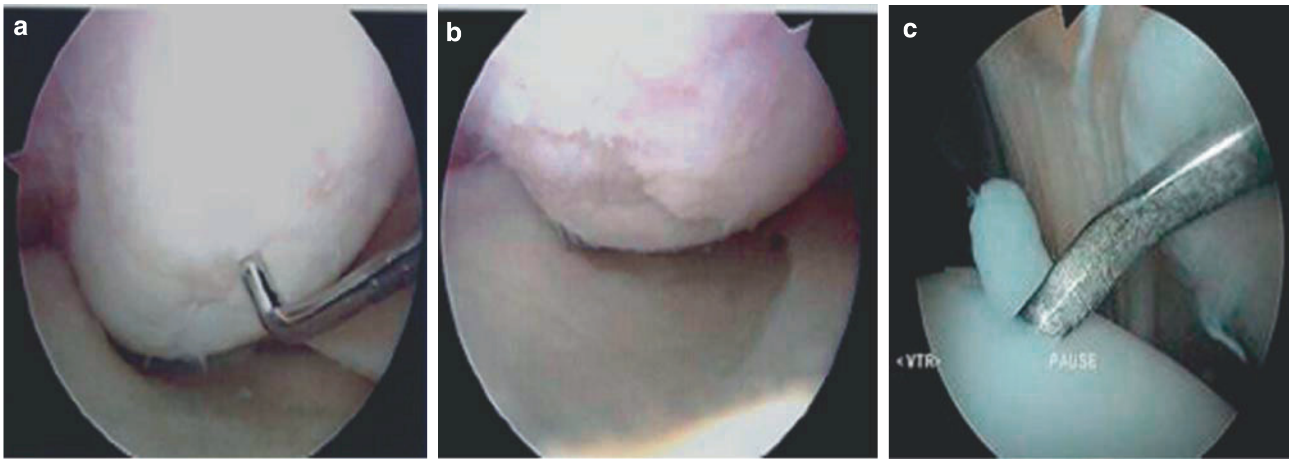


Fig. 37 (**A–C**) Inspection of MFC reveals a chondral defect with failed osteochondral graft. Cartilage was harvested with use of a ring curette

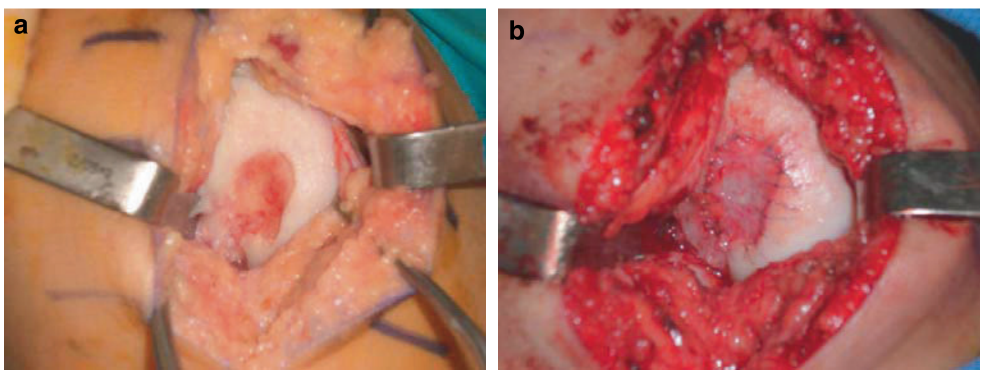


Fig. 38 (**A, B**) Defect was debrided to stable borders, and cells were inserted under the periosteum patch

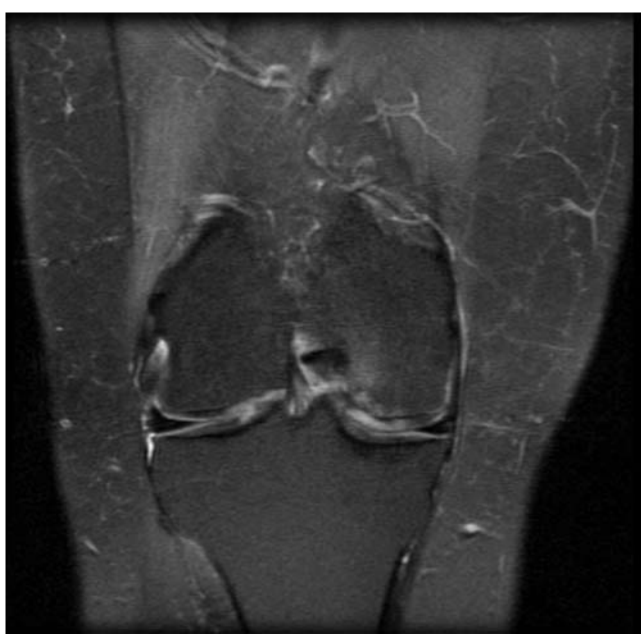


Fig. 39 MRI scan 6 months after surgery demonstrates excellent incorporation of cartilage graft

Fig. 40 MRI scan 9 months after ACT surgery demonstrates minor cartilage overgrowth at region of implanted cells

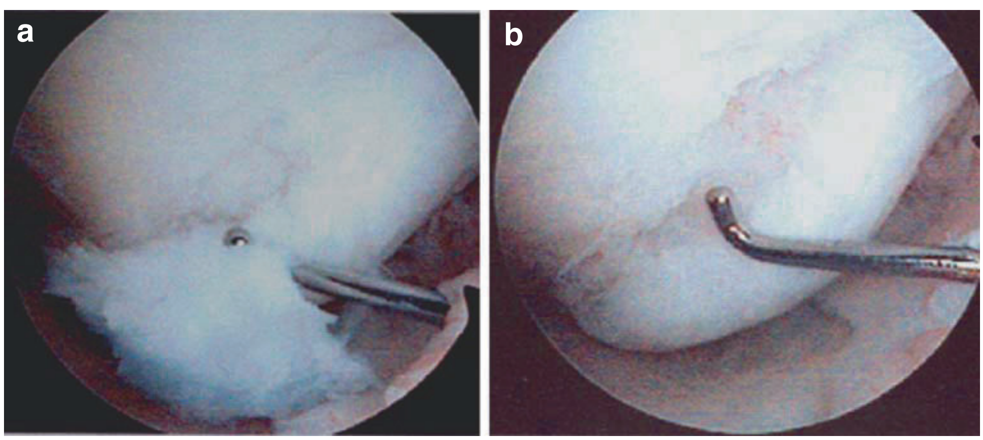


Fig. 41 (**A, B**) Right knee arthroscopy demonstrates failure of a small portion of the graft construct

Associated Bone Deficit/Uncontained Lesions

Chondral lesions with associated bone loss greater than 8–10 mm must be addressed with bone graft. Ipsilateral Gerdy's tubercle is very accessible and an excellent source of autogenous bone. In general, uncontained large defects are bone grafted at the time of chondral biopsy, and other defects can be treated with the "sandwich" technique developed by Peterson [112]. With this technique, the sclerotic bone at the base of the defect is curetted and then drilled followed by packing of cancellous autograft to the level of the adjacent subchondral plate. Two periosteal

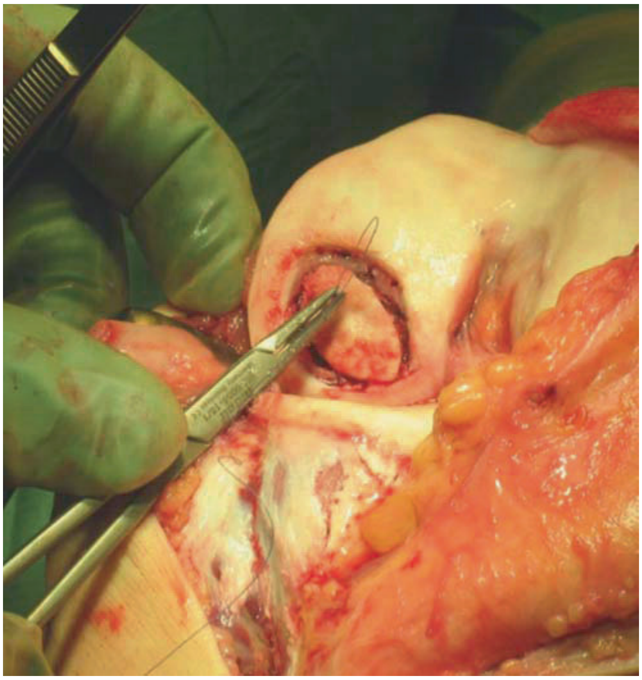


Fig. 42 Intraoperative view demonstrating the "sandwich" technique. The first harvested periosteal patch is placed on morselized bone graft with cambium layer up and secured with horizontal mattress sutures

grafts are harvested, and the first is sewn-in cambium side up with horizontal mattress sutures on the packed bone graft (Fig. 42). The second graft is secured in a routine manner, and the cells are injected between the two layers of the periosteum, hence the "sandwich" technique.

Osteochondritis Dissecans

Osteochondritis dissecans (OCD) is a disease of the subchondral bone that presents in variable sizes and locations in the knee joint, but most commonly on the condylar surfaces. Many theories have been proposed as an etiology of OCD including hereditary, traumatic, and ischemic [113]. As a result of a nonviable subchondral plate, the AC surface begins to soften and can deteriorate to complete destruction and entire AC loss. Often, the clinician has to deal with not only a sizable lesion but also a deep lesion with extensive bone loss (Fig. 43). The combination of a large and deep defect presents the greatest challenge of all AC maladies to the clinician.

In the presence of open growth plates, the OCD lesion is termed a juvenile OCD (JOCD); however, the same pathologic process to the subchondral bone applies. It is likely

Fig. 43 Lateral radiograph reveals a large OCD of the posterior femoral condyle, which required bone grafting and internal fixation

that most adult OCD lesions are a result of un-united JOCD, but adult OCD may occur in and of itself [114]. OCD of the knee has been classified based on many variables including anatomic location and the appearance on radiographs, bone scans, and MRI scans [115–117]. OCD lesions present in many variable stages; however, the two initial key distinctions are to assess whether the growth plates are open and if the lesion is stable. Typically, an unstable lesion is more symptomatic and, in the case of an adult, almost always requires operative intervention. The arthroscopic classification of Ewing and Voto (grade I, intact lesion; grade II, early cartilage separation; grade III, partially detached lesion; grade IV, crater with loose body) is straightforward and can assist in the surgical decision process [118].

Juvenile Osteochondritis Dissecans

JOCD presents in patients aged up to15 years or when the physis is closed. Patients often present with vague symptoms, and the physical examination may be normal. All juveniles with a painful knee should have radiographs on initial evaluation. MRI should follow if an OCD is recognized or suspected [119]. Most lesions appear on the posterolateral aspect of the medial femoral condyle, with lateral condylar, trochlear, and patella areas accounting for approximately 30% [120].

Nonoperative management is the preferred treatment option for stable lesions. A period of cessation of sports, bracing, protected weight-bearing combined with a physiotherapy program can heal more than 50% of JOCD according to Cahill and others [121–123]. However, 10–18 months were required for complete healing in Cahill's series. Treating JOCD is a true art, and no protocol can apply to any patient. For stable JOCD lesions, the author advocates an individualized program based on symptoms. Initially, PWB with a brace until symptoms have resolved is recommended. This is followed by no impact activities for at least another 6 weeks. Around 8–12 weeks, a repeat MRI will assess healing, and, depending on MRI findings, increases in activity are allowed. Comparative radiographs are useful, but the author finds MRI more helpful. If the adolescent remains asymptomatic at 3 months, gradual impact activity is permitted with close follow-up. Recurrence of symptoms warrants repeat MRI and possible consideration of surgical intervention, or another round of activity modification can be attempted.

Surgical options for a stable symptomatic defect are either antegrade or retrograde drilling [124, 125]. Both methods create channels for revascularization and decompress the subchondral bone to allow for healing. Unstable OCD lesions are treated in a similar fashion to adult OCD lesions.

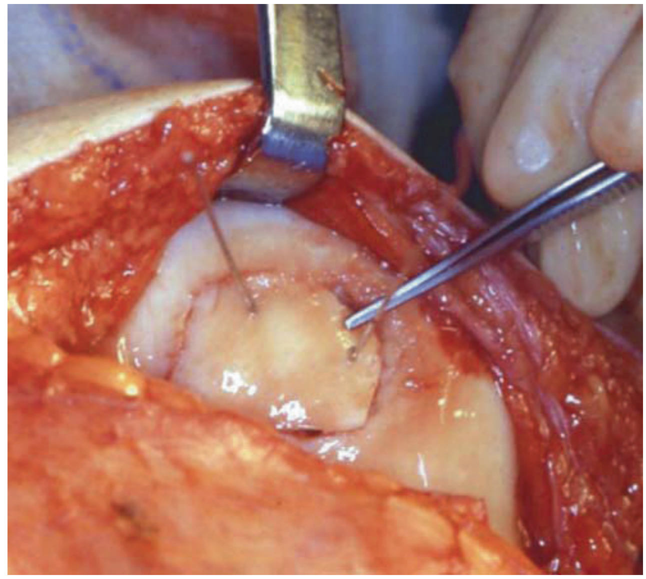


Fig. 44 Intraoperative view depicts a bone grafted and reduced, unstable OCD with pins positioned in preparation for cannulated screws

Adult Osteochondritis Dissecans

Symptomatic adult OCD lesions almost always require operative intervention. Like JOCD, the first priority is to establish stability of the fragment. Grade I lesions defined by intact cartilage but soft surface can be treated with simple stabilization. A variety of methods including bioabsorbable or metal pins and screws have been reported with good success [126–129]. In the unusual case of a stable symptomatic OCD in the adult, the author will use a bioabsorbable device such as the poly-lactic acid smart nail (Arthrex Inc., Naples, FL). Because of both personal and reported issues with use of bioabsorbable fixation in the setting of a unstable OCD fixation [130, 131], the author currently only uses metal fixation in these cases.

The unstable OCD or JOCD lesion can be approached in the same fashion. Many authors confirm success with various surgical methods for OCD stabilization [127–133]. Others have reported favorable results with other resurfacing treatment options discussed in this chapter including ACT, mosaicplasty, and allograft [106, 132, 133]. Preserving the OCD fragment should be the first priority. Often, this is a difficult decision; however, if there is at least 2 mm of acceptable bone, then the author recommends to proceed with fixation with autogenous bone graft (Fig. 44). If a portion of the fragment can be saved, then a hybrid method combining OCD fixation with a restoration procedure should be performed (Fig. 45). Removal of the fragment alone can lead to arthrosis [134]; hence, proceeding with a restorative procedure, at least in the younger population, must be considered. The author advocates miniarthrotomy, autogenous bone grafting, and internal metal fixation for all unstable OCD.

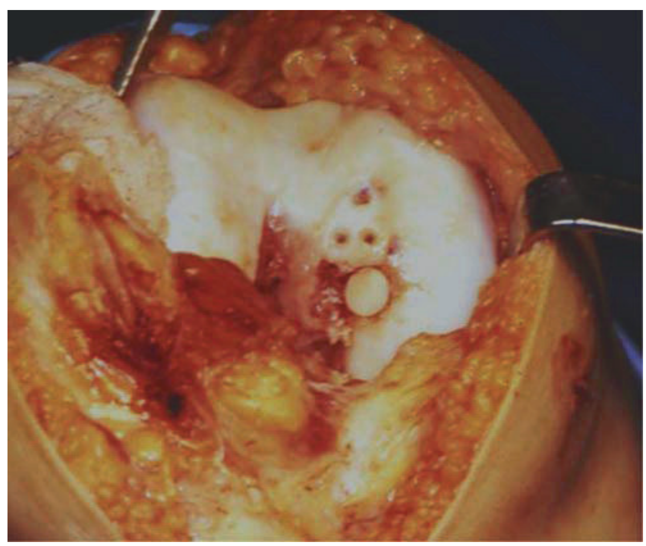


Fig. 45 Intraoperative view demonstrates hybrid treatment of a OCD. The nonviable portion of the OCD was replaced with a single osteochondral graft

Case Report

Case 5: Osteochondritis Dissecans

Chief Complaint/Patient History: The patient is a 17-year-old male athlete who has been experiencing left knee pain and crepitus for the past 6 years. Over the past few months, he has had several locking episodes. He attributes his symptoms to a football collision during which the knee impacted an opposing player. Five years ago, he underwent a microfracture procedure. It appears that he was rehabilitated appropriately, including a period of non-weight bearing (NWB). This surgery failed to provide any symptom relief. The patient is a division I collegiate hockey prospect and now experiences the majority of pain when skating in the knee-flexed position. The knee swells after hockey participation.

Physical Exam: The patient is 5′11″ tall, weighs 210 lb, and is a muscular male. There is small effusion and no meniscal joint-line tenderness. Alignment and ligament testing are normal. There is minor quadriceps atrophy with a 15-degree loss of flexion compared with the opposite knee. There is obvious posterior pain and catching sensation with deep flexion.

Imaging: See Fig. 46A, B.

Surgery/Treatment: The patient underwent a diagnostic arthroscopy and subsequent reduction and internal fixation of a lateral femoral condyle OCD via arthrotomy (Fig. 47A–D). Bone autograft was harvested from the proximal tibia and used. At the time, the author was using bioabsorbable screws for OCD fixation.

Postoperative Care/Follow-Up: The patient used TDWB for the initial 6-week postoperative period. CPM was initiated immediately, and traditional physiotherapy was initiated a week later. The patient continued to progress over the next 4 months. Despite recommendations from the author to refrain from competitive skating, the patient returned at 4½ months. At the 5½-month mark, he collided with an opponent at high speed, resulting in a direct impact to a flexed knee. This resulted in a dramatic knee effusion and pain (Fig. 48A, B).

Surgery/Treatment: The patient underwent left knee arthroscopy for removal of chondral fragments and loose screws (Fig. 49A–D). The bone graft had incorporated, which left the patient with a bleeding subchondral plate. Essentially, this was the equivalent of an abrasion chondroplasty.

Postoperative Care/Follow-Up: The patient was placed PWB with brace locked in extension to avoid loading graft for 6 weeks. Functional activity was progressed over the course of 2 months to include biking and elliptical trainer. At 5 months, free skating with no competition or impact was initiated. The knee at this point was essentially benign on exam with the exception of minor crepitus, and no effusion was appreciated. After 8 months, the patient returned to competitive skating, and there are no new issues to date.

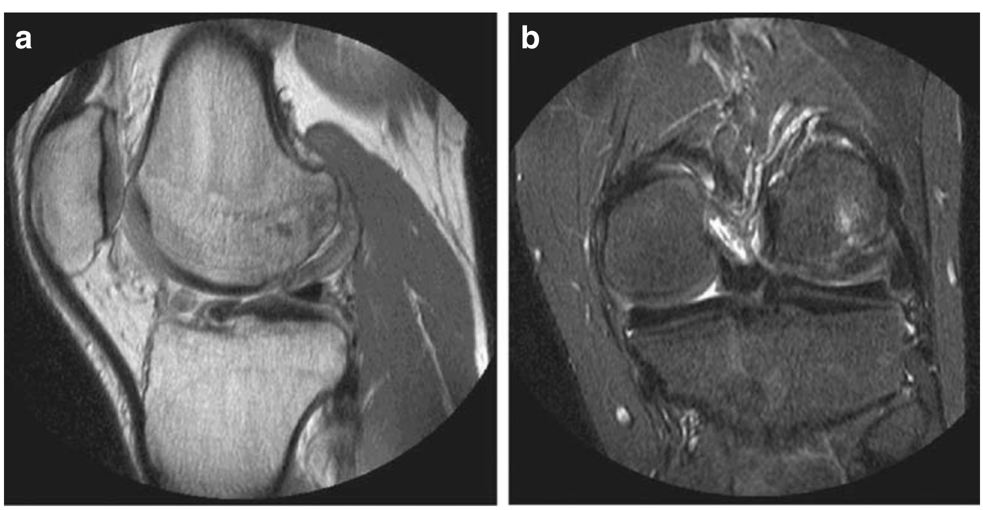


Fig. 46 (**A, B**) MRI views demonstrate an osteochondral flap that is 2.3 cm in length

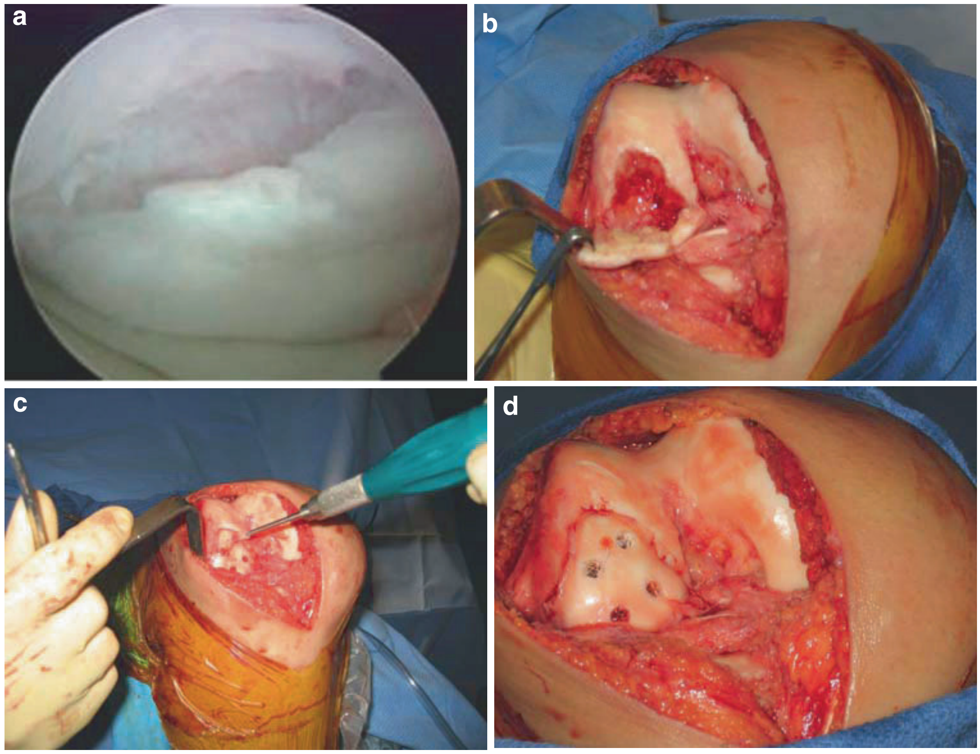


Fig. 47 (**A–D**) Diagnostic arthroscopy demonstrating the chondral defect and subsequent open grafting and open reduction and internal fixation (ORIF) of fragment using four bioabsorbable screws

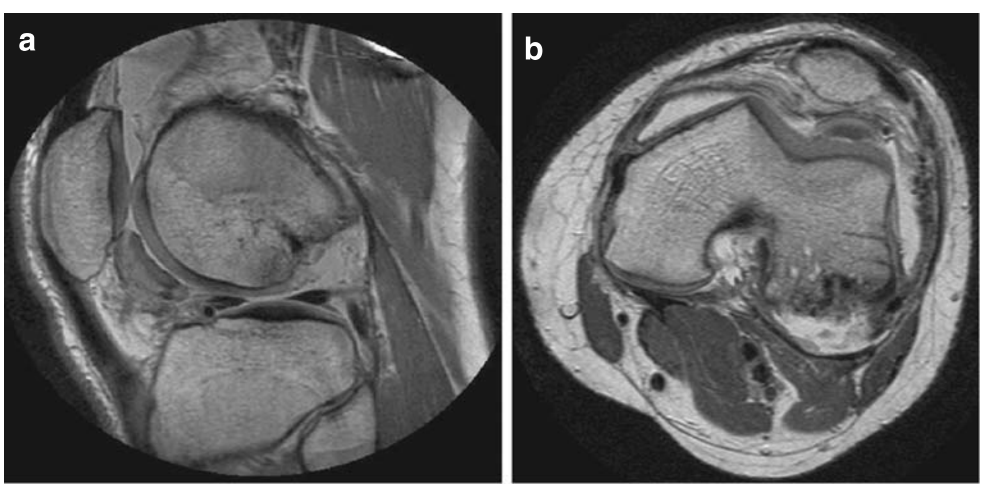


Fig. 48 (**A, B**) Posttraumatic MRI views demonstrate displaced chondral fragment located within the inferior aspect of the patellofemoral joint

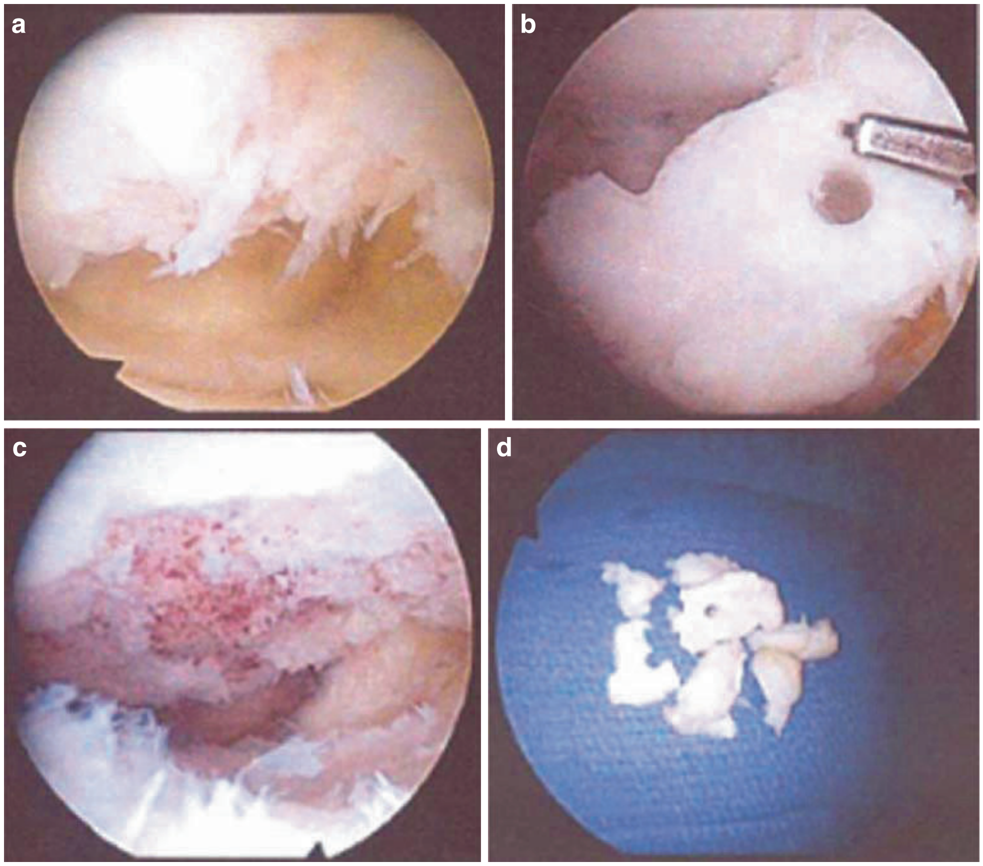


Fig. 49 (**A–D**) Intraoperative arthroscopic views demonstrate absent chondral surface of a posterior lateral OCD. The chondral fragments and screw remnants were removed. A crater at the region of the defect demonstrated healthy bleeding bone. The subchondral bone graft remained intact

Future of Cartilage Repair

There is no doubt that the autologous chondrocyte tissue engineered scaffolds that have become available for clinical use in Europe will eventually replace our techniques available in the United States. Gobbi et al. [135] reported excellent results with treatment of normally troublesome patellofemoral defects with a hyaluronan-based scaffold seeded with autologous chondrocytes. This mesh-like scaffold can be inserted arthroscopically or through a very small arthrotomy. The scaffold has bioadhesive properties; hence, no suturing is required. Twenty-four–month MRI follow-ups and second-look biopsies in six cases revealed "nearly normal" results. Hundreds of other bioscaffolds with added biomaterials are being studied throughout the world. The 2007 ICRS meeting in Warsaw, Poland, introduced several novel cell-seeded scaffolds, which confirms the resilient nature of the chondrocyte [40].

Summary

AC injuries will continue to challenge the orthopaedic discipline. However, recent awareness of the incidence and natural history has led to an explosion of research efforts to search for the ideal resurfacing method. Coordinated efforts between clinicians and basic scientists have brought this common goal within our grasp in the future. Cell-based repairs (ACT) appear to be the basis for the next generation of repair. However, a completely integrated repair tissue with hyaline differentiation has yet to be achieved. Delivery of important chondrogenic factors via gene transfer techniques may overcome important biological obstacles required to produce a true hyaline repair. Despite these shortcomings, current options discussed in this chapter can produce an acceptable clinical result in a properly selected patient.

References

1. Curl WW, Krome J, Gordon ES, Rushing J, Smith BP, Poehling CG. Cartilage injuries: a review of 31,516 knee arthroscopies. *Arthroscopy* 1997;13:456–460.
2. Hjelle K, Solheim E, Strand T, Muri R, Brittberg M. Articular cartilage defects in 1000 knee arthroscopies. *Arthroscopy* 2002;18:730–734.
3. Villalobos CFE Jr, Izaguirre A, Almazan A, Cruz F, Perez F, Ibarra JC. Articular cartilage injuries in 1309 knee arthroscopies: a public health problem in developing countries? ICRS Abstract Proceedings, 2007, 56.
4. Hunter W. On the structure and diseases of articulating cartilage. *Philos Trans R Soc Lond B Biol Sci* 1743;9:267.
5. Mankin HJ. The response of articular cartilage to injury. *J Bone Joint Surg Am* 1982;64:460–466.
6. Linden B. Osteochondritis dissecans of the femoral condyles: a long-term follow-up study. *J Bone Joint Surg Am* 1977;59: 769–776.
7. Messner K, Maletius W. The long-term prognosis for severe damage to weight-bearing cartilage in the knee: a 14-year clinical and radiographic follow-up in 28 young athletes. *Acta Orthop Scand* 1996;67:165–168.
8. Buckwalter JA, Hunziker EB. Articular cartilage morphology and biology. In: Archer CW, Caterson B, Benjamin M, Ralphs JR (eds.), *Biology of the Synovial Joint*. Amsterdam: Harwood Academic Publishers, 1999:75–100.
9. Genda E, Li G, Barrance PJ et al. Functional analysis of hip joint contact pressure. *Trans Orthop Res Soc* 1996;21:416.
10. Buckwalter JA, Hunziker EB. Articular cartilage biology and morphology. In Mow VC, Ratcliff A (eds.), *Biomechanics of Diarthrodial Joints*. New York: Springer-Verlag, 1993.
11. Malmonge SM, Arruda AC. Artificial articular cartilage: mechanoelectrical transduction under dynamic compressive loading. *Artif Organs* 2000;24:174–178.
12. Buckwalter JA. Articular cartilage: injuries and potential for healing. *J Orthop Sports Phys Ther* 1998;28:192–208.
13. Locker M, Kellermann O, Boucquey M, Khun H, Huerre M, Poliard A. Paracrine and autocrine signals promoting full chondrogenic differentiation of a mesoblastic cell line. *J Bone Miner Res* 2004;19:100–110.
14. Maroudas A, Bullough P, Swanson SAV, Freeman MAR. The permeability of articular cartilage. *J Bone Joint Surg Br* 1968;50:166–177.
15. Maroudas A. Physico-chemical properties of articular cartilage. In Mar F (ed.), *Adult Articular Cartilage*. Tunbridge Wells, England: Pitman Medical, 1979, pp. 215–290.
16. Sun DD, Guo XE, Likhitpanichkul M, Lai WM, Mow VC. The influence of the fixed negative charges on mechanical and electrical behaviors of articular cartilage under unconfined compression. *J Biomech Eng* 2004;126:6–16.
17. Likhitpanichkul M, Guo XE, Mow VC. The effect of matrix tension-compression nonlinearity and fixed negative charges on chondrocyte responses in cartilage. *Mol Cell Biomech* 2005;2:191–204.
18. Curl WW, Krome J, Gordon ES, Rushing J, Smith BP, Poeling GG. Cartilage injuries: a review of 31,516 knee arthroscopies. *Arthroscopy* 1997;13:456–460.
19. Aoren A, Loken S, Heir S, Alvik E, Ekeland A, Granlund OG, Engebretsen L. Articular cartilage lesions in 993 consecutive arthroscopies. *Am J Sports Med* 2004;32;211–215.
20. Sahlstrom A, Johnell O, Redlund-Johnell I. The natural course of arthrosis of the knee. *Clin Orthop* 1997;340:152–157.
21. Messner K, Maletius W. The long-term prognosis for severe damage to weight-bearing cartilage in the knee: a 14-year clinical and radiographic follow-up in 28 young athletes. *Acta Orthop Scanda* 1996;67:165–168.
22. Linden B. Osteochondritis dessicans of the femoral condyles: a long–term follow-up study. *J Bone Joint Surg Am* 1977;59: 769–776.
23. Rudd RG, Visco DM, Kincaid SA, Cantwell HD. The effects of beveling the margins of articular defects in mature dogs. *Vet Surg* 1987;16:378–383.
24. Simonian PT, Sussmann PS, Wickiewicz TL, Paletta GA, Warren EF. Contact pressures at osteochondral donor sites in the knee. *Am J Sports Med* 1998;26:491–494.
25. Cicuttini FM, Forbes A, Yuanyuan W, Rush G, Stuckey SL. Rate of knee cartilage loss after partial menisectomy. *J Rheumatol* 2002;29:1954–1956.
26. Mandelbaum BR, Brown JE, Fu F, Micheli L, Mosely JB Jr, Erggelet C, Minas T, Peterson L. Articular cartilage lesions of the knee. *Am J Sports Med* 1998;26:853–861.
27. Outerbridge RE. The etiology of chondromalacia patellae. *J Bone Joint Surg Br* 1961;43:752–757.
28. Lewandrowski KU, Ekkernkamp A, David A, Muhr G, Schollmeier G. Classification of articular lesions of the knee at arthroscopy. *Am J Knee Surg* 1996;9:121–128.
29. Hunt N, Sanchez-Ballester J, Pandit R, Thomas R. Strachan R. Chondral lesions of the knee: a new localization and correlation with associated pathology. *Arthroscopy* 2001;17;481–490.
30. Brittberg M. Evaluation of cartilage injuries and cartilage repair. *Osteologie* 2000;9:17–25.
31. Blackburn WD, Bernreuter WK, Rominger M, Loose LL. Arthroscopic evaluation of knee articular cartilage: a comparison with plain radiographs and MRI. *J Rheumatol* 1994;21: 675–679.
32. Ochi M, Sumen Y, Kanda T, Ikuta Y, Itoh K. The diagnostic value and limitation of magnetic resonance imaging on chondral lesions in the knee joint. *J Arthrosc* 1994;10:176–183.
33. Potter H, Linklater JM, Allen AA, Hannafin JA, Haas SB. Magnetic resonance imaging of articular cartilage in the knee. An evaluation with use of fast spin echo imaging. *J Bone Joint Surg Am* 1998;80:1276–1284.
34. Oei EH, Nikken JJ, Verstijnen AC, Ginai AZ, Hunink MGM. MR imaging of the menisci and cruciate ligaments: a systematic review. *Radiology* 2003;226:837–848.
35. Bashir A, Gray ML, Hartke J, Burstein D. Nondestructive imaging of human cartilage glycosaminoglycan concentration by MRI. *Magn Reson Med* 1999;41:857–865.
36. Williams A, Gillis A, McKenzieC, Po B, Sharma L, Micheli L, McKeon, B, Burstein B. Glycosaminglycan distribution in cartilage as determined by delayed gadolinium-enhanced MRI of cartilage (dGEMRIC): potential clinical applications. *Am J Radiol* 2004;182:167–172.
37. Young AA, Stanwell P, Williams A, Rohrsheim JA, Parker DA, Giuffre B, Ellis AM. Glycosaminaglycan content of knee cartilage following PCL rupture demonstrated by delayed gadolinium-enhanced MRI of cartilage (dGEMRIC): a case report. *J Bone Joint Surg Am* 2005;87:2763,2767.
38. Gillis A, Bashir A, McKeon B, Scheller A, Gray M, Burstein D. Magnetic resonance imaging of relative glycosaminoglycan distribution in patients with autologous chondrocyte transplants. *Invest Radiol* 2001;36:743–748.
39. Rosenberg T, Paulos L, Parker R, Coward DB, Scott SM. The forty-five-degree posterioanterior flexion weight-bearing radiograph of the knee. *J Bone Joint Surg Am* 1988;70: 1479–1483.
40. International Cartilage Repair Society, ICRS recommended MRI protocol for articular cartilage, www.cartilage.org
41. Khetia E, McKeon BP. Meniscal allografts: biomechanics and techniques. *Sports Med Arthrosc* 2007;15:114–120.

42. Gross AE. Fresh osteochondral allografts for post-traumatic knee defects: surgical technique. *Oper Tech Orthop* 1997;7: 334–339.
43. Alford J, Cole B. Cartilage restoration, part 2. *Am J Sports Med* 2005;33:443–460.
44. Brittberg M, Peterson L, Sjogren-Jansson E, Tallheden D. Linddal A. Articular cartilage engineering with autologous chondrocyte transplantation: a review of recent developments. *J Bone Joint Surg Am* 2003;85(Suppl 3):109–115.
45. Mandelbaum B, Browne J, Fu F, Micheli LJ, Moseley B Jr, Erggelet C, Anderson AF. Treatment outcomes of autologous chondrocyte implantation for full-thickness articular cartilage defects of the trochlea. *Am J Sports Med* 2007;35:915–921.
46. Farr J, Schepsis A, Cole B, Fulkerson J, Lewis B. Anteromedialization: review and technique. *J Knee Surg* 2007;20:120–128.
47. Mihalko W, Boachie-Adjei Y, Spang J, Fulkerson JP, Arendt EA, Saleh KA. Controversies and techniques in the surgical management of patellofemoral arthritis. *J Bone Joint Surg Am* 2007;89:2788–2802.
48. Magnusson PB. Technique of debridement of the knee joint for arthritis. *Surg Clin North Am* 1946;26:226–249.
49. Jackson RW, Marans HJ, Silver RS. Arthroscopic treatment of degenerative arthritis of the knee. *J Bone Joint Surg Br* 1988;70:332.
50. Hubbard MJ. Articular debridement versus washout for degeneration of the medial femoral condyle. A five-year study. *J Bone Joint Surg Br* 1996;78:217–219.
51. Merchan EC, Galindo E. Arthroscope-guided surgery versus nonoperative treatment for limited degenerative osteoarthritis of the femorotibial joint in patients over 50 years of age: a prospective comparative study. *Arthroscopy* 1993;9:663–667.
52. Chang RW, Falconer J, Stulberg SD. A randomized, controlled trial of arthroscopic surgery versus closed-needle joint lavage for patients with osteoarthritis of the knee. *Arthritis Rheum* 1993;36:289–296.
53. Ike RW, Arnold WJ, Rothschild EW, Shaw HL. Tidal irrigation versus conservative medical management in patients with osteoarthritis of the knee: a prospective randomized study. Tidal Irrigation Cooperating Group. *J Rheumatol* 1992;19:772–779.
54. Moseley JB, O'Malley K, Petersen NJ, Menke TJ, Brody BA, Kuykendall DH, Hollingsworth JC, Ashton CM, Wray NP. A controlled trial of arthroscopic surgery for osteoarthritis of the knee. *N Engl J Med* 2002;347:81–88.
55. Pirdie KH. A method of resurfacing knee joints. *J Bone Joint Surg Br* 1959;41:618–619.
56. Freedman KB, Nho SJ, Cole BJ. Marrow stimulating technique to augment meniscus repair. *Arthroscopy* 2003;19:794–798.
57. Buckwalter JA, Mow VC, Ratcliffe A. Restoration of injured or degenerated articluar cartilage. *J Am Acad Orthop Surg* 1994;2:192–201.
58. Athanasiou KA, Rosenwasser MP, Spiker RL, et al. Effects of passive motion on the material properties of healing articular cartilage. *Trans Orthop Res Soc* 1991;15:156.
59. Bert JM, Maschka K. The arthroscopic treatment of unicompartmental gonarthrosis: a five year follow-up of abrasion arthroplasty plus arthroscopic debridement and arthroscopic debridement alone. *Arthroscopy* 1989;5;25–32.
60. Steadman JR, Rodkey WG, Singleton SB. Microfracture technique for full thickness chondral defects: Technique and clinical results. *Oper Tech Orthop* 1997;7:300–304.
61. Steadman JR, Briggs KK, Rodrigo JJ, Kocher M, Gill T, Rodkey W. Outcomes of microfracture for traumatic chondral defects of the knee. Average 11-year follow-up. *Arthroscopy* 2003;19: 477–484.
62. Mithoefer K, Williams R, Warren R, Wickiewicz TL, Marx RG. High-impact athletics after knee articular cartilage repair: a prospective evaluation of the microfracture technique. *Am J Sports Med* 2006;34:1413–1418.
63. Kreuz P, Erggelet C, Steinwachs MR, Krause S, Lahm A, Niemeyer P, Ghanem N, Uhl M, Südkamp N. Is microfractue of chondral defects in the knee associated with different results in patients aged 40 years or younger? *Arthroscopy* 2006;26: 1180–1186.
64. Frisbie DD, Morisset S, Ho CP, Rodkey WF, Steadman JR, McIlwraith CW. Effects of calcified cartilage on healing of chondral defects treated with microfracture in horses. *Am J Sports Med* 2006;34:1824–1831.
65. Mankin H. The response of articular cartilage to mechanical injury. *J Bone Joint Surg Am* 1982;64:460–465.
66. Rodrigo J, Steadman JR. Improvement of full-thickness chondral defect healing in the human knee after debridement and microfracture using continuous passive motion. *Am J Knee Surg* 1994;7:109–116.
67. Salter RB. The biologic concept of continuous passive motion of synovial joints. The first 18 years of basic research and its clinical application. *Clin Orthop* 1989;242:12–25.
68. Salter RB, Simmons DF, Malcolm BW, Rumble EG, MacMichael D, Clements ND. The biological effect of continuous passive motion on the healing of full-thickness defects in articular cartilage. *J Bone Joint Surg Am* 1980;62:1232–1250.
69. Wilk KE, Briem K, Reinold MM, Devine KA, Dugas J, Andrews JR Rehabilitation of articular lesions in the athlete's knee. *J Sports Phys Ther* 2006;36:815–827.
70. Gill TJ, Asnis P, Berkson E. The treatment of articular defects using the microfracture technique. *J Sports Phys Ther* 2006;36:728–738.
71. Judet H. Essai sur la greffe des tissues articulaires. *C R Acad Sci III* 1908;146:193–196, 600–603.
72. Hangody L, Karpati Z, Toth J, et al. Autogenous osteochondral grafting in the knee of German shepherd dogs: radiographic and histologic analysis. *Hung Rev Sports Med* 1994;37: 237–243.
73. Lane JG, Tontz WL, Ball ST, Massie JB, Chen AC, Bae WC, Amiehl ME, Sah RL, Amiehl D. A morphologic, biochemical, and biomechanical assessment of short-term effects of osteochondral autograft plug transfer in an animal model. *Arthroscopy* 2001;17:856–863.
74. Garret JC. Fresh osteochondral allografts for treatment of articular defects in osteochondritis dessicans of the lateral femoral condyle in adults. *Clin Orthop* 1994;303:33–37.
75. Bugbee WD. Fresh osteochondral allografts. *Semin Arthroplasty* 2000;11:1–7.
76. Ghazavi MT, Pritzker RP, Davis AM, Gross EA. Fresh osteochondral allografts for popsttraumatic osteochondral defects of the knee. *J Bone Joint Surg Br* 1997;79:1008–1113.
77. Duchow J, Hess T, Kohn D. Primary stability of press-fit-implanted osteochondral grafts: influence of graft size, repeated insertion, and harvesting technique. *Am J Sports Med* 2000;28:24–27.
78. Gudas R, Stankevicius E, Monastyreckiene E, Pranys D, Kalesinskas, RJ. Osteochondral autologous transplantation versus microfracture for the treatment of articular cartilage defects in the knee joint in athletes. *Knee Surg Traumatol Arthrosc* 2006;14:834–842.
79. Hangody L, Fules P. Autologous osteochondral mosaicplasty for the treatment of full thickness defects of weight-bearing joints: ten years experimental and clinical experience. *J Bone Joint Surg Am* 2003;85:25–32.
80. Pearce SG, Hurtig MB, Clarnette R, Kalra M, Cowan B, Miniaci A. An investigation of 2 techniques for optimizing joint surface congruency using multiple cylindrical osteochondral autografts. *Arthroscopy* 2001;17:50–55.

81. Makino T, Fujioka H, Kurosaka M, Matsui N, Yoshihara H, Tsunoda M, Mizuno K. Histological analysis of the implanted cartilage in an exact-fit osteochondral transplantation model. *Arthroscopy* 2001;17:747–751.
82. Diduch DR, Chhabra A, Blessey P, Miller MD. Osteochondral autograft plug transfer: achieving perpendicularity. *J Knee Surg* 2003;16:17–20.
83. Bobic V. Arthroscopic osteochondral autogenous graft transplantation in anterior cruciate reconstruction: a preliminary report. *Knee Surg Sports Traumatol Arthrosc* 1996;3:262.
84. Simonian PT, Sussman PS, Wickiewicz TL, Paletta GA, Warren RF. Contact pressures at osteochondral donor sites in the knee. *Am J Sports Med* 1998;26:491–494.
85. Garretson RB III, Katolik LI, Verma N, Beck PR, Bach BR, Cole BJ. Contact pressure at osteochondral donor sites in the patellofemoral joint. *Am J Sports Med* 2004;32: 967–974.
86. Cain EL, Clancy WG. Treatment algorithm for osteochondral injuries of the knee. *Clin Sports Med* 2001;20:321–342.
87. Hunziker EB, Quinn TM. Surgical removal of articular cartilage leads to loss of chodrocytes from cartilage bordering the wound edge. *J Bone Joint Surg Am* 2003;85(Suppl 2): 85–92.
88. Matsusue Y, Yamamuro T, Hama H. Arthroscopic multiple osteochondral transplantation to the chondral defect in the knee associated with anterior cruciate ligament disruption. *Arthroscopy* 1993;9:318–321.
89. Whiteside RA, Jakob RP, Wyss UP, Mainil-Varlet P. Impact loading of articular cartilage during transplantation of osteochondral autograft. *J Bone Joint Surg Br* 2005;87:1285–1291.
90. Reinold MM, Wilk K, Macrina L, Dugas JR, Cain EL. Current concepts in rehabilitation following articular cartilage repair procedures in the knee. *JOSPT* 2006;10:774–814.
91. Maury AC, Safir O, Las Heras F, Pritzker KP, Gross AE. Twenty-five year chondrocyte viability in fresh osteochondral allograft: a case report. *J Bone Joint Surg Am* 2007;89:159–165.
92. Pearsall AW 4th, Tucker JA, Hester RB, Heitman RJ. Chondrocyte viability in refrigerated osteochondral allografts used for transplantation within the knee. *Am J Sports Med* 2004;32: 125–131.
93. Gross AE, Silverstein EA, Falk J, Falk R, Langer F. The allotransplantation of partial joints in the treatment of osteoarthritis of the knee. *Clin Orthop Relat Res* 1975;108:7–14.
94. McDermott AG, Langer F, Pritzker KP, Gross AE. Fresh small-fragment osteochondral allografts. Long-term follow-up study on first 100 cases. *Clin Orthop Relat Res* 1985;197:96–102.
95. McGovernan BM, Pritzker KP, Shasha N, Price J, Gross AE. Long-term chondrocyte viability in osteochondral allograft. *J Knee Surg* 2002;15:97–100.
96. Standards for tissue banking. Arlington, VA: American Association of Tissue Banks, 1987.
97. Williams R, Ranawat A, Potter H, Carter T, Warren RF. Fresh stored allografts for the treatment of osteochondral defects of the knee. *J Bone Joint Surg Am* 2007;89:718–726.
98. Davidson P, Rivenburgh D, Dawson P, Rozin R. Clinical, histologic, and radiographic outcomes of distal femoral resurfacing with hypothermically stored osteoarticular allografts. *Am J Sports Med* 2007;35:1082–1090.
99. Brittberg M, Lindahl A, Nilsson A, Ohlsson C, Isaksson O, Peterson L. Treatment of deep cartilage defects in the knee with autologous chondrocyte implantation. *New Engl J Med* 1994;331: 889–895.
100. Peterson L, Brittberg M, Kiviranta I, Akerlund EL, Lindhal A. Autologous chondrocyte transplantation: biomechanics and long-term durability. *Am J Sports Med* 2002;30:2–12.
101. Vasara AI, Nieminen MT, Jurvelin JS, Peterson L, Lindahl A, Kiviranta I. Indentation stiffness of repair tissue after autologous chondrocyte transplantation. *Clin Orthop Relat Res* 2005;433:233–242.
102. Knutsen G, Drogset J, Engebretsen L, Grøntvedt T, Isaksen V, Ludvigsen TC, Roberts S, Solheim E, Strand T, Johansen O. A randomized trial comparing autologous chondrocyte implantation with microfracture. Findings at five years. *J Bone Joint Surg Am* 2007;89:2105–2112.
103. Henderson I, Lavigne P, Valenzuela H, et al. Autologous chodrocyte implantation. *Clin Orthop Relat Res* 2006;455:253–261.
104. Nehrer S, Spector M, Minas T. Histologic analysis of tissue after failed cartilage repair procedures. *Clin Orthop Relat Res* 1999 Aug;(365):149–62.
105. Peterson L, Brittberg M, Kiviranta I, Akerlund EL, Lindahl A. Autologous chondrocyte transplantation, biomechanics, and long-term durability. *Am J Sports Med* 2002;30:2–12.
106. Peterson L, Minas T, Brittberg M, Nilsson A, Sjogren-Jansson E, Lindahl A. Two- to 9-year outcome after autologous chondrocyte transplantation of the knee. *Clin Orthop Relat Res* 2000;374: 212–234.
107. Gillogly SD, Voight M, Blackburn T. Treatment of articular cartilage defects of the knee with autologous chondrocyte implantation. *J Orthop Sports Phys Ther* 1998;28:241–251.
108. Mandelbaum B, Browne J, Fu F, Moseley JB Jr, Erggelet C, Anderson AF. Treatment outcomes of autologous chondrocyte implantation for full-thickness articular defects of the trochlea. *Am J Sprots Med* 2007;35:915–922.
109. Mithofer K, Peterson L, Mandelbaum BR, Minas T. Articular cartilage repair in soccer players with autologous chondrocyte transplantation: functional outcome and return to competition. *Am J Sports Med* 2005;33:1639–1646.
110. Mithofer K, Minas T, Peterson L, Yeon H, Micheli LJ. Functional outcome of knee articular cartilage repair in adolescent athletes. *Am J Sports Med* 2005;33:1147–1153.
111. Kreuz P, Steinwachs M, Erggelet C, Lahm A, Krause S, Ossendorf C, Meier D, Ghanem N, Uhl M. Importance of sports in cartilage regeneration after autologous chondrocyte implantation. *Am J Sports Med* 2007;35:1261–1268.
112. Roberts S, McCall IW, Darby AJ, Menage J, Evans H, Harrison PE, Richardson JB. Autologous chondrocyte implantation for cartilage repair: monitoring its success by magnetic resonance imaging and histology. *Arthritis Res Ther* 2003;5:R60–R73.
113. Schenk R, Goodnight JM. Current concepts review: osteochondritis dissecans. *J Bone Joint Surg Am* 1996;78:439–456.
114. Garrett JC. Osteochondritis dissecans**.** *Clin Sports Med* 1991;10:569–593.
115. Cugat R, Garcia M, Cusco X, Monllau JC, Vilaro J, Juan X, Ruiz-Cottoro A. Osteochondritis dissecans: a historical review and its treatment with cannulated screws. *Arthroscopy* 1993;9: 675–684.
116. Fairbank H. Osteochondritis dissecans. *Br J Surg* 1933;21:67–82.
117. Linden B. Osteochondritis dissecans of the femoral condyles: a long-term follow-up study. *J Bone Joint Surg Am* 1977;59: 769–776.
118. Ewing JW, Voto SJ. Arthroscopic surgical management of osteochondritis dissecans of the knee. *Arthroscopy* 1988;4:37–40.
119. Kocher MS, DiCanzio J, Zurakowski D, Micheli LJ. Diagnostic performance of clinical examination and selective magnetic resonance imaging in the evaluation of intraarticular knee disorders in children and adolescents. *Am J Sports Med* 2001;29: 292–296.
120. Kocher MS, Tucker R, Ganley T, Flynn JM. Management of osteochondritis dissecans of the knee. *Am J Sports Med* 2006;34:1181–1190.

121. Cahill BR. Osteochondritis dissecans of the knee: treatment of juvenile and adult forms. *J Am Acad Orthop Surg* 1995;3: 237–247.
122. Linden B. Osteochondritis dissecans of the femoral condyles: a long-term follow-up study. *J Bone Joint Surg Am* 1977;59: 769–776.
123. De Smet AA, Fisher DR, Graf BK, Lange RJ. Osteochondritis dissecans of the knee: value of MR imaging in determining lesion stability and the presence of articular cartilage defects. *AJR Am J Roentgenol* 1990;155:549–553.
124. Lebolt J, Wall E. Retrograde drilling and bone grafting of juvenile osteochondritis dissecans of the knee. *Arthroscopy* 2007;23:794e1–794e4.
125. Bradley J, Dandyy D. Results of drilling osteochondritis dissecans before skeletal maturity. *J Bone Joint Surg Br* 1989;71: 642–644.
126. Kocher M, Czarnecki J, Andersen J, Micheli LJ. Internal fixation of juvenile osteochondritis dissecans lesions of the knee. *Am J Sports Med* 2007;35:712–718.
127. Johnson LL, Uitvlugt G, Austin MD, Detrisac DA, Johnson C. Osteochondritis dissecans of the knee: arthroscopic compression screw fixation. *Arthroscopy* 1990;6:179–189.
128. Zuniga RSJ, Blasco L, Grande M. Arthroscopic use of Herbert screws in osteochondritis dissecans of the knee. *Arthroscopy* 1993;9:668–670.
129. Dervin GF, Keene GC, Chissell HR. Biodegradable rods in adult osteochondritis dissecans of the knee. *Clin Orthop Relat Res* 1998;356:213–221.
130. Friederichs MG, Greis PE, Burks RT. Pitfalls associated with fixation of osteochondritis dissecans fragments using bioabsorbable screws. *Arthroscopy* 2001;17:542–545.
131. Scioscia TN, Giffin JR, Allen CR, Harner CD. Potential complication of bioabsorbable screw fixation for osteochondritis dissecans of the knee. *Arthroscopy* 2001;17:E7.
132. Miura K, Ishibashi Y, Tsuda E, Sato H. Toh. S. Results of arthroscopic fixation of osteochondritis dissecans lesions of the knee with cylindrical autogenous osteochondral plugs. *Am J Sports Med* 2007;35:216–222.
133. Emmerson B, Gortz S, Jamali A, Chung C, Amiehl D, Bugbee WD. Fresh osteochondral grafting in the treatment of osteochondritis dissecans of the femoral condyle. *Am J Sports Med* 2007;35:907–914.
134. Anderson AF, Pagnani MJ. Osteochondritis dissecans of the femoral condyles: long-term results of excision of the fragment. *Am J Sports Med* 1997;25:830–834.
135. Gobbi A, Kon E, Berruto M, Francisco R, Filardo G, Marcacci M. Patellofemoral full-thickness chondral defects treated with hyalograft-C: a clinical, arthroscopic, and histologic review. *Am J Sports Med* 2006;34:1763–1773.

Patellofemoral Disorders

Jeffrey T. Spang and John P. Fulkerson

Introduction

Patients presenting with patellofemoral pain represent a challenging population in any orthopaedic practice. Most patients can be successfully managed with a systematic nonoperative program. Patient selection is the key to achieving successful surgical outcomes. A thorough understanding of patellofemoral anatomy coupled with a complete patient history and physical will increase the chances of success with arthroscopic intervention. A variety of pathologic conditions exist in the patellofemoral joint, including cartilage disease, malalignment, instability, arthritis, and soft tissue disorders. Arthroscopy may be the first line of surgical treatment for a complicated problem or the definitive solution to a complex issue. A clear preoperative understanding of the potential pathology that might be encountered at the time of arthroscopy will assist in correctly applying one of several arthroscopic surgical procedures.

Anatomy

The goal of the surgeon treating anterior knee pain should be to understand the main structural sources of pain (synovium, retinaculum, nerves, muscle, and subchondral bone) in the setting of the complex mechanics of the patellofemoral joint. When the balance of the patellofemoral joint is disturbed, nerve damage and pain may result. Fulkerson et al. first described retinacular small nerve neuromatous degeneration as an important cause of anterior knee pain [1]. Biedert et al. [2] later confirmed that free nerve endings are concentrated in the patellar tendon, retinacular tissue, synovial tissues, and fat pad and can be a significant source of pain in the anterior knee by elaborating neurokinins. Subchondral bone has also been described as richly innervated [3]. Excessive pressure or tension on any of these structures can lead to patellofemoral problems, which may require arthroscopic evaluation. The anatomy of the patellofemoral joint must be viewed as a system in motion with loads increasing and decreasing on different structures through the knee flexion cycle.

Both active and passive restraints provide the balance required for normal patellofemoral tracking. The rectus femoris and vastus intermedius muscles act along the femoral axis, and the vastus lateralis and vastus medialis act in oblique planes to exert medial and lateral forces on the patella [4]. Passive restraints contributing to a properly balanced patella include the medial and lateral retinaculum and the patellar tendon (Figs. 1 and 2). The lateral retinaculum

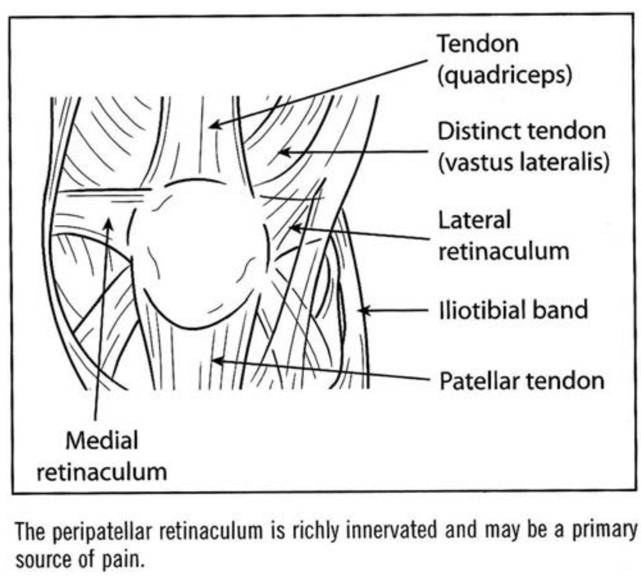


Fig. 1 Forces that must be in balance for proper patellar alignment. (Modified from Fulkerson JP. Patellofemoral realignment: principles and guidelines. In Fulkerson JP (ed.), *Common Patellofemoral Problems.* Rosemont, IL: American Academy of Orthopaedic Surgeons, 2005, pp. 11–17. Modified with permission.)

J.T. Spang (✉)
Department of Orthopaedic Surgery, University of North Carolina School of Medicine, Chapel Hill, North Carolina

B.P. McKeon et al. (eds.), *Knee Arthroscopy*, DOI 10.1007/978-0-387-89504-8_7,

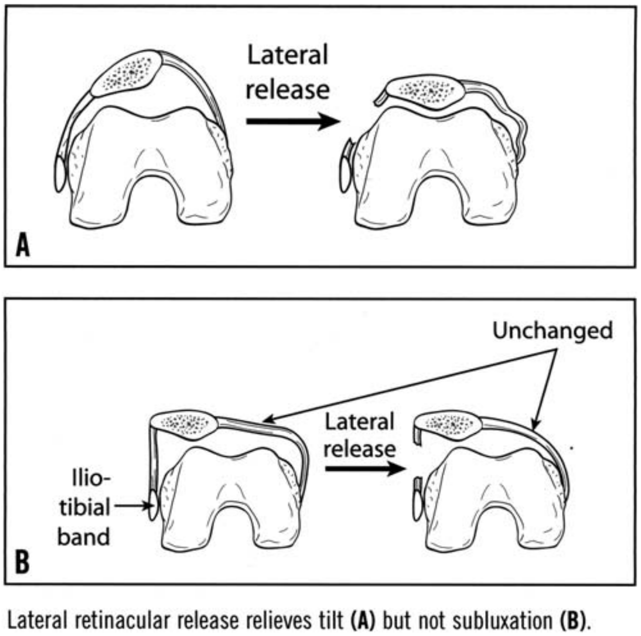


Fig. 2 Effect of imbalance of the lateral retinacular tissues and the possibility of force redistribution with lateral release. (Modified from Fulkerson JP. Patellofemoral realignment: principles and guidelines. In Fulkerson JP (ed.), *Common Patellofemoral Problems.* Rosemont, IL: American Academy of Orthopaedic Surgeons, 2005, pp. 11–17. Modified with permission.)

is a complex structure with two major components: the deep transverse retinaculum and the superficial oblique retinaculum [5]. The medial retinaculum is composed of the medial meniscopatellar ligament and the medial patellofemoral ligament (MPFL) [6–8]. The medial structures, in particular the MPFL, provide the critical restraint to lateral patellar movement while the patella sits out of the trochlea during the early stages of flexion [9]. During early knee flexion, the MPFL acts to draw the patella into the trochlea. Once the patella is contained by the trochlea, tension on the MPFL decreases with further flexion [10]. The majority of patellofemoral stability is derived from the bony surroundings of the trochlea after about 30–45 degrees of knee flexion. If the trochlea is dysplastic, the lateral condyle does not adequately contain the patella, and the demands on the medial retinacular structures will increase. Excessive hip internal rotation can also lead to increased forces acting on the static medial restraints. With the trauma of a dislocation or over time in the malaligned individual, the MPFL may become biomechanically unsound and fail to deliver the patella adequately into the trochlea.

Different areas of the patella contact the femur during flexion. At the start of flexion, the inferior pole of the patella contacts the trochlea. With increasing flexion of the knee, the area of patella femoral contact moves more proximally on the patella [4]. As flexion reaches 120 degrees, only the most medial and lateral aspects of the patella contact the femoral condyles. Increasing flexion also increases the area of contact between the patella and the trochlea.

Before attempting an arthroscopic intervention, the orthopaedist must understand the broader concept of the balanced forces at work on the patella through the range of motion. Not unlike the shoulder, patellar stability is provided by both bony and ligamentous restraints, with supplemental support from muscular actions. The arthroscope is a powerful tool that may be used to restore balance to patellar tracking and improve tissue homeostasis.

Patient History

As with any history, the main focus should be the patient's perception of the disability. Important questions include the quality of pain experienced, the frequency, and any inciting events. It is critical to establish whether the pain resulted from a specific injury or spontaneously appeared [11]. Specific traumatic events should be explored in detail to establish their relationship to current symptoms. Blunt trauma may, for example, be the inciting cause of cartilage destruction that causes ongoing pain.

Once basic details have been obtained, patellofemoral symptoms may be broadly grouped into two categories: pain and pain with symptomatic instability [3]. Whereas pain generally accompanies patellar instability, the fundamental pathologic event associated with patellar instability is subluxation or dislocation of the patella. Instability events may require a reduction or may spontaneously reduce, but the common thread is a movement of the patella off the articulation with the femur, most often toward the lateral side. Such events are typically associated with weight-bearing and torsional trauma, but they may require less precipitating mechanical circumstances with recurrent events. It is important to rule out secondary causes of "knee giving way" such as quadriceps inhibition secondary to pain, deconditioning, or effusion, which may masquerade as instability [12]. Other common causes of anterior knee pain should be explored before focusing on the patellofemoral joint. Both patellar and quadriceps tendonitis can be mistaken for more complicated diagnostic entities, but a focused soft tissue examination of the knee should expose these problems.

Once instability has been ruled out as the primary problem, the clinician's attention may turn to other causes of patellofemoral pain. Ongoing pain is often ascribed to malalignment within the patellofemoral system, but a careful examiner should be able to discover how increasing demands may have stressed a system that previously was quite capable of functioning without pain. If no discrete injury can be established, recent increases in activity levels should be explored as potential causes of overuse phenomena. In such an overuse scenario, the examiner must establish if the pain or disability was present prior to the increase in functional demands. Equally important is the recognition of patellofemoral pain that arrives as an entirely new problem related to new demands on the joint.

The location of the pain is also a vital component of correct diagnosis, and research has shown that patient-generated "pain maps" may help narrow the anatomic focus of any examination [13]. Listening carefully to the patient will yield vital information. Is the pain vague and diffuse or sharp and well localized? Is the pain "deep" or behind the patella? Is it intermittent or constant? To what extent does activity influence the pain? If the pain is associated with activity, it is important to gather as much detail as possible about the activity in questions (i.e., flexion angle of the knee that elicits maximum pain, pain after running, pain while coming down stairs but not up, etc.) [11].

Another vital component of a complete history is review of prior surgical procedures. Such procedures may be patellofemoral related (lateral release; chondroplasty) or they may be general knee procedures (anterior cruciate ligament [ACL] reconstruction with patellar tendon graft; meniscectomy). It is important to establish the type of pain and disability that existed before any surgical procedures and to further define how symptoms changed after a procedure, especially if prior surgical procedures were targeted at the patellofemoral joint. Prior operative notes and any available presurgical imaging can be invaluable in assessing a postoperative patient.

Finally, be aware of other medical issues that may manifest as patellofemoral joint or anterior knee pain. Consideration must be given to inflammatory arthropathies, metabolic disorders, and infections such as Lyme disease that may masquerade as knee pain [12]. At the close of the history, patient expectations about desired activities and work requirements should be explored to ensure that both patient and surgeon maintain realistic goals.

Physical Examination

Recommended references for the patellofemoral exam include the clinical approaches to patients with patellofemoral disorders written by Post [3, 12]. Important points regarding the general examination include the following:

1. The primary goal is to appreciate factors that influence articular and retinacular forces and alignment.
2. Physical exam maneuvers often have no "normal" value, so assessing the contralateral (if asymptomatic) leg provides a valuable reference.
3. Measurable malalignment may or may not be a key factor in the specific disorder experienced by the patient. Malalignment may exist asymptomatically and may be important only in the presence of injury, repetitive overload, or neuromuscular decompensation.

With these principles in mind, a careful physical examination is the primary tool in detecting pathology that may be amenable to arthroscopic intervention.

Observation of the knee may reveal skin changes, calluses from kneeling, surgical scars, or alterations of skin color. Standing alignment of the legs should be viewed from both the front and back of the patient, taking care to evaluate foot pronation [11]. Excessive pronation may contribute to patellofemoral malalignment [14]. Gait patterns should be evaluated while visualizing the entire limb. While the patient is standing, core stability and hip strength may be assessed. Weakness of the pelvic stabilizers and hip external rotators has been implicated in functional internal rotation of the femur, which can contribute to patellar maltracking and pain. Asking the patient to lift the unaffected limb off the ground and then perform a simple one-legged squat can reveal excessive hip internal rotation and pelvic instability [7, 11, 15]. Once corrected, these muscle imbalances may dramatically improve symptoms. A step-down, step-up test may be conducted in the office with a standing stool. The patient should step-down and then step-up with the unaffected knee first, then reverse the order. Pain in the affected leg in early flexion may indicate a distal pole lesion, contacting the femur as the patella enters the trochlea.

Once the patient is supine, palpation of the knee can reveal hypersensitivity. It is especially important to explore prior surgical areas for potential neuromas. Examine the knee in both the flexed and extended positions. View the patella as the knee flexes to determine if it centralizes early in flexion. Palpation of the patella during flexion and extension of the knee can detect crepitation that is linked to pain. A painful plica may be palpated during these cycles, particularly on the medial side [16]. If cartilage lesions are suspected, the patella may be loaded with the knee in any fixed position of flexion to try and determine the location of damage. If the pain is greater near extension, this may indicate a lesion in the inferior pole of the patella, whereas pain with loading of the patella in greater flexion may indicate a proximal patellar lesion [17]. Patients with a history of prior lateral release or prior medial soft tissue procedures should be carefully evaluated for medial subluxation. With the knee extended, displace the patella medially. Flex the knee while maintaining a medially directed force. In cases of medial subluxation, this produces a disabling, painful reaction [18].

The lateral retinacular structures should be thoroughly evaluated. Patella mobility should be carefully examined, with emphasis on patellar tilt. In general, the lateral patella border should be free enough to be elevated above the neutral axis of the knee (parallel to the floor for the supine patient). The iliotibial band should be palpated, and the patient may be placed in the lateral position to adequately assess the band as the knee is flexed and extended.

The competency of the medial retinacular structures may be examined throughout the range of motion. The extended leg is brought into flexion while a mild laterally directed force is applied to the patella. If the MPFL is intact, the examiner should feel the pull of the patella as it is directed into the trochlear groove by the MPFL in early flexion. Lack of this medially directed force on the patella during early flexion indicates a disruption of tissue homeostasis surrounding the patella. Once discovered, this medial laxity should be carefully compared with lateral retinacular tightness and overall limb alignment (Q angle and tibial tubercle–trochlear groove) [19] to assess the current forces at work on the patellofemoral joint.

A prone examination is required during any initial meeting. With the pelvis stabilized, the knees should be maximally flexed in order to determine joint flexibility. For those patients with excessive quadriceps tightness and limited mobility, a sustained stretching program can bring symptomatic relief.

In general, the clinician should attempt to understand the balance, or lack thereof, surrounding the affected knee. Determining which restraints are deficient or overly tight allows a rational approach to stabilization and restoration of balance. Ultimately, this may require releasing tight restraints and restoring appropriate stability surgically [11].

Imaging

Each patient should have a weight-bearing posterior-anterior radiograph in slight flexion, a precise lateral radiograph (posterior condyles overlapped), and an axial radiograph in 30–45 degrees of flexion [11, 12]. The axial and lateral views provide valuable information, and correct technique must be employed. Axial views in an extreme degree of flexion will draw the patella into the trochlea, missing more subtle instabilities that may be present. Criteria for normal alignment have been well established [20–22]. Briefly, the Merchant congruence angle as later studied by Aglietti et al. [20] at 45 degrees of flexion is abnormal if it is >6 degrees in males and >2 degrees in females. In addition to congruence, axial radiographs are useful in identifying subluxation, joint space narrowing, osteophytes, and other patellar deformities [12]. A proper lateral radiograph can reveal trochlear dysplasia and rotational patellar malalignment [23–25].

Further evaluation of patella alignment may be accomplished by computed tomography (CT) at increasing angles of knee flexion (0 degrees, 15 degrees, 30 degrees, and 45 degrees). Midpatellar transverse images will allow a comparison of patellar location within the trochlea and evaluation of the tibial tubercle [19]. This information may be helpful in determining the need for an open realignment procedure. It can be very helpful also to establish the medial-lateral tomographic distance between the tibial tubercle (TT) and the trochlear groove (TG) and thus determine the TT-TG index. A TT-TG index >15 is generally accepted as abnormal.

Magnetic resonance imaging (MRI) capabilities have increased, allowing evaluation of cartilage surfaces and an assessment of subchondral bone. Soft tissue contributors to anterior knee pain may be visible, such as irritated synovium, excessive scarring from prior procedures, or other intraarticular pathology presenting as patellofemoral pain. Post cautions that higher-level radiographic studies such as CT and MRI "are most valuable when used to confirm and refine the clinical impression" [12].

Operative Techniques

Lateral Release

Indications

Indications for lateral release remain controversial [26]. A survey of international surgeons with a specific interest in patellofemoral surgery noted most surgeons performed isolated lateral release less than 10 times a year [27]. With the advent of arthroscopy, lateral release was used to treat generic anterior knee pain, patella instability, and osteoarthritis of the patellofemoral joint. Results for such broad applications were disappointing [28]. Lateral release has the best reported results when patients have clear tightness of the lateral retinacular tissues and evidence of excessive pressure on the lateral patellofemoral joint as defined by Ficat, but not instability or severe patellofemoral osteoarthritis [29–32]. Physical examination should support the diagnosis by confirming the inability to evert the patella to neutral from the lateral side, restricted medial mobility due to lateral retinacular tightness, and radiographic confirmation of excessive lateral tilt [32–34]. With the advent of improved MRI capabilities, bone edema visible on the lateral femur and on the lateral facet of the patella or cartilage compromise on the lateral facet of the patella can help confirm clinical suspicions [35]. Otherwise, radiographs, CT, or MRI should be used

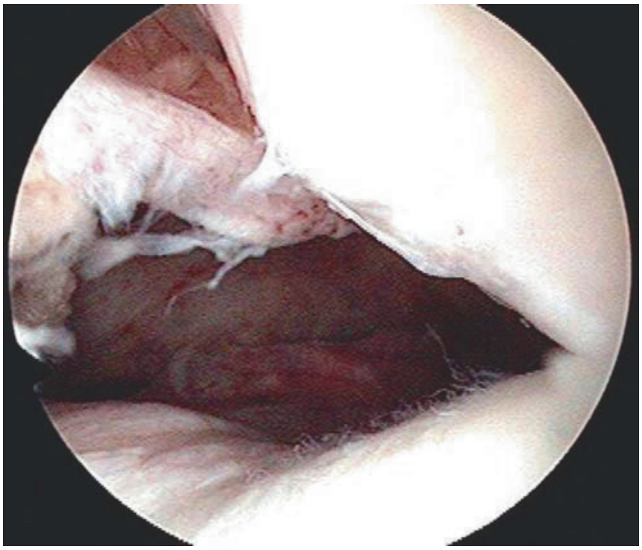


Fig. 3 Patellar tilt visible at arthroscopy

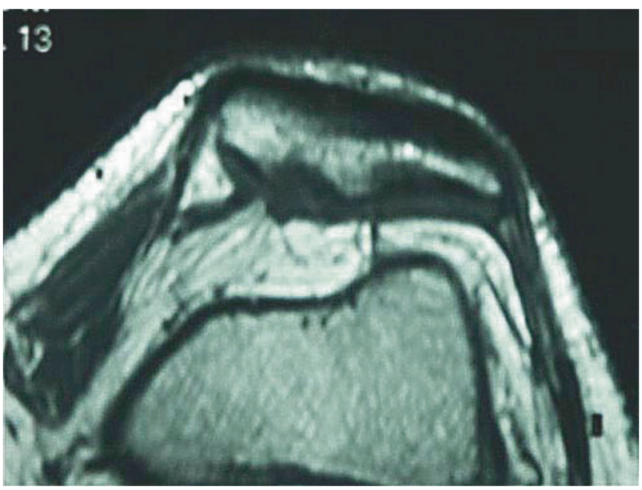


Fig. 4 Patellar tilt visible on preoperative MRI scan

confirm excessive tilt of the patella (Figs. 3 and 4). Of the clinically available reports, one of the longest follow-up studies reported 70% satisfaction in patients with isolated lateral release who did not have patellofemoral instability or major cartilage lesions at the time of surgery, whereas patients with either of those two confounding factors reported only 50% satisfaction [36].

Lateral release may also be employed as an adjunct technique. If severe cartilage disease is to be treated on either the lateral patella or lateral trochlea, lateral release may be considered as an additional step to offload the damaged area. Lateral release is also an important addition if arthroscopic medial imbrication is to be attempted. When considering a medial tightening procedure, a surgeon should critically assess the need for a lateral release to offload pressure on the lateral side while allowing appropriate balancing of the medial tissues.

Contraindications

In contemplating an arthroscopic approach to the patellofemoral joint, it is critical to understand the limitations of arthroscopy, and the treatment of patella instability with a simple arthroscopic lateral release seems to be contraindicated. A review by Lattermann et al. [28] concluded that isolated lateral release (an arthroscopic-only approach to patella instability) "has little or no role in the treatment of acute or chronic patella instability." This review cited studies by Aglietti et al. [37] and Dainer et al. [38], who showed generally poor outcomes in patients with instability treated by lateral release alone. Other authors have reported the results of lateral release alone in the treatment of patellar instability, also with disappointing results [39].

In addition, patients with hypermobility of the patella (excessive medial and lateral translation of the patella; evidence of generalized ligamentous laxity) have been reported to have poor results with lateral release [26, 40].

Technique

Patient positioning in the supine position with the leg extended on the operating room table will allow good mobility and visualization for an anticipated lateral release. The arthroscope may be inserted through an inferior-medial portal to begin the procedure, allowing better assessment of patellar positioning and the lateral structures during initial arthroscopic evaluation. It is possible to complete a diagnostic arthroscopy with visualization of the patellofemoral joint, medial joint space, notch, and lateral joint space through the initial medial portal. Attention can then be returned to the patellofemoral joint. The knee should be flexed and extended to ensure a complete view of patella tracking. Excessive lateral contact and tight lateral retinacular tissues should be confirmed, even with fluid insufflation of the joint. Careful examination of the cartilage on both major patella facets and the trochlea is important to predict outcomes (Figs. 5 and 6). Basic research has shown that lateral release does not significantly decrease contact forces in the patellofemoral joint, but it does shift the contact areas more medial [41]. Thus, it is important to document the status of the surface for the medial trochlea and patella. Needle localization may be used to create an inferior-lateral portal to ensure proper access to the lateral tissues. Electrocautery of some type, including radiofrequency or conventional Bovie, is helpful in minimizing bleeding during the procedure. Although a tourniquet is applied, slow and careful use of electrocautery can obviate

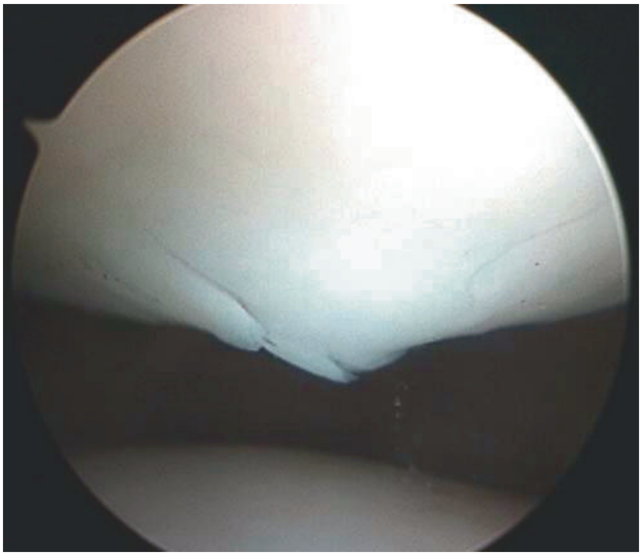


Fig. 5 Cartilage fissuring without fragmentation

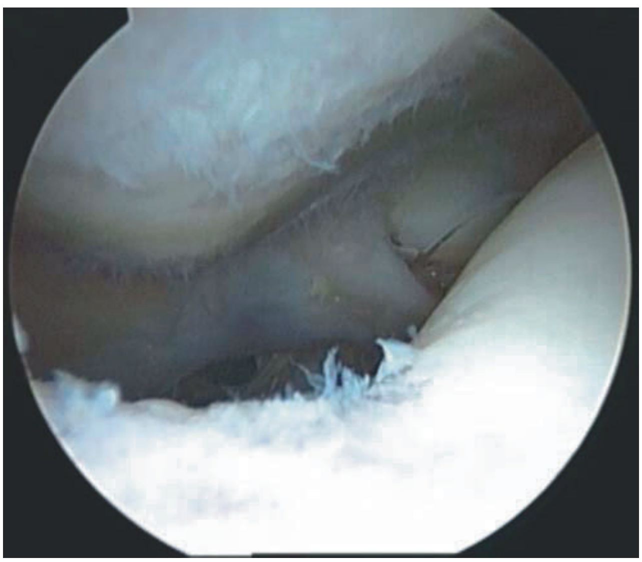


Fig. 6 Global patella and trochlear cartilage wear

the need for routine tourniquet use while ensuring proper homeostasis postoperatively. Ideally, the release should begin at the proximal pole of the patella, taking care to avoid the vastus lateralis insertion (Fig. 7). Release is complete when the subcutaneous fat is visible. The surgeon should concentrate in one small area with limited need to move the arthroscope during the release. This will allow timely cautery as vessels are encountered. Once the subcutaneous fat is visualized, the release may be carried inferiorly. Intermittent and careful application of the electrocautery is required to ensure heat does not penetrate to the skin level. Care should be taken to release inferiorly enough to ensure that the inferior pole of the patella is not tethered by tissue bands.

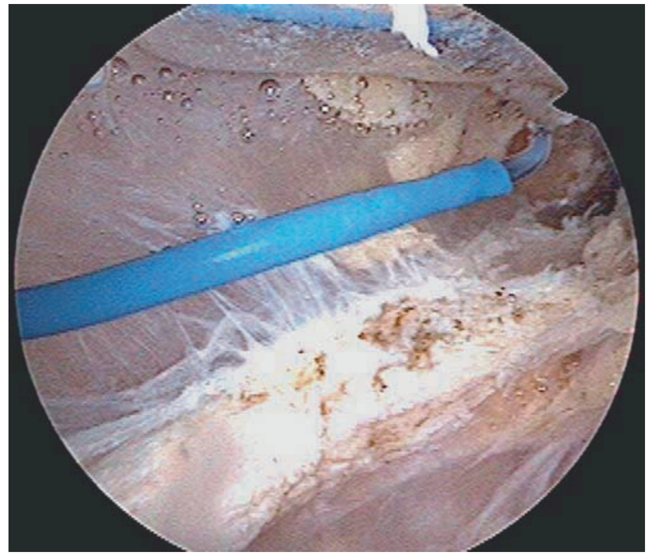


Fig. 7 Lateral release to proximal pole of patella

Once the release is completed, a repeat flexion-extension cycle may be visualized to ensure adequate mobilization of the lateral patella. A standard Merchant axial radiograph or postoperative CT should reveal correction of abnormal patellar tilt after lateral release (Fig. 8A, B).

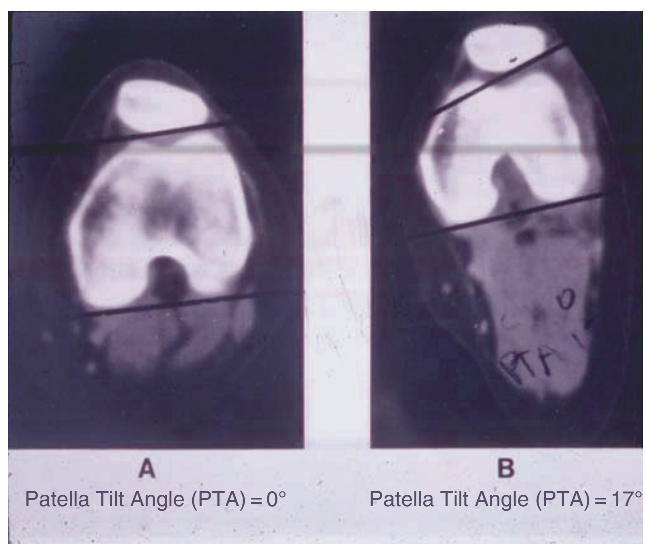


Fig. 8 (**A**) Preoperative CT scan showing patellar tilt. (**B**) Postoperative CT scan showing improved patellar tilt. (From Shea KP, Fulkerson JP. Preoperative computed tomography scanning and arthroscopy in predicting outcome after lateral retinacular release. *Arthroscopy* 1992;8:327–334. Copyright © 1992 Arthroscopy Association of North America. Reprinted with permission of Elsevier.)

Rehabilitation

Rehabilitation for lateral release procedures attempts to respect the soft tissue healing while encouraging appropriate range of motion. An early follow-up visit allows assessment of postoperative swelling and tissue reaction. Aspiration of any inhibitory hemarthrosis is helpful. During this time, the patient may gradually move quickly to full weight-bearing as comfort allows. When the soft tissue swelling is reduced, the patient should start gentle bicycle exercise. As motion improves, the patient should transition to a more formal physical therapy regimen that increases to full flexion. Strengthening exercises for the quadriceps should start on day 1. An aggressive strengthening program may be begun once full motion is obtained and motion does not cause inhibitory lateral-sided discomfort.

Complications

Hemarthrosis after lateral release has been reported at rates ranging from 0 to 42%. A recent review noted most series have reported rates of <10% [26, 42–44]. Another review from Digiulio and Donaldson noted an incidence of 4.5% [45]. Careful attention to homeostasis with appropriate "post-release" visualization will limit this complication. Routine use of tourniquet without removal prior to end of procedure may result in missed vessel transaction and inadequate homeostasis.

Incomplete release of the inferior tissues can result in continued lateral tilt and inadequate symptom relief. Care should be taken to visualize the inferior tissues through the medial portal. Instruments may be inserted through the inferior-lateral portal to confirm no tissue bands exist connecting the lateral retinaculum and the inferior patella.

Excessive release may lead to the devastating problem of medial patellar instability [11, 46]. Release should ideally end at the proximal pole of the patella, taking care to preserve the vastus lateralis. In addition, preoperative screening should prevent the patient with patella hypermobility from receiving a procedure that may increase patella imbalance. Aggressive release also has resulted in at least one report of quadriceps rupture [47].

Excessive pain may be present if medial articular lesions on the patella or the trochlea were not recognized at the time of lateral release. Increasing contact forces on areas of damaged cartilage are possible if a lateral release is performed in the setting of notable medial patellofemoral disease.

Although the use of an electrocautery device is recommended, reports of skin burns secondary to lateral release have been reported [48]. Careful and intermittent use of cautery is advisable.

Cartilage Procedures

Indications for Chondroplasty

The cartilage of the patella and trochlea are easily accessible for the arthroscopist; thus the temptation to operate is great. Patients who have failed a comprehensive nonoperative management based on their symptoms and history may benefit from arthroscopic examination and a cartilage procedure. As with many patellofemoral problems, a restrained approach to cartilage debridement is warranted. In a review of arthroscopic treatment of the patellofemoral joint with severe articular disease, Lee and Kelly advocated careful debridement [49]. They recommended not converting partial-thickness lesions to full-thickness lesions, removal of cartilage flaps only if they appear unstable, and maintenance of the subchondral plate except in the case of small lesions with an intact surrounding rim. This moderate approach is a reasonable theme when approaching patellofemoral articular disease.

Those patients with a distinct traumatic event should have clinical evidence of cartilage damage (positive compression test at a reproducible degree of knee flexion) and should ideally have imaging studies that corroborate the clinician's suspicion of cartilage damage. MRI may be particularly helpful in diagnosing discrete lesions of the patellofemoral joint. Debridement of loose bodies created by the trauma and treatment of cartilage damage may be helpful (Fig. 9). In one report, those patients with traumatic cartilage damage and

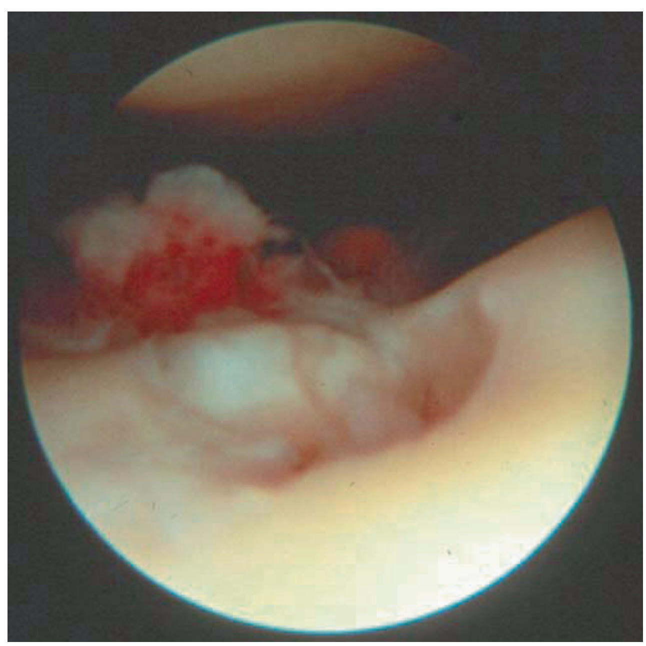


Fig. 9 Posttraumatic lesion trochlea

reported Outerbridge grade II or III lesions of the patella benefited from arthroscopic debridement. Good or excellent results were reported in 58% of the group, and all but 4 of 36 patients thought the surgery was beneficial [50]. One group reported on 37 patients with supposed atraumatic osteochondritis dissecans of the patellofemoral joint (24 patella, 13 trochlea), but the study did not report on the results of treatment [51].

Patients with evidence of long-standing lateral compression syndrome or patellar maltracking may have physical findings consistent with retro–patellar cartilage damage. Painful crepitus through range of motion, pain with a simulated step-up or step-down, and MRI evidence of unhealthy cartilage may warrant arthroscopic examination and treatment. In comparison with their above-reported results for traumatic patella lesions, Federico and Reider [50] reported a good to excellent outcome rate of 41% in patients with atraumatic Outerbridge II or III lesions of the patella. Again, when combined with the traumatic group, all but 4 of 36 patients believed the surgery provided some benefits. Other authors have advocated use of radiofrequency probes for cartilage debridement of the patellofemoral joint [52, 53], with at least one randomized comparison with mechanical debridement favoring radiofrequency ablation [52]. Although radiofrequency probes have been studied in the basic science and clinical area [54–56], their safety and efficacy over the long term is not yet ensured.

For those patients with long-standing patellofemoral disease, clear osteoarthritis may be noted on axial radiographs. If the pain is debilitating, arthroscopic debridement in combination with a lateral release for objective tilt and lateral tether is a viable, minimally invasive option for symptom reduction of primarily lateral facet disease [57].

Technique for Chondroplasty

Starting with an inferior-medial portal in the extended leg is an excellent arthroscopic approach to the patellofemoral joint. From this portal, an evaluation of all knee compartments can be accomplished, ruling out other intraarticular pathology. Once this has been accomplished, a thorough arthroscopic assessment of the patella and trochlea is possible with both static (stationary in any flexion angle) and dynamic (knee flexion-extension to evaluate congruence and tracking) positioning. Typically, the lateral facet of the patella and the lateral condyle are the areas of interest, but the inferior-medial viewing portal allows excellent visualization of the entire joint. A probe is a vital instrument in assessing the state of the cartilage. If softened cartilage is encountered, a probe will help determine if it is stable (Fig. 10). If the cartilage in question does not have large fissures and is in fact in continuity with the surrounding cartilage rim, it is preferable to leave this barrier in place.

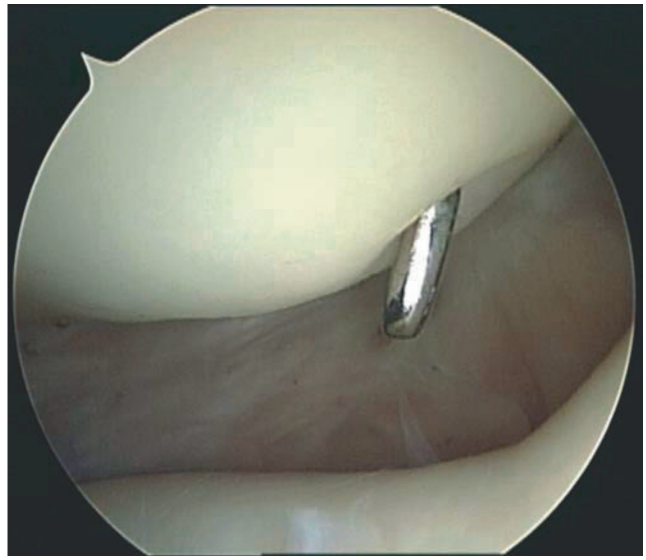


Fig. 10 Soft cartilage tested with arthroscopic probe

If debridement is required, needle localization may be used to create either inferior or superior-lateral portals. This should allow good access for any debridement or chondroplasty procedure. Occasionally, the superior-medial portal can be used for instrument passage. Keep in mind that the arthroscope can also be shifted to any portal quickly and easily with the use of a switching stick to move the cannula quickly and efficiently. This allows different angles of visualization and instrumentation to carry out a thorough treatment of the patella. Be sure to maximize debridement and instrumentation with the arthroscope in one position before chang-

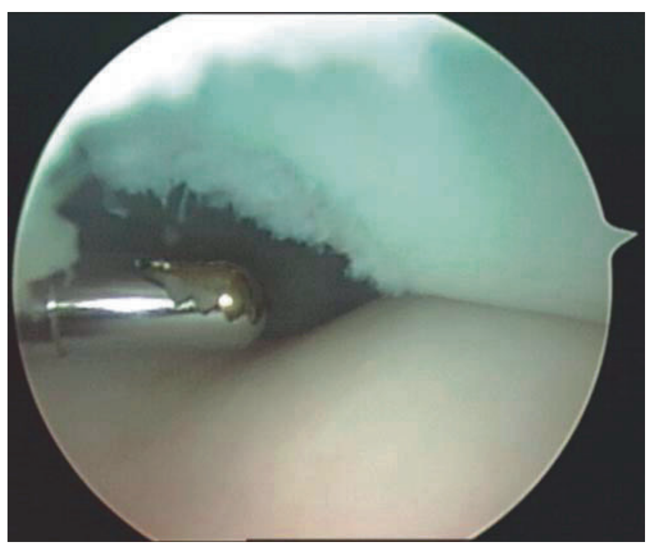


Fig. 11 Chondroplasty of distal patella

ing portals in order to avoid repeated instrument insertion and soft tissue trauma (Fig. 11). It can be helpful to have an assistant stabilize or tilt the patella to improve access when the knee is fully extended. Keeping in mind the “less is more” theory of cartilage debridement, concentrate on removing all loose cartilage fragments and creating stable borders for any treated lesion.

Rehabilitation for Chondroplasty

Chondroplasty is a well-tolerated procedure that in most cases has a relatively benign postoperative course. Although occasional effusions may arise in the immediate postoperative period, there are no major contraindications to movement or weight-bearing in the postoperative period. Immediate range of motion and full weight-bearing may begin in the postoperative period, with caution to limit the range of motion to a nonpainful arc. After 3–5 days of limited motion, a more aggressive focus on increasing range of motion may be undertaken. After 1 week, patients should strive to have full extension and flexion of 90 degrees. Strengthening exercises may be begun once 90 degrees of flexion is comfortably achieved. A short course of focused physical therapy can help with patient instruction for strengthening and range of motion after the initial postoperative visit.

Indications for Microfracture

When cartilage damage is suspected, MRI may indicate a discrete area of damage on the patella or the trochlea. Encountering discrete cartilage lesions in the patellofemoral joint is uncommon, but different options do exist. Although reports continue to be published about the success of autologous chondrocyte implantation [58, 59], other larger-scale studies do not yet show an overwhelming benefit when compared with microfracture [60, 61]. Therefore, it remains reasonable to treat discrete cartilage lesions of intermediate size (about 1.5 cm × 1.5 cm) in the trochlea with microfracture as a first line of treatment.

Discrete lesions on the patella present a more challenging dilemma. Appropriate preparation of a suitable microfracture bed with vertical walls is difficult on the patella, and obtaining the correct angle for microfracture entry is challenging. Coupled with the strength of the subchondral bone, microfracture for the patella is technically demanding. It may be advisable to simply remove all loose cartilage back to a stable rim. If a marrow stimulation procedure is desired, drilling of the subchondral bone may be considered. Ultimately, the patient’s postoperative course may indicate the need for a more involved procedure (autologous chondrocyte implantations (ACI), Osteochondral autograft transfer system (OATS), allograft plug) for discrete cartilage lesions.

Technique for Microfracture

The theory and techniques for microfracture have been reviewed in detail by Steadman et al. [62] Particular attention must be paid to achieving a stable rim in the trochlear region. Ring curettes and small osteotomes can be helpful in trying to achieve the vertical, stable rim of healthy cartilage associated with good outcomes (Fig. 12). When the time comes for a microfracture on the lateral side, consideration should be given to a lateral release to offload the affected area. This course of action is not automatic, and it is only recommended if the surgeon believes excessive lateral tightness exists. If excessive lateral retinacular tightness is confirmed on preoperative exam, and intraoperative examination confirms the affected area will likely continue to see excessive articular forces, then lateral release may be added to a microfracture technique. There are no reliable results in the literature about microfracture of the patella.

Rehabilitation for Microfracture

The rehabilitation for microfracture has been a source of debate in the orthopaedic literature [62, 63]. In general, the authors recommend range of motion in the immediate postoperative period and touch-down weight-bearing only. Crutches are a requirement in order to limit load across the knee. Physical therapy may be begun to increase motion, with a focus on achieving 90 degrees of flexion in the first few weeks. After 4 weeks, partial weight-bearing is allowed and full knee flexion may be sought. If required, additional strengthening programs may begin at this point. At 6 weeks,

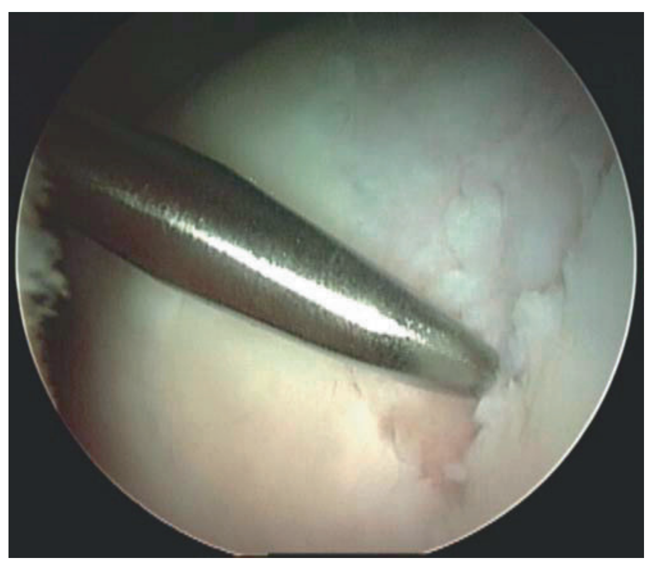


Fig. 12 Microfracture of the trochlea

all limitations to weight-bearing are removed, and the patient may be instructed in a final strengthening program and weight-bearing tolerated.

Complications for Chondroplasty and Microfracture

Routine complications associated with arthroscopy such as infection [64–66] or thrombosis [67] may be anticipated. Aggressive debridement has been reported to cause cystic degeneration of the patella in at least one report [68]. The most likely negative outcome from a cartilage debridement or marrow stimulation procedure is likely to be continued symptoms. Careful surgical technique should assure the surgeon that he or she may proceed with the confidence that he or she will not increase postoperative symptoms.

Medial Retinacular Imbrication

Indications

The decision to approach patella malalignment or instability arthroscopically must be carefully considered. Recent attention has focused on the MPFL as the main source of medial balance for the patella [8, 10]. For patients with disruption of this medial restraint, arthroscopic MPFL/medial retinacular tightening may be considered. Important physical exam findings would include a lax MPFL with knee flexion and normal bony alignment. Bicos et al. noted that MPFL reconstruction should not be seen as a tether designed to compensate for extreme bony malalignment (noted as a TT-TG of $>$20 mm, where the TT-TG is a CT comparison of the tibial tubercle and trochlear groove on overlying images) [10]. Consideration of the natural course of the MPFL (from its origin just distal to the adductor tubercle to its insertion on the intersection of the proximal and middle thirds of the medial patella) is important when considering arthroscopic tightening procedures. Overzealous imbrication of the medial tissues may upset the natural balance of the MPFL (tightest at 30 degrees of flexion, then progressively lax) and lead to medial joint overload [69]. Tom and Fulkerson reported that patients who have experienced dislocations often have healing of the MPFL after injury [70]. They reported that more than 90% of patients examined with use of an open medial patellar approach had objective healing of the MPFL and were therefore candidates for MPFL advancement or imbrication rather than tendon graft reconstruction. Thus, patients who have suffered a dislocation and have lost the medial restraint may, in fact, have sufficient tissue to imbricate in the correct anatomic alignment of the native MPFL. Multiple authors have reported case series of arthroscopic medial imbrication using some type of suture. All have reported good short-term clinical results, with failure rates roughly approximating most open technique reports [71–75]. Among those reports, Shoettle et al. was the only group that did not routinely add a lateral release along with the medial tightening procedure [75]. At least one group has reported on the successful combination of lateral release with medial thermal tissue shrinkage [76], but the authors of this chapter do not advocate this. Another group has reported on the repair of the MPFL using suture anchors into the patella [77].

If a patient does not have bony malalignment, arthroscopy must establish viable tissues exist for tightening in the medial retinaculum. In addition, the patella insertion of the tissues must not be compromised or any tightening procedure will not extend to the medial patella. When combined with an appropriate lateral release, medial imbrication of the retinaculum in the line of the native MPFL appears to restore patella stability in select cases. Our approach is to use less invasive options first and save open tendon graft reconstruction of the MPFL for failures and more severe cases.

Technique

A thorough arthroscopic examination should include the status of the medial tissues. Ideally, a band in the appropriate anatomic direction of the MPFL should be visualized at the time of arthroscopy. If considered, a lateral release should be performed prior to beginning medial imbrication. The medial tissue may be lightly roughed by a shaver without suction or a rasp to stimulate a healing response. A spinal needle can be introduced from the medial side through the medial MPFL remnant and into the joint. An absorbable PDS, Ethicon, located in Summit, NJ suture may be passed into the joint. With the addition of a small stab incision on the medial knee, a penetrating device may be introduced to grasp the free end of the suture and retrieve it outside of the knee (Fig. 13). Tissue between the medial entry of each suture limb should be released so the suture will be tied directly on the capsule. At this point, additional sutures may be placed prior to final tying in order to adjust tension (Fig. 14). Alternatively, the surgeon may take one free end of the already passed suture and direct it again through a carefully directed spinal needle above the previous loop of suture. The surgeon may again use a penetrator to grasp the free suture end below the first loop in order to create a cruciform suture.

This process may be repeated, taking care to stay in the anatomic course of the native MPFL. Tension may be adjusted by differential tying, and visualization of patellar tracking should provide instant feedback as to the appropriateness of the tension. Each suture should be tied over the capsule at the close of the procedure.

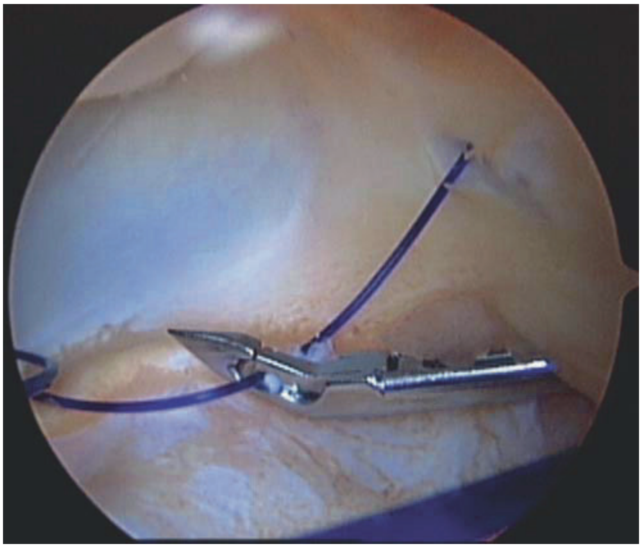


Fig. 13 Grasping medial suture with tissue penetrator

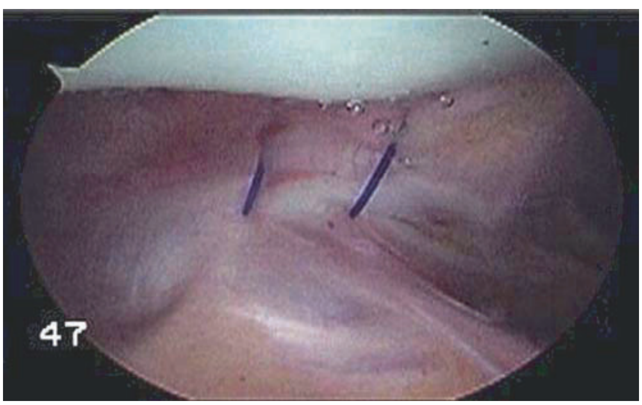


Fig. 14 Completed medial imbrication

Rehabilitation

Medial imbrication rehabilitation relies on soft tissue healing and limited postoperative motion to achieve optimal tissue healing. The protocol is very similar to the lateral release plan, and medial imbrication is often paired with lateral release, but initially patients remain in a knee immobilizer for 2–3 weeks to allow soft tissue healing. Partial weight-bearing is tolerated with advance to full weight-bearing as pain allows. After the healing period, patients begin by flexing the knee to 90 degrees once daily to maintain mobility. As motion and comfort increase, formal physical therapy may begin with the goal of restoring normal motion. After 5–6 weeks, a formal strengthening program may begin, along with proprioceptive training, core stability, and full weight-bearing.

Complications

Medial tissue imbrication has been reported without major complications. The most common complication is failure of the repair. Failure of medial imbrication should be minimized by careful evaluation of the patella after both a lateral release and medial imbrication. Examination of patellar tracking with and without joint insufflation should allow the surgeon to critically asses the amount of stability afforded by the procedure. This should allow fine tuning with addition or subtraction of imbrication stitches to achieve adequate medial-lateral balance in early flexion. If arthroscopic medial stabilization is not sufficient, the surgeon must be ready and able to proceed with an appropriate MPFL reconstruction in an open fashion.

It is clear that overtightening the medial tissues is possible; thus care must be taken to achieve the goal of restoring patellar stability in early flexion without excessive tightening. Again, the ability to review and adjust tension using arthroscopic visualization of multiple flexion-extension cycles is paramount. If notable cartilage damage exists on the medial facet of the patella (not uncommon after dislocation) or the medial trochlea, extra care must be taken not to overload these damaged surfaces.

Entrapment or damage to the branches of the saphenous vein and nerve are possible, depending on the technique used for imbrication. Such complications may be avoided by concentrating the sutures over a small area in line with the MPFL and only slightly medial to the patella.

Clinical Pearls

Evaluating Patients with Prior Surgical History

Patients with anterior knee pain have often had prior surgical procedures, sometimes well removed from the current visit. It bears repeating that critical information that changes the treatment plan can be contained in prior operative reports and arthroscopic images. Often explaining to the patient the value of prior surgical information can provide the stimulus needed for the patient to assist with record recovery in a proactive manner. A comprehensive soft tissue examination of the peripatellar regions often will reveal a postarthroscopy portal neuroma. Special attention should be paid to the areas surrounding portal scars. This postoperative complication of arthroscopy often is neglected in the patient who initially presented with anterior knee pain.

Limiting Bleeding After Lateral Release

After the majority of the lateral release has been completed, additional operating time to ensure adequate homeostasis can prevent may problems. A small area of the release can be viewed with the arthroscope, and the electrocautery device should be left in the visual field. Inflow of fluid into the joint may be stopped, and fluid may be slowly released out of the joint. If done carefully, small bleeding vessels will present themselves. Outflow may be stopped, and the well-positioned electrocautery can cauterize any vessels in the field of view. Inflow may be reapplied, the knee reinsufflated, and the process repeated. Typically, a few cycles are required to achieve complete homeostasis. Although this process does extend the surgical time, its potential benefits are well worth the investment.

Evaluating the Medial Knee Capsule when Considering Medial Imbrication

The techniques described above require a healed medial capsule with a biomechanically present, but lengthened, MPFL. In order for arthroscopic medial imbrication to be effective, the medial tissue must insert on the medial patella and maintain its longitudinal orientation. During the initial arthroscopy, the medial patella should be carefully examined. Often, a complete medial capsular failure may be noted with increasing exposure of the medial patella and a ballooning of the medial capsule. In these cases, medial imbrication will not deliver the desired medial pull on the patella. More commonly, a thickening of the medial capsule as in the shoulder can still be visualized, with a clear attachment to the medial patella. In this case, medial imbrication may be considered.

Summary

The technical skills required for effective patellofemoral joint arthroscopic surgery are well within the grasp of any surgeon with arthroscopic experience. Far more important is a better understanding of the possible causes of pain in the anterior knee and the appropriate application of surgical techniques. The forces acting on the patella and the soft tissues of the anterior knee can be changed with the application of careful arthroscopic techniques. A comprehensive physical exam, when supplemented by good imaging (including MRI), can help the surgeon target procedures to specific pain-generating regions and improve outcomes.

Case Report

Case 1: Chondroplasty

Chief Complaint and Patient History: A 37-year-old woman presents with long-term (15-year) vague anterior knee pain. She is active as a recreational tennis player, but recent pain has limited her play. Recent increasing symptoms include problems climbing stairs and an increase in painful crepitus with walking. She is no longer able to complete a tennis match. She localizes the worst of the pain to the middle of the knee "on the inside." She reports no recent trauma and has not had prior surgery. No effusions are reported. She has undergone 3 weeks of well-designed physical therapy and has had two trials of prescribed nonsteroidal medications in the past 3 months.

Physical Exam: Physical exam reveals a mild limp with walking, good core stability, and normal leg alignment. Palpation of the anterior knee yields no areas of discrete pain. Prone knee flexion is mildly limited on the affected side (Fig. 15A). The patella is stable and tracks well through a complete range of motion. The lateral retinaculum is mildly tight, but the patella may be tilted to neutral or parallel to the floor. Patella compression at different angles of knee flexion is markedly positive, with painful crepitus in early flexion that resolves after 45 degrees of flexion (Fig. 15B). The step-down test causes notable pain when the affected side remains on the stool in early flexion.

Imaging: Radiographs in the office setting are normal. MRI scan brought by the patient to the office does not show discrete pathology. A CT scan shows mild patellar tilt (Fig. 15C).

Surgery/Treatment: The decision to proceed to operative treatment comes from the physical exam findings that indicate a distal patella lesion. Given the recent increase in pain with stairs and the positive step-down test, this anatomic location is considered the prime target area. The lack of recent trauma and the progressive and long-term nature of the patient's anterior knee pain make a cartilage/wear lesion more likely. Findings at the time of arthroscopic evaluation included a grade III lesion of the distal pole of the patella (2 cm × 2 cm) that was debrided back to stable cartilage (Fig. 15D). The patient experienced a significant, although not complete, reduction in symptoms that allowed a return to the desired activities with occasional nonsteroidal drug use for pain reduction.

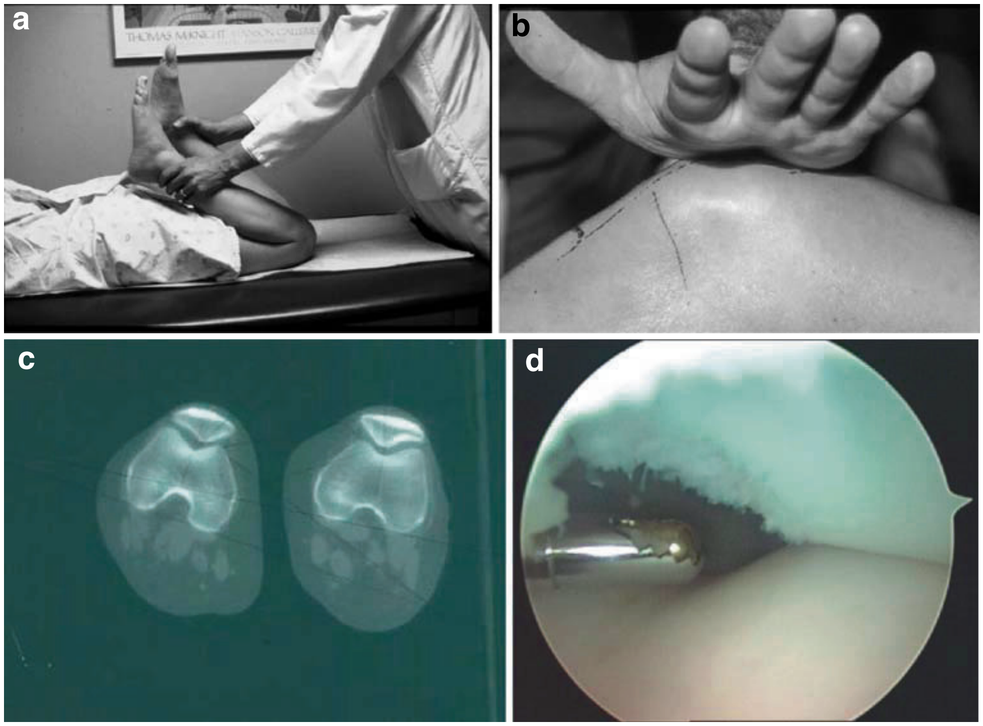


Fig. 15 Case 1. (**A**) Slightly limited knee flexion. (**B**) Compression elicits specific pain at specific flexion angle. (**C**) CT scan with minimal tilt. (**D**) Chondroplasty of distal patella

References

1. Fulkerson JP, Tennant R, Jaivin JS, Grunnet M. Histologic evidence of retinacular nerve injury associated with patellofemoral malalignment. *Clin Orthop Relat Res* 1985;197: 196–205.
2. Biedert RM, Stauffer E, Friederich NF. Occurrence of free nerve endings in the soft tissue of the knee joint. A histologic investigation. *Am J Sports Med* 1992;20:430–433.
3. Post WR. Anterior knee pain: diagnosis and treatment. *J Am Acad Orthop Surg* 2005;13:534–543.
4. Farahmand F, Senavongse W, Amis AA. Quantitative study of the quadriceps muscles and trochlear groove geometry related to instability of the patellofemoral joint. *J Orthop Res* 1998;16: 136–143.
5. Fulkerson JP, Gossling HR. Anatomy of the knee joint lateral retinaculum. *Clin Orthop Relat Res* 1980;153:183–188.
6. Nomura E, Horiuchi Y, Kihara M. Medial patellofemoral ligament restraint in lateral patellar translation and reconstruction. *The Knee* 2000;7:121–127.
7. Arendt EA, Fithian DC, Cohen E. Current concepts of lateral patella dislocation. *Clin Sports Med* 2002;21:499–519.
8. Amis AA, Firer P, Mountney J, Senavongse W, Thomas NP. Anatomy and biomechanics of the medial patellofemoral ligament. *The Knee* 2003;10:215–220.
9. Senavongse W, Amis AA. The effects of articular, retinacular, or muscular deficiencies on patellofemoral joint stability. *J Bone Joint Surg Br* 2005;87:577–582.
10. Bicos J, Fulkerson JP, Amis A. Current concepts review: the medial patellofemoral ligament. *Am J Sports Med* 2007;35:484–492.
11. Fulkerson JP. Diagnosis and treatment of patients with patellofemoral pain. *Am J Sports Med* 2002;30:447–456.
12. Post WR. Clinical evaluation of patients with patellofemoral disorders. *Arthroscopy* 1999;15:841–851.
13. Post WR, Fulkerson J. Knee pain diagrams: correlation with physical examination findings in patients with anterior knee pain. *Arthroscopy* 1994;10:618–623.
14. Tiberio D. The effect of excessive subtalar joint pronation on patellofemoral mechanics. *J Orthop Sports Phys Ther* 1987;9: 160–165.
15. Ireland ML, Willson JD, Ballantyne BT, Davis IM. Hip strength in females with and without patellofemoral pain. *J Orthop Sports Phys Ther* 2003;33:671–676.
16. Dupont JY. Synovial plicae of the knee. Controversies and review. *Clin Sports Med* 1997;16:87–122.
17. Fulkerson JP. Patellofemoral pain disorders: evaluation and management. *J Am Acad Orthop Surg* 1994;2:124–132.
18. Fulkerson JP. Office evaluation of patients with anterior knee pain. *Am J Knee Surg* 1997;10:181–183.
19. Goutallier D, Bernageau J, Lecudonnec B. [The measurement of the tibial tuberosity. Patella groove distanced technique and results (author's transl.).] *Revue de chirurgie orthopedique et reparatrice de l'appareil moteur* 1978;64:423–428.
20. Aglietti P, Insall JN, Cerulli G. Patellar pain and incongruence. I: measurements of incongruence. *Clin Orthop Relat Res* 1983;176:217–224.

21. Laurin CA, Dussault R, Levesque HP. The tangential x-ray investigation of the patellofemoral joint: x-ray technique, diagnostic criteria and their interpretation. *Clin Orthop Relat Res* 1979;144: 16–26.
22. Merchant AC, Mercer RL, Jacobsen RH, Cool CR. Roentgenographic analysis of patellofemoral congruence. *J Bone Joint Surg Am* 1974;56:1391–1396.
23. Dejour H, Walch G, Nove-Josserand L, Guier C. Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc* 1994;2:19–26.
24. Dejour H, Walch G, Neyret P, Adeleine P. [Dysplasia of the femoral trochlea.] *Revue de chirurgie orthopedique et reparatrice de l'appareil moteur* 1990;76:45–54.
25. Malghem J, Maldague B. [Profile of the knee. Differential radiologic anatomy of the articular surfaces.] *J Radiol* 1986;67: 725–735.
26. Williams JS, Jambor C. Role of lateral release in patellofemoral instability. *Tech Knee Surg* 2006;5:187–192.
27. Fithian DC, Paxton EW, Post WR, Panni AS. Lateral retinacular release: a survey of the International Patellofemoral Study Group. *Arthroscopy* 2004;20:463–468.
28. Lattermann C, Toth J, Bach BR Jr. The role of lateral retinacular release in the treatment of patellar instability. *Sports Med Arthrosc Rev* 2007;15:57–60.
29. Ficat P. [The syndrome of lateral hyperpressure of the patella.] *Acta Orthop Belg* 1978;44:65–76.
30. Shea KP, Fulkerson JP. Preoperative computed tomography scanning and arthroscopy in predicting outcome after lateral retinacular release. *Arthroscopy* 1992;8:327–334.
31. Christensen F, Soballe K, Snerum L. Treatment of chondromalacia patellae by lateral retinacular release of the patella. *Clin Orthop Relat Res* 1988;234:145–147.
32. Aderinto J, Cobb AG. Lateral release for patellofemoral arthritis. *Arthroscopy* 2002;18:399–403.
33. Ford DH, Post WR. Open or arthroscopic lateral release. Indications, techniques, and rehabilitation. *Clin Sports Med* 1997;16: 29–49.
34. Fulkerson JP, Schutzer SF, Ramsby GR, Bernstein RA. Computerized tomography of the patellofemoral joint before and after lateral release or realignment. *Arthroscopy* 1987;3:19–24.
35. Elias DA, White LM. Imaging of patellofemoral disorders. *Clin Radiol* 2004;59:543–557.
36. Panni AS, Tartarone M, Patricola A, Paxton EW, Fithian DC. Long-term results of lateral retinacular release. *Arthroscopy* 2005;21:526–531.
37. Aglietti P, Pisaneschi A, De Biase P. Recurrent dislocation of patella: three kinds of surgical treatment. *Ital J Orthop Traumatol* 1992;18:25–36.
38. Dainer RD, Barrack RL, Buckley SL, Alexander AH. Arthroscopic treatment of acute patellar dislocations. *Arthroscopy* 1988;4: 267–271.
39. Ricchetti ET, Mehta S, Sennett BJ, Huffman GR. Comparison of lateral release versus lateral release with medial soft-tissue realignment for the treatment of recurrent patellar instability: a systematic review. *Arthroscopy* 2007;23:463–468.
40. Gecha SR, Torg JS. Clinical prognosticators for the efficacy of retinacular release surgery to treat patellofemoral pain. *Clin Orthop Relat Res* 1990;253:203–208.
41. Ostermeier S, Holst M, Hurschler C, Windhagen H, Stukenborg-Colsman C. Dynamic measurement of patellofemoral kinematics and contact pressure after lateral retinacular release: an in vitro study. *Knee Surg Sports Traumatol Arthrosc* 2007;15: 547–554.
42. Simpson LA, Barrett JP Jr. Factors associated with poor results following arthroscopic subcutaneous lateral retinacular release. *Clin Orthop Relat Res* 1984;186:165–171.
43. Bigos SJ, McBride GG. The isolated lateral retinacular release in the treatment of patellofemoral disorders. *Clin Orthop Relat Res* 1984;186:75–80.
44. Henry JH, Goletz TH, Williamson B. Lateral retinacular release in patellofemoral subluxation. Indications, results, and comparison to open patellofemoral reconstruction. *Am J Sports Med* 1986;14:121–129.
45. DiGiulio M, Donaldson WR. Complications of patellofemoral joint surgery. *Sports Med Arthrosc Rev* 2004;12: 172–184.
46. Hughston JC, Deese M. Medial subluxation of the patella as a complication of lateral retinacular release. *Am J Sports Med* 1988;16:383–388.
47. Blasier RB, Ciullo JV. Rupture of the quadriceps tendon after arthroscopic lateral release. *Arthroscopy* 1986;2:262–263.
48. Lord MJ, Maltry JA, Shall LM. Thermal injury resulting from arthroscopic lateral retinacular release by electrocautery: report of three cases and a review of the literature. *Arthroscopy* 1991;7: 33–37.
49. Lee G, Kelly MA. Isolated patellofemoral arthritis without malalignment. In: Fulkerson J, ed. *Common Patellofemoral Problems*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 2005, pp. 73–84.
50. Federico DJ, Reider B. Results of isolated patellar debridement for patellofemoral pain in patients with normal patellar alignment. *Am J Sports Med* 1997;25:663–669.
51. Peters TA, McLean ID. Osteochondritis dissecans of the patellofemoral joint. *Am J Sports Med* 2000;28:63–67.
52. Owens BD, Stickles BJ, Balikian P, Busconi BD. Prospective analysis of radiofrequency versus mechanical debridement of isolated patellar chondral lesions. *Arthroscopy* 2002;18: 151–155.
53. Barber FA, Iwasko NG. Treatment of grade III femoral chondral lesions: mechanical chondroplasty versus monopolar radiofrequency probe. *Arthroscopy* 2006;22:1312–1317.
54. Amiel D, Ball ST, Tasto JP. Chondrocyte viability and metabolic activity after treatment of bovine articular cartilage with bipolar radiofrequency: an in vitro study. *Arthroscopy* 2004;20: 503–510.
55. Yasura K, Nakagawa Y, Kobayashi M, Kuroki H, Nakamura T. Mechanical and biochemical effect of monopolar radiofrequency energy on human articular cartilage: an in vitro study. *Am J Sports Med* 2006;34:1322–1327.
56. Edwards RB 3rd, Lu Y, Uthamanthil RK, Bogdanske J, Muir P, Athanasiou K, Markel M. Comparison of mechanical debridement and radiofrequency energy for chondroplasty in an in vivo equine model of partial thickness cartilage injury. *Osteoarthritis Cartilage* 2007;15:169–178.
57. Saleh KJ, Arendt EA, Eldridge J, Fulkerson JP, Minas T, Mulhall KJ. Symposium. Operative treatment of patellofemoral arthritis. *J Bone Joint Surg Am* 2005;87:659–671.
58. Minas T. Autologous chondrocyte implantation for focal chondral defects of the knee. *Clin Orthop Relat Res* 2001;391(Suppl): S349–S361.
59. Jones DG, Peterson L. Autologous chondrocyte implantation. *J Bone Joint Surg Am* 2006;88:2502–2520.
60. Knutsen G, Drogset JO, Engebretsen L, Grøntvedt T, Isaksen V, Ludvigsen TC, Roberts S, Solheim E, Strand T, Johansen O. A randomized trial comparing autologous chondrocyte implantation with microfracture. Findings at five years. *J Bone Joint Surg Am* 2007;89:2105–2112.
61. Knutsen G, Engebretsen L, Ludvigsen TC, Drogset JO, Grøntvedt T, Solheim E, Strand T, Roberts S, Isaksen V, Johansen O. Autologous chondrocyte implantation compared with microfracture in the knee. A randomized trial. *J Bone Joint Surg Am* 2004;86: 455–464.

62. Steadman JR, Rodkey WG, Rodrigo JJ. Microfracture: surgical technique and rehabilitation to treat chondral defects. *Clin Orthop Relat Res* 2001;391:(Suppl):S362–S369.
63. Marder RA, Hopkins G Jr, Timmerman LA. Arthroscopic microfracture of chondral defects of the knee: a comparison of two postoperative treatments. *Arthroscopy* 2005;21:152–158.
64. Bert JM, Giannini D, Nace L. Antibiotic prophylaxis for arthroscopy of the knee: is it necessary? *Arthroscopy* 2007;23:4–6.
65. Lubowitz JH, Poehling GG. Arthroscopy and antibiotics. *Arthroscopy* 2007;23:1–3.
66. Babcock HM, Carroll C, Matava M, L'Ecuyer P, Fraser V. Surgical site infections after arthroscopy: outbreak investigation and case control study. *Arthroscopy* 2003;19:172–181.
67. Ramos J, Perrotta C, Badariotti G, Berenstein G. Interventions for preventing venous thromboembolism in adults undergoing knee arthroscopy. *Cochrane Database System Rev* 2007(2): CD005259.
68. Galloway MT, Noyes FR. Cystic degeneration of the patella after arthroscopic chondroplasty and subchondral bone perforation. *Arthroscopy* 1992;8:366–369.
69. Elias JJ, Cosgarea AJ. Technical errors during medial patellofemoral ligament reconstruction could overload medial patellofemoral cartilage: a computational analysis. *Am J Sports Med* 2006;34:1478–1485.
70. Tom A, Fulkerson J. Restoration of native MPFL support after patella dislocation. *Sports Med Arthrosc Rev* 2007;15: 68–71.
71. Ali S, Bhatti A. Arthroscopic proximal realignment of the patella for recurrent instability: report of a new surgical technique with 1 to 7 years of follow-up. *Arthroscopy* 2007;23: 305–311.
72. Haspl M, Cicak N, Klobucar H, Pecina M. Fully arthroscopic stabilization of the patella. *Arthroscopy* 2002;18:E2.
73. Small NC, Glogau AI, Berezin MA. Arthroscopically assisted proximal extensor mechanism realignment of the knee. *Arthroscopy* 1993;9:63–67.
74. Halbrecht JL. Arthroscopic patella realignment: an all-inside technique. *Arthroscopy* 2001;17:940–945.
75. Schoettle PB, Scheffler SU, Schwarck A, Weiler A. Arthroscopic medial retinacular repair after patellar dislocation with and without underlying trochlear dysplasia: a preliminary report. *Arthroscopy* 2006;22:1192–1198.
76. Coons DA, Barber FA. Thermal medial retinaculum shrinkage and lateral release for the treatment of recurrent patellar instability. *Arthroscopy* 2006;22:166–171.
77. Fukushima K, Horaguchi T, Okano T, Yoshimatsu T, Saito A, Ryu J. Patellar dislocation: arthroscopic patellar stabilization with anchor sutures. *Arthroscopy* 2004;20:761–764.

Surgical Management of Anterior Cruciate Ligament Injuries

Mark E. Steiner

Introduction

An anterior cruciate ligament (ACL) injury produces an athletic disability that is the most common knee injury to require a ligament reconstruction. Recent research has contributed to a better understanding of all aspects of this injury, but it is particularly in the areas of kinematics, graft placement, and graft fixation that the greatest improvements have occurred. Controversy abounds in the treatment of this injury, partly due to the difficulty in assessing results of treatment, but an understanding of the controversies is necessary in order to make informed treatment plans. This chapter will address the most current methodologies for ACL injury care relevant to the surgeon and team physician.

Anatomy

The ACL has an average length between 31 mm [1] and 38 mm [2] along its anterior border, but the ligament has an hourglass shape, and this shape defies simple length and width measurements. Further complicating a description of the ligament is the observation that the ligament is composed of two bands [3]. Whereas it is not a unanimous opinion that two bands exist, most observers have reported two bands and labeled them based on their tibial attachments: anteromedial band and posterolateral band [4]. A magnetic resonance imaging (MRI) analysis reported the anteromedial band as 27 mm in length and the posterolateral band as 22 mm in length [5]. The two bands are best appreciated when the knee is flexed when the posterolateral band rotates inferior and lateral to the more isometric anterior band.

The alignment of the overall ligament and its component bands is affected by knee flexion angle. In the coronal plane with the knee close to full extension, MRI images reveal a 19-degree angle between the ligament and the tibial shaft [5–7]; however, flexion increases this angle, and a coronal angle of 42 degrees has been recorded when the knee is flexed 90 degrees [8]. In the sagittal plane, MRI measurements with the knee in extension record a 35-degree angle between the central axis of the ACL and the tibial shaft [5, 7] and a 47-degree angle for just the posterolateral band. Biomechanical models dictate a greater angle between the ACL and the tibial shaft with increased knee flexion [9], and a measurement of 55 degrees between the central axis of the ACL and the tibial shaft with the knee flexed 90 degrees has been reported [8]. Because reconstructive procedures are generally performed with the knee in flexion, it is the alignment of the ACL when the knee is flexed that is most relevant.

What is particularly striking about the ACL is its large femoral and tibial attachment areas relative to its midsection. The insertion areas have been reported to be 3.5 times the size of the mid-ligament cross-sectional area [4]. This has significant implications for reconstructive procedures, during which tendon grafts with uniform diameters are implanted to replace a ligament with a very different gross appearance. Curiously, biomechanical models often depict the ligament as composed of bands when in fact the ligament is shaped like an hourglass.

For the surgeon, a thorough understanding of the femoral and tibial insertions of the ACL is particularly important because a common cause for reconstructive failure is placement of the graft outside these insertions [10–13]. Anatomic placement of grafts has been identified as a key to improved surgical outcomes [6].

The tibial insertion of the ACL lies on the intercondylar eminence between the medial and lateral tubercles; however, it does not attach to either the medial or lateral tubercles

This chapter was written concurrently with an article on the same topic published in the *American Journal of Sports Medicine*. Though the article and this chapter are similar in scope, they are markedly different and neither was used as a source for the other.

M.E. Steiner (✉)
Orthopaedic Sports Medicine, Department of Orthopaedic Surgery, New England Baptist Hospital, Harvard Medical School, Tufts Medical School, Boston, Massachusetts

B.P. McKeon et al. (eds.), *Knee Arthroscopy*, DOI 10.1007/978-0-387-89504-8_8,

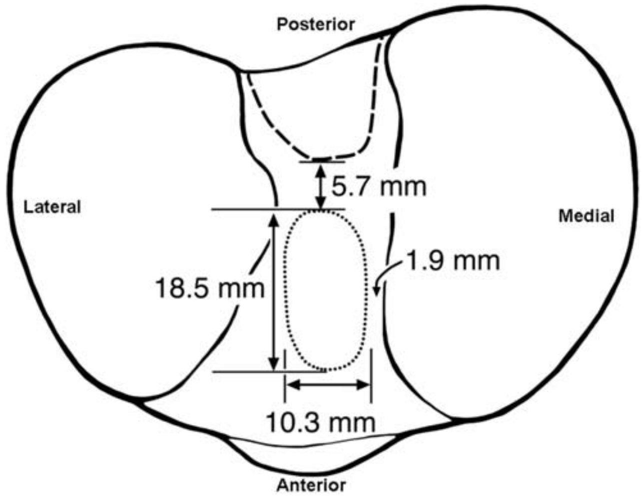


Fig. 1 The tibial insertion of the ACL is drawn with its average length and width measurements. Also shown are the distances to the PCL notch and medial tibial plateau articular cartilage. (From Steiner ME, Murray MM, Rodeo SA. Strategies to improve anterior cruciate ligament healing and graft placement. *Am J Sports Med* 2008;36:176–189. Copyright © American Orthopaedic Society for Sports Medicine. Reprinted with permission of Sage Publications. Based on data from Heming JF, Rand J, Steiner ME. Anatomic limitations of transtibial drilling in ACL reconstruction. *Am J Sports Med* 2007;35:1708–1715. Copyright © American Orthopaedic Society for Sports Medicine. Used with permission of Sage Publications.)

[2, 8, 14]. Although the peak of the medial eminence in some cases is difficult to identify, it has been observed that the entire footprint of the ACL is anterior to this point [15, 16]. The oval attachment, as seen in Fig. 1, which has an approximate length of 18 mm and an approximate width of 10 mm, is reflective of anatomic studies (Fig. 1) [1, 8, 14].

Whereas general agreement exists on the size and shape of the tibial insertion, debate still occurs over a reliable method to use to identify the anterior and posterior boundaries. Morgan et al. placed the center of the tibial footprint 7 mm anterior to the posterior cruciate ligament (PCL) at the level of the intercondylar eminence [17]. However, this would place the posterior border of the ACL practically adjacent to the most posterior margin of the tibial plateau. Recently, attempts have been made to reference the tibial insertion to the indentation on the posterior tibial plateau occupied by the PCL. This notch has been described as the posterior fovea, retro-eminence ridge, or PCL notch [8, 18].

Colombet et al. placed the center of the tibial insertion 19 mm anterior to the PCL notch [18], whereas Heming et al. placed the center 15 mm anterior to the PCL notch [8]. The implication here is as follows: ACL reconstructions looking for a tunnel in the center of the tibial insertion need to direct a guide pin approximately 10 mm more anterior than the location suggested by Morgan et al. [17]. Because grafts usually lie in the posterolateral region of a tibial tunnel, tunnels should be located slightly anterior and medial to the final destination of the graft [19].

Radiographically, the tibial insertion has been described relative to the boundaries of the tibial plateau. The center of the insertion has been reported by Amis and Jakob as 43% of the distance from the anterior to the posterior margin of the tibial plateau [20]. Other measurements have been similar [16]. The medial-lateral center of the tibial insertion averages 44% of the width of the plateau from the medial tibial cortex [1, 16, 20, 21]. These parameters are used by image-guided surgical guidance systems to identify the location of the tibial tunnel.

The femoral insertion has been measured by multiple authors [1, 2, 8, 14, 18], and there has been some variability; but consolidating the results would place the length at 18 mm, width 10 mm, and separation from the posterior articular cartilage of at most 4 mm (Fig. 2) [8]. The anterior border of the ligament's insertion can be variable, but it has been observed to lie directly posterior to a ridge on the medial aspect of the lateral condyle termed the *resident's ridge*. Viewed in the sagittal plane, the long axis of the femoral insertion is rotated 25–30 degrees to the long axis

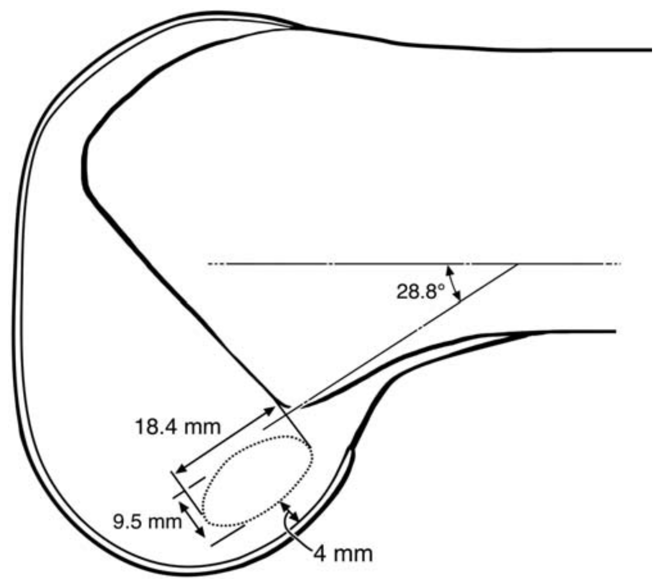


Fig. 2 The femoral insertion of the ACL is drawn with its average length and width measurements. The long axis of the insertion lies at a 28.8-degree angle to the shaft of the femur in the sagittal plane, and the posterior border lies no more than 4 mm from the articular cartilage of the femoral condyle. (From Steiner ME, Murray MM, Rodeo SA. Strategies to improve anterior cruciate ligament healing and graft placement. *Am J Sports Med* 2008;36:176–189. Copyright © American Orthopaedic Society for Sports Medicine. Reprinted with permission of Sage Publications. Based on data from Heming JF, Rand J, Steiner ME. Anatomic limitations of transtibial drilling in ACL reconstruction. *Am J Sports Med* 2007;35:1708–1715. Copyright © American Orthopaedic Society for Sports Medicine. Used with permission of Sage Publications.)

of the femur reflecting its congruity to the posterior border of the femoral condyle (Fig. 2). The femoral insertion has separate insertion sites for the anteromedial and posterolateral bands of the ACL. A transverse partition of the femoral insertion into a slightly larger proximal attachment region for the anteromedial band and slightly smaller distal region for the posterolateral band can be made [14, 18].

Visualizing the femoral insertion during an arthroscopic procedure can be a challenge. Generally, the overhang of articular cartilage at the anterior border of the notch obscures the posterior notch, and a notchplasty is necessary to gain a view of the ACL insertion. Some surgeons prefer to visualize the notch from a central or anteromedial portal, but this can crowd the instrumentation. Further compromising an appreciation of the insertion's relationship to the surrounding anatomy is the variable appearance of the notch based on knee flexion. The top of the notch on average is parallel to the tibia when the knee is flexed 55 degrees [22], and, in this position, the best view of the posterior notch is provided. When a knee is flexed beyond 55 degrees, the top of the notch appears to angle down toward the tibial plateau and obscure visualization of the posterior notch. Therefore, the relative position of the femoral insertion relative to the notch changes with flexion. The anteromedial band will be at the top of the notch regardless of knee flexion, but the posterolateral band appears to rotate anteriorly and laterally with knee flexion. Even more complexity enters because of the inconsistency in the shape of the arch of the intercondylar notch. Thus, in some knees, it can be extremely challenging to identify and place an anatomically correct femoral tunnel.

To identify the proximal-distal position of a femoral tunnel during ACL reconstruction, it is common to characterize the femoral position as it relates to the face of a clock [8, 23, 24]. Whereas this method can be useful, it requires two conditions to be exact: (1) the flexion angle of the knee must be given; (2) a transverse axis for the clock face must be indicated. However, many clock-face descriptions of the femoral ACL insertion have been presented without these conditions being met. In a laboratory setting with the knee flexed 90 degrees and with a clock face referenced to the posterior femoral condyles, the footprint of the femoral ACL insertion has been established (Fig. 3). However, visualizing the top of the notch and posterior border of the femoral condyles is difficult arthroscopically. A practical method is to image the apex of the notch and the lowest point on the lateral wall at the margin of the articular cartilage with the knee flexed 90 degrees. Viewed in this fashion, the center of the femoral origin is halfway between the apex of the notch and the edge of the articular cartilage at the base of the notch.

A radiographic description of the femoral insertion generally has been based on a quadrant division of the lateral wall of the notch [25]. By this convention, the center of the femoral ACL insertion was determined relative to the anterior-posterior length of the lateral condyle and the proximal-distal height of the notch. The center of the ACL was approximately 25% of the anterior-posterior distance from the posterior condyle and 28.5% of the proximal-distal distance from the top of the notch [26]. Lateral radiographs of the knee are very hard to interpret for proximal-distal position of the femoral tunnel, and generally only tunnel location in the anterior-posterior direction can be clearly identified [10, 27].

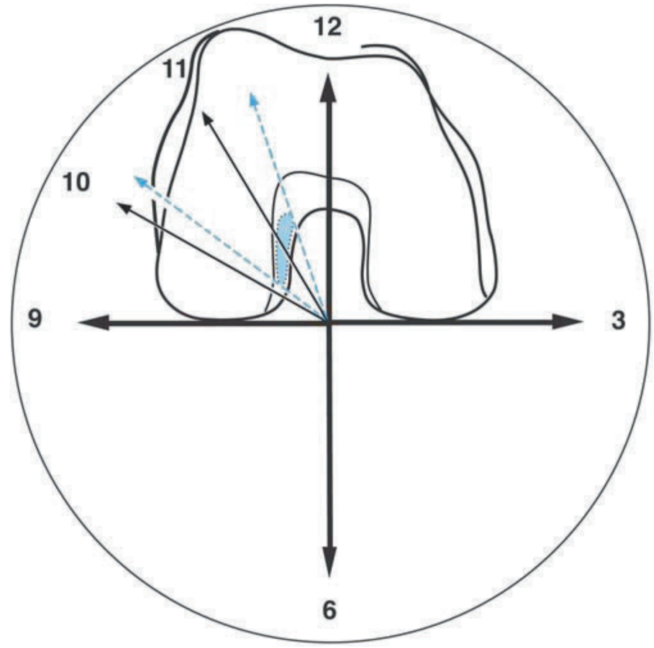


Fig. 3 The insertion of the ACL along the lateral wall of the intercondylar notch is depicted using the analogy of a clock face with the 3–9 o'clock axis aligned with the posterior femoral condyles and the knee flexed 90 degrees. Viewed in this fashion, the insertion lies between approximately 10:15 and 11:20. A practical approach is to place the center of the femoral insertion halfway between the apex of the notch and the articular cartilage margin at the bottom of the lateral wall. (From Steiner ME, Murray MM, Rodeo SA. Strategies to improve anterior cruciate ligament healing and graft placement. *Am J Sports Med* 2008;36:176–189. Copyright © American Orthopaedic Society for Sports Medicine. Reprinted with permission of Sage Publications. Based on data from Heming JF, Rand J, Steiner ME. Anatomic limitations of transtibial drilling in ACL reconstruction. *Am J Sports Med* 2007;35:1708–1715. Copyright © American Orthopaedic Society for Sports Medicine. Used with permission of Sage Publications.)

Biomechanics

The strength or ultimate load of the ACL has been reported to be as high as 2,160 N, with a stiffness of 242 N/mm when specimens between the ages of 22 and 35 years were tested [28]. Age diminishes the strength of the ligament both within its substance and at its insertions. The strength of the ligament is affected by how it is tensioned. There is the greatest strength when all fibers are tensioned in line with the ligament, but, if the ligament is distracted at an angle to its fibers, then it has less strength. This has implications for injury mechanisms where it is noted that a valgus combined with anterior motions is more prone to injure the ligament.

The ACL is well known to be the primary restraint to anterior translation of the knee. The increase in anterior translation secondary to an ACL injury is greatest at 30 degrees flexion, but it can sometimes be documented at flexion angles up to 90 degrees [29, 30]. Measurements have varied, but, in a clinical setting, an increase of more than 3 mm in anterior translation has been used as a diagnostic determinant for a significant ACL injury [31].

Recently, there has been a particular interest in the interaction of internal rotation and anterior translation in the ACL-deficient knee and particularly whether changes in graft placements or the use of two-stranded grafts are necessary to control abnormal rotation [32]. In laboratory studies, cutting the ACL can increase maximum internal rotation of the tibia up to approximately 4 degrees [33, 34]. There is also a coupled anterior translation with the application of a combined internal rotation-valgus torque [24]. Although the increase in internal rotation produced by an ACL injury is small relative to the absolute internal rotation of the knee (an approximate 20% increase), it is particularly the interaction of internal rotation and anterior translation in the ACL-deficient knee that is most interesting. There is evidence that ACL-injured and ACL-reconstructed knees demonstrate abnormalities in axial rotation during functional activities [35–39]. However, the implications of these studies are controversial, and it may be simply that restoring a normal limit to anterior translation will result in normal functional axial rotation to the knee [40]. The ACL does not affect the limit of external rotation, but it does act as a secondary restraint to varus and valgus rotations.

In the ACL-deficient knee, the tracking of the patella is also affected secondary to increased anterior translation. This occurs because an unopposed quadriceps force translates the tibia anteriorly at flexion angles under 60 degrees, and this results in a dynamic anterior translation of the tibial tubercle. The normal internal rotation with anterior translation is also lost when the ACL is torn. The net effect is a lateral translation and lateral tilt of the patella in the ACL-deficient knee when the quadriceps is contracted. This abnormal patellar tracking may return to normal when the ACL is reconstructed. Therefore, patellofemoral symptoms in ACL-injured and ACL-reconstructed knees may relate to altered patellar tracking [41, 42].

Diagnosis and Imaging

The history of an ACL injury is that of an acute giving way of the knee often secondary to a deceleration and rotation motion. Most often, the injury is an isolated noncontact event, and patients will report a “pop” or sense of something giving way in the knee. The isometric or eccentric contraction of the quadriceps while the knee is flexed secondary to the body’s inertia can cause the ACL to rupture [43]. It is important to identify contact injuries because they will often involve the collateral ligaments. The other common injuries in which the history can mimic an ACL injury are a patellar dislocation, an acute severe meniscus tear with a displaced fragment, and large articular chondral injuries. PCL tears can occur rarely in a noncontact situation, and quadriceps and patellar tendon injuries can have acute giving way and disability.

The *sine qua non* to the diagnosis of an ACL tear is a positive Lachman test. There are multiple methods to perform the test, but relaxing the thigh on the edge of a table and securing the patient’s lower leg between the examiner’s legs encourages relaxation of the thigh [44]. The lack of an end point indicates an ACL injury, but this finding requires good muscle relaxation. Often, the knee can be examined without spasm in the first hour after injury before a hemarthrosis develops. The pivot-shift test will also be positive in comparison with the opposite normal knee, but this test provokes more pain and is usually less reliable except in the first hour after injury or after swelling diminishes and motion returns to the knee.

A positive MRI finding of an ACL tear includes the loss of continuity of the ligament and a change in the direction of the ligament’s fibers. However, the ligament runs obliquely across the knee, and sometimes the full length of the ligament cannot be well visualized. Additional information is provided by measuring the angle between the ACL and the shaft of the tibia. On sagittal images, the normal ACL lies at a 45-degree angle or less to the axis of the tibial shaft. However, a torn ACL falls down in the notch and subtends a greater angle [7]. Geographic bone bruises also are seen in more than 80% of knees with acute ACL injuries. Most of these lesions will be in the posterior lateral tibial plateau and midlateral femoral condyle. Long-term articular cartilage injury has been documented when there is a disruption or depression of the cortical surface. Most patients have resolution of these bone bruises over several months, and they do not experience long-term consequences [45–48].

Instrumented laxity testing is useful to document abnormal anterior translation of the injured knee compared with the anterior translation of the normal knee, but it is infrequently used in routine clinical practice. A 3-mm or greater difference in anterior translation using the KT1000

(MedMetric Corporation, San Diego, CA) with a maximum manual anterior force is highly correlated with ACL injury. In general, a knee with 3–5 mm increased translation will have a pivot glide motion, and knees with greater than 5 mm increased anterior translation will have a frank 2+ pivot shift with a "clunk" when the tibia reduces. It has been proposed that knees with greater than 5 mm increased anterior translation or a 2+ pivot shift do poorly with conservative care [19, 49]. Although instrumented laxity testing is critical to the reporting of results, it has been demonstrated that the clinical examination is equal to instrumented laxity testing in diagnostic accuracy [31].

Meniscal and Chondral Injuries

The majority of patients sustain associated meniscus and chondral injuries at the time of an ACL injury, but, fortunately, most of these are minor and do not need treatment [19, 48]. Chondral injuries seen as bone bruises on MRI have ultrastructural injury and may rarely need microfracture or very rarely cartilage restoration surgery. However, the regenerative capacities of the cartilage to these acute injuries mitigates against overly aggressive treatment in the acute setting. The chronic ACL-injured knee may acquire full-thickness cartilage injuries that require more definitive treatment.

Similar to chondral injuries, most meniscus tears are stable and do not need repair or resection. The meniscus tear in the ACL-injured knee is classically a vertical tear in either the medial or lateral meniscus within several millimeters of the peripheral rim where there is a blood supply to promote healing. Those that are less than 1–1.5 cm in length and that extend no more than halfway through the meniscus do not need treatment. The tears that are posterior to the popliteus can be treated without resection and often do well [50]. Occasionally, trephination with a spinal needle has been employed in these small tears to increase vascularity and promote healing. The availability of recent meniscal repair technologies that do not require a posterior incision has promoted an increased number of meniscus repairs. Healing rates of meniscus tears associated with ACL tears are clearly higher than those of isolated meniscus tears that are often degenerative.

The injured knee with a locked bucket-handle meniscus is a special case, and it needs treatment tailored to the demands of the patient and the physiology of the knee. In an acute-injury setting with a knee that is quite stiff, there is the option to repair the meniscus only, then rehabilitate the knee to regain motion, and then perform the ACL reconstruction after approximately 8 weeks. This approach minimizes the concern with loss of motion. An alternative is to try an approximate 2-week period of rehabilitation with protected weight-bearing to regain motion prior to combined ACL reconstruction and meniscus repair. This latter alternative is often used in the chronic ACL-injured knee that presents with a locked meniscus tear. Caution should be used because prolonged weight-bearing on a displaced meniscus will damage the fragment and impair the potential to repair the meniscus subsequently.

The loss of the meniscus, particularly on the medial side, diminishes the capacity of dynamic joint compression to stabilize the knee. In addition, the loss of the medial meniscus will further increase anterior translation in the ACL-deficient knee. This has implications for ACL reconstructions because medial meniscus–deficient knees will place increased forces on a graft in the healing process, and the rehabilitation program may need to be adjusted accordingly.

Indications for Reconstruction

The primary reason to reconstruct the ACL is to provide an individual with a greater capacity to cut, pivot, and quickly decelerate. It has been well documented that an athlete with a stable knee (absence of an abnormal pivot-shift test) after reconstructive surgery will be able to participate in vigorous sports without increased risk of meniscus tears or episodes of giving way [49, 51–53]. Interestingly, Kocher et al. reported that the absence of a pivot shift was the clinical sign that had the highest correlation with the patient's subjective rating of his or her knee after reconstruction [52]. Therefore, an athlete who competes in level 1 sports (jumping, pivoting, hard cutting sports, e.g., basketball, football, and soccer) will need an ACL reconstruction if they are to return to those sports [54]. Particularly vulnerable is an athlete with an acute ACL injury in the midst of a season who attempts to return in the same season with an abbreviated rehabilitation or without a brace. This approach is not endorsed because there is an associated high incidence of giving way and subsequent meniscus tears [55].

A second group for reconstruction consists of those who have returned to moderate activities after an ACL tear only to have recurrent episodes of giving way, resulting in meniscus tears, pain, and repeated effusions. These knees have stretched secondary restraints from repeated injuries and can be quite unstable. If the medial meniscus has been resected, anterior translation is further increased. This group with symptomatic chronic ACL-deficient knees will require reconstruction.

This analysis leaves a large number of ACL-deficient patients who cannot be categorized as athletes competing in level 1 sports or individuals with significant chronic instability. Bracing, improved proprioception, and muscle strengthening have been found to help some of these patients cope with their injury if their activity level is moderated. It is believed that this group will not suffer meniscus tears or develop significant arthrosis, provided they do not have episodes of giving way or effusions. Thus, the question arises in counseling patients of whether to pursue ACL reconstruction or to recommend moderating activities. Certainly, if a person has a job or participates in a sport that requires hard deceleration and pivoting, then an ACL reconstruction is necessary; otherwise, it is good care to rehabilitate the knee and reevaluate the function periodically. Evidence suggests that it takes at least 6 months from the original injury for muscle and neurologic control mechanisms to improve to allow individuals to resume moderate activities [56].

There are also groups that consist of patients who may require an ACL reconstructive procedure because of special circumstances. This includes those with open physes, because children are inherently active and will damage their knees further if stability is not returned to the joint. Another group consists of those with persistent anterior knee pain after ACL injury that is related to quadriceps weakness and dynamic anterior tibial translation during activities. This latter motion can result in patellar tilt. In some cases, knee stability is required to diminish this anterior pain [41]. There is also a group with hypermobility: individuals in this group have lax secondary restraints that have large pivot-shift motions after the ACL is injured. This group will often have instability with even daily activities and will require surgical intervention.

Lax secondary restraints can occur with the original injury, and, if these associated injuries result in a greater pivot shift, then an ACL reconstruction will be required. There are no firm criteria on which to base this decision with collateral ligament injuries. Clearly, there are patients for whom an isolated ACL reconstruction will fail because of collateral ligament injuries, and the decision to reconstruct or reef these collateral structures is based on the severity of the pivot-shift motion that occurs. An isolated ACL reconstruction will not be adequate in some of these cases.

Partial tears that will heal and result in a stable knee must be differentiated from partial tears that represent a critical injury that will result in instability when the individual returns to vigorous activity. Unfortunately, many MRI reports identify partially torn ligaments because of uncertainty in the imaging process. A partial tear that can be treated conservatively often will have only a mild effusion associated with the original injury and a firm end point on the Lachman test at all points in the rehabilitation process. There are also knees that will redevelop an end point to a Lachman test in the weeks after injury as the torn ACL scars to the PCL. These knees may be acceptable for moderate activities, but they are not truly knees with partial tears and they will not stand up to level 1 sports. It is said that an ACL torn less than 50% will generally not require reconstruction and will function well. This may be so, but it is very difficult even during arthroscopy to evaluate the entirety of the ligament when only its anterior aspect can be visualized.

Timing of Surgery

In the first days after ACL injury, patients have varying degrees of pain, limited motion, and quadriceps inhibition secondary to hemarthrosis, associated injuries, inflammation, and neurogenically mediated muscle inhibition [57]. Patients sometimes incorrectly associate the immediate morbidity of the ACL injury with the long-term disability of the injury and therefore acutely desire an ACL reconstruction to relieve the early pain and limited function of the knee. Particularly, the dynamic instability engendered by quadriceps inhibition can result in “giving way” episodes that may be confused with the true pivot-shift instability of an ACL-deficient knee. Also, athletes often request early surgery to commence the rehabilitation process. Unfortunately, early surgery increases the risk of arthrofibrosis and associated comorbidities including patellofemoral pain. Complicating the issue is the occasional patient who may have a retracted ACL stump that impinges in the intercondylar notch that prevents full extension (Fig. 4). Rarely, such a patient will require a preliminary arthroscopy to debride this stump, thereby removing this impediment to full motion before proceeding with ACL reconstruction.

There are no established preoperative motion or muscle function criteria to permit reconstructive surgery, but certainly full extension with a good quadriceps contraction and close to full flexion is desirable. Often in the acute setting, it is difficult to perform a quadriceps setting maneuver and to hyperextend the knee from a supine position, but a patient

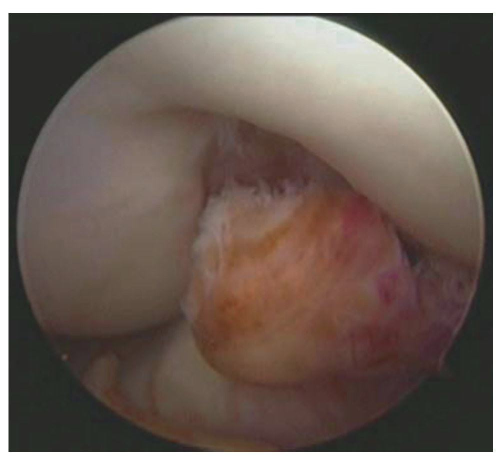


Fig. 4 The retracted stump of the native ACL as visualized arthroscopically 6 weeks after injury. A slow return of extension or the loss of extension after injury may indicate impingement of a retracted ACL stump

who can perform this maneuver will have the best opportunity to regain full extension after surgery. It has been reported particularly in skiers that several days of vigorous rehabilitation including use of constant passive motion can permit an early reconstructive surgery with a small incidence of long-term arthrofibrosis. However, this very early surgery group of patients may require subsequent procedures before regaining full function.

In general, the inflammation after injury mirrors that seen in other joints and tissues after injury. Often, it takes at least 3 weeks for motion and quadriceps strength to return and truly 8 weeks for the knee to look close to normal. Some athletes, for example interior lineman in football, seem to particularly have a prolonged loss of motion with injury, and they may need extensive rehabilitation prior to surgery.

Graft Options

Each patient needs to know his or her graft options and be guided through the graft choice process. However, patients and surgeons have to contend with the reality that inside and outside the medical community there are advocates for various grafts, and the dilemma of which graft to use may be based on such outside influences. Given the small differences in results between different grafts, a choice based on the advice of peers, coaches, or others may have little long-term impact. This is supported by the reality that support exists for all the commonly used grafts for almost any clinical situation. It is a common teaching that graft choice is relatively unimportant, and concerns with graft placement and graft fixation are more important. However, there are differences between grafts as they impact muscle strength, range of motion, healing, stability, pain, cost, and potential for disease transmission.

Hamstring tendons were the first reported graft to reconstruct the ACL, and they continue to be a very popular graft [58, 59]. The pes anserinus anatomy varies between patients, and particularly the semitendinosus fascial attachments to the crural fascia can make harvesting this tendon a challenge for the inexperienced. Postoperative soreness in the posterior thigh and lower leg in the first weeks after surgery can be a burden after hamstring harvest, but rarely does this have a long-term effect. “Strains” in the area of harvest can occur with an accidental eccentric stretch in the first weeks after surgery, and patients should be advised of this possibility and that it can be disabling for 2 or 3 weeks. Hamstring curls should be avoided for the first 8 weeks after harvest.

Clinically, patients feel their hamstring deficit has resolved by 4 to 6 months after reconstruction, but isokinetic testing will reveal slight deficits in knee flexion torque for up to 1 year, but only very rarely will a patient continue have long-term discomfort in the hamstring area. After harvesting the gracilis and semitendinosus tendons, MRI studies have documented healing of these muscles to the semimembranosus and posterior knee fascia. Knee flexion torques ultimately return to normal, but a small loss of internal rotation torque can be measured because the muscles never reattach to the anteromedial tibia [60]. It has been conjectured that a soccer player who requires internal rotation strength to kick the ball will be affected by this loss, and it is debated whether competitive sprinters may have some functional weakness after hamstring harvest. These are concerns based on individual observations.

It is generally accepted that a four-strand gracilis-semitendinosus graft functions better than a two-strand semitendinosus graft, but this necessitates tensioning all four strands equally at the time of fixation [61, 62]. The size of the graft is related to the height, weight, and thigh circumference of the patient, and, although there is variability, four-strand grafts are virtually always between 7 and 10 mm in diameter [63]. An individual with a smaller graft does not seem to have a compromised result.

The central third patellar tendon bone-tendon-bone graft is reported to still be the most commonly used graft in the United States, but concerns with its morbidity have diminished its use. Most patients will have discomfort if they kneel on the distal pole of the patella after its harvest, but rarely does this cause a functional deficit. Most athletes will have symptoms referable to the harvest area for 6 to 9 months after surgery, and it is common for them to ice this area when first returning to sports. Only rarely will there be permanent soreness in the proximal harvest site [25, 64]. It is incumbent upon the surgeon to initiate an accelerated rehabilitation program if a patellar tendon graft is used, and this includes the avoidance of bracing, use of constant passive motion in the first few weeks, and an emphasis on regaining quadriceps function and full knee motion early after surgery. Particularly in high school and college athletes who are aggressively rehabilitated, this graft can be very successful. Care should be taken to not overtension the graft and to place the knee in full extension at the time of fixation. There has been evidence that patellar tendon grafts predispose the knee to arthrosis over hamstring tendon grafts, but this may not be the case if the knees are properly rehabilitated in the first weeks after surgery.

Quadriceps strength is impaired in virtually all ACL-injured patients as a response to the loss of afferent nerves from the native ACL, and 0.25-inch to 0.5-inch thigh atrophy is commonly documented with or without ACL reconstruction. Use of the patellar tendon graft accentuates this quadriceps atrophy, and it takes more than a year from surgery to regain close to normal strength. To minimize the impact of ipsilateral harvest on quadriceps strength and to speed recovery, it has been proposed that the graft be harvested from the contralateral side [65]. Though logical, this approach creates a bilateral disability that can increase the ramifications of a complication.

In the young athlete who will aggressively rehabilitate his or her knee and in an individual with a very loose knee, the patellar tendon continues to be a good choice. The highly rigid fixation afforded by metal interference screws limits any stretching at the fixation site in the first weeks after surgery. There is evidence that this graft has the fastest incorporation into the intraosseous tunnel with rapid bone-to-bone healing.

A partial-thickness quadriceps tendon graft with or without a bone plug from the proximal patella is another autograft option. It is common to harvest a graft with a thickness of 7 mm, width of 10 mm, and length of a least 7 cm [66]. Unfortunately, there is some painful morbidity with use of this graft, and it has not become popular probably because of this fact. Pain may be related to harvesting technique, and clearly, removing a full-thickness graft is more disabling than the 7-mm partial-thickness harvest required for an ACL reconstruction. If an autograft option is needed and the patellar tendon and hamstrings are not preferred, then this provides a good alternative.

Allografts have become more popular in the past few years because of their good clinical results and modest surgical morbidity. Operating times are reduced with allografts versus autografts, and many patients are struck with the minimal morbidity of the procedure. Unfortunately, there are no good comparative studies between autografts and allografts, and generally there has been a selection bias whereby older, less athletic patients tend to be the group choosing allografts. This may be a reflection of concerns with disease transmission and a desire to minimize these risks in the young or a clinical impression supported by some animal data that allografts heal more slowly than do autografts and they may have higher failure rates in young aggressive athletes.

The risk of disease transmission with allografts must be explained to patients and the history of infections associated with allograft use recounted. There was a case of HIV transmission more than 25 years ago and some cases of hepatitis C transmission to patients through use of allografts [51]. There is also documentation of multiple bacterial infections (approximately 40) that can be traced to allograft use. Patients should be informed that this represents the numerator of the equation, and data suggest that perhaps 40,000 allografts are used per year for knee reconstructions in the United States, so the chances of contracting an infection are very, very small. It is strongly suggested that hospitals procure grafts from an accredited tissue bank, and surgeons should know the proprietary processing method used to render grafts statistically sterile.

The tibialis tendon allograft has become very popular and possibly replaced the patellar tendon allograft in usage because it allows smaller tunnels, greater options for fixation, and no dependence on allograft bone quality. Particularly, the common availability of only bisected patellar tendon grafts can result in bone plugs of poor quality.

Unfortunately, there is limited clinical data comparing the tibialis allografts with patellar tendon allografts, and ultimately graft placement and fixation may be the major variables explaining results. Interestingly, the many studies documenting success with allografts have reviewed the use of bone-tendon-bone allografts.

It is a concern that irradiation is still used to terminally sterilize some allografts, because even low doses (1–2.5 Mrad) diminish the biomechanical properties of the graft [67]. Laboratory data have supported the practice of pretensioning irradiated allografts to remove crimp and more closely restore the grafts to a state of "nonirradiated" stiffness.

Healing is slower with allografts, and often surgeons slow the rehabilitation protocol slightly when they are used. Interestingly, allografts are often used for revisions where there may be stretched secondary restraints that will place greater tension on the reconstructed graft. It would perhaps be more logical to use a patellar tendon autograft in revision surgery because this graft allows a strong and stiff fixation. However, the possible prior use of this graft and the desire to minimize surgical morbidity often results in use of an allograft for a revision.

The two variables most commonly sited for choosing one graft over another are concerns with the extensor mechanism and concerns with limited range of motion. There is a clinical sense that allografts will produce the least stress to the knee and will be preferred when motion concerns are greatest, that hamstring grafts would be intermediate in their effect on motion, and that patellar tendon autografts would theoretically pose the greatest risk to the return of full motion. There is logic to this approach and some evidence to support the observation, but patient variability is great and the art of medicine is discerning when these concerns are real and if they should affect the choice of a particular graft.

Reconstruction Technique

The patient's operative leg is either positioned in a leg holder with the foot of the bed flexed down or positioned with a side post to the bed and the foot of the bed up. The leg holder method facilitates the application of varus and valgus torques to accomplish meniscal procedures, but it prevents full flexion of the knee, which compromises drilling through an anteromedial portal. If the foot of the bed is kept up, padded posts can be used to stabilize the knee in flexion and facilitate the application of valgus torque. One post is placed lateral to the thigh, and another is placed across the bed to buttress the foot. These posts can be positioned to stabilize the knee in slightly less than 90 degrees of flexion.

A tourniquet is not necessary, but operation times are reduced if a tourniquet is used, particularly during the tunnel-reaming portion of the procedure. There is evidence that quadriceps weakness in the first weeks after surgery is less if tourniquet times are kept to under 1 hour [68].

The notchplasty provides several benefits: visualization of the ACL femoral attachment, prevention of graft impingement on the roof of the notch, and prevention of graft abrasion on the lateral wall of the notch [69, 70]. A well-chosen anteromedial portal should provide easy access to the notch without traumatizing the anteromedial condyle. Generally, an abrader is used to clear approximately 2–3 mm of the lateral and superior walls. Care is taken not to resect the lateral wall deep within the notch to prevent lateralization of the graft. Extending the knee while the notch is visualized provides a perspective on the potential for impingement and may direct the surgeon on how much bone to remove. A rare complication to avoid is the resection of the anterior notch to a point where the patella will fall into the notch when the knee is fully flexed and result in patellar crepitus. Knees that are chronic ACL-deficient will have developed spurring or bossing at the anterior notch and will need a greater resection.

Tunnel placement is the critical key to ACL surgery [71–76]. Whereas some knees are forgiving because the roof of the notch is more horizontal and open anteriorly, other knees will require very well placed tunnels for the surgery to succeed. If the transtibial drilling technique is used, this compromises tunnel placement, and in some knees the tunnels may not be satisfactory [77, 78]. The tibial tunnel should exit on the tibial articular surface slightly medial to the anatomic center of the insertion site (Fig. 5). Slight medialization of the tibial tunnel will prevent the graft from abrading on the lateral wall of the notch. The center of the tibial insertion should be approximately 15 mm anterior to the PCL notch, and the posterior border of the tunnel should be anterior to the peak of the medial tibial eminence. Transtibial drilling may result in a posterior tibial tunnel that could lead to a more vertical graft [79].

The portal used to introduce the drill guide affects the ability to start the tunnel on the anterior tibia, and an auxiliary anteromedial portal can be made slightly superior to the articular surface to introduce the drill guide. The tunnel can be started just medial to the tibial tubercle where the bone quality is best and the drill guide angle chosen to create a tunnel that has a 30-mm minimum length. A starting point too far medial may jeopardize the medial tibial articular surface when it enters the joint [80]. If a patellar tendon graft is used, consideration should be given to graft-tunnel mismatch where the bone plug extends out the tibial tunnel. A helpful method is to measure the distance between the two bone plugs on the patellar tendon graft and set the drill guide angle approximately 7 degrees greater than this measurement [81]. It should be remembered that the portal used to introduce the aiming arm of the drill guide and the inclination in which the guide is held will strongly affect this method. Use of a lower anteromedial portal and holding the aiming arm horizontal will tend to lengthen the tibial tunnel. Still, a patellar tendon graft with more than 55 mm in length between the bone plugs is difficult to position to facilitate intraosseous tibial fixation.

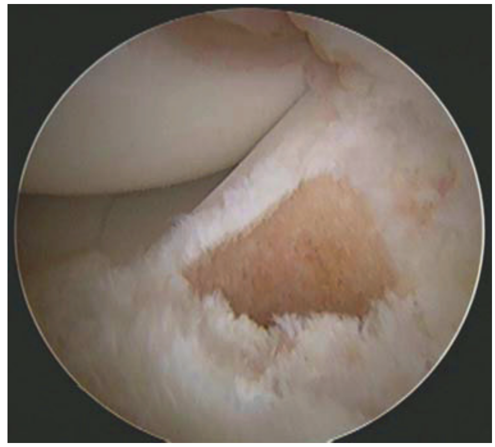


Fig. 5 A reamed tibial tunnel visualized arthroscopically. The tunnel is placed slightly medial in the footprint with its center approximately 15 mm anterior to the PCL fossa. The medial tibial eminence also serves as a landmark based on the observation that the posterior border of the ACL inserts anterior to the peak of the medial tibial eminence

Once the tibial tunnel guide pin is placed, a helpful technique to move the tunnel slightly is to first ream with a small reamer, 6 mm for example. The guide pin can then be manually replaced eccentrically in the small tunnel and the tunnel reamed again with the appropriate-sized reamer. This method can fine-tune the placement of the tunnel by a few millimeters.

Placement of the femoral tunnel is challenging because the visualization of the notch varies based on the angle of knee flexion. Tunnel placements based on clock-face descriptions of the notch are imprecise unless a reference is provided for the 3–9 o'clock axis. A practical method to place the femoral tunnel based on cadaveric observations is to first position the knee in 90 degrees of flexion. Two points are then identified: first, the apex of the notch, and second, the lowest point on the lateral wall. The point halfway between these two points is the anatomic center of the ACL (Fig. 6).

An auxiliary anteromedial portal is necessary to drill the femoral tunnel because the standard anteromedial portal is generally too superior and medial to provide the correct access for pin placement and reaming. The auxiliary anteromedial portal is placed just superior to the medial meniscus and lateral to the medial condyle to avoid femoral

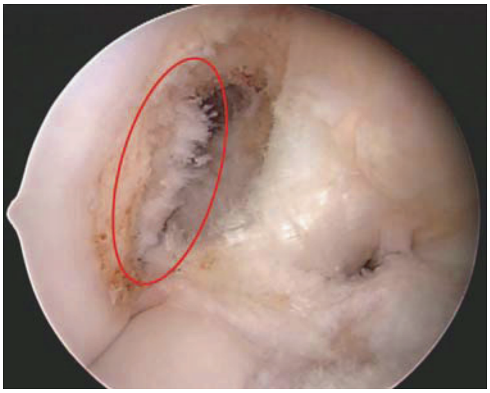


Fig. 6 The femoral footprint of the ACL visualized arthroscopically (oval outline imposed on the image). The insertion is on the lateral wall of the intercondylar notch. When visualized arthroscopically from the anterolateral portal, the center of the insertion is approximately halfway between the apex of the notch and the lowest point on the lateral wall

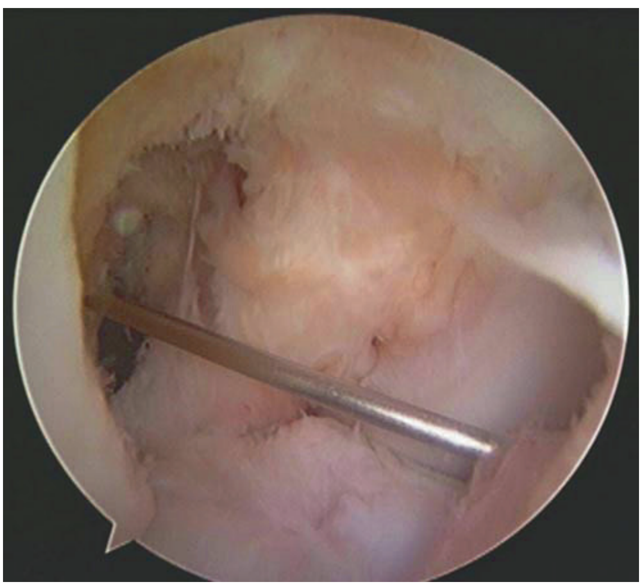


Fig. 7 A trial placement of a spinal needle can identify the correct position of an auxiliary anteromedial portal. This portal should be just above the anterior horn of the medial meniscus and directed toward the femoral insertion. It must be correctly located to avoid articular injury to the medial condyle during the reaming process. In this revision procedure, there is ample room for a new femoral tunnel on the lateral wall away from the prior femoral tunnel that lies high in the notch

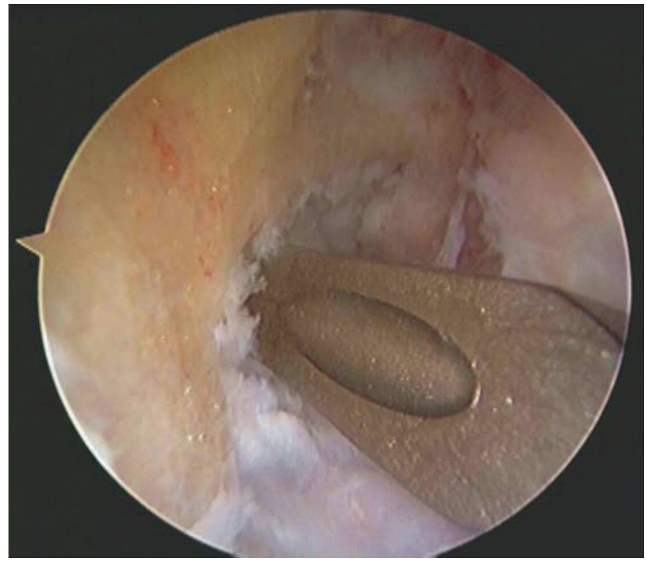


Fig. 8 The correct placement of an offset guide for guide-pin placement is shown arthroscopically. The tunnel should be located halfway between the apex of the notch and the lowest point on the lateral wall. This is a view with the knee flexed approximately 90 degrees, but the knee must be flexed more than 125 degrees during guide-wire placement and during reaming to prevent broaching the posterior femoral cortex

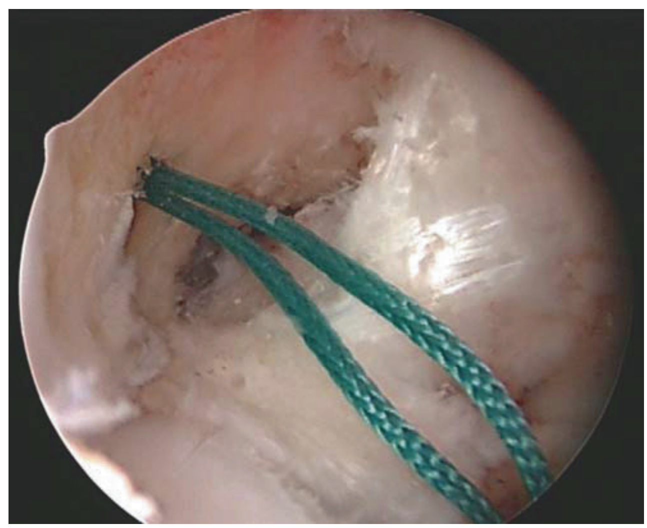


Fig. 9 A femoral tunnel centered in the ACL insertion on the lateral wall of the intercondylar notch

articular injury during reaming (Fig. 7). In the anteromedial drilling method, a 7-mm over-the-top drill guide is introduced to place a guide pin at the anatomic center of the ACL (Fig. 8). While maintaining pressure on the guide pin to ensure it does not move, the knee is flexed to more than 125 degrees. This is challenging because visualization of the notch is impaired at greater flexion angles. This is secondary to displacement of the fat pad back into the notch and because, at higher flexion angles, the roof of the notch angles directly down to the tibial plateau. Once the knee is maximally flexed, the guide pin is drilled out through the lateral thigh and then reamed with the appropriate-sized reamer. A graft passing suture is attached to the base of the guide pin and pulled out the lateral thigh, then pulled down through the tibial tunnel to facilitate graft passage. At this point, the knee can be extended again to 90 degrees and the placement of the tunnel evaluated (Fig. 9).

The graft is then passed across the knee using the passing suture and secured on the femoral side either with a cross pin, button, or intraosseous method. Tensioning and fixation of the graft on the tibial side is critical and dependent on the type of graft, method of fixation, and laxity of the knee. Most grafts when placed by this method will lengthen with knee extension and have a capacity therefore to restrict extension not because of impingement but because of excessive tension. Practically, this is important for patellar tendon grafts because the bone-to-bone fixation of patellar grafts results in a stiff construct that may not lengthen sufficiently to allow extension. Therefore, it is suggested that patellar tendon grafts be tensioned and secured in close to full extension. Hamstring and allograft tendons exhibit slightly greater viscoelastic creep, and they may be tensioned in some knee flexion to provide a graft with greater tension. Generally, for revision procedures, greater tension is required in the graft and some greater knee flexion is helpful.

Tensioning of multistranded grafts requires that all strands be tensioned equally to obtain the full strength of the graft [62]. A practical method to accomplish this is to tie the sutures together at the ends of each tendon graft, thereby creating essentially a loop attached to both ends. For a two-strand graft, tension can be applied equally to each strand by pulling on this loop. For a four-strand graft, first the sutures at the ends of each tendon are tied together to create two independent loops. Then a separate suture is passed through the two loops and tied to itself. Pulling on this last suture between the two tendon loops equally tensions all four strands.

If the transtibial drilling method is used, then compromises must be made in the positioning of the tibial tunnel to facilitate an acceptable femoral tunnel (Fig. 10). Starting further medial on the tibia will slightly lower the femoral tunnel when drilled transtibial, but another critical variable is the length of the tibial tunnel. In general, a transtibial tunnel should begin close to the joint line – a practical method would be 20 mm below the joint line – to facilitate femoral tunnel drilling. The drill guide angle for this might be as little as 40 degrees. The tibial tunnel may require positioning in the posterior tibial footprint as another compromise.

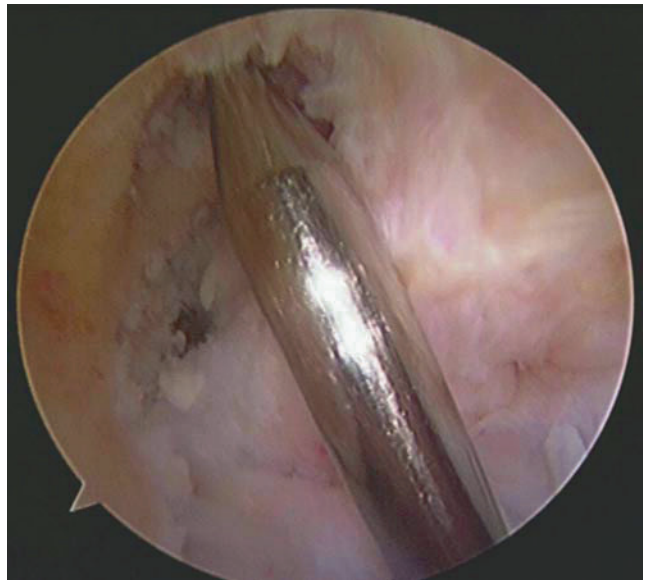


Fig. 10 A switching stick placed through the tibial tunnel identifies a point where the transtibial drilling method would place a femoral tunnel. The point identified is high in the intercondylar notch and outside the footprint of the ACL

After fixation, the graft should be checked for tension, impingement, and alignment (Fig. 11A, B). Patellar tendon grafts may appear slightly lax if secured with the knee in extension, but this is expected and often no retensioning is necessary to produce a knee with symmetric laxity to the other side. If tension has been lost in soft tissue grafts, they

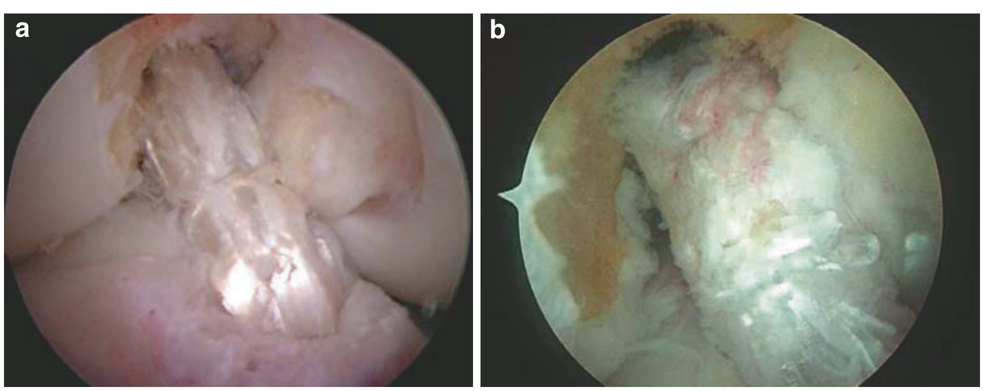


Fig. 11 Two graft placements produced by different drilling methods are compared. In (**A**), a more horizontal hamstring graft has been placed in a femoral tunnel produced by anteromedial drilling, whereas in (**B**), a more vertical patellar tendon graft has been placed in a femoral tunnel produced by transtibial drilling.

can be retensioned and resecured. Care should be taken to check for abrasion of the graft on the lateral wall or impingement on the roof of the notch. A rasp passed through the anteromedial portal can provide further notch resection if it is necessary.

Rehabilitation

Before surgery, the goal of full extension and good flexion should be underscored with the patient and the important role of a quadriceps contraction to regain extension explained. A strong quadriceps contraction can translate the patella superiorly to stretch the parapatellar tissues and thereby minimize many of the injurious compressive forces on the patella. Whereas excessive quadriceps strengthening in the first 8 weeks can cause chondral injuries, such overtraining should be differentiated from the necessity of leg raises and knee extensions with light weights (1–2 kg) and use of a stationary bike. Graft fixation must allow full extension with a good quadriceps contraction immediately after surgery. At the first postoperative visit, the goal of motion and particularly full extension can be taught again and the ability to do a short arc squat promoting a quadriceps contraction demonstrated.

Flexion is facilitated by use of a continuous passive motion (CPM) machine for the first 2 weeks after surgery. The appropriate time and motion settings for the CPM machine should be less than would be used for a cartilage restorative procedure, because excessive use of the CPM machine can contribute to hemarthrosis in the first days after surgery. An initial setting of 0–30 degrees for two 30-minute sessions per day is a good beginning, and, if this is tolerated, the flexion angle and time in the machine can be increased based on comfort.

Bracing after surgery has not been shown to have an impact on results, but it is a good method to slow the overzealous patient and physical therapist in some situations. The disadvantage of bracing is its weight and general encumbrance, which may limit motion. Crutches may be the best device to limit excessive activity in the first few weeks after surgery. A weight-bearing as-tolerated regimen with an adherence to crutch use in the first 2 weeks after surgery will promote normal full knee extension and limit excessive loads produced by a rapid gait. In the occasional patient with questionable graft fixation or in a revision situation in which added protection is necessary, the extended use of the crutches for up to 4 weeks can be useful. It should be explained that crutches are used to limit forces and to encourage normal gait mechanics and not for patient comfort.

The first 8 weeks after surgery is a time for graft-to-bone healing, and vigorous resistance training should be minimized. This is particularly a concern when an allograft is used because there is often minimal postoperative pain. When the goals of full motion and a good quadriceps contraction are met, it is helpful to use a stationary bicycle as an excellent method to regain knee function without overly stressing the reconstruction. If a hamstring graft is used, knee flexion curls should be avoided until 8 weeks after surgery to prevent injury to the graft harvest site.

The 2- to 4-month period after surgery is a time for vigorous strengthening provided the patellofemoral joint is asymptomatic and the parapatellar tissues have regained reasonable flexibility. Jogging and running can begin at 3 to 4 months based on the patient's progress, and full sports at 6 months. It should be remembered that patients heal at different rates, and good proprioception, balance, and strength may not return for up to 12 months or longer in some cases. A strength and conditioning program that underscores sports-specific training beginning at 3 to 4 months is an excellent method to return an athlete to competition.

Complications

Infection, deep vein thrombosis, loss of motion, patellofemoral pain, and general pain syndromes are the major complications that can significantly diminish the results of ACL surgery. Particularly for infection and deep vein thrombosis, the early diagnosis of these problems can minimize their morbidity.

The diagnosis of intraarticular infection is challenging because some patients will have postoperative temperature elevations even up to 102°F in association with a painful hemarthrosis that may look like an infection in the first 5 days after surgery. Generally, this benign elevated temperature and pain condition will start to resolve by the fifth postoperative day. The clinical signs of infection 5 days or more after surgery include increased pain, diminished ability to bear weight, loss of motion, and the persistence of a large effusion. The practical approach is to aspirate a knee if these features are present and send the fluid for a cell count, Gram stain, and culture, protein, glucose, and crystal analysis. Blood work is helpful but sometimes difficult to interpret. Generally, an erythrocyte sedimentation rate (ESR) up to 50 will be observed in the first 2 weeks after surgery; then it will slowly return to normal by 2 to 3 postoperative weeks. Certainly, a knee should be aspirated when a concern arises.

The debate over leaving the graft in place or removing it if an infection is present is based on personal experience and anecdotal reports. If there has been a delay in the diagnosis or if the knee is not responding to an initial arthroscopy and lavage, then removal of the graft should be strongly considered. There is evidence that early graft removal and prompt eradication of infection will diminish the arthrosis that can occur after infection [82]. A revision ACL reconstruction can be performed when antibiotic treatment is completed and the markers for inflammation (ESR and C-polysaccharide reactive protein (CRP)) have returned to normal.

Deep vein thrombosis and pulmonary embolism do occur after ACL reconstructions, and preventative measures should be used. Women taking birth control pills should be strongly encouraged to stop these before surgery and for several weeks after surgery. Aspirin prophylaxis should be routinely used unless there are contraindications. During surgery, the well leg should be padded and the operative leg prevented from dangling in the leg holder. Techniques to support the lower leg during surgery rather than have it hang with pressure from the leg holder on the posterior thigh include supporting the foot in a basin or directly supporting the foot with the surgeon's thigh. Often, a deep vein thrombosis will present with a patient noticing a loss of ability to bear weight because of calf pain a few days after surgery. A venous ultrasound can make the diagnosis, and appropriate care should be initiated. Patients are at risk for deep vein thrombosis for at least several weeks after surgery, and they should be cautioned of this risk if prolonged travel is contemplated.

Loss of motion problems occur but they are less common over the past two decades because of a greater emphasis on obtaining normal motion prior to surgery [57, 83]. Loss of motion problems are also diminished by proper graft placement and proper graft tension. An improperly placed graft can result in loss of motion, and it may have a poor biomechanical axis to resist anterior translation (Fig. 12).

Fig. 12 A CT scan of a failed ACL reconstruction demonstrates a steep (22-degree) inclination of the intercondylar roof, which predisposes the graft to impingement when the knee is brought into extension. An average inclination of the roof relative to the femoral shaft is 36 degrees. In this failed reconstruction, significant osteolysis surrounds the tibial tunnel. A tibial tunnel placed further posterior and a femoral tunnel placed further posterior and lower would be necessary with this type of anatomy.

Patellar tendon graft tensioning is particularly critical because the graft is stiff and because of quadriceps inhibition that can occur after its harvest. Caution should be exercised to not overtension a patellar tendon graft, and attempts to posteriorly translate the tibia and fix the patellar tendon graft with the knee in flexion may result in a flexion contracture. The one exception to this approach may be revision procedures in which graft motion within previously used tunnels may preclude rigid fixation.

If loss of extension does occur, it is best to let the inflammation of surgery resolve before attempting any remedies in the operating room. A good physical therapist who emphasizes early extension and the addition of electrical stimulation to generate a strong quadriceps contraction can obviate the problem. If motion is improving, it may be better to allow the healing process to evolve, and often knees will regain the last degrees of extension over several months. If there is a greater loss of motion or if little improvement is seen, then waiting at least 2 or 3 months is advisable. Arthroscopy may then be helpful to clear the notch of scar combined with a manipulation of the knee into extension and flexion. Rarely, a cylinder cast may be used to hold the knee in extension for 10–14 days. The thigh portion of the cast is applied first and allowed to harden before the knee is held in extension, and the lower portion of the cast is applied. This is a very rare occurrence and may need to be combined with an open debridement of infrapatellar scar if patella infera is identified. Flexion is generally easier to recover by simply manipulating the knee. Very rarely, a knee with severe loss of motion secondary to poor graft placements may require graft removal and prolonged rehabilitation (see Case 2 later in this chapter).

Unfortunately, patellofemoral pain is a common occurrence with ACL injuries, and it may be combined with loss of motion. The etiology for patellofemoral pain may relate to quadriceps inhibition that can be quite striking in some individuals when afferent nerves within the ACL are disrupted. Another factor is the altered patellar tracking with a quadriceps contraction in the ACL-deficient knee near extension. The dynamic anterior translation of the tibia may redirect the patella laterally to change patellar contact forces and result in anterior knee pain. Lastly, the general inflammation of the joint can result in decreased patellar mobility and increased patellar compression forces. Sometimes all three of these factors – quadriceps inhibition, altered patellar tracking, and increased patellar pressures

– are present, and it can be quite challenging to treat. Hopefully, these problems can be identified before surgery and a rehabilitation program initiated to improve the knee before reconstruction. A lucid explanation of the etiology for the patellofemoral pain will dampen the intuitive desire of many patients to proceed with the surgery to alleviate their symptoms. Unfortunately, a further stress to the knee of a surgical reconstruction may exacerbate patellofemoral pain and loss of motion. The one exception is the patient with a full range of motion and patellofemoral pain secondary to quadriceps inhibition relating to the dynamic tibial translation. In some cases, an ACL reconstruction in this setting is warranted provided the surgeon and patient are confident that full motion can be achieved after surgery. Whereas a painful knee may be limiting, a painful stiff knee may be disabling.

Clinical Pearls

Visualization of anatomy is the key to all surgery whether open or arthroscopic, and, in ACL surgery, this principle particularly applies to obtaining a good visualization of the intercondylar notch. This is particularly important for referencing the locations of the tibial and femoral tunnels. Interestingly, the best visualization of the notch is provided when the knee is flexed approximately 60 degrees. In this position, the roof of the notch is parallel to the tibial plateau, and an excellent view is afforded from anterior to posterior. Extending the knee from this position allows the surgeon to visualize the possible tibial tunnel locations relative to the top and lateral wall of the intercondylar notch. Referencing the notch to the tibial plateau in this manner combined with an understanding of the tibial footprint anatomy are the key components to proper tibial tunnel placement.

A simple method to improve the tibial tunnel location, if the first guide pin placement is as close to the desired position but not ideal, is to first ream over the guide pin with a reamer slightly smaller than the desired diameter. The guide pin can then be removed and repositioned by hand eccentrically in the tunnel toward the desired direction and impacted into the roof of the notch to secure its position. This second guide pin placement is then over-reamed to the appropriate diameter to place the tunnel in an ideal position.

Tensioning of grafts is an art based on the surgeon's understanding of the knee's laxity combined with an intuitive sense of how well the patient will regain quadriceps strength after surgery. Furthermore, in almost all reconstructions, grafts will come under greater tension with knee extension. Based on these understandings, the following generalizations can be made for graft tensioning: (a) Patellar tendon grafts are stiff, and these grafts should be tensioned with the knee in close to full extension; (b) hamstring tendon and allograft replacements will stretch despite equal tensioning of all limbs, so some slight flexion of the knee is appropriate when these grafts are secured; (c) lastly, most revision surgeries are in knees with some secondary collateral ligament laxity and in knees with less than ideal tunnel to graft apposition, so in revision surgeries grafts should be tensioned with greater force and with the knee flexed approximately 30 degrees.

Summary

ACL surgery has evolved over the past few years secondary to a better understanding of ACL insertional anatomy and secondary to a better understanding of graft placement effect on knee stability. Femoral tunnels should be placed in the center of the femoral footprint, and tibial tunnels should be placed as anterior as possible in the footprint, provided impingement on the roof and lateral wall of the notch does not occur. The choice of graft is controversial and unresolved. In rehabilitation, all agree that full motion should be regained rapidly to avoid the clear disability of prolonged stiffness. Lastly, despite our best efforts, the ACL-reconstructed knee appears to never be a normal knee, but a good ACL reconstruction can allow athletes to return fully to their sports, and, if the menisci are intact and if there is no residual stiffness, the chance of arthritis in the future is minimal.

Case Reports

Case 1

Chief Complaint and Patient History: A 27-year-old man presented 5 days after a direct contact valgus injury sustained in a flag football game. He wished to return to level 1 sports and desired urgent surgical care to remedy his condition.

Physical Exam: He had a fair quadriceps contraction, moderate knee effusion, and range of motion from 20 to 65 degrees. His examination was notable for an intact extensor mechanism, a positive Lachman test, and 5 mm greater medial joint-line opening on valgus stress in flexion compared with that of the opposite knee.

Imaging: His MRI scan was consistent with a grade 2–3 medial collateral ligament (MCL) sprain, a lateral meniscus tear, and an ACL tear (Fig. 13A).

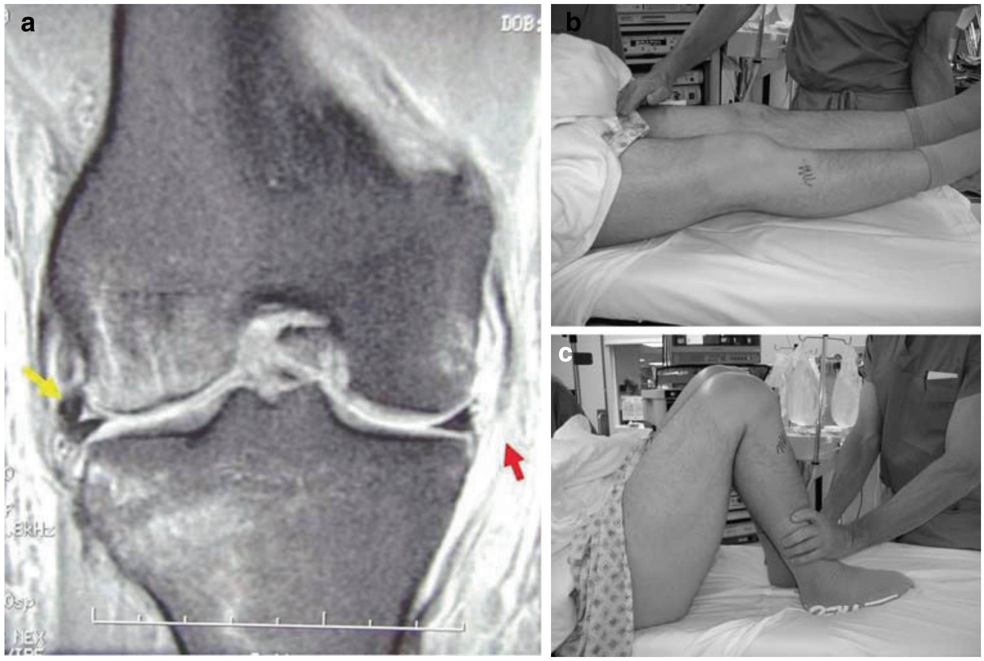


Fig. 13 Case 1. (**A**) The MRI scan demonstrates bone contusion predominately of the lateral femoral condyle and lateral tibial plateau consistent with ACL and MCL injuries. The MCL identified by the *red arrow* is discontinuous in some areas, representing a grade 3 injury. There is also a displaced fragment of the lateral meniscus identified by the *yellow arrow*. (**B**) At the time of surgery, it is demonstrated that the knee had regained very close to full extension. (**C**) It required 8 weeks of rehabilitation to regain close to full flexion in this case. Flexion is often the slowest motion to return after an MCL injury. The inflammation at the sight of injury limits the extensibility of the healing tissues, and motion is lost because the ligament must lengthen to allow flexion.

Surgery/Treatment: Concerns with arthrofibrosis were explained, and he was referred to physical therapy, but he returned in 2 weeks with motion only slightly improved from that at his initial examination. His knee was aspirated for 50 mL of serosanguineous fluid. A home CPM machine was prescribed, and a physical therapy program was prescribed that included electrical stimulation to the quadriceps. After 8 weeks, he had regained all but several degrees of extension and flexion (Fig. 13B). At this point, he underwent an allograft ACL reconstruction and partial lateral meniscectomy, and over several months he regained the last degrees of extension and flexion (Fig. 13C). At the time of surgery, his medial compartment did not open more than normal, and he was judged to be stable to valgus stress.

Discussion: This case underscores the vulnerability to motion loss with direct contact injuries of the ACL and MCL. Some knees will have a significant period of inflammation that will require an understanding by the patient of the rationale for postponing reconstruction. Very few of these knees will require surgery on the MCL because the ligament will usually heal well, and concerns are greater for permanent loss of motion rather than for valgus instability. This type of case provides a relative indication for use of an allograft and the avoidance of a patellar tendon graft to avoid concerns with arthrofibrosis and loss of motion.

Case 2

Chief Complaint and Patient History: A 17-year-old girl who was the outstanding player and captain of her high school basketball team presented 11 months after a hamstring graft ACL reconstruction with loss of motion. The ACL was ruptured in a noncontact basketball injury, and she underwent reconstruction 10 days later. It was unknown what her motion was before surgery, but she underwent a manipulation 3 weeks after surgery and an arthroscopy 8 weeks after surgery to help regain motion. The reconstruction technique employed transtibial drilling of the femoral tunnel and fixation with a femoral cortical button and a tibial bioabsorbable screw. Postoperatively, she worked with a physical therapist for several months, but her range of motion did not improve beyond 15–110 degrees of flexion. She was coping with her injury and had even returned to basketball, but she was limited in her capabilities.

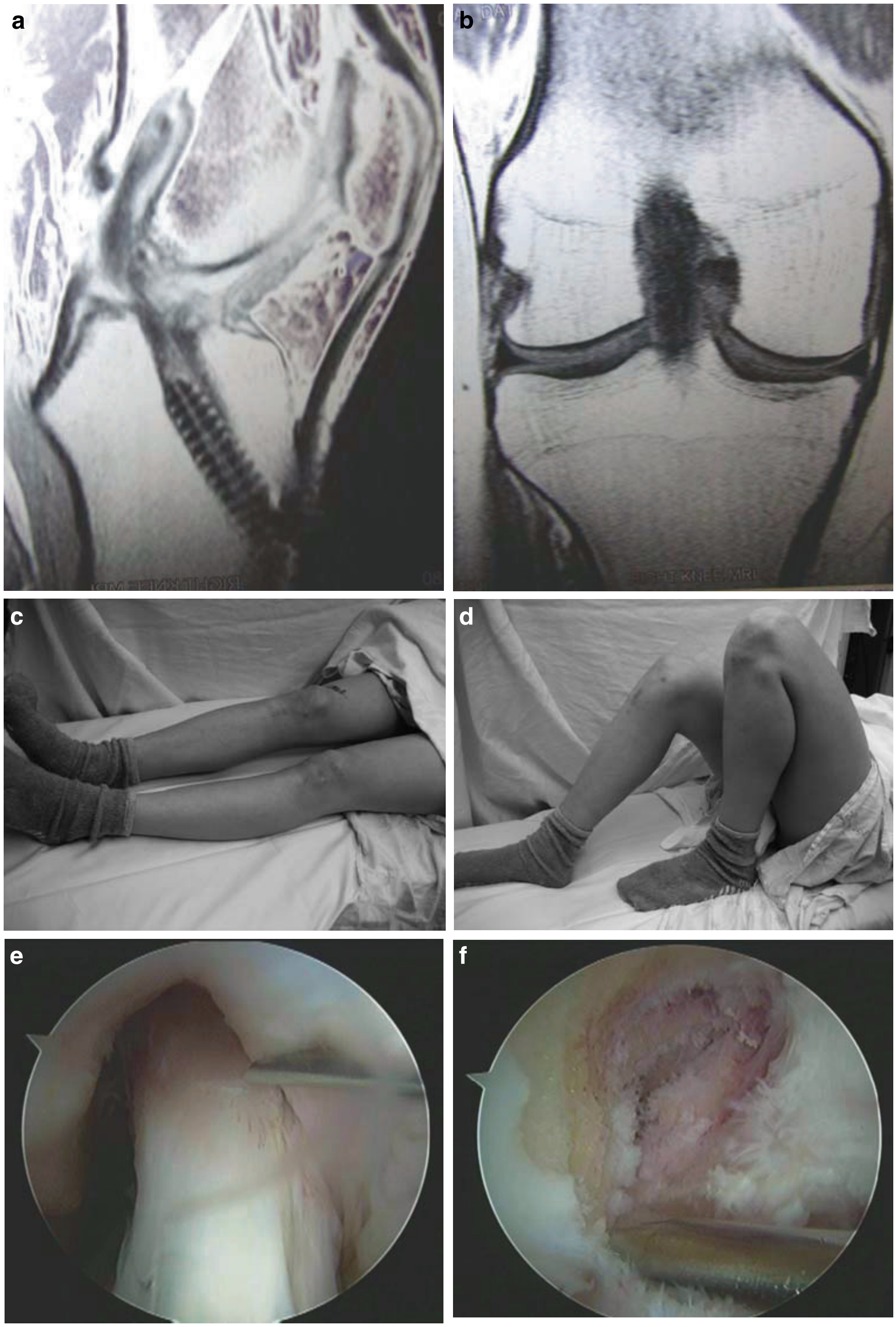


Fig. 14 Case 2. (**A**) The sagittal MRI scan demonstrates a graft that appears posteriorly placed on the tibia to prevent impingement but a femoral tunnel located in the third quadrant from anterior to posterior along Blumensaat's line. (**B**) The coronal-plane MRI scan demonstrates an almost vertical graft. (**C, D**) More than a year after the patient's initial surgery, there was an approximate 15-degree flexion contracture, and full flexion was limited to 110 degrees. (**E**) A vertical graft was identified at arthroscopy and verified by the ability to visualize the entire lateral wall of the notch. (**F**) After graft removal, the femoral tunnel is seen to lie outside the anatomic insertion of the native ACL. The native ACL inserts on the lateral wall of the ACL notch.

Physical Exam: Her examination revealed no tenderness, no effusion, and a good quadriceps contraction, but 0.5-inch of thigh atrophy. Medial-lateral patellar mobility was slightly limited compared with that of the opposite side.

Imaging: Radiographs revealed only mild infrapatellar contracture. The MRI scan demonstrated a vertical graft that had a femoral tunnel positioned in the third quadrant from anterior to posterior along Blumensaat's line (Fig. 14A, B).

Treatment/Surgery: After much discussion, it was resolved that she would finish her senior basketball season before addressing the limited motion of her knee. She returned in 3 months with an examination unchanged from that at presentation. At surgery, the knee could be manipulated only a few degrees beyond her preoperative motion, and, during arthroscopy, a vertical graft was identified that did not impinge in the notch (Fig. 14C–E). It had been preoperatively discussed with the patient that removal of the graft would probably be necessary to regain motion, and this was done, revealing a femoral tunnel near the top of the notch (Fig. 14F). Also, a medial parapatellar incision scar was excised from beneath the patellar tendon down to the tibial tubercle. Her patellar mobility was judged as satisfactory at this point, and further releases were not performed. Postoperatively, she began use of a CPM machine, and physical therapy was initiated, and by 8 months her extension deficit was 5 degrees and her flexion had increased to 130 degrees.

Discussion: In this case, a contributing factor to the loss of motion was the vertical graft placed somewhat anteriorly in the notch. The tibial tunnel was placed posteriorly on the tibial plateau to prevent graft impingement, but the graft had healed with such tension and in such a vertical position that motion could not be regained. The recalcitrant stiffness of the knee despite manipulation was a major factor in the decision to remove the graft. This case underscores the concerns with poor graft placement. There was no impingement, but a vertical graft was placed that probably contributed to the loss of motion.

Case 3

Chief Complaint and Patient History: A 42-year-old woman presented with functional knee instability after two failed allograft ACL reconstructions.

Physical Exam: On examination, she had close to full extension and flexion and only minimally increased valgus, varus, and posterolateral rotation compared with that of her contralateral normal knee.

Imaging: The MRI scan demonstrated significant lysis around the tibial and femoral tunnels measuring more than 20 mm in diameter for both tunnels (Fig. 15A).

Surgery/Treatment: It was elected to primarily graft the defects at a preliminary procedure prior to a revision reconstructive procedure. At surgery, her tibial tunnel was cleared of soft tissue by first reaming the tibial tunnel, then curetting the fibrous tissue from the tunnel. The femoral tunnel had been drilled using the transtibial method; therefore, it could be cleared of old graft and scar through the tibial tunnel using a curette and shaver. The defect in the lateral wall of the notch created by the femoral tunnel precluded any chance of proper graft placement or fixation (Fig. 15B). A generous quantity of autologous cancellous bone was harvested from the ipsilateral iliac crest and inserted through a metal cannula into the femoral tunnel (Fig. 15C). The tibial tunnel was filled by placing an instrument over the intraarticular opening of the tibial tunnel, then grafting the tibial tunnel from its extraarticular end. Six months was provided for the graft to heal within the tunnels prior to reconstruction with an autologous patellar tendon graft. At revision surgery, the tunnels were reamed through bone that was indistinguishable from normal bone, and the fixation with metal interference screws was very good. Three years after surgery, the knee continued to be functionally stable with an intact graft by examination.

Discussion: Particularly after two failed ACL reconstructions with allografts, there will be osteolysis in the tunnels. In order to have the best probability of graft incorporation, staged autograft bone grafting then patellar tendon autograft reconstruction were used to provide the best potential for healing.

Case 4

Chief Complaint and Patient History: A 25-year-old graduate student presented with knee instability 4 years after an ACL reconstruction was performed with a hamstring graft using the transtibial drilling method. The patient had minor trauma to the knee during an exercise class and felt a "pop," then experienced instability in the knee. The patient denied concerns with swelling or significant pain in the knee prior to the recent injury.

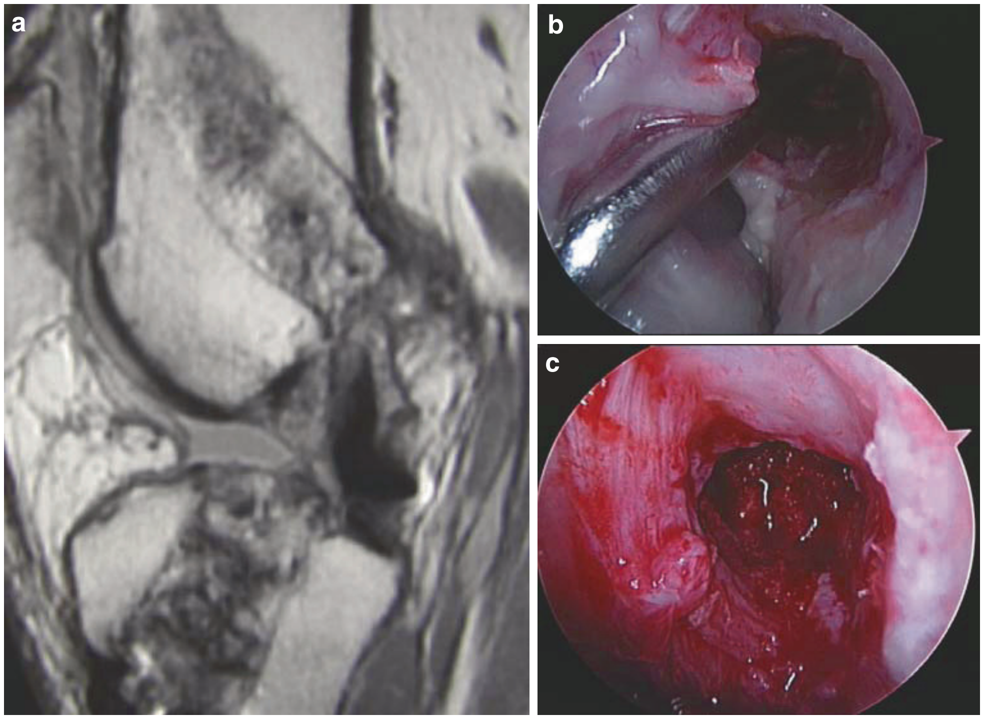


Fig. 15 Case 3. (**A**) Sagittal MRI image demonstrates image after two failed allograft ACL reconstructions using the transtibial drilling method, there have been two failures with extensive osteolysis in both tunnels measuring more than 20 mm in diameter. (**B**) At the bone grafting surgery, the femoral tunnel is seen after removing the remnants of the graft and fibrous tissue from the femoral tunnel. The size of the defect precludes the correct placement and fixation of a new graft; therefore, autologous bone grafting is necessary. (**C**) A generous quantity of bone graft has been packed into the femoral tunnel through a cannula placed through the tibial tunnel. The femoral tunnel appears to lie too high in the notch for ideal reconstructive technique.

Physical Exam: On examination, she had a positive Lachman test with approximately 6 mm of increased anterior translation compared with that of the opposite knee and a grade 2 pivot-shift test.

Imaging: The radiographs and MRI scan revealed a ruptured graft and a tibial tunnel through the medial tibial articular surface (Fig. 16A–C).

Surgery/Treatment: At the time of a revision ACL reconstruction, it was noted that indeed the tibial tunnel did traverse the medial tibial articular surface, and abrasion of the graft on the medial femoral condyle had occurred (Fig. 16D). However, the femoral tunnel was within the anatomic footprint of the ACL located halfway between the top of the notch and the lowest point on the lateral wall (Fig. 16E). The tibial interference screw was easily removed, but the staples were not symptomatic and therefore were not removed. The revision was performed using a tibialis tendon allograft placed through an anatomic tibial tunnel that did not contact the prior tunnel. This new tunnel was started close to the tibial tubercle. The previous femoral tunnel was reused and re-reamed from an auxiliary anteromedial portal (Fig. 16F).

Discussion: This case illustrates the difficulty coordinating tibial and femoral tunnels using the transtibial drilling method, particularly if the tibial tunnel is started from the posteromedial tibia. The femoral tunnel was ideally placed, but it was drilled through a tibial tunnel drilled medial to the anatomic footprint. Despite the medial tibial tunnel placement, the patient experienced minimal symptoms prior to graft rupture.

Case 5

Chief Complaint and Patient History: A 26-year-old man presented with a chief complaint of instability in his right knee for the past year. He had undergone a hamstring ACL reconstruction in the same knee 3 years prior to his presentation, but he sustained an injury 1 year after the reconstruction after which his knee was unstable. Despite the instability, he continued to play basketball and had repeated effusions.

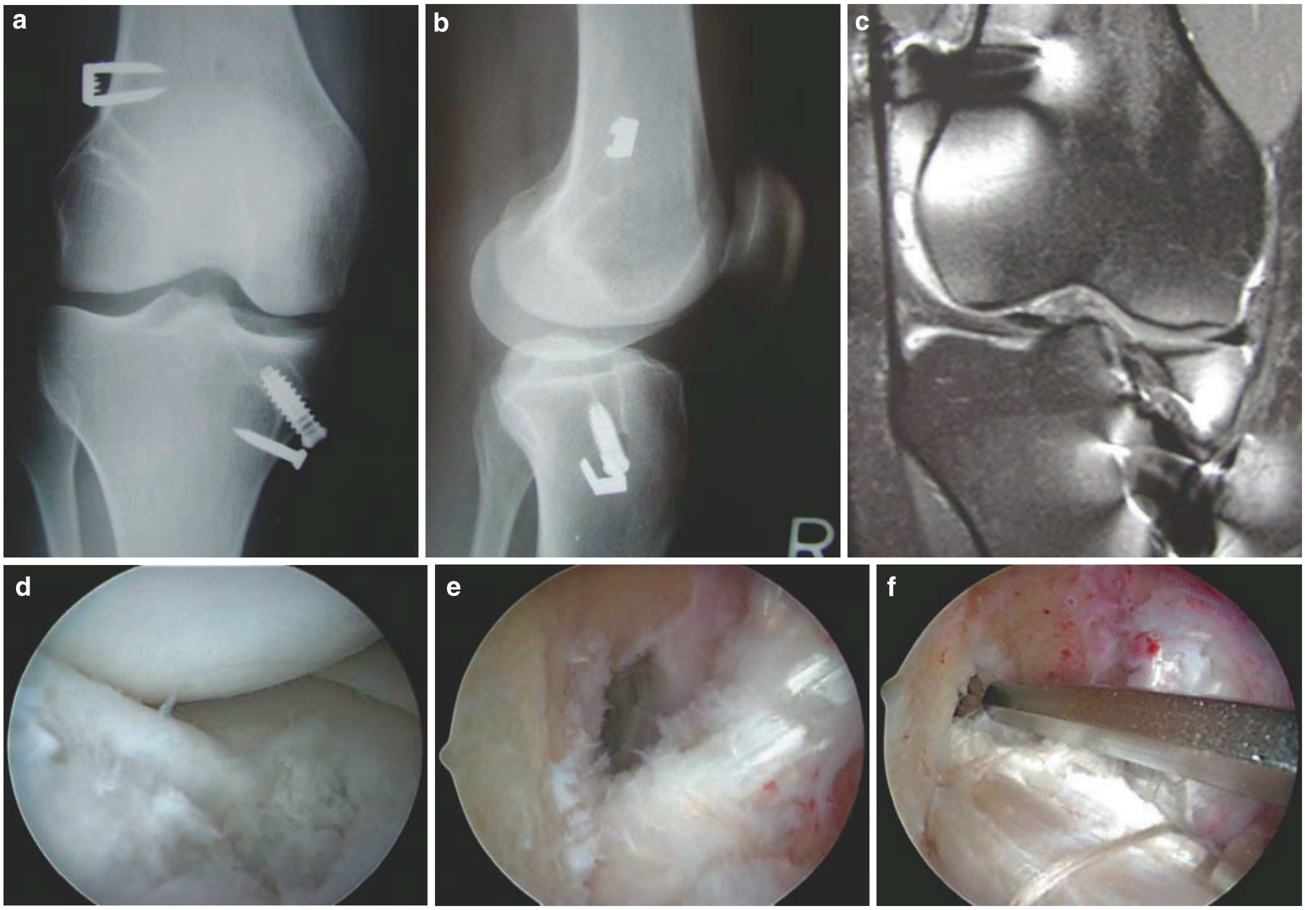


Fig. 16 Case 4. (**A**) The anterior radiograph demonstrates the tibial tunnel traversing the medial tibial articular surface and having an oblique angle to the tibial plateau of approximately 50 degrees. This inclination of the tibial tunnel will help for transtibial drilling of a correct femoral tunnel, but it places the tibial tunnel through the tibial articular surface. (**B**) The lateral radiograph demonstrates a tibial tunnel that starts approximately halfway between the tibial tubercle and posteromedial tibia and a femoral tunnel located in the posterior quadrant along Blumensaat's line. (**C**) The anterior MRI scan delineates the medial position of the tibial tunnel relative to the intercondylar notch. (**D**) The arthroscopic image of the tibial tunnel demonstrates its location slightly within the tibial articular plateau and some evidence of abrasion on the medial femoral condyle. (**E**) The arthroscopic image of the femoral tunnel demonstrates a tunnel located within the anatomic femoral footprint of the ACL and without extensive osteolysis. (**F**) A tibialis allograft has been placed in the previous femoral tunnel and secured with a bioabsorbable interference screw after re-reaming the tunnel from an auxiliary anteromedial portal.

Physical Exam: His examination was significant for a positive Lachman test without an end point and a grade 2 pivot-shift test.

Imaging: His radiographs and MRI scan were consistent with a prior reconstruction using a transverse femoral pin technique and a new medial meniscus tear (Fig. 17A, B).

Surgery/Treatment: At revision reconstruction, a new tibial tunnel could be placed anterior to the prior tibial tunnel that had been placed very close to the PCL (Fig. 17C, D). Drilling through an anteromedial portal, a new femoral tunnel could be placed in the anatomic insertion of the ACL with an apparent 3-mm bone bridge between the prior misplaced femoral tunnel and the new tunnel that was located essentially out of the femoral footprint (Fig. 17E, F).

Discussion: This case illustrates the challenge of placing correct tunnels using transtibial drilling and the femoral cross-pin technique. This method will tend to create graft impingement on the PCL because of the need to place the tibial tunnel posterior to avoid notch impingement anteriorly. Despite these maneuvers, a graft may still be more vertical than ideal and be susceptible to failure. A revision procedure in this situation can be accomplished by placing a new tibial tunnel anterior to the prior tunnel and a new femoral tunnel inferior to the prior tunnel.

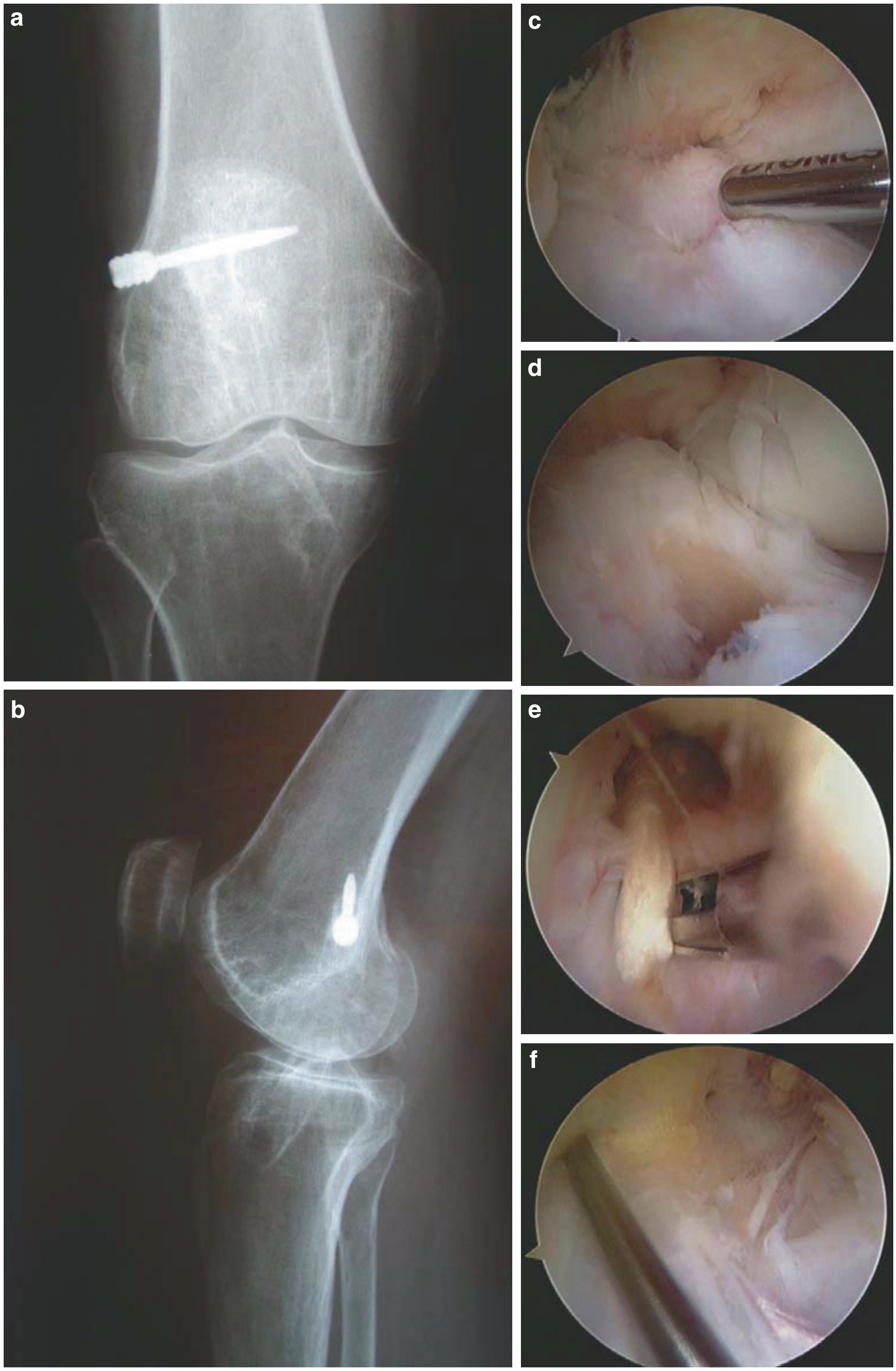


Fig. 17 Case 5. (**A**) The anterior radiograph demonstrates mild disuse osteopenia and a tibial tunnel at an angle of 60 degrees to the tibial plateau. Angulation of the tibial tunnel at less than 70 degrees to the tibial plateau may help to correctly place a femoral tunnel using the transtibial drilling method. (**B**) The lateral radiograph demonstrates osteopenia and a femoral tunnel placed correctly in the posterior quadrant along Blumensaat's line. (**C**) The arthroscopic image of the prior tibial tunnel placed very close to the PCL and probably posterior to the anatomic tibial insertion of the ACL. (**D**) The arthroscopic image of the revision tibial tunnel placed completely anterior to the prior tunnel and within the anatomic ACL tibial footprint. (**E**) The original femoral tunnel and remnant of the prior graft reveal a graft placed too superior in the intercondylar notch. (**F**) The revision graft has been placed in a new anatomic femoral tunnel separated from the original femoral tunnel by several millimeters.

References

1. Odensten M, Gillquist J. Functional anatomy of the anterior cruciate ligament and a rational for reconstruction. *J Bone Joint Surg* 1985;67A:257–262.
2. Girgis FG, Marshall JL, Monajem A. The cruciate ligaments of the knee joint. *Clin Orthop Relat Res* 1975;106:216–231.
3. Cha PS, Brucker PU, West RV, Zelle BA, Yagi M, Kurosaka M, Fu FH. Arthroscopic double-bundle anterior cruciate ligament reconstruction: an anatomic approach. *J Arthrosc* 2005;21:1275e1–1275e8.
4. Harner CD, Baek GHB, Vogrin TM, Carlin GJ, Kashiwaguchi S, Woo SL. Quantitative analysis of human cruciate ligament insertions. *J Arthrosc* 1999;15:741–749.
5. Steckel H, Vadala G, Davis D, Fu FH. 2D and 3D 3-tesla magnetic resonance imaging of the double bundle structure in anaterior cruciate ligament anatomy. *Knee Surg Sports Traum Arthrosc* 2006;14:1151–1158.
6. Howell SM, Gittins ME, Gottlieb JE, Traina SM, Zoellner TM. The relationship between the angle of the tibial tunnel in the coronal plane and loss of flexion and anterior laxity after anterior cruciate ligament reconstruction. *Am J Sports Med* 2001;29:567–574.
7. Mellado J, Calmet J, Olona M, Gine J, Sauri A. Magnetic resonance imaging of anterior cruciate ligament tears: reevaluation of quantitative parameters and imaging findings including a simpolified method for measurirng the anterior cruciate ligament angle. *Knee Surg Sports Traumatol Arthrosc* 2004;12:217–224.
8. Heming JF, Rand J, Steiner ME. Anatomic limitations of transtibial drilling in ACL reconstruction. *Am J Sports Med* 2007;35: 1708–1715.
9. O'Connor J, Shercliff T, FitzPatrick D, et al. Geometry of the knee. In Daniel D, Akeson W, O'Connor J (eds.), *Knee Ligaments: Structure, Function, Injury and Repair.* New York: Raven Press, 1990, p. 194.
10. Khalfayan EE, Sharkey PF, Alexander AH, Bruckner JD, Bynum EB. The relationship between tunnel placement and clinical results after anterior cruciate ligament reconstruction. *Am J Sports Med* 1996;24:335–341.
11. Yagi M, Wong EK, Kanamori A, Debski RE, Fu FH, Woo SL. Biomechanical analysis of an anatomic anterior cruciate ligament reconstruction. *Am J Sports Med* 2002;30:660–665.
12. Yasuda K, Kondo E, Ichiyama H, Kitamura N, Tanabe Y, Tohyama J, Minami A. Anatomic reconstruction of the anteromedial and posterolateral bundles of the anterior cruciate ligament using hamstring tendon grafts. *J Arthrosc* 2004;20:1015–1025.
13. Zavras TD, Race A, Bull AMJ, Amis AA. A comparative study of 'isometric' points for anterior cruciate ligament graft attachment. *Knee Surg Sports Traumatol Arthrosc* 2001;9:28–33.
14. Mochizuki T, Muneta T, Nagase T, Shirawawa S, Akita K, Sekiya I. Cadaveric knee observation study for describing anatomic femoral tunnel placement for two-bundle anterior cruciate ligament reconstruction. *J Arthrosc* 2006;22:356–361.
15. Hutchinson M, Bae T. Reproducibility of anatomic tibial landmarks for anterior cruciate ligament reconstructions. *Am J Sports Med* 2001;29:777–780.
16. Stäubli HU, Rauschning W. Tibial attachment area of the anterior cruciate ligament in the extended knee position. *Knee Surg Sports Traumatol Arthrosc* 1994;2:138–146.
17. Morgan CD, Kalman KR, Grawl DM. Definitive landmarks for reproducible tibial tunnel placement in anaterior cruciate ligmanent reconstruction. *J Arthrosc* 1995;11:275–288.
18. Colombet P, Robinson J, Christel P, Franceschi JP, Djian P, Bellier G, Sbihi A. Morphology of anterior cruciate ligament attachments for anatomic reconstruction: a cadaveric dissection and radiographic study. *J Arthrosc* 2006;22:984–992.
19. Clancy WG, Nelson DA, Reider B, Narechania RG. Anterior cruciate ligament reconstruction using one-third of the patellar ligament, augmented by extra-articular tendon transfers. *J Bone Joint Surg* 1982;64A:352–359.
20. Amis AA, Jakob RP. Anterior cruciate ligament graft positioning, tensioning and twisting. *Knee Surg Sports Traumatol Arthrosc* 1998;6(Suppl 1):S2–S12.
21. Takahashi M, Doi M, Abe M, Suzuki D, Nagano A. Anatomical study of the femoral and tibial insertions of the anteromedial and posterolateral bundles of human anterior cruciate ligament. *Am J Sports Med* 2006;34:787–792.
22. Howell SM, Barad SJ. Knee extension and its relationship to the slope of the intercondylar roof: implications for positioning the tibial tunnel in anterior cruciate ligament reconstructions. *Am J Sports Med* 1995;23:288–294.
23. Loh JC, Fukuda Y, Tsuda E, Steadman RJ, Fu FH, Woo SL. Knee stability and graft function following anterior cruciate ligament reconstruction: comparison between 11 o'clock and 10 o'clock femoral tunnel placement. *J Arthrosc* 2003;19:297–304.
24. Yamamoto Y, Hsu WH, Woo SL, Van Scyoc AH, Takakura Y, Debski RE. Knee stability and graft function after anterior cruciate ligament reconstruction: a comparison of a lateral and an anatomical femoral tunnel placement. *Am J Sports Med* 2004;32: 1825–1832.
25. Berg EE. Intrinsic healing of a patellar tendon donor site defect after anterior cruciate ligament reconstruction. *Clin Orthop* 1992;278:160–163.
26. Bernard M, Hertel P, Hornung H, Cierpinski T. Femoral insertion of the ACL radiographic quadrant method. *Am J Knee Surg* 1997;10:14–22.
27. Kohn D, Busche T, Caris J. Drill hole position in endoscopic anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc* 1998;6(Suppl 1):S13–S15.
28. Woo S, Hollis J, Adams D, Lyon R, Takai S. Tensile properties of the human femur-anterior cruciate ligament-tibia complex. *Am J Sports Med* 1991;19:217–225.
29. Woo SL, Kanamori A, Zeminski J, Yagi M, Papageorgiou C, Fu JG. The effectiveness of reconstruction of the anterior cruciate ligament with hamstrings and patellar tendon: a cadaveric study comparing anterior tibial and rotational loads. *J Bone Joint Surg* 2002;84A:907–914.
30. Zantop T, Herbort M, Raschke M, Fu FH, Petersen W. The role of the anteromedial and posterolateral bundles of the anterior cruciate ligament in anterior tibial translation and internal rotation. *Am Sports Med* 2007;35:223–227.
31. Steiner M, Brown C, Zarins B, Brownstein B, Koval BS, Stone P. Measurement of anterior-posterior knee displacement: comparison of instrumented devices and clinical examination. *J Bone Joint Surg* 1990;72A:1307–1315.
32. Hamada M, Shino K, Horibe S, Mitsuoka R, Miyama T, Shiozaki Y, Mae T. Single versus bi-socket anterior cruciate ligament reconstruction using autogenous multiple-stranded hamstring tendons with endobutton femoral fixation: a prospective study. *J Arthrosc* 2001;17:801–807.
33. Battaglia TC, Rand JD, Festa A, Steiner ME. Tunnel placement and knee kinematics after standard versus computer-assisted ACL reconstruction. Presented at: AOSSM Annual Meeting; June 29–July 2, 2006; Hershey, PA.
34. Scopp JM, Jasper LE, Belkoff SM, Moorman CT. The effect of oblique femoral tunnel placement on rotational constraint of the knee reconstructed using patellar tendon autografts. *J Arthrosc* 2004;20:294–299.
35. Georgoulis AD, Papadonikolakis A, Papageorgiou CD, Mitsou A, Stergiiou N. Three-dimensional tibiofemoral kinematics of the anterior cruciate ligament-deficient and reconstructed knee during walking. *Am J Sports Med* 2003;31:75–79.

36. Logan M, Dunstan E, Robinson J, Williams A, Gedroyc W, Freeman M. Tibiofemoral kinematics of the anterior cruciate ligament (ACL)-deficient weightbearing living knee employing vertical access open "interventional" multiple resonance imaging. *Am J Sports Med* 2004;32:720–726.
37. Mahfouz MR, Komistek RD, Dennis DA, Hoff W. In vivo assessment of the kinematics in normal and anterior cruicate ligament-deficient knees. *J Bone Joint Surg* 2004;86A(Suppl 2):56–61.
38. Ristanis S, Stergiou N, Patras K, Vasiliadis JS, Giakas G, Georgoulis AD. Excessive tibial rotation during high-demand activities is not restrored by anterior cruciate ligament reconstruction. *J Arthrosc* 2005;21:1323–1329.
39. Tashman S, Collon D, Anderson K, Kolowich P, Anderst W. Abnormal rotational knee motion during running after anterior cruciate ligment reconstruction. *Am J Sports Med* 2004;32: 975–983.
40. Muneta T, Koga H, Morito T, Yagishita K, Sekiya I. A retrospective study of the midterm outcome of two-bundle anterior cruciate ligament reconstruction using quadrupled semitendinosus tendon in comparison with one-bundle reconstruction. *J Arthrosc* 2006;22:252–258.
41. Steiner M, Koskinen S, Winalski C, et al. Dynamic lateral patellar tilt in the ACL deficient knee. *Am J Sports Med* 2001;29: 593–599.
42. Hsieh Y-F, Draganich L, Ho S, Reider B. The effects of removal and reconstruction of the anterior cruciate ligament on the contact characteristics of the patellofemoral joint. *Am J Sports Med* 2002;30:121–127.
43. DeMorat G, Weinhold P, Blackburn T, Chudik S, Garrett W. Aggressive quadriceps loading can induce noncontact anterior cruciate ligament injury. *Am J Sports Med* 2004;32:477–483.
44. Adler G, Hoekman R, Beach D. Drop leg Lachman test: a new test of anterior knee laxity. *Am J Sports Med* 1995;23:320–323.
45. Costa-Paz M, Muscolo D, Ayerza M, Makino A, Aponte-Tinao L. Magnetic resonance imaging follow-up study of bone bruises associated with anterior cruciate ligament ruptures. *Arthroscopy* 2001;17:445–449.
46. Nakamae A, Engebretsen L, Bahr R, Krosshaug T, Ochi M. Natural history of bone bruises after acute knee injury: clinical outcome and histopathological findings. *Knee Surg Sports Traumatol Sports* 2006;14:1252–1258.
47. Graf B, Cook D, De Smet A, Keene JS. "Bone bruises" on magnetic resonance imaging evaluation of anterior cruciate ligament injuries. *Am J Sports Med* 1993;21:220–223.
48. Johnson D, Urban W, Caborn D, Vanarthos WJ, Carlson CS. Articular cartilage changes seen with magnetic resonance imaging-detected bone bruises associated with acute anterior cruciate ligament rupture. *Am J Sports Med* 1998;26:409–414.
49. Daniel D, Stone M, Dobson B, Fithian DC, Rossman DJ, Kaufman KR. Fate of the ACL-injured patient: a prospective outcome study. *Am J Sports Med* 1994;22:632–644.
50. Fitzgibbons R, Shelbourne KD. "Aggressive" nontreatment of lateral meniscal tears seen during anterior cruciate ligament reconstruction. *Am J Sports Med* 1995;23:156–159.
51. Beynnon B, Johnson R, Abate J, Fleming BC, Nichols CE. Treatment of anterior cruciate ligament injuries, Part 2. *Am J Sports Med* 2005;33:1751–1767.
52. Kocher MS, Steadman JR, Briggs KK, Sterett WI, Hawkins RJ. Relationships between objective assessment of ligament stability and subjective assessment of symptoms and function after anterior cruciate ligament reconstruction. *Am J Sports Med* 2004;32: 629–634.
53. Fithian D, Paxton E, Stone M, Luetzow WF, Csintalan RP, Phelan D, Daniel DM. Prospective trial of a treatment algorithm for the management of the anterior cruciate ligament–injured knee. *Am J Sports Med* 2005;33:335–346.
54. Feagin JA Jr, Curl WW. Isolated tear of the anterior cruciate ligament: 5-year follow-up study. *Am J Sports Med* 1976;4:95–100.
55. Shelton W, Barrett G, Dukes A. Early season anterior cruciate ligament tears: a treatment dilemma. *Am J Sports Med* 1997;25: 656–658.
56. Wojtys E, Huston L. Neuromuscular performance in normal and anterior cruciate ligament-deficient lower extremities. *Am J Sports Med* 1994;22:89–104.
57. Shelbourne KD, Wilckens JH, Mollasbashy A, DeCarlo M. Arthrofibrosis in acute anterior cruciate ligament reconstruction. *Am J Sports Med* 1991;19:332–336.
58. Hey Groves WE. The crucial ligaments of the knee joint: their function, rupture, and the operative treatment of the same. *Br J Surg* 1920;7:505–515.
59. Pinczewski P, Lyman J, Salmon L, et al. A 10-year comparison of anterior cruciate ligament reconstructions with hamstring tendon and patellar tendon autograft: a controlled, prospective trial. *Am J Sports Med* 2007;35:564–574.
60. Armour T, Forwell L, Litchfield R, Kirkley A, Amendola N, Fowler PJ. Isokinetic evaluation of internal/external tibial rotation strength after the use of hamstring tendons for anterior cruciate ligament reconstruction. *Am J Sports Med* 2004;32:1639–1643.
61. Adachi N, Ochi M, Uchio Y, Iwasa J, Kuriwaka M, Ito Y. Reconstruction of the anterior cruciate ligament single versus double bundle multistranded hamstring tendons. *J Bone Joint Surg* 2004;86B:515–520.
62. Hamner D, Brown C, Steiner M, Hecker AT, Hayes WC. Hamstring tendon grafts for reconstruction of the anterior cruciate liagament: biomechanical evaluation of multiple strands and tensioning techniques. *J Bone Joint Surg* 1999;81A:549–557.
63. Tuman J, Diduch D, Rubino L, Baumfeld JA, Nguyen HS, Hart JM. Predictors for hamstring graft diameter in anterior cruciate ligament reconstruction. *Am J Sports Med* 2007; Nov; 35(11): 1945–9.
64. Adriani E, Mariani PP, Maresca G, Santori N. Healing of the patellar tendon after harvesting of its mid-third for anterior cruciate ligament reconstruction and evolution of the unclosed donor site defect. *Knee Surg Sports Traumatol Arthrosc* 1995;3:138–143.
65. Shelbourne KD, Urch SE. Primary anterior cruciate ligament reconstruction using the contralateral autogenous patellar tendon. *Am J Sports Med* 2000;28:651–658.
66. Adams D, Mazzocca A, Fulkerson J. Residual strength of the quadriceps versus patellar tendon after harvesting a central free tendon graft. *J Arthrosc* 2006;22:76–79.
67. Curran A, Adams D, Gill J, Steiner M, Scheller A. The biomechanical effects of low-dose irradiation on bone-patellar tendon-bone allografts. *Am J Sports Med* 2004 July-Aug; 32(5):1131–1135.
68. Daniel D, Lumkong G, Stone M, Pedowitz, R. Effects of tourniquet use in anterior cruciate ligament reconstruction. *J Arthrosc* 1995;11:307–311.
69. Muneta T, Yamamoto H, Ishibashi R, Asahina S, Murakami S, Furuya K. The effects of tibial tunnel placement and roofplasty on reconstructed anterior cruciate ligament knees. *J Arthrosc* 1995;11:57–62.
70. Tanzer M, Lenczner E. The relationship of intercondylar notch size and content to notchplasty requirement in anterior cruciate ligament surgery. *J Arthrosc* 1990;6:89–93.
71. Cooper DE, Small J, Urrea L. Factors affecting graft excursion patterns in endoscopic anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc* 1998;6(Suppl 1):S20–S24.
72. Hefzy MS, Grood ES, Noyes FR. Factors affecting the region of most isometric femoral attachments. part II the anterior cruciate ligament. *Am J Sports Med* 1989;17:208–216.
73. Howell SM, Deutsch ML. Comparison of endoscopic and two-incision techniques for reconstructing a torn anterior cruciate ligament using hamstring tendons. *J Arthrosc* 1999;15:594–606.

74. Jackson DW, Gasser SI. Tibial tunnel placement in ACL reconstruction. *J Arthrosc* 1994;10:124–131.
75. Panni AS, Milano G, Tartarone M, Demontis A, Fabbriciani C. Clinical radiographic results of ACL reconstruction: a 5- to 7-year follow-up study of outside-in versus inside-out reconstruction techniques. *Knee Surg Sports Traumatol Arthrosc* 2001;9: 77–85.
76. Paessler H, Rossis J, Mastrokalos D, Kotsovolos I. Anteromedial vs. transtibial technique for correct femoral tunnel placement during arthroscopic ACL reconstruction with hamstrings: an in vivo study. *J Bone Joint Surg Br.* 2004; 86(Suppl III):234.
77. Arnold MP, Kooloos J, van Kampen A. Single-incision technique misses the anatomical femoral anterior cruciate ligament insertion: a cadaver study. *Knee Surg Sports Traumatol Arthrosc* 2001;9:194–199.
78. Romano VM, Graf BK, Keene JS, Lange RH. Anterior cruciate ligament reconstruction the effect of tibial tunnel placment on range of motion. *Am J Sports Med* 1993;21: 415–418.
79. Hantes M, Zachos V, Basdekis G, Zibis A, Dailiana Z, Malizos K. Differences in graft orientation using the transtibial and anteromedial portal technique in ACL reconstruction: an MRI prospective study. *J Bone Joint Surg Br.* 2006; 88(Suppl I):93.
80. Chhabra A, Diduch DR, Blessey PB, Miller MD. Recreating an acceptable angle of the tibial tunnel in the coronal plane in anterior cruciate ligament reconstruction using external landmarks. *J Arthrosc* 2004;20:328–330.
81. Miller M, Hinkin D. The "N + 7 Rule" for tibial tunnel placement in endoscopic anterior cruciate ligament reconstruction. *J Arthrosc* 1996;12:124–126.
82. Burks R, Friederichs M, Fink B, Luker MG, West HS, Gries PE. Treatment of postoperative anterior cruciate ligament infections with graft removal and early reimplantation. *Am J Sports Med* 2003;31:414–418.
83. Sommer C, Friederich NF, Müller W. Improperly placed anterior cruciate ligament grafts: correlation between radiological parameters and clinical results. *Knee Surg Sports Traumatol Arthrosc* 2000;8:207–213.

Posterior Cruciate Ligament and Posterolateral Corner Reconstruction

Gregory C. Fanelli, Craig Edson, Kristin N. Reinheimer, and Daniel J. Tomaszewski

Introduction

Much has been learned and written about the anterior cruciate ligament (ACL). Interest in the posterior cruciate ligament (PCL) is increasing, and more articles are appearing in the literature. The natural history of PCL tears has not been well defined. The general consensus has been that isolated PCL tears do well when treated nonoperatively, and multiple ligament injuries about the knee should be surgically stabilized [1–3].

The benign natural history of the isolated PCL tear has been challenged recently [4–6]. Trickey, in 1980, calling the PCL the central pivot point of the knee, recommended early surgical treatment of all PCL tears [6].

Dandy and Pusey studied 20 patients treated conservatively for a mean interval of 7.2 years and found that 14 continued to have pain while walking, whereas 9 had episodes of giving way [4].

Keller et al. studied 40 patients with isolated PCL tears treated nonoperatively [5]. At an average follow-up interval of 6 years from the time of injury, 90% continued to experience pain, and 65% noted that their activity level was limited despite excellent muscle strength. Additionally, 65% of patients had radiographic evidence of degenerative changes that increased in severity as the time interval from injury increased [5]. This supports Trickey's earlier recommendation that PCL tears should be treated early surgically [6].

The purpose of this chapter is to review the anatomy and biomechanics of the PCL and posterolateral corner (PLC), describe the authors' surgical technique, present the authors' results of PCL-posterolateral reconstruction, and briefly discuss rehabilitation after PCL surgery.

PCL Anatomy and Biomechanics

The PCL has been considered by some to be the strongest knee ligament [7]. More recent studies indicate that the ACL and PCL are of approximately equal strength [8–10]. The PCL is the primary restraint to posterior tibial translation at the knee and plays an integral part in knee joint stability.

The PCL is named because of its posterior insertion on the tibia [11, 12]. PCL fibers are more vertically aligned than those of the oblique ACL fibers. The PCL originates on the posterolateral aspect of the medial femoral condyle where its attachment is in the form of a segment of a circle. The tibial attachment of the PCL is situated in a depression between the two tibial plateaus. This attachment in the PCL fossa extends for a few millimeters below the tibial articular surface [13].

Synovial tissue reflected from the posterior capsule covers the ligament on its medial, lateral, and anterior surfaces. Distally, the posterior portion of the PCL blends with the posterior capsule and periosteum. Strictly anatomically speaking, the PCL is extraarticular while lying within its own synovial sheath [12].

Girgis et al. found in cadaver and fresh knee dissections that the PCL averaged 38 mm in length and 13 mm in width, whereas the ACL averaged 38 mm in length and 11 mm in width [13].

The PCL has been shown to consist of two major inseparable bundles. The anterior bundle makes up the bulk of the ligament and is tight in flexion and lax in extension. The posterior bundle is much thinner, and these fibers are tight in extension and lax in flexion. In reality, there is a gradually changing pattern of fiber tension going from anterior to posterior as the knee is extended [11–14]. Recent studies suggest that the PCL consists of four fiber regions: anterior, central, posterior longitudinal, and posterior oblique. These fiber regions are based on fiber orientation and osseous attachment sites, with the anterior and central groups composing approximately 85% of the PCL bulk [15].

The fiber regions should not be confused with the meniscofemoral ligaments, which are distinct and separate

G.C. Fanelli (✉)
Department of Orthopaedic Surgery, Geisinger Clinic, Danville, Pennsylvania

B.P. McKeon et al. (eds.), *Knee Arthroscopy*, DOI 10.1007/978-0-387-89504-8_9,

structures. In approximately 70% of knees, an accessory meniscofemoral ligament is present [13, 16]. The anterior meniscofemoral ligament of Humphry lies anterior to the PCL, arising from the posterior horn of the lateral meniscus, and inserting on the femur with the PCL. It is approximately one third the diameter of the PCL. The posterior meniscofemoral ligament of Wrisberg arises as a continuation of the posterior horn of the lateral meniscus and is closely associated with the PCL. It has been measured to be up to one half the diameter of the PCL [16]. There are no attachments between the PCL and the medial meniscus.

The majority of the blood supply to the PCL stems from the middle geniculate artery, a branch of the popliteal artery [17]. The middle genicular artery also supplies the synovial sheath, which itself is a major contributor of nourishment to the PCL [18, 19]. Capsular vessels also supply the base of the PCL via branches from the popliteal and inferior genicular arteries [19].

Katonis et al. observed three types of nerve endings in the PCL in a histologic study [20]. They observed Ruffini corpuscles (type I, pressure receptors), Vater-Pacini corpuscles (type II, velocity receptors), and free nerve endings (type IV, pain receptors). They further postulated that damage to the PCL not only creates a mechanical disturbance but a central neurologic one as well. This is most likely secondary to lack of feedback mechanisms [20].

The PCL is the primary restraint to posterior tibial translation at all flexion angles >30 degrees [21, 22]. It provides 95% of the total restraining force for the straight posterior drawer [21]. Gollehon et al. found in a biomechanical study of cadaveric knees that isolated sectioning of the PCL did not affect varus or external rotation of the tibia at any position of knee flexion [22]. As expected, isolated sectioning of the PCL increased posterior tibial translation with a posteriorly directed force at all angles of flexion (maximum at 90 degrees). Sectioning of the lateral collateral ligament and the posterolateral complex, while leaving the PCL intact, resulted in small but significant increases in posterior translation at all angles of flexion (maximal at 30 degrees) [22].

As the knee progresses from flexion to extension, the tibia externally rotates relative to the femur. This has been traditionally called the “screw-home” mechanism of the knee. Possible hypotheses of this mechanism include bony anatomy and relative lengths of the cruciates [23]. Van Dommelen and Fowler suggested that the PCL plays an important role in the screw-home mechanism because of variable region taughtness at different flexion angles [12].

Covey and Sapega have conducted a biomechanical study of cadavers to determine the effects of normal knee joint motion and loading on end-to-end fiber length behavior of the four fiber regions. They found obvious differences in taughtness of the region when comparing passive joint motion with simulated quadriceps force. This data may help in determining optimum graft placement and post–PCL reconstruction rehabilitation programs [24].

Posterolateral Corner Anatomy and Biomechanics

The posterolateral corner consists of the lateral collateral ligament, the arcuate ligament, the popliteus tendon, the popliteofibular ligament, the short lateral ligament, the fabellofibular ligament, and the posterolateral capsule. The fibular attachment of the popliteus tendon, the popliteofibular ligament, is a common supporting structure of the posterolateral corner of the knee. This structure reinforces the posterolateral capsule. Its oblique anatomic orientation indicates that it may act as a static restraint to varus and external rotation movements [25].

The posterolateral corner structures serve to resist varus stress, posterior tibial translation near full extension, and external rotation of the tibia relative to the femur. Sectioning of the posterolateral corner structures results in small increases in posterior tibial translation but also in major increases in varus rotation and external tibial rotation [25].

Incidence of PCL and Posterolateral Corner Injuries

The incidence of PCL injuries has been reported to be in the range of 1% to 40% of acute knee injuries [2, 26–31]. This appears to be patient population dependent, and PCL injury occurs more frequently in trauma patients than in athletic injury patients [28, 29]. The authors have reported the incidence of PCL injuries in acute knee injuries from their own tertiary care regional trauma center [32]. The authors have shown a 38% incidence of PCL tears in acute knee injuries from the authors’ center. The two most frequent combined PCL injuries were ACL-PCL injuries (45.9%) and PCL–posterolateral corner injuries (41.2%). PCL–posterolateral corner injuries are the second most frequently encountered multiple ligament injuries of the knee involving the PCL [2, 28, 29, 32].

Clinical Presentation

Patients with PCL-posterolateral instability in the chronic situation will present with a functionally unstable knee. Symptoms will include hyperextension instability, posterolateral knee pain, and different degrees of a varus thrust when ambulating. Depending on the degree of instability, peroneal nerve symptoms also may be present [33]. Physical examination features of an isolated PCL instability include the following:

- Abnormal posterior laxity less than 5–10 mm (tibial step-off is still palpable).
- No abnormal varus.
- Abnormal external rotation of the tibia on the femur <10 degrees compared with the uninvolved side tested with the knee at 30 and 90 degrees of flexion.

Physical examination features of combined PCL-posterolateral instability include the following:

- Abnormal posterior laxity >5–10 mm. Tibial step-off is flat or negative.
- Abnormal varus rotation at 30 degrees of knee flexion (variable).
- Abnormal external rotation thigh foot angle of >10 degrees compared with the normal lower extremity tested at 30 and 90 degrees of knee flexion.
- Positive external rotation recurvatum test (variable).
- Positive posterolateral drawer test.
- Positive reversed pivot-shift test (variable).

Treatment

The surgical treatment principles for PCL-posterolateral instability are to correct the abnormal motion by addressing all the injured ligaments [34–48]. PCL reconstruction is performed as an arthroscopically assisted procedure using either autograft or allograft. The authors believe that the success for posterolateral reconstruction consists in creating a strong "posterolateral post" of autograft or allograft tissue to re-create the function of the popliteofibular ligament. In acute cases, the authors perform a direct repair of all injured posterolateral structures and augment this repair with a "posterolateral post" of soft tissue to reinforce the primary repair. Chronic posterolateral instability is addressed by capsular repair, if possible, and by reconstructing the function of all the injured posterolateral and lateral structures. Graft choices for the posterolateral reconstruction include free graft procedures, split biceps tendon transfer, biceps tendon transfer, and capsular advancement procedures. Certain cases of chronic PCL-posterolateral instability may require high tibial valgus osteotomy to correct bony varus deformity prior to ligament reconstruction. Failure to correct the bony varus deformity exposes the ligament reconstruction to high tensile loads, increasing the risk of ligament reconstruction failure [25].

Surgical Indications

The authors' indications for surgical treatment of acute PCL injuries include insertion site avulsions, tibial step-off decreased 5 mm or greater, and PCL tears combined with other structural injuries. The authors' indications for surgical treatment of chronic PCL injuries are when an isolated PCL tear becomes symptomatic or when progressive functional instability develops.

Surgical Timing

Surgical timing of acute PCL–posterolateral corner injuries depends upon the grade of the lateral side injury (A, B, or C) and/or presence or absence of a bony avulsion of the lateral ligament complex. The authors' preferred timing for PCL–posterolateral corner tears is to allow capsular sealing to occur over 2–3 weeks, followed by arthroscopic PCL reconstruction and posterolateral corner primary repair/reconstruction. The use of strong graft material in the posterolateral corner independently or to augment primary repair is essential for the success of this procedure. Cases of bony avulsion or fibular head avulsion are repaired acutely, and arthroscopic PCL reconstruction is performed 2–6 weeks later.

Surgical Technique

PCL surgical reconstructions may be unsuccessful because of failure to recognize and treat associated ligament instabilities (posterolateral instability and posteromedial instability), failure to treat varus osseous malalignment, and incorrect tunnel placement [49–51]. The keys to successful PCL reconstruction are to identify and treat all pathology, use strong graft material, accurately place tunnels in anatomic insertion sites, minimize graft bending, use a mechanical graft-tensioning device, use primary and back-up graft fixation, and employ the appropriate postoperative rehabilitation program. Adherence to these technical points results in successful single-bundle and double-bundle arthroscopic transtibial tunnel PCL reconstruction documented with stress radiography, arthrometer, knee ligament rating scales, and patient satisfaction measurements [35, 36, 40, 48, 52–56]. The purpose of this chapter is to describe the authors' surgical techniques for arthroscopic transtibial tunnel PCL reconstruction and posterolateral reconstruction.

Graft Selection

The authors' preferred graft source for PCL and posterolateral reconstruction is allograft tissue. The anterolateral bundle of the PCL is reconstructed with Achilles' tendon allograft, and the posteromedial bundle of the PCL is reconstructed with tibialis anterior allograft tissue. Posterolateral reconstruction is performed with semitendinosus allograft for fibular-based reconstructions combined with a posterolateral capsular shift procedure. Fibular head and tibia-based posterolateral reconstructions are performed with a split Achilles' tendon allograft, or semitendinosus allograft for the fibular arm and a tibialis anterior allograft for the tibial arm, also combined with a posterolateral capsular shift procedure.

Fig. 1 The surgeon is able to palpate the posterior aspect of the tibia through the extracapsular extraarticular posteromedial safety incision. This enables the surgeon to accurately position guide wires, create the tibial tunnel, and to protect the neurovascular structures. (Illustration courtesy of Biomet Sports Medicine, Inc., Warsaw, IN.)

PCL Reconstruction Surgical Technique

The patient is positioned on the operating table in the supine position, and the surgical and nonsurgical knees are examined under general or regional anesthesia. A tourniquet is applied to the operative extremity, and the surgical leg is prepped and draped in a sterile fashion. Allograft tissue is prepared prior to beginning the surgical procedure, and autograft tissue is harvested prior to beginning the arthroscopic portion of the procedure. Standard arthroscopic knee portals are used. The joint is thoroughly evaluated arthroscopically, and the PCL is evaluated using the three-zone arthroscopic technique [35]. The PCL tear is identified, and the residual stump of the PCL is debrided with hand tools and the synovial shaver.

An extracapsular posteromedial safety incision approximately 1.5–2.0 cm long is created [35, 36, 40, 48, 52–58]. The crural fascia is incised longitudinally, taking precautions to protect the neurovascular structures. The interval is developed between the medial head of the gastrocnemius muscle and the posterior capsule of the knee joint, which is anterior. The surgeon's gloved finger is positioned so that the neurovascular structures are posterior to the finger, and the posterior aspect of the joint capsule is anterior to the surgeon's finger. This technique enables the surgeon to monitor surgical instruments such as the over-the-top PCL instruments and the PCL-ACL drill guide as they are positioned in the posterior aspect of the knee. The surgeon's finger in the posteromedial safety incision also confirms accurate placement of the guide wire prior to tibial tunnel drilling in the medial-lateral and proximal-distal directions (Fig. 1). This is the same anatomic surgical interval that is used in the tibial inlay posterior approach.

The curved over-the-top PCL instruments are used to carefully lyse adhesions in the posterior aspect of the knee and to elevate the posterior knee joint capsule away from the tibial ridge on the posterior aspect of the tibia. This capsular elevation enhances correct drill guide and tibial tunnel placement (Fig. 2).

The arm of the drill guide (Biomet Sports Medicine, Warsaw, IN) is inserted into the knee through the inferomedial patellar portal and positioned in the PCL fossa on the posterior tibia (Fig. 3). The bullet portion of the drill guide contacts the anteromedial aspect of the proximal tibia approximately 1 cm below the tibial tubercle, at a point midway between the tibial crest anteriorly and the posteromedial border of the tibia. This drill guide positioning creates a tibial tunnel that is relatively vertically oriented and has its posterior exit point in the inferior and lateral aspect of the PCL tibial anatomic insertion site. This positioning creates an angle of graft orientation such that the graft will turn two very smooth 45-degree angles on the posterior aspect of the tibia, eliminating the "killer turn" of 90-degree graft angle bending (Fig. 4).

The tip of the guide in the posterior aspect of the tibia is confirmed with the surgeon's finger through the extracapsular posteromedial safety incision. Intraoperative anteroposterior and lateral x-rays also may be used, as well as arthroscopic

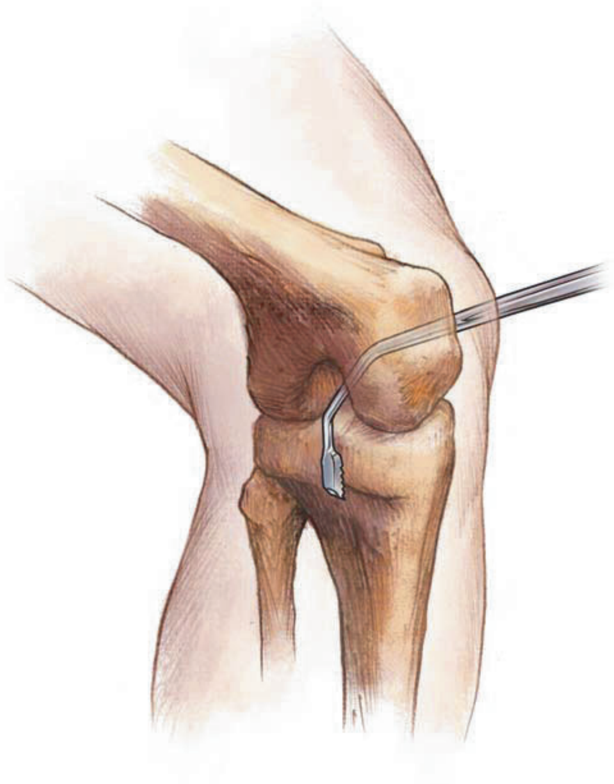


Fig. 2 Posterior capsular elevation using the Arthrotek Biomet Sports Medicine PCL instruments. (Illustration courtesy of Biomet Sports Medicine, Inc., Warsaw, IN.)

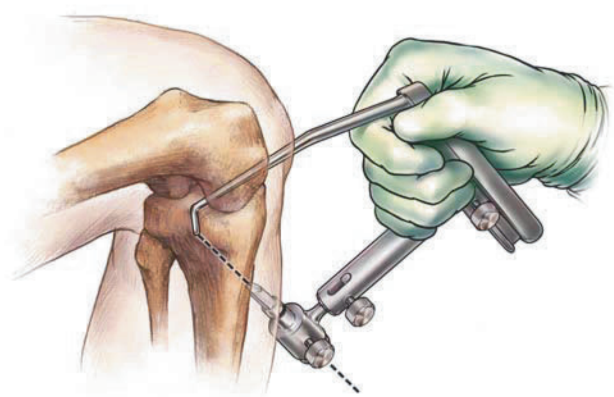


Fig. 3 Arthrotek Biomet Sports Medicine PCL-ACL Drill Guide positioned to place guide wire in preparation for creation of the transtibial PCL tibial tunnel. (Illustration courtesy of Biomet Sports Medicine, Inc., Warsaw, IN.)

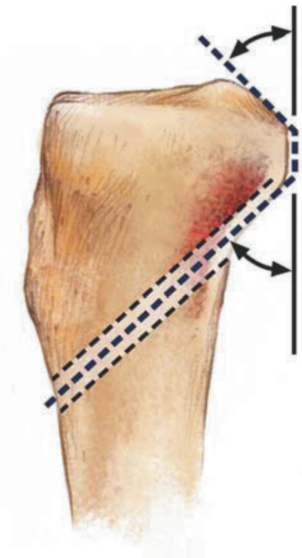


Fig. 4 Illustration demonstrating the desired turning angles the PCL graft will make after the creation of the tibial tunnel. (Illustration courtesy of Biomet Sports Medicine, Inc., Warsaw, IN.)

visualization to confirm drill guide and guide pin placement. A blunt spade-tipped guide wire is drilled from anterior to posterior and can be visualized with the arthroscope, in addition to being palpated with the finger in the posteromedial safety incision. The authors consider the finger in the posteromedial safety incision the most important step for accuracy and safety.

The appropriately sized standard cannulated reamer is used to create the tibial tunnel. The closed curved PCL curette may be positioned to cup the tip of the guide wire. The arthroscope, when positioned in the posteromedial portal, may visualize the guide wire being captured by the curette and may help in protecting the neurovascular structures in addition to the surgeon's finger in the posteromedial safety incision. The surgeon's finger in the posteromedial safety incision is monitoring the position of the guide wire. The standard cannulated drill is advanced to the posterior cortex of the tibia. The drill chuck is then disengaged from the drill, and completion of the tibial tunnel reaming is performed by hand. This gives an additional margin of safety for completion of the tibial tunnel. The tunnel edges are chamfered and rasped with the PCL-ACL system rasp.

The PCL-ACL drill guide is positioned to create the femoral tunnel. The arm of the guide is introduced through the inferomedial patellar portal and is positioned such that the guide wire will exit through the center of the stump of the anterolateral bundle of the PCL (Fig. 5). The spade-tipped guide wire is drilled through the guide, and just as it begins to emerge through the center of the stump of the PCL anterolateral bundle, the drill guide is disengaged. The accuracy of the placement of the wire is confirmed arthroscopically with probing and visualization. Care must be taken to ensure the patellofemoral joint has not been violated by arthroscopically examining the patellofemoral joint prior to drilling and that

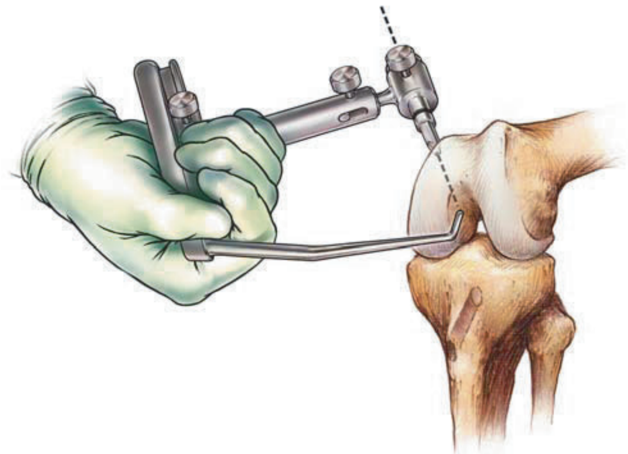


Fig. 5 The Biomet Sports Medicine PCL-ACL Drill Guide is positioned to drill the guide wire from outside-in. The guide wire begins at a point halfway between the medial femoral epicondyle and the medial femoral condyle trochlea articular margin, approximately 2 to 3 cm proximal to the medial femoral condyle distal articular margin, and exits through the center of the stump of the anterolateral bundle of the PCL stump. (Illustration courtesy of Biomet Sports Medicine, Inc., Warsaw, IN.)

there is adequate distance between the femoral tunnel and the medial femoral condyle articular surface. The appropriately sized standard cannulated reamer is used to create the femoral tunnel. A curette is used to cap the tip of the guide wire so that there is no inadvertent advancement of the guide wire, which may damage the ACL or articular surface. As the reamer is about to penetrate interiorly, the reamer is disengaged from the drill, and the final reaming is completed by hand. This adds an additional margin of safety. The reaming debris is evacuated with a synovial shaver to minimize fat pad inflammatory response with subsequent risk of arthrofibrosis. The tunnel edges are chamfered and rasped.

When the double-bundle PCL reconstruction is performed, the PCL-ACL drill guide is positioned to create the second femoral tunnel. The arm of the guide is introduced through the inferomedial patellar portal and is positioned such that the guide wire will exit through the center of the stump of the posteromedial bundle of the PCL. The blunt spade-tipped guide wire is drilled through the guide, and, just as it begins to emerge through the center of the stump of the PCL posteromedial bundle, the drill guide is disengaged. The accuracy of the placement of the wire is confirmed arthroscopically with probing and visualization. Care must be taken to ensure that there will be an adequate bone bridge (approximately 5 mm) between the two femoral tunnels prior to drilling. This is accomplished using the calibrated probe and direct arthroscopic visualization. The appropriately sized standard cannulated reamer is used to create the posteromedial bundle femoral tunnel. A curette is used to cap the tip of the guide wire so there is no inadvertent advancement of the guide wire, which may damage the ACL or articular surface. The tunnel is reamed in identical fashion to the anterolateral (AL) bundle.

The PCL single-bundle or double-bundle femoral tunnels can be made from inside-out using the double-bundle aimers (Biomet Sports Medicine). Inserting the appropriately sized double-bundle aimer through a low anterolateral patellar arthroscopic portal creates the PCL anterolateral bundle femoral tunnel. The double-bundle aimer is positioned directly on the footprint of the femoral anterolateral bundle PCL insertion site. The appropriately sized guide wire is drilled through the aimer, through the bone, and out a small skin incision. Care is taken to ensure there is no compromise of the articular surface. The double-bundle aimer is removed, and an acorn reamer is used to endoscopically drill from inside-out the anterolateral PCL femoral tunnel. When the surgeon chooses to perform a double-bundle double femoral tunnel PCL reconstruction, the same process is repeated for the posteromedial bundle of the PCL. Care must be taken to ensure that there will be an adequate bone bridge (approximately 5 mm) between the two femoral tunnels prior to drilling. This is accomplished using the calibrated probe and direct arthroscopic visualization (Fig. 6A, B).

Retrieving the graft can be facilitated by a suture passer. One such device is the Magellan suture-passing device (Biomet Sports Medicine). The suture-passing device is introduced through the tibial tunnel and into the knee joint and is retrieved through the femoral tunnel with an arthroscopic grasping tool. The traction sutures of the graft material are attached to the loop of the suture-passing device, and the PCL graft material is pulled into position.

Fixation of the PCL substitute is accomplished with primary and backup fixation on both the femoral and tibial sides. The authors' most commonly used graft source for PCL reconstruction is the Achilles' tendon allograft alone for single-bundle reconstructions and Achilles' tendon and tibialis anterior allografts for double-bundle reconstructions, although other allografts and autografts may be used as preferred by an individual surgeon. Femoral fixation is accomplished with cortical suspensory back-up fixation using polyethylene ligament fixation buttons and aperture opening fixation using bioabsorbable interference screws. The graft-tensioning boot (Biomet Sports Medicine) is applied to the traction sutures of the graft material on its distal end and tensioned to restore the anatomic tibial step-off. The knee is cycled through several sets of 25 full flexion-extension cycles for graft pretensioning and settling (Fig. 7). The PCL reconstruction graft is tensioned in physiologic knee flexion ranges. Graft fixation is achieved with primary aperture opening fixation using the bioabsorbable interference screw and back-up fixation with a ligament fixation button, or screw and post or screw and spiked ligament washer assembly (Fig. 8).

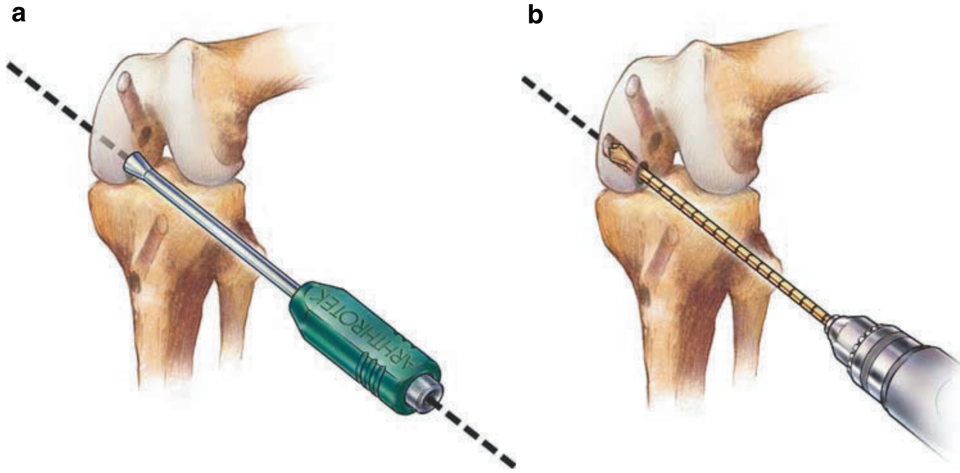


Fig. 6 (**A**) Biomet Sports Medicine Double Bundle Aimer positioned to drill a guide wire for creation of the PCL posteromedial bundle femoral tunnel through the low anterolateral patellar portal. (Illustration courtesy of Biomet Sports Medicine, Inc. Warsaw, IN.) (**B**) Endoscopic acorn reamer is used to create the PCL posteromedial bundle through the low anterolateral patellar portal. (Illustration courtesy of Biomet Sports Medicine, Inc., Warsaw, IN.)

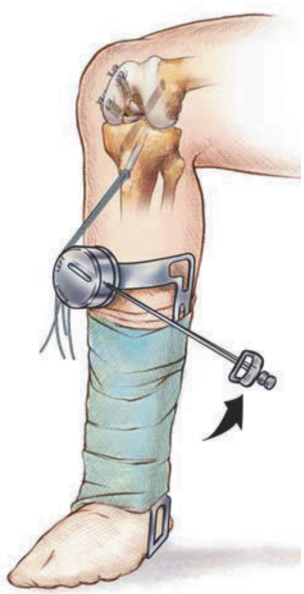


Fig. 7 Biomet Sports Medicine Knee Ligament Graft-Tensioning Boot. This mechanical tensioning device uses a ratcheted torque wrench device to assist the surgeon during graft tensioning. (Illustration courtesy of Biomet Sports Medicine, Inc., Warsaw, IN.)

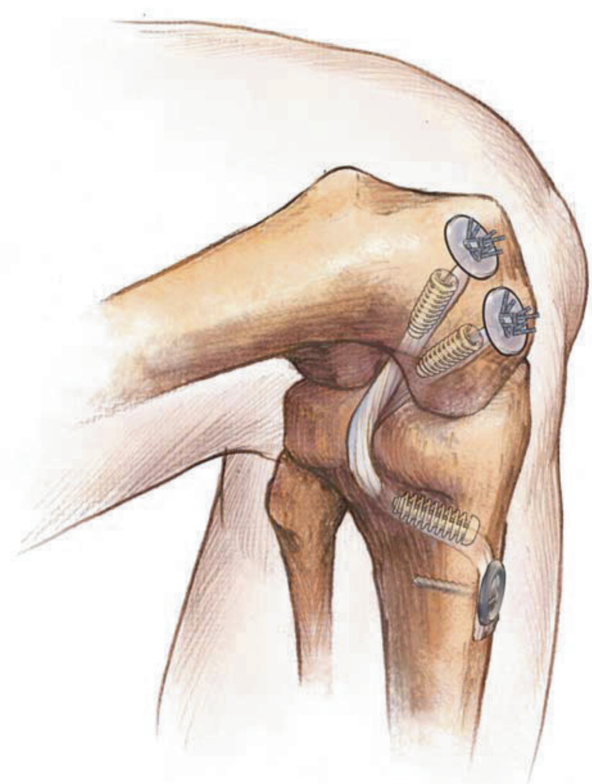


Fig. 8 Final graft fixation using primary and back-up fixation. (Illustration courtesy of Biomet Sports Medicine, Inc., Warsaw, IN.)

Posterolateral Reconstruction Surgical Technique

The free graft figure-of-eight technique for posterolateral reconstruction uses semitendinosus autograft or allograft, Achilles' tendon allograft, or other soft tissue allograft material. This technique combined with capsular repair and/or posterolateral capsular shift procedures mimics the function of the popliteofibular ligament and lateral collateral ligament, tightens the posterolateral capsule, and provides a post of strong autogenous tissue to reinforce the posterolateral corner [59–62]. A curvilinear incision is made in the

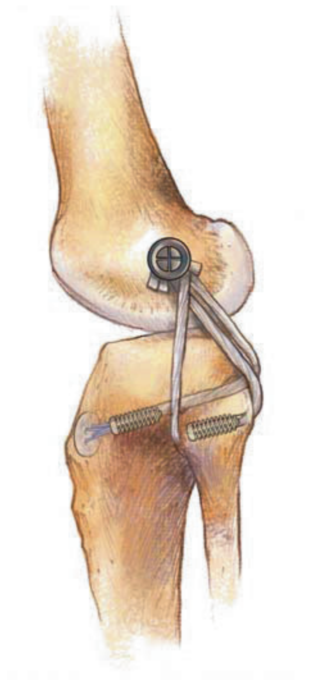


Fig. 9 Posterolateral reconstruction using two-tailed graft. Transfibular head figure-of-eight semitendinosus allograft mimics the force vectors of the fibular collateral ligament and the popliteofibular ligament. Transtibial tibialis anterior allograft mimics the force vectors of the popliteus tendon. Posterolateral capsular shift is also performed. (Illustration courtesy of Biomet Sports Medicine, Inc., Warsaw, IN.)

lateral aspect of the knee extending from the lateral femoral epicondyle to the interval between Gerdy's tubercle and the fibular head. The peroneal nerve is dissected free and protected throughout the procedure. The fibular head is exposed, and a 7-mm tunnel is created in an anterior inferior to posterior superior direction at the area of maximal fibular diameter. The tunnel is created by passing a guide pin followed by a cannulated drill usually 7 mm in diameter. The peroneal nerve is protected during tunnel creation and throughout the procedure. The free tendon graft is then passed through the fibular head drill hole. An incision is made in the iliotibial band in line with the fibers directly overlying the lateral femoral epicondyle. A longitudinal incision is made in the lateral capsule just posterior to the fibular collateral ligament. The graft material is passed medial to the iliotibial band and secured to the lateral femoral epicondylar region with a screw and spiked ligament washer with the allograft insertion sites corresponding with the anatomic insertion sites of the fibular collateral ligament and the popliteus tendon. The posterolateral capsule that had been previously incised is then shifted and sewn into the strut of figure-of-eight graft tissue material to eliminate posterolateral capsular redundancy. The anterior and posterior limbs of the figure-of-eight graft material are sewn to each other to reinforce and tighten the construct. The final graft-tensioning position is approximately 30–40 degrees of knee flexion with the tibia in maximal internal rotation. The iliotibial band incision is closed.

When there is a disrupted proximal tibiofibular joint or hyperextension external rotation recurvatum deformity, a two-tailed (fibular head, proximal tibia) posterolateral reconstruction is used [59–62]. The semitendinosus allograft is passed through the fibular head and secured to the lateral femoral epicondylar area as described earlier. A tibial arm of the reconstruction is passed through a 7-mm drill hole made 2 cm below the joint line through the proximal lateral tibia. This tibial arm of the posterolateral reconstruction follows the course of the popliteus tendon, providing additional support to the posterolateral corner. The procedures described are intended to eliminate posterolateral and varus rotational instability (Fig. 9).

Incorporation of autologous platelet-rich fibrin matrix into the grafts used in the PCL and posterolateral reconstructive procedures has been found to enhance the biologic healing response and graft incorporation [63]. The authors' clinical results demonstrate earlier soft tissue graft incorporation documented radiographically, enhanced wound healing, and decreased wound inflammation and pain (Fig. 10A, B).

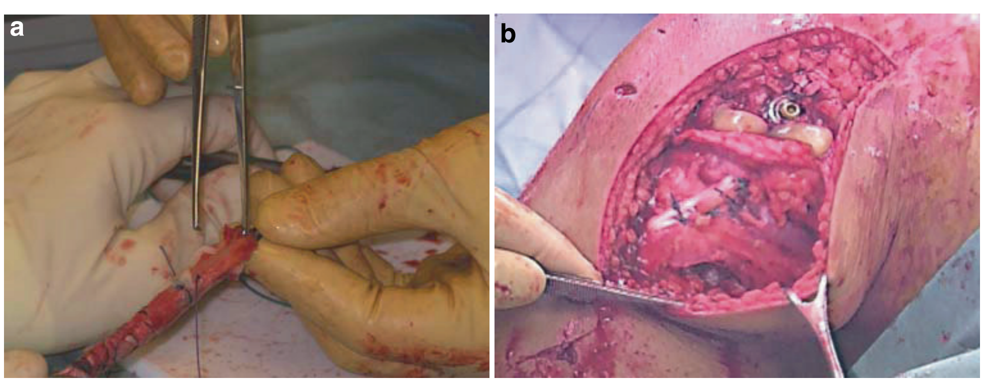


Fig. 10 (**A**) Cascade (Musculoskeletal Transplant Foundation, Edison, NJ) platelet-rich fibrin matrix incorporated into the PCL graft preparation and (**B**) the posterolateral reconstruction. The authors' clinical results demonstrate earlier soft tissue graft incorporation documented radiographically, enhanced wound healing, and decreased wound inflammation and pain

Rehabilitation

The knee is immobilized in a long leg brace in full extension, with non-weight-bearing using crutches. Progressive range of motion occurs during weeks 3 through 6. The brace is unlocked between 4 and 6 weeks, and progressive weight-bearing at 25% body weight per week is allowed during postoperative weeks 7–10. The crutches are discontinued at the end of postoperative week 10. Progressive strength training and range of motion exercises are performed. Return to sports and heavy labor occurs after the sixth to ninth postoperative month, when sufficient strength, range of motion, and proprioceptive skills have returned.

Complications

ACL surgery is a frequently performed orthopaedic surgical procedure in the United States. PCL injuries occur less frequently in this country, and the experience of the orthopaedic surgeon is correspondingly less for PCL examination, diagnosis, and surgical reconstructive procedures. Studies indicate that acute PCL injuries are related to geographic region, frequency of blunt trauma, and the population density of orthopaedic surgeons. It is estimated that relatively few orthopaedic surgeons perform PCL surgery compared with ACL surgery, and complications may result from lack of experience in diagnosis, surgical techniques, and postoperative care. PCL reconstruction is technically demanding surgery. Complications encountered with this surgical procedure include failure to recognize associated ligament injuries, neurovascular complications, persistent posterior sag, osteonecrosis, knee motion loss, anterior knee pain, and fractures. A comprehensive preoperative evaluation that includes an accurate diagnosis, a well-planned and carefully executed surgical procedure, and a supervised postoperative rehabilitation program will help to reduce the incidence of these complications.

Results: Single-Bundle PCL Reconstruction

In 2004, Fanelli and Edson published the 2- to 10-year (24- to 120-month) results of 41 chronic arthroscopically assisted combined PCL-posterolateral reconstructions evaluated preoperatively and postoperatively using Lysholm, Tegner, and Hospital for Special Surgery (HSS) knee ligament rating scales, KT1000 (MedMetric Corporation, San Diego, CA) arthrometer testing, stress radiography, and physical examination [48]. PCL reconstructions were performed using the arthroscopically assisted single femoral tunnel–single-bundle transtibial tunnel PCL reconstruction technique using fresh-frozen Achilles' tendon allografts in all 41 cases. In all 41 cases, posterolateral instability reconstruction was performed with combined biceps femoris tendon tenodesis and posterolateral capsular shift procedures. Postoperative physical exam revealed normal posterior drawer/tibial step-off for the overall study group in 29 of 41 (70%) knees. Normal posterior drawer and tibial step-offs were achieved in 91.7% of the knees tensioned with the mechanical graft tensioner (Biomet Sports Medicine). Posterolateral stabilit was restored to normal in 11 of 41 (27%) knees and was tighter than the normal knee in 29 of 41 (71%) knees evaluated with the external rotation thigh foot angle test. Thirty-degree varus stress testing was normal in 40 of 41 (97%) knees and grade 1 laxity in 1 of 41 (3%) knees. Postoperative KT1000 arthrometer testing mean side-to-side difference measurements were 1.80 mm (PCL screen), 2.11 mm (corrected posterior), and 0.63 mm (corrected anterior) measurements. This is a statistically significant improvement from preoperative status for the PCL screen and the corrected posterior measurements ($p = 0.001$). The postoperative stress radiographic mean side-to-side difference measurement measured at 90 degrees of knee flexion and 32 pounds of posterior directed force applied to the proximal tibia using the Telos device (Telos GmbH, Laubscher, Holstein, Switzerland) was 2.26 mm. This is a statistically significant improvement from preoperative measurements ($p = 0.001$). Postoperative Lysholm, Tegner, and HSS knee ligament rating scale mean values were 91.7, 4.92, and 88.7, respectively, demonstrating a statistically significant improvement from preoperative status ($p = 0.001$). The authors concluded that chronic combined PCL-posterolateral instabilities can be successfully treated with arthroscopic PCL reconstruction using fresh-frozen Achilles' tendon allograft combined with posterolateral corner reconstruction using biceps tendon tenodesis combined with posterolateral capsular shift procedure. Statistically significant improvement is noted ($p = 0.001$) from the preoperative condition at 2- to 10-year follow-up using objective parameters of knee ligament rating scales, arthrometer testing, stress radiography, and physical examination.

In 2005, Fanelli et al. published the 2-year follow-up results of 15 allograft ACL-PCL reconstructions using a mechanical graft-tensioning device (Biomet Sports Medicine) [55]. This study group consisted of 11 chronic and 4 acute injuries. These injury patterns included six ACL PCL PLC injuries, four ACL PCL medial collateral ligament (MCL) injuries, and five ACL PCL PLC MCL injuries. All knees had grade III preoperative ACL-PCL laxity and were assessed preoperatively and postoperatively using Lysholm, Tegner, and HSS knee ligament rating scales, KT1000 arthrometer testing, stress radiography, and physical examination.

Arthroscopically assisted combined ACL-PCL reconstructions were performed using the single-incision endoscopic ACL technique and the single femoral tunnel–single-bundle transtibial tunnel PCL technique. PCLs were reconstructed with allograft Achilles' tendon in all 15 knees. ACLs were reconstructed with Achilles' tendon allograft in all 15 knees. MCL injuries were treated surgically using primary repair, posteromedial capsular shift, and allograft augmentation as indicated. Posterolateral instability was treated with allograft semitendinosus free graft, with or without primary repair, and posterolateral capsular shift procedures as indicated. The graft-tensioning boot (Biomet Sports Medicine) was used in this series of patients.

Postreconstruction physical examination results revealed normal posterior drawer/tibial step-off in 13 of 15 (86.6%) knees. They also revealed normal Lachman test in 13 of 15 (86.6%) knees and normal pivot-shift tests in 14 of 15 (93.3%) knees. Posterolateral stability was restored to normal in all knees with posterolateral instability when evaluated with the external rotation thigh foot angle test (nine knees equal to the normal knee, and two knees tighter than the normal knee). Thirty-degree varus stress testing was restored to normal in all 11 knees with posterolateral instability. Thirty- and zero-degree valgus stress testing was restored to normal in all nine knees with medial-side laxity. Postoperative KT1000 arthrometer testing mean side-to-side difference measurements were 1.6 mm (range, –3 to 7 mm) for the PCL screen, 1.6 mm (range, –4.5 to 9 mm) for the corrected posterior, and 0.5 mm (range, –2.5 to 6 mm) for the corrected anterior measurements, a significant improvement from preoperative status. Postoperative stress radiographic side-to-side difference measurements measured at 90 degrees of knee flexion and 32 pounds of posteriorly directed proximal force using the Telos stress radiography device were 0–3 mm in 10 of 15 knees (66.7%), 4 mm in 4 of 15 knees (26.7%), and 7 mm in 1 of 15 knees (6.67%). Postoperative Lysholm, Tegner, and HSS knee ligament rating scale mean values were 86.7 (range, 69–95), 4.5 (range, 2–7), and 85.3 (range, 65–93) respectively, demonstrating a significant improvement from preoperative status.

The authors concluded that the study group demonstrates the efficacy and success of using allograft tissue and a mechanical graft-tensioning device/graft-tensioning boot (Biomet Sports Medicine) in single-bundle single femoral tunnel arthroscopic PCL reconstruction.

Double-Bundle Compared with Single-Bundle PCL Reconstruction Results and Single-Bundle Versus Double-Bundle Arthroscopic Transtibial PCL Reconstruction Results

Ninety consecutive arthroscopic transtibial PCL reconstructions were performed by a single surgeon [64]. Forty-five single-bundle and 45 double-bundle reconstructions were performed using fresh-frozen Achilles' tendon allograft for the anterolateral bundle and tibialis anterior allograft for the posteromedial bundle. Postoperative comparative results were assessed using Telos stress radiography, KT1000, Lysholm, Tegner, and HSS knee ligament rating scales. Postoperative period ranged from 15 to 72 months.

Three groups of data were analyzed: Single- and double-bundle all; single-bundle PCL–collateral and double-bundle PCL–collateral; and single-bundle PCL–ACL–collateral and double-bundle PCL–ACL–collateral. Mean postoperative side-to-side difference values for Telos, KT1000 PCL screen, KT1000 corrected posterior, and KT1000 corrected anterior measurements for the overall single-bundle group in millimeters were 2.56, 1.91, 2.11, and 0.23, respectively.

Mean postoperative side-to-side difference values for Telos, KT1000 PCL screen, KT1000 corrected posterior, and KT1000 corrected anterior measurements for the overall double-bundle group in millimeters were 2.36, 2.46, 2.94, and 0.15, respectively. Mean postoperative values for Tegner, Lysholm, and HSS knee ligament rating scales for the single-bundle group were 5.0, 90.3, and 86.2, respectively. Mean postoperative values for Tegner, Lysholm, and HSS knee ligament rating scales for the double-bundle group were 4.6, 87.6, and 83.3, respectively.

Mean postoperative side-to-side difference values for Telos, KT1000 PCL screen, KT1000 corrected posterior, and KT1000 corrected anterior measurements for the single-bundle PCL–collateral group in millimeters were 2.59, 1.63, 2.03, and 0.25, respectively. Mean postoperative side-to-side difference values for Telos, KT1000 PCL screen, KT1000 corrected posterior, and KT1000 corrected anterior measurements for the double-bundle PCL–collateral group in millimeters were 1.85, 2.03, 2.83, and –0.17, respectively. Mean postoperative values for Tegner, Lysholm, and HSS knee ligament rating scales for the single-bundle PCL–collateral group were 5.4, 90.9, and 87.7, respectively. Mean postoperative values for Tegner, Lysholm, and HSS knee ligament rating scales for the double-bundle PCL–collateral group were 4.9, 89.0, and 86.5, respectively.

Mean postoperative side-to-side difference values for Telos, KT1000 PCL screen, KT1000 corrected posterior, and KT1000 corrected anterior measurements for the single-bundle PCL–ACL–collateral group in millimeters were 2.53, 2.19, 2.19, and 0.22, respectively. Mean postoperative side-to-side difference values for Telos, KT1000 PCL screen, KT1000 corrected posterior, and KT1000 corrected anterior measurements for the double-bundle PCL–ACL–collateral group in millimeters were 3.16, 2.86, 3.09, and 0.41, respectively. Mean postoperative values for Tegner, Lysholm, and HSS knee ligament rating scales for the single-bundle PCL–ACL–collateral group were 4.7, 89.6, and 84.6, respectively. Mean postoperative values for Tegner, Lysholm, and HSS knee ligament rating scales for the double-bundle PCL–ACL–collateral group were 4.3, 86.0, and 79.4, respectively.

Both single-bundle and double-bundle arthroscopic transtibial tunnel PCL reconstructions provide excellent results in these complex knee instability patterns. The authors' results did not indicate that one surgical procedure was clearly superior to the other.

Clinical Pearls/Summary

Both the single-bundle and double-bundle arthroscopically assisted transtibial PCL reconstruction techniques are successful surgical procedures. The authors have documented results demonstrating statistically significant improvements from preoperative to postoperative status evaluated by physical examination, knee ligament rating scales, arthrometer measurements, and stress radiography. Factors contributing to the success of this surgical technique include identification and treatment of all pathology (especially posterolateral and posteromedial instability), accurate tunnel placement, placement of strong graft material at anatomic graft insertion sites, minimizing graft bending, performing final graft tensioning at 70–90 degrees of knee flexion using the graft tensioning boot (Biomet Sports Medicine), using primary and backup fixation, and the appropriate postoperative rehabilitation program.

References

1. Parolie JM, Bergfeld JA. Long term results of nonoperative treatment of isolated posterior cruciate ligament injuries in the athlete. *Am J Sports Med* 1986;14:35–38.
2. Fanelli GC. PCL tears—who needs surgery? Presented at: AANA Annual Meeting; April 1–4, 1993; Palm Dessert, CA:
3. Torg JS, Barton JM. Natural history of the posterior cruciate deficient knee. *Clin Orthop* 1989;246:208–216.
4. Dandy DJ, Pusey RJ. The long term results of unrepaired tears of the posterior cruciate ligament. *J Bone Joint Surg Br* 1982;64:92–94.
5. Keller PM, Shelbourne KD, McCarroll JR, Rettig AC. Nonoperatively treated isolated posterior cruciate ligament injuries. *Am J Sports Med* 1993;12:132–136.
6. Trickey EL. Injuries to the posterior cruciate ligament. *Clin Orthop* 1980;147:76–81.
7. Johnson JC, Back BR. Current concepts review, posterior cruciate ligament. *Am J Knee Surg* 1990;3:143–153.
8. Carlin GJ. Personal communication. Pittsburgh, PA: University of Pittsburgh Biomechanics Laboratory; March 1994.
9. Prietto MP, Bain JR, Stonebrook SN, Settlage RA. Tensile strength of the human posterior cruciate ligament. Presented at: 34th Annual Meeting, Orthopaedic Research Society; February 1–4, 1988; Atlanta, GA.
10. Woo SL, Hollis JM, Adams DJ, Lyon RM, Takai S. Tensile properties of the human femur-anterior cruciate ligament-tibia complex. *Am J Sports Med* 1991;19:217–225.
11. Cooper DE, Warren RF, Warner JJ. The posterior cruciate ligament and posterolateral structures of the knee: anatomy, functions, and patterns of injury. *Instr Course Lect* 1991;40:249–270.
12. Van Dommelen BA, Fowler PJ. Anatomy of the posterior cruciate ligament, a review. *Am J Sports Med*1989;17:24–29.
13. Girgis FG, Marshall JL, Al Monajem ARS. The cruciate ligaments of the knee joint: anatomical, functional, and experimental analysis. *Clin Orthop* 1975;106:216–231.
14. Kennedy JC, Grainger RW. The posterior cruciate ligament. *J Trauma* 1967;7:367–377.
15. Covey DC, Sapega AA, Sherman GM, Torg JS. Testing for "isometry" during posterior cruciate ligament reconstruction. *Trans Orthop Res Soc* 1992; 17:665.
16. Heller L, Langman J. The menisco-femoral ligaments of the human knee. *J Bone Joint Surg Br* 1964;46:307–313.
17. Vladimirov B. Arterial sources of blood supply of the knee joint in man. *Acta Med* 1968;47:1–10.
18. Arnoczky SP, Rubin RM, Marshall JL. Microvasculature of the cruciate ligaments and its response to injury. *J Bone Joint Surg Am* 1979;61:1221–1229.
19. Scapinelli R. Studies of the vasculature of the human knee joint. *Acta Anat* 1968;70:305–331.
20. Katonis PG Assimakopoulos AP, Agapitos MV, Exarchou EI. Mechanoreceptors in the posterior cruciate ligament. *Acta Orthop Scand* 1991;62:276–278.
21. Butler DL, Noyes FR, Grood ES. Ligamentous restraints to anterior-posterior drawer in the human knee. *J Bone Joint Surg Am* 1980;62:259–270.
22. Gollehon DL, Torzilli PA, Warren RF. The role of the posterolateral and cruciate ligaments in the stability of the human knee. *J Bone Joint Surg Am* 1987;69:233–242.
23. Brantigan OC, Voshell AF. The mechanics of the ligaments and menisci of the knee joint. *J Bone Joint Surg* 1941;23:44–66.
24. Covey DC, Sapega AA. Posterior cruciate ligament fiber length behavior under varied joint motion and loading conditions. Presented at: AAOS Annual Meeting; February 24–March 1 1994; New Orleans, LA.
25. Veltri DM, Warren RF. Posterolateral instability of the knee. *J Bone Joint Surg Am* 1994;76:460–472.
26. Clancy WG, Shelbourne KD, Zoellner GB, Keene JS, Reider B, Rosenberg TD. Treatment of knee joint instability secondary to rupture of the posterior cruciate ligament. Report of a new procedure. *J Bone Joint Surg Am* 1983;65:310–322.
27. Degenhardt TC, Hughston JC. Chronic posterior cruciate instability: nonoperative management. *Orthop Trans* 1981;5:486–487.
28. Fanelli GC. PCL injuries in trauma patients. *Arthroscopy* 1993;9:291–294.
29. Fanelli GC, Edson CJ. PCL injuries in acute traumatic hemarthrosis of the knee. Presented at: AAOS Annual Meeting; February 24–March 1 1994; New Orleans, LA.
30. O'Donoghue DH. An analysis of end results of surgical treatment of major injuries to the ligaments of the knee. *J Bone Joint Surg Am* 1955;37:1–13.
31. Parolie JM, Bergfeld JA. Long term results of non operative treatment of isolated posterior cruciate ligament injuries in the athlete. *Am J Sports Med* 1986;14:35–38.
32. Fanelli GC, Edson CJ. PCL injuries in trauma patients. Part II. *Arthroscopy* 1995;11:526–529.
33. Stanish WO, Rubinovich M, Armason T, Lapenskie G. Posterior cruciate ligament tears in wrestlers. *Can J Appl Sports Sci* 1986; 4:173–177.
34. Malek MM, Fanelli GC. Technique of arthroscopic PCL reconstruction. *Orthopaedics* 1993;16:961–966.
35. Fanelli GC, Giannotti BF, Edson CJ. Current concepts review. The posterior cruciate ligament: arthroscopic evaluation and treatment. *Arthroscopy* 1994;10:673–688.

36. Fanelli GC, Giannotti BF, Edson CJ. Arthroscopically assisted PCL/posterior lateral complex reconstruction. *Arthroscopy* 1996;12:521–530.
37. Fanelli GC, Monahan TJ. Complications of posterior cruciate ligament reconstruction. *Sports Med Arthrosc Rev* 1999;7:296–302.
38. Fanelli GC. Point counter point. Arthroscopic posterior cruciate ligament reconstruction: single bundle/single femoral tunnel. *Arthroscopy* 2000;16:725–731.
39. Fanelli GC, Monahan TJ. Complications in posterior cruciate ligament and posterolateral complex surgery. *Oper Tech Sports Med* 2001;9:96–99.
40. Fanelli GC, Edson CJ. Arthroscopically assisted combined ACL/PCL reconstruction. 2–10 year follow-up. *Arthroscopy*2002;18:703–714.
41. Fanelli GC, Edson CJ. Management of posterior cruciate ligament and posterolateral instability of the knee. In Chow J (ed.), *Advanced Arthroscopy*. New York: Springer-Verlag, 2001, pp. 545–558.
42. Fanelli GC, Monahan TJ. Complications and pitfalls in posterior cruciate ligament reconstruction. In Malek MM (editor-in-chief), *Knee Surgery: Complications, Pitfalls, and Salvage.* New York: Springer-Verlag, 2001, pp. 121–128.
43. Fanelli GC. Arthroscopic evaluation of the PCL. In Fanelli GC (ed.), *Posterior Cruciate Ligament Injuries. A Guide to Practical Management.* New York: Springer-Verlag, 2001, pp. 95–108.
44. Fanelli GC. Arthroscopic PCL reconstruction: transtibial technique. In Fanelli GC (ed.), *Posterior Cruciate Ligament Injuries. A Guide to Practical Management.*New York: Springer-Verlag, 2001, pp. 141–156.
45. Fanelli GC. Complications in PCL surgery. In Fanelli GC (ed.), *Posterior Cruciate Ligament Injuries. A Guide To Practical Management.* New York: Springer-Verlag, 2001, pp. 291–302.
46. Miller MD, Cooper DE, Fanelli GC, Harner CD, LaPrade RF. Posterior cruciate ligament: current concepts. In Beaty JH (ed.), *American Academy of Orthopaedic Surgeons Instructional Course Lectures,* Vol. 51. Rosemont, IL: American Academy of Orthopaedic surgery 2002, pp. 347–351.
47. Fanelli GC. *Arthrotek PCL Reconstruction Surgical Technique Guide. Fanelli PCL-ACL Drill Guide System*. Warsaw, IN: Arthrotek, Inc., 1998.
48. Fanelli GC, Edson CJ. Combined posterior cruciate ligament–posterolateral reconstruction with Achilles tendon allograft and biceps femoris tendon tenodesis: 2–10 year follow-up. *Arthroscopy* 2002;18:703–714.
49. Noyes FR, Barber-Westin SD. Posterior cruciate ligament revision reconstruction, part 1. *Am J Sports Med* 2005;33:646–654.
50. Sekiya JK, Haemmerle MJ, Stabile KJ, Vogrin TM, Harner CD. Biomechanical analysis of a combined double bundle posterior cruciate ligament and posterolateral corner reconstruction. *Am J Sports Med* 2005;33:360–369.
51. Robinson JR, Bull AMJ, Thomas RR, Amis AA. The role of the medial collateral ligament and posteromedial capsule in controlling knee laxity. *Am J Sports Med* 2006;34:1815–1823.
52. Fanelli GC, Giannotti BF, Edson CJ. Arthroscopically assisted combined anterior and posterior cruciate ligament reconstruction. *Arthroscopy* 1996;12:5–14.
53. Fanelli GC, Feldmann DD. Management of combined ACL/PCL/posterolateral complex injuries of the knee. *Oper Tech Sports Med* 1999;7:143–149.
54. Fanelli GC, Orcutt DR, Edson CJ. Current concepts: the multiple ligament injured knee. *Arthroscopy* 2005;21:471–486.
55. Fanelli GC, Edson CJ, Orcutt DR, Harris JD, Zijerdi D. Treatment of combined ACL PCL medial lateral side injuries of the knee. *J Knee Surg* 2005;28:240–248.
56. Fanelli GC. Surgical treatment of ACL-PCL-medial side-lateral side injuries of the knee. *Oper Tech Sports Med* 2003;11:263–274.
57. Fanelli GC. *Rationale and Surgical Technique for PCL and Multiple Knee Ligament Reconstruction. Surgical Technique Guide.* Warsaw, IN: Sports Medicine Inc., 2006.
58. Fanelli GC, Edson CJ, Reinheimer KN. Posterior cruciate ligament reconstruction: transtibial tunnel surgical technique. *Orthop Today* 2007;27:40–46.
59. Fanelli GC. Surgical treatment of lateral posterolateral instability of the knee using biceps tendon procedures. *Sports Med Arthrosc Rev* 2006;14:37–43.
60. Fanelli GC. Surgical reconstruction for acute posterolateral injury of the knee. *J Knee Surg* 2005;28:157–162.
61. Muller W (ed.). *The Knee: Form, Function, and Ligamentous Reconstruction*. New York: Springer-Verlag, 1983, pp. 246–248.
62. Fanelli GC, Lason RV. Practical management of posterolateral instability of the knee. *Arthroscopy* 2002;18(Suppl 1):1–8.
63. Fanelli GC. *The Cascade System in Knee Ligament Reconstruction. Surgical Technique Manual.* Edison, NJ: Musculoskeletal Transplant Foundation, 2005.
64. Fanelli GC. Posterior cruciate ligament reconstruction. Presented at: AOSSM Specialty Day 2007. American Academy of Orthopaedic Surgeons Annual Meeting; February 17, 2007; San Diego, CA.

Arthrofibrosis and Synovial Lesions of the Knee

John C. Richmond and Iván Encalada-Diaz

Arthrofibrosis

Introduction

Restriction of motion of any joint can result from arthrofibrosis. When this involves the knee, any loss of extension is particularly bothersome, because serious gait abnormalities can result. Although limitation of knee motion may result from minor injury or even diagnostic arthroscopy, it is much more common after major injury or surgery, as in multiligament injury of the knee or periarticular fractures. Factors associated with the incidence and severity of arthrofibrosis after surgery include the magnitude of the operative procedure, restriction of motion prior to surgery, and the duration of immobilization employed postoperatively [1–4].

Loss of knee motion after surgery or injury is not a new problem. In the past, open treatment was relied upon to address motion limitations about the knee. Thompson, in the 1940s, described an extensive open quadricepsplasty to treat motion limitations about the knee [5]. Recognition of the areas that contribute to motion limitation should be attributed to Nicoll [6]. He identified four potential sites that could contribute to the problem. These included fibrosis of the vastus intermedius in and around the suprapatella pouch, intraarticular adhesions between the patella and femur, fibrosis of the vastus lateralis with adhesions to the lateral femoral condyle, and shortening of the rectus femoris. As with Thompson before him, Nicoll recommended open surgical release of all these areas until motion was regained. With the advent of arthroscopy in the 1970s, Sprague et al. recognized its potential for the treatment of arthrofibrosis about the knee [1]. Many authors since that time have furthered our understanding, evaluation, and treatment of this condition. Arthroscopic surgery, with its reduction in joint trauma, has substantially reduced the incidence and severity of postoperative arthrofibrosis about the knee. Current classification systems rely on defining the restriction of tibiofemoral motion as well as patellar mobility. An accepted classification system is presented in Table 1 [7].

Table 1 Classification of the types of arthrofibrosis of the knee

Type	Extension loss (degrees)	Flexion loss (degrees)	Patellar mobility
1	>5	None	Normal
2	None	>20	Limited inferior glide
3	>10	>25	Limited in all planes
4	>10	>30	Patella infera

Patella infera and the infrapatella contraction syndrome (IPCS) were initially recognized by Paulos et al. [3]. This is a specific and very difficult to treat contracture that results from abnormal fibrosis of the infrapatella fat pad and the peripatella tissues. Progressive shortening of the patellar tendon in this condition makes it particularly resistant to treatment.

Whereas prevention of arthrofibrosis cannot be completely attained to date, there are a number of mechanisms whereby its frequency can be reduced after surgery. Attention to these details includes the timing of surgical procedures such that full motion is attained preoperatively, positioning of grafts anatomically, and rehabilitation protocols that encourage early mobilization, when appropriate [4, 7, 8].

Anatomy

The majority of the pathology related to arthrofibrosis is intraarticular. Most commonly, the condition begins with the formation of intraarticular adhesions. Motion limitation by pain, hemarthrosis, or an incorrectly positioned graft

J.C. Richmond (✉)
Orthopaedic Surgery, New England Baptist Hospital, Tufts University School of Medicine, Boston, Massachusetts

B.P. McKeon et al. (eds.), *Knee Arthroscopy*, DOI 10.1007/978-0-387-89504-8_10,

will lead to scar formation within the joint. If rehabilitation techniques are inadequate to overcome the problem, the scar will contract. Motion limitation may become progressive, and, in the worst case, progressive fibrosis of the infrapatella fat pad results in IPCS and patella infera [2, 3]. Secondary contractures can occur in either the quadriceps or hamstring muscles [6]. Capsule contractures from prolonged limitation of motion also need to be considered. With limitation of flexion, secondary contracture in the suprapatellar pouch and gutters may occur. Whereas the arthroscopic surgeon will tend to focus on intraarticular pathology, it is important to understand there are extraarticular anatomic components to these contractures as well. The most common issue presented is a shortening of the quadriceps mechanism. This can occur proximal to the patella with an extension contracture where the quadriceps muscle is shortened and normal gliding over the anterior femur reduced [5, 6]. With IPCS, the quadriceps mechanism is shortened below the patella, with a patella baja as evidenced radiographically [3, 4].

Pathophysiology

The normal healing process is regulated by numerous growth factors, which are produced locally by cells that migrate to an area of injury. This leads to a process that includes a series of interrelated steps to produce scar. At the time of injury and active healing that follows, the expression of growth factors is highest and then progressively declines. Mature scar tissue does not produce growth factors. In the case of IPCS, the regulatory process to stop the production of growth factors and inflammatory kinins does not function properly. The scar that forms in IPCS is very similar histologically in response to stress to the fibrotic tissue from Dupuytren′s contracture [9–13]. Both have been studied and respond abnormally by the expression of transforming growth factor beta and interleukin-6 in response to cyclic loading in tissue culture. Interleukin-6 is a potent mediator of inflammation, and transforming growth factor beta is a potent mediator of scar formation. This abnormal expression of these two regulatory molecules may explain the frequently noted inflammatory response to repetitive stretching in arthrofibrosis [14]. Aggressive stretching of these joints after surgery may in fact stimulate and perpetuate the inflammatory and scar-forming processes. Alpha smooth muscle contractile fibroblastic cells also have been identified in arthrofibrosis tissue from the knee. This contractile protein produced by myofibroblast may contribute to contraction of the scar and shortening of the periarticular tissues in arthrofibrosis [15].

Indications and Techniques

The early signs of arthrofibrosis may be a delay in return of motion after injury or surgery. Magnetic resonance imaging (MRI) for soft tissue injuries and computed tomography (CT) scans for fractures about the knee are important adjuncts to identify mechanical blocks to motion after injury. Malpositioned anterior cruciate ligament (ACL) grafts remain a common cause of postoperative knee stiffness. Routine radiographs are often adequate to identify malpositioned ACL grafts, although MRI scanning may be necessary to identify notch impingement limiting motion. If a patient fails to regain adequate early extension after ACL surgery, then an aggressive approach for diagnosis of the etiology of this motion loss is appropriate, because a lack of extension may lead to early patellofemoral arthrosis, a frequent complication of flexion contractures of the joint [3, 4, 7, 16].

If a mechanical block to motion is identified through imaging studies, then early surgery, such as meniscal repair for a displaced bucket-handle tear or notchplasty for impinging ACL graft, is indicated early on to reduce the risk of secondary damage to the joint. If no mechanical block to motion is identified, then clinical evaluation in the joint is important in decision-making. If there is significant warmth about the knee, this is evidence of a developing IPCS, and aggressive physical therapy is contraindicated. Repetitive forceful stretching of the joint will perpetuate the inflammation and may increase the contracture [3]. If the joint is cool, without inflammation, then a focused mobilization program through physical therapy may assist in regaining motion.

Pain is often a symptom of a developing IPCS. When IPCS is recognized in the inflammatory phase, the authors believe this is very similar to the inflammatory phase of adhesive capsulitis of the shoulder. The authors rely on oral corticosteroids over a 4-week taper in conjunction with a very gentle physical therapy program to maintain motion and relieve this inflammation. It is crucial that the inflammatory phase is eliminated prior to undertaking more aggressive surgical treatment of the arthrofibrosis.

When there is a functional limitation to the patient as a result of this loss of motion, then surgical treatment is appropriate. The type of surgery, the location where it is carried out (surgicenter vs. hospital), and postoperative rehabilitation are determined by the type of arthrofibrosis as noted in Table 1. Whereas types 1 and 2 are often easily addressed in an outpatient surgicenter, followed by immediate outpatient physical therapy, types 3 and 4 typically require inpatient hospitalization for several days for pain management and optimal mobilization postoperatively [17].

Type 1 limitation of motion, with a pure extension loss, typically results from scar that is either anterior in the notch or about the fat pad. An anteriorly placed tibial tunnel for an ACL graft, impinging on the notch, also may lead to a flexion contracture. A flexion contracture of as little as 5–8 degrees may result in a limp and anterior knee pain, necessitating surgical treatment. Contractures of 10 degrees or more are almost always symptomatic. The authors rely on arthroscopy for evaluation and treatment in this condition, although a combination of arthroscopic and limited open techniques may be employed for scarring of the fat pad to the anterior tibia. The authors prefer general or spinal anesthesia for type 1 or 2 arthrofibrosis, with monitored anesthesia care in conjunction with local anesthesia typically being inadequate to allow the extensive resection of scar tissue that is appropriate. The authors typically do not use a tourniquet during these procedures and rely on the injection of a local anesthetic with epinephrine into the joint prior to beginning the procedure and meticulous hemostasis with electrocautery during the procedure to control bleeding. Radiofrequency devices can be used instead of the shaver to resect the scar, as the hemostatic effect is beneficial. Most commonly, adhesions between the fat pad and the intercondylar notch or a Cyclops lesion anterior to an ACL graft are present. In type 1 arthrofibrosis, one rarely encounters adhesions in the suprapatella pouch or gutters, but, if these are identified, they should be resected. If full extension cannot be obtained after resection of all intraarticular scar, then the extrasynovial region between the fat pad extending to the distal patellar tendon and the tibia should be addressed. Although this can be done arthroscopically via the joint, it is the authors' preference to do this through a small incision just lateral to the tendon, using a blunt obturator or periosteal elevator to free the fat pad and patellar tendon from the tibia, reestablishing the bursa. This surgery may be carried out in a surgicenter on an outpatient basis, if it is perceived that outpatient pain management and physical therapy are appropriate for the patient [4, 17]. Certainly, hospitalization on an observation status for 23 hours or as an inpatient for longer may be appropriate. Splinting in extension or the use of a commercial extension board is important in the early postoperative period. Immediate physical therapy (which can be done either as an inpatient or outpatient) to maintain full extension and mobilization of the joint is appropriate. Use of continuous passive motion (CPM) machines in this situation is actually counterproductive, because full extension (or natural hyperextension) can be difficult to obtain with CPM. Patients should be encouraged to use crutches (partial weight-bearing) until they have full active extension and can maintain this extension with gait.

Type 2 arthrofibrosis typically results from immobilization in extension or excessive pain limiting flexion early on after surgery or injury [2, 7, 16]. Clinical evaluation shows that only flexion is limited and that there is decreased inferior excursion of the patella (but no significant limitation of medial or lateral glide). When appropriate physical therapy fails to regain adequate motion, then surgery is indicated. As with type 1 arthrofibrosis, it may or may not be necessary to use a tourniquet. Again, we favor distension of the joint with use of a local anesthetic with epinephrine prior to beginning the procedure to assist in hemostasis and also facilitate insertion of instrumentation. Anteromedial and anterolateral portals are usually all that are needed. Typically, the suprapatella pouch is either obliterated with scar or has significant adhesions from the quadriceps mechanism to the anterior femur (Fig. 1). There may also be scar anterior in the notch. Initial resection of all scar using motorized shaver or radiofrequency electrocautery should be accomplished (Fig. 2). Meticulous hemostasis is necessary. The distal quadriceps muscle also should be freed from the anterior femur to allow its normal excursion. We typically do this with a blunt obturator, dissecting medial, anterior, and lateral to the femur. At the completion of the resection of all scar and freeing of the quadriceps from the femur, the joint should be manipulated into full flexion. This is accomplished with gentle sustained pressure. The tourniquet, if used during scar resection, should be deflated prior to manipulation to allow normal quadriceps muscle excursion. If any excessive bleeding is encountered at this point, it is the authors' preference to drain the joint with suction drains placed into the medial and lateral gutters and to admit the patient for a minimum of 23 hours. As with surgery for type 1 arthrofibrosis, this surgery often may be carried out in a surgicenter on an outpatient basis, if it is perceived that outpatient pain management and physical therapy are appropriate for the patient. Certainly,

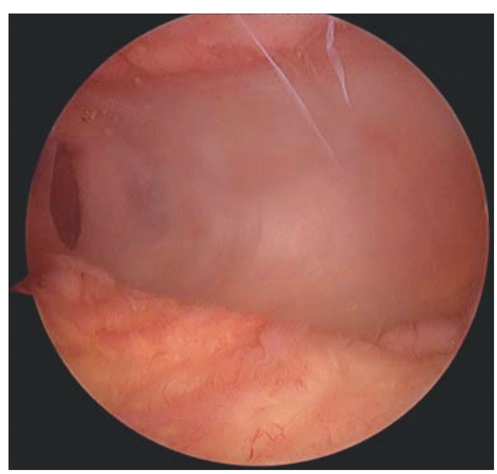


Fig. 1 Arthroscopic view of the suprapatella pouch, which is markedly reduced in size due to dense adhesions from the quadriceps tendon to the anterior femur

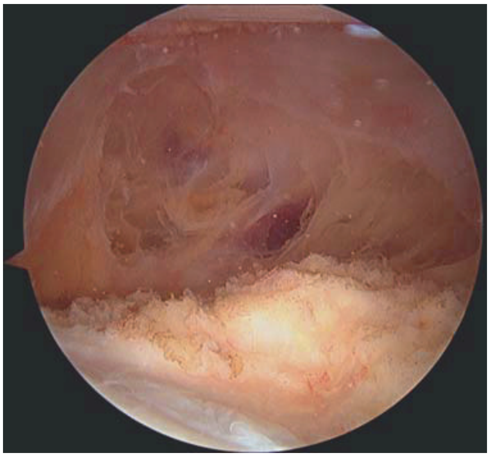


Fig. 2 Restoration of the normal suprapatella pouch, with muscle fibers visible beyond the synovium proximally

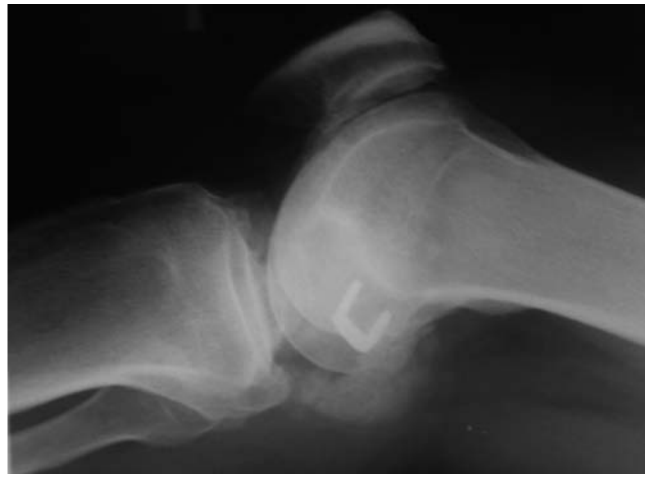


Fig. 3 Lateral radiograph demonstrating the ratio of patella tendon length to patella length equals 0.66, indicative of patella infera. Comparison with the contralateral knee as illustrated in Fig. 4 is necessary to determine if an osteotomy of the tubercle is appropriate and the correction that would be needed

hospitalization on an observation status for 23 hours or as an inpatient for longer may be appropriate. Immediate CPM machine use at the maximum flexion tolerated is encouraged. Physical therapy should begin immediately. If postoperative pain management is an issue, then a femoral nerve block (continuous, repeated, or one-time) may be exceedingly beneficial to control pain and maintain motion. A continuous epidural catheter also may be of benefit in pain management.

Types 3 and 4 arthrofibrosis include patellar entrapment syndromes, with involvement of the soft tissues about the patella and the patella tendon. When the tendon itself is involved, this results in true IPCS [2–4]. Surgery is almost always necessary to treat these conditions. Surgical treatment should be delayed until there is complete resolution of the inflammatory stage from the injury of surgery. The development of patella infera is the hallmark of IPCS. A flexed knee lateral radiograph at 45 degrees will show a reduced Insall-Salvati ratio, which is the ratio of the patella tendon length divided by the length of the patella (Fig. 3). There is a wide variability between this ratio of patellar tendon length to patellar length (range, 0.75–1.46, mean = 1.02), although there is almost no variability side-to-side in an individual patient. Comparison with the normal knee at the same flexion angle is key. More than 8 mm of shortening of the patellar tendon will require that this problem be addressed. It is the authors' preference to do this through osteotomy and slide the tubercle proximally to restore normal patellar height. Oftentimes, the DeLee type tibial tubercle osteotomy is an appropriate procedure to restore normal patellar tendon height (Fig. 4A–D) [18].

Surgical treatment for types 3 and 4 arthrofibrosis can be accomplished via arthroscopic, open, or combined procedures. Good results can be obtained with any of these techniques, and selection depends on surgeon preference [1–4, 16, 19]. To be successful, all intraarticular adhesions must be resected in a systematic fashion, beginning in the suprapatella pouch, proceeding to both gutters, and finally addressing the infrapatella fat pad and the intercondylar notch. Lateral (and oftentimes medial) retinacular releases are necessary to regain patellar mobility. The fat pad is typically scarred, and all scarring should be resected from this area, only preserving normal-appearing fat, if any remains. If a malpositioned ACL graft has been the inciting event for the arthrofibrosis, it should be resected at the same time because regaining motion will be impossible if the graft remains. In the authors' experience, fewer than 50% of those individuals that require graft excision will develop late instability. This is in part due to the periarticular scar and in part due to reduced activity levels in these patients. There is typically significant patellofemoral arthrosis that has developed during the patella entrapment, and symptoms from this arthrosis oftentimes result in reduced activity levels. We always drain these joints with suction drains positioned in both gutters to reduce the possibility of a postoperative hemarthrosis.

Manipulation of the joint at the completion of the arthrolysis should be gentle. Reestablishing of the quadriceps gliding through blunt dissection about the femur as described for the treatment of type 2 arthrofibrosis is crucial. These surgeries should all be carried out in the hospital, with inpatient hospitalization for several days to manage pain and aggressively mobilize the joint. CPM, cryotherapy, and regional pain

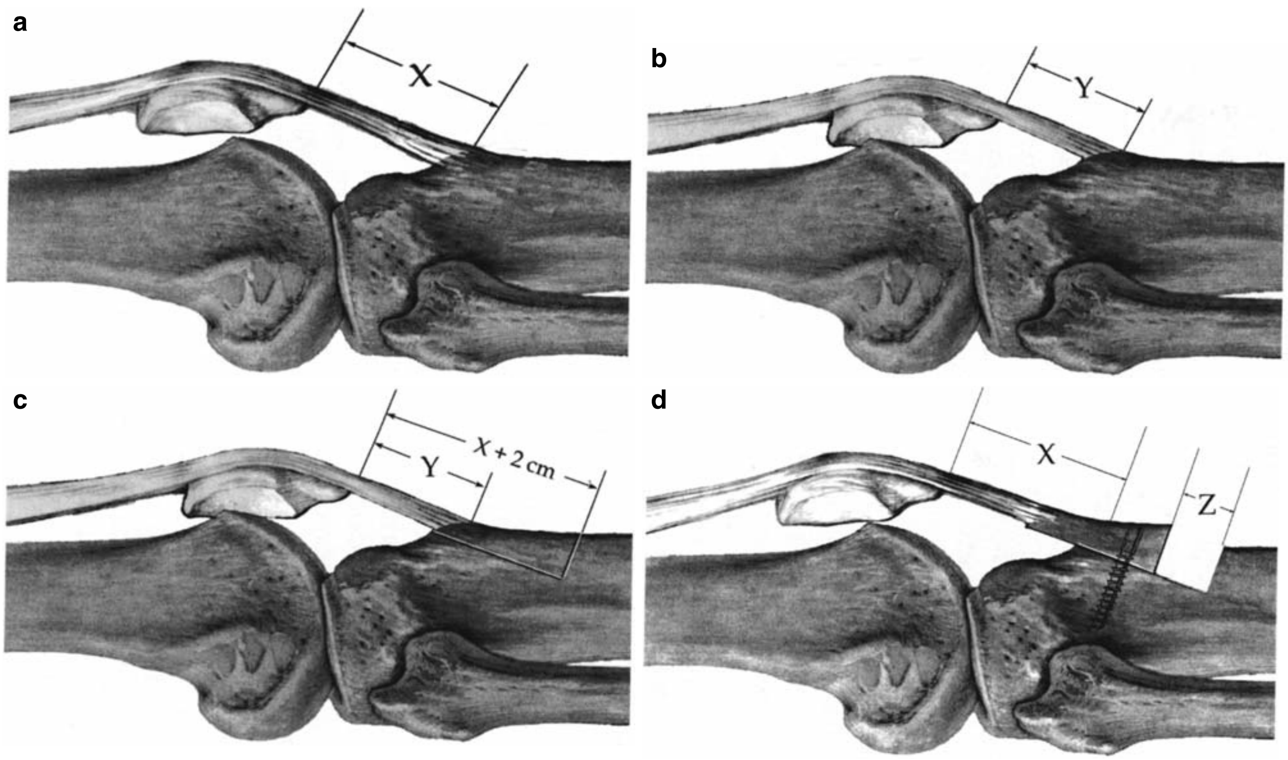


Fig. 4 This is a DeLee osteotomy, which can be used to restore the normal position of the patella in relation to the femoral trochlea. The patient's lateral radiographs, obtained at the same degree of flexion, are used to make the measurements. (**A**) The noninvolved knee is used to measure the length of the normal patella tendon (X). (**B**) The involved knee is used for the measurement of the shortened tendon (Y). (**C**) The overall length of the shortened tendon plus the osteotomy is X + 2 cm. (**D**) When the bone is cut, the tibial tubercle is slid proximally the distance $Z = X - Y$, and fixed with one or two screws. The defect Z is grafted, typically with demineralized bone matrix and allograft cancellous chips. (From Richmond JC. Arthofibrosis following knee surgery (arthroscopic/open). In Malek MM (ed.), *Knee Surgery: Complications, Pitfalls, and Salvage.* New York: Springer-Verlag, 2001. Reprinted with permission.)

management techniques (femoral nerve blocks or indwelling epidural catheter) are important adjuncts. Twice-daily physical therapy sessions for patella and joint mobilization as well as for improvement of the quadriceps muscle strength are employed while the patient is hospitalized. After discharge, daily physical therapy sessions should be planned for the ensuing 2–3 weeks. The authors often find that there is excellent early motion immediately after surgery, which declines over the ensuing 7–10 days, in the early inflammatory phase after surgery. Oral corticosteroids can be an important adjunct to limit this inflammation. The authors use a 4-week taper with 40 mg/day for the first week, 30 mg/day for the second week, 20 mg/day for the third week, and 10 mg/day for the final week. Outpatient physical therapy should be continued daily until motion begins to increase. CPM, although helpful in maintaining overall mobility, will not increase range of motion. Protected weight-bearing is necessary for a prolonged period of time, particularly if DeLee osteotomy has been employed. One should not wean these patients from crutches until there is radiographic evidence of incorporation of the graft that was used to fill the defect created by the tubercle slide.

Whereas results of treatment of types 1 and 2 arthrofibrosis are typically excellent, with restoration of near-normal range of motion and function, patellar entrapment and infrapatella contracture syndromes are typically associated with less satisfactory results. This is due in large part to damage to the articular surfaces of the patella and femoral trochlea [1–4, 16, 19]. Functional recovery and return to preinjury athletic or work status usually is determined by whether there has been significant damage to the patellofemoral cartilaginous surfaces. Whereas significant improvement in range of motion can be anticipated, function may remain limited. In those patients who have had an ACL graft removed in order to regain motion, it is crucial that revision ACL reconstruction be delayed until normal motion of the joint with good compliance of soft tissues is reattained. As noted earlier, not all patients will require revision ACL reconstruction, depending on their functional status and decreased overall joint laxity.

Complications

Hemarthrosis after arthrolysis, although not frequent, can be a devastating complication because it may lead to recurring limitation of motion. We only use the tourniquet for patellar entrapment syndromes and avoid its routine use for the treatment of type 1 and 2 arthrofibrosis. Meticulous hemostasis and a low threshold to drain the joint through vacuum drainage systems are key. Development of a synovial cutaneous fistula through the drainage site is possible, and avoiding the use of previously scarred portal sites for the drains is an important pearl. If a fistula does develop, there must be concern for the development of septic arthritis.

Vigorous manipulation at the completion of the resection of scar should be avoided [2, 3]. It is possible to avulse the patellar tendon from the tibial tubercle, particularly when the patient has been relatively inactive as a result of ongoing arthrofibrosis. The defect created in the anterior tibia when a DeLee osteotomy is performed can be problematic. If the patient resumes activity too soon, it is possible to develop a stress fracture through the remaining tibia at this site. This can be avoided by prolonged protection until there is clear radiographic evidence of incorporation of the graft and then very gradual return to strenuous activities.

Clinical Pearls and Summary

Early recognition of developing motion problems is important to limit the effects of this potentially devastating complication in patients. Identification of the inflammatory phase of patellar entrapment/infrapatella contraction syndrome, as well as its treatment with oral corticosteroids, can be exceedingly beneficial. As long as there is no mechanical block to motion, surgical intervention for motion limitations after injury or surgery can be delayed for a period of time to allow for resolution of any inflammation. Surgical principles for the treatment of the arthrofibrosis include the following:

1. Complete removal of all intraarticular and extraarticular scar.
2. Elimination of any mechanical block, such as a malpositioned ACL graft.
3. Restoration of patellar position and mobility.

These goals can be accomplished via open, arthroscopic, or combined means. It should be at the discretion of the surgeon. Any persistent limitation of motion that results from failure to achieve one of these may result in further cartilage injury. Postoperative rehabilitation should be tailored to the specific condition treated, and a skilled physical therapist is crucial to regain and maintain mobility.

Case Report

Case 1

Chief Complaint and Patient History: A 35-year-old woman was treated elsewhere with an autologous quadrupled hamstring ACL reconstruction 2 weeks after a ski injury. She is now 9 months after the initial surgery and has been unable to regain adequate flexion to kneel in her garden, in spite of vigorous physical therapy. She comes seeking a second opinion as to possible treatment options.

Physical Examination: Physical examination reveals a stable knee. She has full active extension, but lacks 30 degrees of full flexion, with decreased inferior translation of her patella.

Imaging: She brings an MRI scan obtained within the past few weeks. A representative view of the notch (Fig. 5A) demonstrates the graft to be well positioned, without impingement. With the diagnosis of type 2 arthrofibrosis, outpatient arthroscopy is recommended to address intraarticular scarring, reassuring her that the graft is functioning well and not limiting motion.

Treatment: Arthroscopy reveals that the suprapatella pouch is markedly limited due to scar (Fig. 5B). Removal of the scar, with blunt freeing of the quadriceps from the anterior femur, and gentle manipulation restores full flexion (Fig. 5C), which she is able to maintain through an early, guided physical therapy program.

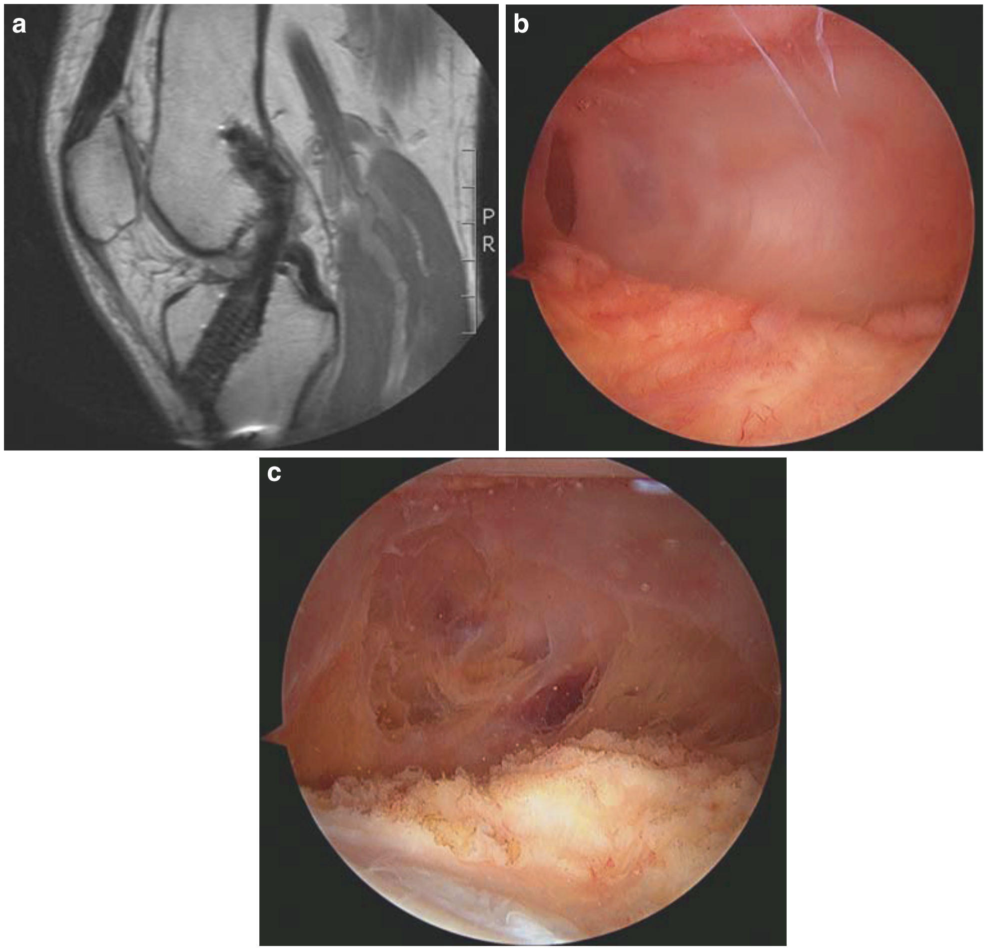


Fig. 5 Case 1. (**A**) MRI scan of a well-positioned ACL graft, without evidence of notch impingement. (**B**) Arthroscopic view of the suprapatella pouch, which is markedly reduced in size due to dense adhesions from the quadriceps tendon to the anterior femur. (**C**) Restoration of the normal suprapatella pouch, with muscle fibers visible beyond the synovium proximally

Synovial Lesions

Introduction

The synovial membrane represents the innermost lining of the capsule of the knee joint. It is richly supplied with blood vessels, lymphatics, and nerves. Histologically, the membrane is a sheet of loose fibrous connective tissue. There are two types of synovial cells. Type A synovial cells are similar to macrophages in the rest of the body. Type B is a secretory cell rich in rough endoplasmic reticulum and is the source of glycoprotein and hyaluronic acid in the synovial fluid.

In dealing with synovial lesions, it makes most sense to categorize them as either diffuse (such as rheumatoid arthritis or other inflammatory synovial conditions) or focal (such as intraarticular ganglions or lipoma arborescens). Pigmented villonodular synovitis may be either a focal localized condition or a diffuse synovial process, which may become locally invasive into the surrounding soft tissue and bone. The arthroscopic treatment of specific synovial conditions in the knee depends on the pathology, symptoms, and general status of the articular cartilage.

Anatomy

The synovium lines the entire joint cavity of the knee, with the exception of the articular surfaces. There are several remnants of embryologic invaginations into the joint, which are called plicae. Most frequent among these is the ligamentum mucosum running from the fat pad to the anterior aspect of the notch and occasionally invaginating around the ACL. The medial patella plica (sometimes referred to as the suspensory ligament or the fat pad) runs from beneath the medial retinaculum down to the fat pad. It is present in upwards of 40% of knees. It is the one frequent plica of the knee joint that is believed to potentially become pathologic and cause symptoms. Although the synovium is attached throughout the knee above the articular margins of the femur and below the articular margins of the tibia, the menisci interrupt its attachment medially and laterally. In the posterior aspect of the knee, the synovium invaginates from the posterior capsule around both cruciate ligaments. This invagination divides the posterior compartment of the knee to form the medial and lateral recesses. Frequently, there is a communication between the posteromedial compartment of the knee to the semimembranosus bursa. When this communication leads to filling of the bursa with synovial fluid and/or tissue, this is referred to as a Baker's cyst or popliteal cyst.

Indications and Techniques

Addressing diffuse synovial lesions requires specific preoperative planning and intraoperative equipment. For each diffuse synovial condition, there are specific indications for surgery and special considerations. Those conditions for which the role of arthroscopic total synovectomy has been established to have significant benefit include rheumatoid arthritis, seronegative arthritis, pigmented villonodular synovitis (PVNS), synovial chondromatosis, nonspecific monoarticular synovitis, hemophilic arthropathy of the knee, and chronic Lyme arthritis of the knee. Because of the diverse pathology involved, each of these conditions will have specific indications for arthroscopic synovectomy.

Rheumatoid Arthritis

Arthroscopic synovectomy is only indicated in patients with rheumatoid arthritis when they have failed appropriate medical management of the condition and have reasonable preservation of joint spaces [20, 21]. The optimum medical management should be determined and overseen by a rheumatologist. It is the authors' impression, although not substantiated in the literature, that the newer biologic treatments (genetically engineered proteins derived from human genes, designed to inhibit specific components to the immune system) have significantly reduced the need for arthroscopic synovectomy in rheumatoid arthritis. It has been established, however, that arthroscopic synovectomy is effective in attenuating the symptoms for those patients in whom biologics have not been successful [21]. The results of arthroscopic synovectomy in rheumatoid arthritis have been in the 70% to 80% success range over midterm (4–5 years) follow-up. It remains a viable treatment for those cases in which optimum medical management has been unsuccessful to control local disease of the knee [22, 23]. Because destruction of the joint surfaces by ongoing rheumatoid arthritis significantly reduces the efficacy of synovectomy, it should be carried out early in the course after the failure of medical treatment [20].

Seronegative Arthritis

Seronegative arthritis includes patients with various conditions such as ankylosing spondylitis, psoriatic arthritis, and Reiter's disease. As with rheumatoid arthritis, optimal medical management for at least 6 months that fails to control the synovitis is an indication for arthroscopic synovectomy. The results of surgical treatment of the seronegative arthropathies are not as good as in rheumatoid arthritis, with only 50% to 60% of patients showing significant benefit [24]. Stiffness due to the involvement of the enthesis in these conditions makes early mobilization, including CPM use, an important part of the rehabilitation program.

Pigmented Villonodular Synovitis

PVNS is a condition that has two forms, one of which is localized and the other generalized. Although histologically it may be impossible to differentiate these two, they behave very differently. The localized form often presents with mechanical symptoms related to the nodule. The diffuse form is widespread and may invade bone. Symptoms are of recurrent effusions with synovial thickening [25]. MRI is key for this diagnosis. Hemosiderin, due to its iron content and deposition within the proliferative synovium, results in areas of low signal within the synovium on both T1- and T2-weighted images, best seen on fast field echo sequence MRI scans [26, 27]. Synovial biopsy for documentation of this condition is crucial, because it is frequently resistant to treatment and adjunctive therapy such as irradiation, which may be necessary if it recurs. The arthroscopic appearance of PVNS is distinctive, with thick, hemosiderin-stained fronds (Fig. 6). Local invasion of the soft tissue mass into the popliteal space can occur, and this precludes arthroscopic treatment in the posterior compartment in the knee. In such cases, open posterior removal of the invading soft tissue mass may be indicated [28].

Synovial Chondromatosis

As with PVNS, synovial chondromatosis comes in two major forms: one localized with minimal synovial reaction; the other a diffuse form with many cartilaginous loose bodies

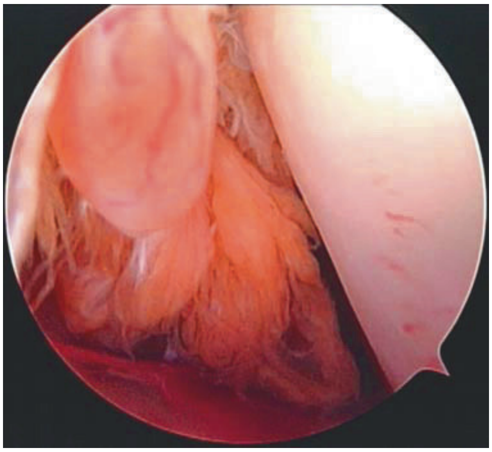


Fig. 6 Dense hemosiderin-stained fronded synovium is the typical arthroscopic appearance of PVNS

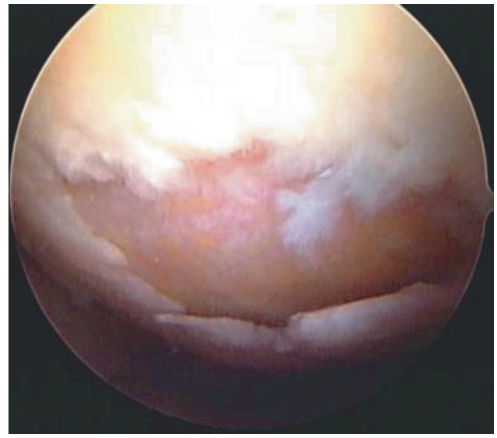


Fig. 7 Articular cartilage full-thickness loss may be a relatively early consequence of recurrent hemarthroses from hemophilia

and an actively inflamed synovium. Ogilvie-Harris and Salah demonstrated that synovectomy in conjunction with removal of all loose bodies is necessary to eradicate this disease [29]. Because the loose bodies may be small and lodged in any of the interstices about the knee, we recommend careful, thorough repeat examination of the knee at completion of the synovectomy to be sure that all loose bodies have been removed to the maximum extent possible. Recurrence rates after synovectomy with loose body removal are low, ranging from 0 to 10%.

Nonspecific Synovitis

Patients who have a mono-arthropathy, for which no specific diagnosis can be established, are believed to have nonspecific synovitis. Serologic testing for spirochetes is indicated [30]. Arthroscopy and biopsy may be warranted, as well as culturing for bacteria, tuberculosis, and fungi. If no specific diagnosis can be established, then arthroscopic synovectomy is indicated if there is failure of medical management [24]. Some of these patients will go on, at a later date, to develop classic inflammatory arthropathy involving multiple joints, such as rheumatoid arthritis or seronegative arthritis.

Hemophilic Arthropathy

A common complication of hemophilia is recurrent, often spontaneous, intraarticular hemorrhage into the knee or other joints. It is believed that the bleeding episodes produce synovitis through hypertrophy of the synovium caused by hemosiderin deposition. This results in the release of catabolic enzymes and degradation of the articular surfaces. In recurrent hemarthrosis, the primary indication for arthroscopic synovectomy is failure of medical management after 6 months of prophylaxis with appropriate clotting factor replacement. Relatively rapid destruction of the articular surface may result from the recurrent hemarthroses, as is seen in Fig. 7, where only 14 months of recurrent bleeds into the knee resulted in marked damage to the articular cartilage of the medial femoral condyle of this 26-year-old man. Advanced arthropathy is a contraindication to arthroscopic synovectomy, because the persistent pain and motion loss will continue to limit function. Careful perioperative management of factor replacement is key to success in the treatment of these patients with synovectomy. It is typically recommended that patients be maintained above 100% normal factor levels for at least 3 days after surgery and then gradually tapered to maintenance levels over several weeks to months during rehabilitation to regain motion [31, 32].

Lyme Arthritis

Lyme arthritis is caused by an infection with the spirochete *Borrelia burgdorferi,* which is transmitted by the deer tick. Lyme arthritis typically occurs late in the course of the disease (weeks, months, or years after the initial infection). Oral antibiotics are usually curative for the arthritis, but a small percentage of patients with a specific human leukocyte antigen (HLA) haplotype and related alleles may develop treatment-resistant arthritis, very similar to rheumatoid arthritis. Arthroscopic synovectomy can be considered for those patients in whom antibiotics have not been curative of the condition. Unfortunately, fewer than 50% of patients respond well to this treatment in chronic Lyme arthritis [30].

Surgical Technique and Planning

Preoperative planning is extremely important in dealing with diffuse synovial conditions of the knee when synovectomy is indicated. Because it is often important to be working in the posterior compartment of the knee, a 70-degree arthroscope should be available. High fluid flow is necessary both to maintain distension and also to limit bleeding. Optimally, a pump, which has both volume and pressure controls, should be used. One should begin with as low a pressure setting as can be used to maintain distension and visualization (typically 35–40 mm Hg) and only increase the pressure as needed for visualization. It is essential the pump maintain a clear field when working in the posterior compartment of the knee, to reduce the risk for neurovascular injury.

Tourniquet use is almost always indicated when synovectomy is being performed for a diffuse synovial conditions. Otherwise, bleeding during the course of the procedure will make it difficult to accomplish the procedure. Although new radiofrequency electrocautery devices may be effective in performing a synovectomy, they should be used with caution when there is significant hypertrophy of the synovium (such as in rheumatoid arthritis or PVNS), because heating of the fluid and secondary thermal damage to the joint may occur. It remains the authors' preference to use motorized shavers to perform the bulk of the synovectomy and then complete the procedure with thermal devices for cauterization of the bleeding points.

One must be systematic in performing a total (or near-total) synovectomy of the knee. The authors prefer to inflate the tourniquet at the beginning of the procedure, start with routine anterolateral and anteromedial portals for diagnosis, and synovial biopsy if indicated. Once a thorough diagnostic exam has been completed, synovectomy is carried out in a stepwise fashion as indicated in Table 2.

As the authors proceed through each step of the synovectomy, they begin with the motorized shaver, using the largest full radius resector blade available that can be safely inserted into the given location, to remove as much synovium as rapidly as possible. There are time constraints on use of the tourniquet, and a total synovectomy can be a complicated and tedious procedure, pushing the time limits for tourniquet use. One will typically begin with a 5.5 mm resector in the suprapatella pouch and both gutters and reduce down to a 3.5 mm to work under the menisci and in the posterior compartments. The authors routinely cauterize the areas treated prior to moving their instrumentation to the next position. To access the posterior compartments in steps 5 and 6, the knee should be flexed 70–90 degrees. This opens the intercondylar notch for the instrumentation, and the posterior capsule distension displaces the neurovascular contents (popliteal artery and vein, tibial nerve) from the synovialized space. There are several ways to identify the appropriate start point externally. The authors' preference is to use palpation or transillumination to identify the external start point and then to insert a spinal needle percutaneously to confirm appropriate position and direction of the portal [33, 34]. Incise the skin and then use blunt dissection with either a straight hemostat or blunt obturator down to the synovium. It is often easier to penetrate the synovium with a sharp obturator. Once the synovium has been entered, use a switching stick technique with either a small metal or plastic cannula to allow instrumentation to be placed. The authors prefer small plastic cannulas with a resealable diaphragm in order to maintain distension and to switch between various-sized motorized instruments and radiofrequency electrocautery devices. In establishing the posteromedial portal, the saphenous nerve and vein are the neurovascular structures at risk, and these can be safely avoided by this technique.

Visualizing and instrumenting the posterolateral compartment can be accomplished in a similar fashion to that for the posteromedial compartment [33, 34]. It is feasible to either leave the arthroscope in the anterolateral portal and pass it under the ACL or, as is the authors' preference, to place the arthroscope in the anteromedial portal and pass it between the lateral femoral condyle and the ACL into the posterolateral compartment. Again, the knee should be flexed 70–90 degrees. By palpation, one should stay several centimeters anterior to the biceps tendon to establish the entry site, use transillumination, and then localize with a spinal needle as described earlier. Again, incising the skin sharply, using blunt dissection down to the capsule and then a blunt or sharp obturator to puncture the synovial space, is a safe technique. When instrumenting the posterolateral compartment, the popliteal artery and vein, the tibial nerve posteriorly, and the common peroneal nerve laterally are the structures at risk.

In performing synovectomy in either of the posterior compartments, it is crucial that visualization be maintained. An alternative technique for working from one compartment to the other posteriorly is to use the transseptal portal as described by Ahn and Ha [35]. This technique was simplified by Louisia et al., describing a back-and-forth "modification" of the original technique [36]. A detailed knowledge of the anatomy is necessary for this. It is best performed with the arthroscope in the posteromedial portal using a blunt obturator from the posterolateral portal to penetrate the posterior septum behind the midportion of the posterior cruciate ligament (PCL). A small full radius resector can then be used to clear the synovium from the septum to allow easy back-and-forth instrumentation of the posterior compartments. It is key that the synovial resector be aimed anteriorly when enlarging the opening in the septum so that the popliteal artery and vein can be avoided (Fig. 8). It is recommended that one practice this technique in a cadaver lab prior to using it clinically.

Table 2 Steps and positioning of the arthroscope and instrumentation for performing an arthroscopic synovectomy of the knee

Step	Arthroscope location	Resector location	Areas treated
1	Anterolateral	Superolateral	Suprapatella pouch Medial gutter Lateral gutter
2	Anterolateral	Anteromedial	Medial gutter Intercondylar notch Fat pad Beneath menisci
3	Anteromedial	Anterolateral	Lateral gutter Intercondylar notch Fat pad Beneath menisci
4	Anteromedial	Superomedial	Medial gutter Remaining suprapatella pouch
5	Anterolateral Pass through the notch	Posteromedial	Posteromedial
6	Anteromedial/anterolateral Pass through the notch	Posterolateral	Posterolateral

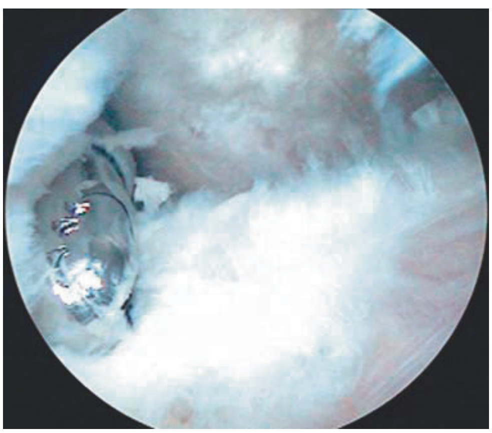


Fig. 8 Arthroscopic view from the posteromedial portal through the defect created in the septum behind the PCL, which is to the right. The lateral femoral condyle is above and to the right of the shaver

Postoperative Management

The authors find it is advisable to drain the knee after a complete synovectomy. It is the authors' preference to thread each arm of a vacuum drain down through the superolateral and superomedial portals into the lateral and medial gutters, under direct vision with the arthroscope. The drains are sewn to the skin with the nylon sutures used to close the portal sites to prevent their migration out of the joint early on. It has been our preference to use immediate passive motion while in the hospital to facilitate regaining motion. The drains are removed as soon as drainage is below 75 mL per 8-hour shift. An outpatient physical therapy program, supervised by a therapist, is necessary after discharge to ensure that the patient progresses with increasing motion, muscle control, and return to function.

Localized Pathology of the Synovium

Multiple conditions can cause local synovial pathologies. The site of the disease often will determine the degree of symptoms it causes. The most common focal synovial problem is the medial patellar plica syndrome [37]. Other less common conditions include focal PVNS, Hoffa's disease of the infrapatella fat pad, intraarticular ganglion, and lipoma arborescens [28, 38, 39]. Multiple other pathologic entities have been described in the synovium, typically published as case reports, as in intraarticular hemangioma and non–Hodgkin's lymphoma [40, 41]. Any pathologic tissue recognized within the joint at arthroscopy should be biopsied, with tissue sent for pathologic examination [42].

The popliteal or Baker's cyst represents a common condition associated with other intraarticular pathology of the knee. Traditionally, it has been the teaching that correcting the underlying pathology, which leads to the recurrent intraarticular effusion, will result in resolution of the cyst. If the cyst does not resolve, then directly addressing the cyst should be considered if it remains symptomatic [43]. It should also be addressed if there is a significant intraarticular synovial process (such as rheumatoid arthritis or PVNS) that has invaded the cyst as evidenced by imaging. With the arthroscope in the anterolateral portal, passed through the notch into the posteromedial compartment, the opening to the Baker's cyst/semimembranosus bursa can be identified.

Through the posteromedial portal a shaver can be brought to the mouth of the cyst, and the opening can be enlarged. Using caution, the lining of the cyst can be excised by passing a small (3.5–4.5 mm) full radius resector from the posteromedial portal into the cyst. Care must be maintained to keep the tip of the shaver within the cyst to avoid injury to other popliteal contents.

Symptomatic Plica

The medial patella plica is the only synovial fold located within the knee, which is widely believed to be associated with significant symptomatology [37]. This plica is present in somewhere between 20% and 80% of the population (depending on regional variations, with the incidence being higher in Asians and lower in Caucasians). A specific symptom complex should be looked for preoperatively to identify a symptomatic plica. This includes pain located in the medial parapatellar area, with snapping or popping when flexing and extending the knee. With palpation, if the plica can be rolled between the medial retinaculum and the medial border of the trochlea to reproduce the patient's symptoms, then the presumptive diagnosis of symptomatic medial patellar plica can be made. Conservative treatment is indicated, including nonsteroidal anti-inflammatory drugs and physical therapy modalities. Local corticosteroid injection into the subsynovial tissues (not intraarticularly) in the region of the plica also can be of diagnostic and therapeutic value. Chronic thickening of the plica from rubbing against the articular surface of the trochlea over long periods of time can occur and may erode the articular surface in this locus (Fig. 9). Treatment of a symptomatic medial patella plica, if more conservative modalities are unsuccessful, is arthroscopic resection. This can be accomplished with the arthroscope in the anterolateral portal and the shaver brought in from the anteromedial portal. Sometimes, due to the angles involved, this is technically difficult to accomplish and may be facilitated by establishing a superolateral portal for resection.

Hoffa's Disease: Fibrosis of the Infrapatella Fad Pad

This is truly a diagnosis of exclusion. The condition results from inflammatory fibrosis within the fat pad. One must rule out all other synovial diagnoses before reaching this. An MRI scan can be very helpful in ruling these out. Hoffa's sign (pain produced by pressure on the medial side of the fat pad as the knee is brought from 90 degrees of flexion to full extension, entrapping the fat pad under the patellar tendon) is believed to be diagnostic of the condition. Local excision of all fibrotic fat should be curative.

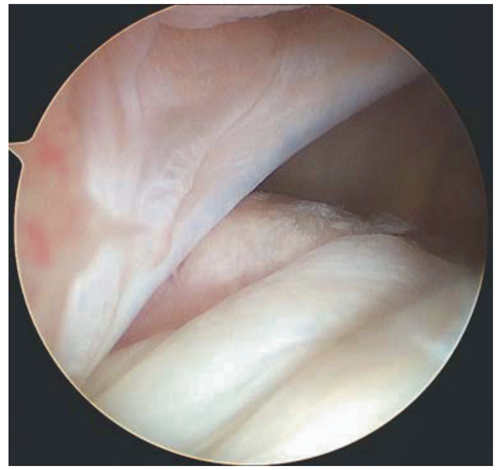


Fig. 9 Left knee view of a long-standing symptomatic mediopatellar plica, which has led to significant erosion of the articular cartilage of the medial femoral trochlea

Ganglions

Ganglions have been recognized as an intrasynovial pathology that can be associated with painful symptoms. They have been identified in multiple locations about the joint, including around the cruciates and in the fat pad. They are readily diagnosable through use of MRI scan (Fig. 10). Arthroscopic surgical treatment with excision of the ganglion can be easily accomplished, leading to excellent relief of the symptomatology (Fig. 11) [38].

Localized PVNS

The localized form of PVNS is actually more common in the knee joint than the diffuse variety. Focal pedunculated lesions are typically identified in the suprapatella pouch or the gutters. They often are associated with mechanical symptoms because of their pedunculated nature. MRI scans have the same signature as the more diffuse form. It is not known whether local PVNS will spread to form the diffuse variety if not recognized and treated early. The series that has been reported uniformly in the literature notes no clinical signs of recurrence in those patients treated with either open or arthroscopic excision of the localized PVNS [28].

Synovitis and Osteoarthritis of the Knee

Osteoarthritis of the knee is a progressive condition in which the synovium may become focally inflamed and hypertrophic. This contributes to recurrent effusions and pain of osteoarthritis. The hypertrophic synovium also may be

Fig. 10 MRI scan demonstrating intraarticular ganglions, just anterior to the ACL in the fat pad. These often cause localized pain, which can be relieved by arthroscopic removal

Fig. 11 Arthroscopic view of the ganglion imaged in Fig. 10, prior to resection

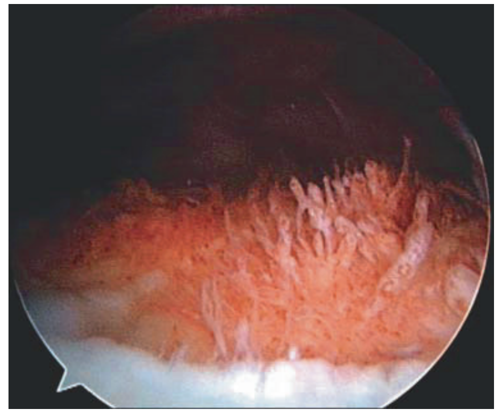


Fig. 12 Hypertrophic synovium in the suprapatella region in a patient with moderate degenerative arthritis and recurrent effusions

an accelerant to the degradative processes by the catabolic enzymes produced. When significant synovitis is identified in the osteoarthritic knee at the time of arthroscopy for mechanical conditions (Fig. 12), then focal synovectomy is indicated [44]. This is best carried out with either radiofrequency electrocautery or whisker-type motorized shaving blades, followed by cauterization of the bleeding points (Fig. 12). Although the arthroscopic treatment of osteoarthritis is palliative, it may significantly improve function for moderate (2–5 years) periods of time prior to more definitive surgical procedures.

Various other intrasynovial conditions have been recognized and identified through the years. Arthroscopic treatment of these may be appropriate depending on the findings. Whereas these lesions are usually benign, it is possible that a malignant condition can present intraarticularly. This has been noted in non–Hodgkin's lymphoma, which was recognized at arthroscopy for presumed meniscus tear. As noted previously, any pathologic synovial tissue, particularly of an unusual appearance, should be biopsied for pathologic examination.

Complications

Because of the extensive nature of total synovectomy when performed arthroscopically, limitations of motion after this procedure have been described [20, 21, 24, 25, 28]. The aggressive approach to physical therapy we have outlined has been successful in our hands in regaining motion after total or subtotal synovectomy. When the posterior compartment is instrumented, neurovascular injury is possible and meticulous technique is necessary to avoid this. Minor complications around portal sites can occur. Use of radiofrequency wands to cauterize the synovial surface after synovectomy is of significant benefit in potentially reducing the risk of hemarthrosis. Care must be used to maintain high fluid flow when using radiofrequency wands, such that thermal injury to the articular cartilage does not occur.

Clinical Pearls and Summary

For diffuse synovial lesions and inflammatory conditions, it is key that a thorough diagnostic evaluation and appropriate medical management be carried out prior to undertaking arthroscopic synovectomy of the knee. A systematic six-step approach (Table 2) using both a motorized shaver and a radiofrequency wand will facilitate as complete a resection of the pathologic synovium as is possible while reducing the risk of postoperative hemarthrosis. For total synovectomy, suction drainage, short-term hospitalization, and early passive motion facilitate regaining motion and can reduce the risk of postoperative stiffness of the knee. Any unusual-appearing synovial lesion should be removed and the resected tissue sent for pathologic evaluation. Limited synovectomy should be considered in those patients with osteoarthritis undergoing arthroscopic treatment of a symptomatic meniscal tear, when they have demonstrated recurrent preoperative effusions and when the arthroscopy reveals significant inflammatory synovitis.

Case Report

Case 2

Chief Complaint and Patient History: A 43-year-old man presents to your office with a recent ACL tear, confirmed by MRI, with symptomatic buckling of the knee when cutting or turning. He has had a long history of anterior knee pain that is slowly worsening. It is medial to the patella and associated with snapping as he ascends or descends stairs.

Physical Exam: His examination reveals a 2+ anterior drawer and Lachman, with a positive pivot-shift. He is tender over the medial retinaculum, and you can roll a palpable band, which is tender, over the condyle.

Treatment: At the time of ACL reconstruction, a thickened mediopatellar plica is identified, with an erosion of the cartilage of the medial trochlea (Fig. 13). Resection of the plica is readily accomplished. Although symptomatic plicae are not uncommon, it is distinctly unusual to identify any articular cartilage injury associated with them. Resection of any plica that is causing symptoms, while one is arthroscoping the knee for another reason, is appropriate.

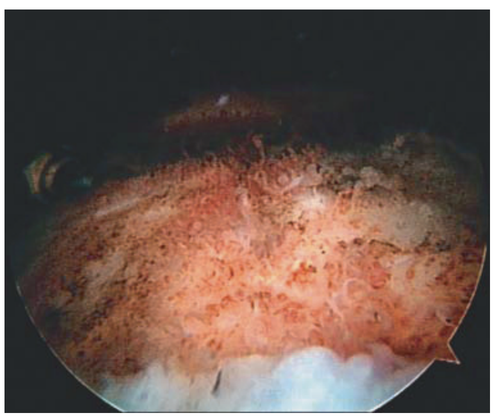


Fig. 13 Case 2. Synovectomy of the knee depicted in Fig. 12 was accomplished with a whisker-type shaver blade, and the synovium was then cauterized using a radiofrequency wand

References

1. Sprague NF III, O'Connor RL, Fox JM. Arthroscopic treatment of postoperative knee fibroarthrosis. *Clin Orthop Relat Res* 1982;166:165–172.
2. Richmond JC, Assal MA. Arthroscopic management of arthrofibrosis of the knee, including infrapatellar contraction syndrome. *J Arthrosc Relat Surg* 1991;7:144–147.
3. Paulos LE, Wnorowski DC, Greenwald AE. Infrapatellar contracture syndrome: diagnosis, treatment, and long-term follow-up. *Am J Sports Med* 1994;22:440–449.
4. Magit D, Wolff A, Sutton K, Medvecky M. Arthrofibrosis of the knee. *J Am Acad Orthop Surg* 2007;15: 682–694.
5. Thompson TC. Quadricepsplasty to improve knee function. *J Bone Joint Surg* 1944;15:366–379.
6. Nicoll EA. Quadricepsplasty. *J Bone Joint Surg Br* 1963;45: 483–490.
7. Richmond JC. Arthrofibrosis. In McGinty JB, Burkhart SS, Jackson RW, Johnson DH, Richmond JC (eds.), *Operative Arthroscopy*, 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2003, pp. 335–346.

8. Shelbourne KD, Nitz P. Accelerated rehabilitation after anterior cruciate ligament reconstruction. *Am J Sports Med* 1990;18: 293–299.
9. Richmond JC, Alman BA, Pojerski M. Growth factor expression in arthrofibrosis. *Arthroscopy* 1996;12:352–353.
10. von Deck MD, Richmond JC, Alman BA. Arthrofibrosis: a potential treatment based on growth factor manipulation. *Arthroscopy* 1997;13:393–394.
11. Alman BA, Goldberg MJ, Nabor SP, Galanopoulous T, Antoniades HN, Wolfe HJ. Aggressive fibromatosis. *J Pediatr Orthop* 1992;12:1–10.
12. Alman BA, Nabor SP, Terek RM, Jiranek WA, Goldberg, MJ, Wolfe, HJ. Platelet-derived growth factor in fibrous musculoskeletal disorders: a study of pathologic tissue sections and primary cell cultures. *J Orthop Res* 1995;13:66–77.
13. Terek RM, Jiranek WA, Goldberg MJ, Wolfe HJ, Alman BA. The expression of platelet-derived growth factor gene in Dupuytren contracture. *J Bone Joint Surg Am* 1995;77:1–9.
14. Skutek M, van Griensven M, Zeichen J, Brauer N, Bosch U. Cyclic mechanical stretching enhances secretion of Interleukin 6 in human tendon fibroblasts. *Knee Surg Sports Traumatol Arthrosc* 2001;9:322–326.
15. Unterhauser FN, Bosch U, Zeichen J, Weiler A. Smooth muscle actin containing contractile fibroblastic cells in human knee arthrofibrosis tissue. *Arch Orthop Trauma Surg* 2004;124: 585–591.
16. Kim DH, Gill TJ, Millett PJ. Arthroscopic treatment of the arthrofibrotic knee. *J Arthrosc Rel Surg* 2004;20:187–194.
17. Shelbourne KD, Johnson GE. Outpatient surgical management of arthrofibrosis after anterior cruciate ligament surgery. *Am J Sports Med* 1994;22:192–197.
18. Richmond JC. Arthrofibrosis following knee surgery. In Malek MM, Fanelli G, Johnson D, Johnson D (eds.), *Knee Surgery, Complications, Pitfalls, and Salvage*. New York: Springer-Verlag, 2000, pp. 450–460.
19. Millett PJ, Williams RJ, Wickiewicz TL. Open debridement and soft tissue release as a salvage procedure for the severely arthrofibrotic knee. *Am J Sports Med* 1999;27:552–5651.
20. Klug S, Wittmann G, Weseloh G. Arthroscopic synovectomy of the knee joint in early cases of rheumatoid arthritis: follow-up results of a multicenter study. *J Arthrosc Relat Surg* 2000;16: 262–267.
21. Kanbe K, Inoue K. Efficacy of arthroscopic synovectomy for the effect attenuation cases of infliximab in rheumatoid arthritis. *Clin Rheumatol* 2006;25:877–881.
22. Carl H-D, Klug S, Seitz J, Swoboda B, Kinne R. Site-specific intraoperative efficacy of arthroscopic knee joint synovectomy in rheumatoid arthritis. *J Arthrosc Relat Surg* 2005;21: 1209–1218.
23. Kim SJ, Jung KA, Kwun JD, Kim JM. Arthroscopic synovectomy of the knee joint in rheumatoid arthritis: surgical steps for complete synovectomy. *J Arthrosc Relat Surg* 2006;22: 461.el–461.e4.
24. Roch-Bras F, Daures JP, Legouffe MC, Sany J, Combe B. Treatment of chronic knee synovitis with arthroscopic synovectomy: long-term results. *J Rheumatol* 2002;29:1171–1175.
25. Tyler WK, Vidal AF, Williams RJ, Healey JH. Pigmented villonodular synovitis. *J Am Acad Orthop Surg* 2006;14:376–385.
26. Araki Y, Tanaka H, Yamamoto H, Yamamoto T, Shino K, Nakamura H, Tsukagushi I. MR imaging of pigmented villonodular synovitis of the knee. *Radiat Med* 1994;12:11–15.
27. Cheng XG, You YH, Liu W, Zhao T, Qu H. MRI features of pigmented villonodular synovitis (PVNS). *Clin Rheumatol* 2004;23:31–34.
28. Dines JS., DeBerardino TM., Wells JL. Long-term follow-up of surgically treated localized pigmented villonodular synovitis of the knee. *J Arthrosc Relat Surg* 2007;23:930–937.
29. Ogilvie-Harris DJ, Saleh K. Generalized synovial chondromatosis of the knee: a comparison of removal of the loose bodies alone with arthroscopic synovectomy. *J Arthrosc Relat Surg* 1994;10: 166–170.
30. Massarotti EM. Lyme arthritis. *Med Clin N Am* 2002;86: 297–309.
31. Yoon KH, Bae DK, Kim HS, Song SJ. Arthroscopic synovectomy in haemophilic arthropathy of the knee. *Int Orthop* 2005;29:296–300.
32. Verma N, Valentino LA, Chawla A. Arthroscopic synovectomy in haemophilia: indications, technique and results. *Haemophilia* 2007;13:38–44.
33. Lubowitz JH, Rossi MJ, Baker BS, Guttmann D. Arthroscopic visualization of the posterior compartments of the knee. *J Arthrosc Relat Surg* 2004;20:675–680.
34. Kramer DE, Bahk MS, Cascio BM, Cosgarea AJ. Posterior knee arthroscopy: Anatomy, technique, application. *J Bone Joint Surg Am* 2006;88 (Supp 4): 110–121.
35. Ahn JH, Ha CW. Posterior trans-septal portal for arthroscopic surgery of the knee joint. *J Arthrosc Relat Surg* 2000;16:774–779.
36. Louisia S, Charrois O, Beaufils P. Posterior "back and forth" approach in arthroscopic surgery on the posterior knee compartments. *J Arthrosc Relat Surg* 2003;19:321–325.
37. Ewing JW. Plica: pathologic or not? *J Am Acad Orthop Surg* 1993;1:117–121.
38. Kang CN, Kim DW, Kim DJ, Kim SJ. Intra-articular ganglion cysts of the knee. *J Arthrosc Relat Surg* 1999;15:373–378.
39. Hirano K, Deguchi M, Kanamono T. Intra-articular synovial lipoma of the knee joint (located in the lateral recess): a case report and review of the literature. *The Knee* 2007;14:63–67.
40. Akgun I, Kesmezacar H, Ogut T, Dervisoglu S. Intra-articular hemangioma of the knee. *J Arthrosc Relat Surg* 2003;19:E17.
41. Mileti J, Mileti L, Kaeding C. Non-Hodgkin's lymphoma of the knee diagnosed by arthroscopy. *J Arthrosc Relat Surg* 2007;23:447.e1–447.e4.
42. Ozalay M, Tandogan RN, Akpinar S, Cesur N, Hersekli M, Ozkoc G, Uysal M. Arthroscopic treatment of solitary benign intra-articular lesions of the knee that cause mechanical symptoms. *J Arthrosc Relat Surg* 2005;21:12–18.
43. Ahn JH, Yoo JC, Lee SH, Lee YS. Arthroscopic cystectomy for popliteal cysts through the posteromedial cystic portal. *J Arthrosc Relat Surg* 2007;23:559.el–559.e4.
44. Steadman JR, Ramappa AJ, Maxwell RB, Briggs K. An arthrosopic treatment regimen for osteoarthritis of the knee. *J Arthrosc Relat Surg* 2007;23:948–955.

Arthroscopic Treatment of Knee Fractures

S.L. Mortimer and Robert Hunter

Introduction

Tibial plateau fractures can be a challenging injury to manage for orthopaedic surgeons. Management of these fractures has evolved from immobilization, skeletal traction, open reduction, and internal fixation to (all) arthroscopic reduction and internal fixation for some fracture types. Although not all fractures are amenable to arthroscopic reduction and fixation techniques, the arthroscope is still an important instrument for intraarticular diagnostic and treatment purposes. Arthroscopy provides potential advantages over other methods of treatment, by allowing for complete and accurate diagnosis of intraarticular pathology, earlier and better recovery of joint motion, less soft tissue dissection, and anatomic restoration of the joint surface.

Another proximal intraarticular fracture of importance is the tibial eminence fracture. This represents an avulsion injury of the insertion of the anterior cruciate ligament (ACL) at the tibia and is considered the equivalent of an ACL tear [1–2]. Poncet first described the tibial eminence fracture in 1875, and, since then, the treatment algorithm has changed significantly from nonoperative management to what is now considered state-of-the-art arthroscopic management [3]. This chapter will discuss in detail a current review of the anatomy, mechanism of injury, diagnosis, treatment, rehabilitation, and potential complications that can occur with tibial plateau and tibial eminence fractures.

Anatomy

The tibia is the primary weight-bearing bone of the knee joint. The most proximal aspect of the tibia is composed of the medial and lateral tibial condyles. The articular surfaces of the condyles are the medial and lateral tibial plateaus, which articulate with the corresponding medial and lateral femoral condyles. The plateaus are separated by the intercondylar eminence, which serves as the site of attachment for the anterior and posterior cruciate ligaments and the fibrocartilaginous menisci [4]. Specifically, the midpoint of the intercondylar eminence serves as the distal attachment of the ACL.

The medial tibial plateau is larger and has a concave shape [5]. The lateral tibial plateau is smaller and elevated in comparison with the medial side and has a convex shape. In addition to these anatomic differences, the medial articular surface and its supporting medial condyle are stronger than their lateral counterparts. These differences help to explain why fractures of the lateral tibial plateau are more common and why they are associated with a lower-energy mechanism of injury [5].

Mechanism of Injury

Tibial Plateau Fractures

Tibial plateau fractures occur as a result of a valgus or varus force, an axial compressive force, or a combination of these forces. Varus forces (medial force moment) are often referred to as “bumper fractures,” as they are frequently the result of pedestrian versus motor vehicle injuries. The femoral condyle applies a compressive and shearing force on its corresponding tibial plateau, resulting in a split fracture, a depression fracture, or both [5].

Tibial Eminence Fractures

The mechanism of injury for tibial eminence fractures is similar to that for an ACL tear; however, it involves an avulsion

S.L. Mortimer (✉)
Department of Orthopaedic Surgery, Arizona Institute for Sports Medicine University of Arizona Tucson Arizona

B.P. McKeon et al. (eds.), *Knee Arthroscopy*, DOI 10.1007/978-0-387-89504-8_11,

fracture at the ACL insertion. The injury may be associated with valgus and external rotation. It is often seen in skiers and is related to either a boot-induced injury after the skier lands on the tail of the ski or to the phenomenon referred to as a "phantom foot" injury, which involves forced internal rotation with knee flexion. Although seen frequently in skiers, it is also seen in other sports, bicycle accidents, motor vehicle accidents, and pedestrian versus motor vehicle injuries.

Tibial eminence fractures are seen in children usually between the ages of 8 and 15 years [3, 6, 7]. Although this fracture pattern is commonly associated with a childhood injury, it is also seen in adults [2, 8, 9]. It is theorized that this occurs more commonly in children because of the relative weakness of the incompletely ossified tibial eminence compared with the fibers of the ACL. It also has been proposed that the injury occurs secondary to greater elasticity of the ligaments in young people [10].

Indications

Tibial Plateau Fractures

A patient with a tibial plateau fracture generally presents with acute knee pain, inability to weight-bear, a tense knee effusion, joint-line tenderness, and a history of a traumatic event. Although patients usually are not able to accurately describe the precise mechanism of injury, a good history does provide insight to the degree of force, which helps to identify a high-energy versus low-energy mechanism of injury. This is a crucial aspect of the initial evaluation because high-energy injuries are associated with other injuries and may require more urgent treatment.

A complete evaluation of the extremity should be performed beginning with inspection of the skin and grading of the soft tissue damage. Palpation of the joint line and origins and insertions of the collateral ligaments should be performed. Range of motion, both active and passive, will be limited as a result of pain and swelling. Although pain and voluntary and involuntary muscle guarding may cause difficulty in examination of ligamentous stability, an effort to examine the stability of the collateral ligaments and cruciate ligaments is warranted. Popliteal, dorsalis pedis, and posterior tibial pulses must be palpated and, if absent, Doppler examination performed. Any suspicion of an associated knee dislocation and vessel injury necessitates angiogram and vascular surgery consultation. Doppler ultrasound should never be used as an alternative to angiography, as it does not reliably predict intimal arterial injury [11]. Assessment of neurologic status should also be performed with specific evaluation of the peroneal and tibial nerve function.

The most commonly used and accepted classification system is that described by Schatzker. This classification scheme divides tibial plateau fractures into six types based on fracture pattern and fragment anatomy (Fig. 1). Injury patterns, severity of injury, and prognosis are defined by these six categories, which reflect increased energy expenditure and a worse prognosis as the number increases [5].

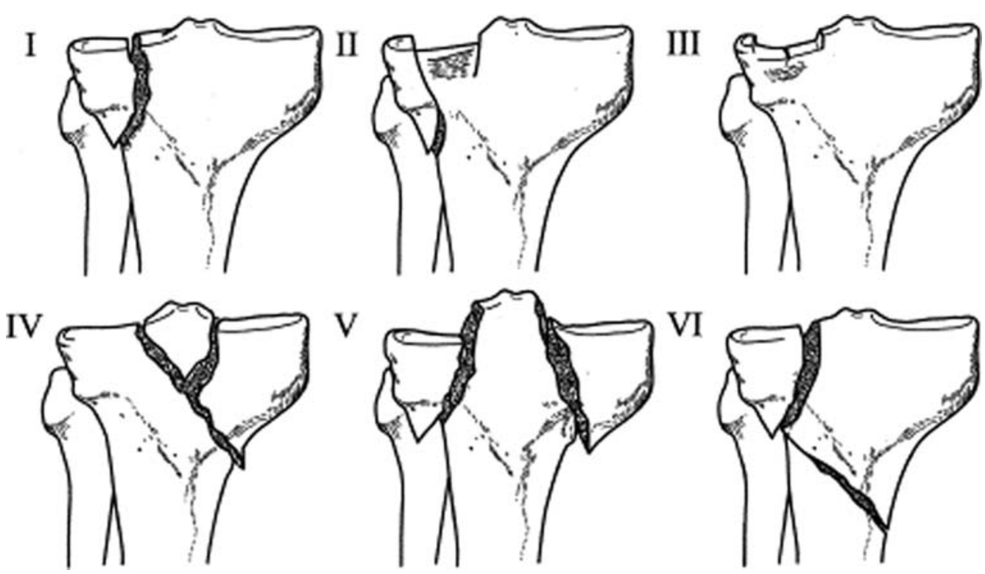


Fig. 1 The Schatzker classification of tibial plateau fractures. (From Lubowitz JH, Elson WS, Guttmann D. Part I: arthroscopic management of tibial plateau fractures. *Arthroscopy* 2004;20:1063–1070. Copyright © 2004 Arthroscopy Association of North American. Reprinted with permission of Elsevier.)

Type I is a split fracture of the lateral tibial plateau, which occurs secondary to a valgus and axial force. There is no compression (depression) of the fragment(s), and this is usually seen in young adults in whom the cancellous bone is strong. If the fragment is displaced, it is commonly associated with a meniscal injury, which can be entrapped in the split blocking reduction.

Type II is a split fracture associated with compression (depression) of the lateral tibial plateau. This fracture pattern has a similar mechanism of injury to a type I injury, a lateral bending force with axial loading, but generally is seen in patients with bone unable to resist compression secondary to osteoporosis or increased forces.

Type III is a pure compression fracture of the lateral tibial plateau. The mechanism of injury is usually an axial force resulting in a central or lateral depression; however, the depression may involve any portion of the plateau. Although not associated with a split, there is frequently a plastic deformation of the plateau, resulting in widening of the tibia.

Type IV is a fracture involving the medial tibial plateau. This fracture pattern may be a result of a varus or axial compression force and thus may be a split or a split combined with a depression. In comparison with the lateral plateau, the medial plateau is stronger and tends to resist fracture formation. Therefore, these injuries are associated with a higher mechanism of injury and greater force.

Medial tibial plateau fractures also are frequently associated with an avulsion of the intercondylar eminence, which may indicate rupture of the cruciate ligaments. The varus force also commonly results in rupture of the lateral collateral ligamentous complex. With increasing energy, subluxation or dislocation of the knee may be associated with an injury to the popliteal artery or peroneal nerve. Therefore, caution and careful examination of the patient must be used when a type IV injury presents, as it may represent a dislocation of the knee that spontaneously reduced prior to radiographic examination.

Type V fractures involve split elements of both the medial and lateral femoral condyles and may involve varying degrees of depression on either the medial or lateral articular surface. The mechanism of injury usually involves a pure axial load applied to an extended knee.

Type VI is a fracture defined as a bicondylar fracture combined with a metaphyseal fracture that separates the condylar components from the diaphysis. This fracture pattern is associated with a high-energy mechanism of injury resulting in depression and impaction of fracture fragments and severe soft tissue injury with potential neurovascular compromise and compartment syndrome.

Associated injuries in tibial plateau fractures are common and include injuries to the menisci, collateral and cruciate ligaments, arteries, and nerves. Meniscal injuries may be seen as peripheral, flap, or radial tears, and cruciate ligament injuries may occur as avulsion, attenuation, or midsubstance injuries [11]. It has been suggested that up to 47% of knees with closed tibial plateau fractures have associated injuries of the menisci that require surgical intervention [12]. Fracture pattern does not correlate with meniscal injury, which makes diagnosis difficult without the aid of arthroscopy or magnetic resonance imaging (MRI) [13]. Recognizing the association of other injuries with tibial plateau fractures is important, as these injuries usually require operative intervention.

Imaging studies are crucial for classifying the type of injury and for preoperative planning. Radiographs must include anteroposterior, lateral, and oblique views. The fracture pattern, degree of comminution, and articular depression are often underappreciated with plain films [5]. In addition to these shortcomings, the true incidence of concomitant soft tissue injury has only recently begun to be understood, emphasizing the need for additional imaging modalities to better classify the extent of bony injury as well as soft tissue pathology.

MRI has become the imaging modality of choice because of the ability to evaluate the soft tissues and osseous structures. It has been shown to be highly sensitive at evaluating soft tissue injuries, and it also is accurate in depicting fracture comminution, depression, and displacement in patients with tibial plateau fractures [14–17]. A recent large series of operative tibial plateau fractures, using MRI as the imaging modality, found that 99% of patients had additional soft tissue injury [15]. Although there is debate regarding the treatment of these concomitant injuries, this study also found that MRI changed the treatment plan in 23% of 52 patients studied with tibial plateau fractures compared with the operative plan based on conventional radiographs and computed tomography (CT) imaging [15]. The ability to use MRI to reliably predict soft tissue and bony injury has made it the examination of choice for evaluation of tibial plateau fractures.

Although MRI provides optimal visualization of osseous and soft tissue structures, CT imaging can be helpful in a complete understanding of the osseous components. Axial combined with coronal plane reconstructions provide precise interpretation of articular as well as extraarticular components of the fracture pattern [11]. Despite the excellent osseous interpretation, CT has limited ability to adequately visualize soft tissue structures, which can lead to incomplete diagnosis and inadequate management of soft tissue injuries [4, 18].

In summary, MRI is an excellent preoperative planning tool and is the imaging modality of choice for soft tissue and bony injury involving the tibial plateau. CT imaging provides excellent imaging for bony injury and has some application in the visualization of soft tissue injuries as well. It cannot be used to predict meniscal pathology, and this must be realized during preoperative planning.

Tibial Eminence Fractures

Similar to patients with other fractures involving the knee joint, patients presenting with fractures of the tibial eminence present with a painful swollen knee and have difficulty bearing weight. Initial examination is often difficult secondary to pain and may limit evaluation of the ligaments. A complete neurologic and vascular examination must be performed.

Plain radiographs are usually diagnostic and involve anteroposterior, lateral, and oblique views. CT scanning may be used to better define bony architecture, and MRI is useful for determining additional injuries to chondral surfaces, menisci, and ligaments. Arteriography and vascular surgery consultation must be considered in the presence of diminished pulses or abnormal vascular examination.

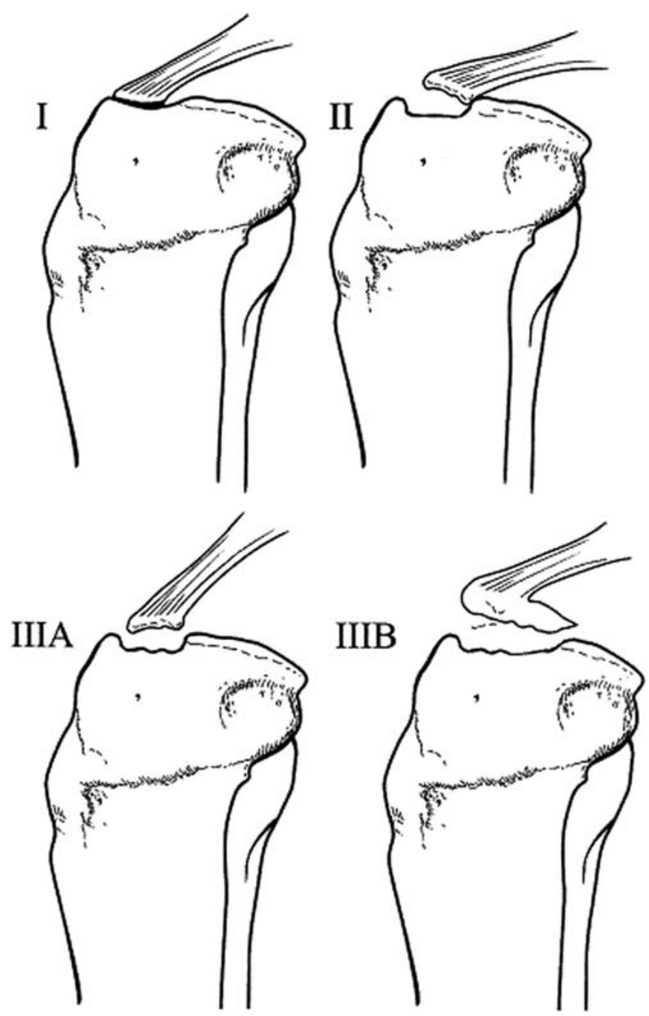


Fig. 2 The Meyers and McKeever classification of tibial intercondylar eminence fractures. (From Lubowitz JH, Elson WS, Guttman D. Part II: arthroscopic treatment of tibial plateau fractures: intercondylar eminence avulsion fractures. *Arthroscopy* 2005;21:86–92. Copyright © 2005 Arthroscopy Association of North America. Reprinted with permission of Elsevier.)

Meyers and McKeever first described the classification scheme for tibial eminence fractures in 1959 [2]. Their classification divides these fractures into three types based on displacement of the avulsed fracture fragment (Fig. 2). Type I represents a nondisplaced or minimally displaced fracture at the anterior margin. Type II fractures involve the anterior third or half of the avulsed bone displaced proximally with an intact posterior hinge, resembling a bird's beak. Type III fractures have a completely displaced fracture. These have been further subdivided into IIIA and IIIB fracture classifications [19]. Type IIIA fractures involve the ACL insertion only, whereas the IIIB includes the entire intercondylar eminence. Some have labeled comminuted fractures as type IV [19].

Associated injuries with fractures of the tibial intercondylar eminence are common. Meniscus injuries are the most common injuries seen; however, these fractures may be associated with chondral and ligamentous injuries as well [20, 21]. In addition to intraarticular pathology, tibial eminence fractures also are seen with tibial plateau fractures, specifically type V and VI fractures [5].

Treatment

Tibial Plateau Fractures

The goal for treatment of tibial plateau fractures is restoration of joint function and prevention of posttraumatic osteoarthritis. This is accomplished by reestablishing alignment, joint surface congruity, stability, and mobility. Management is dictated by the condition of the soft tissue envelope, amount of articular depression, comminution, and degree of diaphyseal-metaphyseal comminution.

Careful assessment of the soft tissues is crucial when first examining the patient. The compartments must be evaluated for compartment syndrome, and any neurovascular deficit must be identified immediately. Recognizing the presence of an open or closed fracture also is important when trying to determine a treatment plan.

Although anatomic reduction of the articular surface is thought to prevent posttraumatic arthritis, there has not been a general consensus regarding the maximal acceptable depression. Brown et al., using a cadaveric model, found significant departures from anatomic pressure occurring with fragment step-off of more than 1.5 mm [22]. Bai et al., using cadavers and a dynamic pressure sensor, found increased average and maximum contact pressures and decreased contact areas with progressively greater step-off heights. Their maximum step-off of 6 mm increased contact pressures 154% with an intact meniscus and 254% with a meniscectomy specimen. The contact areas were decreased by 26%

with the intact specimen and 39% with the meniscectomy specimen [23]. An increase in local pressures secondary to articular depression is thought to result in proteoglycan depletion, fibrillation, and late osteoarthritic changes [22]. Excessive joint contact pressures have been shown to result in a high incidence of joint degeneration [24]. In addition to the laboratory measurements, clinical evidence also has revealed that excessive pressures on the cartilage have a direct relationship to degenerative changes in the knee, and any articular depression or axis deviation bears the risk of posttraumatic arthrosis [25]. Therefore, fractures that demonstrate articular displacement, instability, and deviation in the mechanical axis may result in posttraumatic arthrosis and should be evaluated for surgical management.

If nonoperative management is pursued, close follow-up of the patient is required with radiographs every 2 weeks for the first 4–6 weeks to monitor depression, displacement, and axis deviation [11]. Early range of motion is instituted, and a hinged knee brace is used to control motion [26–28]. Weight-bearing is prohibited, and non–weight-bearing status is continued until fracture consolidation is present. After 6–8 weeks, the patient is generally moved to a partial weight-bearing status, and by 12 weeks full weight-bearing is allowed if the fracture is healed.

Although open reduction and internal fixation has been the mainstay for treatment of displaced or compressed fractures, arthroscopic reduction and internal fixation is a viable and attractive alternative as it may reduce morbidity associated with surgical management [29, 30]. Arthroscopic management of these fractures allows for a minimally invasive technique and a direct and accurate fracture reduction that does not require an extensive exposure [31, 32]. The advent of arthroscopy has provided a bridge to the gap that exists between the extremes of open versus nonoperative management of tibial plateau fractures [11].

There are other advantages of arthroscopy, which make this technique attractive for certain fracture patterns. It allows for a thorough lavage of the joint and evacuation of the hemarthrosis and fracture debris [33]. The fracture and entire joint surface can be visualized, and the remainder of the intraarticular structures also can be directly visualized and treated. It has been shown that arthroscopic management of meniscal and ligamentous injuries is superior to reconstruction or repair using open procedures [31, 33]. The avoidance of an arthrotomy and meniscal detachment requiring open repair is another advantage with the arthroscopic technique. The less invasive nature of arthroscopic management of tibial plateau fractures may allow for a more rapid recovery, less pain, improved fracture and soft tissue healing, early full range of motion, and a more functional recovery [13, 31, 32].

There are also disadvantages with arthroscopic treatment of tibial plateau fractures. The technique required is technically demanding and may be difficult if not employed by a surgeon who routinely uses arthroscopy. Bleeding can be a challenge, but use of a fluid pump assists in controlling this problem. Caution should be used, and the pump should be kept on a low-pressure setting to avoid extravasation of fluid. Development of compartment syndrome is possible, and the compartments should be carefully monitored for tightness and extravasation of fluid.

Tibial plateau fractures with split, split depression, or pure depression (types I–IV) are the fracture patterns most amenable to arthroscopic reduction and internal fixation. This is generally combined with percutaneous screws, buttress screws, and/or percutaneous versus open plating. More complex injury patterns associated with higher energy (types V and VI) may not be amenable to complete arthroscopic management; however, arthroscopy still may be used as an aid in diagnosis and an adjunct for treatment of associated intraarticular pathology [31].

Tibial Eminence Fractures

The goal for management of tibial eminence fractures should be no different than for any other intraarticular fracture. Anatomic reduction and rigid fixation that allows for early range of motion should be the treatment for these fractures. Debate has ensued over anatomic reduction versus overreduction. It has been proposed that overreduction may result in excessive tension of the ACL, which results in limited knee range of motion [34]. Others have countered this by stating that plastic deformation of the ACL occurs prior to the avulsion fracture and thus overreduction would result in a better outcome [10]. Numerous studies have documented residual laxity in well-reduced tibial eminence fractures, and most conclude that the laxity is not symptomatic [35–37]. More studies are needed to answer the question of anatomic versus overreduction; however, there is consensus that any displacement requires at least an anatomic reduction.

Management has been based on the Meyers and McKeever classification with recommendations for immobilization in extension for type I fractures [2]. Some controversy exists as to what degree the knee is to be extended for nonoperative management. Meyers and McKeever recommend immobilization in 20 degrees of flexion [2, 9]. Similarly, Beaty and Kumar recommend immobilization in 10–15 degrees of flexion [38]. Fyfe and Jackson based their recommendations of flexing the knee to 30–40 degrees because the ACL is taut in extension and with some flexion the tension on the avulsion fragment would be less [39]. These authors favor immobilization in full extension to avoid a flexion contracture, which can occur if the knee is kept in a flexed position. We encourage straight leg raises and quadriceps isometrics and allow full weight-bearing as tolerated in a brace locked in full extension. The knee should not be immobilized in hyperextension, as extensive stretch on the popliteal artery may result in a compartment syndrome [5]. Regardless of the position of immobilization, close follow-up with radiographs weekly for 4 weeks should be taken to confirm maintenance of reduction.

Treatment of type II fractures has been controversial. Closed reduction may be attempted by aspiration of the hemarthrosis and knee extension performed to allow the femoral condyles to help reduce the fracture [11]. Anteroposterior and lateral radiographs should be taken to verify reduction, and, with difficult visualization, CT or MRI should be performed. Oftentimes, anatomic reduction is not achieved secondary to interposition of the medial meniscus, lateral meniscus, or the intermeniscal ligament. Persistent displacement, despite attempted reduction maneuvers, warrants arthroscopic evaluation and treatment. Many reports identify associated soft tissue injuries, including chondral, meniscal, and ligamentous structures. The need for anatomic reduction of these fractures to allow for postoperative stability and motion combined with the need to identify and treat these associated injuries make arthroscopic evaluation necessary for successful treatment of type II and III fractures [36, 40–43]. These authors also believed that most, if not all, type II fractures were likely a type III level of displacement at the time of disruption. Based on that, the authors take an aggressive operative approach to the majority of type II injuries.

Closed reduction may be attempted for type III injuries; however, anatomic reduction and maintenance of reduction is difficult. Most experts agree that type III fractures require reduction and fixation [5, 44, 45]. Arthroscopic reduction and fixation of these injuries have become the standard of care and have made open reduction and internal fixation a treatment infrequently necessary or used.

Techniques involving use of cannulated screws or suture have been described, and the results with either method have been excellent. Risks of cannulated screw placement involve comminuting the fracture fragment, hardware removal, and posterior neurovascular injury. Repair using nonabsorbable suture fixation provides the benefit of eliminating these risks and still maintaining an excellent reduction and result.

Technique

Tibial Plateau Fractures

General anesthesia is used in most cases. Examination under anesthesia is routine to help identify ligamentous injuries. The leg is secured in an arthroscopic leg holder, and the end of the bed is flexed. The contralateral leg is supported with a foam pad and abducted to the side to allow fluoroscopy in the anteroposterior and lateral projections. A tourniquet is placed around the leg, but it is not routinely used. Standard anterolateral and anteromedial portals are used. The anteromedial portal is often used to view lateral fractures. An accessory lateral portal made at the level of the joint line and lateral to the standard anterolateral portal is often made to assist with meniscal retraction with a meniscal hook and to provide better visualization of anterior plateau fractures (Fig. 3).

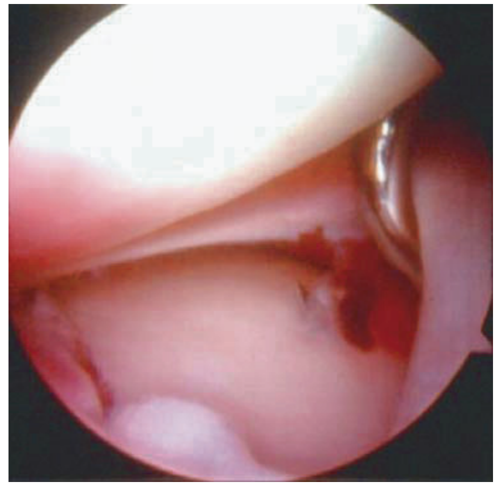


Fig. 3 A meniscal hook may be used through the accessory lateral portal to retract the meniscus for better visualization

The knee must first be thoroughly lavaged and the hemarthrosis and loose bodies evacuated. Careful attention must be directed to fluid extravasation, and compartments should be routinely palpated to assess pressure. In cases of suspected increased compartment pressure, pressure measurements are recommended and fasciotomy is required should compartment syndrome occur. This is particularly important in split fractures where fluid extravasation may occur through the fracture line. Once the hemarthrosis is evacuated, debridement of the ligamentum mucosum, the intermeniscal ligament, and fat pad is performed as needed to fully visualize the entire fracture.

See Fig. 4 for the specific instruments needed to aid in reduction and fixation of tibial plateau fractures.

Split fractures with no depression (type I fractures) are reduced first with a reduction forceps. If the apex of the split is displaced, a small incision can be made to allow for anatomic reduction of the distal fracture spike while the forceps are used to close the fracture at the level of the joint. Arthroscopy is used to verify reduction and for visualization of the joint and other associated pathology. Fluoroscopy is used to aid in reduction as well as to verify adequate placement of wires for provisional reduction. Two percutaneous cannulated screws, used with washers, may be placed over

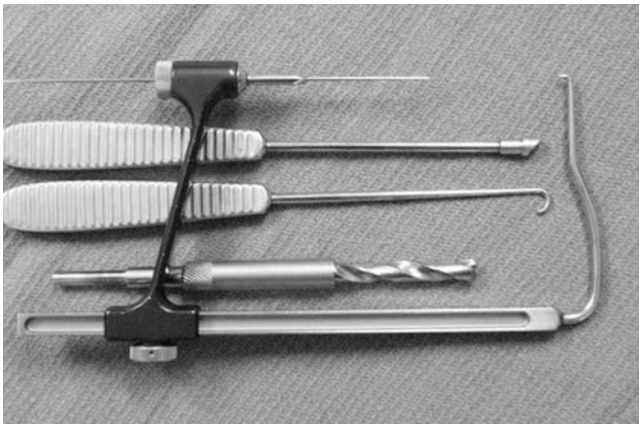


Fig. 4 Specific instruments are needed to aid in reduction and fixation of tibial plateau fractures

the wires. If residual fragment instability is suspected, a buttress screw may be placed at the inferior apex of the split. In cases with comminution, fragment instability or poor bone quality requires a buttress plate with or without additional compression screws, which may be placed percutaneously or with a standard extraarticular incision.

Type II, III, and IV fractures require reduction of all depressed elements first. Under arthroscopic guidance, a modified ACL guide is used to place a 2.4-mm drill-tipped guide pin in the center of the depressed fragment starting on the anterolateral or anteromedial tibial cortex. With the guide pin properly placed, an incision is made at the pin entrance and enlarged enough to allow for a cannulated 9.0-mm drill to pass (Fig. 5). The 9.0-mm drill is used to

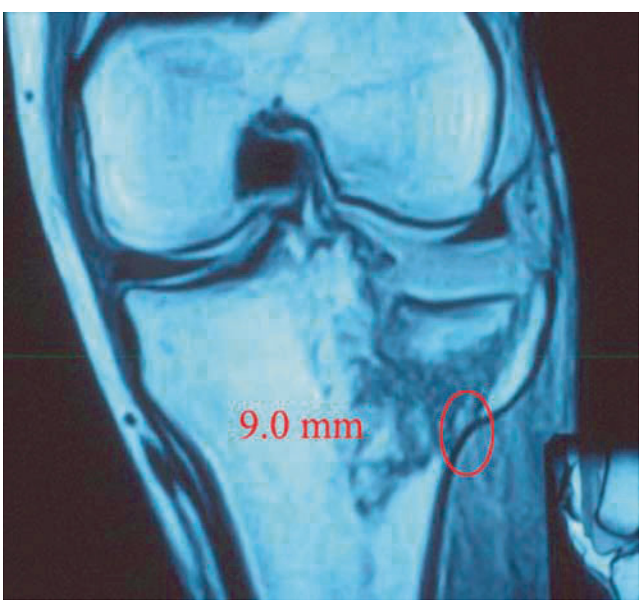


Fig. 5 A 9.0-mm drill bit is used to penetrate the cortex at the base (circled) of the split depression and is used to cut a hole without violating the joint surface

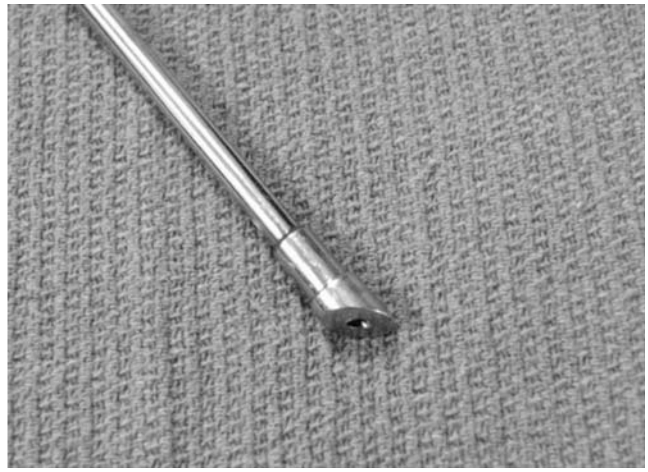


Fig. 6 A cannulated tamp is used to elevate the fracture site

penetrate the cortex, taking care to just cut a cortical hole without violating the joint surface. A cannulated tamp is used to elevate the fracture site under arthroscopic visualization (Fig. 6). It is imperative that this is performed gently and with a slight overcorrection. Adequate reduction is verified by probing the fracture and the metaphyseal defect created by the elevation, and reduction of the fracture fragment is grafted (Figs. 7 and 8). Graft options include autograft from the comminuted metaphysis, allograft freeze-dried croutons, demineralized bone matrix, calcium triphosphate, and other bone substitutes. The graft material is packed gently and is used to fill the defect. Fixation is then performed under arthroscopic and fluoroscopic guidance. In type III fracture patterns, cannulated screws with washers are placed directly under the subchondral plate to act as a buttress for the elevated fragments (Figs. 9 and 10). For types I, II, and IV

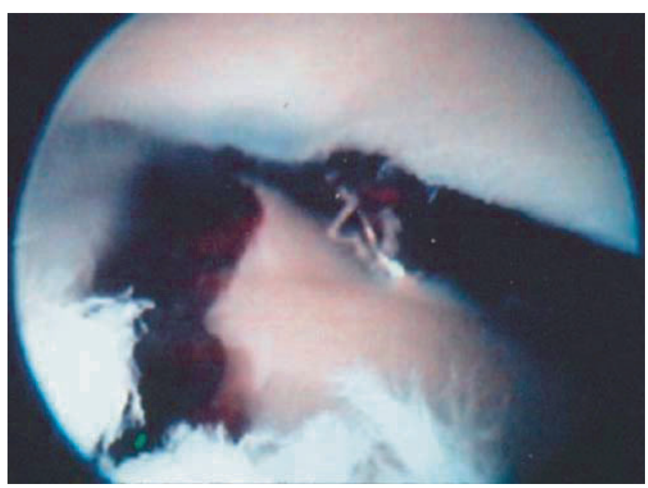


Fig. 7 The fracture is visualized with the arthroscope with significant displacement and depression being appreciated

Fig. 8 The fracture is reduced with anatomic realignment of the articular surface being visualized

Fig. 9 Anteroposterior radiograph revealing subchondral placement of cannulated screws and bone graft placement

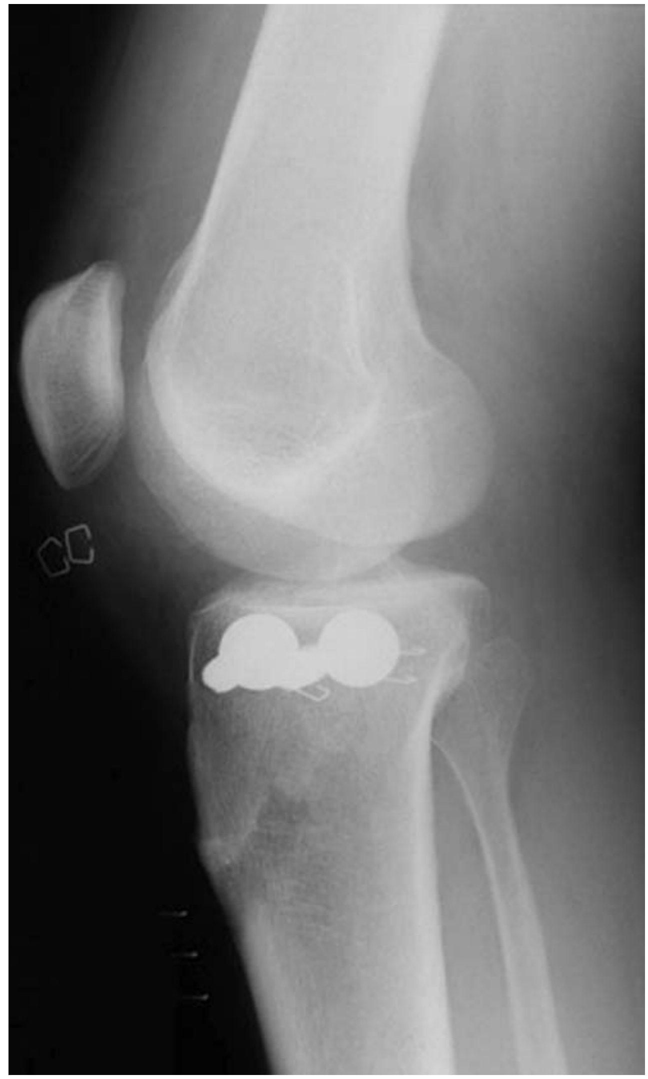


Fig. 10 Lateral radiograph revealing subchondral placement of cannulated screws and bone graft placement

fracture patterns, similar cannulated screw techniques will buttress the compression, but a buttress screw placed at the inferior apex or a buttress plate may be needed for additional stability to prevent subsidence or distal migration of the fracture fragment. In types II and III fractures that demonstrate plateau widening, the first cannulated screw can be used to close this widening by allowing the screw head and washer to compress the lateral cortex as the screw threads engage the medial cortex. This will result in a screw that is too long and prominent medially. To avoid postoperative pain, the screw should be replaced with a shorter screw that just engages the medial side of the bone.

Tibial Eminence Fractures

General anesthesia or epidural anesthesia may be used. The leg is secured in an arthroscopic leg holder, and the foot of the bed is flexed. The contralateral leg is supported with a foam pad and abducted to the side to allow fluoroscopy of the involve extremity in both anteroposterior and lateral projections. A tourniquet is placed around the thigh, but it is not routinely used. After prepping and draping, an anterolateral portal is established. A fluid pump is used to promote hemostasis and adequate visualization. The hematoma is evacuated until good visualization is possible. Once pathology can be visualized, an anteromedial portal is established after localization with a spinal needle. An arthroscopic probe is then used to dislodge any clotted blood or debris at the site of fracture (Fig. 11).

A synovial resector (4.5 mm) is used to further debride the region and to remove any debris from the fracture bed. Once the fracture site has been debrided, the probe is used to attempt a reduction. Interposition of the intermeniscal ligament or the menisci requires use of the probe to hold the soft tissue structures out of the way while attempting to reduce the fragment with an ACL guide or probe. In cases in which the intermeniscal ligament prevents reduction and also cannot be mobilized, resection is performed. Once the fracture has been reduced, a 0.062-inch Steinmann pin is placed percutaneously from a medial parapatellar position to hold the fracture reduced (Fig. 12).

If there is a large fragment and the piece is large enough to consider placing a cannulated screw, fixation is achieved by

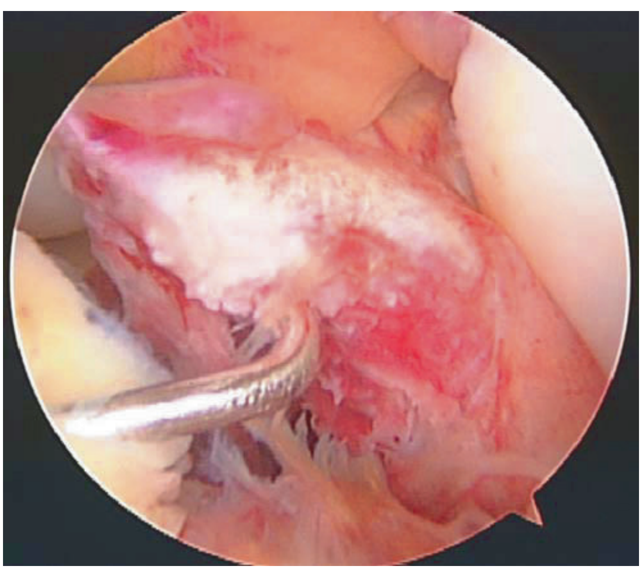


Fig. 11 A probe is used to hold the fracture site open to debride clot, intermeniscal ligament, or other debris

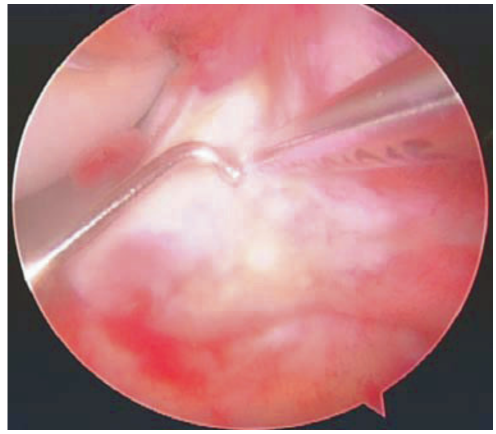


Fig. 12 A probe and Steinmann pin are used to reduce the fracture anatomically

using one or two 4.0-mm cannulated screws (Synthes USA, Paoli, PA). If the Steinmann pin that is holding the reduction is in good position, it may be used as the guide pin for the cannulated screw. If not, a second wire may be placed under fluoroscopic control. The goal is to have the pin(s) just penetrate the posterior cortex of the tibia. Frequent use of fluoroscopy is recommended to ensure accurate placement of the wire and screw and also to make sure the wire is not being advanced as the screw is placed.

Suture fixation should be used when the fracture is small, comminuted, or in the presence of open growth plates. Some have advocated using the suture methods for all cases as less risk (neurovascular bundle) and less secondary procedures (hardware removal) are seen [44, 45]. After the fracture is reduced and held in position with the Steinmann pin, the ACL tibial tunnel guide is used to pass a 2.4-mm wire through the anteromedial tibial metaphysis and through the

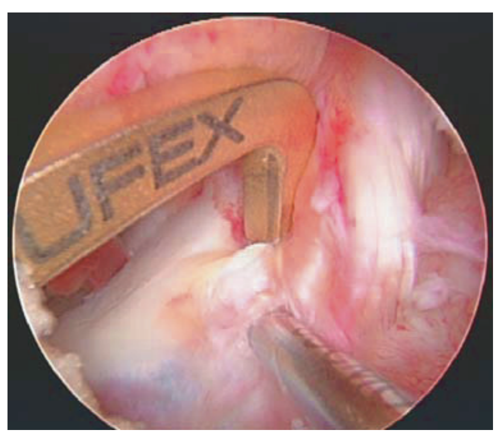


Fig. 13 The Acufex ACL tibial tunnel guide is used to pass a 2.4-mm wire on the medial side of the fragment. (Courtesy of Smith & Nephew Endoscopy, Andover, MA.)

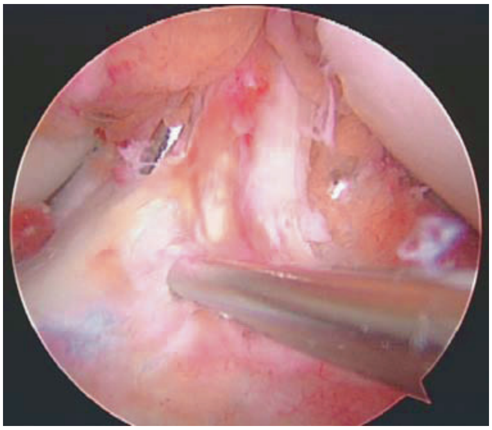


Fig. 14 A second wire is passed on the lateral side of the ACL

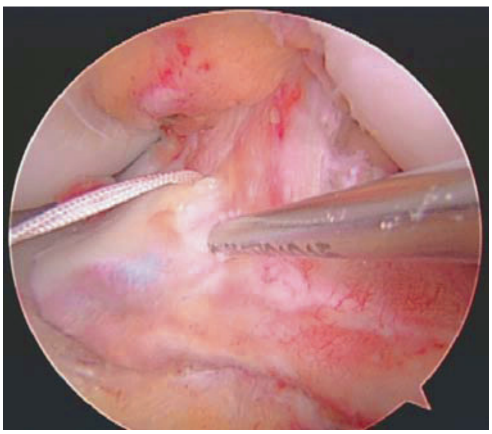


Fig. 15 The medial 2.4-mm wire is withdrawn, and a suture passer is placed in the hole and two Ultrabraid sutures are passed. (Courtesy of Smith & Nephew Endoscopy, Andover, MA.)

medial side of the fragment (Fig. 13). A second wire is passed starting 1–2 cm lateral to the first hole on the tibial cortex, entering the knee at the lateral side of the fragments (Fig. 14). The first wire is removed, and, immediately after removal, a suture passer is passed up the hole and two Ultrabraid sutures (Smith & Nephew Endoscopy, Andover, MA) are delivered into its loop through the anteromedial portal and pulled out the anteromedial tibia (Fig. 15). The second wire is taken out, and the suture passer is immediately placed into the joint (Fig. 16). The opposite end of the Ultrabraid suture(s) are delivered into its loop and pulled out the tibia (Fig. 17). A crochet hook or a blunt probe is passed into the subcutaneous tissue through one of the suture holes, hooking the opposite sutures, and pulling them out the same hole. A knot is tied and passed through the skin puncture hole and subcutaneous tissue and is secured tightly to the tibial cortex. Each suture is tied and secured individually (Fig. 18). This provides firm fixation of the fracture fragment and can be directly visualized.

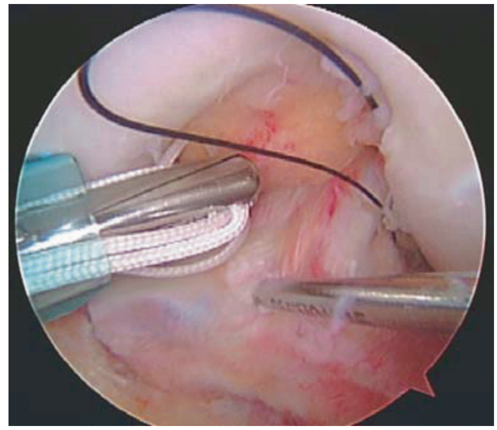


Fig. 16 The lateral 2.4-mm wire is removed, and the suture passer is passed, and the other ends of the Ultrabraid are brought out of the tibial cortex. (Courtesy of Smith & Nephew Endoscopy, Andover, MA.)

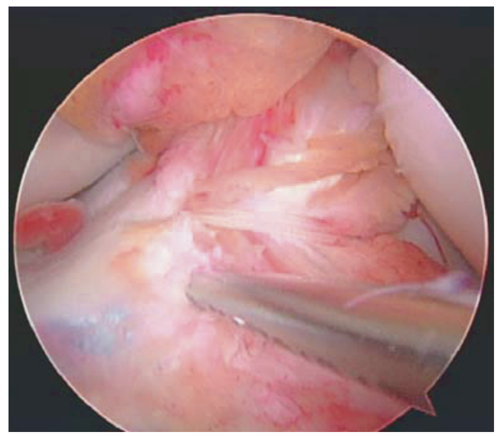


Fig. 17 The Ultrabraid suture has been passed through both tibial holes and now holds the ACL and its fracture fragment reduced. (Courtesy of Smith & Nephew Endoscopy, Andover, MA.)

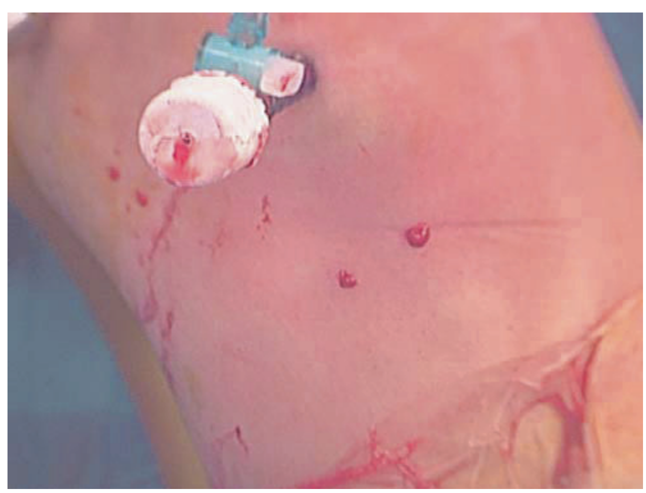


Fig. 18 The sutures are tied through one of the wire holes leaving no incision along the tibia

Rehabilitation

Tibial Plateau Fractures

Postoperative management initially focuses on return of range of motion. Continuous passive motion may be used postoperatively if the soft tissue envelope is not severely compromised. This may need to be delayed for a few days to allow wound healing to occur and swelling to subside. The continuous passive motion device may be discontinued after a week as a patient's swelling and pain is improved, and he or she may continue with active or gentle active assist motion. Early range of motion is encouraged with a goal of 0–90 degrees to be achieved by the first week. Full range of motion should be obtained by postoperative week 6. Active and active-assist range of motion may begin once wound healing has occurred. Use of a hinged knee brace is used for 6 weeks in cases of associated collateral ligament injury or in environments where an unexpected fall might occur.

Physical therapy should be initiated immediately for crutch weaning, gait training, range of motion, and maintenance of muscle strength. Strict non-weight-bearing (NWB) is maintained for the initial 6–8 weeks. Time for NWB is dependent on the fracture pattern and the patient's bone quality and needs to be maintained to prevent subsidence of the articular surface [46]. Once the 6- to 8-week mark is reached and there is radiographic evidence of articular consolidation, partial weight-bearing with 50% body weight may be initiated. For most fracture patterns, patients may begin full weight-bearing by 12–14 weeks. Throughout the postoperative course, quadriceps and hamstring strengthening is emphasized and advanced based on activity demands [5]. It has been shown that residual quadriceps strength loss directly correlates with decreased functional results; thus, postoperative protocols should be directed at the quadriceps mechanism [47].

Tibial Eminence Fractures

Patients are placed in a hinged knee brace locked in 0 degrees of flexion for the first 4 weeks and allowed to perform passive or active-assist range of motion exercises in the prone position through an arc of 0–90 degrees. Patients may weight-bear as tolerated, with the brace locked at 0 degrees. Crutches are generally discontinued by postoperative day 10. At 4 weeks, the brace is removed, and closed-chain quadriceps exercises are begun. At 8 weeks, easy straight-ahead running is initiated and pivot-twist maneuvers are avoided until at least 12 weeks after surgery.

Complications

Tibial Plateau Fractures

The results of treatment of tibial plateau fractures have improved as concepts of preoperative planning have evolved, less invasive surgical techniques have been adopted, and methods of exposure and implant placement have become less traumatic. Despite these improvements, the ability to recognize potential and existing complications is important and must be realized.

With arthroscopic-assisted fracture fixation, it is possible to have an iatrogenic compartment syndrome develop due to fluid extravasation. Continuous monitoring of compartment tightness must be routine. Measurement of pressures must be done if suspicion of compartment syndrome is present and fasciotomy performed if pressures are elevated. This may be avoided by keeping inflow pressure low and by performing the reduction maneuver in a dry field. It also has been suggested that an incision may be made, before arthroscopy is begun, where placement of the fixation device is planned so the fluid will leak out through the wound instead of into the compartment [34].

Incisions made before soft tissue swelling has diminished may result in postoperative wound slough and infection. The risk of wound complications can be significantly reduced with careful examination of the soft tissues, delaying surgery as needed, and careful preoperative planning. Employing techniques that limit large skin flaps, minimize soft tissue stripping, and use indirect reduction methods help to decrease surgical trauma to the soft tissue envelope [5].

Nonunion can occur and is more common in higher-energy fractures that involve the metaphysis and diaphysis [48]. This complication is extremely rare in cases treated with an arthroscopic approach. Once identified, the nonunion site should be bone grafted, and fixation may need to be revised. Recognition of a loss of articular surface reduction necessitates immediate revision because delayed revision is difficult and may not result in a good outcome. Total knee arthroplasty is a reasonable salvage procedure in elderly patients with loss of articular reduction. Mechanical axis deviation may occur and, when identified, may be addressed by an osteotomy to restore the normal mechanical axis [5].

Arthrofibrosis may occur if early range of motion is not instituted postoperatively. Use of a continuous passive range of motion device may aid in preventing loss of motion. If patients have not reached 90 degrees of flexion within the first 4–6 weeks postoperatively, manipulation under anesthesia combined with arthroscopic lavage and lysis of adhesions should be performed.

Deep venous thrombosis and pulmonary embolism may occur with fractures occurring around the knee. Preoperative, perioperative, and postoperative use of mechanical prophylaxis should be routine. Postoperative chemical prophylaxis should also be used. Although swelling is expected, concern for deep venous thrombosis warrants surveillance with duplex ultrasound or other venous studying methods.

Tibial Eminence Fractures

Although a good outcome is usually expected for fractures of the tibial eminence, complications do occur. Residual laxity after fixation is commonly found after arthroscopic reduction and fixation. Although multiple studies have reported results verifying positive Lachman tests and a difference in laxity from the contralateral uninjured extremity, the majority of patients have functional stability and are not adversely affected [8, 35–37, 49]. Evidence of clinical instability warrants revision with ACL reconstruction.

Arthrofibrosis is a potential complication, but it is rare if the patient undergoes arthroscopic reduction with fixation, because the goal of the operation is to promote early range of motion. Development of arthrofibrosis warrants aggressive therapy and possible manipulation under anesthesia with arthroscopic lavage and debridement of adhesions. Loss of full extension can be avoided by immobilization in full extension and attention to quadriceps and hamstring strengthening.

Painful or symptomatic hardware is common with use of cannulated screws. Loss of full knee extension can occur secondary to scar tissue or a prominent screw in the intercondylar notch. It has been demonstrated that at the time of hardware removal, soft tissue interposition is the rule and that excision combined with implant removal results in excellent outcomes [50].

Clinical Pearls and Summary

Tibial Plateau Fractures

Arthroscopy has many applications in the treatment of tibial plateau fractures. It serves as the best method of identifying and treating intraarticular pathology. In addition, arthroscopic reduction and internal fixation of certain types of tibial plateau fractures allows for anatomic reduction of the joint surface and rigid fixation with less morbidity and better visualization. Arthroscopic reduction and internal fixation may be used for types I–IV tibial plateau fractures and, given the right clinical scenario, may be superior to open reduction and internal fixation.

Tibial Eminence Fractures

Arthroscopy is a safe and preferable alternative to closed management of types II and III tibial eminence fractures. Arthroscopic exam, reduction, and fixation can be accomplished in virtually all patients. In addition, this technique provides superior reduction and fixation when compared with that of closed or open methods. Nearly all patients return to sports at their previous level when treated with arthroscopic reduction and internal fixation, which further supports this as the approach that best predicts a good result.

Case Reports

Case 1

Chief Complaint and Patient History: G.T. is a 32-year-old man who presents to the emergency department with left knee pain after falling while skiing. The patient reports he caught an edge when skiing and describes a valgus/compression to the joint. He is unable to bear weight and states his knee is quite tight. He denies any numbness or tingling or pain in any other extremity and is otherwise healthy.

Physical Exam: Vital signs are stable. Examination of left lower extremity reveals no lacerations or other cutaneous abnormalities. He has a large tense effusion. He is tender to palpation at the lateral joint line and along the lateral plateau of the tibia. Compartments are soft. Range of motion is guarded secondary to pain, and the knee is held in 20 degrees of flexion. The knee is stable with varus/valgus and Lachman examinations. Posterior drawer and pivot shift are deferred secondary to pain and inability to flex the knee. Sensation is intact in the L2 to S1 nerve distribution. Motor examination reveals 5/5 foot dorsiflexion, plantar flexion, and extensor hallucis longus. There is a 2+ dorsalis pedis and posterior tibialis pulse.

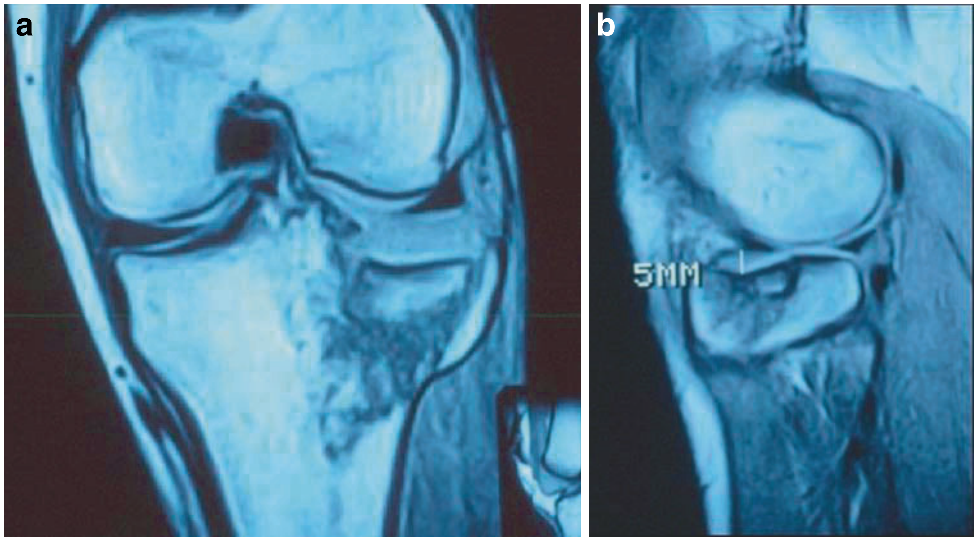


Fig. 19 Case 1. (**A**) Anteroposterior and (**B**) lateral MRI scans of Schatzker type II fracture pattern

Imaging: Anteroposterior and lateral radiographs reveal a split-depression fracture of the lateral tibial plateau consistent with a Schatzker type II fracture pattern. MRI reveals the same (Fig. 19A, B).

Surgery/Treatment: The patient was admitted to the hospital, and the extremity was elevated and iced overnight. The effusion had not worsened, and the soft tissue envelope was intact with only mild periarticular swelling present. The patient was taken to the operating suite, and arthroscopic reduction and internal fixation was performed (see Figs. 3–10). Intraoperatively, arthroscopic evaluation revealed no other significant intraarticular pathology. The joint was visualized, and reduction with direct visualization was performed. The patient tolerated the procedure and was made non-weight-bearing for 8 weeks. Range of motion was regained early, with active range of motion of 0–90 degrees at the first postoperative visit, and a full range of motion was seen at 1-month status postsurgery. The patient began partial weight-bearing at 8 weeks, and there was radiographic evidence of fracture consolidation at that visit. The patient was full weight-bearing and had no significant complaints of knee pain at 12 weeks from initial surgery.

Discussion: This patient presents a classic example of a fracture that can be managed with a minimally invasive and structurally rigid fixation technique that results in an excellent outcome.

Case 2

Chief Complaint and Patient History: K.M. is a 24-year-old college student who presents to clinic with a painful left knee. She reports she was playing intramural rugby and sustained the injury with a noncontact valgus and pivot movement. She was initially seen at a local emergency department and was discharged with a knee immobilizer and sent to an orthopaedic surgeon for follow-up. On presentation to the clinic, she was 1 week from the initial injury, was non-weight-bearing, and was wearing the knee immobilizer. She denied any complaints of numbness or tingling or complaints in any other extremity.

Physical Exam: Vital signs were stable. Examination of left lower extremity revealed no lacerations or other cutaneous abnormalities. There was a moderate effusion. There was no significant tenderness to palpation of the medial or lateral joint lines. Compartments were soft. Range of motion was guarded secondary to pain, but passive range was from 0 to 90. Stability examination revealed a positive Lachman and pivot-shift test. Varus and valgus stress were normal. Sensation was intact in the L2 to S1 nerve distribution. Motor examination revealed 5/5 foot dorsiflexion, plantar flexion, and extensor hallucis longus. There was a 2+ dorsalis pedis and posterior tibialis pulse.

Imaging: Plain radiographs revealed a displaced tibial eminence fracture consistent with a type III avulsion fracture (Fig. 20A, B). There were no other significant plain film findings. MRI also was performed, which revealed a complete avulsion of the tibial eminence with no other significant pathology present.

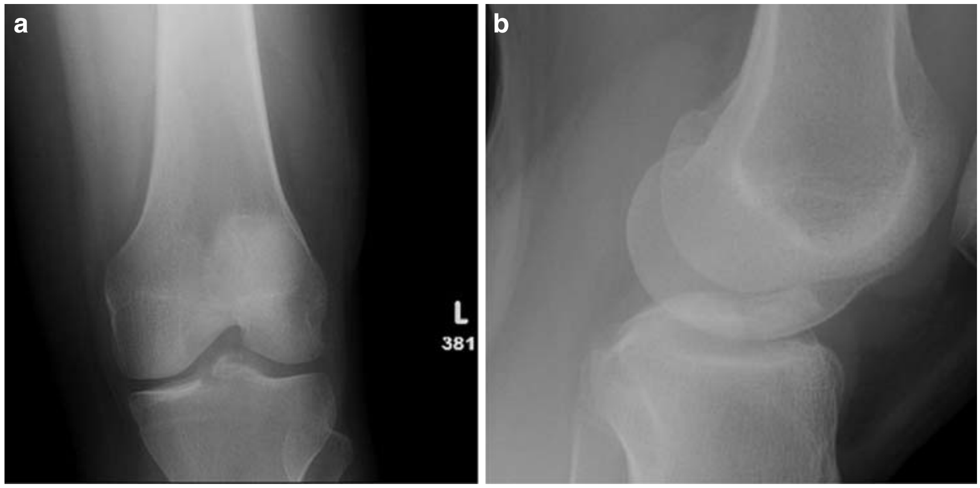


Fig. 20 Case 2. (**A**) Anteroposterior and (**B**) lateral radiographs of a type III tibial eminence fracture

Surgery/Treatment: The patient was taken to the operating room and underwent initial arthroscopic lavage and evaluation of the joint. No other significant intraarticular injury was appreciated. The intermeniscal ligament was anterior to the anterior fracture line and was not interposed. There was cancellous debris within the fracture bed that initially prevented adequate reduction. After debriding the fracture bed with the synovial resector, the fracture was easily reduced with a probe, and a Steinmann pin was placed percutaneously through the reduced fracture. The fracture was then stabilized with two sutures of Ultrabraid as described in the technique earlier, with anatomic reduction being achieved. The knots were tied and brought to the anterior tibial cortex with fixation below the joint using this incisionless surgical technique (see Figs. 11–19). The patient began prone range of motion exercises immediately and was weight-bearing as tolerated with knee locked in full extension for the first 4 weeks. She healed well with no significant complaints. At 3 months postoperatively, she returned to rugby and her other previous activities with no complaints of laxity or pain.

Discussion: This patient represents an example of a nontraditional injury to the ACL. Tibial eminence fractures often are misunderstood to be only childhood injuries, when in fact they also occur in the adult population. Treating this injury pattern with arthroscopic reduction and internal fixation allows for anatomic reduction and early range of motion. The advantage of bony healing provides a rapid healing rate and good clinical outcomes. This patient did well postoperatively and represents the success that is possible with treating tibial eminence fractures with this method.

References

1. Kendall NS, Hsu SYC, Chan K. Fracture of the tibial spine in adults and children. *J Bone Joint Surg Br* 1992;74:848–852.
2. Meyers MH, McKeever FM. Fracture of the intercondylar eminence of the tibia. *J Bone Joint Surg Am* 1959;41:209–222.
3. Gronkvist H, Hirsch G, Johansson L. Fracture of the anterior tibial spine in children. *J Pediatr Orthop* 1984;4:465–468.
4. Wiss DA, Watson JT. Fractures of the tibial plateau. In Rockwood CA, Green DP, Bucholz RW, Heckman JD (eds.), *Rockwood and Green's Fractures in Adults.* Philadelphia: Lippincott-Raven, 1996, pp. 1920–1953.
5. Schatzker J. Tibial plateau fractures. In Browner BD, Jupiter JB, Levine AM, Trafton PG (eds.), *Skeletal Trauma.* Philadelphia: WB Saunders, 1992, pp. 1745–1770.
6. Chandler JT, Miller TM. Tibial eminence fracture with meniscal entrapment. *Arthroscopy* 1995;11:499–502.
7. Owens BD, Crane GK, Plante T, Busconi BD. Treatment of type III tibial intercondylar eminence fractures in skeletally immature atrhletes. *Am J Orthop* 2003;33:103–105.
8. Wiley JJ, Baxter MP. Tibial spine fractures in children. *Clin Orthop Relat Res* 1990;255:54–60.
9. Meyers MH, McKeevor FM. Fracture of the intercondylar eminence of the tibia. *J Bone Joint Surg Am* 1970;52:1677–1683.
10. Noyes FR, DeLucas JL, Torvik PJ. Biomechanics of anterior cruciate ligament failure; an analysis of strain-rate sensitivity and mechanism of failure in primates. *J Bone Joint Surg Am* 1974;56: 236–153.
11. Lubowitz JH, Elson WS, Guttman D. Part II: arthroscopic treatment of tibial plateau fractures: intercondylar eminence avulsion fractures. *Arthroscopy* 2005;21:86–92.
12. Vangness Ct, Gharderi B, Hohl M, Moore TM. Arthroscopy of meniscal injuries with tibial plateau fractures. *J Bone Joint Surg Br* 1994;76:488–490.
13. McClellan RT, Comstock CP. Evaluation and treatment of tibial plateau fractures. *Curr Opin Orthop* 1999;10:10–21.
14. Kode L, Lieberman JM, Motta AO, Wilber JH, Vasen A, Ya-gan R. Evaluation of tibial plateau fractures: efficacy of MR imaging compared with CT. *AJR Am J Roentgenol* 1994;63:141–147.
15. Yacoubian SV, Nevins RT, Sallis JG, Potter H, Lorich D. Impact of MRI on treatment plan and fracture classification of tibial plateau fractures. *J Orthop Trauma* 2002;16:632–637.
16. Holt MD, Williams LA, Dent CM. MRI in the manament of tibial plateau fractures. *Injury* 1995;26:595–599.

17. Brophy DP, O'Malley M, Lui D, Denison B, Eustace S. MR imaging of tibial plateau fractures. *Clin Radiol* 1996;51:873–878.
18. Mui LW, Engelsohn E, Umans H. Comparison of CT and MRI in patients with tibial plateau fracture: can CT findings predict ligament tear or meniscal injury? *Skeletal Radiol* 2007;36: 245–251.
19. Zaricznyj B. Avulsion fracture of the tibial eminence treated by open reduction and pinning. *J Bone Joint Surg Am* 1997;59: 1111–1114.
20. McLennan JG. The role of arthroscopic surgery in the treatment of fractures of the intercondylar eminence of the tibia. *J Bone Joint Surg Br* 1982;64:477–480.
21. Falstie-Jensen S, Sondergard Peterson PE. Incarceration of the meniscus in fractures of the intercondylar eminence of the tibia in children. *Injury* 1984;15:236–238.
22. Brown TD, Anderson DD, Nepola JV, Singerman, RJ, Pedersen DR, Brandet RA. Contact stress aberrations following imprecise reduction of simple tibial plateau fractures. *J Orthop Res* 1988;6:851.
23. Bai B, Kummer FJ, Sala DA, Koval JK, Wolinsky PR. Effect of articular step-off and meniscectomy on joint alignment and contact pressures of fractures of the lateral tibial plateau. *J Orthop Trauma* 2001;15:101–106.
24. Walker S, Erkman MJ. The role of the menisci in force transmission across the knee. *Clin Orhthop* 1975;109:184.
25. Kettlekamp DB, Hillberry BM, Murrish DE, Da H. Degenerative arthritis of the knee secondary to fracture malunion. *Clin Orthop* 1988;234:159.
26. Daniel D, Rice T. Valgus-varus stability in a hinged cast used for controlled mobilization of the knee. *J Bone Joint Surg Am* 1979;61:135.
27. DeCoster TA, Nepola JV. Cast brace treatment of proximal tibial plateau fractures: ten year follow-up study. *Clin Orthop* 1988;231:196.
28. Delamarter R, Hohl M. The cast brace and tibial plateau fractures. *Clin Orthop* 1989;242:26.
29. Cassard X, Beaufils P, Blin JL, Hardy P. Osteosynthesis under arthroscopic control of separated tibial plateau fractures. 26 case reports. *Rev Chir Orthop Reparatice Appar Mot* 1999;85: 257–266.
30. Lemon RA, Bartlett DH. Arthroscopic assisted internal fixation of certain fractures about the knee. *J Trauma* 1985;25: 355–358.
31. Buchko GM, Johnson DH. Arthroscopically assisted operative management of tibial plateau fractures. *Clin Orthop Relat Res* 1996;332:29–36.
32. Jennings JE. Arthroscopic management of tibial plateau fractures. *Arthroscopy* 1985;1:160–168.
33. Gill TJ, Moezzi DM, Oates K, Sterett WI. Arthroscopic reduction and internal fixation of tibial plateau fractures in skiing. *Clin Orthop Relat Res* 2001;383:243–249.
34. Lubowitz JH, Elson WS, Guttmann D. Part I: arthroscopic management of tibial plateau fractures. *Arthroscopy* 2004;20:1063–1070.
35. Baxter MP, Wiley JJ. Fractures of the tibial spine in children. *J Bone Joint Surg Br* 1988;70:228–230.
36. Willis RB, Blokker C, Stoll TM, Paterson DC, Galpin RD. Long-term follow-up of anterior tibial eminence fractures. *J Ped Orthop* 1993;13:361–364.
37. Lee Y, Chin L, Wang N, Hou CH, Lo WH. Anterior tibial spine fracture in children: follow-up evaluation by biomechanical studies. *Chin Med J* 1996;58:183–189.
38. Beaty JH, Kumar A. Fractures about the knee in children. *J Bone Joint Surg Am* 1994;76:1870–1880.
39. Fyfe IS, Jackson JP. Tibial intercondylar fractures in children: a review of the classification and the treatement of malunion. *Injury* 1981;13:165–169.
40. Mah JY, Otsuka NY, Mclean J. An arthroscopic technique for the reduction and fixation of tibial-eminence fractures. *J Ped Orthop* 1996;16:119–121.
41. Janarv P, Westblad P, Johansson C, Hirsch G. Long-term follow-up of anterior tibial spine fractures in children. *J Ped Orthop* 1995;15:63–68.
42. Osti L, Merlo F, Bocchi L. Our experience in the arthroscopic treatment of fracture-avulsion of the tibial spine. *Chir Organi Mov* 1997;82:295–299.
43. Matthews DE, Geissler WB. Arthroscopic suture fixation of displaced tibial eminence fractures. *Arthroscopy* 1994;10:418–423.
44. Hunter RE, Willis JA. Arthroscopic fixation of avulsion fractures of the tibial eminence: technique and outcome. *Arthroscopy* 2004;20:113–121.
45. Lubowitz JH, Grauer JD. Arthroscopic treatment of anterior cruciate ligament avulsion. *Clin Orthop Relat Res* 1993;294:242–246.
46. Biyani A, Reddy NS, Chaudhury J, Simison AJ, Klenerman L. The results of surgical management of displaced tibial plateau fractures in the elderly. *Injury* 1995;26:291.
47. Honkonen SE, Kannus P, Natri A, Latvala K, Jarvinen MJ. Isokinetic performance of the thigh muscles after tibial plateau fractures. *Int Orthop* 1997;21:323.
48. Benirschke SK, Agner SG, Mayo KA, et al. Open reduction internal fixation of complex proximal tibial fractures. *J Orthop Trauma* 1991;5:236.
49. Smith JB. Knee instability after fractures of the intercondylar eminence of the tibia. *J Ped Orthop* 1984;4:462–464.
50. Senekovic V, Veselko M. Anterograde arthroscopic fixation of avulsion fractures of the tibial eminence with a cannulated screw: five-year results. *Arthroscopy* 2003;19:54–61.

Index