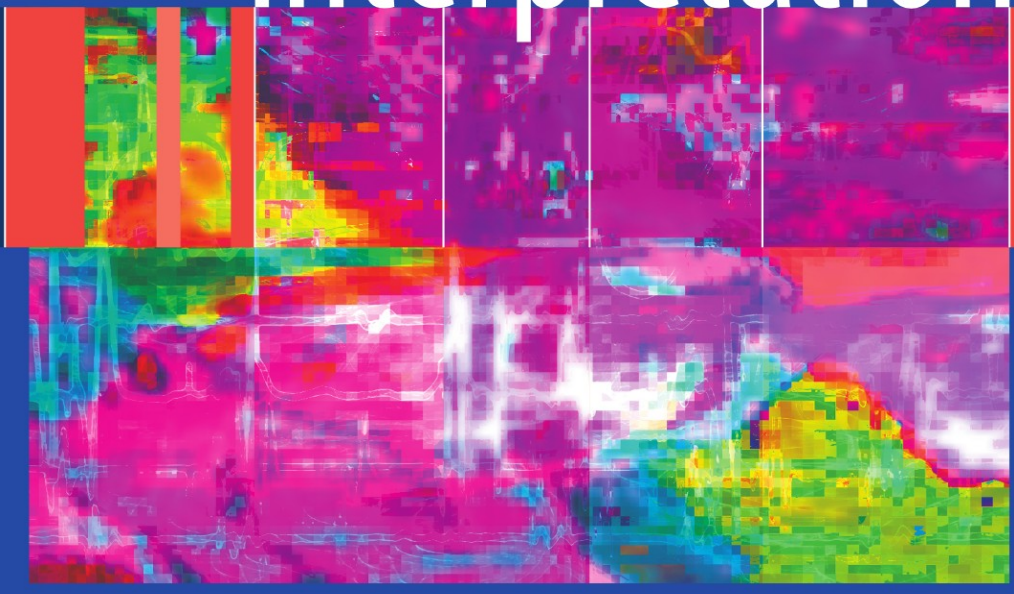
Fred Kusumoto

ECG Interpretation



From Pathophysiology to Clinical Application

Springer

ECG INTERPRETATION: FROM PATHOPHYSIOLOGY TO CLINICAL APPLICATION

ECG INTERPRETATION: FROM PATHOPHYSIOLOGY TO CLINICAL APPLICATION

by

Fred Kusumoto, MD
Electrophysiology and Pacing Service
Division of Cardiovascular Diseases
Department of Medicine
Mayo Clinic
Jacksonville, Florida, USA

Springer

Fred Kusumoto, MD
Division of Cardiovascular Diseases
Department of Medicine
Mayo Clinic
Jacksonville, Florida, USA
Kusumoto.Fred@mayo.edu

ISBN 978-0-387-88879-8 e-ISBN 978-0-387-88880-4
DOI 10.1007/978-0-387-88880-4

Library of Congress Control Number: 2008937757

Printed on acid-free paper

springer.com

To Laura, Miya, Hana, and Aya for their patience and understanding,
and to my parents for putting up with a very inquisitive child.

Preface

Why write another book on ECG analysis and interpretation? Although there are a number of superb introductory and comprehensive books on ECG interpretation, there are very few books that provide the reader information beyond the basics, other than encyclopedic texts. In addition, ECG reading has been traditionally taught using "pattern recognition." However, over the past two decades there has been a tremendous explosion of basic research that has transformed our understanding of the basis of the ECG. Finally, teaching ECGs has often been done by "stand-alone" lectures that have little clinical context; or worse, no organized teaching of ECGs is available because of the tremendous demands of the increasing depth and breadth of medical knowledge that must be mastered during medical school, training, and beyond to become a consumate clinician.

This book has been written to fill these gaps. Although this book provides basic information on ECG analysis it also attempts to explain the electrophysiologic underpinnings for the ECG. Traditional findings such as ST segment elevation are explained with a "framing" case for each chapter with a series of clinically based questions at the end designed to help the student understand the importance of the ECG in clinical medicine. Finally, the book ends with a discussion and series of clinical problems that will help the reader develop a personal style for ECG analysis. In the end I hope the reader finds this text useful for learning how to interpret ECGs in the context of patient care.

This book grew out of a series of lectures on ECG analysis I have given at the University of California, San Francisco; the University of New Mexico; and the Mayo Clinic, Jacksonville. I would like to thank the many students, residents, and colleagues that contributed to this project. I would also like to thank my three mentors that taught me ECG analysis over the years: Nora Goldschlager, Mel Scheinman, and Tom Evans. I appreciate the patience of Melissa Ramondetta for letting this project evolve over a very long time. Finally I would like to thank my family for putting up with the constant typing and the missed soccer games and school plays that a task like this inevitably requires.

Table of contents

Part I: ***Basic electrophysiology and electrocardiography*** 1

1. Cardiac anatomy and electrophysiology 3
2. Physics of electrocardiography 11
3. The normal electrocardiogram 21

Part II: ***Abnormal depolarization*** 35

4. Chamber enlargement 37
5. Conduction abnormalities in the His-Purkinje tissue 49

Part III: ***Abnormal repolarization*** 63

6. Ventricular repolarization: T waves and U waves 65
7. ST segment elevation and other ECG findings in myocardial infarction 81
8. ST segment elevation not associated with myocardial infarction 111

Part IV: ***Arrhythmias*** 127

9. Premature beats 129
10. Bradycardia 139
11. Supraventricular tachycardia 155
12. Wide complex tachycardia 183
13. Pacemakers 205

Part V: ***"Putting it all together"*** 215

14. Analyzing ECGs: Methods, techniques, and identifying abnormalities 217

15. Analyzing ECGs: Putting it together with case studies 225

16. Electrolyte disorders 249

17. Orphans 259

Appendix 277

Extra practice "So you're a glutton for punishment" 283

Index 293

Part I

Basic electrophysiology and electrocardiography

Chapter 1
Cardiac anatomy and electrophysiology

Since its development in the early 1900s by Einthoven, the electrocardiogram (usually referred to by its acronym, ECG) has become an important tool for evaluating the heart. During the last twenty years, our understanding of the basic electrophysiology of the heart has dramatically increased, which has provided further insight into the physiologic basis of the electrocardiogram. In this first chapter basic electrophysiology and cardiac anatomy will be reviewed. Although these principles can be difficult to understand, they provide an important foundation for understanding the physiologic and pathophysiologic basis for the ECG. In this way, rather than evaluating the ECG using "pattern recognition," the mechanisms for ECG changes can be understood and hopefully more easily remembered. Readers are encouraged to refer back to this chapter as they read about specific conditions observed in an ECG in later chapters.

ECG: electrocardiogram; EKG: elektrokardiogramm

Although Einthoven perfected the string galvanometer in Leiden, The Netherlands, and used the acronym EKG to describe his tracings, as English has become more dominant in today's world, the acronym ECG has now become more common.

Cardiac electrophysiology

All cells have a cell membrane that separates the interior and exterior of the cell. The cell membrane allows different ion concentrations to be maintained in the intracellular space and extracellular space. The cell membrane is composed of a phospholipid bilayer, within which cholesterol molecules and proteins are found. Proteins are a critical component of the cell membrane; they allow selective movement of different ions at different times in the cardiac cycle. For the cardiac cells, voltage differences between the inside and outside of the cell are generated by sequential opening and closing of different ion channels. Ion channels are simply "pores" that, when open, allow passive movement of ions across the cell membrane down the electrical or concentration gradient of the ion. The concentration differences of ions between the inside and outside of the cell are formed and maintained by the action of protein pumps and channels, including the Na^+-K^+-ATPase

F. Kusumoto, *ECG Interpretation: From Pathophysiology to Clinical Application*,
DOI 10.1007/978-0-387-88880-4_1, © Springer Science+Business Media, LLC 2009

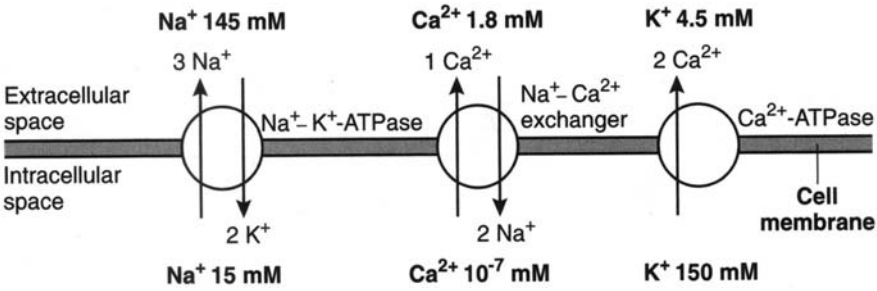


Figure 1: Different ion concentrations are established between the intracellular and extracellular spaces by the action of several protein pumps and exchangers. The Na^+-K^+ ATPase is the main pump behind these differences by transporting 3 Na^+ out and 2 K^+ in, using the energy from ATP breakdown. Higher extracellular Ca^{2+} concentrations are maintained by the Ca^{2+} ATPase and the Na^+-Ca^{2+} exchanger. The Na^+-Ca^{2+} exchanger is driven by Na^+ traveling into the extracellular space down its electrochemical gradient (reprinted with permission from Kusumoto FM, *Cardiovascular Pathophysiology*, Hayes Barton Press, Raleigh, NC, 1999).

(Figure 1). At rest the intracellular concentration of K^+ is relatively high and concentrations of Na^+ and Ca^{2+} are relatively low. For this reason if Na^+ and Ca^{2+} channels were to open these ions would flow into the cell.

At rest, cells are permeable to K^+ ions via a specific potassium channel called the inwardly rectifying current (I_{K1}). The concentration gradient favors outward flow of K^+ ions. Since the predominant negatively charged particles in the cell are large proteins that cannot cross the membrane, a negative charge builds up

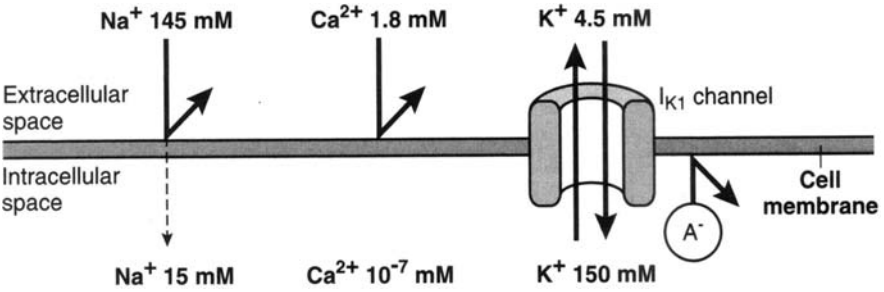


Figure 2: At baseline, the membrane is impermeable to Na^+ and Ca^{2+}. K^+ flows freely through open K^+ channels. At rest, K^+ is at equilibrium with outward flow down the K^+ concentration gradient balanced by inward flow to the development of intracellular negative charge from large anionic proteins that cannot travel across the membrane (reprinted with permission from Kusumoto FM, *Cardiovascular Pathophysiology*, Hayes Barton Press, Raleigh, NC, 1999).

inside the cell. At rest, K^+ ions are in equilibrium with the concentration gradient favoring outward K^+ flow, while the negative charge inside the cells favors flow of K^+ into the cells. Although K^+ channels are "open" at rest, Na^+ and Ca^{2+} channels are "closed;" these ions are not at equilibrium and represent potential energy (Figure 2).

For the resting cardiac myocyte the intracellular concentration of K^+ is relatively high and the concentrations of Na^+ and Ca^{2+} are relatively low.

Action potential

If a small amount of voltage is applied to a cardiac myocyte, the voltage of the myocyte will change in a characteristic repeatable pattern. These voltage changes are mediated by Na^+, K^+, and Ca^{2+} ions traveling across the membrane down their concentration and electrical gradients as their respective ion channels open and close. Electrophysiologically, there are two characteristic action potentials that can be observed in cardiac cells: fast response action potentials, and slow response action potentials.

Fast response action potential

Atrial and ventricular myocytes exhibit fast response action potentials. In fast response action potentials, depolarization of the cell membrane leads to opening of specialized Na^+ channels (Figure 3). Opening of these channels allows Na^+ to flow rapidly into the cell, since both the concentration gradient and electrical gradient favor inward flow of Na^+. The influx of Na^+ leads to a rapid upstroke of the action potential (phase 0) and the membrane potential becomes approximately 10 mV (the interior is 10 mV more positive than the exterior). The majority of these Na^+ channels are open for milliseconds. Once most of the Na^+ channels have closed, the myocyte maintains a voltage of approximately 0 mV (voltages in the intracellular and extracellular spaces are roughly equal) for a relatively long period of time called the plateau phase (phase 2). This plateau phase is maintained by an inward flow of Ca^{2+} (via I Ca-L channels) and a continued inflow of Na^+ through a few channels that remain open, and an outward flow of K^+ mediated by a series of different protein ion channels that have different timing characteristics. In particular, the K^+ channels responsible for repolarization are unique from the K^+ channels that are open at baseline and are called "delayed rectifier channels," because there is a delay in pore opening with membrane depolarization. The plateau phase ends when Ca^{2+} and Na^+ flows decrease as these channels become inactive and K^+ permeability increases due to the delayed opening of the K^+ channels. The cell returns to the original membrane potential when K^+ returns to equilibrium—K^+ channels (I_{K1}) open and Na^+ and Ca^{2+} channels closed. In the background, the Na^+-K^+-ATPase continues to pump Na^+ out of the cell and bring K^+ into the cell, so that the

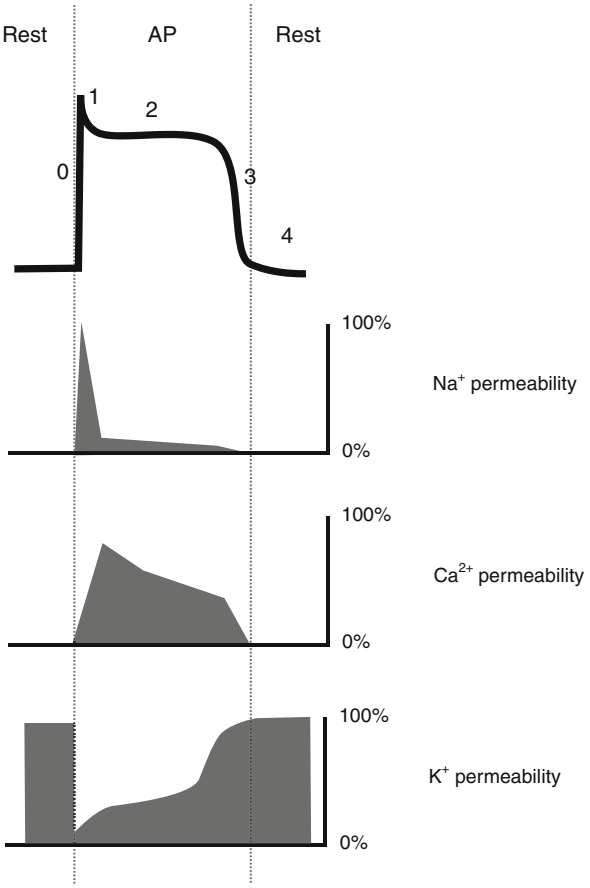


Figure 3: Ion permeabilities at rest and during the cardiac action potential (AP). At rest the membrane is permeable to K^+, but impermeable to Na^+ and Ca^+. During phase 0, K^+ permeability drops precipitously (K^+ channels close), and Na^+ permeability and Ca^{2+} permeability increase (Na^+ and Ca^{2+} channels open). A slight rise in K^+ permeability due to opening of specialized K^+ channels (I_{to}) leads to phase 1. The plateau (phase 2) is mediated by inward Na^+ and Ca^{2+} balanced by outward K^+. Phase 3 occurs as K^+ permeability increases and Na^+ and Ca^{2+} permeability decrease and the cell returns to baseline (phase 4).

membrane potential always returns to its baseline value of approximately -90 mV with high intracellular K^+ and high extracellular Na^+. For the interested reader, a diagram showing the individual K^+ currents that are responsible for different parts of the action potential is shown in Chapter 6, Figure 3.

Slow response action potential

Slow response action potentials are found in AV node cells and sinus node cells (Figure 4). The action potentials of slow response cells have three basic differences from those of fast response cells: depolarization is less rapid, no plateau phase exists, and there is no true resting membrane potential. In slow response cells, Na^+ channels do not contribute to the action potential. Instead, depolarization of the cells is mediated by the opening of Ca^{2+} channels (I Ca^{2+}-L current) that allow inward flow of Ca^{2+}. The opening of Ca^{2+} channels is slower than the opening of Na^+ channels and this results in a lower velocity upstroke. In slow response cells, no plateau phase is present; instead, cells repolarize slowly through the opening of K^+ channels. Finally, slow response cells do not have a resting I_{K1} current. For this reason the action potential approaches but does not reach the K^+ equilibrium value. Slow

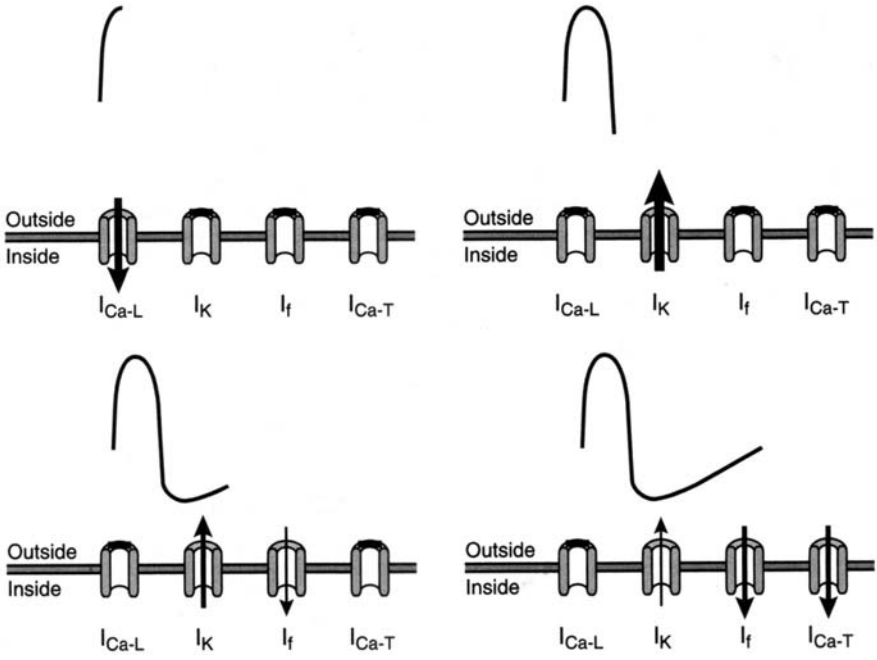


Figure 4: Ion channel opening and closing in slow response cells. Since no Na^+ channels are present, the upstroke is due solely to opening of Ca^{2+} channels (I_{Ca-L}). Repolarization occurs as K^+ channels open. Since slow response cells have no resting I_{K1} current, gradual diastolic depolarization is noted. Diastolic depolarization results from multiple factors, including gradual decrease in K^+ permeability and inward flow of Ca^{2+} (I_{Ca-T}) and Na^+ (I_f) (reprinted with permission from Kusumoto FM, *Cardiovascular Pathophysiology*, Hayes Barton Press, Raleigh, NC, 1999).

response cells reach a maximal negative potential of approximately −65 mV and then slowly depolarize spontaneously (diastolic depolarization). Diastolic depolarization is mediated by three ion currents. First, the delayed rectifier current begins to decay (the cell becomes less permeable to K^+). Second, there is a small amount of inward Na^+ flow due to the action of a small current called I_f. Third, Ca^{2+} inflow occurs mediated by specialized I Ca-T channels. When the cell reaches threshold, the I Ca^{2+}-L channels are activated and the cycle repeats itself.

The electrophysiologic differences of slow response cells lead to two fundamental clinical observations. First, since the cells exhibit spontaneous depolarization they activate repetitively and act as pacemaker cells. This allows for spontaneous and repetitive activation of the heart. Second, the slower depolarization upstroke means that these cells conduct impulses less rapidly. Slow conduction properties of cells in the AV node allow a temporal delay which coordinates atrial and ventricular contraction, and also "protect" the ventricles from any rapid atrial arrhythmias.

Fast response cells have a sharp upstroke, a prolonged plateau phase, and usually minimal pacemaker activity. Slow response cells have a slow upstroke, no plateau phase, and pacemaker activity.

Cardiac anatomy

The left and right atria and ventricles contract in coordinated fashion to pump blood to the body and lungs. Under normal conditions, the heart is "driven" by the sinus node since these cells have the highest natural pacemaker rate (the fastest phase 4 depolarization) (Figure 5). The sinus node is a fairly large structure, often

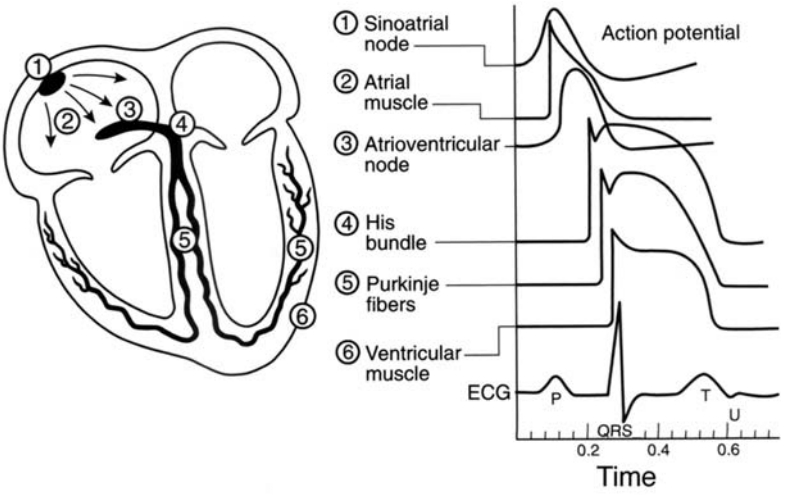


Figure 5: Normal activation of the heart (reprinted with permission from Kusumoto FM, *Cardiovascular Pathophysiology*, Hayes Barton Press, Raleigh, NC, 1999).

1–1.5 cm long, located near the junction of the superior vena cava and the right atrium. Once the electrical impulse leaves the sinus node, the atria are activated. Since the sinus node is located in the right atrium, right atrial activation occurs slightly earlier and is completed slightly sooner than left atrial activation.

Within the septal region of the atria, the electrical impulse travels through the AV node, where conduction delay allows the ventricles to optimally fill (remember that electrical activity is faster than the time required for contraction of the myocytes and transfer of blood from the atria to the ventricles). Conduction delay occurs within the AV node both because the AV node cells have slower activation and because of slower cell-to-cell propagation. Once the electrical impulse passes through the AV node, the impulse travels rapidly through the His bundle and the left and right bundles to activate the ventricles rapidly and almost simultaneously via an intricately branching network of cells called the Purkinje system. The myocytes from the His bundle to the terminal portions of the Purkinje system are characterized by large phase 0 upstrokes and rapid intercellular conduction that leads to efficient spread of the electrical impulse throughout both ventricles.

The atria and ventricles are separated by a fibrous framework (annulus) that is electrically inert, so that the AV node and the contiguous His bundle form the only electrical connection between the atria and ventricles under normal conditions. This anatomic arrangement along with electrical delay within the AV node allows the atria and ventricles to beat in a synchronized fashion and minimizes the chance of electrical feedback between the chambers.

Key points

1. The resting membrane potential is determined by (1) extracellular and intracellular ion concentration differences formed by the Na^+-K^+-ATPase and (2) baseline membrane permeability to K^+.
2. There are two types of action potentials: fast response and slow response.
3. Fast response action potentials have a rapid phase 0 upstroke due to the presence of Na^+ channels. The upstroke of slow response tissues is due to the influx of Ca^{2+}.
4. Cardiac activation proceeds systematically from (through) the sinus node, atria, AV node, His Purkinje system, and ventricles.

Review questions

1. The drug dofetilide blocks K^+ channels (delayed rectifier current). What effect would be noted on the action potential?

A. Repolarization would be delayed
B. Repolarization would occur more quickly
C. Phase 0 upstroke would become steeper
D. Phase 0 upstroke would become less steep

2. The drug flecainide blocks Na^+ channels. What effect on the action potential would be observed?

A. Phase 0 would become less steep
B. Spontaneous automaticity would be observed
C. Repolarization would be delayed
D. Repolarization would occur earlier

3. Normally cardiac activation is initiated in the:

A. AV node
B. Sinus node
C. Bachmann's bundle
D. His-Purkinje fibers

Answers to the review questions

1. A. The delayed rectifier K^+ current is important for repolarization of cardiac myocytes. Blocking the activity of this channel would tend to delay repolarization. K^+ channels do not mediate depolarization of cardiac myocytes and would not affect phase 0.
2. A. By blocking the opening of Na^+ channels, the phase 0 upstroke would be less steep. Na^+ channels contribute very little to repolarization. Similarly Na^+ channels do not generally contribute to automaticity. In His-Purkinje tissue, Na^+ channels can mediate automaticity, but blocking slow inward current of Na^+ would tend to decrease automaticity.
3. B. The sinus node initiates the normal heart beat.

Chapter 2
Physics of electrocardiography

If there is any word that strikes fear in the hearts of many students in the health care field, it is "physics." Much of this fear is unnecessary, at least in relation to the ECG, because a thorough understanding of the physical basis for the ECG provides an important foundation for the understanding and interpretation of ECGs. In this chapter, the physical processes that are important for understanding the ECG will be explored.

Physics of electrocardiography

Depolarization

As described in the prior chapter, at baseline, most cardiac cells have a stable resting membrane potential in which the inside of the cell has a relatively negative voltage when compared to the outside of the cell. A three-cell model of the heart with three electrode pairs is shown in Figure 1. If one places several electrode pairs at various positions around our cell model, no voltage differences will be recorded, since the electrodes are exposed to a similar excess positive charge over the entire model (Figure 1, top left panel).

Now a small depolarizing pulse is applied to the cell at one end of our model. Na^+ channels open and Na^+ flows down its electrochemical gradient, the inside of the cell becomes positive, and the surface of the cell develops a relatively negative charge. Now a measurable voltage difference exists in our model, with one end having a relatively negative surface charge (due to inward flow of Na^+) compared to an adjacent area that is still at rest (Figure 1, middle left panel). As the three-cell model is progressively depolarized the area of activation spreads from left to right until all three cells are depolarized (Figure 1, bottom left panel).

1. *When a wave of depolarization approaches the positive electrode of a recording system, a positive signal will be recorded.*
2. *When a wave of depolarization travels away from the positive electrode, a negative signal will be recorded.*

F. Kusumoto, *ECG Interpretation: From Pathophysiology to Clinical Application*,
DOI 10.1007/978-0-387-88880-4_2, © Springer Science+Business Media, LLC 2009

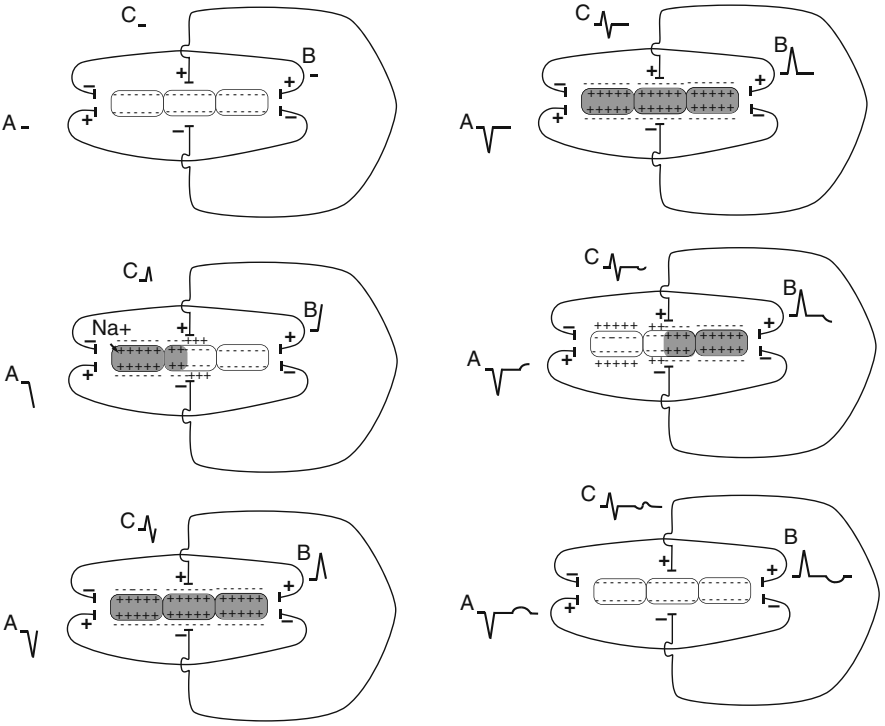


Figure 1: Three-cell model for measuring ECG signals. Please see the text for discussion.

The movement of this wave of depolarization can be measured by our three lead recording systems (Figure 1, left column). Different voltages will be recorded by our three lead systems depending on the orientation between the electrode positions and the wave of activation. By convention, when a wave of depolarization approaches the positive electrode of a recording system, a positive signal will be recorded. Conversely, when a wave of depolarization travels away from the positive electrode, a negative signal will be recorded. In our model, for lead system **B**, the wave of depolarization approaches the positive electrode and a positive signal is recorded (Figure 1, bottom left panel). For lead system **A**, the wave of activation travels away from the positive electrode and a negative signal is recorded. For lead system **C**, the wave of activation first approaches and then travels away from the positive electrode so that the recorded signal has an initially positive signal and then a negative signal.

The surface ECG measures the sum of electrical activity of the heart. Since the ventricles are the largest chambers, ventricular depolarization leads to the largest signal. The amplitude of the signal will depend on the amount of tissue depolarized (more tissue leads to a larger signal), the direction of depolarization (a wave that travels directly toward the positive electrode will have the largest signal), and whether any "canceling" forces are present (for example, one wave of depolarization

traveling toward the positive electrode and another wave simultaneously traveling away from that electrode).

Repolarization

After activation, the cells remain in the plateau phase (Figure 1, top right panel). Since the surface charge of all three cells is similar, no voltage differences are recorded by our three lead systems. During the plateau phase of the action potential there will be an isoelectric period on all of the lead systems, since all of the cells in our three-cell system have a similar voltage (0–10 mV). However, as K^+ permeability increases, the cell begins to repolarize, and a voltage difference will be detected by our lead systems (Figure 1, middle right panel). Since the change in voltage is more gradual (phase 3 is less steep than phase 0), the electrical signal measured during repolarization is usually of lower amplitude. At the initial region of repolarization the cell surface will be relatively positive to the other portions of the three-cell model and a negative deflection will be detected in system **B** and a positive deflection will be observed in system **A** (Figure 1, bottom right panel).

In the normal ECG, the T wave generally has the same direction as the QRS complex: if the QRS is positive the T wave is positive, and if the QRS is negative the T wave is negative. Given this information, what must be the normal relative directions of depolarization and repolarization? The answer will be provided in Chapter 3.

Standard leads

The standardized electrode recording system has changed little from the 1940s. While in certain cases specialized lead recording systems are used, an ECG composed of twelve leads is by far and away the most commonly used. The 12 ECG leads are divided into six limb leads and six precordial leads (Figure 2).

Limb leads

There are six limb lead systems: three "bipolar" leads that are derived from Einthoven's triangle, and three "unipolar" lead systems that were developed in the 1940s.

Bipolar leads: Einthoven's triangle

The first lead systems used buckets of salt water as electrodes (one can see why limb leads were for many years the only type of leads that were developed and that patients must have had a lot of confidence in their physicians). If the arms are

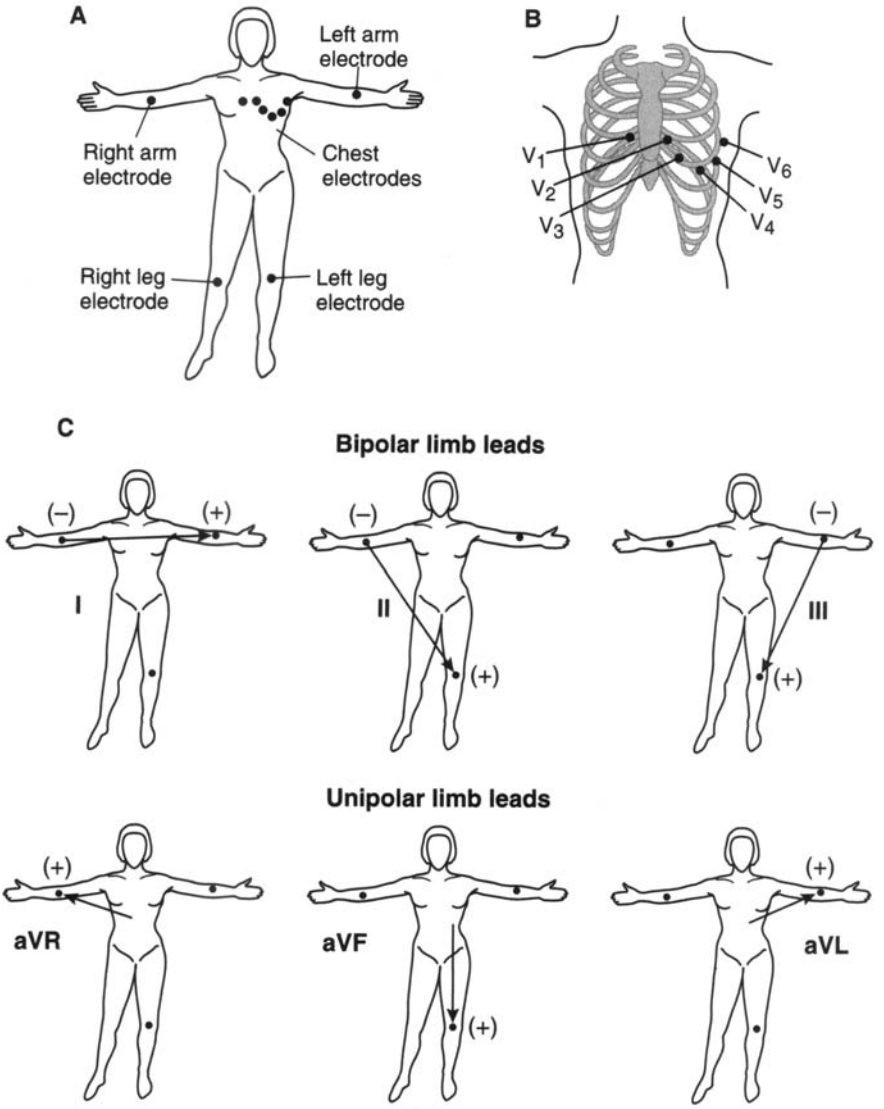


Figure 2: **A**. Location of the standard positions for the electrodes. **B**. Position of the chest electrodes relative to the ribcage (see text for discussion) (reprinted with permission from Kusumoto FM, *Cardiovascular Pathophysiology*, Hayes Barton Press, Raleigh, NC, 1999).

spread out, the electrodes are equidistant from the heart and an equilateral triangle is formed. Einthoven measured the potential difference between the right and left arm (I), the right arm and the left leg (II), and the left arm and the left leg (III). For lead I he defined the left arm as positive, and for leads II and III, he defined the left leg as positive. Since the heart can be defined as a closed circuit, the three leads can be summed algebraically. Since he defined the left leg as positive for both leads II and III, lead II = lead III + lead I (Figure 2). In fact, in modern ECG machines only lead I and lead II are measured, and lead III is actually derived mathematically.

It was pointed out by Wilson in the late 1920s that the limbs are really "extension cords" and the recorded electrical signal is similar regardless of the specific position of the lead on the limb. However, it is important to remember that electrodes for the limbs should not be placed on the torso, because the signal can become attenuated and distorted. Modern ECG recording systems that are designed for exercise testing often use a mathematical algorithm that will "reconstruct" a standard 12-lead ECG from limb positions on the torso. The upper extremity leads are placed just below the clavicle and the lower extremity leads are placed in the left and right iliac fossa. Collectively, this electrode placement system is sometimes called the Mason-Likar limb leads in honor of the investigators that introduced this concept in the mid 1960s.

Unipolar or augmented leads

In the late 1940s unipolar leads were developed. Unipolar is really a misnomer, since any electrical recording system requires two electrodes to "complete the circuit." However, with unipolar leads the positive electrode is the right arm, left arm, and the left leg, and the negative electrode is the sum of the remaining two limb leads. These leads have traditionally been called unipolar, because the signal is measured from an exploring electrode and an indifferent electrode (the voltage sum of the remaining two electrodes). They are associated with a small "a" because the leads must be Augmented, amplifying the signal by a small factor (1.1), in order to have voltages equal to those obtained through the bipolar signals. The term unipolar is gradually being phased out, but the concept remains useful for conceptually separating the frontal leads: I, II, and III developed by Einthoven vs. aVR, aVL, and aVF.

The relationship between the bipolar leads and the unipolar leads can be better understood by placing the negative electrode of each of the lead systems in the center and plotting the vectors (Figure 3). The common center is then placed in the middle of the heart. Each of the vectors radiates from the heart in a single plane called the frontal plane. The leads can be grouped based on their relative directions in the frontal plane. The positive electrodes can then be oriented as a clockface. By convention 0° is defined as horizontal and to the left, clockwise movement is positive, and counterclockwise movement is negative. With this configuration it becomes obvious that I and aVL are in the same general direction, 0° and −30° respectively, and are often grouped together as the "lateral leads." Similarly, leads II, aVF, and III are called the "inferior leads" and are oriented at 60°, 90°, and 120° respectively.

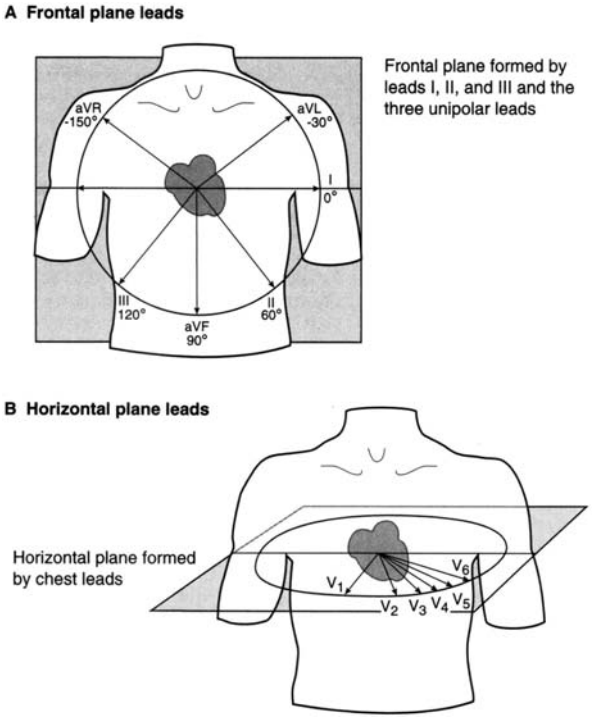


Figure 3: **A**. Frontal plane leads with the negative electrodes aligned to a central point. The approximate location of the heart is shown for reference. The leads are described relative to lead I. Counterclockwise is defined as the negative direction and clockwise is the positive direction. **B**. The horizontal or precordial plane and the relative position of the chest leads (reprinted with permission from Kusumoto FM, *Cardiovascular Pathophysiology*, Hayes Barton Press, Raleigh, NC, 1999).

Chest or precordial leads

The leads in the frontal axis look at the electrical forces of the heart in only a single vertical plane. In the 1930s Wilson developed a lead system that looked at the heart in the horizontal plane. Six leads were described, which are located around the front of the chest. For all of the chest leads, the negative electrode is the sum of the limb leads and the exploring electrode is placed in different positions on the anterior chest wall. For lead V_1, the positive electrode is placed in the right fourth intercostal space, lead V_2 is placed in the left fourth intercostal space, lead V_3 is placed midway between V_2 and V_4, lead V_4 is placed in the fifth intercostal space in the left midclavicular line (an imaginary line drawn from the clavicle downward), lead V_5 is placed at the same level of V_4 at the left anterior axillary line, and lead V_6 is also placed at the level of V_4 but positioned in the midaxillary line. These lead

locations were initially chosen to standardize research but have become the clinical standard for ECG acquisition. The positive electrodes of the chest leads encircle the heart anteriorly (V_1–V_4) and laterally (V_5–V_6). It is important to try to consistently place the precordial leads using anatomic landmarks; misplacing the leads too high or too low on the chest wall can significantly alter the recorded signal.

Again, correct lead position is critical for obtaining the ECG. As will be outlined in Chapter 17, incorrect placement of the precordial leads in a higher interspace will alter the signal significantly. In women, electrodes should generally be placed below the breast if possible, but location remains the most critical criterion. If the electrode must be placed on top of the breast, the amount of signal attenuation is minimal.

Derived 12-lead ECG

There are certain situations where placing all ten electrodes for a 12-lead ECG can be difficult. In response to this need, a simpler four-lead system has been developed called the EASI system. Four leads are placed in a roughly perpendicular orientation: Electrodes are placed on the upper part of the sternum (S electrode), the lower sternum at the fifth intercostal space (E electrode), the right midaxillary line on the fifth intercostal space (I electrode), and the left midaxillary line at the fifth intercostal space (A electrode). A fifth ground electrode can be placed anywhere on the torso. From these lead positions a derived 12-lead ECG that correlates with the traditional 12-lead ECG can be obtained.

Similarly, algorithms are available that use 6 standard electrode sites: Mason–Likar limb leads and V2 and V5. The remainder of the precordial leads are mathematically derived. It should be noted that most ECG machines measure two limb leads, most often I and II, and derive by calculation the other four limb leads (remember Einthoven's Law).

Although derived 12-lead ECGs can be useful in monitoring situations where continuous and complete ECG acquisition is unnecessary or inconvenient, it should be remembered that the derived ECG does not replace the standard 12-lead ECG.

Standard ECG display

With modern ECG machines, all 12 leads are active simultaneously and usually obtain 10 s of data from the heart. Since the usual paper speed is 25 mm/s, each "large box" represents 0.20 s. Display formats vary, but in general the 12 leads are displayed in four columns: column 1 – I, II, III; column 2 – aVR, aVL, and aVF; column 3 – V_{1-3}; column 4 – V_{4-6} (Figure 4). Thus the first two columns represent the frontal leads and the right two columns represent the precordial leads. Usually a "rhythm strip" with one to three leads is displayed on the bottom of the page. As will be apparent when we discuss arrhythmias, simultaneous activation is very

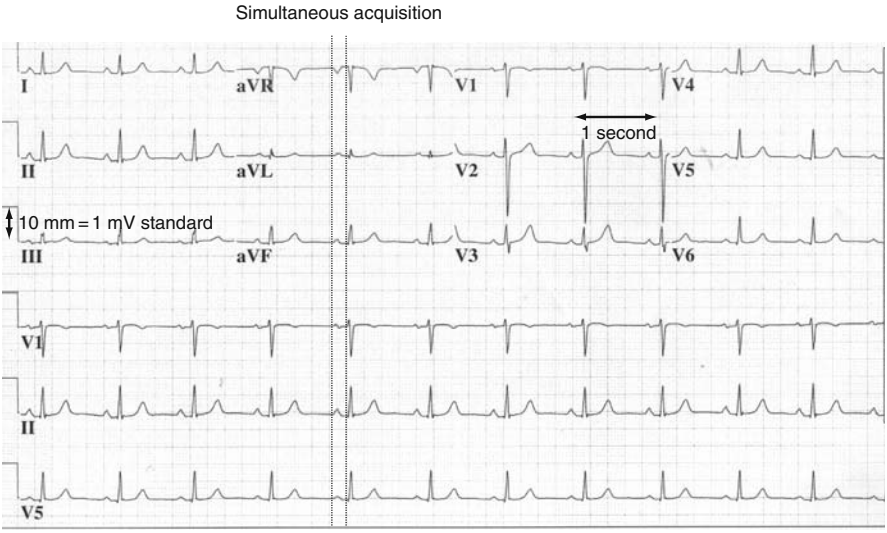


Figure 4: Standard ECG format in columns. The first column are leads I, II, and III. The second column is leads aVR, aVL, and aVF. The third column is V_1, V_2, and V_3. The fourth column is V_4, V_5, and V_6. The first and second columns are the frontal plane leads and the third and fourth columns are the precordial plane leads. Usually at the bottom of the standard ECG are one or more continuous recordings from a single lead called a rhythm strip. From left to right all of the signals in a vertical axis are acquired simultaneously so that signals from the heart will "line up." In this example, the beginning of the P wave and QRS can be observed in leads aVR, aVL, aVF, V_1, II, and V_5. This can be important if there is uncertainty about the origin of signals, particularly in patients with abnormal heart rhythms.

useful since it allows simultaneous comparison of waveforms from multiple leads that can help discern QRS morphology and P wave location in patients with slow heart rates (bradycardia) or fast heart rates (tachycardia).

Summary

The electrical activity from the heart can be measured from the surface using the ECG. The signal obtained will depend on the relationship between the direction of depolarization or repolarization and the orientation of the lead system. The standard ECG is composed of 12-lead systems. There are six frontal leads in which the positive electrodes are oriented within the vertical plane of the body. There are six precordial leads in which the positive electrodes are located in the horizontal plane at chest level.

Key points

1. Depolarization and repolarization of cardiac cells can be recorded from the surface by the ECG.
2. The frontal leads include the "bipolar" leads I, II, and III and the "unipolar" leads aVR, aVL, and aVF.
3. The frontal leads can be grouped together with a common center (negative electrode) with the vectors of the positive electrodes radiating outward like spokes on a wheel.
4. The precordial leads are V_1 through V_6 and measure electrical activity in the horizontal plane (a plane roughly parallel to the ground).

Review questions

1. A wave of depolarization traveling directly away from the positive electrode of a lead system will generate a:

   A. Negative deflection
   B. Positive deflection
   C. Biphasic deflection
   D. No electrical activity

2. Normally which chamber will generate the largest electrical signal?

   A. Left atrium
   B. Right atrium
   C. Left ventricle
   D. Right ventricle

3. Name the six limb leads and the relative positions of the positive electrodes.
4. Describe the difference between the precordial and frontal leads.

Answers to the review questions

1. A. A wave of depolarization traveling away from the positive electrode of a recording system will generate a negative signal on the ECG.
2. C. The size of the electrical signal recorded on a surface ECG correlates with the size/mass of the cardiac chamber. Since the left ventricle has the largest mass of the four cardiac chambers, it generates the largest signal.
3. I, II, III, aVL, aVR, and aVF. The orientation of the leads is defined relative to a horizontal ray traveling right to left, so that the orientations of the electrodes are 0°, 60°, 120°, −30°, −150°, and 90° respectively.
4. The precordial leads are in the horizontal plane and the frontal leads are located in the vertical plane.

Chapter 3
The normal electrocardiogram

The electrical activity generated during the cardiac cycle can be measured from the body surface as the ECG. This chapter will summarize the correlation between cardiac events and the ECG in the normal heart and will follow the time course of cardiac activation.

Case study: Joan Miera is a 22-year-old student that is being evaluated in a pre-sports physical by her physician. Her ECG is shown in Figure 1.

Atrial depolarization

Atrial activation begins at the sinus node. Remember from Chapter 1 that the sinus node is located at the right atrial-superior vena cava junction. For this reason the atria are normally depolarized from right to left and superior to inferior ("high-low"). Atrial depolarization can be observed on the ECG as the P wave (Figure 2). Since lead II is oriented at 60°, and aVR is oriented at −150°, the P wave is usually positive in lead II and negative in lead aVR. However, there are exceptions to this generalization. In some cases the P wave will be flatter and almost isoelectric in lead II because a lower region of the sinus node becomes the dominant pacemaker of the heart (Figure 3). This is most frequently observed during sleep, where increased parasympathetic tone encourages pacing from lower sites in the sinus node. These lower regions have slower phase 4 depolarization slopes and slower heart rates.

Case study: Notice that the P wave in Ms. Miera's ECG is positive in lead II and negative in aVR. This suggests that the sinus node is "driving" the heart and atrial activation is normal.

Atrioventricular conduction

Electrical activation travels through the right atrium, and the AV node is depolarized at some point in the middle-terminal portion of the P wave. The AV node has slow conduction properties due to the absence of Na^+ channels, and electrical

F. Kusumoto, *ECG Interpretation: From Pathophysiology to Clinical Application*,
DOI 10.1007/978-0-387-88880-4_3, © Springer Science+Business Media, LLC 2009

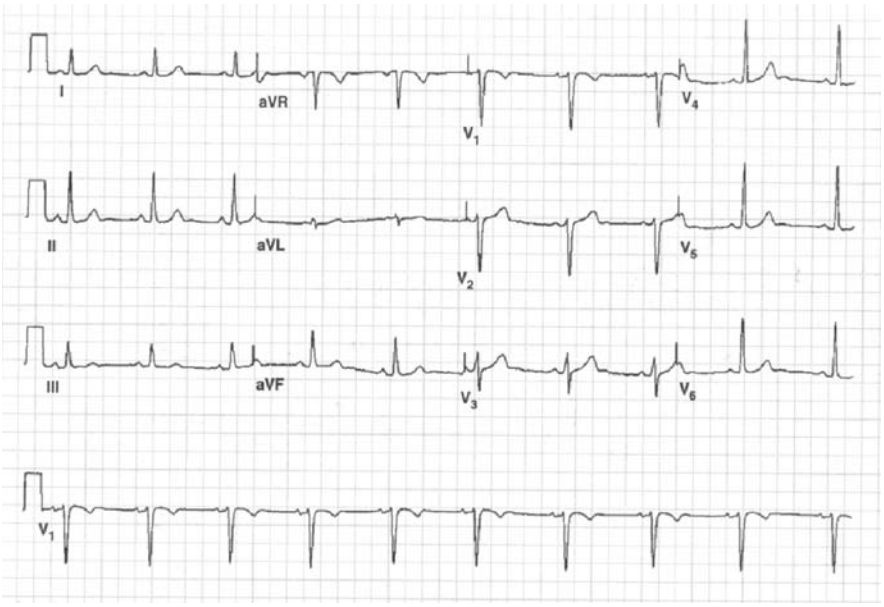


Figure 1: Ms. Miera's ECG.

activation of the heart slows significantly. This slow conduction is important because it allows more efficient sequential mechanical contraction of the atria and ventricles. Remember that once atrial contraction occurs, it takes some time for blood to travel from the atria to the ventricles. Since the atria have already depolarized and are in the plateau phase and the ventricles have not been activated during this period, there are no large electrical gradients that can be measured from the surface and no deflections are recorded on the ECG. Electrical activation within the AV node is too small to be measured from the electrocardiogram.

Once the AV node activation is completed, the electrical activation travels through the His-Purkinje system. Activation of the His-Purkinje system is rapid, but again the total mass of cardiac depolarization is too small to be measured from the surface using the ECG, and an isoelectric signal is recorded in all leads. Atrioventricular conduction occurs during the isoelectric period separating the P wave and the QRS complex.

Atrial repolarization is usually not observed on the ECG for several reasons. First, atrial repolarization results in a smaller signal than atrial depolarization (think of the relative sizes of the QRS complex and the T wave). Second, atrial tissue mass is usually relatively small. Third, atrial repolarization is often obscured by the QRS complex.

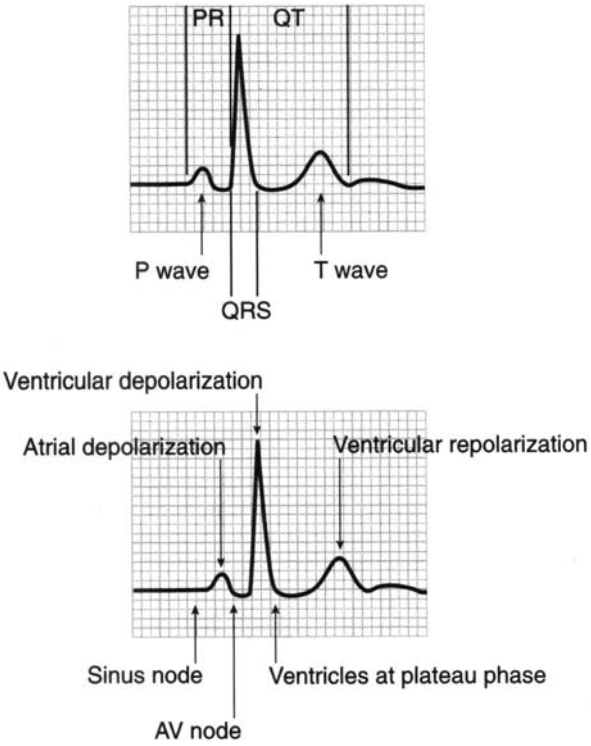


Figure 2: The P wave represents atrial depolarization. During the isoelectric portion of the PR interval the AV node is being depolarized. Ventricular depolarization produces the QRS complex. After the QRS complex the ventricular myocytes are at the plateau phase and an isoelectric ST segment is observed. Gradual repolarization of the ventricles leads to the broad-based T wave (reprinted with permission from Kusumoto FM, *Cardiovascular Pathophysiology*, Hayes Barton Press, Raleigh, NC, 1999).

Ventricular depolarization

Activation of the His-Purkinje system leads to rapid spread of electrical activity throughout the ventricles, and the right and left ventricles are usually activated quickly and almost simultaneously. Simplistically, left ventricular activation can be considered to have two phases: first where the entire endocardium is activated as an "internal shell" by the termination points of the Purkinje fibers within a few milliseconds, followed immediately by endocardial to epicardial depolarization. Transmural depolarization occurs by means of cell-to-cell propagation via the gap junctions. In toto, ventricular activation leads to the largest signal on the ECG, called the QRS

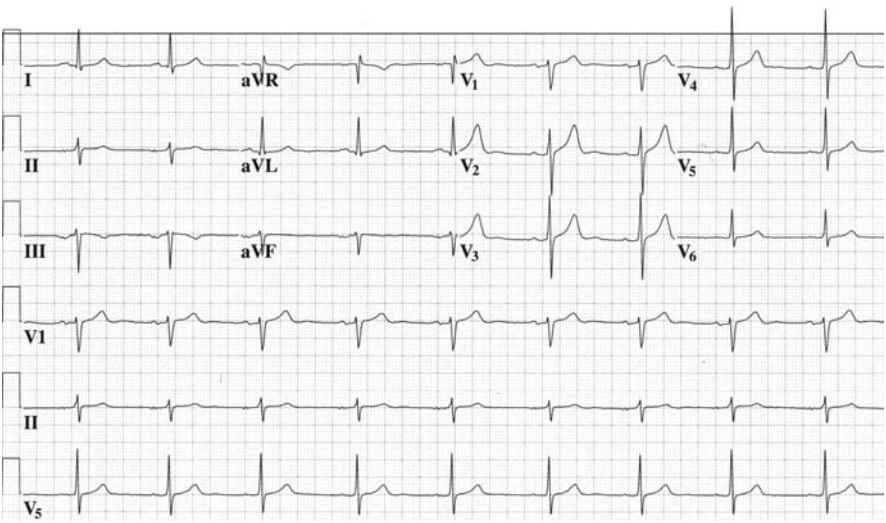


Figure 3: A normal 12-lead ECG from a young healthy man. Notice that the P wave is flatter in II. The lowest portion of the sinus node is "driving" the heart.

complex. The QRS complex is usually large (5–15 mV) and is complete within 0.1 s, or 100 ms. As discussed in detail later in this chapter, the ECG normally records at 25 mm/s, so that each small box (1mm) represents 0.04 s and the normal QRS complex is usually less than two and a half "little boxes."

ECG nomenclature for ventricular activation

The QRS complex can often be composed of several discrete signals. A standardized method for describing the positive and negative deflections has been developed to more accurately describe nuances within the QRS complex (Figure 4). The first negative deflection is called a "q" wave, the first positive deflection is called an "r" wave, the second negative deflection is called an "s" wave, and a second positive deflection is referred to as r'. Capital letters are used for larger deflections and lower case letters for smaller deflections. No specific amplitude is uniformly used to mark the transition between using lower case letters and capital letters. A completely positive QRS complex is referred to as an R wave and a completely negative complex is called a QS complex. The specific morphology of the QRS complex will depend on the relationship between the lead orientation and the pattern of ventricular activation.

Case study (continued): The QRS complexes in the frontal leads for Ms. Miera would be classified as I: R, II:R , III: R, aVR: QS, aVL: RS, and aVF: R.

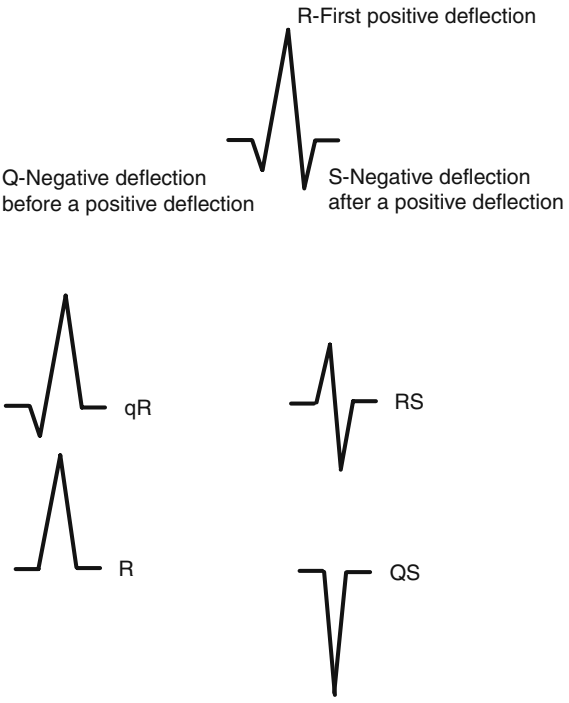


Figure 4: Nomenclature for ventricular activation (QRS complex) with some commonly observed waveforms.

ECG in the frontal plane

The left ventricle has a significantly larger mass than the right ventricle since the left ventricle normally pumps blood to the entire body while the right ventricle is only responsible for pumping blood to the lungs. Therefore the combined electrical voltages of right and left ventricular activation as measured by the ECG appear to travel from right to left. In addition, ventricular activation starts in the superior portion of the ventricles and cardiac activation is directed from superior to inferior. These two characteristics lead to cardiac activation that generally is directed from the right shoulder to the left hip. The general direction of ventricular activation in the frontal plane is called the cardiac axis (Figure 5). In the normal ECG the cardiac axis can range from $-30°$ to $110°$. This normal variation can be due to the varying position and orientation of the heart within the body. For example, thinner people tend to have a more vertical cardiac axis ($60°$–$110°$) because the ventricles are oriented more downward.

The frontal axis can be calculated in a number of ways. The first and easiest method is to look for the largest QRS complex in the frontal leads. The largest QRS complex will be observed in the general direction of the cardiac axis. If two lead

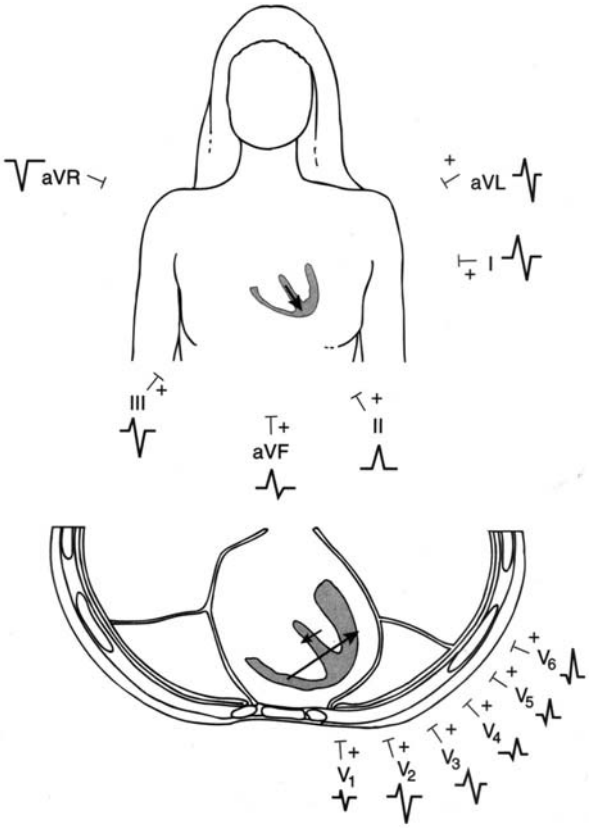


Figure 5: Normal activation of the heart. **Top**. In the frontal plane, activation of the ventricles can be approximated by a vector oriented at roughly 60°. For this reason a monophasic R wave is usually seen in lead II. **Bottom**. In the precordial plane, ventricular activation begins with depolarization of the septum from left to right, and then the ventricles are activated in a general right to left direction (since the left ventricle is larger than the right ventricle) (reprinted with permission from Kusumoto FM, *Cardiovascular Pathophysiology*, Hayes Barton Press, Raleigh, NC, 1999).

systems have similar amplitudes, the axis will be some intermediate value between the two vectors. Another method for calculating the axis is to find a QRS complex that is biphasic. This identifies a lead that is oriented perpendicular to the frontal axis, and the true axis can be calculated by measuring 90° from that lead towards the leads that are positive.

Case (continued): The QRS axis for Ms. Miera is approximately 60°. The largest QRS complex is recorded in lead II. Alternatively, the QRS axis could be estimated by noting the biphasic signal in aVL. The axis will be 90° from aVL. Both methods yield a value of 60° for the cardiac axis. This is a normal value.

ECG in the precordial plane

The septum is the first portion of the ventricles to be activated. Ventricular activation occurs via the left and right bundles; the left bundle splits off first and in most people initiates activation of the septum. Simultaneous activation of the left and right ventricles leads to a summed wave of activation that is directed from right to left (Figure 5). These two components of ventricular activation lead to a characteristic QRS pattern in the precordial leads. In lead V_1, initial septal activation leads to a small initial r wave and left ventricular activation leads to a large negative S wave (rS complex). Conversely, in lead V_6, a small q wave followed by a relatively large R wave will be observed (qR complex). Inspection of the precordial leads between V_1 and V_6 usually reveals a gradual increase in the amplitude of the R wave as the positive electrode becomes oriented more directly "in front" of ventricular activation.

Ventricular repolarization

Once the ventricles have depolarized, the ventricular myocytes are at their plateau phase and a generally isoelectric period is usually observed in the normal ECG. The interval between the QRS complex and the T wave is called the ST segment. As will be discussed in Chapter 8, small amounts of ST segment elevation can be observed, particularly in men. This deviation of the ST segment is due to small differences in cell voltages (magnitude and shape of the plateau phase, small differences in timing of depolarization and repolarization).

As myocytes repolarize, the T wave is observed. As will be described in Chapter 6, repolarization results in the complex interplay of several ion currents, and T wave changes can be very subtle due to small amplitude changes observed on the surface ECG. The T wave is generally smaller than the QRS complex for several reasons. First, repolarization occurs more gradually than depolarization. For ventricular activation, depolarization occurs from the sudden opening of Na^+ channels. Repolarization occurs with a gradual decrease in Na^+ and Ca^{2+} permeability and a gradual increase in K^+ permeability. The His-Purkinje system facilitates simultaneous depolarization of ventricular cells. In other words, the phase 0 s are "lined up." In contrast, there is significant heterogeneity of ventricular repolarization in different cell populations. For example, epicardial cells have a shorter action potential duration than endocardial cells due to different characteristics/populations of K^+ channels in each of these cell populations.

T waves are "flatter" than the QRS complex because cells repolarize at different times, and for a single cell the rate of repolarization is slower than depolarization.

Within the ventricular wall, depolarization of the ventricle usually occurs from endocardium to epicardium, since the terminal portions of the Purkinje fibers are usually located in endocardial tissue. Conversely, the general direction of

repolarization is from epicardium to endocardium because of the shorter action potential duration of epicardial cells. This difference in the direction of depolarization and repolarization leads to T waves that are generally in the same direction as the QRS complex (Figure 6).

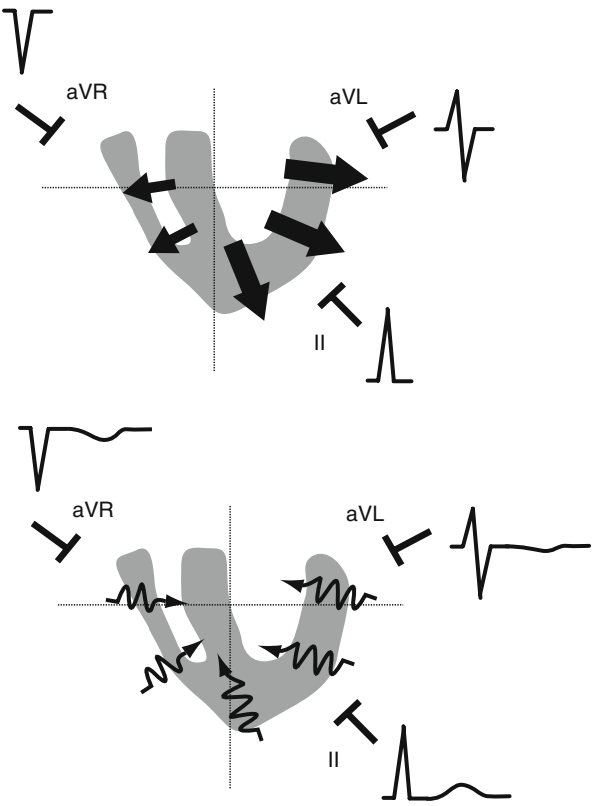


Figure 6: Depolarization (*top*) and repolarization (*bottom*) of the ventricles as observed in the frontal plane. Depolarization occurs almost simultaneously because of the His-Purkinje system. Depolarization occurs from endocardium to epicardium, and since the left ventricle has a larger mass than the right ventricle the overall direction of depolarization is from right to left and from the superior portion of the ventricles to the lower portion of the ventricles. In general this leads to an axis of approximately 60°, so that a large R wave is noted in lead II, an RS complex is observed in aVL (depolarization travels toward and then away from this lead), and a QS complex is noted in lead aVR. Depolarization occurs from epicardium to endocardium in a more gradual fashion. This leads to an upright T wave in II, a flat wave (slightly inverted in this example) in lead aVL, and an inverted T wave in aVR. Notice that since repolarization is generally in the opposite direction than depolarization, the QRS and T wave orientation is usually the same.

The normal direction of the T wave in the precordial leads generally follows the direction of the QRS complex, but in the anterior leads (V_2–V_4) an upright T wave can be observed even in the presence of a predominantly negative QRS complex.

Case (continued): Notice the relative morphologies of the QRS complexes and the T waves in Ms. Miera's ECG. In lead aVr, where a QS complex is observed, the T wave is inverted; and in lead II where the QRS is predominantly positive, the T wave is upright.

ECG intervals

Up to this point we have been focusing on the morphology of the electrical activity observed on the ECG. Another important data component is the temporal relationship of these ECG signals (Figure 7).

The usual paper speed for the ECG is 25 mm/s. ECG paper is usually divided into "large" boxes composed of five 1-mm "small" boxes. Since

$$1\,\text{s} = 25\,\text{mm},$$
$$1\,\text{mm} = 1/25\,\text{s} = 0.04\,\text{s}.$$

Each "small" box is 0.04 s and each "large" box is 0.20 s.

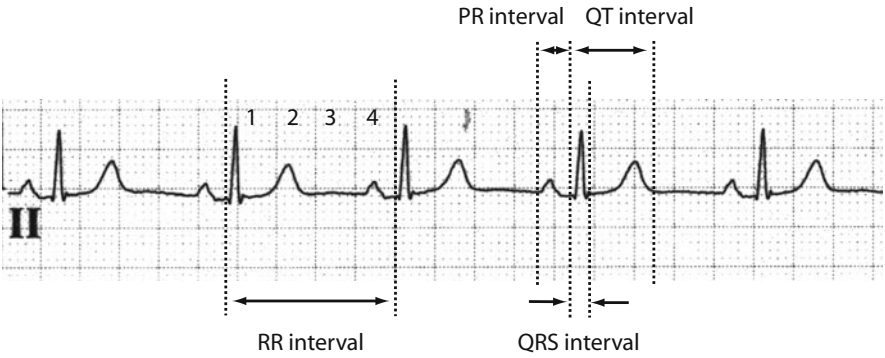


Figure 7: ECG intervals. There are four large boxes between the QRS complexes, so the heart rate is approximately 75 beats per minute (300/4). The PR interval is measured from the beginning of the P wave to the beginning of the QRS. The QRS interval is measured from the beginning of the QRS to the end of the QRS, and the QT interval is measured from the beginning of the QRS complex to the end of the T wave.

Heart rate

The rate of ventricular depolarization can be calculated by measuring the distance between each QRS complex. The approximate heart rate can quickly be calculated using the formula:

300/number of large boxes between QRS complexes = heart rate.

Normally the rate of atrial activation (P waves) is equal to the rate of QRS complexes.

PR interval

The PR interval is measured from the beginning of the P wave to the beginning of the QRS complex and indicates the time interval between the onset of atrial activation and the onset of ventricular activation. The normal PR interval in adults is less than 0.20 s. The normal PR interval in newborns and young children is less than adults, since the atrioventricular (AV node and His bundle) conduction system is physically smaller.

QRS interval

The QRS interval is measured from the beginning of the QRS complex to the end of the QRS complex. Physiologically the QRS interval represents the time between the first phase 0 of ventricular activation and the last phase 0 of ventricular activation. The normal QRS interval is less than 0.12 s, since ventricular activation occurs very rapidly via the His-Purkinje system. Again, the QRS interval is narrower in babies and children because of the smaller ventricular size.

QT interval

The QT interval is measured from the beginning of the QRS to the terminal portion of the T wave. It represents a global measure of the ventricular plateau phase duration from the first ventricular phase 0 to the last ventricular phase 3. The normal QT interval is longer in women than men and shortens with rate. A number of algorithms have been developed to account for rate-related QT interval changes. The usual method is Bazett's formula developed in the 1920s, where the corrected QT interval (QTc) is calculated by:

$$QTc = QT/(RR)^{1/2}$$

where RR is the interval between QRS complexes in seconds. An example of the effects of correcting the QT interval is demonstrated in Figure 8. The QT interval

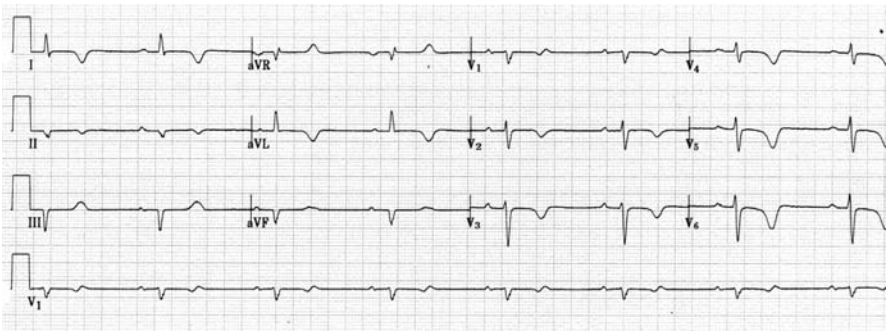


Figure 8: ECG with abnormal repolarization with inverted T waves. The QT interval is only mildly prolonged, once corrected for heart rate.

is approximately 0.55 s. Since the patient has a heart rate of approximately 45 beats per minute, the R-R interval is 1.3 s. The QTc would be $0.55/(1.3)^{1/2}$ or approximately 0.48 s. Bazett's formula has been criticized because it tends to provide an inappropriately short QTc at slow rates and inappropriately long QTc at higher rates. Several competing methods have been developed:

- Friderica: $\mathrm{QTc} = \mathrm{QT}/(\mathrm{RR})^{1/3}$
- Framingham: $\mathrm{QTc} = \mathrm{QT} + 0.154(1 - \mathrm{RR})$
- Hodges: $\mathrm{QTc} = \mathrm{QT} + 105(1/\mathrm{RR} - 1)$

None of the formulas has been shown to be clearly superior; so despite its obvious shortcomings, Bazett's correction is used for automated analysis and large clinical studies. The QT interval can be clinically important. In particular, patients with prolonged QT intervals can be susceptible to life-threatening ventricular arrhythmias (Chapter 6).

Case (continued): Ms. Miera has normal PR interval: 0.18 s, normal QRS duration: 0.09 s, normal QT interval: 0.40 s. Her ECG is normal, and if the rest of her history and physical are normal, Ms. Viera should be cleared for sports.

Summary

The electrical activity from the heart can be measured from the surface using the ECG. The normal ECG is characterized by a P wave from atrial activation, an isoelectric period associated with activation of the AV node and the His bundle/Purkinje system, and a QRS complex from ventricular activation. The axis represents general activation of the ventricles in the frontal plane and varies from 0° to 90°. The precordial QRS complex is usually recorded as an rS complex in lead V_1 and a qR complex in lead V_6. The T wave represents ventricular repolarization. The temporal relationship of cardiac activity is usually defined by the heart rate, PR interval, QRS duration, and QT interval.

Key points

1. Depolarization and repolarization of cardiac cells in different chambers of the heart can be recorded from the surface by the ECG.
2. Atrial depolarization is represented by the P wave, ventricular depolarization by the QRS complex, and AV node and His bundle activation by the isoelectric period between these two waves.
3. The general direction of ventricular activation in the frontal plane is called the axis.
4. The temporal relationship of atrial and ventricular activation can be estimated by the PR interval, the interval between initial and terminal ventricular activation can be measured via the QRS duration, and the global action potential duration of the ventricle can be measured by the QT interval.

Review questions

1. What is the axis of the ECG in Figure 9?

   A. 0°
   B. 60°
   C. 90°
   D. 120°

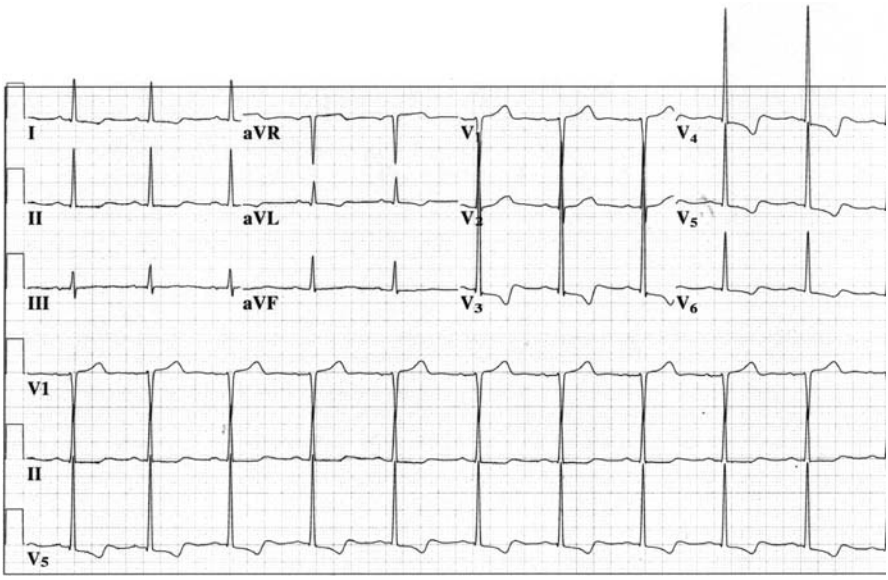


Figure 9: ECG for problems 1 through 4.

2. How would the QRS complex in lead aVL be described in Figure 9?

   A. qR
   B. rS
   C. QS
   D. R

3. The PR interval for the ECG in Figure 9 is:

   A. 0.02 s
   B. 0.16 s
   C. 0.22 s
   D. 0.26 s

4. Ventricular repolarization for the ECG in Figure 9 is:

   A. Normal
   B. Abnormal

Answers

1. B. The axis is approximately 60°; notice that the largest QRS complex is recorded in lead II. The axis is actually closer to 40° since aVL is predominantly positive rather than biphasic. In truth, there is very little use in calculating the exact value for the axis. The main issue is to identify those patients with an abnormal axis (more leftward than −30° to −45° or more rightward than 110°). Causes for an abnormal axis will be covered in greater detail in Chapters 4 and 5.
2. A. The QRS complex in aVL has a qR morphology: small negative wave with a subsequent larger R wave.
3. C. The PR interval is approximately 0.22 s—approximately four and a half "little boxes." Since each "little box" is 0.04 s wide, 0.04*4.5 = 0.22 s. This is an example of a slightly prolonged PR interval that represents slightly delayed conduction within the AV node.
4. B. Repolarization is abnormal. Notice that the T waves are inverted in leads I, L, V_3–V_6. In addition the ST segment is downsloping rather than isoelectric. This patient has left ventricular hypertrophy. Refer back to this ECG after reading Chapter 4.

Part II

Abnormal depolarization

Chapter 4
Chamber enlargement

The twelve-lead ECG has been the traditional tool for the identification of hypertrophy or enlargement of the cardiac chambers. Although this role has been supplanted in part by direct imaging modalities such as echocardiography, the ECG remains a valuable tool for identifying structural abnormalities of the heart and still provides a simple screening tool for important prognostic and clinical information.

Case study: Mr. Vincent Gore is a 67-year-old man that has not been medically evaluated for many years. He is not on any medications. On physical examination he is noted to have a blood pressure of 176/88 mm Hg. His ECG is shown in Figure 1.

Atrial enlargement

Atrial depolarization is represented electrocardiographically by the P wave. Since the atria are relatively small and thin-walled, the electrical activity generated by depolarization is small and the P wave is usually only 1–2 mm (0.1–0.2 mV) tall. The sinus node is located in the superior and lateral portion of the right atrium, so the initial portion of the P wave usually reflects right atrial activation and the terminal portion of the P wave is due to left atrial activation (Figure 2). Since the P wave is so small, it is not a sensitive indicator of atrial abnormalities.

A tall-peaked P wave has traditionally been used as a sign for right atrial enlargement, and in the presence of significant pulmonary disease this finding is often called P pulmonale. The usual criteria used for right atrial enlargement are a P wave > 2 mm in lead V_1 or a P wave > 2.5 mm in lead II. Unfortunately, the finding of tall P waves is nonspecific and can be seen by echocardiography in patients with left atrial enlargement or no atrial enlargement.

Since the left atrium is activated in the terminal portion of the P wave, a prominent terminal negative component (> 1 little box in duration and depth) in lead V_1 has been used as an indicator for left atrial abnormality. The term left atrial abnormality is preferred over left atrial enlargement because the finding can be observed in patients with intraatrial conduction delay due to slow conduction between the right atrium and the left atrium. Another finding suggestive of left atrial abnormality is a P wave longer than 0.12 s in duration.

F. Kusumoto, *ECG Interpretation: From Pathophysiology to Clinical Application*,
DOI 10.1007/978-0-387-88880-4_4,

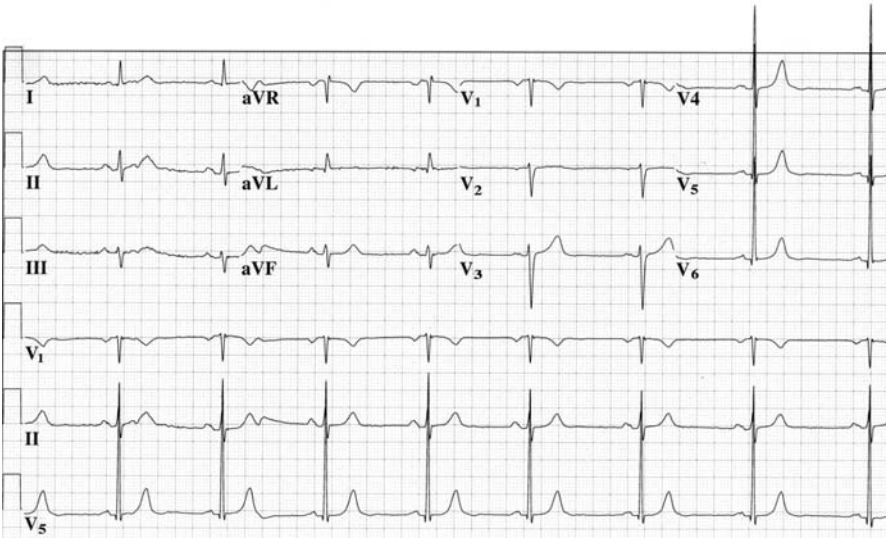


Figure 1: Mr. Gore's ECG.

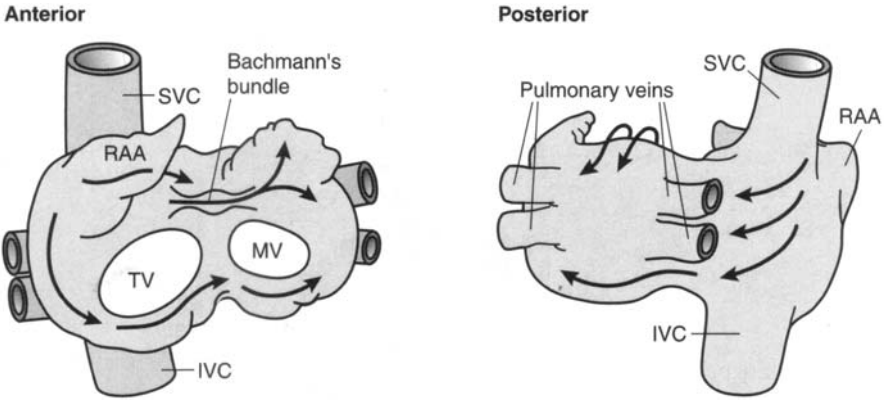


Figure 2: Schematic of normal atrial activation. Atrial activation starts at the sinus node near the right atrial-superior vena cava (SVC) junction. The right atrium is activated first, which is then followed by left atrial activation. For this reason, in general the initial part of the P wave reflects right atrial activation and the terminal portion of the P wave is due to left atrial activation (reprinted with permission from Kusumoto FM, *Cardiovascular Pathophysiology*, Hayes Barton Press, Raleigh, NC, 1999).

Left atrial abnormality: P wave > 0.12 s in duration

> *Terminal negative portion of the P wave in lead V_1 > 1 small box deep and wide (1 mm tall and 0.04 s wide).*

Case study (continued): The ECG shows left atrial abnormality. Notice that the terminal portion of the P wave is deeply inverted in V_1.

Ventricular enlargement

Left ventricle

Any condition that places an abnormal load on the left ventricle can lead to left ventricular hypertrophy. Increased intraventricular pressure from causes such as systemic hypertension or aortic stenosis can cause left ventricular enlargement or hypertrophy. Similarly, conditions associated with an increased volume load on the heart (mitral regurgitation, aortic regurgitation) can also lead to left ventricular enlargement.

Several ECG criteria have been developed to detect the presence of left ventricle hypertrophy. Since the QRS complex becomes larger and more prominent as the left ventricle becomes larger and thicker, most of the criteria for left ventricular hypertrophy use some cut-off for QRS size (voltage criteria) (Figure 3). In addition, some methods evaluate whether conditions associated with left ventricular hypertrophy, such as left atrial abnormality or abnormal left ventricular repolarization, are present.

Left ventricular hypertrophy is associated with:

1. *Larger and slightly wider QRS complexes: more left ventricular tissue to depolarize.*
2. *Left atrial abnormality: thicker and more vigorous left atrial contraction required to fill the thicker and stiffer left ventricle.*
3. *ST segment and T wave changes: thick left ventricle repolarizes abnormally.*

Voltage criteria

Although there is a loose correlation between ventricular (QRS) voltage and ventricular hypertrophy, it is important to remember that normal QRS voltages will vary with age and gender. In general, normal ventricular voltages are highest at adolescence and then decrease with age, reaching a final plateau at 50 years old. Normal voltages are approximately 10% lower in women in all age groups. Normal voltages vary by ethnicity, with highest voltages in Africans and lowest reported voltages in Chinese. Finally, normal QRS voltage is also inversely proportional to body mass index.

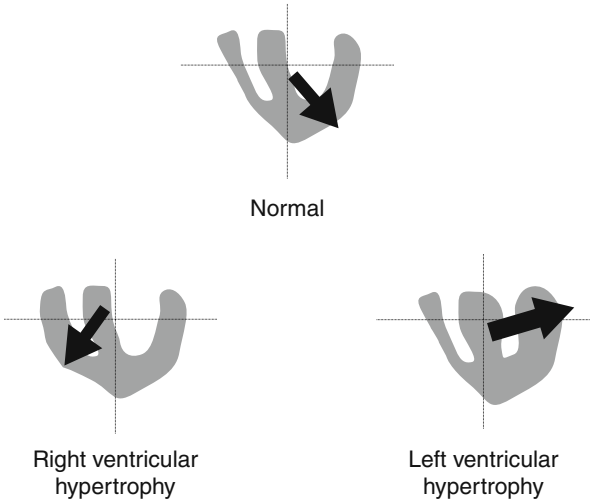


Figure 3: ECG depolarization forces in left ventricular hypertrophy and right ventricular hypertrophy. Normally, the heart is activated from right to left and from superior to inferior. This leads to a frontal QRS axis of approximately 60° and a QRS with an S wave in V_1 and an R wave in V_6. With left ventricular hypertrophy the frontal axis tends to shift leftward, and the S wave in V_1 and the R wave in V_6 become deeper and taller respectively. In contrast with right ventricular hypertrophy, the frontal axis shifts rightward and an R wave will be observed in V_1 and an S wave in V_6.

There are several voltage criteria for left ventricular hypertrophy. The most well known were developed by Sokolow and Lyon in the late 1940s. They used the sum of the S wave in lead V_1 and the R wave in V_5 or V_6 to determine whether left ventricular hypertrophy was present (Figure 4). If this sum is greater than 35 mm, then left ventricular hypertrophy is present. The R wave in aVL can also be used, since left ventricular hypertrophy is associated with a more leftward shift of the frontal plane axis (Figure 3). If the R wave in aVL is $>$ 11 mm, left ventricular hypertrophy is present. As shown in Figure 3, with left ventricular hypertrophy the frontal axis shifts leftward; and in the precordial plane, activation is directed more posteriorly (since the left ventricle lies behind the right ventricle). It is this shift in the direction of the depolarization axis that leads to prominent R waves in the lateral leads (aVL, V_5, and V_6) and the S wave in the rightward anterior leads. Notice that in Figure 4, while the precordial voltage criteria for left ventricular hypertrophy is satisfied, the voltage in aVL does not meet voltage criteria. However, only one criterion must be satisfied to make the diagnosis of left ventricular hypertrophy. The Sokolow criteria have a low specificity in younger patients and a sensitivity of only 20–30%.

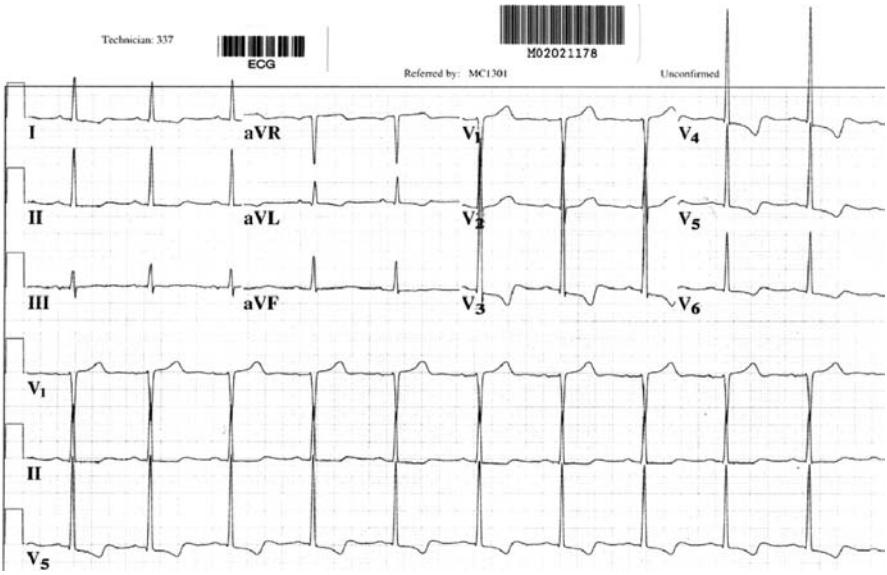


Figure 4: An ECG from a man with a longstanding history of systemic hypertension with severe left ventricular hypertrophy confirmed by echocardiography. Some but not all of the criteria for left ventricular hypertrophy are present. Notice that the sum of the S wave in V_1 and the R wave in V_5 is greater than 35 mm and the repolarization changes in V_5,V_6, (Romhilt-Estes score: 4 (ST changes and QRS $>$ 0.09 s, V_4 meets voltage criteria but V_5 and V_6 do not). The ECG does not meet left ventricular hypertrophy criteria using aVL voltage or Cornell voltage criteria.

Another commonly used voltage criterion for left ventricular hypertrophy was developed by a group at Cornell University: the sum of the R wave in aVL and the S wave in $V_3 > 28$ mm in men and > 20 mm in women. This was the first large study to identify specific criteria for left ventricular hypertrophy in women. The ECG in Figure 4 would not meet the Cornell criteria for left ventricular hypertrophy because of the early transition in the precordial leads (an R wave in lead V_3).

Repolarization changes

In the late 1960s Romhilt and Estes recognized that left ventricular hypertrophy was associated with ST segment and T wave changes. As discussed earlier, depolarization of the ventricle usually occurs from endocardium to epicardium, while the direction of repolarization is reversed, from epicardium to endocardium, due to the shorter action potential duration of epicardial myocytes (Figure 5). However, in patients with left ventricular hypertrophy the direction of repolarization can be reversed, due perhaps to slower depolarization and subsequent changes in the

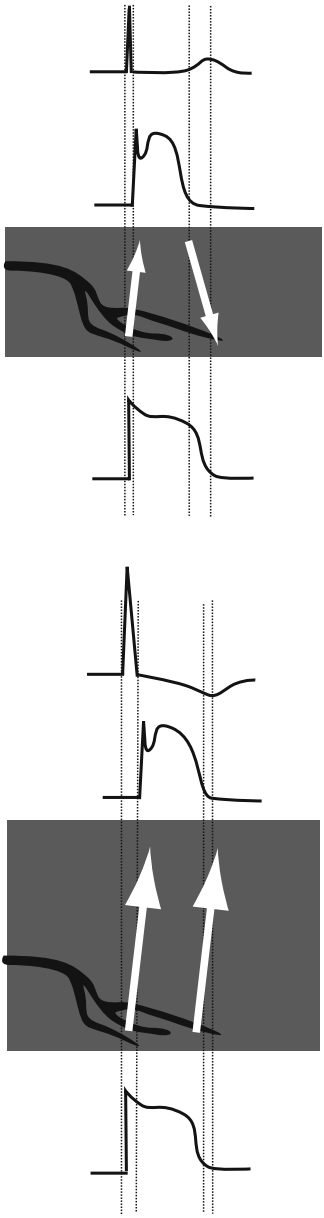


Figure 5: Under normal conditions, the general direction of repolarization is opposite the direction of depolarization (*top*). In left ventricular hypertrophy, thickening of the myocardial wall leads to increased voltage and widening of the QRS complex, and the general direction of repolarization can reverse, leading to ST segment changes and T wave inversion.

sequence of repolarization. Repolarization from endocardium to epicardium leads to inverted T waves, and increased heterogeneity in repolarization leads to downsloping ST segments (Figure 5). Importantly, several studies have shown that the presence of repolarization abnormalities is associated with poorer prognosis.

The Romhilt-Estes method uses a point system for the diagnosis of left ventricular hypertrophy (Table 1). If an ECG has 4 points, left ventricular hypertrophy is suspected, and left ventricular hypertrophy is present if ≥ 5 points are present. In day-to-day clinical practice it is difficult to remember the exact points for each criteria, but it is clinically useful to note that if both left atrial abnormality and repolarization changes (6 points total) are present, left ventricular hypertrophy criteria are met regardless of voltage.

Other/Composite criteria

The Cornell group added an additional criterion, the QRS duration, to calculate the "Cornell Product." Left ventricular hypertrophy is associated with increased total time of depolarization (Figure 5). The Cornell product is calculated by multiplying the Cornell voltage (R wave in aVL + S V_3) and the QRS duration; a value > 24.4 mm/s has been used as the cut-off value for left ventricular hypertrophy. It may be that the Cornell product correlates with the severity of left ventricular hypertrophy; larger values are associated with more severe left ventricular hypertrophy. Remember that all of the previously described criteria provide binary information—is left ventricular hypertrophy present or not? However, serial measurements of the Cornell product have been used as a surrogate for the progression of left ventricular hypertrophy in several studies.

Table 1: Romhilt-Estes scoring system for left ventricular hypertrophy

Criteria	Points
Any limb-lead R or S $>$ 20 mm, or an S wave in V_1 or V_2 deeper than 30 mm, or an R wave in V_5 or V_6 taller than 30 mm	3
ST-T typical of LVH	
no digitalis	3
with digitalis	1
Left atrial involvement (terminal negative P wave in lead V_1 wider than 0.04 s and deeper than 0.1 mV (1 "little box" wide and deep)	3
Left axis deviation $> 30°$	2
QRS duration > 90 ms	1
Intrinsicoid deflection in V_5 or $V_6 \geq 50$ ms	1

5 points: Definite left ventricular hypertrophy; 4 points: Probable left ventricular hypertrophy

Commonly used criteria for left ventricular hypertrophy:

1. *R wave in aVL > 11 mm*
2. *Sum of the S wave in V_1 and R wave in V_5 or V_6 > 35 mm*
3. *Sum of the R wave in aVL and the S wave in V_3 > 20 mm in women and > 28 mm in men*
4. *Romhilt-Estes point system: left atrial abnormality and repolarization abnormalities*

The availability of echocardiography has allowed evaluation of all of the ECG criteria against a "gold standard." Unfortunately, all of the ECG criteria for left ventricular hypertrophy have shortcomings, and although they are all relatively specific they are fairly insensitive markers (Table 2). In other words, if the ECG finding is present the patient probably has left ventricular hypertrophy, with most false positive rates < 10%. However, the ECG markers will identify only a relatively small proportion (11–70%) of patients with left ventricular hypertrophy.

Right ventricle

Several criteria have been developed for detection of right ventricular hypertrophy. Since anatomically the right ventricle is located in the "front" part of the heart, right ventricular hypertrophy can be associated with an increased relative electrical force directed anteriorly and to the right. This leads to a relatively large R wave in lead V_1 (Figure 3).

There are several voltage criteria that have been developed for the diagnosis of right ventricular hypertrophy that take advantage of this change in the direction of depolarization. In the frontal plane the axis is shifted rightward (Figure 6). In the precordial leads an R wave in V_1 > 7 mm or an R wave that is greater than twice the depth of the S wave in V_1 have been used as criteria for the presence of right ventricular hypertrophy. Similarly, an S wave that is larger than the R wave in V_6 has also been used as a criterion for right ventricular hypertrophy.

Just as left ventricular hypertrophy can be associated with repolarization abnormalities, right ventricular hypertrophy can cause changes in the ST segment and T

Table 2: Sensitivity and specificity of ECG criteria for left ventricular hypertrophy

Criteria	Sensitivity (%)	Specificity (%) false positives
aVL R > 11 mm	11–20	0–2
S V1 + R V_5 or V_6 > 35 mm	40–50	5–8
R aVL + S V_3 > 20 mm (women), 28 mm (men)	15–40	2–9
Cornell product	11–30	3–17
Romhilt-Estes	10–70	6–11

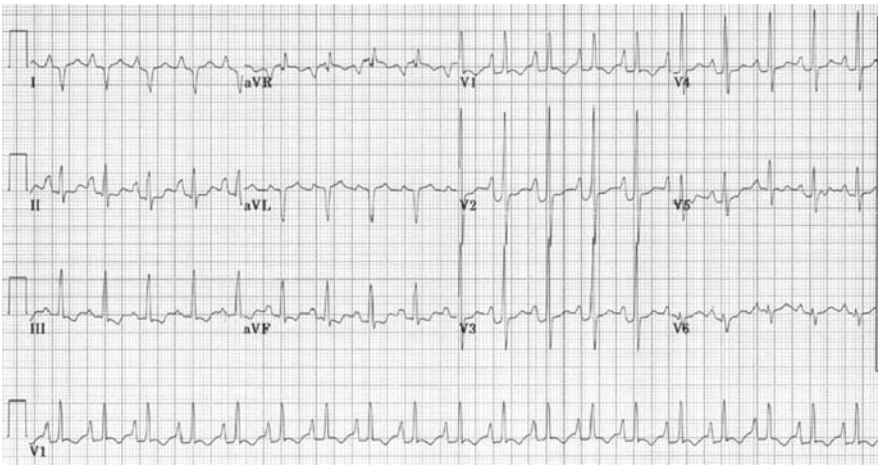


Figure 6: ECG in right ventricular hypertrophy (RVH). In the frontal plane, since the R wave in lead III is more prominent than the R wave in aVF, right axis deviation is present (approximately 120°). In the precordial plane, rightward and anteriorly directed depolarization (since the right ventricle is located just under the sternum) leads to a prominent R wave in V_1 and a deep S wave in V_6. The ECG meets criteria for RVH since the R wave in V_1 is greater than 7 mm. In V_6, although the S wave voltage does not meet criteria for RVH, the morphology (S wave deeper than the R wave height) is characteristic of RVH. The prominent R wave in V_1 is associated with ST depression (repolarization abnormality). Finally, notice that the P wave is "tall and peaked."

wave. Downsloping ST segments and T wave inversion can be observed in right ventricular hypertrophy.

Unfortunately, the ECG criteria for right ventricular hypertrophy are relatively specific (approximately 90%) but are very insensitive (2–20%).

Right ventricular hypertrophy:

- *Right axis deviation* $> 110°$
- *R wave in* $V_1 > 7mm$
- *S wave in* V_5 *or* $V_6 > 7\,mm$
- *R wave more than twice the S wave in* V_1
- *S wave larger than the R wave in* V_6

Case study (continued): Notice the large voltages in lead V_1 *and* V_5*. Notice the ST segment changes and T wave inversion. This is an example of left ventricular hypertrophy. Unfortunately, the presence of left ventricular hypertrophy and associated ST segment changes has important prognostic information and is associated with a 50% increased risk of myocardial infarction or cardiovascular death.*

Key points

1. In general, the ECG diagnosis for chamber hypertrophy is dependent on longer and larger depolarization: broad or large P waves for atrial enlargement, and broad and large QRS complexes for ventricular hypertrophy.
2. Criteria for left atrial abnormality/enlargement include: terminal negative portion of the P wave in lead V_1 greater than 1 mm deep and 0.04 ms in duration; notched P wave with a duration > 0.10 s (2.5 little boxes).
3. Left ventricular hypertrophy is associated with increased QRS voltages, left axis deviation, changes in repolarization, and left atrial abnormality.
4. Right ventricular hypertrophy is more difficult to classify by ECG criteria but such criteria include increased voltage in lead V_1, right axis deviation, and repolarization changes in leads V_1 and V_2.

Questions

1. The ECG in Figure 7 suggests what abnormality

   A. None, this is a normal ECG
   B. Borderline left ventricular hypertrophy
   C. Severe left ventricular hypertrophy
   D. Right ventricular hypertrophy.

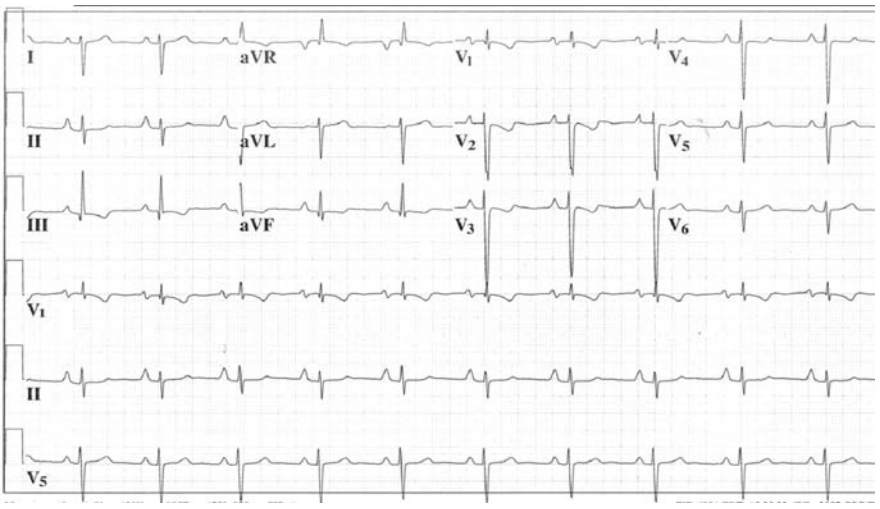


Figure 7: ECG for Problems 1 and 2.

2. What ECG findings support this diagnosis?
3. In a patient with left ventricular hypertrophy the QRS complex will generally:

   A. Be shorter in duration due to more profuse development of the His-Purkinje system.
   B. Be longer due to larger mass of ventricular tissue.
   C. Be unchanged due to minimal histologic changes.

4. The ECG in Figure 8 shows:

   A. Nothing this is a normal ECG.
   B. Borderline left ventricular hypertrophy.
   C. Severe left ventricular hypertrophy.
   D. Right ventricular hypertrophy.

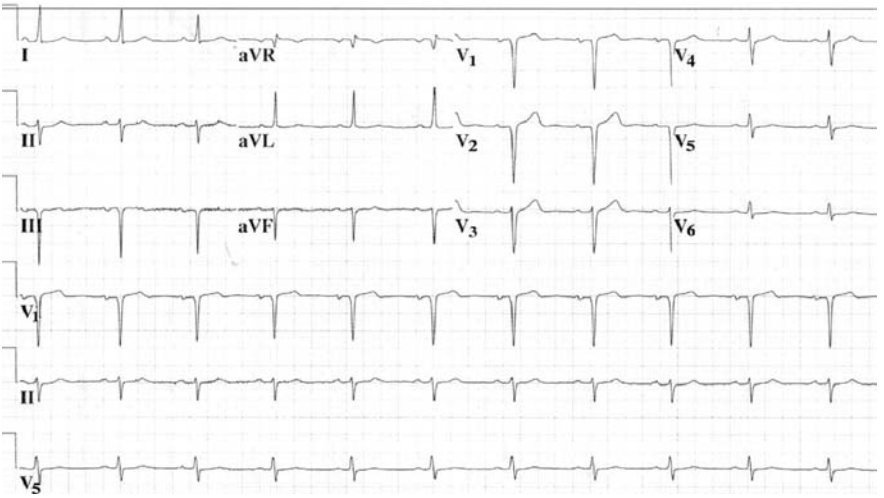


Figure 8: ECG for Problem 4.

Answers

1. The correct answer is D. The ECG shows a prominent R wave in V_1 and a deep S wave in V_6. Right axis deviation is present along with ST-T changes consistent with right ventricular hypertrophy (ST segment depression and an inverted T wave in V_1).
2. See above answer.
3. The correct answer is B. Left ventricular hypertrophy is generally associated with a wider QRS complex, since there is a greater mass of ventricular tissue to be depolarized.
4. The correct answer is B. The patient has a single criterion (aVL $>$ 11 mm) for left ventricular hypertrophy. No other voltage criteria for left ventricular hypertrophy are present.

Chapter 5
Conduction abnormalities in the His-Purkinje tissue

The ventricles are activated by the left and right bundles and the Purkinje system. The rapid conduction velocity of these specialized myocytes leads to almost simultaneous activation of the entire myocardium, which in turn leads to a narrow QRS complex recorded by the ECG (usually less than 0.10 s). However, abnormalities of the bundles will lead to sequential activation of the ventricles that in turn will cause a wide QRS complex. The left bundle is composed of two major divisions: anterior and posterior. Abnormal conduction in one of these divisions or fascicles of the left bundle will not necessarily lead to a wide QRS complex but will cause characteristic QRS patterns on the ECG. The recognition of different types of conduction abnormalities in the bundle branches and fascicles will be the subject of this chapter.

Case Study: Introduction

Ms. Scott is an eighty-five-year-old woman who comes to your office for a routine physical examination. She has no significant medical history, and her physical examination is unremarkable. Her ECG is shown in Figure 1. What ECG abnormality is present? Should any further diagnostic testing be recommended on the basis of this ECG?

Anatomy/electrophysiology

As reviewed in Chapter 1, after the cardiac impulse leaves the AV node it activates the His bundle that penetrates the fibrous annulus that separates the atria from the ventricles. The His bundle bifurcates into two branches: the right bundle and the left bundle. The left bundle separates into a broad band of fibers that spreads out as a large "fan." Traditionally, the left bundle is divided into a left posterior fascicle and a left anterior fascicle. As discussed below, while this provides a useful electrocardiographic classification, the reader should always keep in mind that the classification is based on electrocardiographic criteria and that on actual anatomic dissection the left bundle has a more diffuse, fan-like appearance. The right bundle is a relatively slender structure (approximately 1 mm in diameter) that forms as the natural extension of the His bundle. The anatomy of this region can be visualized by

F. Kusumoto, *ECG Interpretation: From Pathophysiology to Clinical Application*,
DOI 10.1007/978-0-387-88880-4_5, © Springer Science+Business Media, LLC 2009

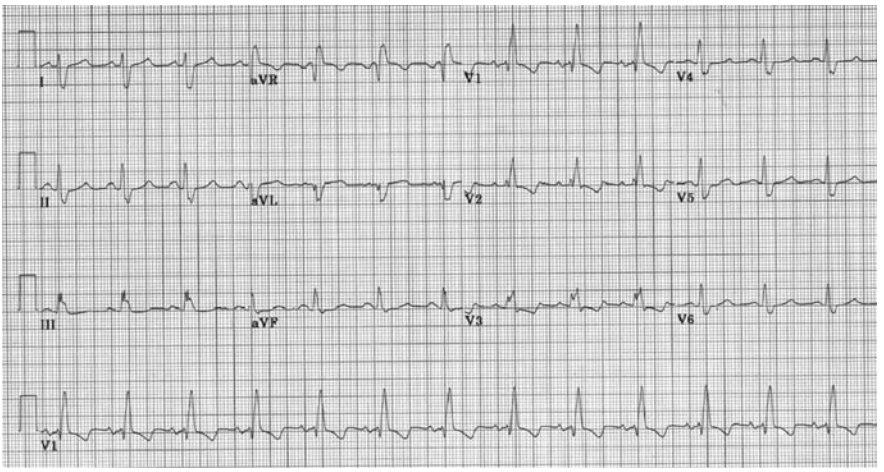


Figure 1: ECG from Ms. Scott.

using your left hand. Place your hand in the position shown in Figure 2. Your wrist represents the His bundle penetrating the fibrous annulus. The palm of your hand represents the left bundle which then divides into a diffuse fan (spread fingers) that are divided into the posterior fascicle (fourth and fifth fingers) and anterior fascicle (second and third fingers). The thumb represents the right bundle. When viewed from above it can be seen that the left bundle splits off first (this is why the septum is usually activated from left to right) and that the right bundle is the continuation of the His bundle. Using the "hand model" also emphasizes the important point that the left bundle is significantly larger than the right bundle.

Right bundle branch block

Blocked conduction in the right bundle causes the right ventricle to be activated abnormally late. Normally, the septum is activated first from left to right, and then the left and right ventricles are activated simultaneously (Figure 4, Chapter 3). Remember from our prior discussion that after septal activation the general activation direction is right to left because of the larger left ventricular mass. The ventricular activation pattern in the right bundle branch block is shown in Figure 4. Since the left bundle activates the septum, in the right bundle branch block septal activation remains normal. Left to right septal activation leads to a small initial positive deflection (r wave) in lead V_1 and a small initial negative wave (q wave) in lead V_6. Left ventricular activation also occurs normally via the left bundle, and a large negative deflection in lead V_1 and a large positive deflection in lead V_6 are recoded on the ECG. However, right ventricular activation is significantly delayed, which leads to a large terminal positive wave (R wave) in lead V_1 and a large terminal negative wave

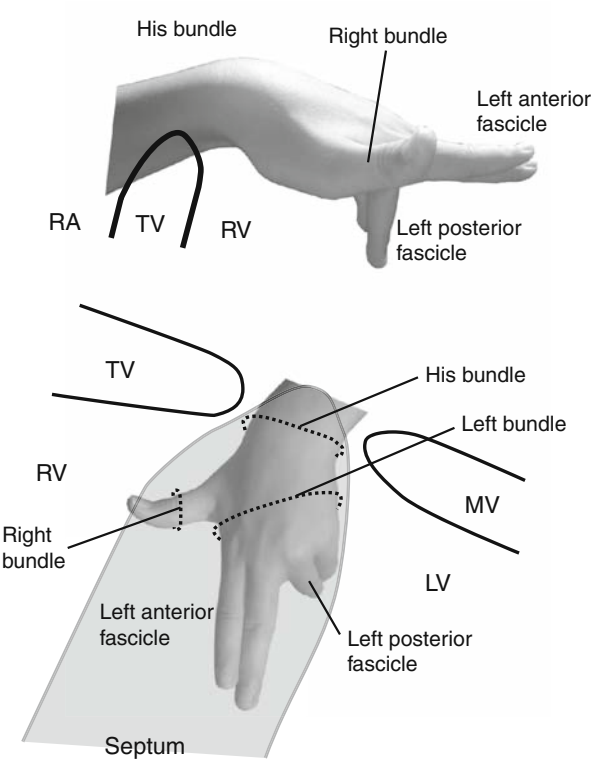


Figure 2: Using the left hand as a model for the bundles from the right atrial and right ventricular side (*top*) or from above (*bottom*). The His bundle provides the only conduction axis from the atria to the ventricles. Notice that the left bundle branch is significantly thicker than the right bundle branch, and that the His bundle and right bundle travel along a similar axis. The left bundle splits into a longer left anterior fascicle and a shorter posterior fascicle. TV: tricuspid valve; MV: mitral valve; RA: right atrium; RV: right ventricle; LV: left ventricle.

(S wave) in lead V_6. In this case, a relatively large deflection due to right ventricular activation is observed on the surface ECG since it occurs late and is "unopposed" by left ventricular activation.

In the right bundle branch block:

1. *QRS duration >0.12 s*
2. *V_1: rSR' complex*
3. *V_6: qRS complex*

It is important to remember that a right bundle branch block pattern on an electrocardiogram does not necessarily mean that conduction through the bundle is blocked. If the conduction velocity of the right bundle branch is sufficiently delayed

to cause relatively late activation of the right ventricle, a right bundle branch block "pattern" will be seen on the ECG.

Right bundle branch block is a relatively common electrocardiographic finding, particularly in older people. The incidence of right bundle branch block is approximately 1:1000 in people under 30 years old and increases to 2–3:1000 in people 30 to 40 years old. In general, the presence of right bundle branch block does not predict an increased future incidence for heart disease. This finding is not unexpected, because the right bundle branch is such a slender structure (remember it is represented by the thumb in our "hand model"). A relatively localized lesion (due to fibrosis, mechanical trauma, etc.) can potentially cause right bundle branch block. This is an important issue in one clinical situation. Patients with left bundle branch block (see below) may be activating their ventricles solely via the right bundle. In some clinical situations a catheter is placed in the pulmonary artery, via a central vein. As the catheter passes through the right ventricle, complete heart block can precipitously occur if the right bundle is traumatized.

Case study (continued):

Ms. Scott has a right bundle branch block pattern on the ECG. Again, remember that we cannot distinguish between conduction "block" and conduction "delay." Regardless, in the absence of accompanying symptoms and the normal physical examination, no further evaluation is necessary. Ms. Scott continues to do well and a follow-up ECG is obtained three years later (Figure 3). What changes are present on the more recent ECG?

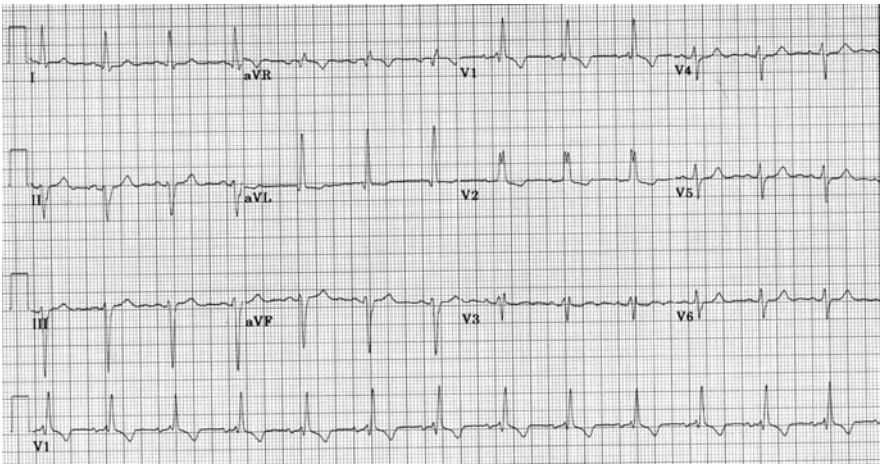


Figure 3: Ms. Scott's follow-up ECG.

Left bundle branch block

In left bundle branch block, the septum is not activated normally. The septum is activated from right to left, and the left ventricle is activated late (Figure 4). In lead V_1 a wide negative QS complex is seen, while in lead V_6 a wide positive (R wave) signal is recorded. The frontal plane leads I and aVL have a similar orientation as V_6, so wide positive QRS complexes are also seen in these leads. Figure 5 shows an example of a patient with left bundle branch block. It is important to remember that any bundle branch block can be intermittent. In Figure 6, the patient develops intermittent conduction over the left bundle, so that beats with a normal QRS morphology alternate with beats with a left bundle branch morphology.

Since the left bundle is larger and involves a more generalized area than the right bundle (palm vs. thumb in the "hand model" shown in Figure 2), a left bundle branch block pattern on the ECG suggests a more extensive myocardial abnormality. Unlike right bundle branch block, left bundle branch block is rarely seen in patients

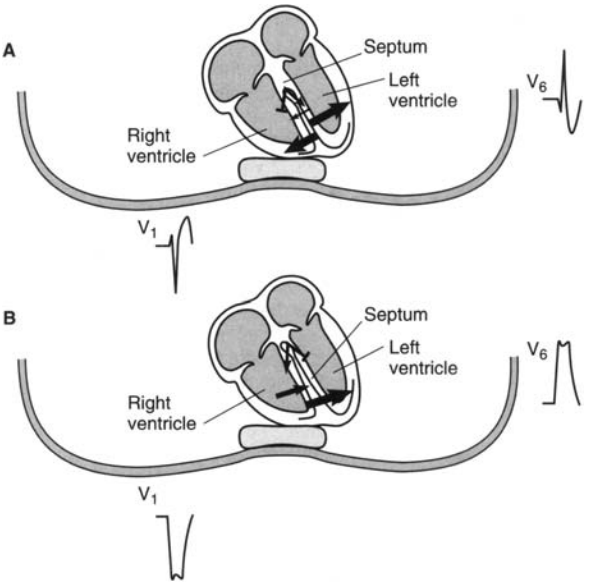


Figure 4: ECG in left and right bundle branch block. **A**. In right bundle branch block left to right septal activation is normal since the left bundle branch depolarizes normally. Depolarization of the left ventricle yields a right to left wave of activation. Finally the right ventricle is activated late from left to right. In V_1 an rSR' complex is recorded and in V_6 a qRS complex is seen. **B**. In left bundle branch block, right to left septal activation and late right to left activation of the left ventricle leads to a wide negative complex in V_1 and a wide positive complex in V_6 (reprinted with permission from Kusumoto FM, *Cardiovascular Pathophysiology*, Hayes Barton Press, Raleigh, NC, 1999).

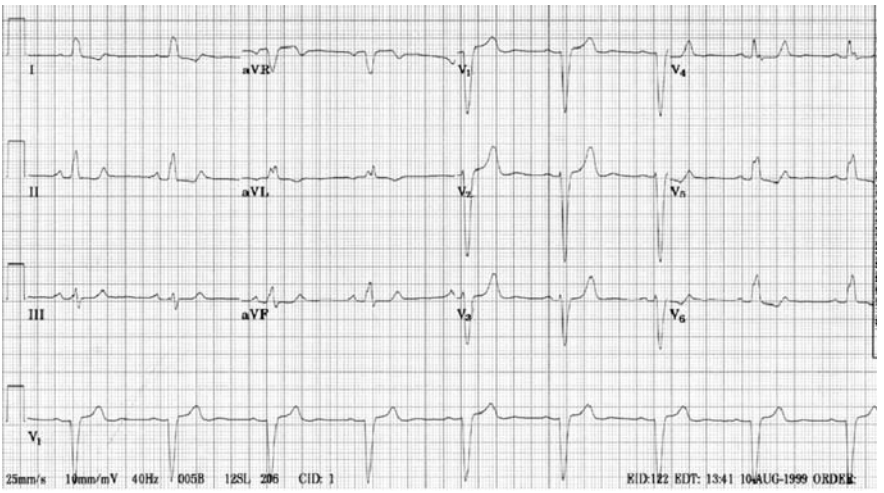


Figure 5: ECG in left bundle branch block. Since both the septum and lateral wall of the left ventricle are activated from left to right a predominantly negative and wide QS complex is recorded in V_1 and a wide monophasic R wave is observed in the most lateral leads (V_6, I, and aVL).

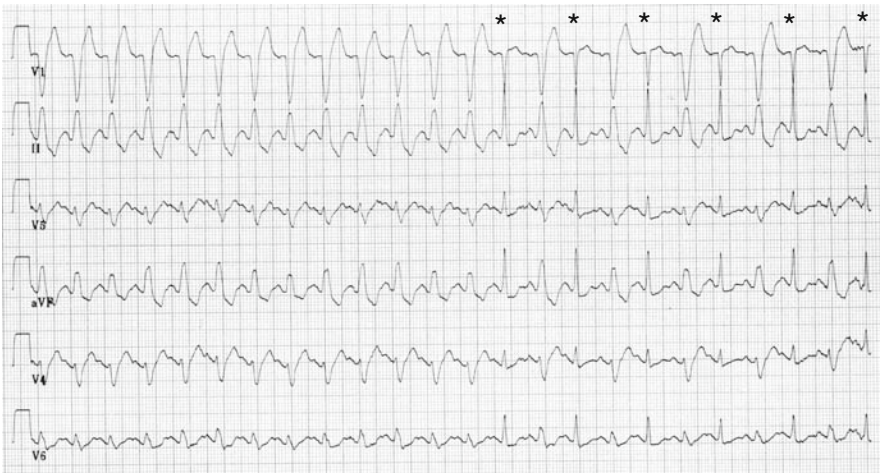


Figure 6: Intermittent left bundle branch block. The left side of the ECG shows left bundle branch block (wide negative QRS complex in V_1). On the right side of the ECG the patient develops intermittent left bundle branch block with normal conduction on alternate beats ($*$).

without some type of heart disease. The most common causes for left bundle branch block are coronary artery disease, hypertensive heart disease, or dilated cardiomyopathy (a condition where the cardiac myocytes do not contract normally; it can be genetic or occur after an infection). Left bundle branch block can also be seen as an isolated finding due to progressive sclerosis and calcification of the bundle with associated fiber loss. This condition is more commonly observed in older patients and is referred to as Lev's disease if the fiber loss is mostly proximal and Lenegre's disease if the sclerosis is present in the more peripheral portions of the left bundle.

In left bundle branch block:

1. *QRS duration >0.12 s*
2. V_1: *negative complex*
3. V_6: *positive complex*

Left anterior fascicular block

Since the left bundle is a large structure, conduction block in a portion of the left bundle can develop and produce characteristic ECG patterns. Partial block of the left bundle is classified electrocardiographically as left anterior fascicular block or left posterior fascicular block.

In left anterior fascicular block the more anterior fibers of the left bundle branch have slowed or blocked conduction, which leads to relatively late activation of the anterior portion of the heart. Think back to the hand model. Initial activation of the left ventricle occurs via the left posterior fascicle (ring and little fingers) that is followed by inferior to superior activation of the rest of the left ventricle (Figure 7). On the electrocardiogram the major finding associated with left anterior fascicular block is left axis deviation in the frontal axis (superior to $-30°$ to $-45°$). In the frontal plane, block of the left anterior fascicle leads to activation of the posterior and inferior portions of the left ventricle first, with later activation of the anterior portion of the heart (via regular myocytes rather than the His-Purkinje system). This leads to a slightly prolonged QRS width, an initial downward force followed by a later superior force (Figure 8). The overall axis of the heart is rotated upward because of the late activation of the anterior portion of the heart. Several authors have argued whether $-35°$ or $-45°$ should be used as the lower cut-off for the diagnosis of left anterior fascicular block; both values are rather arbitrary cut-offs, although the author generally uses $-45°$. Left anterior fascicular block is one of several reasons for left axis deviation. Besides the left axis deviation, the initial activation of the inferior and posterior portions of the left ventricle frequently leads to small q waves in the lateral leads (I and aVL) and small r waves in the inferior leads (II, III, and aVF). An example of an electrocardiogram with left anterior fascicular block is seen in Figure 8. In the precordial leads the late activation of the anterior portion

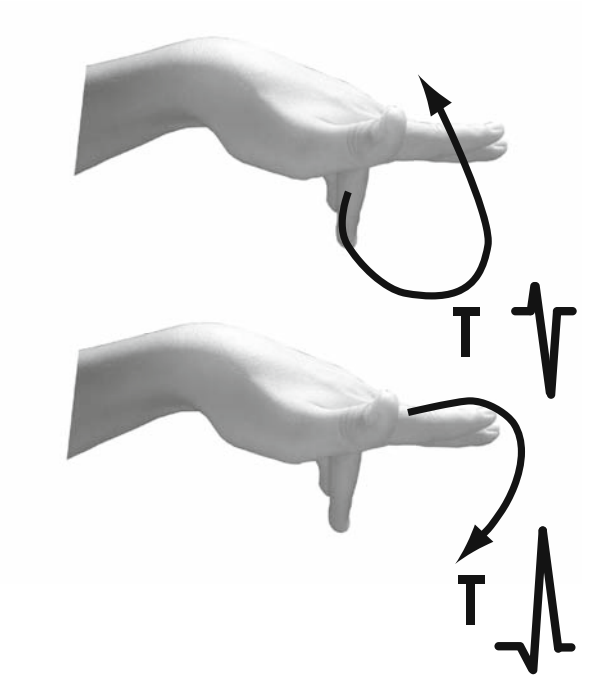


Figure 7: The "hand model" in left anterior fascicular block (*top*). Activation of the left ventricle is initiated by the left posterior fascicle (fourth and fifth fingers). This leads to left axis deviation with an rS pattern in the inferior leads (shown). Conversely, in left posterior fascicular block, activation of the left ventricle occurs over the left anterior fascicle (first and second fingers) and right axis deviation with a qR pattern is noted in the inferior leads (*bottom*).

of the left ventricle via the posterior fascicle leads to relatively deep S waves in the lateral precordial leads (V_5 and V_6), since these leads are actually located below the horizontal plane formed by leads V_1 through V_4 (Figure 8).

Left anterior fascicular block should increase the suspicion of organic heart disease. However, isolated left anterior fascicular block can be observed, particularly in older patients.

In left anterior fascicular block:

1. *QRS duration 0.10–0.12 s*
2. *Left axis deviation: more leftward than 45°*
3. *qR complexes in I and aVL*

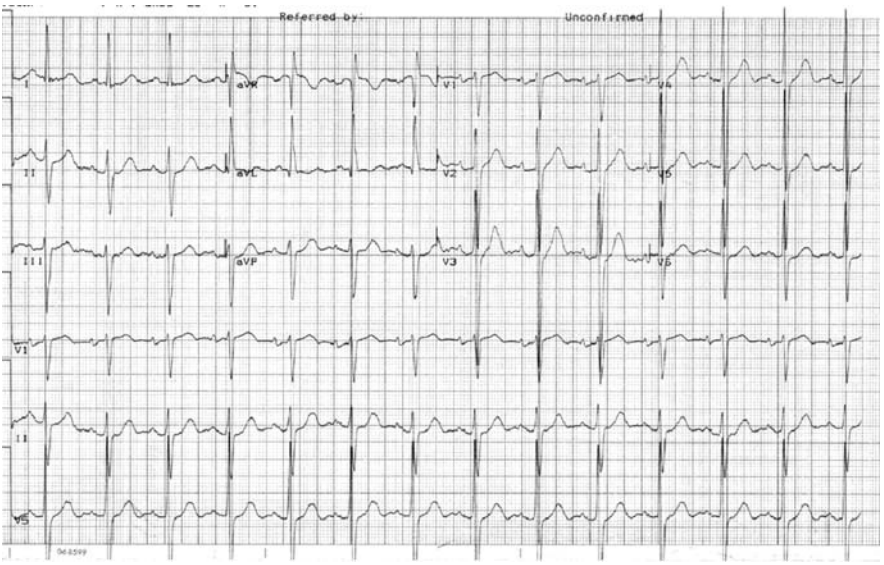


Figure 8: Electrocardiogram in left anterior fascicular block (LAFB). The left ventricle is initially activated inferiorly, then the majority of the left ventricle is activated in an upward direction from the inferior septum to the lateral and anterolateral walls. This leads to left axis deviation. In the lateral leads a qR complex is recorded and in the inferior walls an rS complex is seen. This also leads to relatively deep S waves in the precordium as activation proceeds from the apical region to the back.

Left posterior fascicular block

If conduction is blocked in the left posterior fascicle, the anterior portion of the left ventricle is activated first, and then the remainder of the left ventricle is activated in the superior to inferior direction. For this reason the major finding in left posterior fascicular block is right axis deviation (greater than 90°–110°) in the frontal plane. Left posterior fascicular block is only one cause of right axis deviation (see Appendix). In addition to right axis deviation, left posterior fascicular block is associated with small q waves in the inferior leads and small r waves in the lateral leads. An example of an electrocardiogram in a patient with left posterior fascicular block is shown in Figure 9. The findings in the frontal plane are opposite of the ECG findings associated with left anterior fascicular block. In the precordial plane, both left anterior fascicular block and left posterior fascicular block tend to be characterized with deeper S waves in the anterior chest leads (V_3 and V_4), since in both conditions there is late left ventricular activation traveling from anterior to posterior.

Isolated left posterior fascicular block is fairly uncommon. The low incidence of left posterior fascicular block is probably due to both an anatomic and

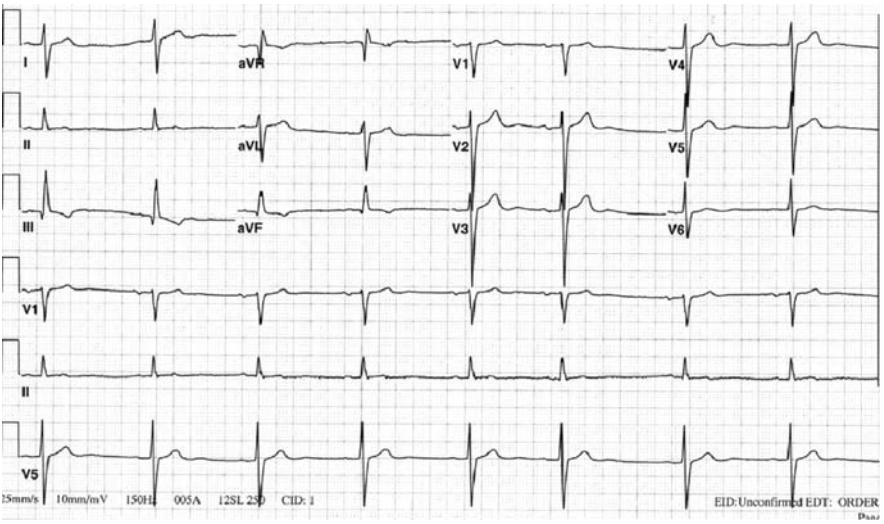


Figure 9: Electrocardiogram in left posterior fascicular block (LPFB). The left ventricle is activated anteriorly in the septal region and then is directed downward to the inferior wall. This leads to right axis deviation with qR complexes now recorded in the inferior leads and rS waves in leads I and aVL. Again, deep S waves are recorded in the precordium.

a physiologic reason. First, the left posterior fascicle is relatively short and broad. Second, the left posterior fascicle usually receives blood supply from both the left anterior descending coronary artery and the posterior descending artery branch of the right coronary artery.

In left posterior fascicular block:

1. *QRS duration 0.10–0.12 s*
2. *Right axis deviation: > 100°*
3. *rS complex in I and aVL*

Case study (continued):

Ms. Scott has developed left anterior fascicular block in addition to right bundle branch block. This is the most commonly observed form of "bifascicular" block, since the left anterior fascicle and the right bundle branch are contiguous. The presence of bifascicular block increases the likelihood of organic heart disease. A thorough physical examination should be performed with further work-up based on symptoms or other findings. In an asymptomatic person, the presence of left anterior fascicular block and right bundle branch block is not an indication for the implantation of a permanent pacing device. However, it should be noted that progression to AV block (Chapter 10) could occur.

Key points

1. Anatomically the left bundle is much larger than the right bundle. The left bundle generally divides into anterior and posterior fascicles. Specific ECG patterns are associated with blocks in each of the fascicles or bundles.
2. In right bundle branch block, an rSR' complex, which is predominantly positive, is seen in lead V_1. Conversely, in left bundle branch block a predominantly negative complex is seen in lead V_1.
3. Block can also occur in one of the two predominant fascicles of the left bundle. In left anterior fascicular block, left axis deviation ($> -30°$) and a qR complex in lead avL or lead I are seen. Conversely, in left posterior fascicular block, right axis deviation and a qR complex in the inferior leads are observed. Both conditions are associated with S waves in the anterior precordial leads.

Questions

1. An electrocardiogram is shown in Figure 10. What is the abnormality?

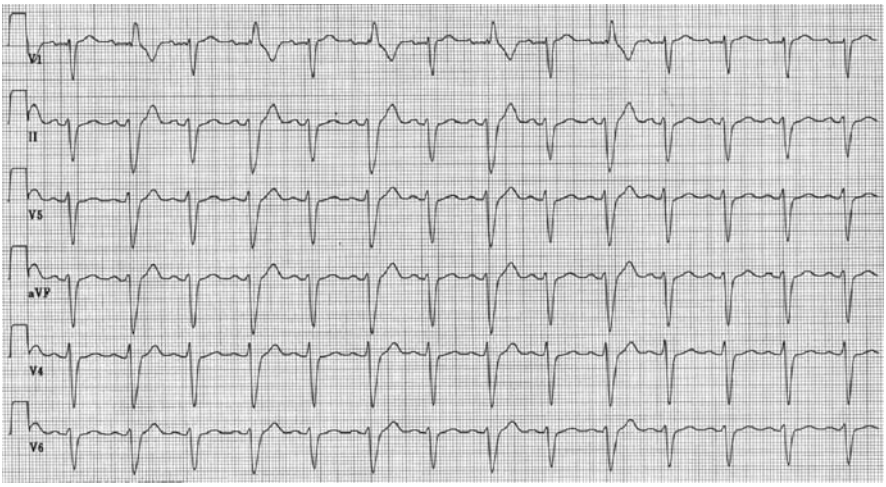


Figure 10: ECG for Problem 1.

2. An electrocardiogram is shown in Figure 11. What is the abnormality?
3. An electrocardiogram is shown in Figure 12. What is the abnormality?

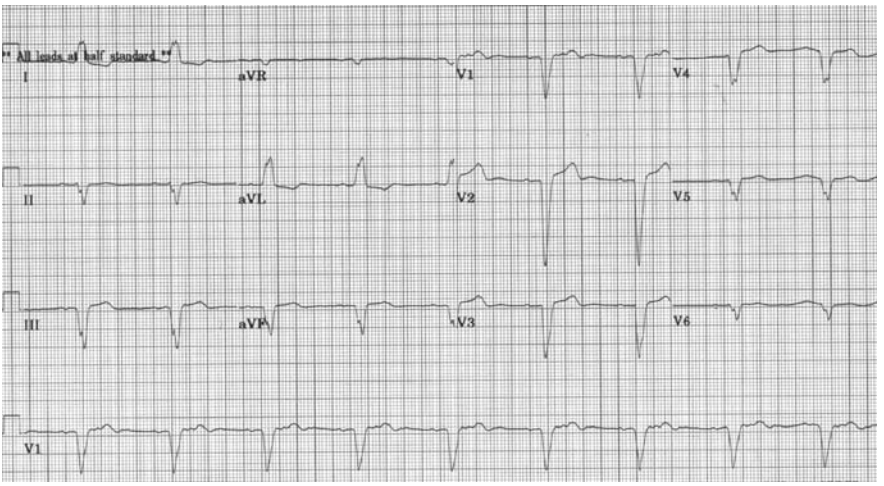


Figure 11: ECG for Problem 2.

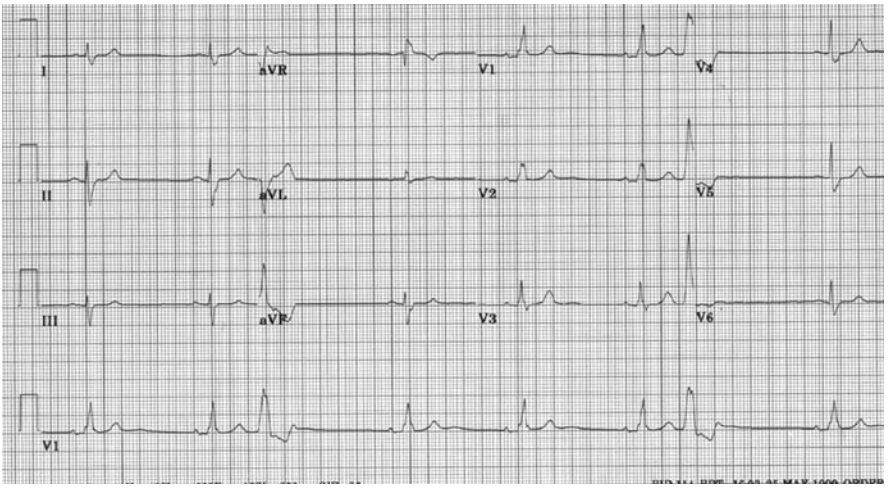


Figure 12: ECG for Problem 3.

Answers

1. The ECG shows a rhythm strip of leads V_1, V_4, V_5, V_6, II, and aVF. The patient has LAFB with intermittent RBBB. Notice that the QRS is wide (>0.12 s) and in lead V_1 every other QRS complex is wider and characterized by an rSR' complex, characteristic of RBBB. Often the right bundle branch has the longest refractory period and intermittent RBBB is often seen.

With continued activation, the refractory period decreases and the intermittent RBBB resolves in the later part of the ECG.

2. The patient has LBBB. Notice the wide QS complex in lead V_1. Since the heart is more posteriorly directed, a monophasic R wave typical of LBBB is not observed in V_6 but is seen in I and aVL (which are more lateral than V_6). The ECG was obtained at half-standard. The 1 mv test produces a 5 mm deflection.
3. The patient has RBBB. Although the classic rSR' complex is not observed in V_1, monophasic R complexes can be observed in RBBB. The late S wave consistent with RBBB in V_6 is still present. The third and seventh QRS complexes are due to premature ventricular contractions (they are not preceded by P waves). Premature ventricular contractions will be discussed in Chapter 9.

Part III

Abnormal repolarization

Chapter 6
Ventricular repolarization: T waves and U waves

Jennifer Yost is a 13-year-old girl who comes to your office accompanied by her parents. They are concerned that she has had episodes of passing out (the medical term for transient loss of consciousness is syncope) while exercising. Her ECG is shown in Figure 1.

T waves

On the ECG, ventricular repolarization leads to inscription of the T wave. Abnormalities of repolarization can lead to abnormalities of the T wave. The T wave shape and the location of the T wave relative to the QRS (ventricular depolarization) provide important data for the clinician.

T wave shape

The T wave is usually flatter and more broad-based then the QRS complex. Since ventricular depolarization occurs over the specialized His-Purkinje tissue and is mediated by the fast response Na^+ channels, ventricular depolarization is complete within 100–120 ms. In contrast, ventricular repolarization occurs cell-to-cell and is mediated by a more gradual increase in K^+ permeability. In general the direction of the T wave will be the same as the direction of the QRS complex in a given lead. This seeming paradox is due to normal depolarization occurring from endocardium to epicardium, while the general direction of normal repolarization occurs in the reverse direction: from epicardium to endocardium (Figure 6, Chapter 3).

Generally, the T wave will be in the same direction as the predominant QRS direction.

It is now clear that the myocytes within the ventricle have different ion channel populations, and it appears that these differences also contribute to the shape of the T waves observed on the surface ECG (Figures 2 and 3). Epicardial cells have shorter action potential durations and a more prominent phase 1 notch than endocardial cells. The phase 1 notch is due to the opening of several different ion channels,

F. Kusumoto, *ECG Interpretation: From Pathophysiology to Clinical Application*,
DOI 10.1007/978-0-387-88880-4_6, © Springer Science+Business Media, LLC 2009

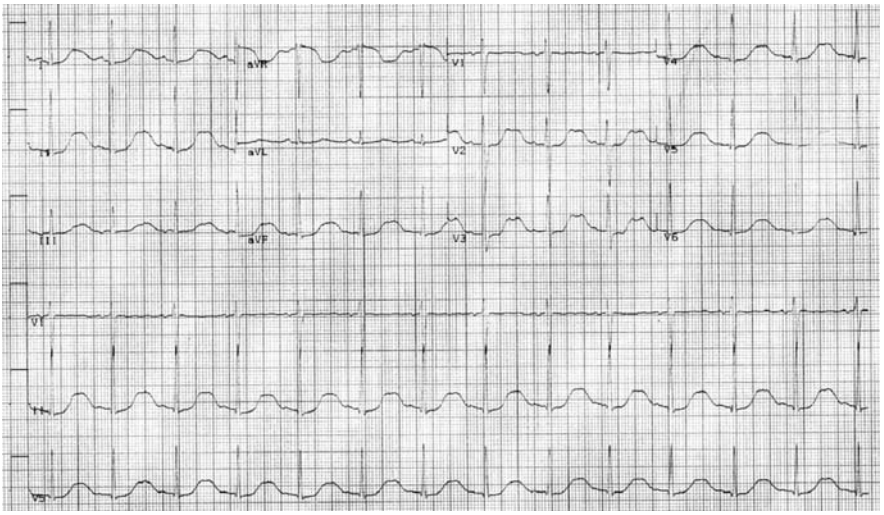


Figure 1: Ms. Yost's ECG.

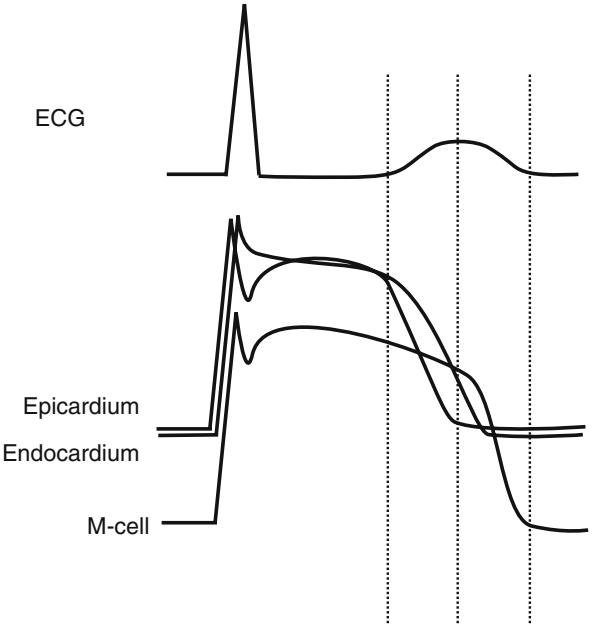


Figure 2: Ventricular repolarization. The ventricle is composed of multiple types of cells with different action potential shapes. In experimental preparations the beginning of the T wave seems to coincide with separation between the action potentials of epicardial cells and endocardial cells, since repolarization begins earlier in epicardial cells. The peak of the T wave coincides with complete repolarization of epicardial cells. The end of the T wave coincides with repolarization of the M cells located in the "middle" portion of the myocardium.

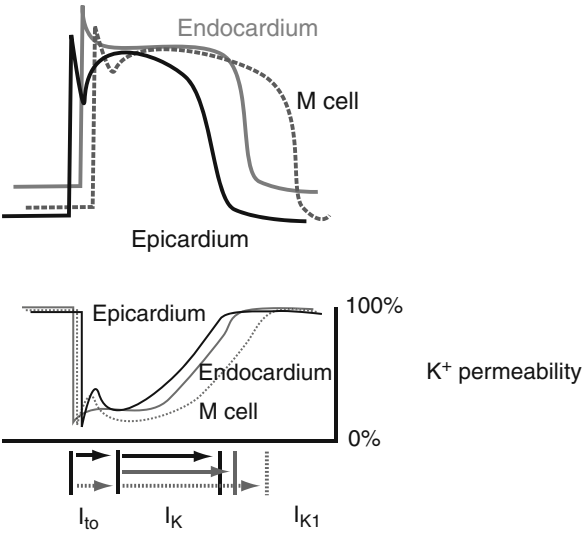


Figure 3: Differences in repolarization properties of different ventricular myocytes appear to be mediated by different populations of K^+ channels that lead to differences in K^+ permeability after the phase 0 upstroke. Endocardial cells do not have an I_{to} current. Epicardial cells have an I_{to} current and an I_K current (delayed rectifier) with more rapid gating properties, leading to increased K^+ permeability and the shortest action potential duration. M-cells have a smaller I_{to} current than epicardial cells and slower activation of the I_K.

one of which is permeable to K^+ ions (thus moving the membrane voltage closer to the resting potential). In addition there appears to be a specialized population of cells called M cells that is in the "mid-myocardium." The M cells have a phase 1 notch that is slightly less prominent than epicardial myocytes but, distinct from endocardial and epicardial cells, have long action potential durations, particularly at slow heart rates. This effect appears to be mediated by continued Na^+ influx and a smaller population of K^+ channels responsible for repolarization (Figure 3). In experimental preparations, preliminary studies suggest that the beginning of the T wave appears to coincide with the separation of the action potential of the ventricular epicardium from the endocardium and M cells. This separation is very gradual, and for this reason it is difficult to accurately identify the "beginning" of the T wave. The peak of the T wave appears to coincide with complete repolarization of the epicardium, and the end of the T wave coincides with repolarization of the M cells (Figure 2).

T wave inversion is usually defined as an inverted T wave in the presence of a positive QRS complex (Figure 4). It suggests that abnormal ventricular repolarization is present. T wave inversion can be seen in a variety of settings and is a nonspecific finding. However, it is important to recognize that abnormal T waves can be the first subtle manifestations of myocardial ischemia or other problems.

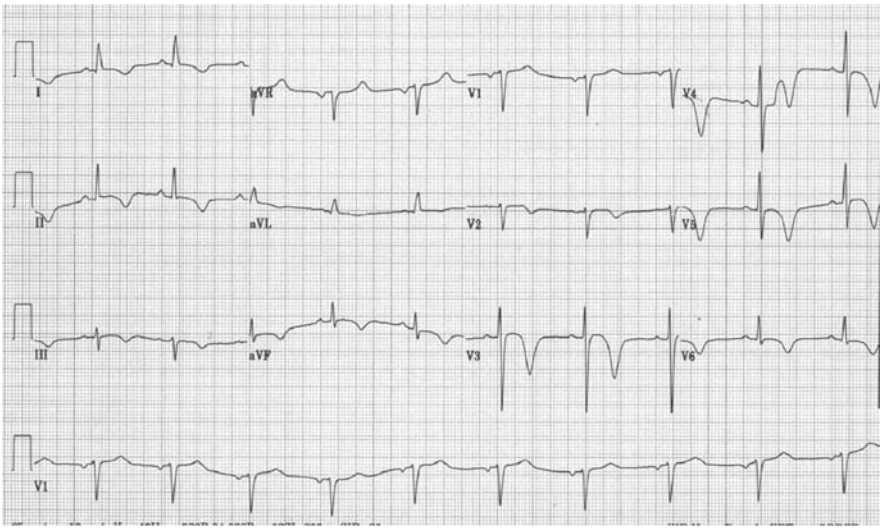


Figure 4: Patient with abnormal T waves. Notice the prominent and symmetric T wave inversion in the inferolateral leads that are in the opposite direction of the predominant depolarization forces (I, II, aVL, aVF, V_5, and V_6). The deeply inverted T waves in V_3 and V_4 are also abnormal.

Importantly, the presence of T wave abnormalities on a baseline ECG appears to be associated with a higher risk of long-term mortality.

There are a number of subtle gradations between normal T waves and T wave inversion, such as T wave flattening or subtle T wave inversion. These changes are usually grouped together as "nonspecific STT wave changes." These subtle changes can be seen in a variety of conditions and do not necessarily indicate significant cardiac problems. For this reason they should be considered adjunctive rather than diagnostic findings. For example, subtle T wave inversion would be more important in a patient with chest pain.

Digoxin can cause downsloping ST segments in the lateral leads (Figure 5). This previously common cause for STT changes is less frequently observed, since digoxin is now prescribed less often. However, digoxin can still be used for the treatment of atrial fibrillation and heart failure. Notice the similarity of the STT wave changes associated with digoxin and those seen in a patient with repolarization abnormalities due to left ventricular hypertrophy (Chapter 4). This is why the presence of repolarization abnormalities is given less weight as a criterion for left ventricular hypertrophy in a patient receiving digoxin in the Romhilt-Estes criteria.

T wave location

Since the T wave represents ventricular repolarization, the time from the QRS complex to the end of the T wave provides a rough estimate for the duration of

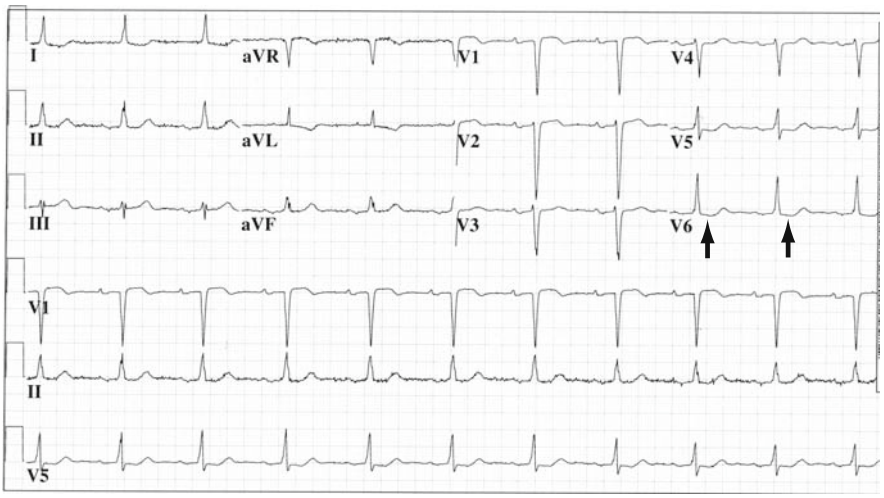


Figure 5: STT changes in a patient on digoxin. The "sagging" ST segments in the lateral leads (I, aVL, V_5, and V_6, arrows) and the inverted T waves in I and aVL are typical in patients with digoxin.

the plateau phase of ventricular tissue. The interval from the beginning of the QRS complex to the end of the T wave is defined as the QT interval. Unfortunately, accurate measurement of the QT interval can be difficult for several reasons. First, the T wave has a smaller amplitude with a broad base, which can make identifying the exact endpoint of the T wave very difficult. Second, a deflection after the T wave, called the U wave, can be observed in some people. Identification of a normal U wave is discussed later in this chapter, but it is important to remember for the purposes of this discussion that the U wave should not be included in the measurement of the QT interval. Since the normal U wave is most obvious in the anterior precordial leads (V_3 and V_4), many experts have recommended leads II and V_5 as the best leads for measuring the QT interval (Figure 6). Others recommend evaluating all twelve leads and using the longest measured value as the QT interval. Given these different opinions, it is not surprising that a recent study found that more than 50% of physicians (including cardiologists) measured the QT interval incorrectly.

Even if the QT interval could be measured by using a standardized methodology, identifying an "abnormal" QT interval can be difficult. The QT interval shortens with higher rates, and a "normal" QT interval at a faster rate may actually be a sign of an underlying problem. In addition, there is significant overlap between "normal" and "abnormal" QT intervals. As a general guide, the upper limit of normal for the QTc is probably 0.45 s in men, 0.47 s in women, and 0.45 s for boys and girls $<$ 15 years old. Despite all of these problems, QT interval measurement is important, and in general the student should remember that there is a rough correlation between the QT interval and risk for ventricular arrhythmias, i.e., the longer the QT interval the higher the risk.

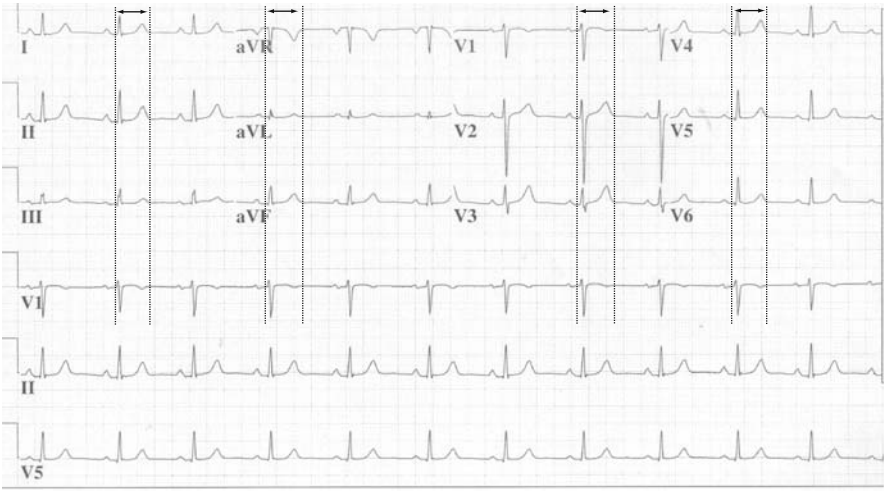


Figure 6: The QT interval can be better estimated by drawing a series of "plumb lines," since the ECG tracings are acquired simultaneously in all of the leads. Notice that the QT estimate from the "unipolar" and the V_1–V_3 leads is slightly longer than the QT estimate from the "bipolar" and the V_4–V_6 leads. In this case the uncorrected QT interval is approximately 430 ms. As a rough estimate for whether the QT interval is normal or abnormal, notice that the QT interval is less than half the distance between QRS complexes (RR interval).

Case Study (continued): Examine Jennifer's ECG. Notice that the QT interval is very prolonged. The automated ECG lists it as 470 ms. Do you agree? At normal heart rates, a general guideline is that the QT interval should be less than half the distance between two consecutive QRS complexes.

Genetic causes for a long QT (Long QT Syndrome)

One of the most fruitful areas of research that has demonstrated the relationship between basic research and clinical medicine has been the long QT syndrome. Patients with the long QT syndrome have a prolonged QT interval at baseline and are at risk for life-threatening ventricular arrhythmias.

Any condition that causes delay in repolarization in ventricular tissue will be associated with a prolonged QT interval (Figure 7). Remember that the plateau phase of the action potential was due to small amounts of continued inward flow of Ca^{2+} and Na^{+} just balanced by outward flow of K^{+}. Repolarization starts as K^{+} permeability gradually increases and the cell returns to the resting membrane potential, where the cell membrane is freely permeable to K^{+} ions. With this in mind, potential causes for prolongation of repolarization include abnormal continuation of Na^{+} or Ca^{2+} inflow, or attenuation or delay of K^{+} outward flow.

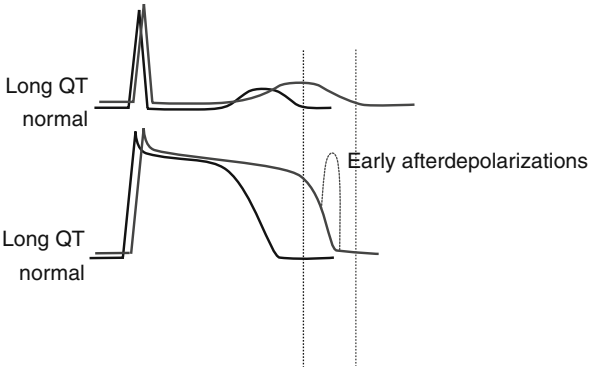


Figure 7: Schematic for the ECG basis of the long QT syndrome. Any abnormality that prolongs ventricular repolarization will lead to a prolonged QT interval on the ECG. Prolongation of repolarization can lead to reactivation of Na and Ca channels that have recovered, leading to membrane depolarization at the end of the plateau phase (early afterdepolarizations). As discussed in the chapter on wide complex tachycardia, repeated afterdepolarizations can lead to sustained life-threatening arrhythmias due to triggered activity.

Several genetic defects have been identified that are associated with these abnormalities. In fact, there are currently twelve genetic abnormalities that have been identified that probably account for about 70% of cases of inherited long QT syndrome. The most commonly observed genetic mutations cause attenuation and/or delay of the K^+ current.

Any condition that is associated with prolongation of repolarization increases the possibility of ventricular arrhythmias due to a specific form of tachycardia called triggered arrhythmias. To understand the genesis of triggered arrhythmias it is necessary to understand the opening and closing properties of ion channels. Ion channels exist in several different states. For example, at baseline, a Na^+ channel is at rest. When the Na^+ channel is exposed to a small electrical current, it opens for a brief period of time and quickly goes to an "inactive" state, where it cannot reopen. The rapid transition from the "open" state to the "inactive" state is mediated by a protein "tail" that plugs the pore that allows the Na^+ ions to enter the cell. Over time the ion channel will change from the "inactive" state to the resting state, where it can reopen. It is these "gating characteristics" that will prevent a cell from being depolarized again even if a large electrical current is applied to the cell. This property is responsible for refractoriness (Figure 8). It is easier to think about refractoriness by thinking about a cell that is stimulated twice. When the interval between the stimuli is long, two normal action potentials are observed. If one gradually restimulates the cell earlier and earlier, first a period of relative refractoriness will be observed, since there are fewer Na^+ channels in the resting state, and often a larger stimulus is required to depolarize the cell during this period. During the relative refractory phase, a less than normal response will be seen, since some of the channels are in

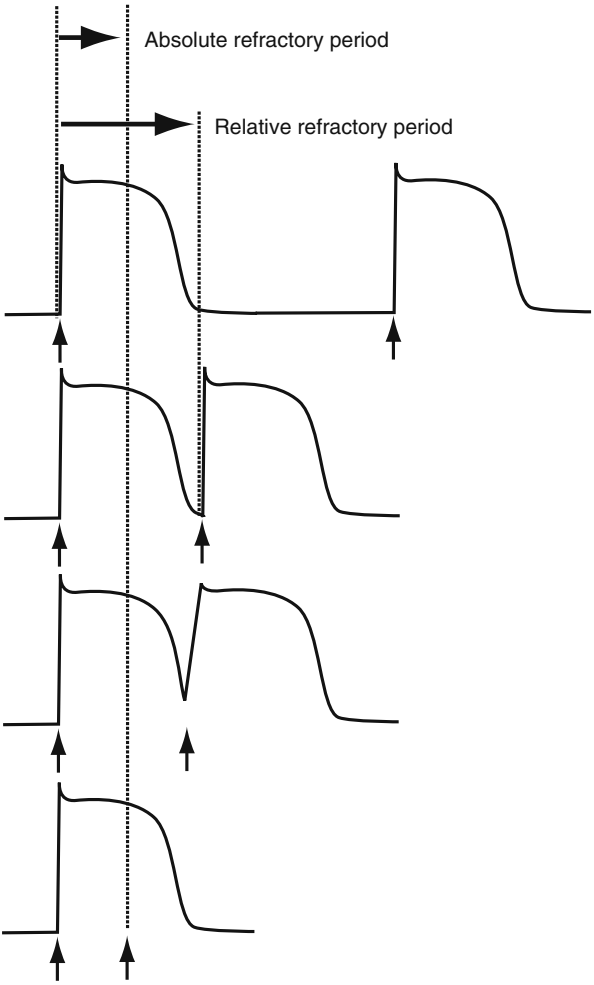


Figure 8: Schematic showing the property of refractoriness. As one progressively restimulates the heart sooner and sooner (top to bottom panels), initially the second action potential is the same as the initial action potential. As the second stimulus encroaches on the initial action potential, the phase 0 upstroke becomes less rapid and high as fewer Na^+ channels are available for activation. Finally the action potential reaches a point where no matter how large the stimulus, no depolarization is observed because no additional Na^+ channels are available to be recruited. This point is called the absolute refractory period.

the inactive state and have not returned to the resting state. As the stimulation is delivered earlier and earlier a point will be reached when there are no Na^+ channels available for activation and depolarization will not occur. This period is called absolute refractoriness, and the cell cannot be depolarized because there are no Na^+ channels in the resting state that are available to open.

In any condition associated with a prolonged plateau phase, Na^+ and Ca^{2+} channels can become reactivated when they return to the resting state and lead to membrane depolarization. Membrane depolarizations after the initial Phase 0 are called "afterdepolarizations" and repetitive afterdepolarizations are often called "triggered activity". Repetitive afterdepolarizations in ventricular tissue can lead to sustained or nonsustained ventricular arrhythmias that are often called "torsade de pointes" because of the characteristic "twisting of the points" appearance on the ECG (Figure 9).

Acquired long QT

There are several clinical causes for prolongation of the QT interval (Table 1). A number of electrolyte abnormalities can cause QT prolongation, including

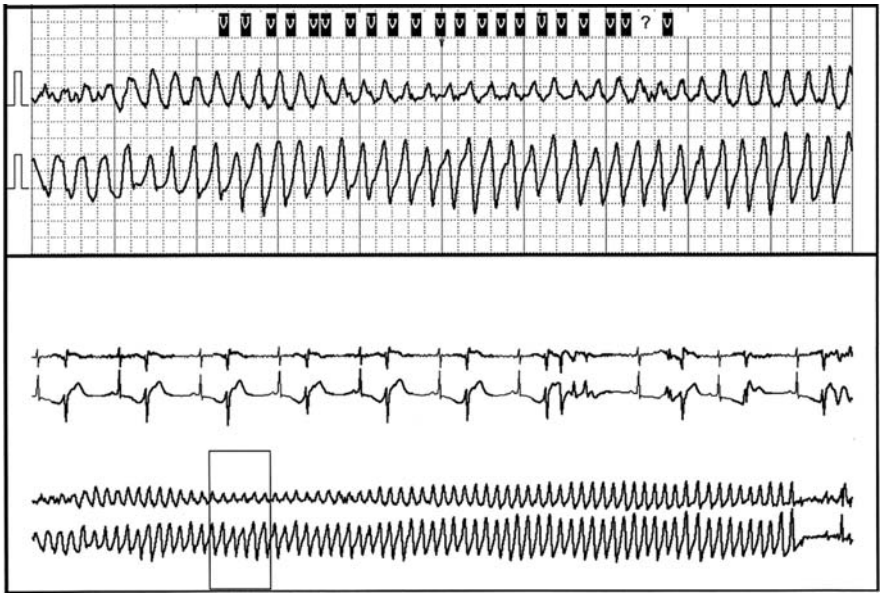


Figure 9: A patient with long QT (due to use of an antiarrhythmic drug that blocks K^+ channels) develops ventricular arrhythmias. First the patient develops single extra ventricular beats at the T wave and then develops a longer burst of ventricular arrhythmia. Notice the oscillating change in the QRS size. Fortunately the arrhythmia is nonsustained and the patient returns to sinus rhythm.

Table 1: Causes of a prolonged QT interval

Cause	Details
Congenital	Multiple genetic types have been identified, most common are due to K^+ channel mutations that cause delayed repolarization.
Acquired	
• Metabolic	Hypokalemia
	Hypocalcemia
	Hypomagnesemia
	Hypoglycemia
• Drugs	See Table 2
• Neurologic	Intracranial hemorrhage
	Stroke
• Endocrine	Hypothyroid, pituitary insufficiency
• Cardiac	Ischemia, myocardial infarction
• Miscellaneous	Liquid protein diet, obesity

hypokalemia, hypocalcemia, and hypomagnesemia (remember all "the hypos"). The ECG abnormalities associated with electrolyte disorders will be covered separately in Chapter 9, but an introductory discussion of the prolonged QT interval associated with hypokalemia is useful here. Traditionally, a prominent U wave inscribed after the T wave has been thought to be the most important ECG characteristic for hypokalemia (Figure 10). Recent experimental data suggest that the "U" wave actually is a bifid T wave due to a notch in the upstroke of the T wave. This is actually part of the reason that there has been confusion on whether to include the U wave in the measurement of the QT interval. Generally, as discussed below, the true physiologic U wave should not be included in the calculation for the QT interval. Hypokalemia is associated with increased heterogeneity of phase 3 repolarization and separation of phase 3 repolarization between M cells and endocardial cells, but the second peak still coincides with full repolarization of epicardial cells.

A number of drugs can cause QT interval prolongation. The most common mechanism for drug-related QT prolongation is blocking the function of K^+ channels. In addition, it has been shown that some drugs may affect intracellular trafficking of ion channel proteins from the endoplasmic reticulum and Golgi body to the cell membrane. The most common class of drugs associated with QT prolongation is the antiarrhythmic drugs, including sotalol, defetilide and ibutilide. Figure 11 shows an ECG recorded from a man receiving dofetilide. Dofetilide is a medication that blocks K^+ channels and is sometimes used for the treatment of atrial fibrillation. In this case, because of QT prolongation observed on ECG and the associated risk for developing torsade de pointes, the medication was stopped. Noncardiac drug classes that can cause QT prolongation and increased risk for torsade de pointes include tri-

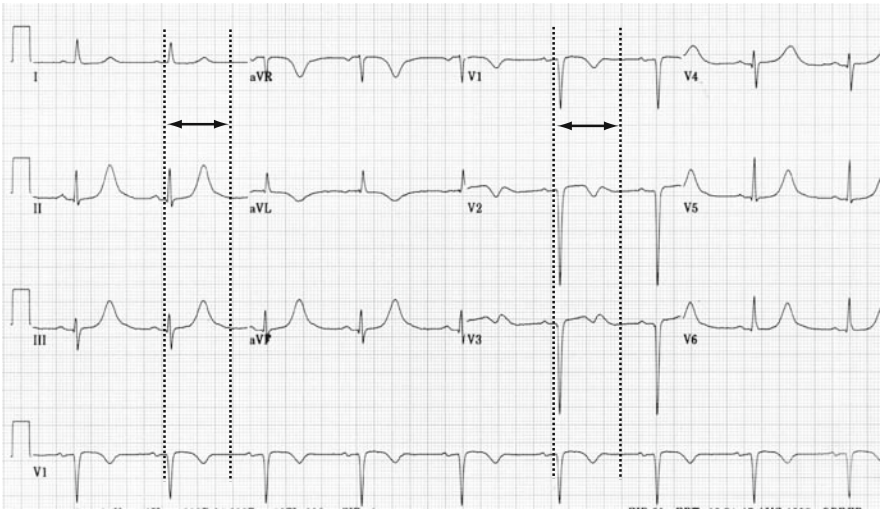


Figure 10: Prolonged QT interval in hypokalemia. The prolonged QT interval can be estimated from lead II. By transposing this value to the chest leads, it is obvious that the "U" wave actually represents a bifid T wave.

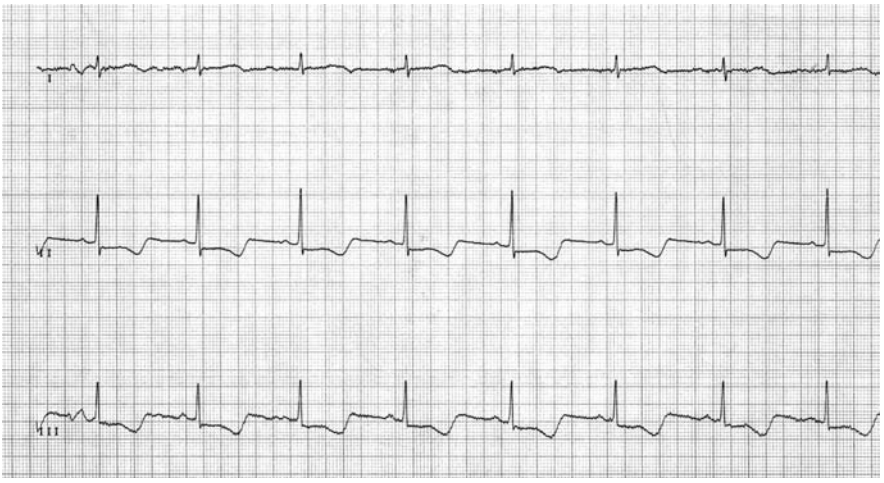


Figure 11: QT prolongation due to the use of dofetilide. Defetilide is a K^+ channel blocker that is used to treat atrial fibrillation.

cyclic antidepressants, phenothiazines, selected antibiotics and antihistamines, and a number of others (Table 2). A number of drugs, including cisapride, ternadine, and astemizole, were withdrawn from the market because of cases of sudden cardiac death due to proarrhythmia.

Table 2: Drugs that cause QT interval prolongation

Cardiac	Antiarrhythmic	Disopyramide, quinidine, dofetilide, sotalol, amiodarone
	Calcium channel blockers	Bepridil, ranolazine
	Diuretics	indapamide
Noncardiac	Antiseizure	phenytoin
	Antibiotics	Amantadine, clarithromycin, erythromycin, pentamidine, ketoconazole, antimalarials (chloroquine, halofantrine)
	Antidepressants	Amitrytyline, desipramine, fluoxetine, imipramine
	Antipsychotic	Chlorpromazine, haloperidol, risperidone
	Antimanic	Lithium
	Lipid lowering drugs	Probucol
	Hormones	Fludrocortisone, vasopressin
	Chemotherapy agents	Tamoxifen
	Miscellaneous	Arsenic, methadone, licorice

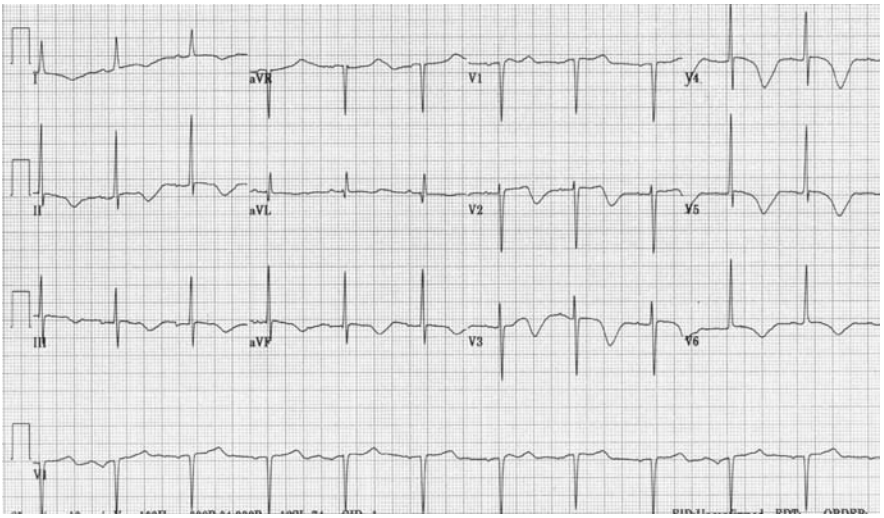


Figure 12: QT prolongation and T wave inversion in a patient that has suffered a subarachnoid hemorrhage.

Prolonged QT intervals, often with associated deeply inverted broad-based T waves, can be observed with injury to the central nervous system, particularly with subarachnoid hemorrhage (Figure 12). The mechanism for QT prolongation and repolarization abnormalities is not understood, but some believe that it is mediated by changes in autonomic nervous system function and surges in catecholamine release.

U waves

After his initial description of the ECG, Einthoven described the presence of another wave after the T wave that is called the U wave. The physiologic U wave is usually defined as a low amplitude wave (less than one-fourth the T wave) that is most prominent in leads V_1, V_2, or V_3. (Figure 13). The U wave can be most easily identified by carefully measuring the QT interval in one of the frontal leads and using that measurement on the precordial leads; any wave that appears after the interval would be defined as a U wave. It is important to remember that the P wave in a patient with first degree AV block can sometimes be confused with a U wave (Figure 14).

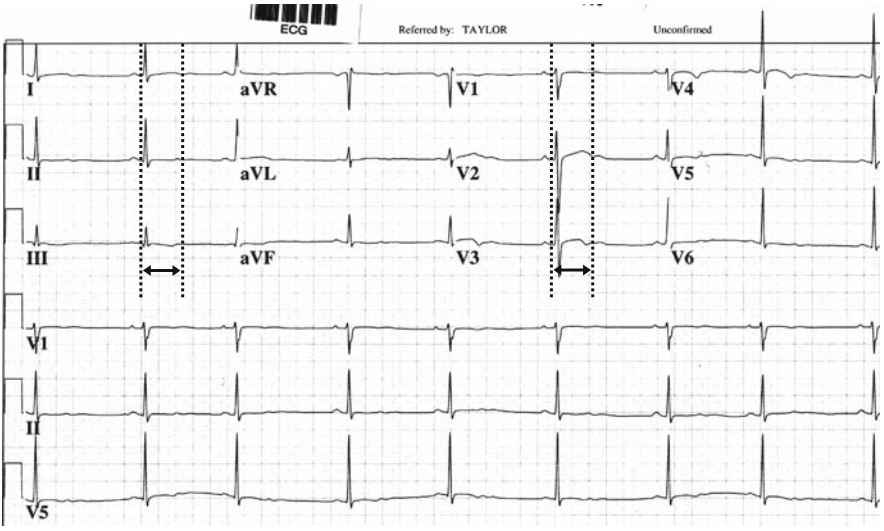


Figure 13: Normal physiologic U waves. The actual QT interval can be measured from lead II. By transposing the QT interval measured in lead II to V_2 and V_3 the reader can see that the U waves occur after the QT interval. Compare this ECG to Figure 10 and the bifid T waves.

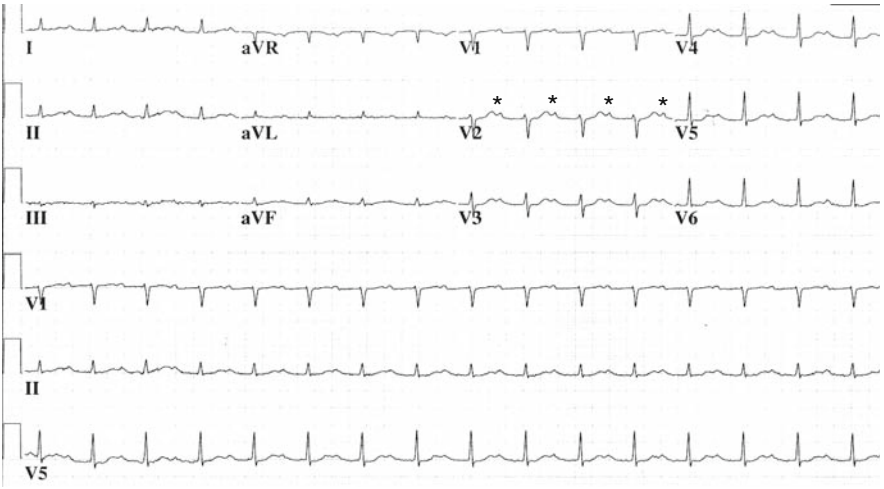


Figure 14: In this situation a patient has a prolonged PR interval. The P waves, marked can sometimes be mistaken for a U wave or a prolonged QT interval.

Previously, the most widely held hypothesis has been that repolarization of His-Purkinje tissue (which has a longer action potential duration than M cells) was responsible for the U wave. The main difficulty with this hypothesis has been the very small relative muscle mass of the His-Purkinje tissue. Recent research suggests that the U wave is actually due to stretch mediated depolarization. Patients with ion channel disorders that are associated with a short QT interval have early repolarization but normal duration of systole. Interestingly, they will have prominent U waves that are not immediately after the T wave (which would be expected if His-Purkinje tissue repolarization was responsible for the U wave) but rather coincide with the early diastole and rapid filling of the left ventricle. Mechanical stretch of the cell membrane can cause local membrane depolarizations that can be observed on the surface ECG as a U wave. It is now believed by many in the field that this is the mechanism for the genesis of the U wave.

Although a prominent U wave has been traditionally associated with hypokalemia, recent studies as outlined above suggest that the U wave in hypokalemia is actually a notched or "two-component" T wave. If the "U" wave in hypokalemia is excluded, there are no identified clinical consequences to the presence of a U wave on the ECG. It is remarkable that since its identification 100 years ago, the mechanism of the U wave is just now being elucidated.

Jennifer has the long QT Syndrome. It will be important for her to avoid certain medications. Long QT Syndrome is often treated with medications that block beta adrenergic receptors. In some severe cases a device that automatically identifies ventricular arrhythmias and delivers a shock (implantable cardiac defibrillator or ICD) is implanted.

Key points

1. Repolarization of ventricular tissue is the mechanism for the T wave. The T wave is broader-based and lower in amplitude because of regional differences in the action potential shape.
2. It is important to consider both the shape and location of the T wave. In general, the normal T wave will be in the same direction as the QRS complex.
3. Prolongation of ventricular repolarization will cause a prolonged QT interval. Although difficult to measure, identification of an abnormally prolonged QT interval is important.

Questions

1. Which statement is false?
   A. The T wave represents ventricular repolarization.
   B. The T wave is generally in the same direction as the QRS complex because depolarization and repolarization occur in the same general direction.
   C. Changes in the T wave are often subtle and nonspecific.
   D. T wave abnormalities are associated with reduced long-term survival.
2. QT interval prolongation is observed in all of the following except:
   A. Hypokalemia
   B. Hypocalcemia
   C. Hyperthermia
   D. Genetic defect associated with attenuated potassium current
3. A drug is developed for gastrointestinal distress, but it is noted that it prolongs the QT interval. A possible mechanism of action is:
   A. Blocks Na^+ channels
   B. Enhances Na^+ channel opening
   C. Enhances K^+ channel opening
   D. Blocks Ca^{2+} channels

Answers

1. B. The T wave and QRS generally have the same direction, but this is due to endocardial-epicardial depolarization and epicardial-endocardial repolarization.
2. C. Hyperthermia is not associated with QT prolongation. This effect is noted in hypothermia.
3. B. Enhancing continued inward flow of Na^+ will be associated with prolongation of the action potential. This is a very rare effect; most drugs that prolong the QT interval are K^+ channel blockers.

Chapter 7

ST segment elevation and other ECG findings in myocardial infarction

The ST segment is isoelectric under normal conditions, since after ventricular depolarization the ventricular myocytes are at the plateau phase (phase 2). During this period the ventricular cells have similar voltages, so that no voltage gradient exists. In certain pathophysiologic conditions the ST segment can change from baseline. The most important cause for changes in the ST segment is myocardial ischemia and myocardial infarction. Evaluation for the presence of myocardial ischemia or myocardial infarction is one of the most important uses of the ECG.

Case Study: John Arbuckle is a 48-year-old man with a history of high blood pressure and diabetes. This morning, approximately two hours ago, he noted a dull ache in the middle of his chest that radiates to his jaw. His ECG is shown in Figure 1.

Pathophysiology of coronary artery disease

Cellular changes associated with acute myocardial infarction

The heart receives blood from the coronary arteries. Within the coronary arteries lipid-filled atherosclerotic plaques can form. These plaques can rupture, exposing the lipid-rich interior (Figure 2). The exposed plaque promotes platelet aggregation and formation of a thrombus. If thrombus formation is extensive the coronary artery can become completely occluded, and the heart muscle that is supplied by this artery does not receive blood. This condition is called a myocardial infarction. Myocardial ischemia is usually defined as the early period in which potentially reversible cellular changes are present. Although the blood flow is not sufficient to provide the metabolic needs of the myocyte, if blood supply is reestablished (reperfusion), no significant permanent damage will occur.

On a cellular level, when the cardiac cell is deprived of blood, the cell membrane becomes increasingly permeable to K^+ (Figure 3). This increase in potassium permeability is initially due to activation of specialized K^+ channels that are sensitive to low levels of ATP (I_{KATP}). Since the K^+ concentration is significantly higher inside the cell, K^+ "leaks" out in the injured region. Increased extracellular K^+ leads to relative depolarization of the membrane in the injured area. If the cell continues

F. Kusumoto, *ECG Interpretation: From Pathophysiology to Clinical Application*,
DOI 10.1007/978-0-387-88880-4_7, © Springer Science+Business Media, LLC 2009

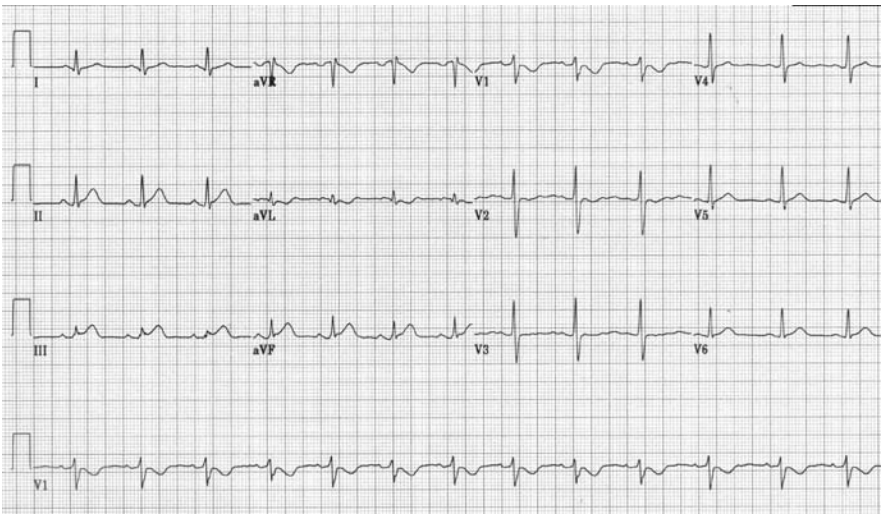


Figure 1: ECG of Mr. Arbuckle.

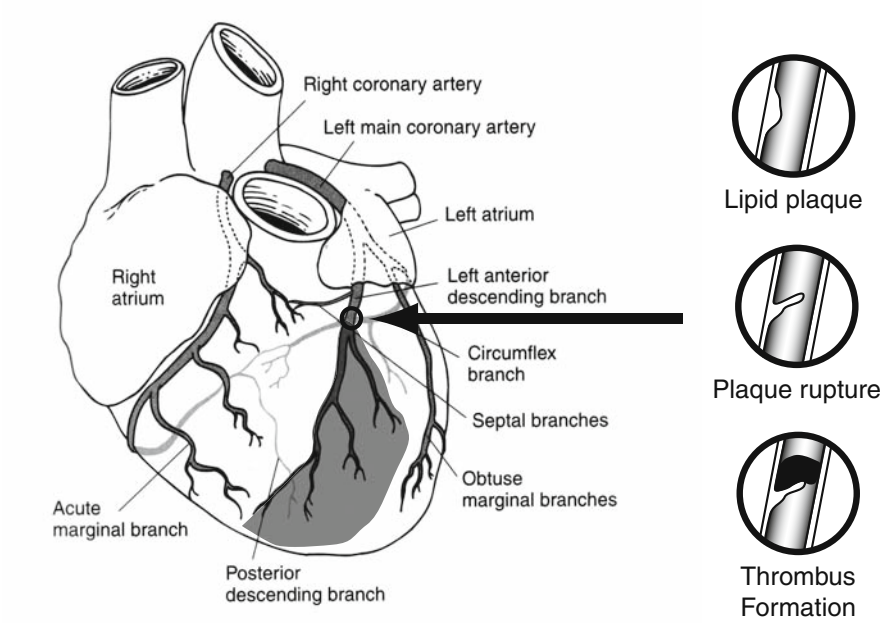


Figure 2: Schematic for the development of myocardial infarction or ischemia. A lipid plaque ruptures and thrombus forms at the exposed lipid/tissue. If the thrombus completely occludes flow, the patient develops a myocardial infarction in the downstream myocardium (shaded area). Sometimes the clot does not completely occlude flow but severe limitation in flow prevents sufficient blood flow to the affected area (ischemia). Adapted with permission from Kusumoto FM, *Cardiovascular Pathophysiology*, Hayes Barton Press, Raleigh, NC, 1999.

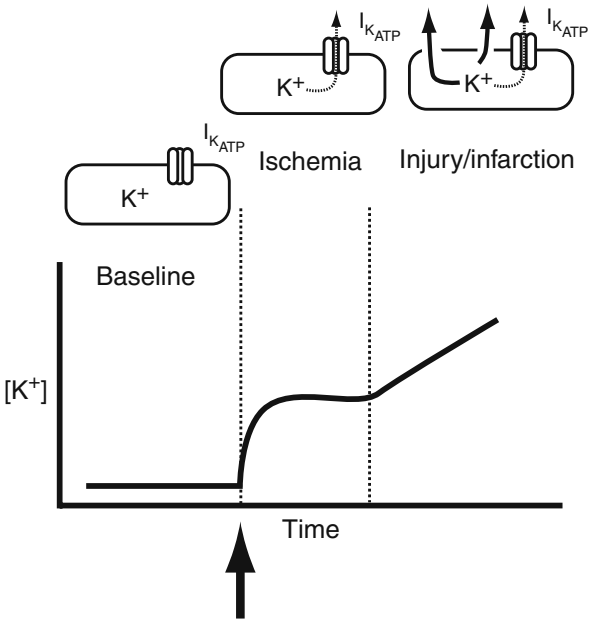


Figure 3: Effects of ligation of a coronary artery (arrow) on extracellular accumulation of K^+ in cells supplied by the ligated coronary artery. Initially there is a fairly rapid rise to an intermediate level of K^+ due to activation of K^+ channels (I_{KATP}) that increase K^+ permeability in the presence of low levels of ATP. If the coronary artery remains occluded, the cells begin to lose membrane integrity and more K^+ accumulates in the extracellular spaces.

to be deprived of blood, irreversible damage occurs and the cell membranes begin to break down and even more intracellular K^+ is lost. Since the K^+ accumulates in the intercellular clefts, very small changes in local ion concentration can lead to significant changes in membrane potential and subsequent ECG changes.

The Nernst equation ($Vm = RT/F \ ln \ K_o^+/K_i^+$): Increase in K_o^+ leads to a less negative value (natural log of a larger fraction) and the membrane becomes partially depolarized.

Evolution of myocardial infarction and ischemia

The ECG findings associated with myocardial ischemia/infarction vary depending on the specific evolutionary phase of myocardial infarction. The first phase of a myocardial infarction lasts several minutes and is called the ischemic phase, in which viability of the myocardium at risk is maintained by anaerobic metabolism. The infarction phase occurs when anaerobic metabolism cannot keep

up with metabolic need, and irreversible damage and cell death begins. The extent of irreversible damage can be attenuated by the presence of collateral blood flow from other coronary artery branches. The reperfusion phase refers to the first few minutes when coronary artery flow is reestablished, either spontaneously with partial resolution of the occlusive thrombus, or with therapeutic intervention using drugs designed to break up the thrombus or mechanically restoring flow through angioplasty.

In the damaged myocardium, the healing phase occurs over the first few weeks as scar and fibrotic changes occur in the regions of necrosis and inflammation. Ventricular contraction in the damaged area may gradually return if myocardial salvage through collateral flow or reperfusion during the infarction phase was present. After the healing phase, the patient enters into the chronic phase, in which scar and functioning heart tissue coexist.

ECG Changes in Acute Myocardial Infarction

Myocardial infarction is associated with several characteristic ECG changes: T wave peaking, ST segment changes, and abnormal ventricular depolarization (Q waves). Although the ECG is a useful tool, it is important to recognize the limitations of the ECG for identifying myocardial infarction and ischemia by noting that 20% of patients will have a normal ECG and the great majority of patients with a myocardial infarction will not have ST segment elevation observed on the ECG.

ECG changes in myocardial infarction/ischemia:

Abnormal ventricular depolarization: Q waves.

Abnormal ventricular repolarization: ST segment elevation, ST segment depression, peaked T waves, inverted T waves.

T wave peaking

The first ECG finding that can be observed during a myocardial infarction is T wave peaking, characterized by prominent T waves with a relatively narrow base. Remember that the T wave represents ventricular repolarization. T wave peaking can be observed in hyperkalemia because the phase 3 downstroke of repolarization becomes steeper. Similarly, during ischemia, activation of K^+ channels that are sensitive to reduced amounts of ATP (I_{KATP}) leads to outward flow of K^+ and accumulation of K^+ in the extracellular spaces of affected myocytes. These ion concentration changes are responsible for peaked or "hyperacute" T waves that can sometimes be observed during acute myocardial infarction/ischemia. Unlike the generalized T wave peaking observed in hyperkalemia, the peaked T waves will be observed only in the leads overlying the affected area (Figure 4). T wave peaking is observed only in the first few minutes of ischemia/infarction and is only rarely seen by the time a patient presents for medical care.

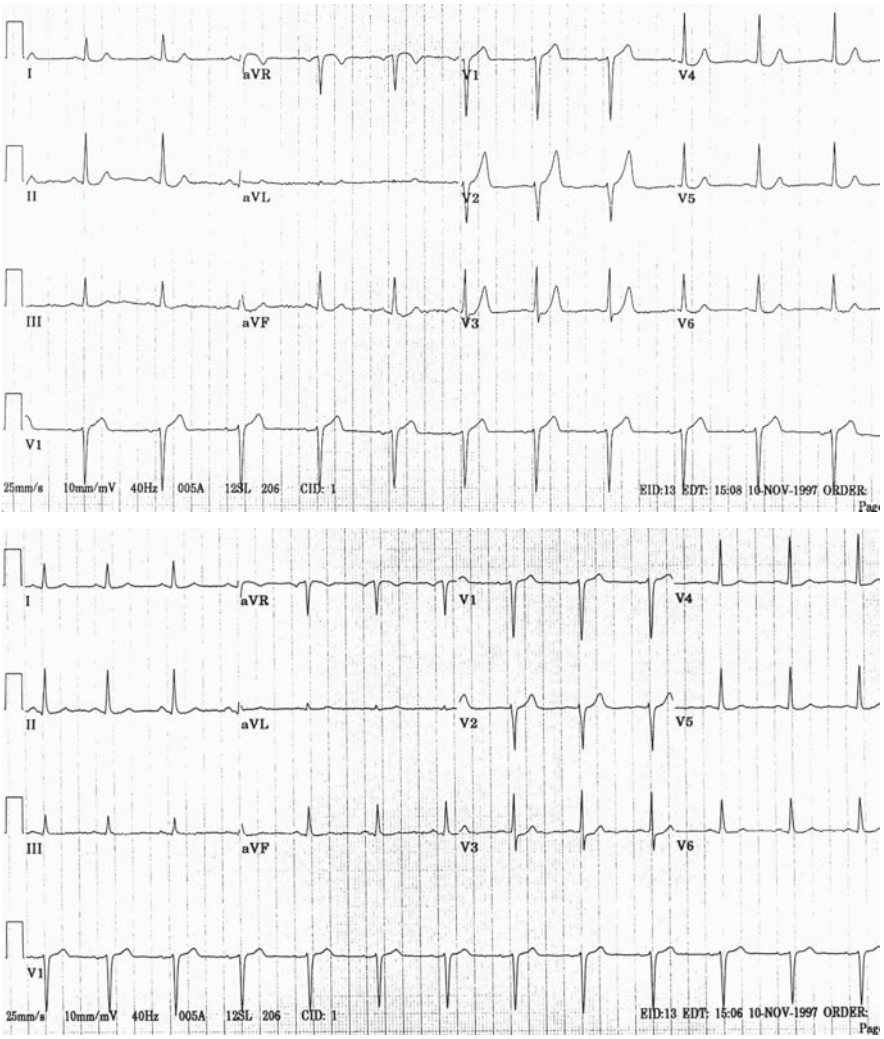


Figure 4: ECGs from a policeman who complained of intermittent chest pain. The *top* ECG was recorded when his pain was present and the *bottom* ECG after the chest pain resolved several minutes later. Notice the transient prominent T waves in leads V_2 and V_3 associated with his chest pain. The patient was found to have a $> 90\%$ blockage in his left anterior descending artery.

ST segment changes

Abnormalities in the ST segment, either elevation or depression, are the most commonly used ECG findings for the diagnosis of ischemia/infarction. Accumulation of extracellular K^+ also appears to be an important mechanism for ST segment

changes observed in ischemia. In an interesting study, mice that did not have the gene responsible for producing the pore-forming subunit for I_{KATP} did not have ST segment elevation with ligation of a coronary artery and cessation of blood flow. Increased K^+ permeability can cause ST elevation due to effects during diastole (the period between ventricular contractions, which coincides with the resting phase 4) and systole (the period from phase 0 to the beginning of the next phase 4).

During phase 4, accumulation of extracellular K^+ at baseline due to increased I_{KATP} will produce a resting voltage difference between injured and noninjured areas of the myocardium. Remember that the value of the resting membrane potential is determined by the ratio of extracellular K^+ and intracellular K^+ (Nernst equation). If extracellular K^+ is higher, the natural log of a "larger fraction" will be a less negative value and the membrane will be depolarized.

For a positive electrode over an injured region of myocardium, the relatively negative surface charge will result in depression of the resting T-P segment (Figure 5). Since all ECG machines take the T-P segment as zero, T-P segment

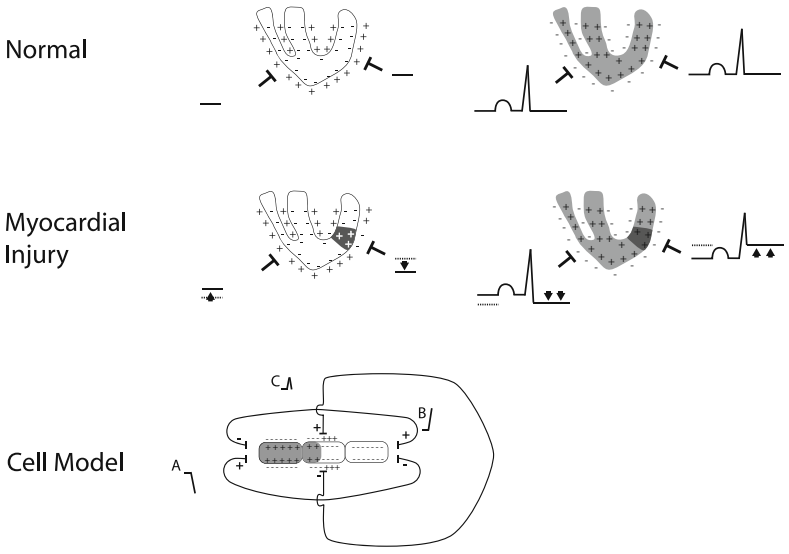


Figure 5: Mechanism for ST segment changes during myocardial infarction. Under normal conditions (*top row*), at rest and after depolarization during the phase 2 plateau, there are no voltage differences present and the T-P segment and the ST segment are isoelectric. If a portion of the heart is injured due to ischemia (*middle row*), depolarization of the membrane leads to T-P segment depression in leads where the positive electrode directly overlies the injured myocardium. T-P segment depression is produced because of the relatively negative surface charge. Think about the three-cell system of depolarization introduced in Chapter 2 (*bottom row*). The lead overlying the injured area would be similar to lead system A and the lead overlying normal tissue would be similar to lead system B. Since the ECG machine uses the T-P segment as "zero," T-P segment depression produces ST segment elevation when the ventricles are at the plateau phase.

depression will cause ST segment elevation. Conversely, for a positive electrode "looking" at this region through normal tissue, T-P elevation will be observed. T-P elevation in these "distant" leads will produce ST segment depression. In the setting of a myocardial infarction associated with ST segment elevation, ST segment depressions are usually called "reciprocal changes" in order to emphasize that both the ST segment elevation and depression arise from the same process.

In addition to changes during diastole, activation of I_{KATP} will also have effects on the action potential itself during systole. Activation of I_{KATP} will cause earlier repolarization of epicardial tissue compared to M cells and endocardial tissue, because epicardial cells have a greater response to low ATP (due either to a larger number of channels that are responsible for I_{KATP} or to channels with different gating characteristics). Earlier repolarization of the epicardium will be associated with ST segment elevation (Figure 6). One can think of the changes as "an early and exaggerated upright T wave."

From the foregoing discussion, it is obvious that ST segment elevation during myocardial infarction is a complex process. However, regardless of whether ST segment elevation is due to diastolic or systolic changes in ion concentrations and gradients, careful review of the ECG with particular attention to the location and magnitude of ST segment elevation and depression can yield important clinical information such as the affected region and the likely site of occlusion.

Complete and sustained occlusion of a coronary artery with development of a myocardial infarction will lead to ST segment elevation in the electrodes that are overlying the injured area, and to ST depression (reciprocal changes) in leads that are looking at the injured area "from a distance" through normal tissue.

At this point, a brief review of the blood supply route to the heart (coronary artery anatomy) is useful. Remember that there are left and right coronary arteries

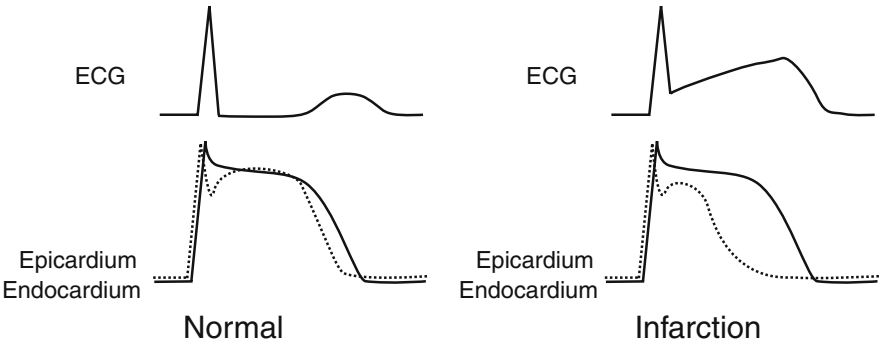


Figure 6: Systolic current of injury. Activation of I_{KATP} leads to greater abbreviation of the epicardial action potential compared to endocardial cells, since epicardial cells, have a greater sensitivity to low ATP: more channels or more sensitive channels.

(Figure 2). The left coronary artery starts out as a single trunk (left main coronary artery) that quickly bifurcates into an artery that travels over the front of the heart (left anterior descending coronary artery) and an artery that travels along the side of the heart in the groove between the left atrium and the left ventricle (circumflex coronary artery). The left anterior descending artery gives off septal branches that supply the interventricular septum, and diagonal branches that supply the anterolateral portion of the left ventricle. The circumflex coronary artery has large branches called obtuse marginals. (This side of the heart is historically called the obtuse side since it forms an obtuse angle relative to the diaphragm.)

The right coronary artery travels in the groove formed by the right atrium and the right ventricle and gives off branches that supply the right ventricle (acute marginal branches), and then divides into the posterior descending artery and the posterolateral branches.

The specific pattern for ST segment elevation and depression in different types of myocardial infarction is discussed in the next section. However, it is worth reemphasizing the importance of reciprocal changes for identifying ST segment changes due to myocardial injury. The presence of reciprocal changes in the setting of localized ST segment elevation is a very specific finding for myocardial infarction ($> 90\%$). As discussed in the next chapter, reciprocal ST segment depression is seen only in lead aVR in pericarditis and is usually not observed in early repolarization.

Coexistence of ST segment elevation and ST segment depression (reciprocal changes) in a 12-lead ECG should always arouse suspicion of myocardial injury.

Q waves

A Q wave is defined as an initial negative deflection in the QRS complex. A Q wave would be expected in lead aVR since depolarization occurs from right to left. An abnormal Q wave would be a negative deflection in a lead that is "unexpected." Abnormal Q waves can arise from a number of causes that will be reviewed in the final chapter. However, one of the most important causes for an abnormal Q wave is myocardial infarction. In a patient with a myocardial infarction, the affected area will contribute less to depolarization, so that an electrode overlying the affected area will record a negative deflection due to measurement of "unopposed" depolarization from the opposite wall (Figure 7). For this reason abnormal Q waves due to myocardial infarction will be observed in a coronary artery distribution (inferior, anterior, or the lateral leads), and should be identified in at least two contiguous leads. Abnormal Q waves should be at least 1 mm deep and > 0.03 s (approximately 1 "little box" x 1 "little box"). Q waves can develop within the first few minutes/hours of myocardial injury, but usually begin becoming more prominent approximately twelve hours after the initial coronary artery occlusion. In leads that develop Q waves, an accompanying decrease in the amplitude of the R waves will sometimes be observed.

Traditionally, Q waves were thought to develop when irreversible damage to the heart occurred, but it has become apparent that Q waves can be observed in the

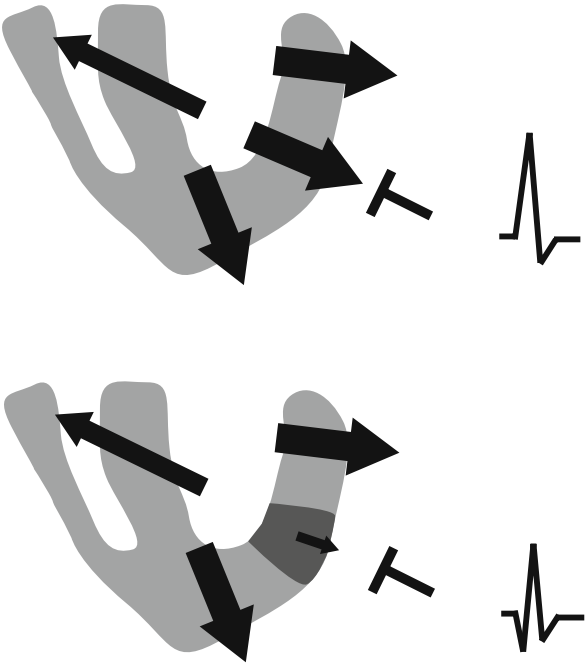


Figure 7: Genesis of Q waves. In the presence of myocardial infarction, the injured portion of the heart will develop a reduced force of depolarization that will lead to an initial negative deflection (Q wave) in an electrode that is overlying the injured area.

acute process of a myocardial infarction and will sometimes resolve if reperfusion is established in a timely fashion. In fact, the incidence of myocardial infarctions associated with Q waves has decreased from 65–70% to 35–40% with the modern treatment emphasis on early reperfusion. In addition, Q waves will regress and sometimes resolve completely in 25–65% of cases.

T wave inversion and ST segment depression

Clinically, myocardial infarctions are usually classified by whether ST segment elevation is present (ST segment Elevation Myocardial Infarction or STEMI) or not (non-STEMI). The distinction is somewhat arbitrary, although useful clinically, since ST segment elevation is often present when there is complete occlusion of one of the major branches of the coronary arteries (left anterior descending, circumflex, or right coronary artery). Non-STEMI myocardial infarctions are often due to transient occlusion of one of the major coronary arteries or transient or total occlusion of one of their branches. The ECG changes associated with non-STEMI infarctions can be subtle and include ST segment depression and T wave inversion (Figure 8).

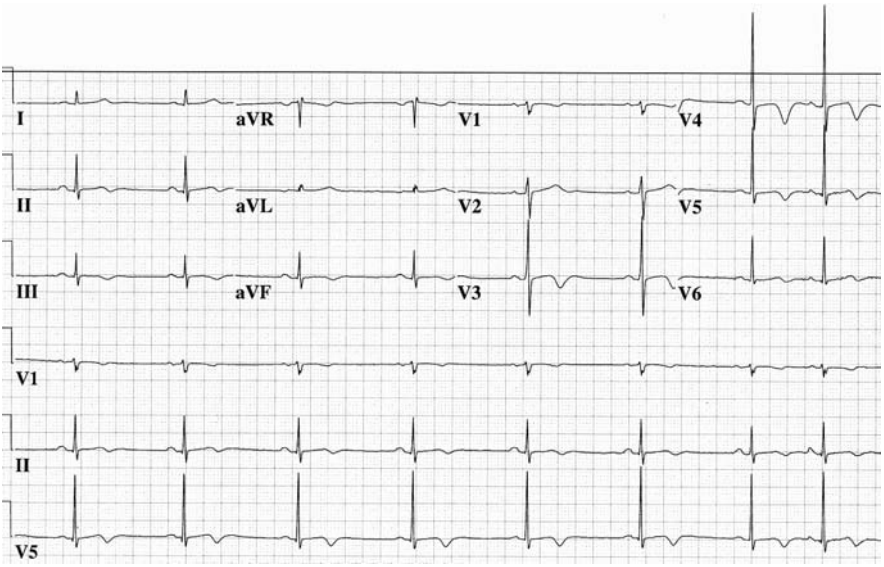


Figure 8: Anterior T wave inversion in the anterior, lateral, and inferior leads in a patient with a significant narrowing in the right coronary artery producing myocardial ischemia. Even though the inferior wall was at risk, diffuse T wave changes were observed. Unlike ST segment elevation, location of the T wave changes provides little information on the location of the "culprit" lesion.

ST segment depression may be due to endocardial ischemia. The major coronary arteries travel over the epicardial surface, and smaller branches penetrate the heart wall to provide flow to the endocardial layers. Since it is "downstream," the endocardium is generally affected more than the epicardium when insufficient coronary artery flow is present. In this case, ST segment depression might be observed, because the positive electrode overlying the injured area "sees" the more ischemic endocardial tissue through less ischemic epicardial tissue. Unfortunately isolated ST segment depression without accompanying ST segment elevation is not very specific for identifying acute myocardial infarction, since ST segment depression is much more commonly observed. In particular, ST segment depression associated with left ventricular hypertrophy can be difficult to separate from ST segment depression associated with myocardial injury.

However, there are several clues that can be used when interpreting the ECG in a patient with isolated ST segment depression. First, ST segment depression associated with chest pain should always arouse suspicion of myocardial ischemia. Second, ST segment depression associated with left ventricular hypertrophy is usually located in the lateral leads (I, aVL, V_5, and V_6). Suspicion of ischemia as a cause of ST segment depression should always be higher if the ST segment depression is located in other leads. For example, isolated ST segment depression in the anterior leads V_2 and V_3 has been associated with occlusion of the circumflex, since the leads are looking at the damaged lateral wall through normal myocardium.

Third, the ST segment depression associated with left ventricular hypertrophy has a downsloping appearance from the end of the QRS complex to the T wave. If present, horizontal ST segment depression rather than downsloping ST segment depression is more characteristic for ischemia/infarction, but is also a relatively nonspecific finding. Finally, dynamic ST segment depression strongly suggests underlying ischemia with transient occlusion and reperfusion of a coronary artery. Reexamine the ECGs in Figure 4. Notice that the ECG obtained during chest pain also has subtle ST segment depression in V_3–V_6 (ST depression in V_3 and V_4 would be unexpected for ST segment depression associated with left ventricular hypertrophy) and in inferior leads that resolves when the patient's chest pain resolves (transient blood flow through the coronary artery is restored). It is important to remember that dynamic changes in the ST segment or T waves observed on an ECG associated with symptoms should always arouse suspicion of an underlying cardiac condition.

Remember to always evaluate the ECG in the context of a patient's symptoms. The presence of dynamic changes in the ECG should always be evaluated if possible.

Although they are abnormal, inverted T waves, like ST segment depression, are a nonspecific finding for ischemia. Inverted T waves can be observed in any condition associated with repolarization abnormalities, including left ventricular hypertrophy. In addition, T wave inversion in the anterior leads can be a normal variant. In general, the same rules for ST segment depression apply for evaluation of T wave inversion. The presence of symptoms, the presence of T wave inversion in leads other than the inferolateral leads, and dynamic T wave changes should always increase the clinician's concern that the T wave inversion is due to myocardial ischemia. However, even in the presence of these findings, patients will not always have myocardial infarction/ischemia. In a group of patients presenting with chest pain and isolated T wave inversion, 60% were found to have myocardial ischemia, 5% had left ventricular hypertrophy, and the remaining 35% had no significant cardiac disease.

Prominent and deeply inverted T waves can be associated with myocardial infarction. In fact, deep symmetric T wave inversion in the anterior chest leads (V_2 and V_3) can be associated with significant narrowing in the left anterior descending artery, and is sometimes called Wellens' sign, for Heins Wellens, who described it initially. However, even prominent T wave inversion can have multiple diverse causes, including pulmonary embolism, antiarrhythmic drug use, central nervous system injury, and can even be a normal variant. The general consensus among experts is that deeply inverted T waves in the setting of myocardial injury/ischemia usually suggest the presence of viable myocardium and usually portend a better prognosis.

In the presence of an ST segment elevation myocardial infarction, there is a general evolution of T wave morphology and ST segment deviation from baseline (Figures 9, 10, and 11). Initially the T waves will be upright, and after time the T waves will invert, while the ST segment is still elevated. There is some data to suggest that T wave inversion develops with reperfusion and that its presence is associated with an improved prognosis. Gradually the ST segment elevation resolves,

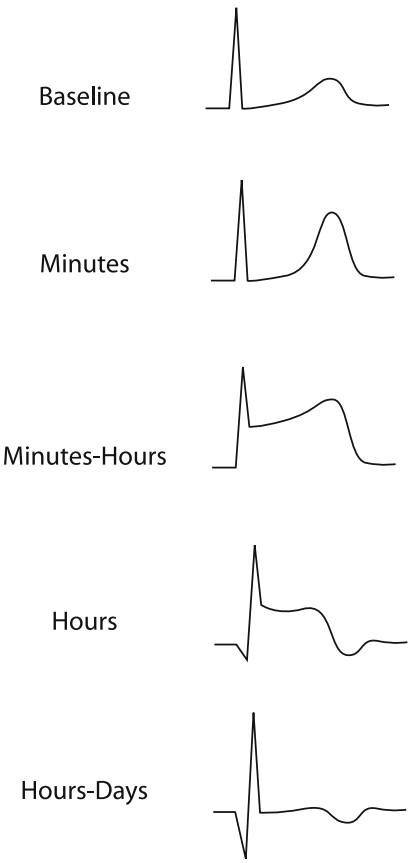


Figure 9: The evolution of ECG changes during an ST elevation myocardial infarction. In the first minutes peaked T waves can be observed; after several minutes to the first several hours, ST segment elevation with upright T waves can be seen. During the later hours, T wave inversion with partial resolution of ST elevation and Q wave formation is observed. Over subsequent days, the Q waves deepen, the ST segment returns to normal, and the T wave is inverted. It is important to keep in mind that all of these stages will not be observed in all patients.

but the T wave remains inverted. This sequence is different in pericarditis, where the ST segment often will normalize before the T waves become inverted. The presence of T wave inversion with concomitant ST segment elevation would be a very unusual finding in pericarditis and should arouse suspicion of a myocardial infarction. Over time, T wave inversion associated with myocardial infarction will sometimes resolve. Normalization of T waves after a myocardial infarction appears to be a marker for improvement in left ventricular function and an indicator of a better prognosis.

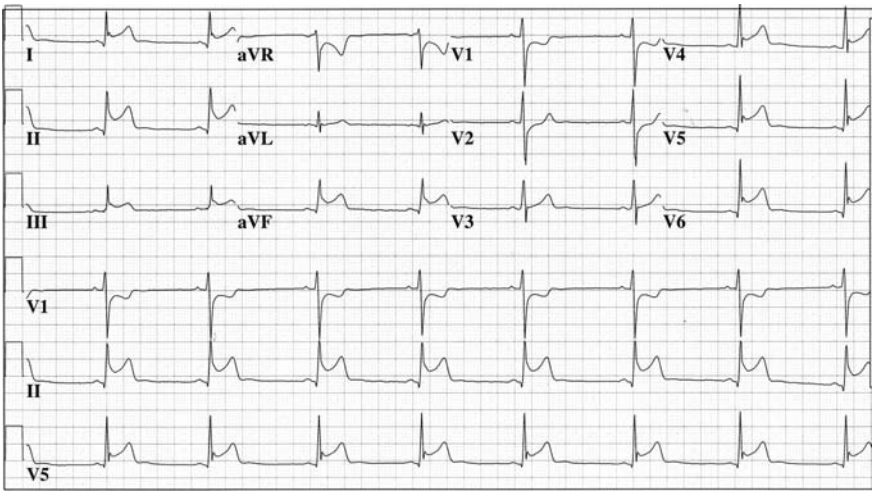


Figure 10: An ECG from a patient with a large inferior and lateral wall myocardial infarction. Notice the significant ST segment elevation in the inferior and lateral leads. Since the ECG is acquired early in the myocardial infarction, the T waves remain upright and very small insignificant Q waves are noted in the inferior and lateral leads.

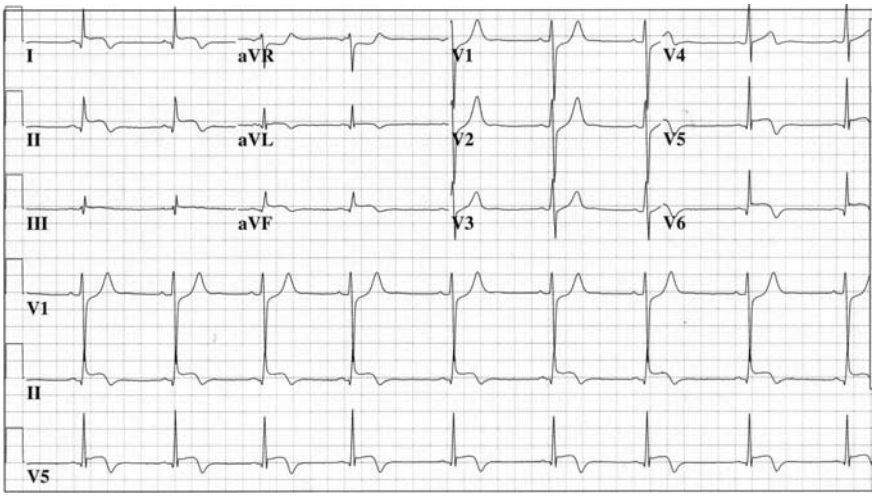


Figure 11: An ECG from the same patient as in Figure 10 on the second day after a myocardial infarction. Notice that the ST segment elevation is gradually resolving and the T waves have become inverted.

Anterior infarction

Occlusion of the left anterior descending coronary artery will cause ST segment elevation in the anterior precordial leads (Figure 12). Usually, leads V_1, V_2, V_3, and V_4 are considered the anterior precordial leads. The presence of ST segment elevation in two adjacent leads is used for the diagnosis of an anterior myocardial infarction. The number of leads involved provides a clue for the location of the occlusion and the extent of the myocardium at risk. The major branches of the left anterior descending coronary artery are septal perforators that supply the septum, and diagonal branches that supply the anterolateral wall. In general, the branch points of the first septal perforator and first diagonal branch are very close to each other. If the blockage is past the first diagonal branch, ST segment elevation will normally be present in V_3 through V_5 (Figure 13). However, if the blockage is in the proximal portion of the left anterior descending coronary artery prior to the branch-off of the septal perforator and diagonal ST segment, elevation will be observed in the lateral leads (aVL, I, and V_6) and the anterior leads because of injury of the anterior and anterolateral walls (Figure 12). Thus the general location of the coronary occlusion and the myocardium at risk can be estimated by the extent of ST segment elevation. It is intuitively obvious that patients with larger myocardial infarctions have a higher short-term risk of death and complications and poorer long-term prognosis.

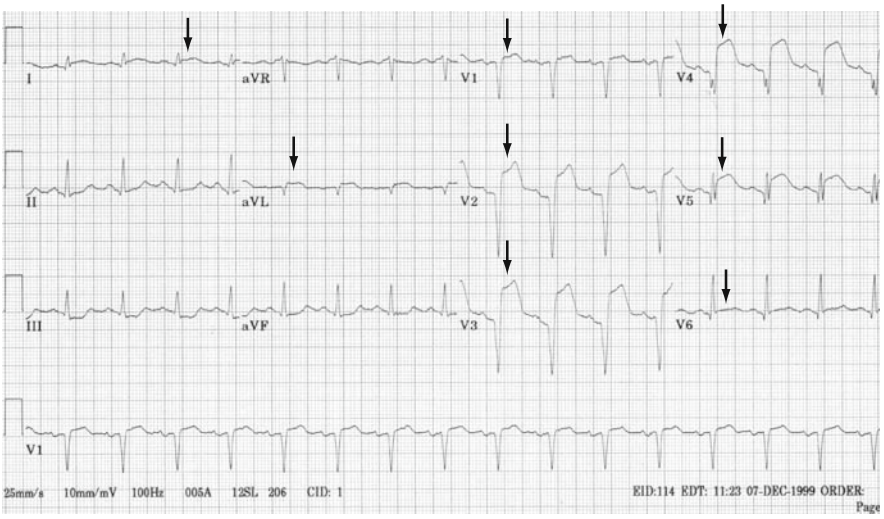


Figure 12: Anterior myocardial infarction involving the proximal left anterior descending artery. Although the ST segment elevation is most prominent in V_1–V_6 (arrows), ST segment elevation is observed in all of the anterior leads and I and aVL (arrows). Notice the reciprocal ST segment depression in the inferior leads. Finally, the patient has abnormal Q waves in the anterior leads and aVL.

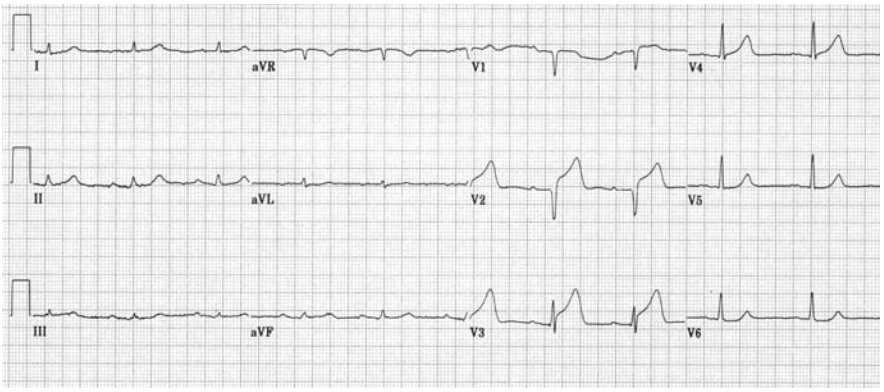


Figure 13: Anterior myocardial infarction due to occlusion in the distal left anterior descending coronary artery. Since the occlusion is beyond the first diagonal artery, ST segment elevation is observed only in the anterior leads V_1–V_3 with associated Q waves in V_1 and V_2.

In some cases the left anterior descending coronary artery will "wrap around" the apex and supply the distal portion of the inferior wall. In this case, occlusion of the left anterior descending can be associated with anterior ST segment elevation and ST segment elevation in the inferior leads. Again, the number of leads that display ST segment elevation provides an estimate for the amount of jeopardized myocardium.

Unless the anterior descending supplies the inferior apical walls, reciprocal ST segment depression will usually be observed in the inferior leads. The absence of inferior reciprocal ST depression is not necessarily a better prognostic sign. The absence of accompanying inferior ST segment elevation may be due to associated injury of the inferior wall (since the leads are looking at the anterior wall through a relatively injured inferior wall). Compare the reciprocal changes in the inferior leads in the anterior wall myocardial infarctions shown in Figures 12–14. Interestingly, the patients with the absence of reciprocal inferior ST depression (Figures 12 and 13), had left anterior descending arteries that went completely to the apex, while the patient in Figure 14 had a left anterior descending artery that did not reach the apex.

Myocardial infarction not only is associated with changes in repolarization but can also affect depolarization. Damage to an area produces Q waves in the area because of relative damage to the heart and reduction in depolarization voltages in the affected area compared to normal myocardium not affected by the coronary occlusion. Q waves were generally thought to represent irreversible damage to the heart and a relatively late manifestation of myocardial infarction. However, it is now recognized that Q waves can be present early in myocardial infarction (so called "hyperacute" Q waves) and can resolve completely with aggressive treatment to reestablish coronary artery blood flow.

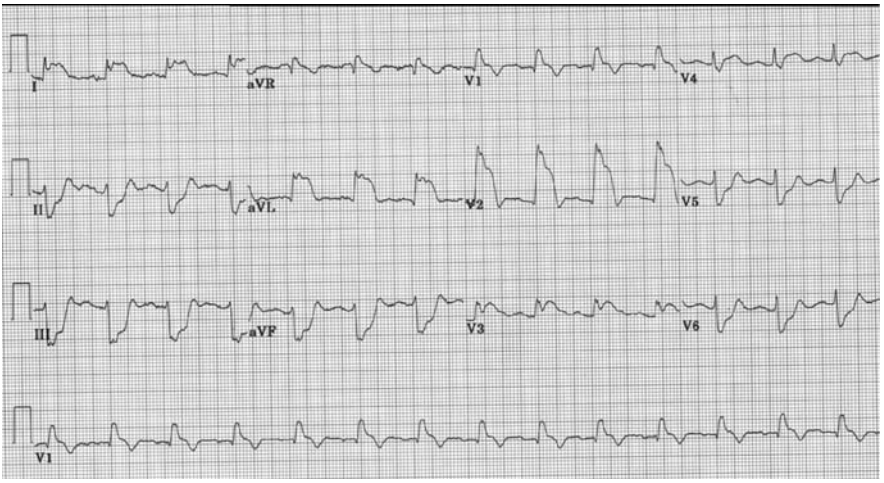


Figure 14: Prominent ST segment elevation due to a proximal lesion in the left anterior descending artery (notice the ST segment elevation in I and aVL). The ST segment elevates larger than the QRS complexes. This ECG pattern is often referred to colloquially as "tombstones" due to the obvious appearance in the anterior leads and the life-threatening situation. Notice that the patient has accompanying right bundle branch block and left anterior fascicular block. The right bundle and left anterior fascicle usually receive blood from the first septal perforator of the left anterior descending coronary artery.

The right bundle branch and the left anterior fascicle usually receive their blood supply from the first septal perforator. Right bundle branch block due to an anterior wall myocardial infarction occurs in approximately 5–10% of anterior wall myocardial infarctions and should arouse suspicion of a proximal lesion in the left anterior descending coronary artery (Figure 14). Left anterior fascicular block also develops in 7–15% of anterior wall myocardial infarctions, frequently in conjunction with right bundle branch block since they have a similar blood supply.

With evolution of the myocardial infarction, the ST segment abnormalities gradually resolve and the T wave frequently becomes inverted. If Q waves are present after the first 24–48 h after the myocardial infarction, they often become less prominent over time (weeks and months), but will generally not resolve completely.

Inferior myocardial infarction

Occlusion of the right coronary artery will cause injury to the inferior wall of the heart and lead to ST segment elevation in the inferior leads (II, III, and aVF) (Figure 15). In most patients the posterior descending coronary artery branches from the right coronary artery ("right dominant"). Approximately 10% of people are

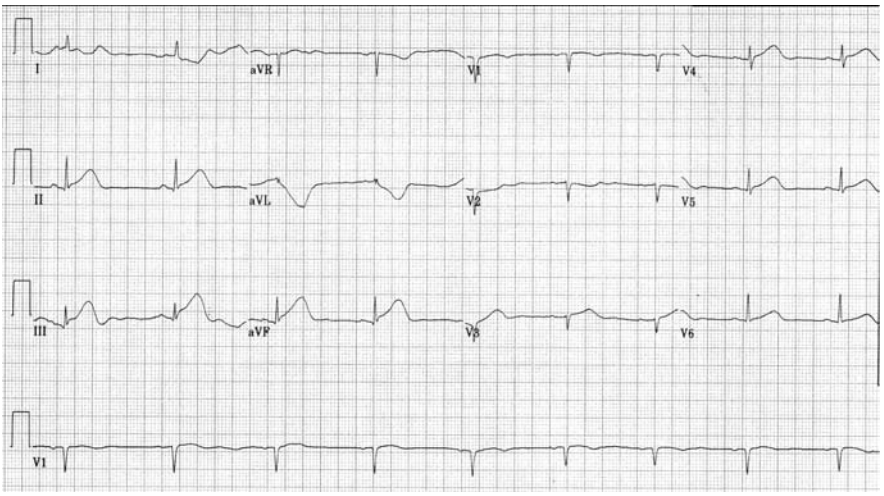


Figure 15: Inferior wall myocardial infarction due to occlusion of the right coronary artery. ST segment elevation in the inferior leads. Notice the reciprocal ST depression in aVL.

"left dominant" and the circumflex coronary artery supplies the posterior descending artery and the inferior wall. In patients where the inferior wall myocardial infarction is due to occlusion of the right coronary artery, the ST segment elevation in lead III is usually more prominent than that in lead II, since lead III "looks" at the left ventricle from a more rightward perspective. In addition, reciprocal ST depression is usually prominently seen in I and aVL, since these lateral leads "look" at the infarcted region through normal tissue (Figure 15). Conversely, inferior wall myocardial infarction associated with occlusion of the circumflex coronary artery will generally have prominent ST segment elevation in lead II compared to the other inferior leads, with a relative lack of reciprocal changes in leads I and aVL.

Generally, the inferior wall is considered the portion of the left ventricle that lies "on top" of the diaphragm. In the 1960s the portion of the inferior wall closest to the spine (adjacent to the mitral valve) was further differentiated as the posterior wall. Although there have been recent attempts to remove or suppress this terminology, the classification "posterior myocardial infarction" remains in common clinical use. This region of the heart is usually supplied by posterolateral branches that often form the most distal portion of the right coronary artery. By ECG criteria, a posterior myocardial infarction is present when a prominent R wave is noted in lead V_1 (Figure 16). In addition to the prominent R wave, posterior myocardial infarction is usually associated with significant ST segment depression and an upright T wave. Since lead V_1 looks almost directly opposite this wall, one has to flip and take the mirror image of the lead to see what a virtual electrode that is directly over this wall

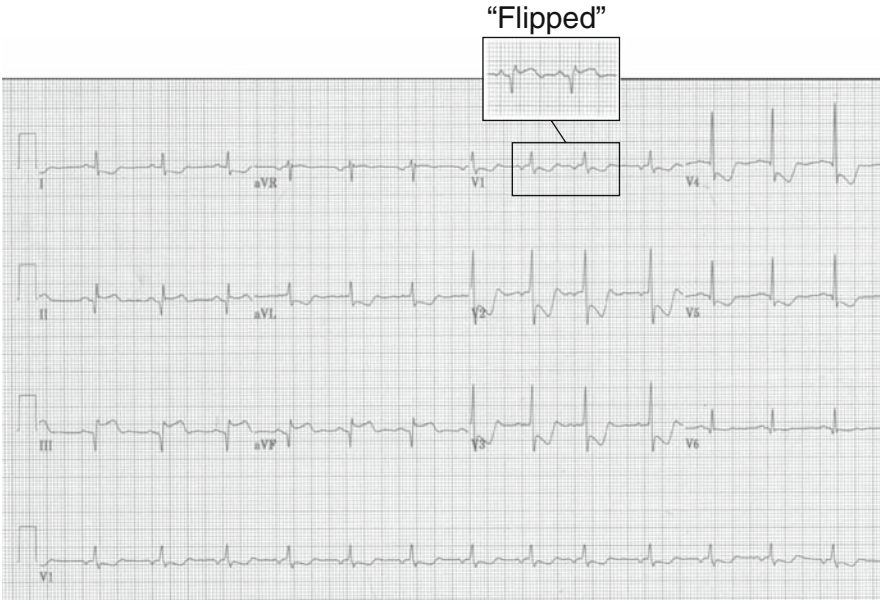


Figure 16: Posterior wall myocardial infarction in conjunction with an inferior wall myocardial infarction. The posterior wall is suspected because of the prominent R wave in V_1 with associated ST segment depression. *Inset*: if the ECG is inverted (since V1 is looking at a mirror image of V_1), the ECG changes in V_1 really represent a Q wave with ST segment elevation and T wave inversion.

would "see." In this way it can be noted that the prominent R wave actually represents a Q wave, the ST depression actually represents ST elevation and the upright T wave is actually an inverted T wave. It is important to keep in mind that an accompanying right bundle branch block may also be associated with a prominent R wave in V_1. However, a right bundle branch block will be associated with a terminal S wave in V_6 and a wide QRS complex.

Case Study (continued): Mr. Arbuckle has ST segment elevation in the inferior leads. Accompanying reciprocal ST depression in aVL suggests that the occlusion is in the right coronary artery. He is taken to the cardiac catheterization laboratory where of an occlusion of the proximal right coronary artery is found.

Lateral myocardial infarction

True lateral wall myocardial infarction is due to occlusion of the circumflex coronary artery. The lateral leads are usually considered to be I, aVL, V_5, and V_6.

Unfortunately, the ECG is least sensitive to myocardial infarction involving the circumflex coronary artery. In many cases occlusion of the circumflex coronary artery or one of its branches will not be associated with ST segment elevation, since the traditional 12-lead ECG records most laterally from the midaxillary line. For this reason some clinicians have advocated using a larger number of leads, with additional leads oriented around the left back.

Lateral infarction can often be seen in the presence of an inferior wall myocardial infarction (Figure 17). In the presence of a right dominant system with extensive posterolateral branches, occlusion of the right coronary artery will often lead to ST segment elevation in the inferior and lateral leads (predominantly V_5 and V_6). In a patient with a left dominant system, occlusion of the circumflex coronary artery will also lead to inferolateral ST segment elevation. As discussed above, the presence or absence of matching reciprocal ST segment depression in aVL can still be helpful for differentiating between these two possibilities (present: right coronary artery, absent: circumflex coronary artery). Compare Figures 15 and 17. Both represent inferior and lateral wall myocardial infarctions, but occlusion of the circumflex artery (Figure 17) is associated with absent reciprocal changes in leads I and aVL. In addition, compare the ST segment elevation in each of the inferior leads. In an inferior wall myocardial infarction due to right coronary artery occlusion, equal ST segment elevation is observed in II, aVF, and III. In contrast, in an inferior wall myocardial infarction due to occlusion of the circumflex artery, ST segment

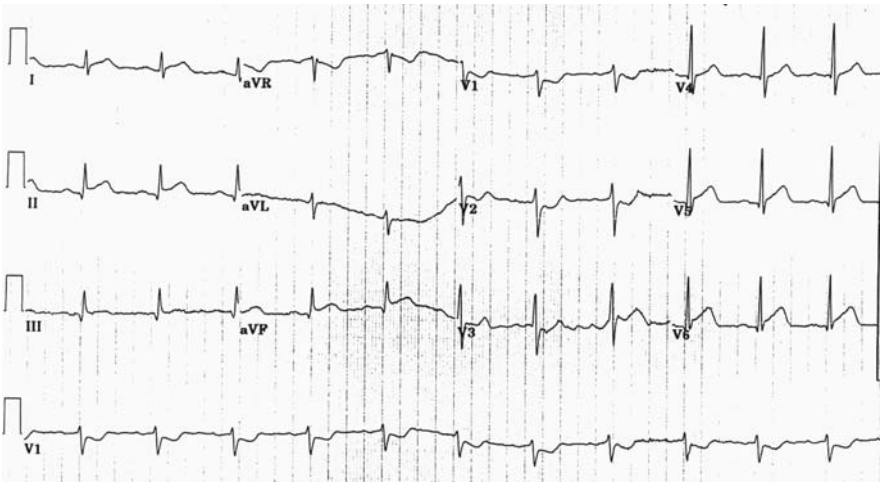


Figure 17: Inferior wall myocardial infarction due to occlusion of the circumflex coronary artery. Notice that reciprocal ST segment depression is not prominent in I and aVL. The ST segment elevation in III is less prominent than the ST segment elevation in II.

elevation is more prominent in lead II when compared to aVF, since II is oriented at 60° and III is oriented farther away from the lateral wall at 120°.

Right ventricular infarction

The right coronary artery gives off an acute marginal branch(es) that supply the right ventricle. Proximal occlusion of the right coronary artery can cause myocardial infarction of the right ventricle (Figure 18). The precordial leads V_1 and V_2 overlie the right ventricle. In most cases reciprocal ST segment depression is observed in V_1 and V_2 in the setting of an inferior wall myocardial infarction, since these leads are looking at the inferior wall through normal tissue. However, if a proximal occlusion of the right coronary artery causes both an inferior wall and right ventricular infarction, lack of reciprocal changes or even frank ST segment elevation will be observed in leads V_1 and V_2 due to injury of the right ventricle (Figure 15).

Another method for detection of a right ventricular infarction is to use right-sided precordial leads. To place right-sided leads, precordial lead RV_1 is placed where the normal lead V_2 should be (left side of the sternum, fourth intercostal space), and lead RV_2 is placed where the standard lead V_1 is placed. The remainder of the leads are placed on the right chest using the analogous landmarks for the standard precordial leads. ST segment elevation in RV_4 is a relatively insensitive but very specific finding in the setting of right ventricular involvement. Figure 19 shows right-sided precordial leads in the patient with the inferior wall myocardial infarction shown in Figure 15. As would be expected, the QRS becomes predominantly negative as one moves laterally to the right. However, ST segment elevation in RV_4 is an excellent ECG sign for the presence of a right ventricular infarction.

It is important that any time an inferior wall myocardial infarction is present, the presence of accompanying right ventricular infarction or extension to the posterior wall should be considered. Evaluation of the reciprocal changes in V_1 and V_2 can provide clues to this important clinical question. In general, the amount of ST segment depression observed in V_1 and V_2 should match the amount of ST segment elevation in the inferior leads. If the reciprocal ST depression is less than expected, one should consider the presence of a right ventricular infarction. Conversely, if the reciprocal ST depression in V_1 and V_2 is more prominent than the inferior ST segment elevation, accompanying posterior wall myocardial infarction should be considered. It is important to always keep in mind the concept of canceling forces and realize that ECG interpretation is more art than algorithm. In the presence of a proximal occlusion of the right coronary artery that has large posterolateral branches, the utility of the reciprocal anterior ST segment will be reduced because the right ventricular infarction will tend to reduce the reciprocal ST depression, while the posterior wall involvement will tend to exaggerate the ST segment depression in the same leads. What is ultimately recorded will depend on the amount and location of the myocardium in jeopardy, the presence or absence of collateral circulation, the relative orientation of the heart within the body, and a number of other factors.

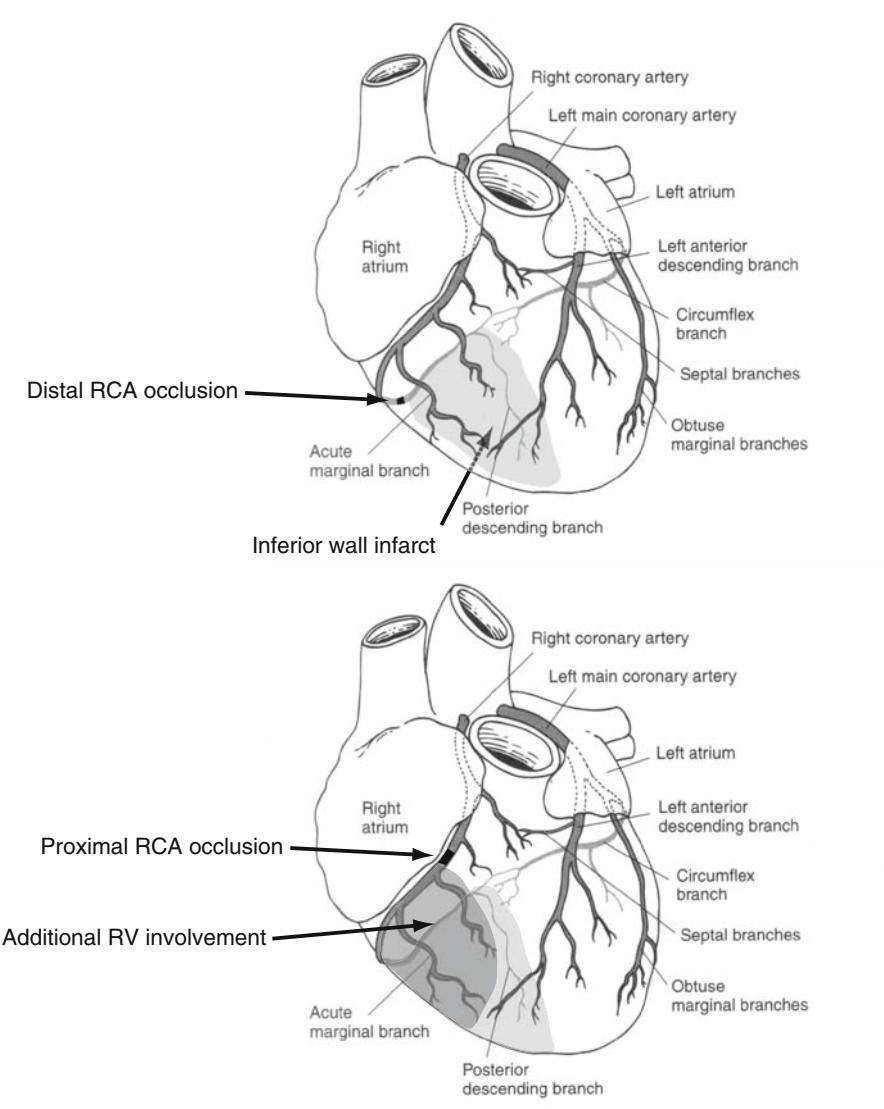


Figure 18: Occlusion of the right coronary artery can develop proximally or distally. In a more distal occlusion (*top*), the inferior wall of the left ventricle is at risk and ST segment elevation is observed in the inferior leads. If the occlusion develops more proximally, additional regions of the right ventricle do not receive blood. In general, proximal lesions are defined as those before the acute marginal branches (that supply the right ventricle), and distal occlusions are those that occur after the acute marginal branch(es) split off.

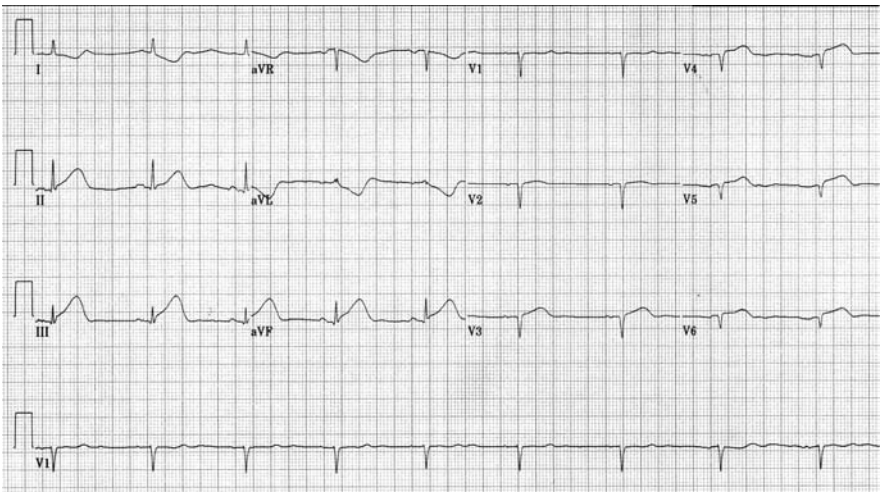


Figure 19: Same patient as in Figure 13 but with right-sided leads. The frontal leads are the same but the precordial leads are positioned in the exact opposite orientation from normal left precordial leads: V_1r is the same as the conventional V_2, V_2r is the same as the conventional V_1, but the remainder of the leads are unique to the right-sided ECG. The normal right-sided QRS complexes will be QS complexes, but the ST segment elevation is abnormal. Lead rV_4 is the most commonly evaluated lead. ST segment televation rV4 confirms the presence of right ventricular myocardial infarction due to a proximal occlusion in the right coronary artery.

Left main

The left coronary system begins with the left main coronary artery, which then usually divides within 1–2 cm into the left anterior descending and left circumflex. Although relatively rare, it is important to recognize this potentially catastrophic problem. Obstruction of the left main results in diffuse ischemia of the left ventricle. Since it is more distal in the watershed area, endocardial ischemia tends to be more prominent than epicardial ischemia and for this reason most of the ECG leads (I, II, III, aVL, aVF, V_1–V_6) look at "more injured" tissue through "less injured" tissue, and diffuse ST segment depression is often observed (Figure 20). The one exception is aVR-since the left ventricle can be considered as a "pot" with its opening directed to the right and posterior, aVR is the only lead that "looks" directly at the endocardium (Figure 21). This is why diffuse ischemia associated with left main occlusion/ischemia will be associated as elevation in aVR (Figure 20). Thus isolated ST segment elevation in aVR should raise concern for occlusion or narrowing of the left main coronary artery. In addition to lead aVR, in some cases, depending on the orientation of the left ventricle, lead V_1 also can "look" at the endocardial surface, and ST segment elevation will be observed in V_1 with occlusion of the left main artery.

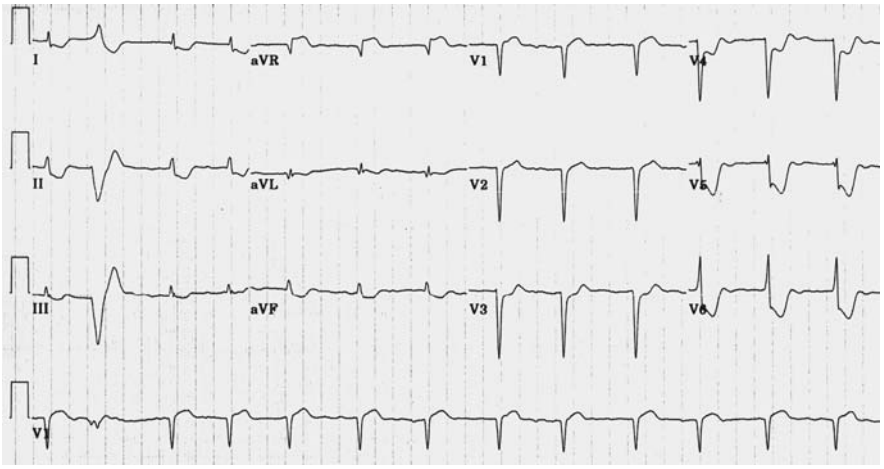


Figure 20: ECG in a patient with a 95% blockage in the left main coronary artery. ST segment elevation is observed in aVR and V_1. In the remainder of the leads, ST segment depression is observed. Profound ST segment depression is observed in the anterolateral leads (V_4–V_6).

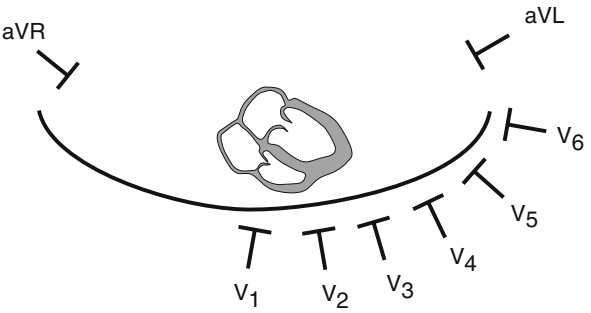


Figure 21: Relative orientation of aVR compared to the other precordial leads. Lead aVR "looks" directly at the endocardium.

Myocardial infarction in the presence of bundle branch block

It is very difficult to determine whether an acute myocardial infarction is present when a patient has left bundle branch block. The most diagnostic finding is the presence of $\geq$1 mm ST segment elevation associated with a positive QRS complex. Remember that left bundle branch block is associated with repolarization changes at baseline. In general, the ST segment and T wave will be in a direction opposite the QRS complex so that a negative QRS complex is associated with ST segment elevation and a positive QRS complex is associated with ST

segment depression. For this reason ST segment elevation would be unexpected in the setting of a positive QRS complex. Similarly ST segment depression ≥ 1 mm in the right precordial leads (V_1 through V_3) should arouse suspicion of myocardial ischemia/infarction in a patient complaining of chest pain. In the top part of Figure 22, a patient with chest pain and left bundle branch block is shown. There is significant anterior ST segment depression in leads with predominantly negative

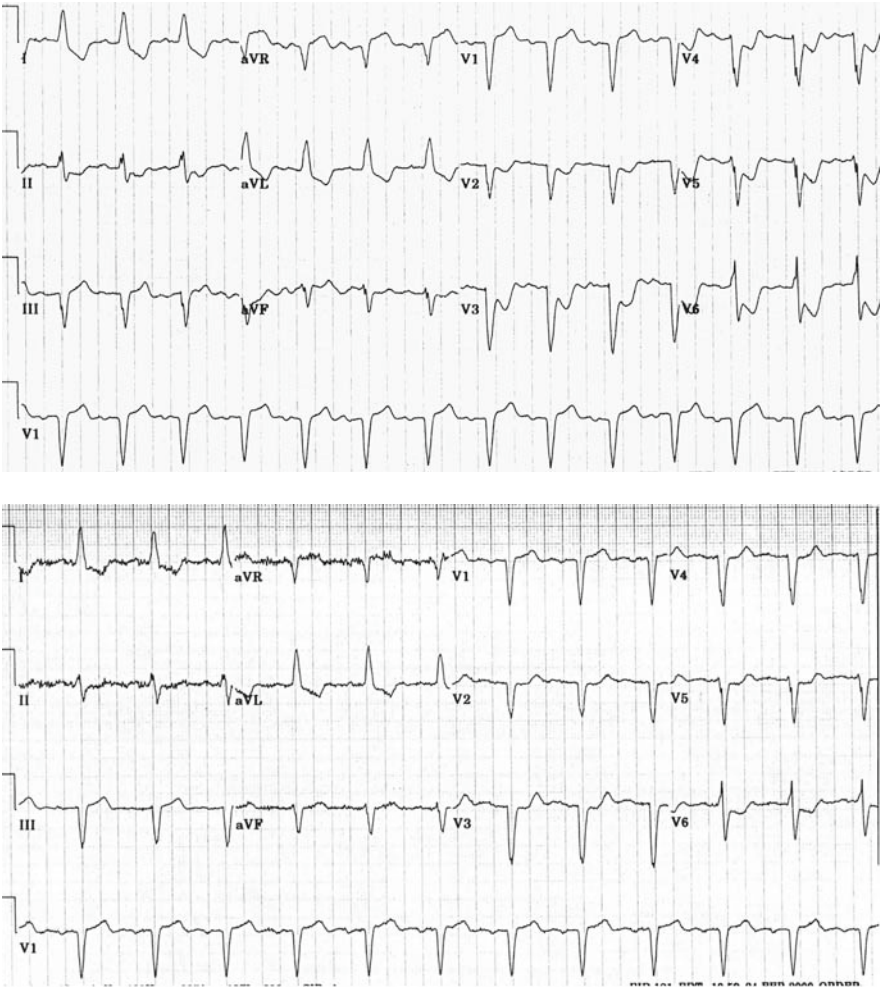


Figure 22: *Top*: Patient with left bundle branch block. *Top*: ECG obtained while the patient has chest pain. Notice the presence of anterior ST segment depression in leads with negative QRS complexes. Since LBBB typically would be associated with ST segment elevation in leads with negative QRS complexes, the presence of ST depression indicates ischemia. *Bottom*: ECG after the chest pain resolves. Notice that the anterior ST depression has almost completely resolved.

QRS complexes. His chest pain resolves spontaneously (probably due to partial spontaneous resolution of the thrombus) and his new ECG is shown in the bottom panel of Figure 22. The anterior ST segment depression has resolved, highlighting the importance of evaluating the ECG for dynamic changes if possible. A patient with an inferior wall myocardial infarction and left bundle branch block is shown in Figure 23. In this case inferior ST segment elevation would not be expected in leads with positive QRS complexes.

Exaggerated ST segment elevation ($\geq$ 5 mm) should also arouse suspicion of a myocardial infarction, but this is the weakest criteria for myocardial infarction and is present in only 6% of clinically stable patients with left bundle branch block.

Since left bundle branch block is associated with abnormal depolarization, diagnosing old myocardial infarction by identifying Q waves is usually not helpful. However, the presence of a Q wave ($>$ 30 ms) in two or more lateral leads (I, aVL, V_5, or V_6) is a fairly specific finding for the presence of a remote anterior myocardial infarction, since Q waves would generally not be observed in these leads in typical LBBB.

Myocardial infarction is easier to identify in the presence of right bundle branch block. Since initial depolarization of the ventricles is normal, evaluation for Q waves is the same as in patients with a normal QRS complex (Figure 24). Since the right ventricle generally has less mass than the left ventricle, the ST segment changes are usually less prominent in right bundle branch block, and any significant ST segment elevation in a patient with chest pain should arouse suspicion for the presence of a myocardial infarction. Right bundle branch block can be commonly

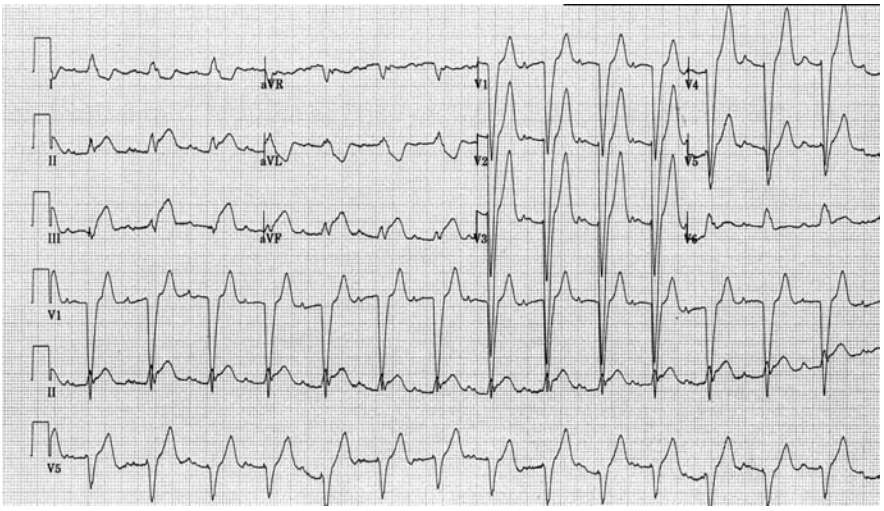


Figure 23: ECG of an inferior wall myocardial infarction in the presence of left bundle branch block. ST segment elevation is evident in the inferolateral leads. In the inferior leads the positive QRS complex would normally be associated with a downsloping ST segment depression.

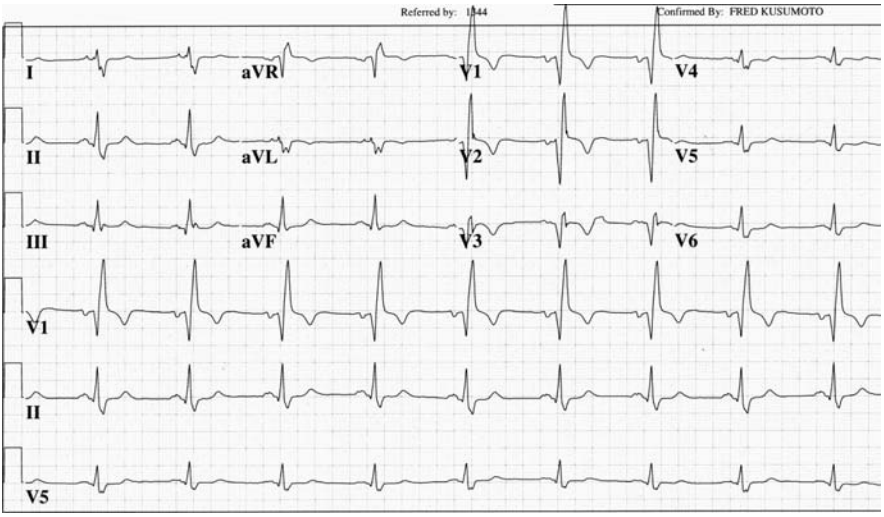


Figure 24: Abnormal Q waves in V_2 and V_3 in the presence of right bundle branch block. In right bundle branch block early activation of the septum is normal, so abnormal Q waves due to a remote myocardial infarction can be assessed by the ECG. A small Q wave in V_1 can sometimes be normally observed, but Q waves in the remainder of the anterior leads are abnormal. In this ECG, abnormal Q waves are present in leads V_2 and V_3.

associated with abnormal T waves, so evaluation of T wave morphology can become problematic.

Diagnosis of myocardial infarction in the presence of left bundle branch block:
Acute: ST segment changes in the same direction as the QRS complex. Exaggerated ST segment elevation (> 5 mm).
Old infarction: Presence of Q waves in two of the lateral leads (I, aVL, V5, V6).

Case Study (continued): Mr. Arbuckle underwent successful angioplasty (a balloon inflated at the site of the coronary occlusion that results in restoration of blood flow). His ECG on the next day is shown in Figure 25. The ECG now shows T wave inversion and the development of Q waves in the inferior leads.

Key points

1. ST segment elevation is an ECG sign of complete coronary artery occlusion and myocardial infarction.
2. ST segment elevation due to myocardial infarction can generally be "localized" to a specific region of the heart supplied by a particular coronary artery: anterior (left anterior descending), inferior (right coronary artery), and lateral (circumflex) (Table 1).

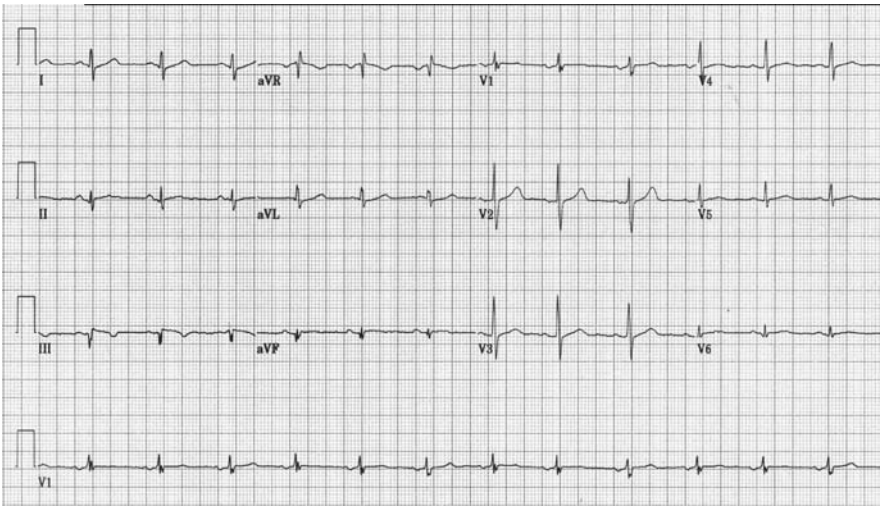


Figure 25: Mr. Arbuckle's ECG on the first hospital day after his myocardial infarction. The ST segment changes have improved. The inferior T waves are now inverted and Q waves have developed in III and aVF.

Table 1: Infarction type and ECG changes

Infarction type	Leads with ST segment elevation	Considerations for ECG interpretation
Inferior	II, III, aVF	Is the right ventricle involved? • Less reciprocal ST depression in V_1, V_2 • ST segment elevation in rV_4 Is the posterior wall involved? • More reciprocal ST depression in V_1 • Prominent R wave in V_1 Is the lateral wall involved? • ST segment elevation in V_5, V_6 RCA or Circumflex? • RCA: Reciprocal ST depression in I, aVL • Cx: Absence of reciprocal ST depression in I, aVL
Lateral	I, aVL, V_5, and V_6	May not have any ST segment elevation
Anterior	V_1, V_2, V_3, and V_4	Is the lesion proximal? • ST elevation in I, aVL Is the inferior apex involved? • ST elevation in II, III, aVF
Left Main	aVR, V_1	Diffuse ST segment depression in the inferolateral leads

RCA: right coronary artery; Cx: circumflex coronary artery

3. Myocardial infarction can also cause changes in the QRS complex, usually an initially negative deflection called a Q wave.
4. Most myocardial infarctions are not associated with ST segment elevation. ST segment depression, T wave inversion, and sometimes no ECG changes may be seen.

Review questions

1. ECG findings associated with myocardial infarction include:

   A. ST segment elevation
   B. ST segment depression
   C. T wave peaking
   D. All of the above.

2. The ECG in Figure 26 shows:

   A. Inferior wall myocardial infarction
   B. Anterior wall myocardial infarction
   C. Lateral wall myocardial infarction
   D. No evidence for a myocardial infarction

3. Atrioventricular block is most commonly associated with:

   A. Inferior wall myocardial infarction
   B. Anterior wall myocardial infarction
   C. Lateral wall myocardial infarction

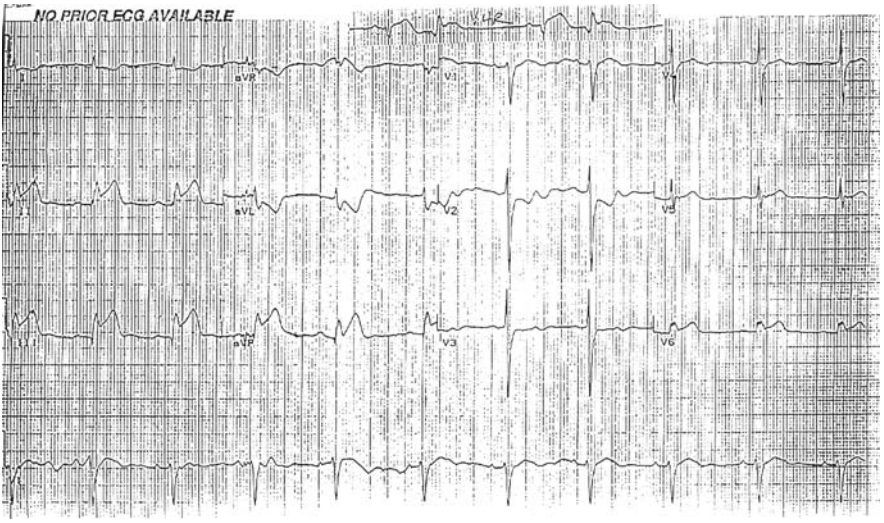


Figure 26: ECG for Problems #2 and #4 (Courtesy Irwin Hoffman).

4. The ECG in Figure 26 is consistent with:
   A. Proximal right coronary artery occlusion
   B. Distal right coronary artery occlusion
   C. Left anterior descending coronary artery occlusion
   D. Circumflex coronary artery occlusion

Answers

1. D. Myocardial infarction can be associated with a number of ECG findings. However, identification of a myocardial infarction associated with ST segment elevation is particularly important (ST elevation myocardial infarction or STEMI), since multiple studies have shown that rapid re-establishment of coronary flow is critical.
2. A. The ECG shows an inferior wall myocardial infarction with ST segment elevation in the inferior wall.
3. A. Inferior wall myocardial infarction is associated with some form of AV block in 1/3 of patients. Usually, the AV nodal artery branches at the same point as the posterior descending artery. A more thorough discussion of AV block in the setting of myocardial infarction is provided in the clinical cases.
4. A. The ECG shows ST segment elevation in the inferior wall with right ventricular infarction (ST segment elevation in RV_4 and absence of reciprocal ST depression in V_1 and V_2). This constellation of findings is consistent only with a proximal occlusion of the right coronary artery.

Chapter 8

ST segment elevation not associated with myocardial infarction

Case: Michael Moore is a 72-year-old man with a long-standing history of hypertension and diabetes. He came to the office with vague complaints of "I do not feel well" but specifically denies chest pain. His ECG is shown in Figure 1.

Although identification of ST segment elevation associated with myocardial infarction is perhaps the most important clinical use for the ECG, there are many other clinical conditions that are associated with ST segment elevation. In fact, it is not uncommon to see small degrees of ST segment elevation in normal ECGs. For example, in a large sample of more than 6000 healthy men in the Unites States Air Force, more than 90% had ST segment elevation of 1–3 mm in the precordial leads. In this chapter the cardiac and noncardiac causes for ST segment elevation and the diagnostic clues for identifying the specific cause for ST segment elevation will be discussed (Table 1).

Although the ECG is an important component for rapidly identifying patients with myocardial ischemia and infarction, it should always be considered a supplement to the clinical history.

Normal ST segment elevation and "early repolarization"

The transition between the QRS complex and the ST segment is usually called the J point (Figure 2). As mentioned above, the J point will be greater than 1 mm from baseline in at least one of the right precordial leads (V_1–V_4) in 90% of young men. In older men the incidence of ST segment elevation appears to be less (30%), but there is no natural history data available that examine the progression of ST changes in a specific person over time. ST segment elevation is less common in women (approximately 5–10%). Generally, in the right precordial leads the ST segment elevation will be associated with QRS complexes that are characterized by deep S waves. The ST segment itself will generally be concave with a slight "sagging" between the J point and the T wave.

Very prominent J point elevation can sometimes be observed as a normal variant and was originally described in the 1940s and 1950s and termed early repolarization. Some but not all epidemiologic studies suggest that early repolarization is particularly prevalent in young African-American men. The prominent J point

F. Kusumoto, *ECG Interpretation: From Pathophysiology to Clinical Application*,
DOI 10.1007/978-0-387-88880-4_8, © Springer Science+Business Media, LLC 2009

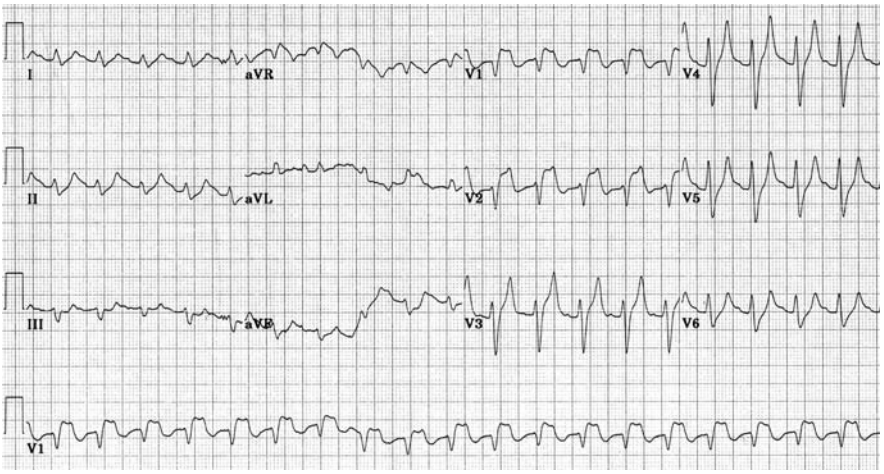


Figure 1: For Mr. Moore ECG.

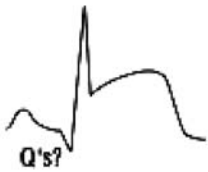
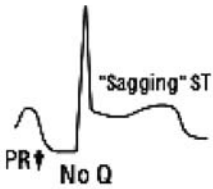
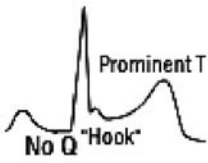
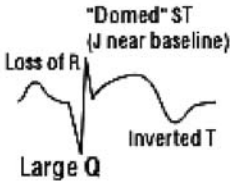
often looks like a "notch" or "hook" (Figure 2). The exact cellular mechanism of the "notch" could be due to either delayed depolarization or early repolarization in a region of the heart. Recent experimental data suggest that a relatively prominent transient outward current (I_{to}) that was discussed in Chapter 6 may be responsible for some of the ECG changes. This transient outward current is largest in epicardial cells from the right ventricle and leads to a prominent phase 1 in the action potential and a "spike and dome" morphology. This leads to a voltage difference at the end of depolarization that can be observed on the surface ECG. The ECG characteristics of early repolarization can be quite varied, but in general can be summarized as precordial ST segment elevation, prominent T waves, and a "notch" or "hook" at the terminal portion of the QRS complex.

Early repolarization has generally been considered benign. However, a recent multicenter study that evaluated 206 patients with unexplained sudden cardiac arrest found that 31% of patients that had undergone cardiac arrest had early repolarization, compared to 5% of matched controls. Recently research has focused on a possible relationship between early repolarization and Brugada Syndrome. Brugada Syndrome is an unusual entity associated with sudden cardiac death and, like early repolarization, is characterized by deflections on the terminal portion of the QRS; this relationship will be discussed later in the chapter. However, since early repolarization is so common (5% of the overall population) and unexplained sudden cardiac arrest in young adults is so uncommon, identifying its presence on an ECG has very little prognostic value.

Left ventricular hypertrophy and left bundle branch block

In both left ventricular hypertrophy and left bundle branch block, abnormal ventricular depolarization leads to reversal of the normal epicardial to endocardial sequence of repolarization. For this reason, in both cases where a predominantly

Table 1: ECG clues to the possible cause of ST segment changes

Cause	Characteristics	Schematic
Myocardial infarction	• Localized to a specific arterial distribution • Presence of reciprocal changes • Q waves may be present	
Pericarditis	• Generalized anterior, lateral, and inferior ST elevation • Reciprocal ST depression only in aVR • PR segment elevation in aVR • PR segment depression in the inferolateral leads • No pathologic Q waves	
Early repolarization	• Most prominent in the anterior and lateral leads • Notch at the terminal portion of the QRS complex • In leads with ST segment elevation, prominent T waves • No pathologic Q waves	
Left ventricular aneurysm	• ST segment elevation in the anterior leads • "Domed" ST segment elevation • Accompanying T wave inversion • Pathologic Q waves present	

negative QRS complex is observed (right precordial leads), ST segment elevation will also be present (Figure 3 and Chapter 5, Figure 5). In particular, very prominent ST segment elevation (> 5 mm) can be observed in patients with left bundle branch block. It is obvious then that when evaluating the ST segment, the QRS complex must be evaluated for width (left bundle branch block) and voltage (left ventricular hypertrophy).

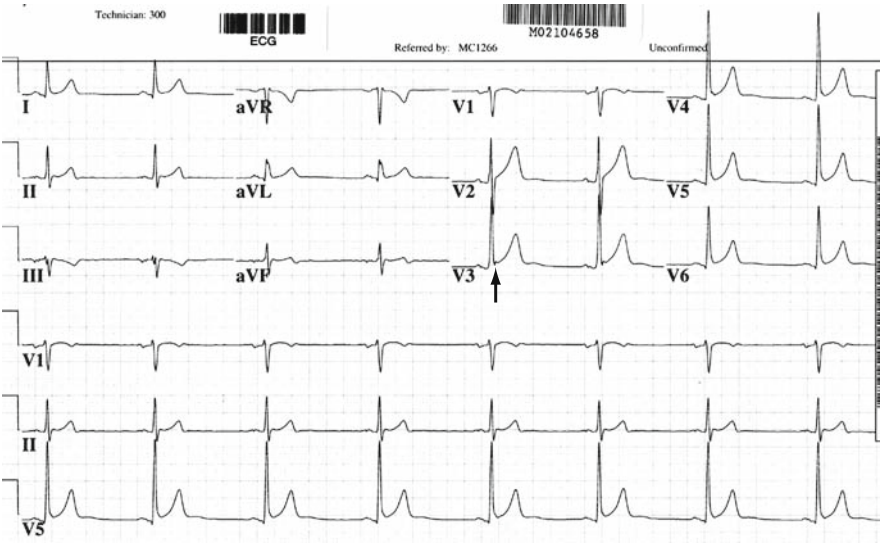


Figure 2: ECG in a young man with early repolarization. ST segment elevation can be observed in the anterior leads. Patients with early repolarization will have a small terminal "hook" at the end of the QRS complex and the beginning of the ST segment (*arrow*).

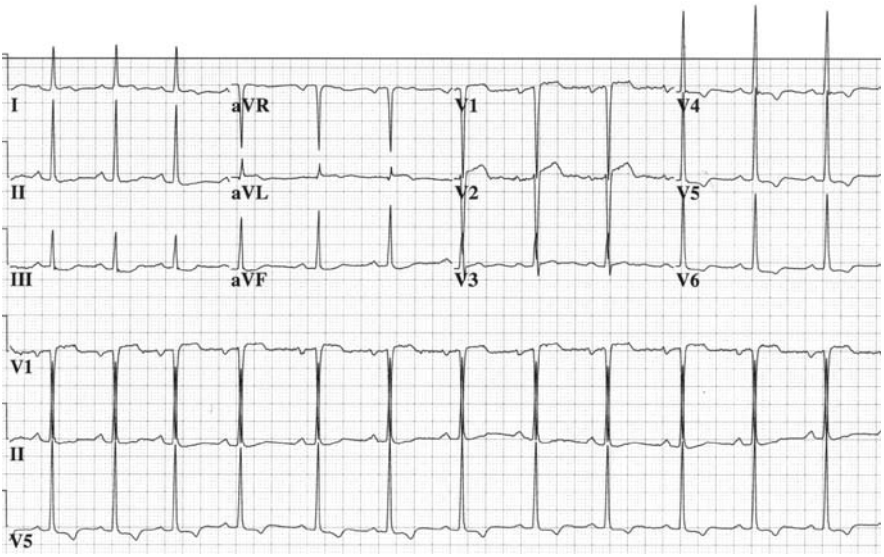


Figure 3: ECG from a patient with left ventricular hypertrophy. ST segment elevation is isolated to V_1 and V_2.

Pericarditis

The pericardium is a sac surrounding the heart. Inflammation of the pericardium or pericarditis can result from a number of causes, including infection, collagen vascular disease, trauma, and renal failure. Since the inflammatory response is observed diffusely in the ventricular epicardium, diffuse ST segment elevation is observed in the precordial leads and the frontal leads, particularly the inferior leads (Figure 4). The exception is aVR. Since the positive electrode of aVR "looks" at the injured pericardium through normal endocardium, ST segment depression is observed (Figure 5). The ST segment elevation in pericarditis seldom exceeds 5 mm and often has a concave appearance. The differentiation between pericarditis and myocardial infarction can sometimes be difficult, because in both cases patients present to the emergency room with chest pain. However, there are a few ECG clues that will help differentiate between the two conditions (Table 2). Unlike pericarditis, the ST segment elevation associated with myocardial infarction is often described as having a convex or "domed" appearance. Pericarditis is usually associated with diffuse ST segment elevation rather than localized ST elevation, although exceptions exist and "localized" pericarditis has been reported. Reciprocal ST depression will usually be observed in myocardial infarctions associated with ST segment ele-

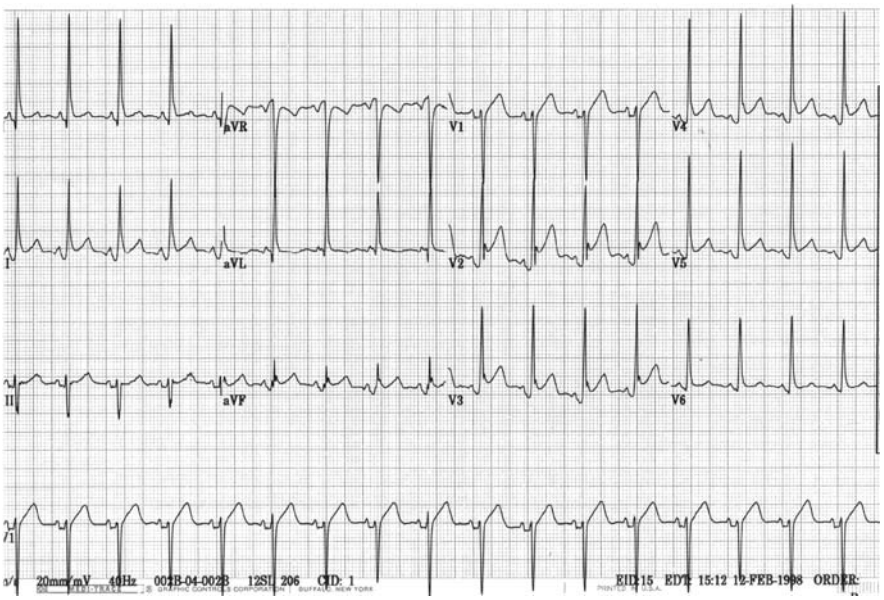


Figure 4: ECG in a patient with pericarditis. ST segment elevation is present in the inferior, lateral, and anterior leads. ST segment depression is noted in aVR. PR depression is observed in the inferior and lateral leads and an elevated PR segment is observed in aVR.

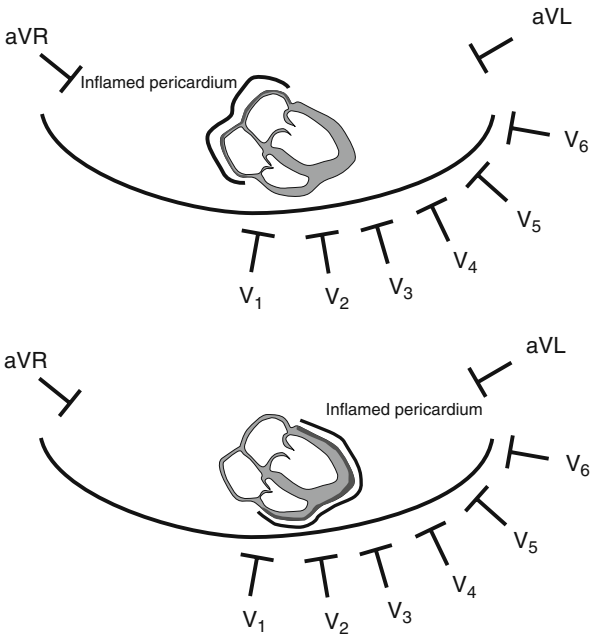


Figure 5: Schematic demonstrating the reason for ECG changes observed in pericarditis. *Top*: Inflamed pericarditis over atrial tissue leads to injury and baseline depolarization of superficial atrial myocytes (*dark grey*). Since aVR looks directly at the myocardial injury, PR segment elevation is observed. The rest of the ECG leads look at the injured region through normal tissue, so PR segment depression is seen. *Bottom*: For the ventricle aVR looks at the injured myocardium through normal tissue, so ST segment depression is noted. The rest of the leads look directly at the injured myocardium, so diffuse ST segment elevation is observed.

Table 2: ECG changes associated with pericarditis vs. myocardial infarction

Myocardial infarction	Pericarditis
ST segments with a convex "domed" appearance	ST segments with a concave "sagging" appearance
Localized ST segment elevation (coronary artery distribution)	Generalized ST segment elevation (inferior, anterior, and lateral)
Reciprocal ST depression	ST depression only in aVR
Q waves may be present	Q waves not observed
T waves becomes inverted while the ST segment is elevated	T waves become inverted after the ST segment returns to baseline
PR segment changes are usually not observed	PR segment elevation in aVR and PR segment depression in the inferolateral leads

vation, whereas in pericarditis ST depression is usually only observed in lead aVR. Q waves are not observed in pericarditis. Finally, the time course of ST segment and T wave changes differ. In myocardial infarction, T waves usually become inverted early in the course while the ST segments remain elevated, whereas in pericarditis the ST segments usually return to normal before the T waves invert. Thus the presence of elevated ST segments in association with T wave inversion is worrisome for an evolving myocardial infarction.

The pericardium surrounds the right atrium and the inferior and superior portions of the left atrium, so pericarditis can also affect atrial tissue. Pericardial inflammation overlying atrial tissue leads to PR segment depression in the inferolateral leads and PR segment elevation in aVR. The situation is the reverse of ventricular replorization: aVR "looks" directly at the inflamed atrial tissue while the inferolateral leads "look" at the inflamed atrial tissue through normal endocardial atrial tissue. In general the PR interval changes are subtler than the ST segment changes, but in the author's experience PR segment elevation in lead aVR is the most specific finding for pericarditis.

Pericarditis: PR segment elevation and ST segment depression in aVR.

Coronary artery spasm

Not surprisingly, coronary artery spasm will also lead to transient ST segment elevation. Some patients will have a sudden spasm of a large epicardial vessel that causes ischemia in the downstream region of the myocardium. ST segment elevation will be observed (using the same mechanism as with myocardial infarction), but the effect will be transient, lasting a few seconds to minutes.

Transthoracic cardioversion

As a therapeutic option some patients will undergo transthoracic cardioversion to treat certain types of rapid heart rates (in clinical use, most commonly ventricular tachycardia or atrial fibrillation). In transthoracic cardioversion a relatively large electrical current (50–300 joules) is delivered through large electrodes applied to the skin. The electrical current resets the heart and hopefully returns the patient to a normal rhythm. In approximately 15–20% of patients, transient ST segment elevation will be observed immediately after the cardioversion (Figure 20, chapter 12). The ST segment elevation generally lasts only several seconds, but can still be observed several minutes after cardioversion in some cases. The mechanism for the ST segment elevation is not known and does not appear to be associated with myocardial damage. This phenomenon appears to be more common in patients with poor left ventricular function.

Left ventricular aneurysm/Takotsubo Syndrome

After a large myocardial infarction, some patients will develop a region of dense scar and fibrosis that is called a left ventricular aneurysm. Left ventricular aneurysms are less commonly observed now that early reperfusion is emphasized for the treatment of myocardial infarction. The most common region for the development of a left ventricular aneurysm is the anterior wall or apex of the left ventricle. Consequently, a left ventricular aneurysm is characterized by ST segment elevation in the anterior leads (Figure 6). The ST segment elevation often will have a distinct "rounded" appearance and is usually associated with inverted T waves. In addition, Q waves will usually be observed in the leads that overlie the area of aneurysm.

Several investigators have described an interesting syndrome of transient ballooning of the apical region not associated with coronary artery occlusion. Transient apical ballooning is also called Takotsubo Syndrome because the heart has the typical appearance of a Japanese pot used to trap octopi, characterized by a thin neck and large bowl. Takotsubo syndrome appears to be more common in women who are exposed to a sudden adrenergic surge. Anterior ST segment elevation similar to occlusion of the left anterior descending artery is observed (Figure 7). Clinically, release of enzymes suggestive of myocardial damage and a large region of the apex that does not contract normally will be observed. Interestingly, over time the left ventricular function returns to normal or near normal and the ECG also normalizes.

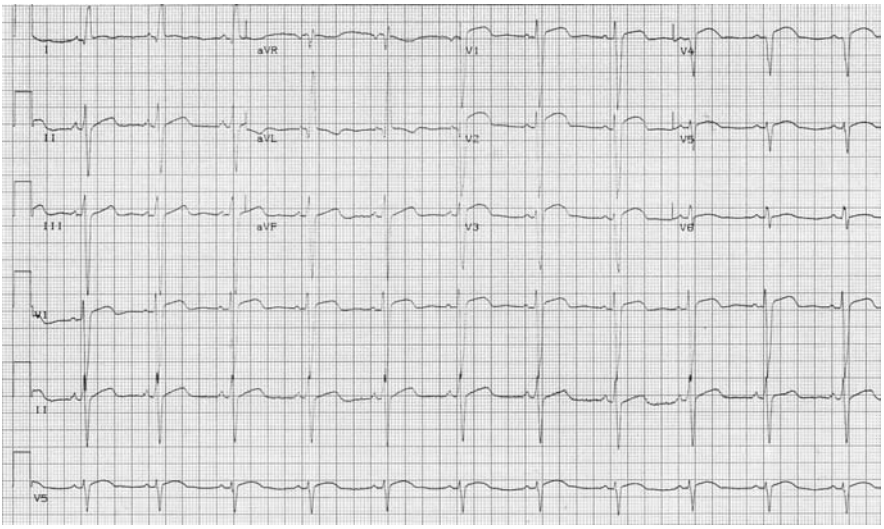


Figure 6: ECG from a patient with a left ventricular aneurysm after a large anterior wall myocardial infarction. Anterior ST segment elevation is present along with an abnormal Q wave in V_4.

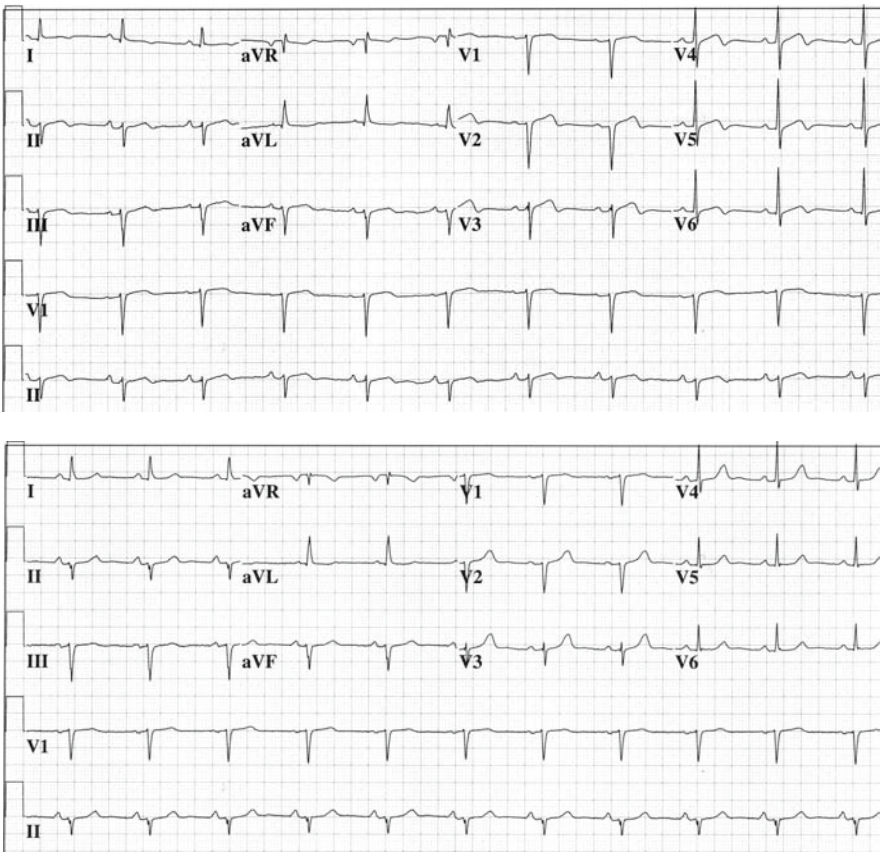


Figure 7: *Top*: ECG from a patient with Takotsubo Syndrome. Apical ballooning caused ST segment elevation and T wave inversion in the precordial leads and an abnormal Q wave in V_2. *Bottom*: The ECG changes resolved completely one month later with normalization of precordial ST segment elevation and T wave inversion. The patient has baseline left anterior fascicular block.

Brugada syndrome

In 1992 Brugada and Brugada described eight patients with sudden cardiac death who also had an ECG with characteristic findings: right bundle branch block morphology and ST segment elevation in the right precordial leads (Figure 8). The Brugada Syndrome is fairly rare worldwide but is more common in Asian men, with a prevalence of 0.12–0.14%. Although the genetic cause for most cases of Brugada Syndrome is unknown, a substantial proportion appears to be due to a Na^+ channel mutation. Attenuation of Na^+ inflow can potentially be associated with both depolarization and repolarization abnormalities. Decreased Na^+ current can cause slow conduction from the endocardium to the epicardium. Reduction of Na^+

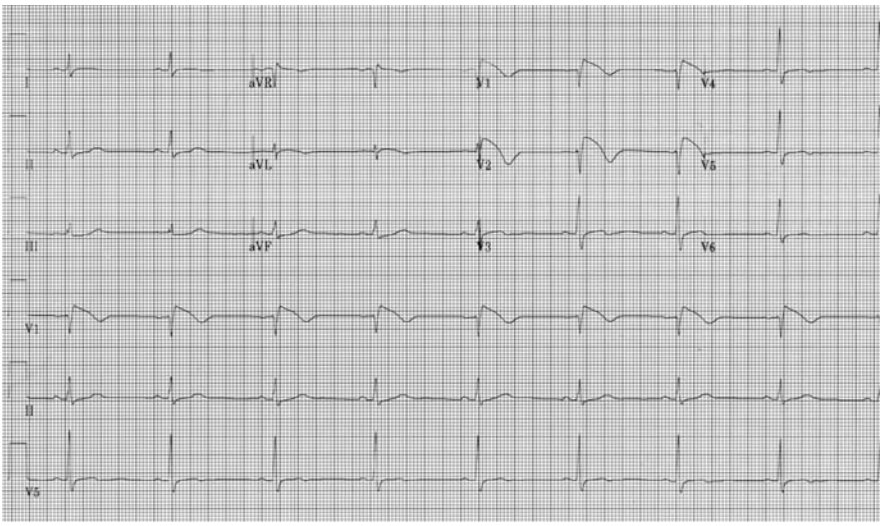


Figure 8: ECG from a patient with Brugada Syndrome. The QRS is wide with a right bundle branch block pattern in the right precordial leads (V_1,V_2) and associated ST segment elevation.

current can also cause repolarization abnormalities because of an unopposed I_{to} current that leads to the widened QRS and right bundle branch block pattern in the right precordial leads (Figure 9). In experimental preparations this leads to an ECG pattern similar to the Brugada Syndrome with a very prominent J wave (the R' of the right bundle branch block) and associated ST segment elevation. Interestingly, it appears that I_{to} is stronger in men and in the right ventricle, providing a possible explanation for the male predilection of the disease and the more prominent ECG findings in the right precordial leads. In some cases the I_{to} notch can become so prominent that Ca^{2+} channels are not activated, leading to extreme abbreviation of the action potential and extreme heterogeneity in repolarization. From a clinical perspective this heterogeneity in repolarization can lead to ventricular arrhythmias due to reentry and to an increased risk for sudden death.

Changes in the relative strength of the I_{to} current may be the common electrophysiologic thread for deflections observed at the end of the QRS in patients with early repolarization, the Brugada Syndrome, and Osborn waves that can be seen in hypothermia and hypercalcemia. A larger phase 1 downstroke could be mediated by an increased I_{to} or other repolarizing current or a decrease in the inward Na^{+} or Ca^{2+} current.

Pulmonary embolism

Rarely, pulmonary embolism can be associated with ST segment elevation, usually in the inferior leads. However, the most common ECG findings in pulmonary embolism are fairly nonspecific and include sinus tachycardia, right axis deviation, and subtle ST and T wave changes.

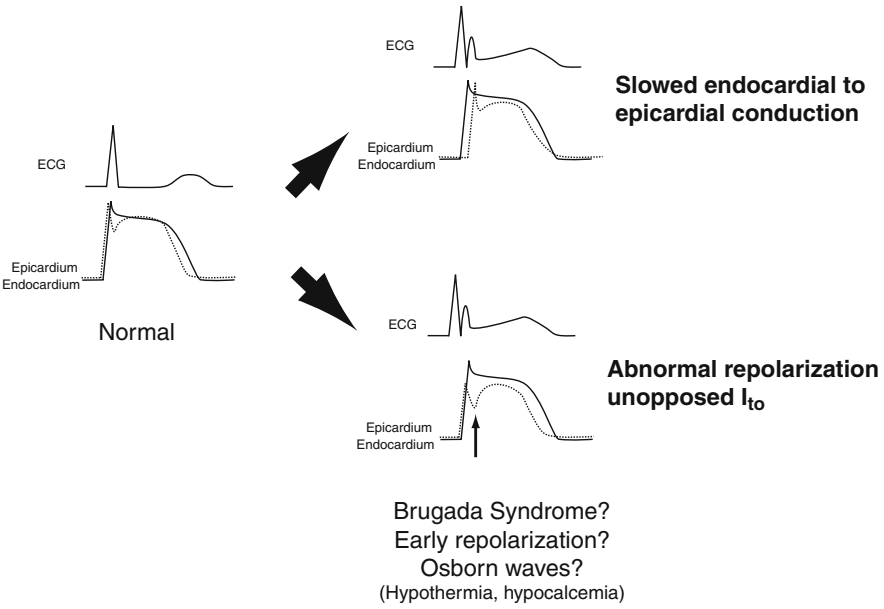


Figure 9: Possible electrophysiologic mechanisms for the development of the ECG findings observed in Brugada Syndrome and any condition-associated deflections in the terminal portion of the QRS complex, such as early repolarization or Osborn waves in hypercalcemia and hypothermia. The additional deflection may be due to slowing of depolarization from one region to another (endocardium to epicardium). An attenuated Na^+ current can also cause repolarization changes by leaving an unopposed I_{to} current that produces a terminal R wave in the right precordial leads. It may be that this is the same mechanism for the notch after the QRS in early repolarization, or the Osborn waves observed in hypothermia and hypercalcemia.

Hyperkalemia

Hyperkalemia also can cause ST segment elevation. Remember from the last chapter that one of the reasons for ST segment elevation in myocardial infarction is T-P segment depression due to localized extracellular accumulation of K^+. It is not surprising that severe hyperkalemia in the blood would also be associated with ST segment elevation. In addition to ST segment elevation, hyperkalemia is associated with other ECG findings, such as T wave peaking and loss of P waves, that are more fully discussed in Chapter 16.

Case (continued): Mr. Moore has acute ST segment elevation due to hyperkalemia associated with acute renal failure as a result of his diabetes and hypertension. After treatment of his hyperkalemia his ECG several minutes later is shown in Figure 10. The ST segment elevation is diffuse. The relatively prominent T waves and the slightly wide QRS suggest the possibility of hyperkalemia, but realistically it would be difficult to differentiate this ECG from acute myocardial infarction. Again, the

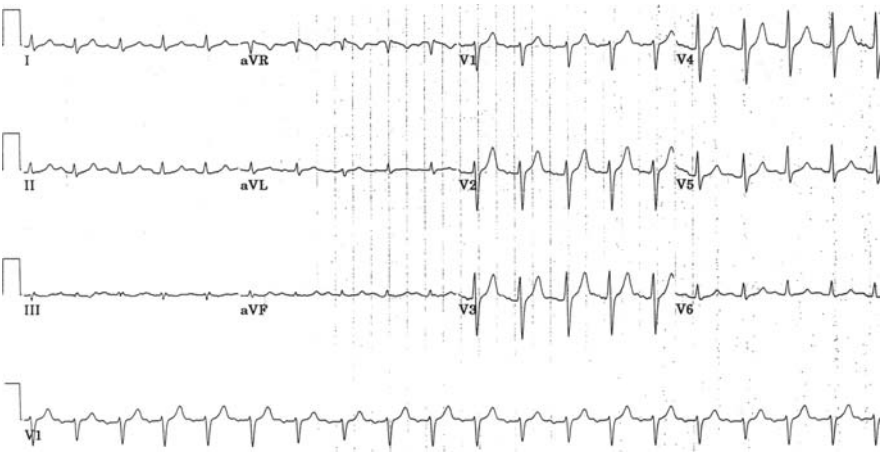


Figure 10: After the patient was treated for hyperkalemia, the ST segments returned to baseline. Patient still has residual anterior prominent T waves.

absence of accompanying chest pain is the most important clinical tool for suggesting an etiology other than acute myocardial infarction for the ST segment elevation.

Summary

The ST segment is usually very close to isoelectric because the ventricular cells all have similar voltages. However on closer inspection some ST changes are commonly present, particularly in the right precordial leads. These small ST segment changes are likely due to small differences in voltage that are present during this period. ST segment changes are most frequently associated with myocardial infarction and myocardial ischemia; but there are several other causes for ST segment elevation, including pericarditis, left bundle branch block, hyperkalemia, and Brugada Syndrome. ECG characteristics for conditions that cause ST segment elevation are summarized in Table 1.

Key points

1. The ST segment generally represents the plateau phase of the ventricular action potential.
2. ST segment elevation can normally be seen in young men in the right precordial leads. More prominent and widespread ST segment elevation can also be observed in healthy men and is often termed “early repolarization.”
3. Pericarditis causes diffuse ST segment elevation in all of the leads except aVR, where ST segment depression is observed. The PR segment will be depressed in most leads but elevated in lead aVR.

4. Although the ECG is an important tool for identifying patients with myocardial infarction, there are a number of clinical conditions in which ST segment elevation can be observed.

Review questions

1. What is the likely diagnosis in Figure 11?

   A. Early repolarization
   B. Pericarditis
   C. Left ventricular aneurysm
   D. Brugada Syndrome

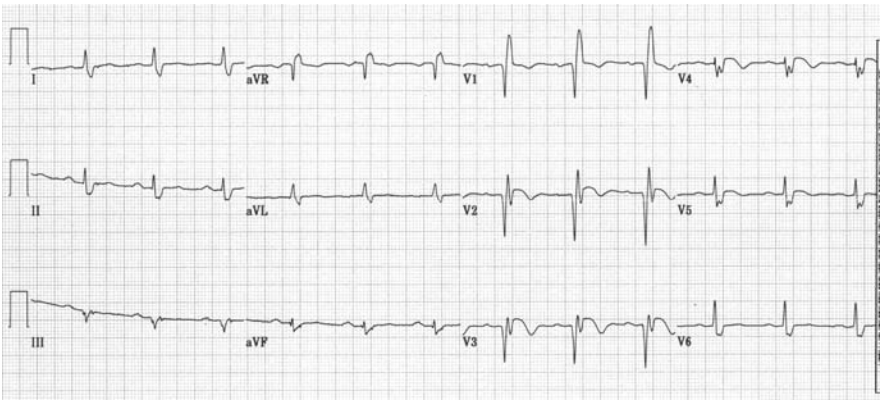


Figure 11: ECG for Problem #1.

2. Which of the following is not associated with ST segment elevation?

   A. Hypokalemia
   B. Pericarditis
   C. Left bundle branch block
   D. Brugada Syndrome

3. ECG findings associated with pericarditis include all of the following except:

   A. ST segment elevation in the inferolateral leads
   B. ST segment elevation in aVR
   C. PR segment elevation in aVR
   D. Diffuse ST segment elevation is observed.

4. Which statement is true?

   A. ST segment elevation is rarely observed in young men
   B. ST segment elevation is a common finding in pulmonary embolus
   C. ST segment elevation is only found in myocardial infarction
   D. As a normal finding, ST segment elevation is less common in women than men.

5. What is the most likely diagnosis in Figure 12?

   A. Early repolarization
   B. Pericarditis
   C. Left ventricular aneurysm
   D. Brugada Syndrome

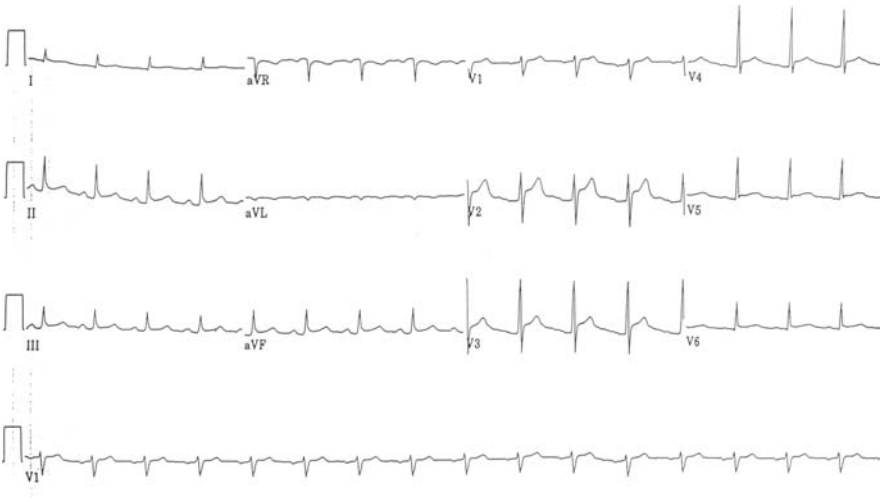


Figure 12: ECG for Problem #5.

Answers

1. C. The ECG is from a patient with a left ventricular aneurysm. The ECG shows "humped" ST segment elevation in the anterior leads with associated Q waves. Pericarditis, Brugada Syndrome, and early repolarization would not be associated with Q waves. The patient also has right bundle branch block and first degree AV block.
2. A. Hypokalemia is not associated with ST segment elevation.
3. B. ST segment depression is normally observed in aVR with pericarditis.
4. D. ST segment elevation is more common in young men than young women, as a normal finding.

5. B. The patient has diffuse ST segment elevation with PR depression in the inferior leads. More importantly, aVR has PR segment elevation and ST segment depression. The patient has no Q waves. Early repolarization would be characterized by more prominent T waves in the precordial leads and would not have ST depression and PR segment elevation in aVR.

Part IV

Arrhythmias

Chapter 9
Premature beats

The sinus node normally initiates activation of the heart because it has the fastest intrinsic rate of automaticity. Frequently, for a variety of reasons, premature beats can arise from atrial tissue, junctional tissue, or ventricular tissue. The most common cellular mechanism for these premature beats is increased automaticity.

Case Study: Introduction

Ms. Jones is an eighty-five-year-old woman who comes to your office for a routine physical examination. She has no significant medical history, and her physical examination is unremarkable. Her ECG is shown in Figure 1. Why is the heart rate irregular?

Premature atrial contractions

Sometimes atrial tissue other than the sinus node will spontaneously reach threshold earlier than the sinus node and produce a premature atrial contraction. Premature atrial contractions are a common and benign finding, occurring in approximately 0.5% of people undergoing a standard 12-lead ECG. The prevalence is actually more common: over a 24-h period, 80% of people will have at least one premature atrial contraction.

By definition, the P wave from the ectopic atrial focus will be early or premature. The shape of the P wave will be dependent on the site of the initiating focus. For example, in Figure 2, the premature beat has a similar morphology to the normal sinus rhythm because the ectopic site is located very close to the sinus node. In Figure 3, the ectopic focus is located near the inferior vena cava, which yields a P wave that is negative in the inferior leads, since atrial activation is occurring from "low" to "high." In Figure 4, a premature atrial contraction arising from tissue in the left upper pulmonary vein is shown. In this case the P wave is negative in aVL.

The response of the AV node and ventricular activation will be determined by the prematurity of the atrial complex. If the premature atrial activation occurs early enough, conduction through the AV node is blocked and an isolated P wave will be seen. This is called a blocked premature atrial complex. This is a normal

F. Kusumoto, *ECG Interpretation: From Pathophysiology to Clinical Application*,
DOI 10.1007/978-0-387-88880-4_9, © Springer Science+Business Media, LLC 2009

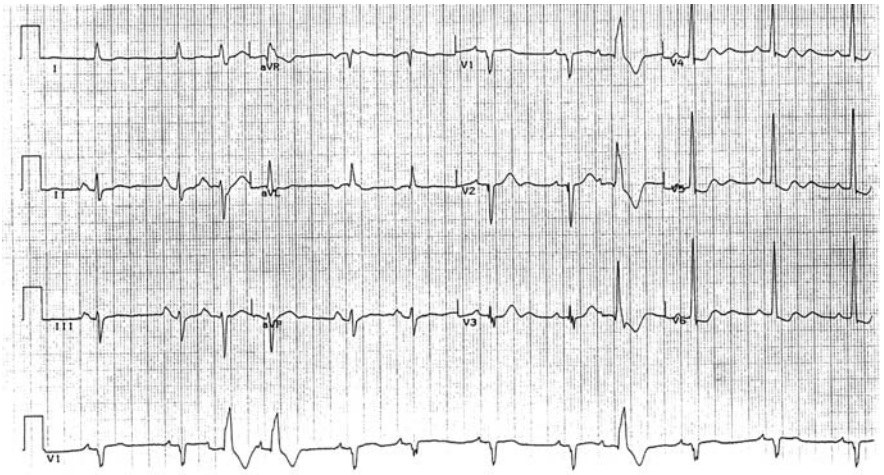


Figure 1: ECG for Ms. Jones.

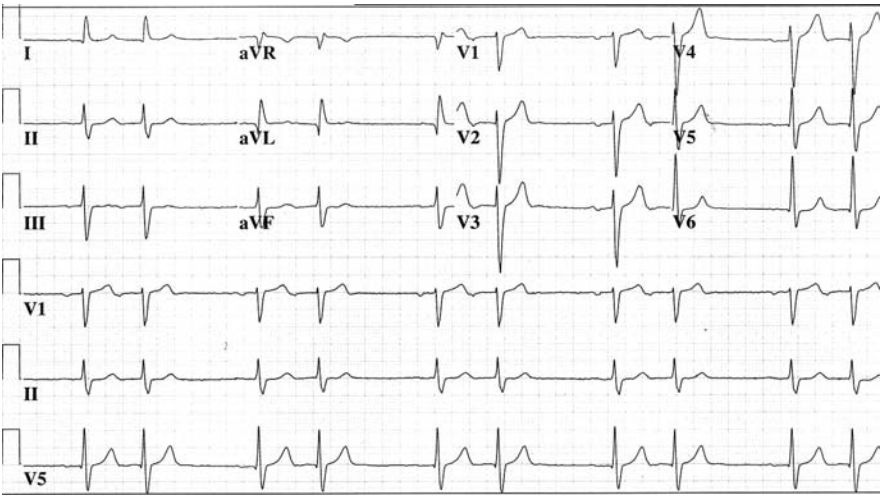


Figure 2: Irregular heart rhythm due to premature atrial complexes. After every sinus beat a premature atrial beat is associated with an early QRS complex. It is very difficult to specifically evaluate the P wave morphology of the premature beats. The P waves are slightly different from the sinus beats, so the rhythm would be classified as sinus rhythm with frequent premature atrial contractions in a bigeminy pattern (a premature beat after every sinus beat). If all of the P waves were the same, the ECG would be classified as sinus arrhythmia since only one site (presumably the sinus node) is generating the P wave.

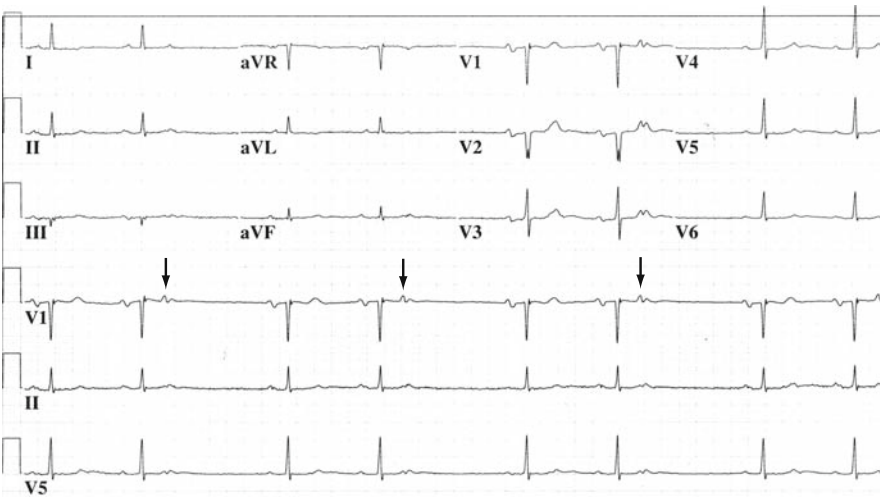


Figure 3: Nonconducted premature atrial complexes. After two sinus beats there is a pause. On examination of the ST segment and T wave an "unexpected" deflection can be observed (arrows). This represents premature atrial activity. The AV node is refractory, so a QRS complex does not follow the premature atrial activity. This is a normal response for the AV node. Remember that the AV node is composed of slow response cells. In this way the AV node can act as a regulator to prevent rapid abnormal atrial activity from causing rapid ventricular activity. This ECG illustrates the importance of comparing the ST segment and T wave in consecutive beats in irregular rhythms to identify atrial activity.

response and should not be confused with atrioventricular block that will be discussed in Chapter 10. It is important to remember our review of refractoriness from Chapter 6. When cardiac tissue is refractory, a stimulus will not result in an action potential. There is a gradation in the cardiac issue refractoriness. During the absolute refractory period, a stimulus, no matter how strong, will not result in another action potential. During the relative refractory period a very strong stimulus will result in a partial or complete tissue response. In general, the AV node has the longest refractory period of all cardiac tissue. This is an important property that will become more apparent in Chapter 11. Because of its long refractory period, the AV node will limit the ventricular rate in the presence of an abnormally fast atrial rate.

Premature atrial contractions:

1. *Preceded by a P wave.*
2. *P wave morphology will be determined by the site of origin.*
   Whether a not a QRS complex is observed after a premature atrial complex will be determined by whether or not the AV node is refractory.

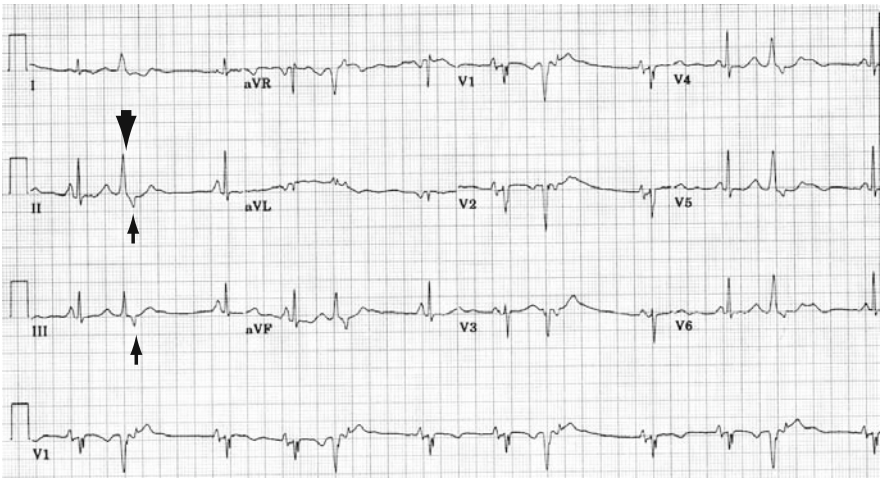


Figure 4: Premature ventricular complexes. In this ECG premature ventricular contractions (PVCs) can be observed (*large arrow*). Since PVCs are due to an early depolarization from a focus within the ventricles, the QRS complex is wide. In some cases the premature ventricular contraction is associated with retrograde activity via the AV node, and a P wave that is inverted (*small arrows*) in the inferior leads can sometimes be observed.

Premature ventricular contractions

Occasionally, ventricular activation will reach threshold early and activate the ventricles. Premature ventricular contractions will appear as wide QRS complexes since the ventricles are not activated over the normal His-Purkinje system (Figure 4). The morphology of the QRS complex will be determined by the site of origin. In Figure 4 the premature ventricular complex has a left bundle branch block morphology that suggests that the ectopic beat was generated by a site in the right ventricle that led to depolarization of the right ventricle before the left ventricle (Figure 5). In addition, the PVC has an inferior axis (predominantly positive QRS complexes in the inferior leads) that suggests that the ectopic site is near the right ventricular outflow tract. This information can be important. In most cases, premature ventricular contractions are not associated with significant clinical consequences, but in some cases an ectopic site can fire repetitively, leading to a series of rapid ventricular beats (ventricular tachycardia).

In some cases, the premature ventricular contraction will activate the His bundle tissue and AV node (retrograde conduction), and lead to atrial activation (Figure 6). Since the AV node is in the inferior part of the atria, the atria will be activated "low-high" and lead to inverted P waves in the inferior leads. Since the AV node has slow conduction properties, when present the P wave will usually be seen in the ST segment.

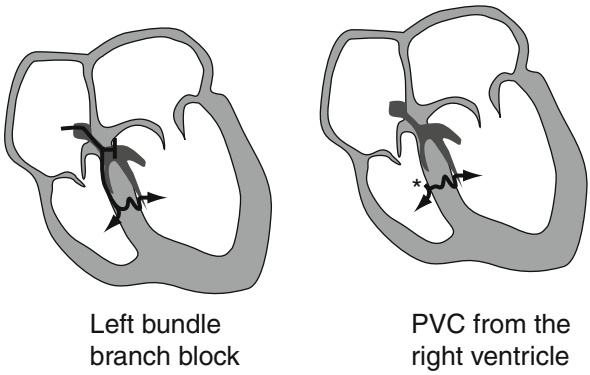


Figure 5: Schematic showing that a premature ventricular contraction arising from the right ventricle will result in a QRS complex with a left bundle branch block morphology, since the right ventricle will be activated before the left ventricle. This will lead to a predominantly negative QRS complex in V_1 and a predominantly positive QRS complex in V_6, aVL, and I.

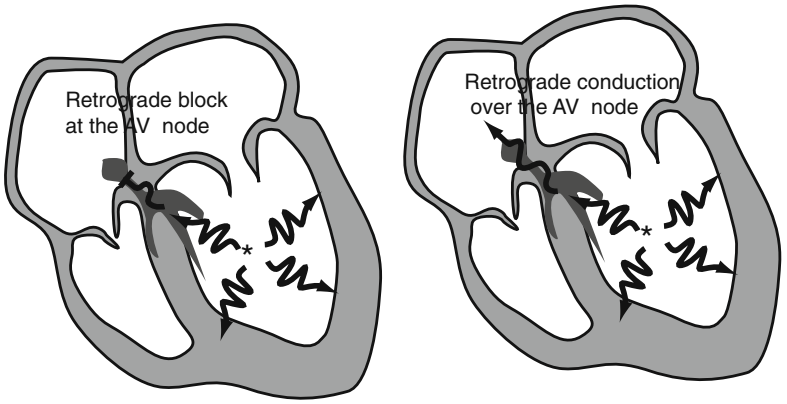


Figure 6: A premature ventricular contraction can be associated with retrograde block in the AV node (no P wave will be observed) or with retrograde conduction (inverted P wave will be observed in the ST segment after the PVC). Retrograde activity will be observed only if the AV node and His bundle are not refractory.

Premature ventricular contractions:
1. *QRS duration* > 0.12 *seconds.*
2. *Not initiated by a P wave.*
3. *Morphology will depend on the site of initiation.*

The significance of premature ventricular contractions depends on whether or not there is accompanying heart disease. It is estimated that premature ventricular

contractions occur in 20–30% of people. In patients with normal hearts, premature ventricular contractions are not associated with a poorer than normal prognosis. However, premature ventricular contractions are observed in some patients after a myocardial infarction or with other heart disease. In patients with heart disease, premature ventricular contractions are generally associated with increased risk for sudden cardiac death and overall mortality.

Case Study (continued):

Ms. Jones has predominant sinus rhythm. The fourth beat occurs early and is wide. Notice that is preceded by a P wave, which has a similar morphology to normal atrial activation via the sinus node. The QRS complex has a right bundle branch block pattern. This is a case of a premature atrial contraction arising from a site near the sinus node that reaches the AV node early (Figure 7). Since the AV node has decremental conduction properties, the PR interval is slightly prolonged. The wave of activation reaches the right bundle branch early, when the right bundle is still refractory. Conduction block occurs in the right bundle, the ventricles are activated via the left bundle, and the QRS complex has a right bundle branch block pattern (see Figure 1).

Premature junctional complex

At times automaticity of the atrioventricular node region (AV junction) can be greater than the sinus rate, so the junction fires early. This is manifested by a normal-appearing QRS complex (since the ventricles are activated normally by the His-Purkinje system. The atria are activated retrogradely. Since the P wave is much smaller than the QRS complex, it is frequently obscured by the QRS. In some cases the P wave can be seen changing the initial deflection of the QRS complex (Figure 8).

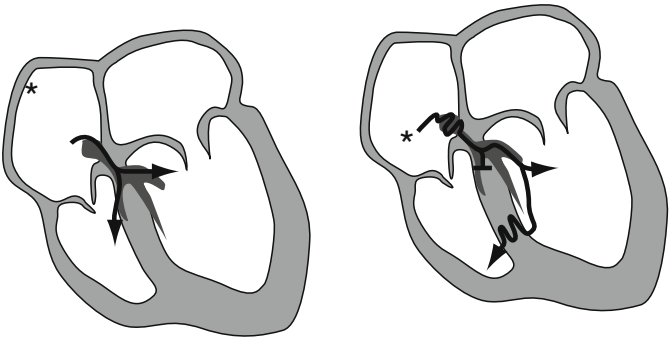


Figure 7: Schematic for the cause of the wide QRS complexes in the case.

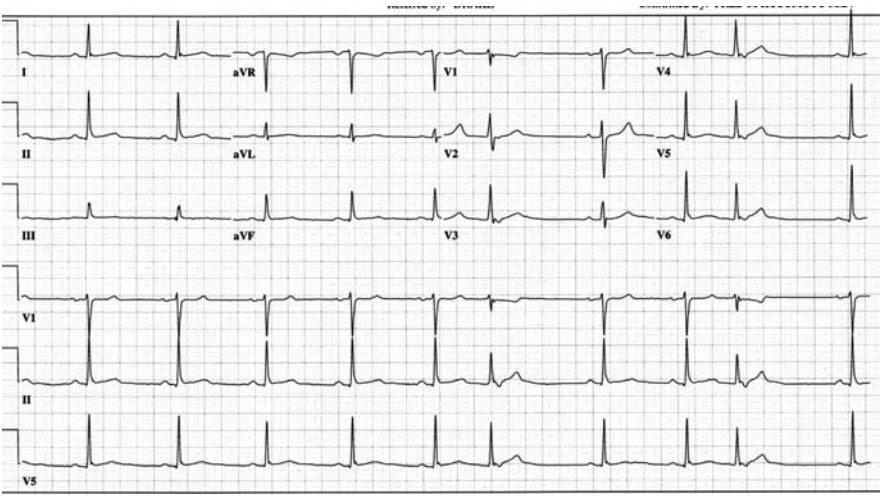


Figure 8: Premature junctional complexes. The early beats are characterized by a narrow QRS complex that is very similar in morphology to the normal sinus beats. In addition, retrograde activation of the atria can be observed just after the QRS complexes of the early beats.

In premature junctional complexes:

1. *QRS duration is normal, and the morphology is frequently normal.*
2. *P waves are usually not seen.*

Key points

1. Premature beats can arise from any type of cardiac tissue: atrial tissue, junctional tissue, and ventricular tissue.
2. In premature atrial complexes an early P wave due to atrial activation is seen. Whether or not a subsequent QRS is observed, and the morphology of the QRS complex, will depend on the refractoriness of the AV node and His-Purkinje tissue.
3. In premature ventricular contractions, an early QRS complex that is not preceded by a P wave is observed. The QRS complex will be wide, since the normal His-Purkinje tissue is not being used.
4. In premature junctional complexes, an early normal-appearing QRS will be seen. The P wave is usually obscured by the QRS complex.

Questions

1. All of the following statements about premature atrial complexes are true except:
   A. The early beat is preceded by a P wave.
   B. The QRS complex can be wide.
   C. Retrograde ventricular-atrial conduction may be present.
   D. The QRS complex is early.

2. A premature beat with a wide negative QRS complex in V_1 could represent all of the following except:
   A. A premature atrial complex with left bundle branch block
   B. A premature junctional complex in a patient with baseline right bundle branch block
   C. A premature ventricular contraction arising from the right ventricle
   D. A premature ventricular complex arising from the left ventricle

3. An electrocardiogram is shown in Figure 9. What is the abnormality?
   A. Premature atrial complexes
   B. Premature junctional complexes
   C. Premature ventricular complexes
   D. Premature atrial complexes and premature ventricular complexes

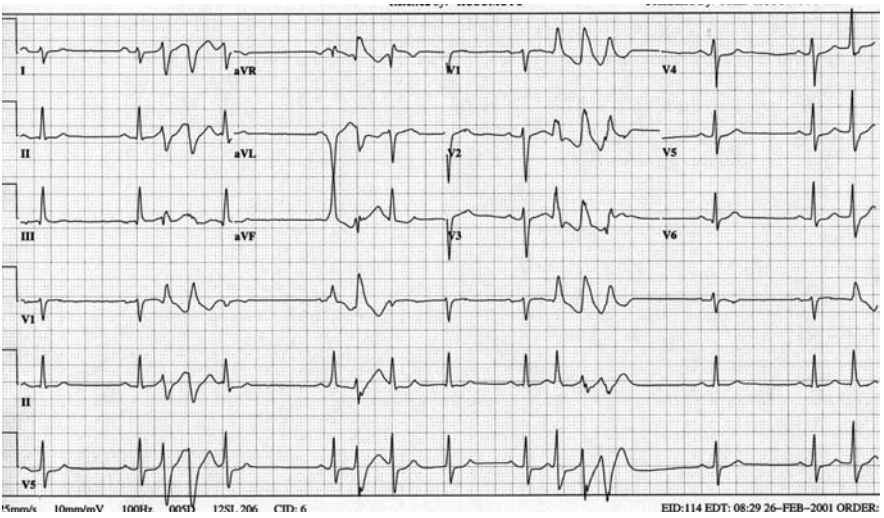


Figure 9: ECG for problem #3.

Answers

1. C. With a premature atrial complex, both the P wave and QRS complex will be early. An isolated premature atrial complex can block in the AV node or conduct from the atria to the ventricles. It will not be associated with retrograde ventriculoatrial conduction.
2. B. A wide negative QRS complex in V_1 is a left bundle branch block pattern and suggests that the right ventricle is activated before the left ventricle. This pattern will be seen if the patient has a premature atrial complex or premature junctional complex associated with left bundle branch block. Premature ventricular complexes from the right ventricle will have a left bundle branch block pattern. Premature ventricular complexes from the left ventricle in a patient with a normal heart will have a right bundle branch block morphology (positive QRS complex in V_1). However, in patients with heart disease, premature ventricular complexes from the left ventricular septum can have a left bundle branch block morphology because of late activation of the lateral left ventricular wall in a diseased heart.
3. C. The patient has multiple ventricular contractions that occur one after the other. When any rhythm presents as salvos of > 3 beats but less than 30 s, the rhythm is generally called nonsustained. The patient is having nonsustained ventricular tachycardia. Notice that the first early beat in each case is not preceded by atrial activity.

Chapter 10
Bradycardia

Case Study: Introduction

Ms. Johnson is a 76-year-old woman with no prior medical history who comes to the office complaining of gradually worsening fatigue. She has had no episodes of passing out (syncope) but does feel intermittent dizziness. She has had hypertension for the past five years and is currently on atenolol (beta-blocker) 50 mg daily.

Her physical examination is notable for intermittent prominent pulsations in her neck. Her ECG is shown in Figure 1.

Anatomy/pathophysiology

The sinus node has the fastest automatic or pacemaker rate and is the usual "driver" of cardiac activation. The impulse generated by the sinus node leads to atrial activation (P wave), and conduction through the AV node and His bundle leads to ventricular activation (QRS complex). Bradycardia (slow heart rate) is usually defined as a rate < 60 beats per minute. There are only two mechanisms for slow heart rates: abnormal automaticity or atrioventricular block.

Bradycardia: Heart rate < 60 beats per minute.

Abnormal automaticity

The sinus node is located on the superior portion of the lateral wall of the right atrium (Figure 2). The cells within the sinus node exhibit spontaneous depolarization (pacemaker activity) due to the gradual inflow of sodium and calcium ions and decreased outward flow of potassium ions. This leads to gradual accumulation of positive charge within the cell and membrane depolarization. Once the cell reaches threshold, activation of the cell via calcium channels occurs and the cell depolarizes (Chapter 2, Figure 4). Each of the individual cells within the sinus node have slightly different rates of diastolic depolarization, but when they are electrically coupled they "mutually entrain" each other and a single depolarization leaves the sinus node region to activate the atrium.

F. Kusumoto, *ECG Interpretation: From Pathophysiology to Clinical Application*,
DOI 10.1007/978-0-387-88880-4_10,

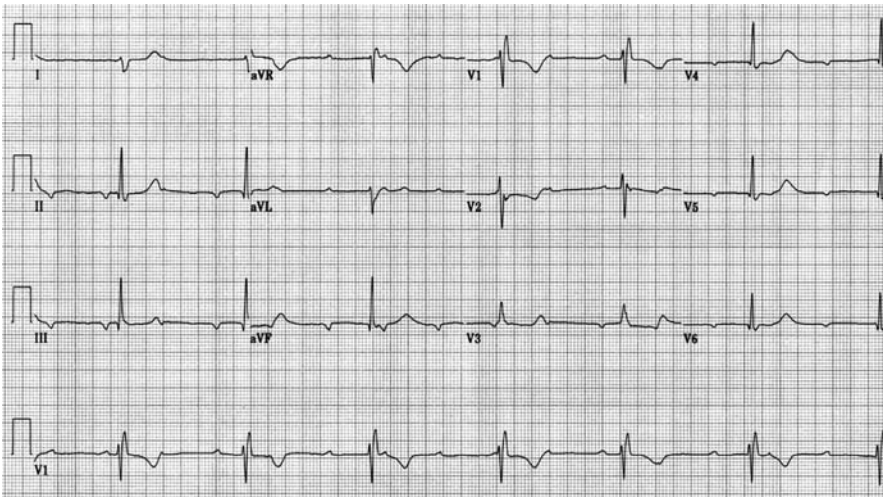


Figure 1: ECG for the case.

The rate of diastolic depolarization is affected by neural input from the sympathetic and parasympathetic systems. Adrenergic activation increases diastolic depolarization by activating sodium and calcium channels which leads to more rapid diastolic depolarization and a faster pacemaker rate. Conversely, parasympathetic activation reduces the rate of diastolic depolarization by favoring potassium permeability. This accounts for the normal variability of the sinus rate: higher rates during exercise due to sympathetic activation, and slower heart rates while sleeping due to higher parasympathetic tone.

Abnormal automaticity occurs when the sinus node region fails to depolarize or depolarizes at a slower than expected rate. This can be due to failure of any of the mechanisms that lead to diastolic depolarization, or from a block of the impulse from the sinus node region to the atrial tissue. As we age, collagen can be deposited within the sinus node region and prevent activation of neighboring atrial tissue. This is one of the reasons that heart rates decrease with age and why some older patients require pacemakers.

Atrioventricular block

Once the atria are activated, the electrical impulse travels through the AV node, His bundle, and bundle branches to activate the ventricles. Since the AV node and His bundle form the single axis for electrical conduction to the ventricles, any pathophysiologic process that blocks conduction in these critical areas can lead to slow ventricular rates.

Atrioventricular block becomes increasingly common with age. Persistent atrioventricular delays (not block) are observed in 1–2% of adults 20–30 years of age, but increases to over 5% in people over 50 years of age. In a pathologic study of

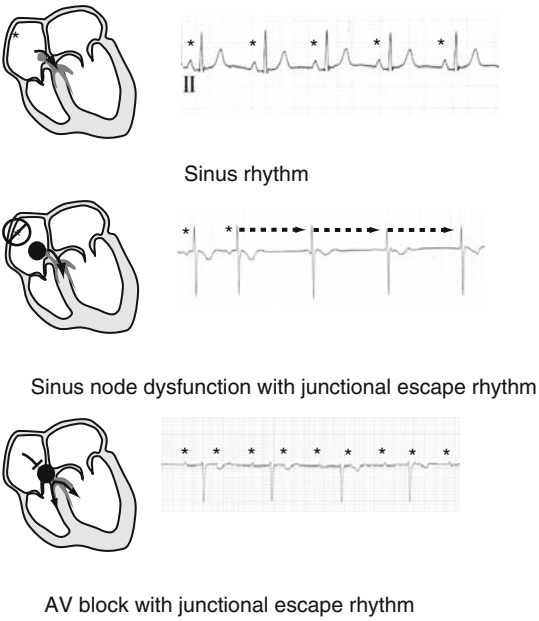


Figure 2: Schematics and ECG manifestations depending on the cause of bradycardia. The *top panel* shows normal sinus rhythm. The sinus node "drives" the heart, and there is 1:1 conduction to the ventricles via the AV node/His bundle. The *middle panel* shows sinus node dysfunction. After the first two beats, the sinus node (*) does not "fire" and a lower subsidiary pacemaker from the AV node "takes over" (•). Notice that there is a small P wave from another atrial source before the first junctional beat. It is unlikely that this atrial activity led to ventricular activation, since the escape rates (dashed arrows) are the same for all three junctional beats shown. The *lower panel* shows complete AV block. The sinus node "fires" (*), but the ventricles are depolarized by a subsidiary pacemaker from the AV node region.

people that died of noncardiac causes, fatty infiltration began to develop in the AV node at 30 years of age, and fatty changes and fibrosis developed in the His bundle after 40 years of age.

Causes for slow heart rate: Sinus node dysfunction and atrioventricular block (Figure 2).

Sinus node dysfunction

Abnormal sinus node function can be observed on the ECG. If the sinus node does not fire, an ectopic atrial site can take over, as shown in Figure 3. In ectopic atrial rhythm a P wave will precede the QRS complex, but the morphology

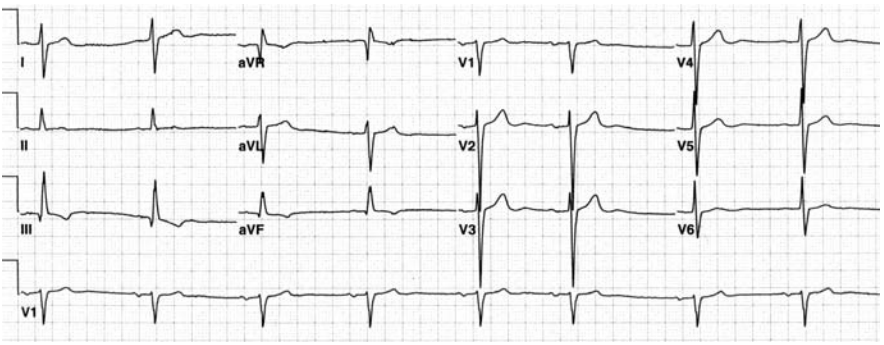


Figure 3: Ectopic atrial rhythm with "flat" P waves in the inferior leads that suggest that a lower atrial pacemaker is "driving" the heart.

of the P wave will not demonstrate typical activation from "high-to-low" and "right-to-left." In this case the P wave is "flatter," suggesting initial atrial activation from a lower portion of the atria. If an ectopic atrial rhythm does not take over, the AV node has diastolic depolarization so that it can generate the impulse at a slower rate. In junctional rhythm due to sinus node dysfunction the QRS complexes are not preceded by a P wave. In some cases a retrograde P wave can be observed (Figure 4, bottom). In other cases the junction does not provide "back-up" and a site within the ventricles becomes the primary pacemaker (Figure 5).

Another ECG manifestation of sinus node dysfunction is a sinus pause. In this case, the sinus node transiently does not depolarize and no subsidiary pacemakers immediately "take over." It is important to remember that sometimes poor electrode contact can lead to apparent sinus node pauses and bradycardia (Figure 6). Usually in this circumstance significant change in the baseline is observed.

ECG manifestations of sinus node dysfunction:

Sinus bradycardia
Ectopic atrial rhythym
Sinus pauses
Junctional rhythm

Atrioventricular block

On an ECG, atrioventricular block is classified by the relationship between atrial and ventricular conduction (Figure 7). The interval from atrial activation to ventricular activation can be measured from the beginning of the P wave to the beginning of the QRS complex, and is called the PR interval. The normal PR interval in adults ranges from 0.12 s to 0.20 s (3–5 little boxes). In first degree AV block a one-to-one relationship between atrial and ventricular activation is maintained, but

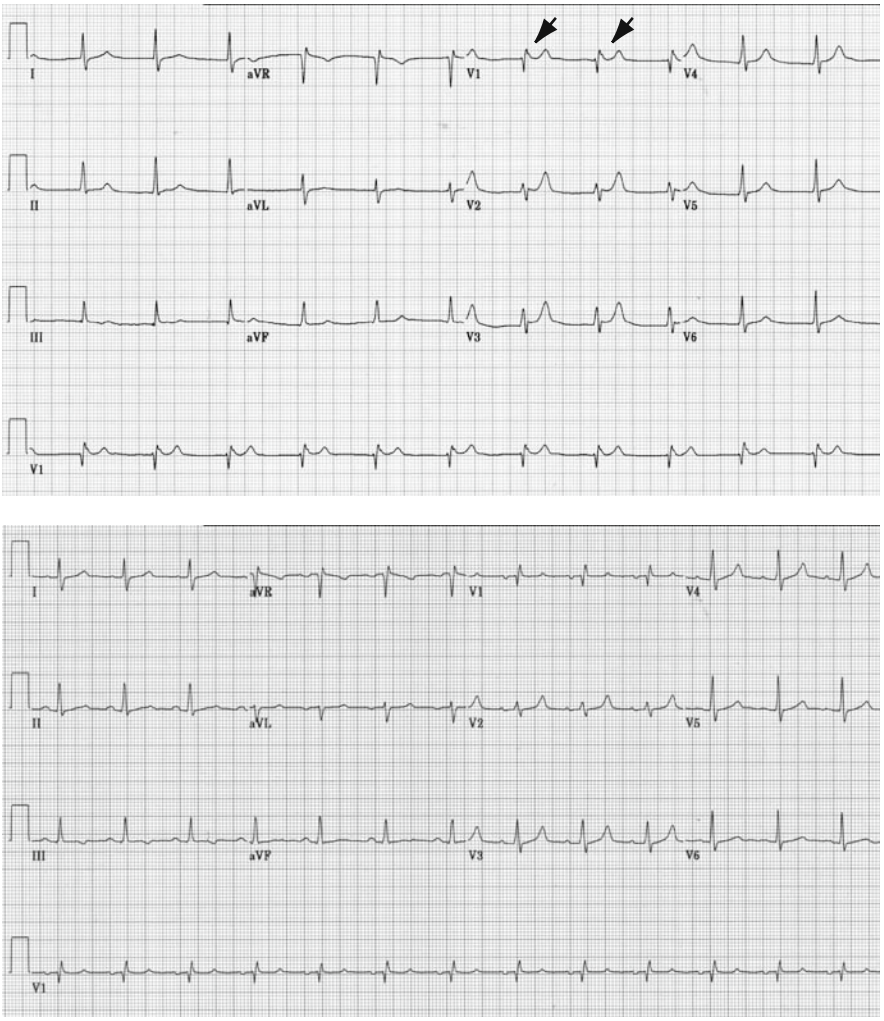


Figure 4: *Top*: Junctional rhythm. The sinus rate has slowed, so the AV node "drives" the heart. This leads to QRS complexes that are not preceded by P waves. *Bottom*: Later, in the same patient, the sinus rate increases, and now P waves can be seen before every QRS complex. Comparison of the two 12-lead ECGs can provide clues for the presence of P waves within the QRS complex. The P waves usually can be observed best in the inferior leads or V_1 (*arrows*).

conduction is delayed and the PR interval is greater than 0.20 s (Figure 8). A simple "eyeball estimate" for normal AV conduction is that the PR interval should be less than one "large box."

In second degree AV block, some but not all normal atrial activity leads to ventricular activity, and more P waves than QRS complexes will be observed. Second

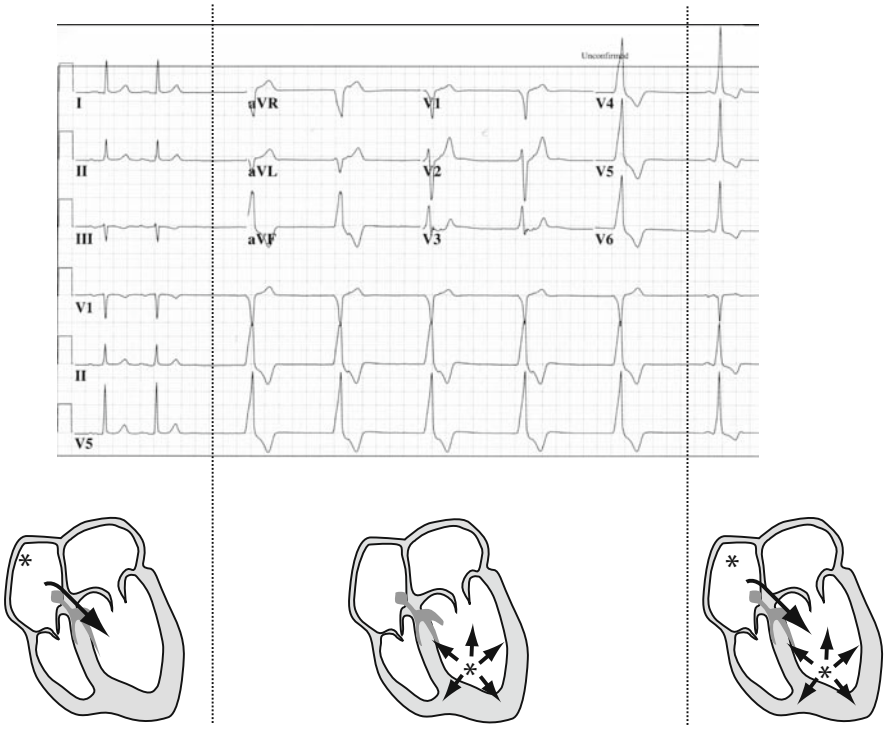


Figure 5: The first two beats show normal sinus rhythm. Unexpectedly, the sinus node suddenly stops "firing." Notice that in this case, subsidiary pacemakers in atrial tissue or the junction do not "take over." In this case, a site within the ventricular tissue becomes the primary pacemaker for the heart. When a ventricular site serves as the heart's pacemaker, slow, wide QRS complexes are observed. The astute observer will notice subtle deflections in the ST segment of the QRS complexes that represent atrial depolarization from the sinus node. These P waves do not lead to ventricular activation, because the AV node is refractory until the last QRS complex. In the last QRS complex, a slightly narrower QRS complex is noted because some ventricular tissue is being activated by the preceding P wave and AV nodal conduction.

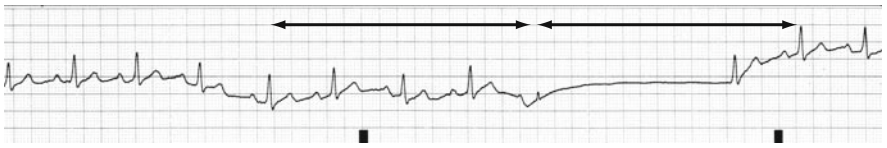


Figure 6: Artifact mimicking a sinus pause. In this rhythm strip, patient movement leads to an apparent sinus pause. Notice that the QRS complex after the "pause" occurs at the expected time. Constant intervals between QRS complexes suggest a stable heart rate and would be extremely unlikely for a sinus pause.

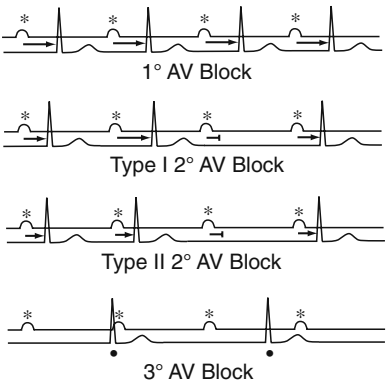


Figure 7: Schematic showing the basic types of AV block. In 1° AV block the P waves (*) and QRS complexes maintain a 1:1 relationship, but the PR interval is abnormally long. In 2° AV block some but not all P waves conduct to the ventricle. In Type I 2° AV block the PR interval gradually prolongs before the P wave blocks. In Type II 2° AV block, the PR interval remains constant before and after the blocked P wave. In 3° AV block, there is no relationship between atrial activity (*) and ventricular activity (•).

degree AV block is further classified as Type I 2° AV block, Type II 2° AV block, and 2:1 2° AV block. Woldemar Mobitz was the first to classify second degree AV block into two types (Type I vs. Type II) based on ECG characteristics. In Type I 2° AV block the PR interval gradually increases until a dropped QRS occurs (P wave without a subsequent QRS complex). An example of Type I 2° AV block is shown in Figure 9. Another patient with inducible second degree AV block is shown in Figure 10. This patient complained of shortness of breath with exertion. At baseline the ECG shows normal sinus rhythm with borderline first degree AV block and right bundle branch block. When the patient exercises on a treadmill the ECG in Figure 11 is obtained. In this case every P wave does not result in a QRS complex because of the development of Type I second degree AV block. Second degree AV block develops because the sinus rate increases and the AV node becomes partially refractory. In Type II 2° AV block the PR interval stays constant and there is a sudden dropped QRS complex (Figure 12). Traditionally, Type I 2° AV block is identified by gradual prolongation of the PR interval until a dropped QRS is observed (Figures 8 and 10). However, in some cases this pattern can be difficult to identify and occasionally the PR interval will actually decrease (Figure 12). The easiest way to distinguish between Type I and Type II 2° AV block is to evaluate the PR interval before and after the dropped QRS. If the PR interval after the dropped beat is shorter (due to more time for the AV node to recover), Type I 2° AV block is present (Figures 9, 11, 13). Type I 2° AV block is often called Wenckebach block for Karl Wenckebach, the German physiologist who first considered the possibility of progressive AV delay by examining the relationship between the arterial pulse and the

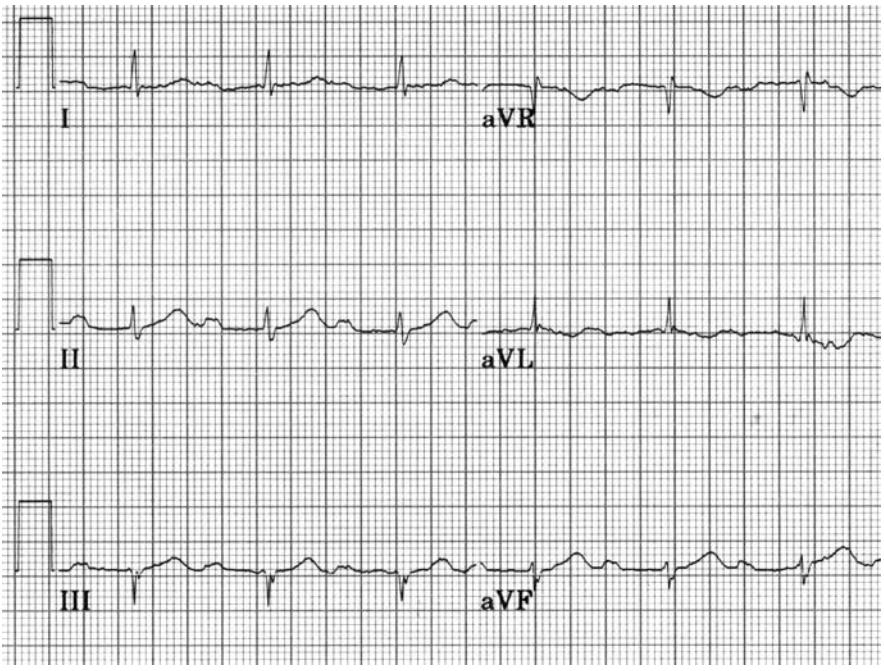


Figure 8: Frontal leads in a patient with first degree AV block. The P waves and the QRS complexes have a 1:1 relationship, but the PR interval is abnormally long (0.27 s).

jugular venous waves. In 2:1 2° AV block, every other P wave is conducted to the ventricle (Figure 14). In this situation it is impossible to tell whether the PR interval is lengthening, since there are no consecutively conducted beats. In this case prolonged continuous ECG monitoring is required to determine whether Type I or Type II 2° AV block is present. As described below, the distinction between Type I and Type II 2° AV block is important because it provides information for the specific site of atrioventricular block. Reexamine Figure 11. The first few beats show 2:1 2° AV block. With continued monitoring, the patient would be classified as having Type I 2° AV block.

In third degree AV block, there is a complete conduction block between the atria and the ventricles. The P waves and QRS complexes will have their own distinct rates, and no relationship between atrial and ventricular activity will be observed. Oftentimes P waves can be seen as deflections within the ST segment and T wave.

There is a final category called advanced or high grade AV block. In this case, as in second degree AV block, some association between atrial depolarization and ventricular depolarization can be observed. However, in advanced AV block more than one P wave not associated with ventricular depolarization—that is > 2 consecutive P waves not leading to QRS complexes—can be observed (Figure 15).

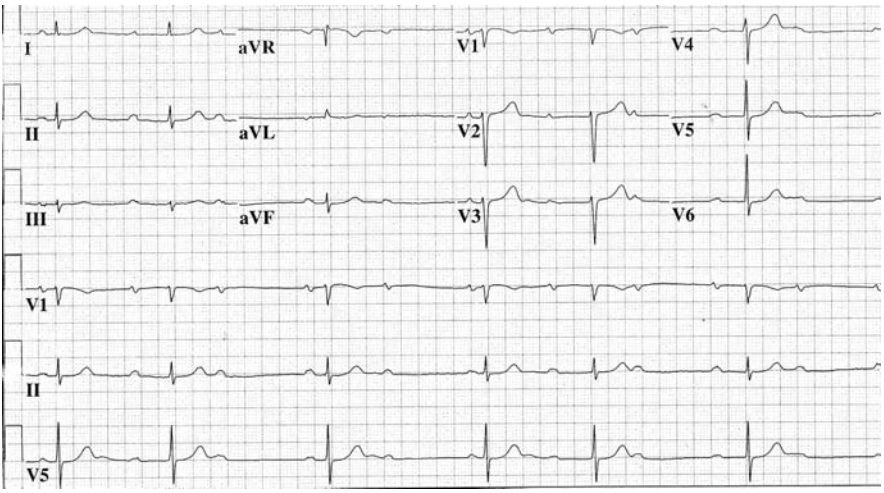


Figure 9: Second degree AV block Type I. The atria are normally activated by the sinus node, leading to upright P waves in II, III, and aVF and an inverted P wave in aVR. However, not every P wave is conducted to the ventricles. The PR interval gradually increases until a nonconducted P wave and a dropped QRS complex is observed.

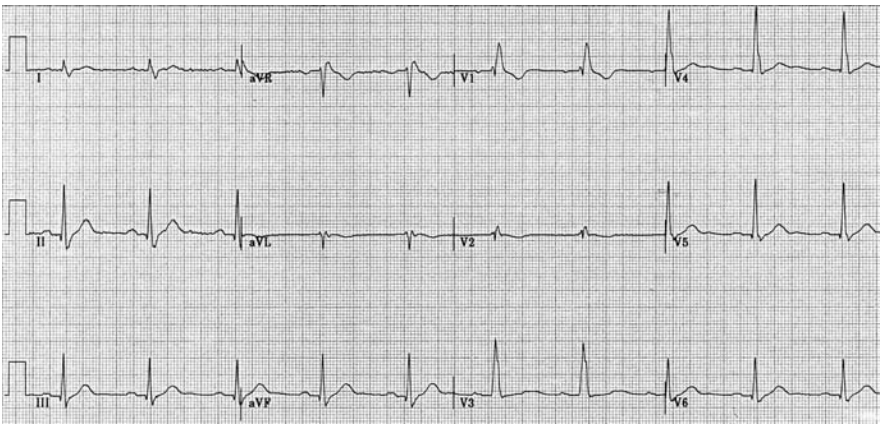


Figure 10: ECG from a patient complaining of shortness of breath when he exercises. At baseline the ECG shows borderline first degree AV block and right bundle branch block.

It is important for the clinician to be aware of a form of AV block that can be observed normally, particularly in younger patients. In "vagal" AV block, transient asymptomatic Type I 2° AV block can be observed, particularly during sleep, due to an increase in parasympathetic or vagal tone. An example of "normal" Type I 2°

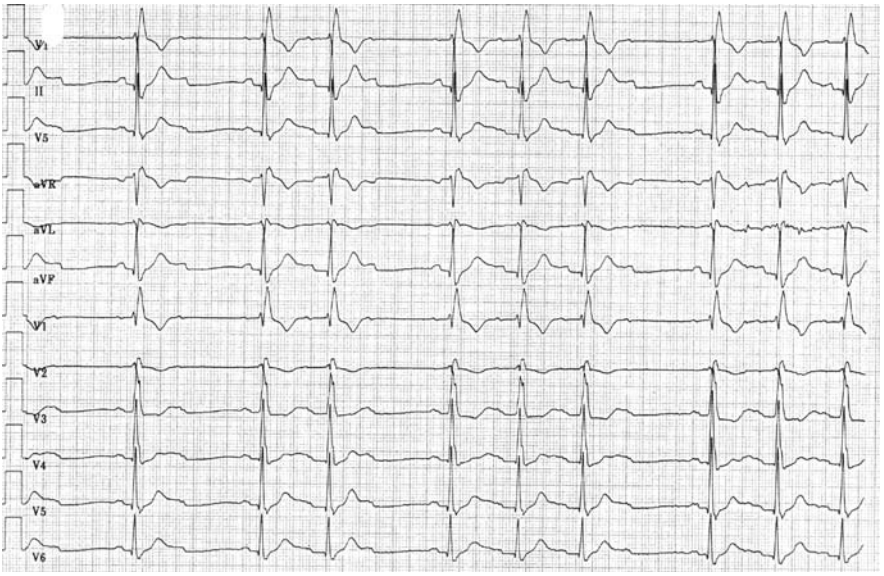


Figure 11: ECG from the same patient as Figure 10 when he exercises. With an increase in the sinus rate, the patient now develops Type I second degree AV block due to refractoriness in the AV node.

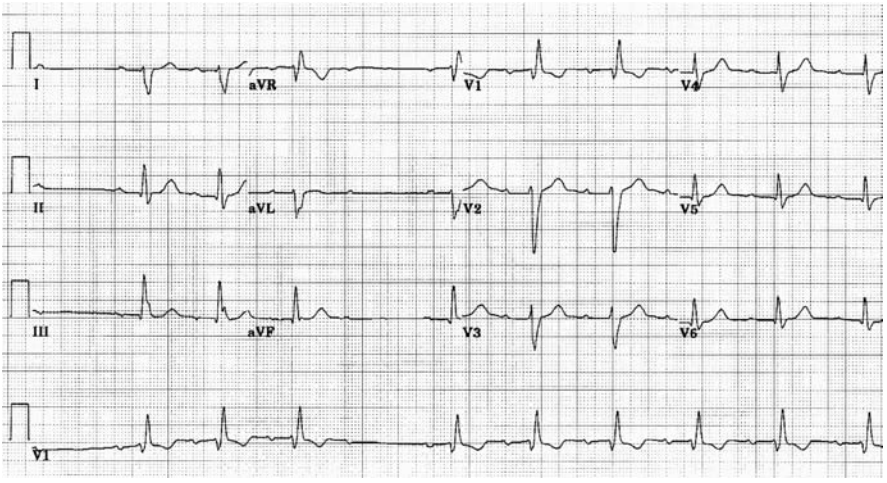


Figure 12: Second degree AV block Type II. The patient has baseline right bundle branch block. The PR interval is constant before the nonconducted P wave. The easiest way to identify 2° AV block Type II is the presence of a constant PR interval both before and after the nonconducted P wave.

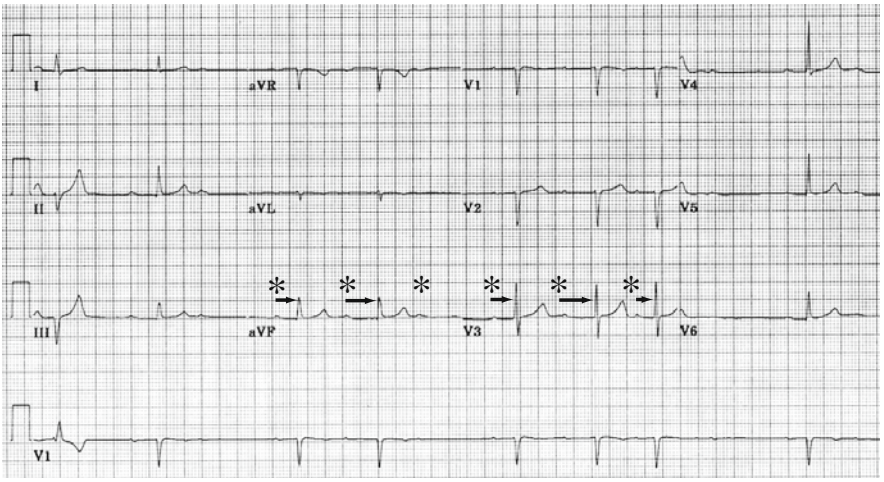


Figure 13: Unusual form of Type I 2° AV block. The typical pattern of gradual PR interval prolongation is not consistently observed. In this case, the PR interval actually decreases during one sequence. Since the PR interval after the dropped QRS complex is shorter than the PR interval preceding the dropped QRS complex this form of second degree AV block would be classified as Type I.

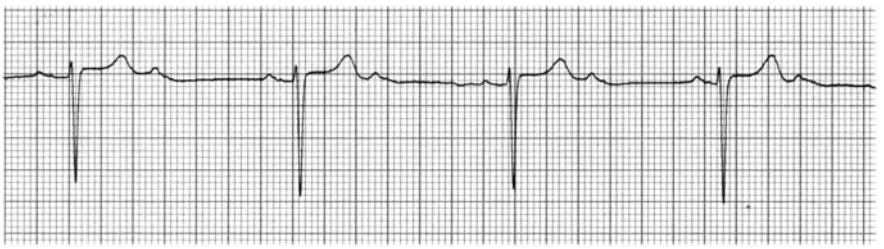


Figure 14: 2:1 AV block. Every other P wave is conducted to the ventricles. In this case, it is impossible to distinguish between Type I and Type II 2° AV block. However, as discussed later, the presence of a narrow QRS complex suggests that Type I 2° AV block is present.

AV block due to increased vagal tone is shown in Figure 16. The diagnosis of vagal AV block can be made if associated sinus node slowing is observed (double-headed arrows), since the increase in vagal tone affects both the sinus node and the AV node. Isolated Type I 2° AV block due to increased vagal tone that develops during sleep should be considered a normal finding and should not raise alarm, particularly if the patient does not have symptoms (dizziness, passing out).

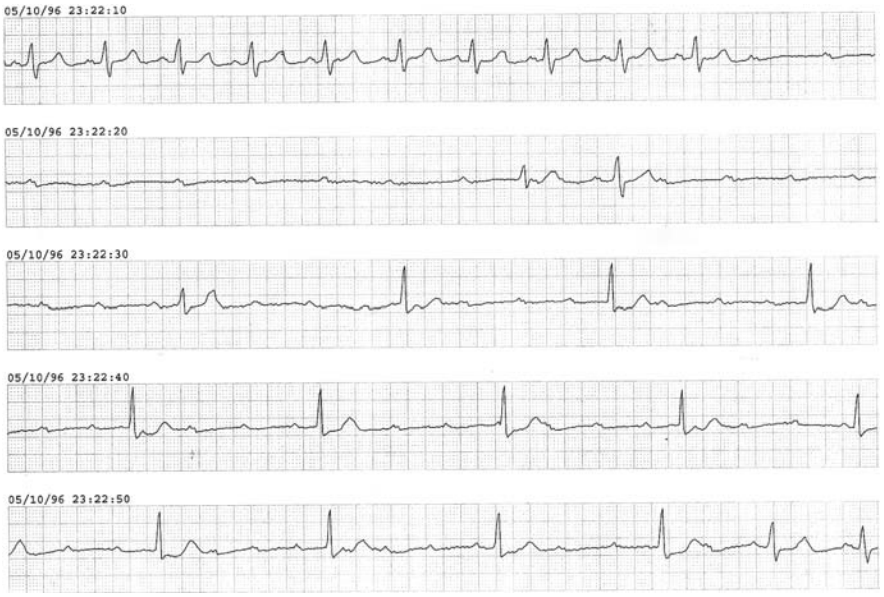


Figure 15: Advanced atrioventricular block. In this continuous strip, a number of nonconducted P waves are observed. Complete heart block is not present since some P waves do conduct to the ventricles and lead to QRS complexes.

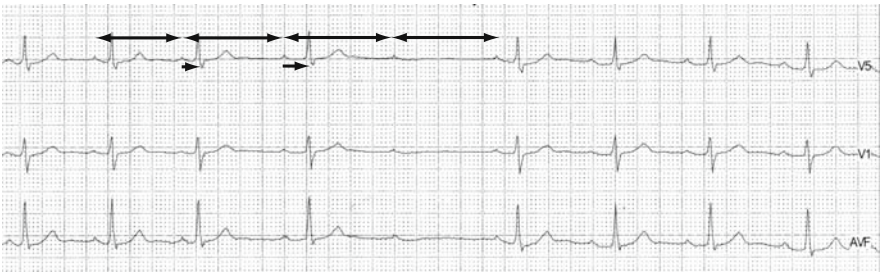


Figure 16: An episode of vagally induced Type I 2° AV block. The development of 2° Type I AV block (*single-headed arrows*) is associated with slowing in the sinus rate (*double-headed arrows*), since increase in vagal tone affects both sinus node and AV node properties.

Types of AV block

- ***First degree AV block:*** *All P waves produce a QRS (1:1 relationship) but the time required for conduction is longer (PR > 0.20 s).*

- ***Second degree AV block:*** *Some but not all P waves conduct to the ventricles and produce a QRS complex:*
  - *Type I 2° AV block*
  - *Type II 2° AV block*
  - *2:1 AV block*
  - *High grade AV block*
- ***Third degree AV block:*** *No P waves conduct to the ventricles.*

Case Study (continued):

The ECG in Figure 1 shows a 3° or complete heart block. The P waves and QRS complexes are not related. The slow ventricular rates are probably responsible for the patient's symptoms of tiredness and fatigue.

When evaluating atrioventricular block the rate and associated symptoms must first be evaluated. The second, related issue is to determine the anatomic site of the block: the AV node or His bundle. If the block is within the AV node, a site in the distal portion of the AV node will pace the heart at 40–50 beats per minute if complete heart block were to develop. On the other hand, if the block is within the His bundle, a pacemaker from fascicular tissue or ventricular tissue will pace the heart. These pacemakers have slow heart rates (20–40 beats per minute) and are notoriously unreliable. Generally, pacemakers are placed in symptomatic patients having block within the AV node, or in any patient having block within the His bundle.

The ECG can provide several clues as to the site of the block (Figure 17). In general, severe first degree AV block (> 0.30 s) is due to block within the AV node because the AV node can be associated with significant delays, while maximal delay in the His bundle is relatively limited because conduction velocity in the His bundle is rapid. For patients with second degree AV block, the presence of Type I second degree AV block suggests block within the AV node. Conversely, Type II 2° AV block is observed with His bundle block; remember that the conduction tissue in the His bundle is sodium channel dependent and tends to be "all or nothing." For patients with second degree AV block, the associated QRS complex can provide an additional clue. If the QRS complex is wide, conduction tissue disease is present and block within the His bundle becomes more likely. For patients with third degree AV block, if the QRS complex is narrow (< 0.12 s), block within the AV node is most likely, while a wide QRS complex suggests block within the His bundle.

Evaluation of the QRS pattern can help differentiate between second degree and third degree AV block. In complete heart block the QRS complexes will be regular, since there are no conducted P waves. In second degree AV block, some irregularity in the QRS pattern will be observed. A patient with second degree AV block is shown in Figure 18. The irregular QRS pattern implies that some AV conduction is present. The one QRS complex with the slightly different morphology is a junctional escape beat (marked by a *).

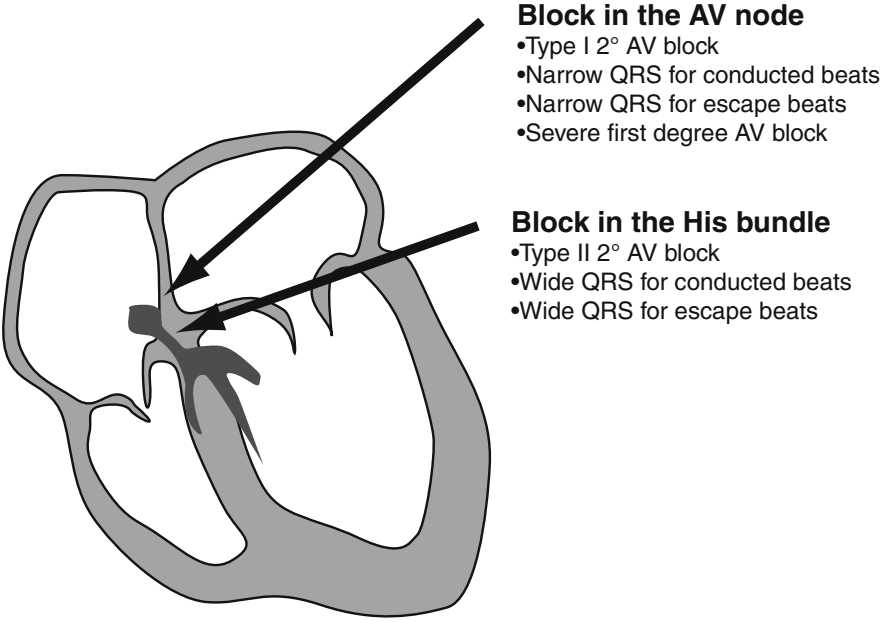


Figure 17: ECG clues for identification of the site of block.

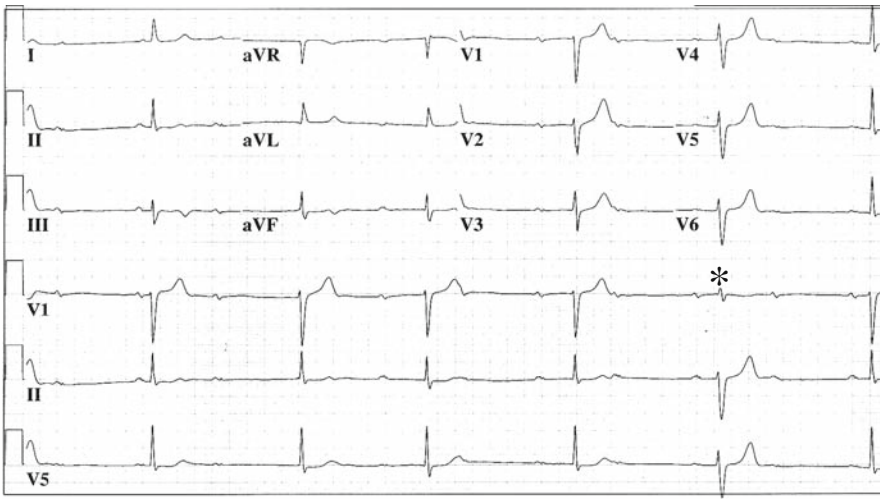


Figure 18: ECG from a patient with atrioventricular block. The third QRS complex is "early" suggesting that some atrioventricular conduction is present (albeit very poor). The QRS marked with an asterisk (*) probably represents an escape beat from a "lower site" on the atrioventricular axis.

Case Study (continued):

The ventricular escape complex is relatively wide. Notice the morphology of the QRS complex. It has a right bundle branch block appearance, which suggests that the block is below the His bundle. If a narrow QRS complex escape beat is noted, the block must be within the AV node.

When evaluating atrioventricular block it is important to keep in mind that the atrial rate must be normal. The slow conduction properties of the AV node "protect" the ventricles from rapid atrial rates. It is normal to observe variable conduction of atrial activity in the presence of rapid atrial rates. For example, as discussed in the next chapter, in atrial flutter the atria are repetitively activated at a rate of 300 beats per minute. If the AV node and His bundle permit conduction of all atrial activity, a ventricular rate of 300 beats per minute will be observed. Such rapid ventricular rates can lead to severe symptoms and can be potentially life-threatening. Fortunately, in this case the AV node will usually allow only some of the atrial activity to lead to ventricular depolarization, which provides a "regulator" to slow the ventricular rate.

Key points

1. Mechanistically, the two causes for slow heart rates are abnormal automaticity and blocked atrioventricular conduction.
2. Sinus node dysfunction (abnormal automaticity) can have a number of manifestations on the ECG, including sinus bradycardia and sinus pauses.
3. Slowing of the sinus rate can be associated with junctional rhythm or ectopic atrial rhythm, as pacemaker sites other than the sinus node "take over."
4. Atrioventicular block can be classified as 1° where every P wave leads to a QRS, but with a prolonged atrioventricular conduction time; 2° where some but not all atrial activity conducts to the ventricles; and 3° where there is no communication between the atria and the ventricles.
5. Atrioventricular block can be due to block in the AV node or His bundle. Clinically this is an important distinction, since block in the His bundle can lead to severe bradycardia because of very unreliable auxiliary pacemakers.

Questions

1. Slow heart rates can be due to:

   (i) Abnormal automaticity of the sinus node
   (ii) Abnormal automaticity of the AV node
   (iii) Block in the AV node
   (iv) Block in the left bundle

   A. (i) and (iii)
   B. (ii) and (iv)

C. (i),(ii), and (iii)
D. All of the above

2. Which statement is false?

A. The sinus node is the normal pacemaker for the heart.
B. Sinus rhythm is the only heart rhythm in which a P wave is seen before the QRS complex with a consistent relationship.
C. Junctional rhythm can be seen in patients with sinus node dysfunction.
D. Collagen deposition in the sinus node increases with age.

3. Which statement is false?

A. Atrioventricular block can be present in either the AV node or His bundle.
B. In atrioventricular block the P waves can be generated by the sinus node.
C. Second degree AV block is always associated with a slow regular pulse.
D. Type I AV block is usually due to conduction block within the AV node.

4. Examine Figure 1. The patient has complete heart block. Where is the pacemaker site that is depolarizing the ventricles?

A. AV node
B. Right bundle
C. Left anterior fascicle
D. Left posterior fascicle

Answers

1. The correct answer is A. Both abnormal sinus node automaticity and block within the AV node will be associated with slow heart rates. Block in the left bundle branch will cause a wide QRS complex but will not alone cause slow heart rates. Similarly, abnormal AV node automaticity by itself will not be associated with slow heart rates, although abnormal AV node automaticity in conjunction with abnormal sinus node automaticity can lead to very slow ventricular rates (due to a ventricular pacemaker) or asystole (no ventricular activation).
2. Statement B is false. In a patient with an ectopic atrial rhythm, an atrial pacemaker separate from the AV node leads to atrioventricular conduction and ventricular depolarization. A P wave due to atrial depolarization will be observed before every QRS complex. However, the P wave will have a different morphology than the P wave generated by sinus rhythm.
3. Statement C is false. In 2° AV block some but not all P waves are conducted to the ventricle. In many cases this leads to an irregular pulse. The one exception is 2:1 AV block in which every other P wave is conducted to the ventricles and a slow regular pulse will be observed.
4. The correct answer is C. The QRS complex has a right bundle branch block, left posterior fascicular block morphology. This suggests that the pacemaker site is within the left anterior fascicle, since a site here would lead to late activation of the posterior left ventricle and right ventricle.

Chapter 11
Supraventricular tachycardia

Traditionally, any heart rate greater than 100 beats per minute is classified as a tachycardia. Tachycardia can frequently be a normal finding; young adults can have heart rates of 150–180 beats per minute at peak exercise due to sympathetic activation. However, tachycardia can be an abnormal finding associated with hemodynamic collapse. Tachycardias are usually classified by their QRS morphology/duration: normal or wide complex. If the QRS complex has a normal duration and morphology, the ventricles are being activated normally and a tachycardia site within the ventricle is excluded. For this reason narrow QRS complex tachycardias are frequently called supraventricular tachycardias.

Although classifying tachycardias by their QRS morphology is useful, it is also important to keep the mechanistic and anatomic classification of tachycardia in mind.

Ms. Jennifer Saltsman is a 23-year-old woman with a history of intermittent rapid heart rates. She was doing well until several hours ago when she felt a "fluttering" in her chest and has had continued fast heart rates and has come to the emergency room. Her ECG is shown in Figure 1.

Tachycardia mechanisms

Three mechanistic causes for a fast heart rate are: increased automaticity, triggered activity, and reentry (Figure 2).

Increased automaticity

The simplest example of increased automaticity would be increased phase 4 depolarization from sympathetic activation of the sinus node, leading to sinus tachycardia. In some conditions, cells that usually do not normally display "pacemaker" activity can develop abnormal phase 4 depolarization and lead to rapid heart rates. The most commonly observed examples of this are accelerated ventricular rhythms that can be observed in the setting of myocardial injury, where ventricular rates of 80–100 beats per minute can be observed, particularly when blood flow is

F. Kusumoto, *ECG Interpretation: From Pathophysiology to Clinical Application*,
DOI 10.1007/978-0-387-88880-4_11, © Springer Science+Business Media, LLC 2009

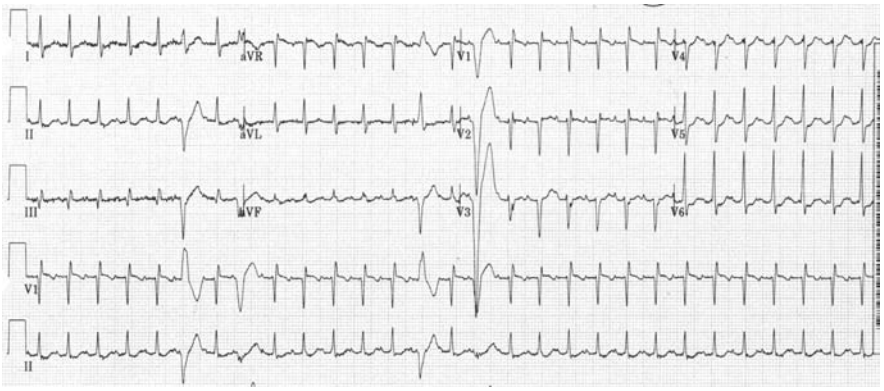


Figure 1: Ms. Saltsman's ECG.

reestablished in a previously occluded coronary artery. This effect appears to be mediated by inward Na^+ flow during phase 4.

Triggered Activity

In triggered activity, reactivation of sodium or calcium channels leads to spontaneous depolarizations. This special form of automaticity is usually called triggered activity because it usually requires preceding depolarization. If the triggered activity occurs in phase 3 of the action potential, the abnormal activity is usually referred to as early afterdepolarization (EAD). If the triggered activity occurs after repolarization has completed and the cell has returned to the resting membrane potential, it is called delayed afterdepolarization (DAD). Early afterdepolarizations are more likely in conditions associated with prolongation of the action potential duration, while delayed afterdepolarizations are associated with conditions involving increased intracellular calcium. Triggered activity is discussed in greater detail in Chapter 6 with the consideration of torsade de pointes.

Reentry

Reentry is the most common mechanistic cause for rapid heart rates. In this chapter for example, we will see that reentry is the mechanism responsible for atrial flutter, AV node reentrant tachycardia, and atrioventricular reentry. In the traditional model of reentry, two parallel, electrically isolated pathways with different electrophysiologic properties exist (Figure 2). Regular heartbeats conduct down the fast pathway and slow pathway. However, a premature beat can block in one pathway (due to refractory tissue) and conduct down the alternate pathway, often with a slower conduction velocity; the electrical impulse "reenters" the first pathway

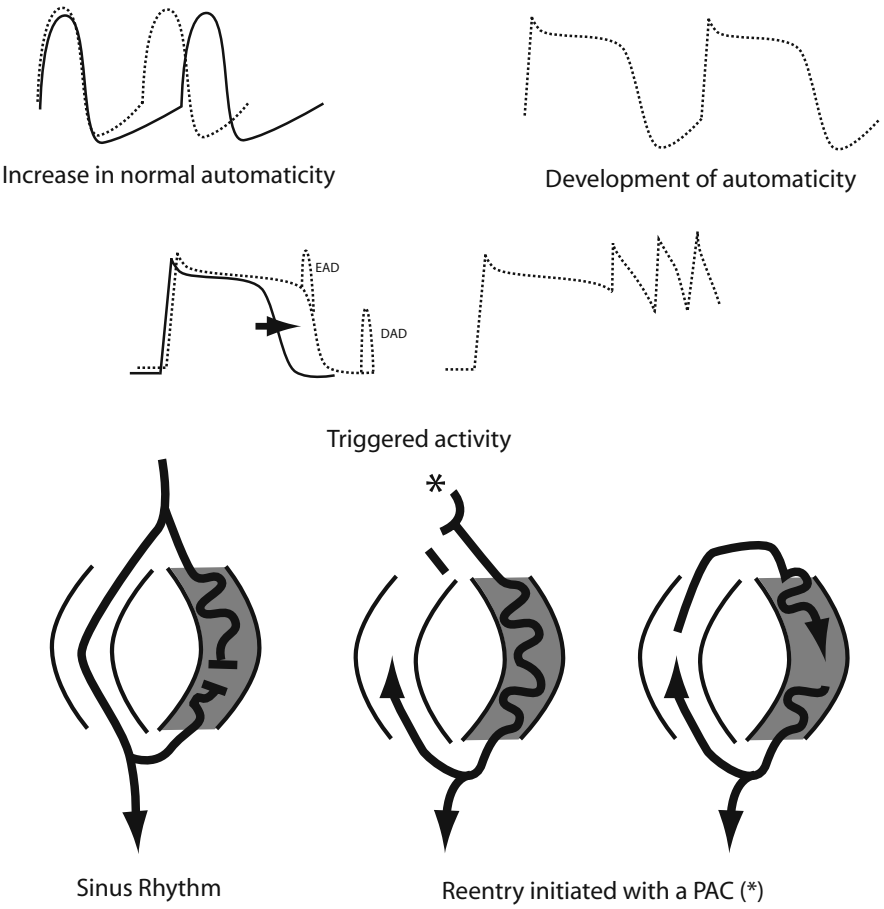


Figure 2: Cellular/tissue mechanisms for tachycardia. *Top row:* Increased automaticity can lead to faster heart rates. The most common example of this would be sinus tachycardia. Another possibility is development of automaticity in tissue that normally does not have pacemaker activity. *Middle row:* Triggered activity is a form of abnormal automaticity characterized by afterdepolarizations due to Na and Ca channel activation either during phase 3 (early afterdepolarizations) or after repolarization has completed (delayed afterdepolarizations). *Bottom row:* In reentry two parallel paths with different conducting and refractory properties are present (slow pathway with shorter refractory periods in *gray*). During sinus rhythm the wave of depolarization conducts over the fast pathway and the depolarization wave is extinguished within the slow pathway. A premature beat can block in one pathway and conduct slowly down the second pathway. The wave of depolarization can enter the first pathway retrogradely, and if it does not encounter refractory tissue, a self-perpetuating circuit can develop.

(which has recovered from refractoriness); and a reentrant wave of electrical activity can be initiated and become sustained (Figure 2). Initiation of reentry requires a specific electrophysiologic substrate: parallel pathways with different electrophysiologic properties and a perfectly timed premature beat. Given these requirements, it would appear that reentry would be a very rare event; but it is actually the most common mechanism for abnormal tachycardias encountered in clinical medicine.

Anatomic classification

Anatomically, tachycardia can arise from atrial tissue, the junctional region, or ventricular tissue, or it can utilize an accessory pathway (Figure 3). A tachycardia arising from the ventricles would yield a wide complex arrhythmia due to abnormal activation of the ventricles and will be specifically discussed in the next chapter; so that from an anatomic standpoint a narrow QRS tachycardia can be due to: atrial tachycardia, junctional tachycardia, or atrioventricular reentry utilizing an accessory pathway.

Atrial tachycardia

1. Sinus tachycardia

The most common reason for a heart rate > 100 bpm with a narrow QRS complex is an increase in the sinus node rate. The sinus node receives input from the sympathetic nervous system. With sympathetic activation (stress, exercise) the sinus rate increases. This appears to be mediated by phosphorylation of ion channels that leads to more rapid diastolic depolarization (increased I_f and increased I_{Ca-T}). Since the sinus node continues to activate the atria, the atria are activated from "superior to inferior" and "right to left," so the P wave is negative in aVR and positive in lead II. Since sympathetic activation also leads to more rapid AV nodal conduction, the P wave and the QRS complex usually maintain the same relationship, with the P wave just before the QRS complex.

2. Focal atrial tachycardia

In focal atrial tachycardia, a single site away from the sinus node activates the atria. This can be due to automaticity or triggered activity. Fast atrial rates due to reentry are usually classified as atrial flutters (see below), although it is important to remember that a small reentrant circuit could appear "focal" in nature. From the ECG alone it is often impossible to determine the mechanism for the tachycardia. From surface recordings, initiation and termination of the tachycardia are usually the only times that mechanistic information can be obtained. Reentrant tachycardias are generally initiated by a premature atrial or ventricular depolarization, while automatic tachycardias simply "start" or "stop" suddenly. Automatic tachycardias will sometimes display a gradual "warm-up" or "warm-down" similar to sinus tachycardia.

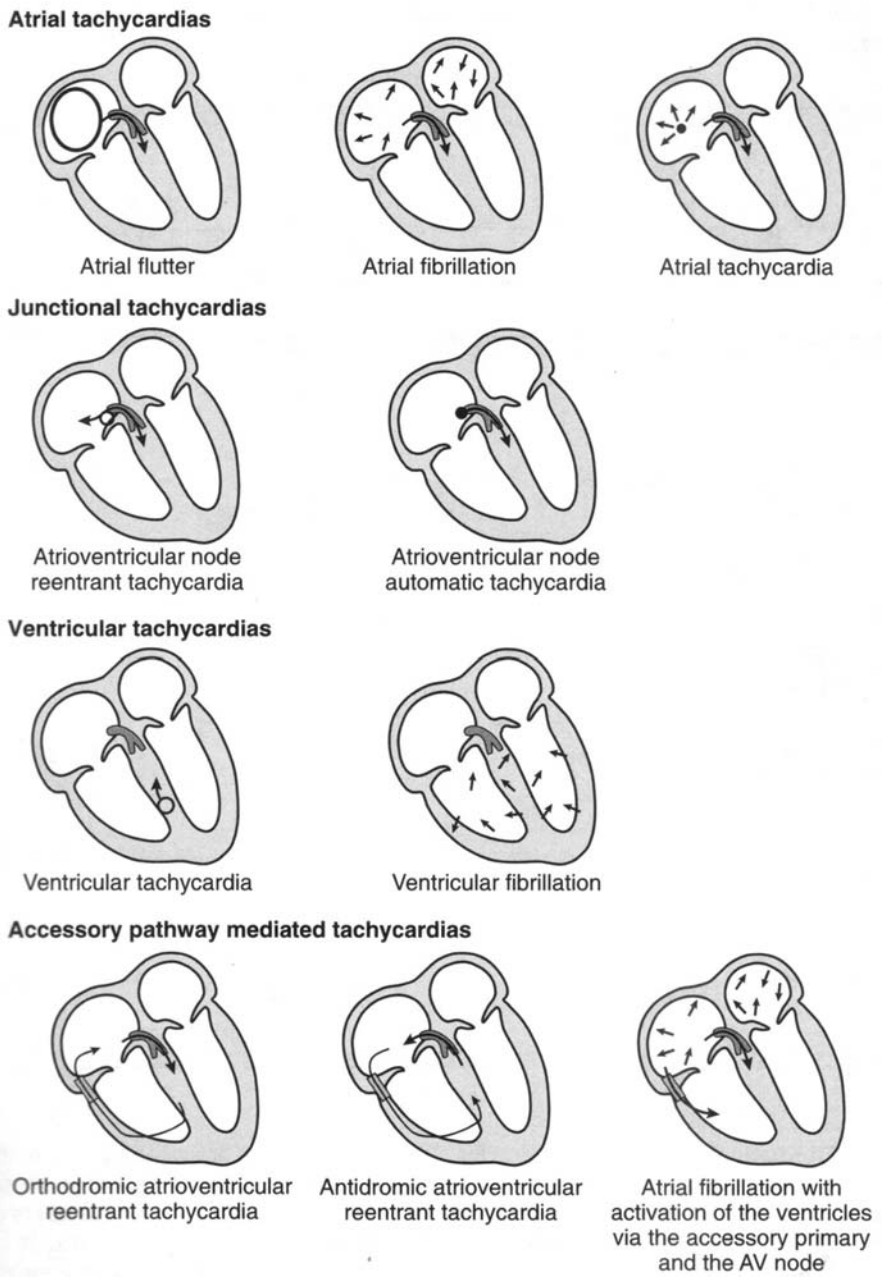


Figure 3: Anatomic tachycardia classification: Tachycardias can arise from atrial tissue, junctional tissue, or ventricular tissue, or by utilizing an accessory pathway. (reprinted with permission from Kusumoto FM, *Cardiovascular Pathophysiology*, Hayes Barton Press, Raleigh, NC, 1999).

The P wave morphology in focal atrial tachycardia will depend on the location of the abnormal site where the rapid depolarization is emanating from. For example, in a tachycardia from the lateral wall of the left atrium, the P wave will be inverted in aVL (Figure 4). Since the nonphysiologic activation of the abnormal site does not necessarily lead to sympathetic activation of the AV node, the relationship of the P wave and the QRS complexes will depend on the tachycardia rate and refractoriness of the AV node. For example, a P wave in the "expected" location could be observed, the P wave could be associated with first degree AV block, or not every P wave can be conducted to the ventricles ("second degree AV block"). In Figure 4, because the tachycardia rate is relatively slow and AV node conduction is excellent,

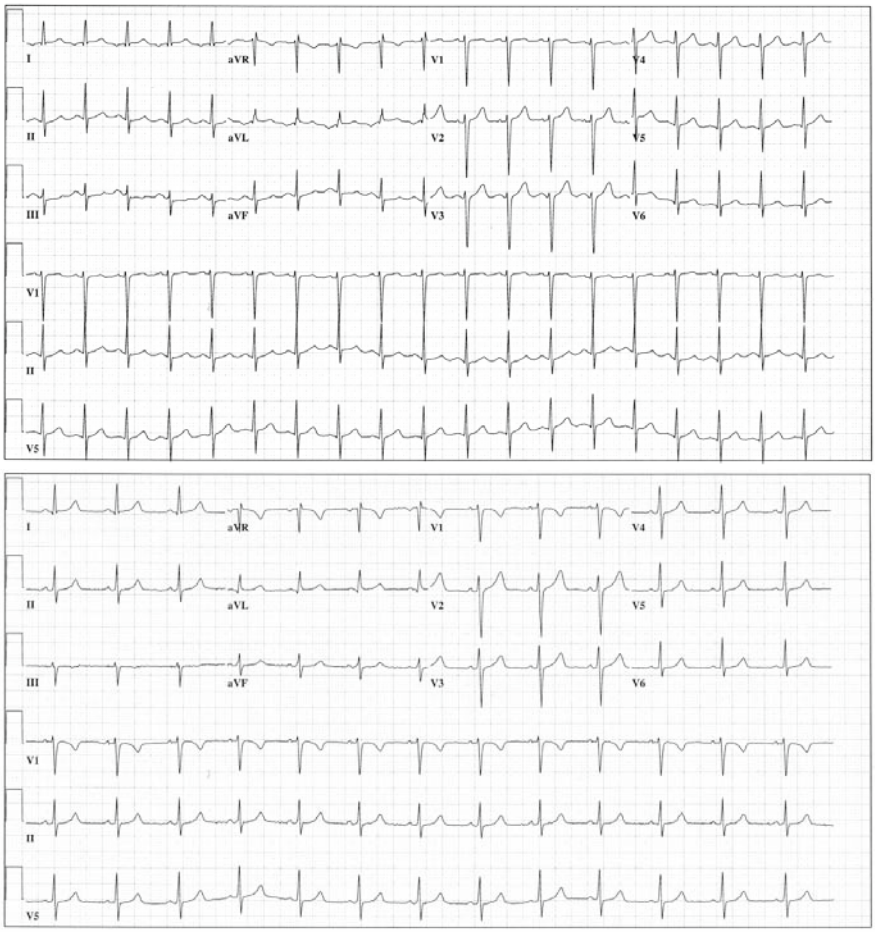


Figure 4: Patient referred for sinus tachycardia and tiredness. *Top*: Baseline ECG. Notice that the P wave is inverted in aVL. This is an atrial tachycardia from near the left upper pulmonary vein. *Bottom*: After a procedure to ablate the abnormal site, the patient's ECG is now normal. Notice the change in the P wave morphology.

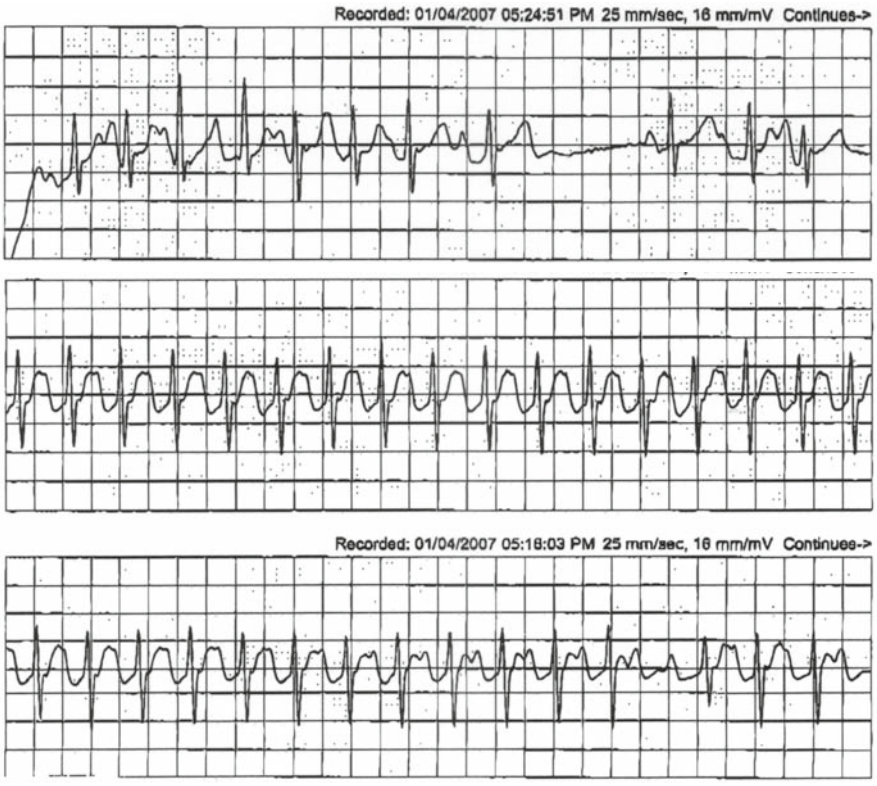


Figure 5: A patient with atrial tachycardia arising from a focal site within the right upper pulmonary vein. *Top:* An ECG tracing from one of the patient's episodes. The tachycardia terminates, one sinus beat is present, and the tachycardia "just starts" without a premature beat, suggesting automaticity as a cause for the arrhythmia. *Bottom:* During another episode the ECG shows block within the AV node due to refractoriness. As will be discussed later, if the tachycardia can continue in the presence of AV block, it is called an AV node independent tachycardia.

the PR interval appears normal. In contrast, in Figure 5, an ECG is shown from a young woman with episodes of rapid heart rate due to an automatic site within the right upper pulmonary vein. The atrial tachycardia rate is rapid, so in some cases variable block occurs within the AV node. As a separate issue, it should be noted that the first and subsequent P waves of the tachycardia are similar in morphology; this suggests a focal tachycardia due to automaticity rather than reentry.

3. Multifocal atrial tachycardia

In multifocal atrial tachycardia, multiple sites have increased pacemaker activity and depolarize the atria. This leads to several different P wave morphologies, with the shape of each P wave dependent on the location of the abnormal site

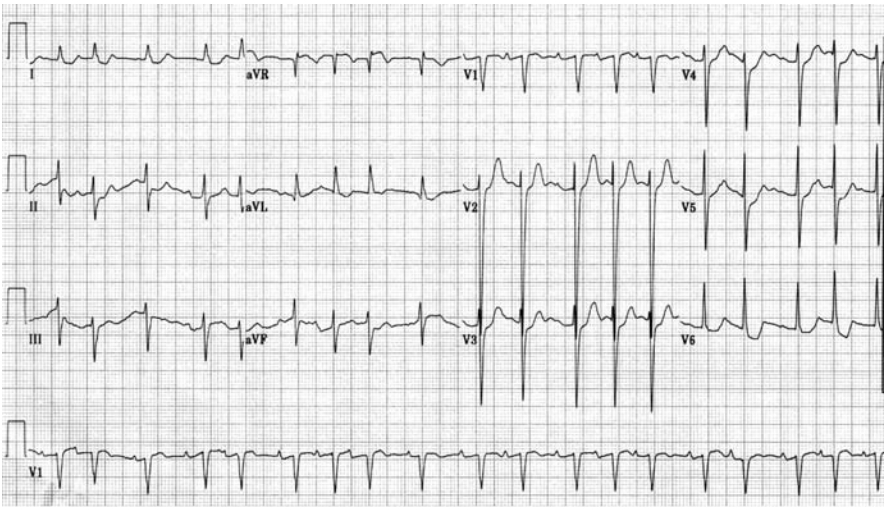


Figure 6: 12-lead ECG from a patient with multifocal tachycardia associated with severe hypoxia due to pulmonary disease.

leading to that specific atrial activation (Figure 6). Since each of the sites has a different depolarization rate, the atrial rate is irregular; and as a consequence, the QRS rate (the measured heart rate) will also be irregular. Multifocal atrial tachycardia is often observed in the presence of other abnormalities, such as hypoxia and acidosis. Experimental and clinical data suggest that multifocal atrial tachycardia is often due to triggered activity.

4. Atrial fibrillation

In atrial fibrillation there is continuous activation of the atria. The traditional explanation for atrial fibrillation is multiple wandering wavelets within the atria that can coalesce or split into different patterns (Figure 7). Another possible mechanism is a single, very rapidly firing site or reentrant circuit in which the rest of the atria cannot follow in an organized matter. More recently, it appears that very small reentrant circuits that produce emanating spiral waves that form "arms" of activation can cause atrial fibrillation (Figure 8). As these arms of depolarization run into obstacles such as a region of refractory tissue or a structure such as a vessel opening, the wavelet form splits into daughter wavelets. Given the anatomy of the atria with its multiple "holes" (tricuspid valve annulus, mitral valve annulus, vena cavae, pulmonary veins, and three-dimensional complexity (two atrial appendages and interatrial septum), it is not surprising that irregular atrial activation associated with atrial fibrillation can develop relatively easily.

Regardless of mechanism, on an ECG atrial fibrillation is defined as irregular atrial activity that does not lead to discrete P waves with intervening isoelectric periods. The fibrillatory waves appear as irregular complex low amplitude activity. Although the ECG manifestations may be similar, effective treatment for atrial fibrillation will be mechanism dependent.

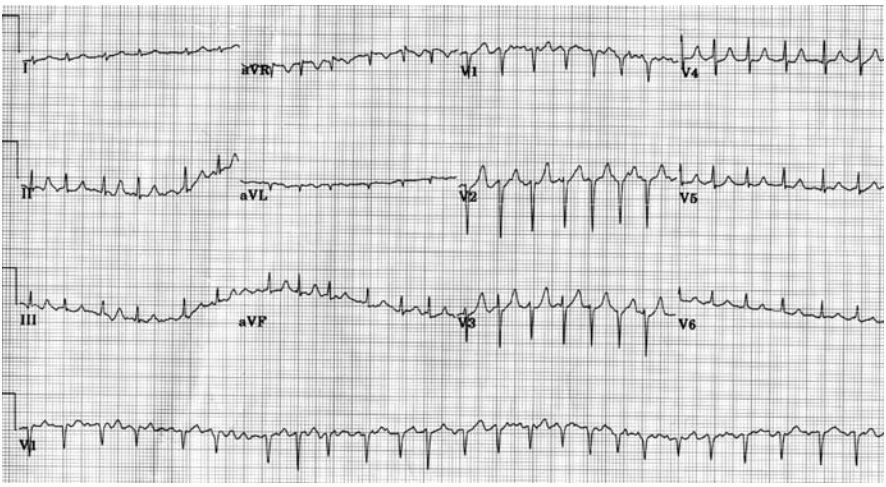


Figure 7: 12-lead ECG of atrial fibrillation. Irregular and continuous atrial activation leads to fibrillatory waves without an intervening isoelectric period. Irregular atrial activation leads to irregular ventricular activation.

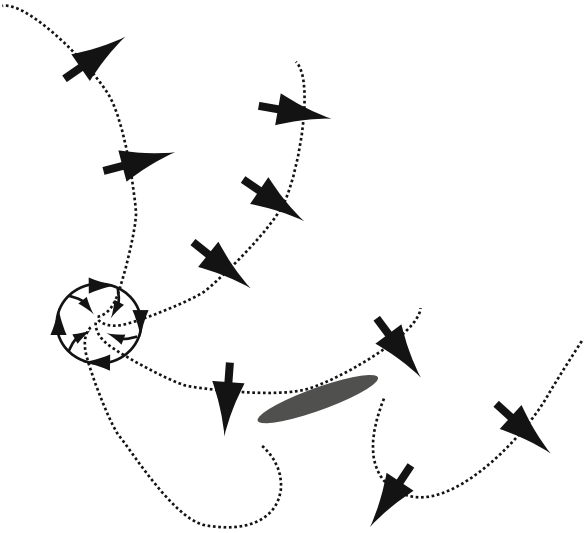


Figure 8: Cartoon showing one of the potential mechanisms of atrial fibrillation. A rotor is a very localized reentrant circuit that appears to propagate "arms" of activation. When the "arms" meet obstacles—a region of refractory tissue, a "hole" such as a valve or vein opening—the arms split into "daughter" rotors.

The atrial rate is very rapid, 300 to 1000 beats per minute. Fortunately, the AV node limits the ventricular response rate, although in younger patients fairly rapid rates can be observed. The ventricular rate will depend on the refractoriness of the AV node.

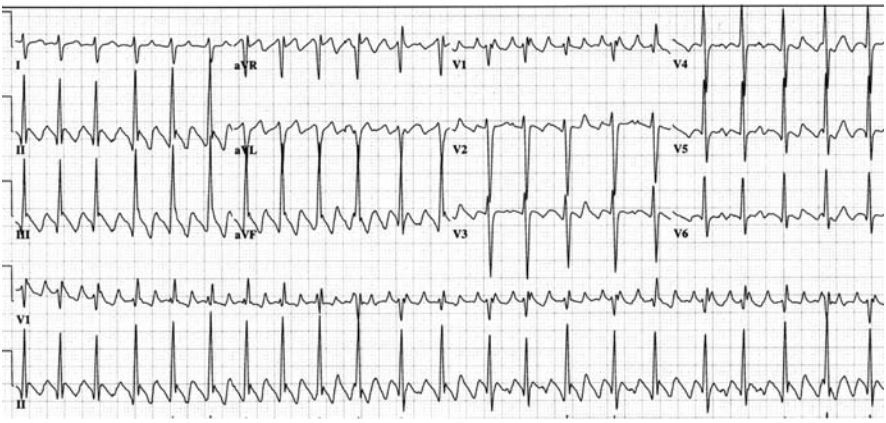


Figure 9: Typical atrial flutter. Negative "sawtooth" flutter waves are seen in the inferior leads.

5. Atrial flutter

Atrial flutter is a term generally used for atrial arrhythmias caused by relatively large and stable reentrant circuits exclusively within atrial tissue. The most common type of atrial flutter uses the tissue isthmus formed by the tricuspid valve and the inferior vena cava. A premature beat blocks at one "end" of the isthmus and activation travels through the isthmus, circles around the tricuspid valve, and reenters the other "end" of the isthmus. This type of flutter is called isthmus dependent flutter or typical atrial flutter. The flutter waves are usually negative in the inferior leads (II, III, and aVF), since the left atrium is activated in the inferior to superior direction (Figure 9).

Atrial flutters can also revolve around other anatomic barriers; for example atrial flutter circuits can revolve around the mitral annulus using the isthmus formed by the mitral valve and the pulmonary veins. These will lead to flutter waves that have a different morphology than typical atrial flutter (Figure 10). In Figure 10, the patient has an atrial flutter that is circulating around the mitral valve. It is almost impossible to tell the exact circuits for atrial flutters, but in general, left atrial flutters will not be associated with the deeply negative flutter waves that are usually observed in typical atrial flutter.

The ventricular rate in atrial flutter is usually slower than the atrial rate because of AV nodal block. This is another example of the AV node "protecting" the ventricles from very rapid rates. As a corollary, the ventricular rate during atrial flutter can provide some clues for the AV conduction properties in a patient. A patient with typical atrial flutter is shown in Figure 11. Notice that AV nodal disease is suggested, since there is one conducted beat for every five to seven flutter waves. Once the atrial flutter has resolved, the patient has sinus rhythm with 2° AV block Type I (*middle row*). After the patient's beta-blocker is stopped, the patient has improvement in AV nodal conduction (*bottom row*).

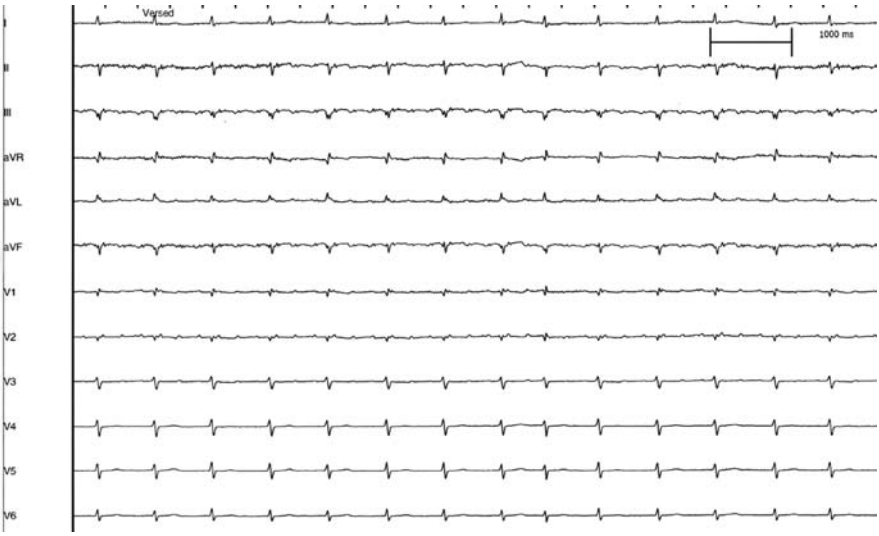


Figure 10: Atypical atrial flutter. Compare the flutter waves to the prior ECG. Notice that the flutter waves are not as prominent.

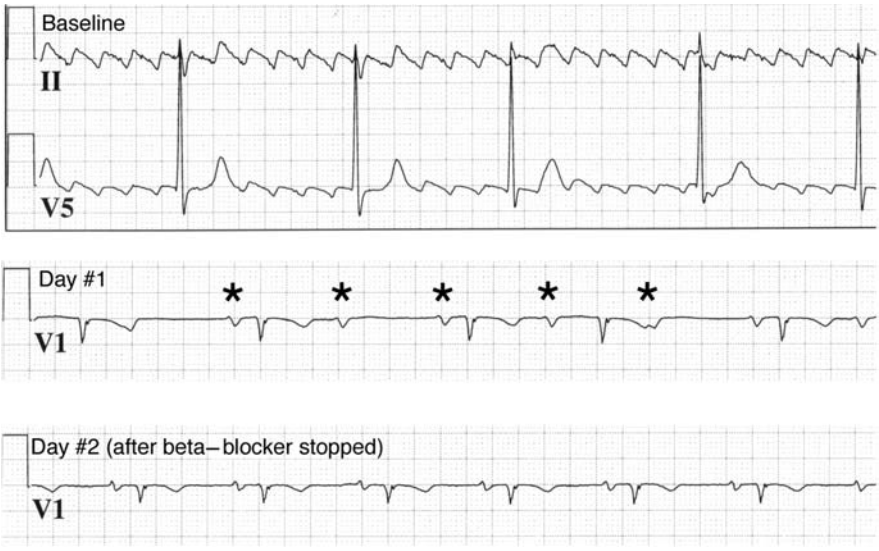


Figure 11: *Top*: Tracing of patient in typical atrial flutter. Since only one QRS complex is present for every 5–7 flutter waves, AV conduction is impaired. After conversion to sinus rhythm, the patient has 2° AV block Type I (*middle row*). When the beta blocker is stopped, 2° AV block resolves and the patient is left with borderline 1° AV block (PR interval 0.22 s).

AV junctional tachycardia

Tachycardias that arise from junctional tissue can be due either to reentry or increased automaticity.

1. AV node reentry

In AV node reentry, a reentrant circuit is formed in the AV node and atrial tissue around the AV node. The AV node can have several inputs that form the parallel pathways required for the development of reentry. In this case there is typically a "fast pathway" and a "slow pathway" into the AV node. During sinus rhythm, the atrial activation travels down both the fast and slow pathways (Figure 12). A premature beat can block in the fast pathway and travel to the AV node solely by the slow pathway. If the fast pathway has recovered, the electrical activation from the premature beat can travel backwards into the fast pathway and initiate a reentrant circuit. Thus for AV nodal reentry to occur, there have to be two electrically sep-

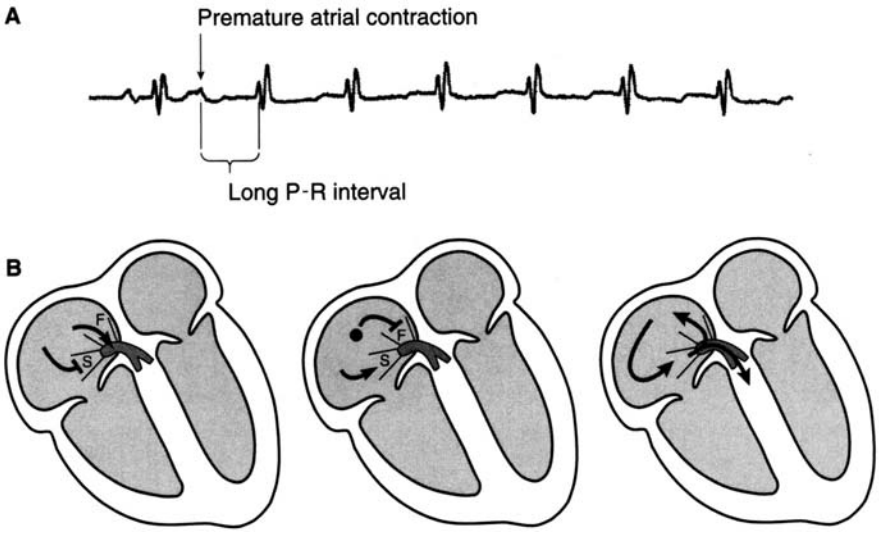


Figure 12: **A**. ECG showing initiation of AV node reentrant tachycardia with a premature atrial contraction. **B**. Schematic showing AV node reentry. In AV node reentry there is a slow and fast "input" into the AV node, so that parallel slow pathways into the AV node are present. At baseline, activation enters both the fast (F) pathway and slow pathway. When the impulse through the slow pathway meets the common endpoint the AV nodal tissue is refractory and depolarization is blocked. However, if a premature atrial complex blocks in the fast pathway and conducts solely down the slow pathway, the depolarization can enter the fast pathway in the retrograde direction and a reentrant arrhythmia can be initiated (reprinted with permission from Kusumoto FM, *Cardiovascular Pathophysiology*, Hayes Barton Press, Raleigh, NC, 1999).

arate pathways with different electrophysiological properties and a perfectly timed premature beat to initiate the fast heart rate. This is why AV node reentry is almost always paroxysmal.

On ECG the P waves are often not easily seen, since the atria and ventricles are activated almost simultaneously. It is often helpful to compare the QRS complex in sinus rhythm to the QRS complex during tachycardia to evaluate for any new deflections that represent atrial activity (P waves). Since the atria are being activated from the AV node, the P waves are usually inverted in the inferior leads and positive in lead V_1. The finding of a small terminal r' wave in lead V_1 can be an important clue for identifying supraventricular tachycardia caused by AV node reentry.

2. Automatic junctional tachycardia

In automatic junctional tachycardia, as the name implies, an automatic site near or within the AV node fires more rapidly than the sinus node and drives the heart. The location of P waves is variable; in some circumstances they can be obscured by the QRS complex if the atria are being retrogradely activated by the junction. In other cases sinus node activity can be observed. Figure 13 shows an ECG of a

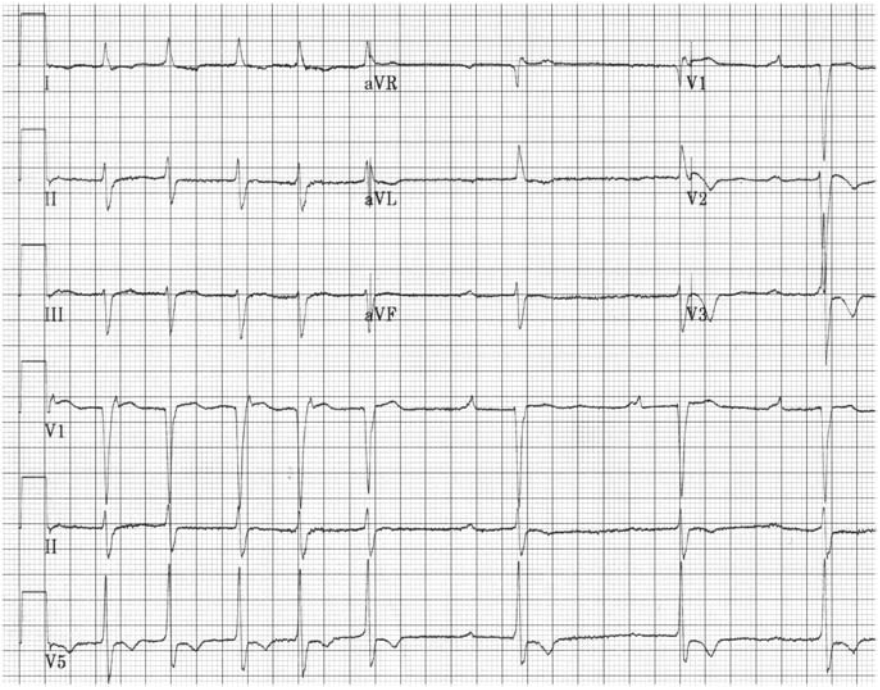


Figure 13: Junctional tachycardia due to automaticity for the first five beats that spontaneously terminates, and the patient has three beats of sinus rhythm with profound 1° AV block. Notice that retrograde P waves can be observed in three of the five junctional beats (best observed in V_1).

patient with an automatic junctional tachycardia after cardiothoracic surgery. On the left side of the strip the patient has a tachycardia at a rate of approximately 100 beats per minute. The tachycardia spontaneously terminates and the patient has sinus rhythm with very profound first degree AV block at baseline. Notice that the QRS during tachycardia and during sinus rhythm is the same. During the tachycardia some P waves are observed on the terminal portion of the QRS complex, and at other times they are not present.

Accessory pathway mediated tachycardia

The AV node usually is the single electrical connection between the atria and the ventricles. In approximately 1:1000 people an additional connection between the atria and ventricles exists. The presence of an accessory pathway increases the likelihood of reentry since there are now two pathways between the atria and ventricles. Accessory pathways can be associated with several different types of tachycardias and will be discussed more fully in Chapter 15. But as a brief introduction, the presence of an accessory pathway means that the ventricles are activated by two sites—the AV node and the accessory pathway. Since the accessory pathway conducts more rapidly than the AV node and inserts into ventricular tissue, the PR interval is short and the QRS complex is wide. The initial upstroke of the QRS complex is due only to accessory pathway conduction (remember that the AV node is still slowly conducting), and this slurred upstroke is called a delta wave. During sinus rhythm, ventricular activation is a "fusion beat" due to partial activation of the ventricles via the accessory pathway. The presence of an accessory pathway means that there are two parallel paths between the atria and the ventricles, and the patient has the perfect substrate for developing reentrant tachycardias. The most common form of tachycardia is called orthodromic atrioventricular reentry (Figure 14). In

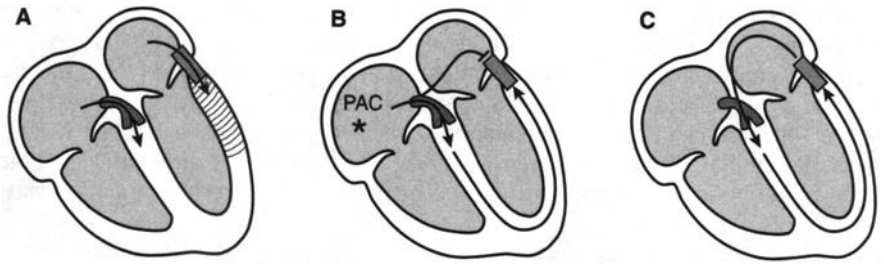


Figure 14: Initiation of orthodromic atrioventricular (AV) reentry in a patient with an accessory pathway. **A**. At baseline atrial depolarization conducts to the ventricles by the AV node and accessory pathway. **B**. A premature atrial complex blocks in the accessory pathway and conducts to the ventricles solely by the AV node. **C**. Retrograde (from ventricles to atria) activation of the accessory pathway can lead to the development of a reentrant circuit (reprinted with permission from Kusumoto FM, *Cardiovascular Pathophysiology*, Hayes Barton Press, Raleigh, NC, 1999).

this reentrant tachycardia the ventricles are activated via the AV node and the atria are activated via the accessory pathway. Since the ventricles are activated normally by the His-Purkinje tissue, a narrow complex tachycardia is observed on the ECG. For atrioventricular reentry, the reentrant circuit is quite large and requires the AV node, ventricular tissue, an accessory pathway, and atrial tissue. The tachycardia is often initiated by a premature atrial complex that blocks in the accessory pathway and conducts slowly to the ventricles via the AV node. The wave of depolarization activates the accessory pathway retrogradely and a reentrant circuit can develop. This type of tachycardia is called orthodromic atrioventricular tachycardia (refer to Figure 3) because the ventricles are activated normally (ortho: Greek for straight or correct) via the AV node.

Electrocardiographic diagnosis

The electrocardiogram is an important tool for the evaluation of supraventricular tachycardia. Important considerations include: Is the tachycardia regular or irregular? Where is the P wave located and what is its shape? Does the tachycardia require AV node activation to continue? Simple flow sheets for evaluation of irregular and regular supraventricular tachycardias are shown in Figures 15 and 16.

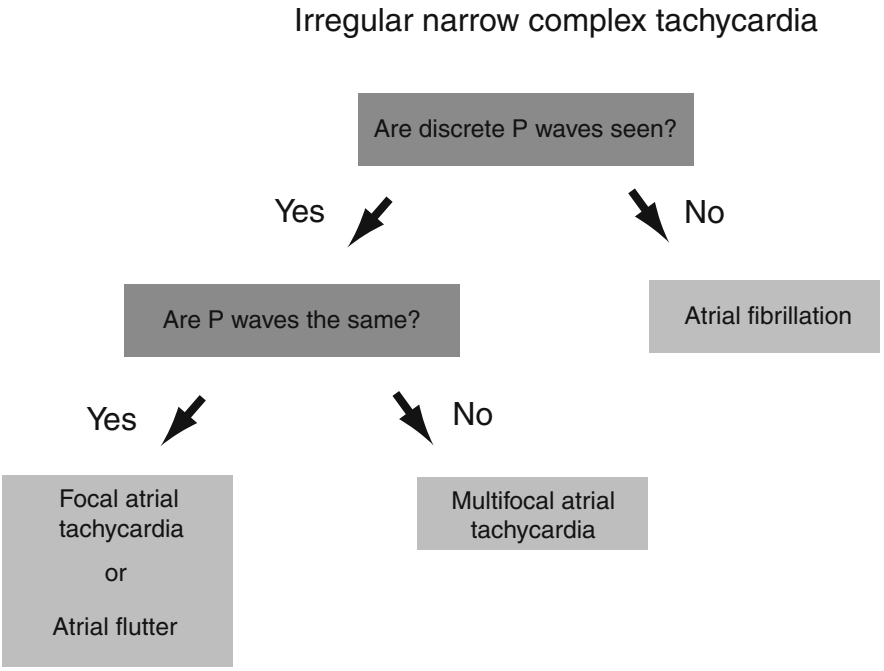


Figure 15: Flow diagram for evaluating irregular tachycardias.

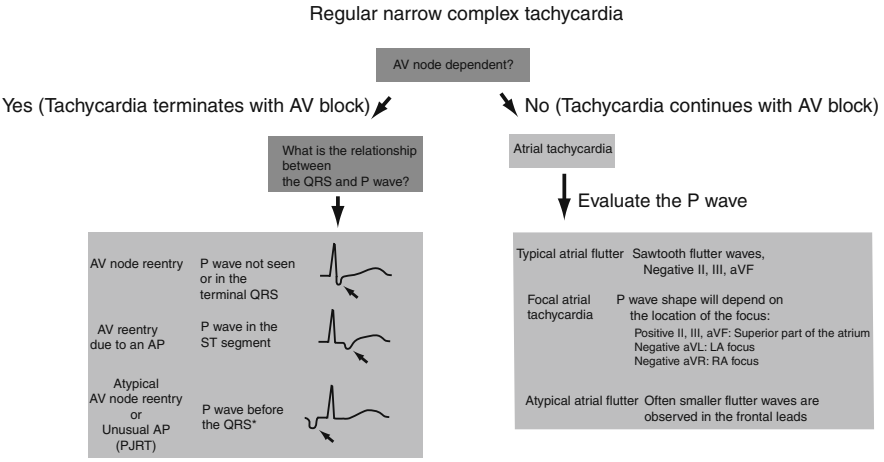


Figure 16: Flow diagram for evaluating regular tachycardias.

Regular vs. irregular

The most common cause for an irregular supraventricular tachycardia is atrial fibrillation. Chaotic atrial activity leads to fast and variable conduction through the AV node. A less common cause for an irregular tachycardia is multifocal

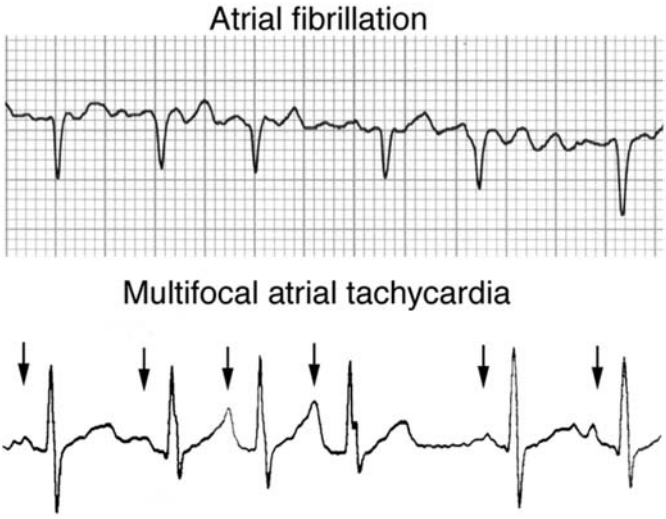


Figure 17: ECG examples of irregular tachycardias. In atrial fibrillation continuous fibrillatory activity will be observed, while in multifocal atrial tachycardia discrete P waves (arrows) with intervening isoelectric periods will be observed (reprinted with permission from: Nicoll D, McPhee S, Pignone M, Chuanyi ML (Eds.). *Pocket Guide to Diagnostic Tests, Fifth Edition* McGraw Hill, 2008).

atrial tachycardia. These two possibilities can usually be distinguished by the ECG (Figure 17). In multifocal atrial tachycardia discrete P waves of different shapes will be observed with intervening isoelectric periods where there is no atrial activation. In contrast, in atrial fibrillation continuous atrial activity will lead to a chaotic appearing baseline with no isoelectric periods. Atrial tachycardias with variable AV nodal block can also lead to irregular rapid heart rates, but since the atrial rate is regular some regular ventricular activity is often observed.

P wave location and morphology

The location of the P wave relative to the QRS complex will provide mechanistic clues for the cause of supraventricular tachycardia. If the tachycardia is arising from the AV junction area, simultaneous activation of the atria and ventricles will lead to the P wave being "hidden" by the QRS complex. In addition, tachycardias arising from the AV junction area will frequently have a small r' noted in V_1 that is due to retrograde atrial activation (Figure 18). In patients with accessory pathway mediated tachycardia the atria are sequentially activated after the ventricles. Since accessory pathway conduction is generally more rapid than conduction via the AV node, the P wave is seen in the ST segment (Figure 19). Many patients with accessory pathways will have abnormal QRS complexes at baseline due to accessory pathway activation of the ventricles. The accessory pathway is a "two-way street" that can conduct in both directions. Interestingly, some patients have accessory pathways that can only conduct backwards, or retrogradely. These patients have a normal QRS complex at baseline (Figure 20). The accessory pathway in this case is a "one-way street." The reason for this apparent paradox appears to be a "source-sink" mismatch (Figure 21). A thin accessory pathway cannot develop enough current to activate ventricular tissue, but can activate atrial tissue. Since preexcitation is not observed at baseline, these patients are said to have "concealed" accessory pathways. Since the P wave is closer to the preceding QRS complex compared to the subsequent QRS for both of these tachycardias, these tachycardias are generally called "short R-P" tachycardias (Figure 22). Oftentimes the ECG can help distinguish between AV node reentry and orthodromic AV reentry by the location of the P wave. In AV node reentry, the P wave is often not observed or is in the terminal portion of the QRS, because the atria and ventricles are activated simultaneously; while sequential activation of the ventricles and atria in orthodromic AV reentry leads to a P wave in the ST segment. A third, less likely cause for a short R-P tachycardia is the presence of an atrial tachycardia with associated first degree AV block.

Short R-P tachycardia

1. *AV node reentry: Most common particularly in women.*
2. *AV reentry: More common in children and adolescents.*
3. *Atrial tachycardia with first degree AV block: The least likely possibility.*

In "long R-P" tachycardias, the P wave is seen closer to the subsequent QRS rather than the preceding QRS complex (Figure 23). In general, long-RP tachycardias are due to focal atrial tachycardias (Figures 4 and 5), although some unusual

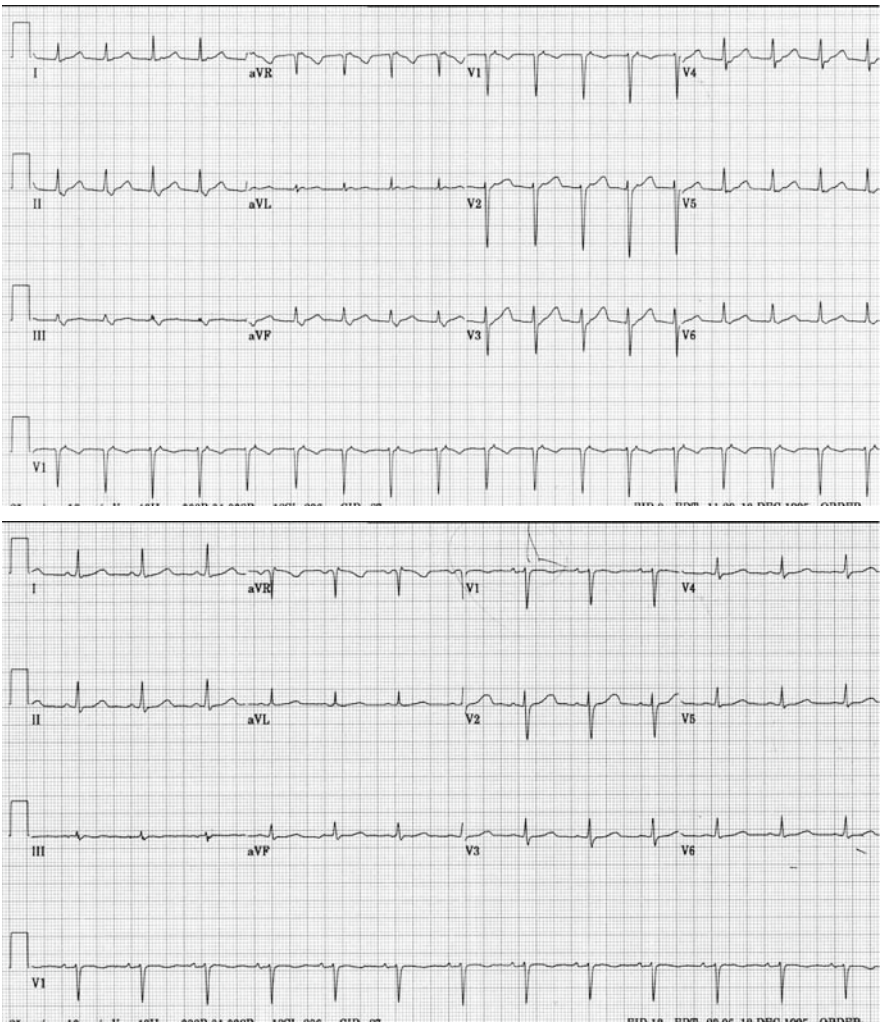


Figure 18: *Top*: ECG from a patient with AV node reentry. In AV node reentry the P wave is usually obscured but can sometimes be seen as a negative deflection on the terminal portion of the QRS in the inferior leads or as a small positive deflection observed in lead V_1. *Bottom*: ECG from the same patient after the tachycardia has resolved. Notice that the small r' during the tachycardia is not present at baseline. This suggests that the r' represents atrial depolarization.

cases of AV node reentry and accessory pathway mediated tachycardias can lead to a long-RP tachycardia.

For example, if the accessory pathway is composed of slowly conducting tissue rather than normal atrial or ventricular tissue with rapid conduction, retrograde

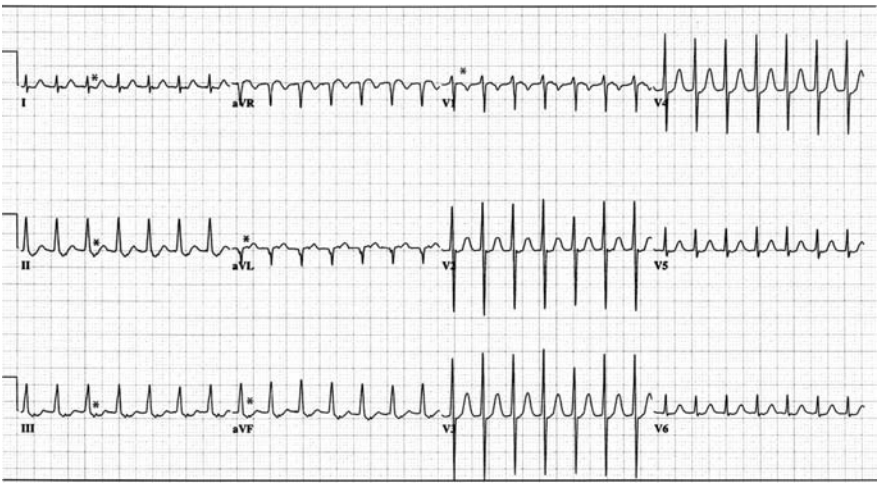


Figure 19: A patient with tachycardia due to an accessory pathway. Retrograde P waves (*) can be observed in 5 of the 6 frontal leads and V_1.

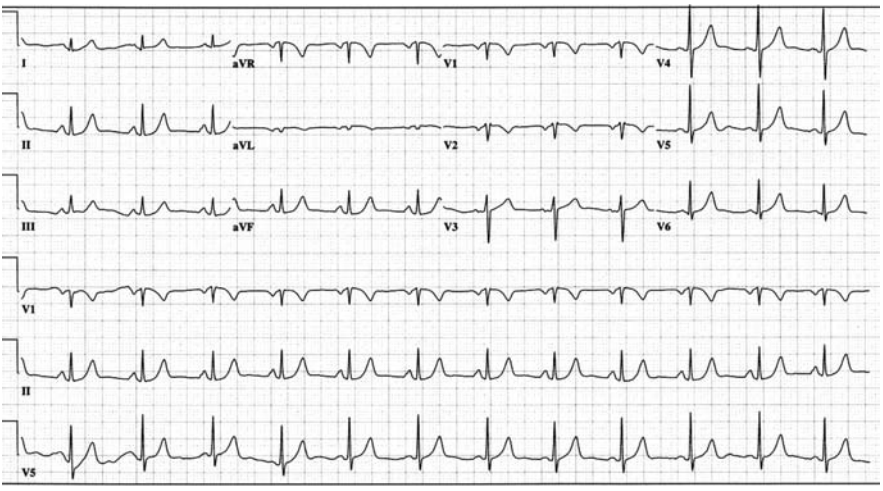


Figure 20: The baseline ECG for the same patient as Figure 19. Notice that the QRS complex is normal despite the presence of an accessory pathway.

conduction via the slowly conducting accessory pathway will cause a long-RP interval (Figure 24). In this case, since the AV node has more time to recover, the PR interval will be normal. Patients with slowly conducting accessory pathways often present with incessant tachycardias, since the reentrant circuit is composed of two slowly conducting pathways, and the tachycardias are easier to initiate and perpetuate because the slow conduction increases the likelihood that any tissue about

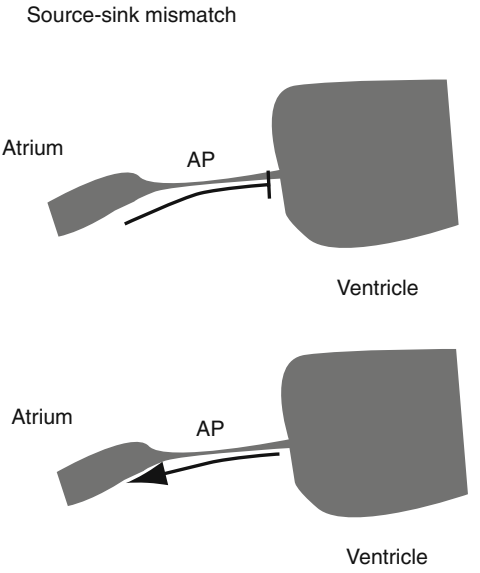


Figure 21: Diagram of proposed reason for "concealed" accessory pathways. The small accessory pathway (AP) cannot depolarize the large ventricular mass but can generate enough current to activate the thinner walled atria.

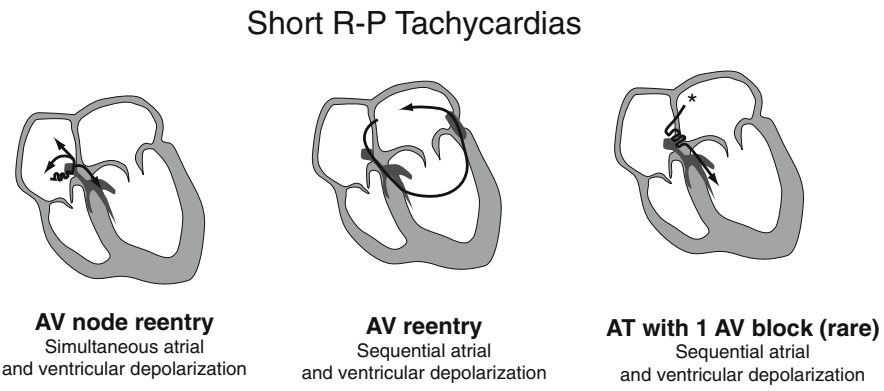


Figure 22: Differential diagnosis for short R-P tachycardias. In short R-P tachycardias the R-P interval is shorter than the PR interval. The most common cause for short R-P tachycardia is AV node reentry. Orthodromic atrioventricular (AV) reentry is a less common cause for short R-P tachycardia, but is a more frequent cause for tachycardia in children. These two possibilities can often be separated by the location of the P wave. A far less common cause of short R-P tachycardia in all age groups is a focal atrial tachycardia (AT) with associated first degree AV block.

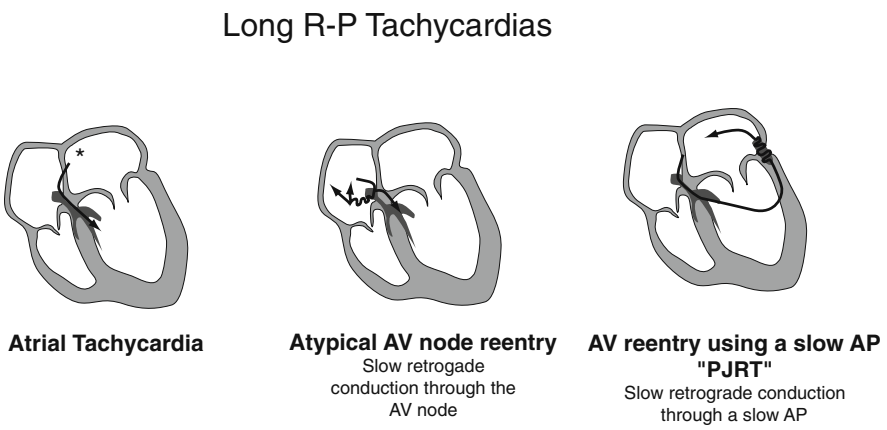


Figure 23: Differential diagnosis for long R-P tachycardias. In long R-P tachycardias the R-P interval is longer than the PR interval. The most common cause for long R-P tachycardia is atrial tachycardia. Less commonly, long R-P tachycardia can be due to AV node reentry where retrograde activation occurs via a slow pathway. Very rarely, a long R-P tachycardia can be due to a slowly conducting accessory pathway usually located near the septum in the region of the AV node. These patients often have incessant tachycardia because of the presence of two slowly conducting pathways. This arrhythmia is often called permanent junctional reciprocating tachycardia (PJRT) to emphasize the incessant nature of this arrhythmia.

to be activated will have fully recovered. This arrhythmia has traditionally been called permanent junctional reciprocating tachycardia (PJRT) because of the incessant nature of the arrhythmia and because the slowly conducting accessory pathway is usually located near the AV node.

Similarly, a long R-P tachycardia can be observed if the patient has AV node reentry in which the reentrant circuit is reversed. Retrograde activation of the atria occurs via the "slow" pathway, and anterograde activation through the AV node occurs via the "fast" pathway. This form of AV node reentry is usually called "atypical AV node reentry" because it is very infrequently observed.

Long R-P tachycardia

1. *Atrial tachycardia: Most common.*
2. *Atypical AV node reentry: Uncommon.*
3. *Slowly conducting accessory pathway (PJRT): Extremely rare.*

From the foregoing discussion it is obvious that identifying the P wave during tachycardia provides important clues. It is extremely helpful to compare the ECG during tachycardia with an ECG during sinus rhythm. This allows the clinician to carefully evaluate the ECG during tachycardia for any deflections in the QRS complex, ST segment, and T wave that represent atrial depolarization. Once the P wave is identified, the P wave shape can be evaluated. If the atria are being retrogradely

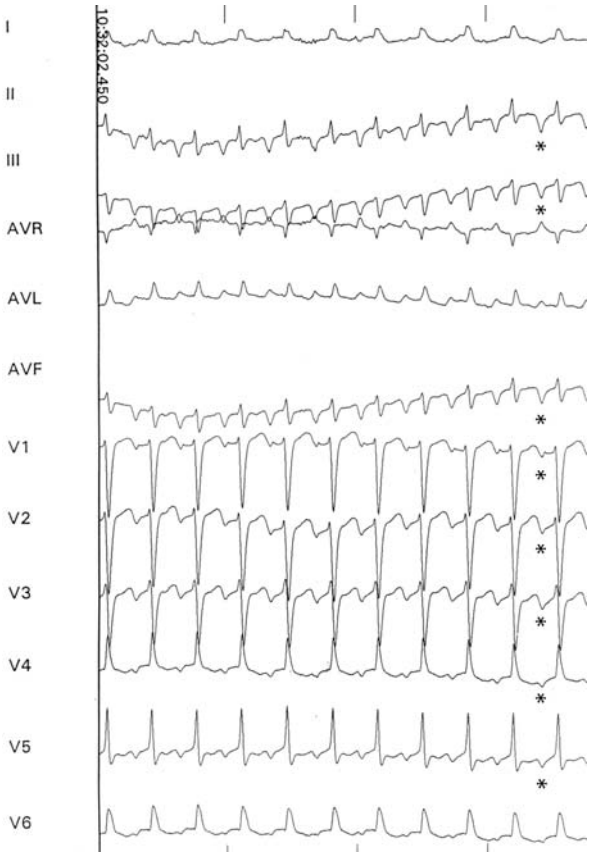


Figure 24: A patient with a slowly conducting accessory pathway. The retrograde P waves (*) are best seen as negative deflections in the inferior and precordial leads.

activated from the AV node or an accessory pathway, inverted P waves will sometimes be observed in the inferior leads. Conversely, if atrial activation appears to be "high-low" or positive in the inferior leads, it suggests that an atrial tachycardia is present. This is particularly useful in a long R-P tachycardia, where P waves are usually more easily identified. The presence of an upright P wave in the inferior leads essentially rules out atypical AV node reentry or PJRT.

AV node dependent vs. AV node independent

Probably the most important clue to determining the etiology of supraventricular tachycardia is determining whether the tachycardia requires AV node activation for its continuation (AV node dependent). AV node reentry and atrioventricular reentry are AV node dependent; if conduction through the AV node is blocked, even

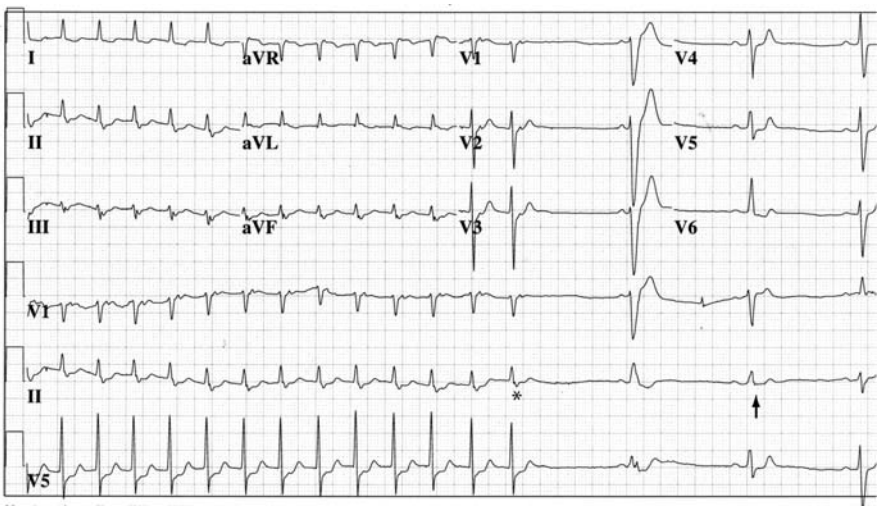


Figure 25: A patient with a tachycardia that terminates with adenosine. Thus the tachycardia would be considered AV node dependent. In this case the patient has AV node reentry. Comparing the QRS complexes during tachycardia and at baseline reveals a retrograde P wave in the terminal portion of the QRS ($*$) during tachycardia that is not present during sinus rhythm (*arrow*).

for one beat, the tachycardia will terminate, because the reentrant circuit is broken. Conversely, if the patient has an atrial tachycardia, activation of the ventricles via conduction through the AV node follows passively; so although the ventricular rate will slow if AV block occurs, the tachycardia in the atrial tissue will continue.

AV node independent: Atrial tachycardia.
AV node dependent: AV node reentry, AV reentry.

The easiest way to clinically attempt to achieve AV block is through a physical maneuver to increase vagal tone. The most commonly employed techniques are carotid sinus massage or a Valsalva maneuver. If the patient has intravenous access, adenosine can be given. By increasing potassium permeability, adenosine will cause transient block in the AV node (Figure 25).

Spontaneous termination of a supraventricular tachycardia can also provide information on whether a tachycardia is AV node independent or dependent. Examine Figure 26. The patient is in a long R-P tachycardia that spontaneously terminates while the ECG is being recorded (what luck!). The tachycardia "ends" on a P wave that can be most easily seen in lead V_1. This implies that the tachycardia is AV node dependent. Atrial tachycardia with 1:1 conduction over the AV node will generally not terminate spontaneously on a P wave. In atrial tachycardia, the AV node conduction follows passively. When the atrial tachycardia terminates, the last P wave should be followed by a QRS complex.

Diagnostic algorithms for evaluating supraventricular tachycardias are shown in Figures 15 and 16. In clinical practice, irregular supraventricular tachycardias are almost always due to some type of atrial tachycardia. If discrete atrial activity is not seen, the patient has atrial fibrillation. If discrete P waves of differing morphology are observed, the patient has multifocal atrial tachycardia. If a single P wave morphology is observed the patient has focal atrial tachycardia or atrial flutter. Since the irregular ventricular rate is due to variable conduction through the AV node, frequently the patient will have bursts of regular activity due to stable 2:1 activation through the AV node interspersed with longer intervals between the QRS complexes due to higher grade (3:1, 4:1, etc.) AV block and some very occasional shorter intervals (1:1 conduction).

Regular supraventricular tachycardias have multiple anatomic causes: atrial tachycardia, AV node reentry, and AV reentry using an accessory pathway (Figure 16). The most important procedure is determining whether the tachycardia is AV node dependent or AV node independent. If the tachycardia is AV node dependent, either AV node reentry or AV reentry using an accessory pathway is present. Identifying the location of the P waves during tachycardia is useful for distinguishing between these two possibilities. If the tachycardia continues in the presence of AV block, the patient has atrial tachycardia due to either a single focus firing rapidly or a stable reentrant circuit within the atria. It is extremely difficult to distinguish between these two choices by a 12-lead ECG, although the presence of typical flutter waves is helpful.

Case Study (continued): The patient was given 6 mg adenosine. Continuous ECG monitoring is shown in Figure 27. Notice that the tachycardia continues in the presence of prolonged high grade AV block, thus confirming that the patient has an atrial tachycardia. Examination of the P wave morphology shows upright P waves in the inferior leads. The upright P waves in the precordial leads suggests that the tachycardia is arising from the superior portion of the posterior left atrium.

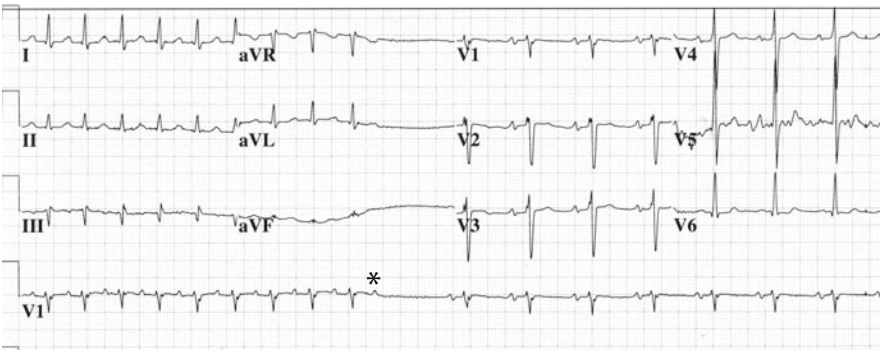


Figure 26: Spontaneous termination of a long R-P tachycardia. The tachycardia ends on a P wave, (*) ruling out atrial tachycardia. The patient has atypical AV node reentrant tachycardia, in which retrograde atrial activation occurred via the slow pathway, thus producing the long R-P interval.

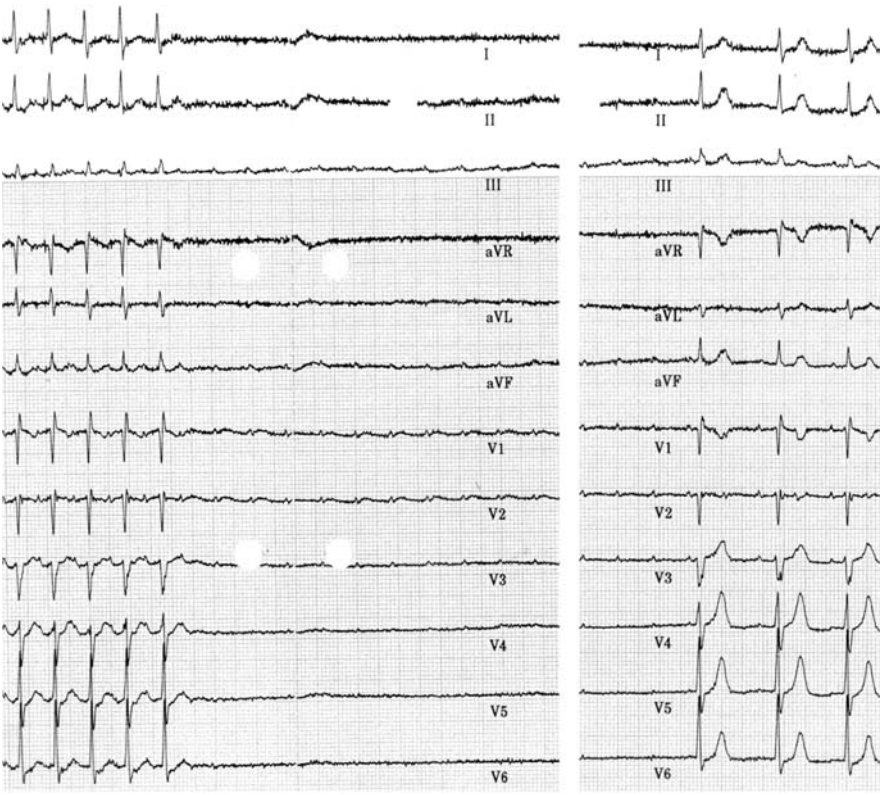


Figure 27: Ms. Saltsman's ECG with adenosine. Notice that the tachycardia (small P waves can be seen that do not conduct to the ventricles via the AV node) continues in the presence of AV block (AV node independent).

Key points

1. Tachycardias can be classified by mechanism, anatomy, or ECG appearance.
2. There are three types of tachycardia mechanisms: automaticity, triggered activity, and reentry.
3. Using an anatomic classification, narrow complex tachycardias can be due to tachycardia within atrial tissue, within junctional tissue, or utilizing an accessory pathway.
4. Irregular narrow complex tachycardias: atrial fibrillation, multifocal atrial tachycardia, atrial tachycardia with variable AV node conduction.
5. Regular narrow complex tachycardias: focal atrial tachycardia, atrial flutter, junctional tachycardias, and accessory pathway mediated tachycardias.
6. To distinguish between these possibilities on an ECG, it is important to determine the location and shape of the P wave and whether or not the tachycardia requires the AV node to continue.

Review questions

1. Generally, tachycardia associated with a narrow QRS complex can be due to all of the following except:

   A. Orthodromic reciprocating tachycardia.
   B. AV node reentrant tachycardia.
   C. Ectopic atrial tachycardia.
   D. Ventricular tachycardia.

2. In patients with atrial fibrillation, the calcium channel blocker diltiazem is often given. What ECG finding will most likely be observed?

   A. Abrupt termination.
   B. Development of a wide QRS due to aberrant conduction through the right or left bundles.
   C. Decreased heart rate.
   D. Decreased amplitude of fibrillatory waves.

3. A narrow complex tachycardia with more P waves than QRS complexes is observed. Which type of arrhythmia is excluded?

   A. Atrial tachycardia.
   B. Atrial flutter.
   C. AV node reentrant tachycardia.
   D. Orthodromic reciprocating tachycardia.

4. In Figure 28, the mechanism for the arrhythmia is most likely:

   A. Abnormal automaticity.
   B. Reentry.
   C. Triggered activity.
   D. Normal increase in automaticity.

5. The arrhythmia in Figure 28 is most likely:

   A. Ventricular tachycardia.
   B. Atrial tachycardia.
   C. Orthodromic AV reentry.
   D. Atrial fibrillation.

Answers

1. D. Ventricular tachycardia would be associated with a wide QRS since the ventricles are not activated normally.

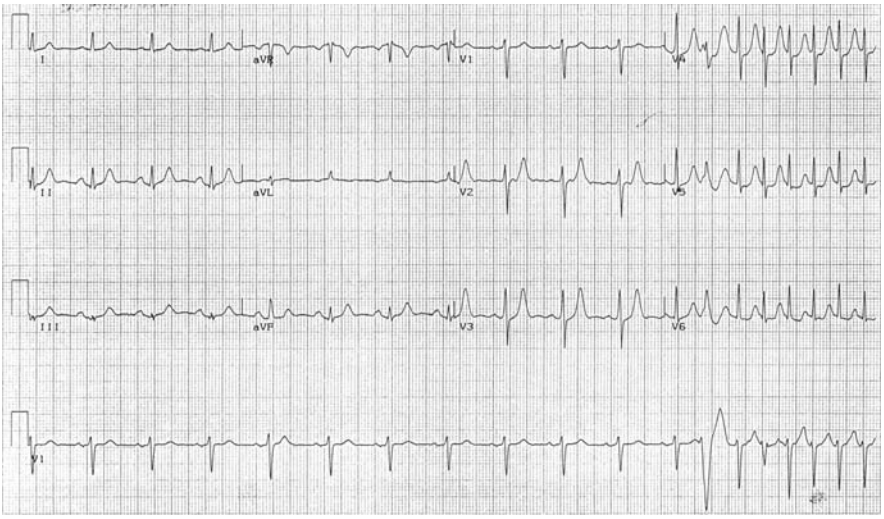


Figure 28: ECG for Questions 4 and 5.

2. C. Diltiazem is a calcium channel blocker whose main electrophysiologic effect is to slow AV node conduction. In a patient with atrial fibrillation this would tend to slow the ventricular rate. Calcium channel blockers are frequently used this way to improve the hemodynamic status by providing a slower and more physiologic heart rate.
3. D. If more atrial activity then ventricular activity is observed, the patient most likely will have an atrial tachycardia. Orthodromic AV reentrant tachycardia is ruled out since in this arrhythmia, the AV node, ventricles, accessory pathway, and atria are activated sequentially in a one to one fashion: "For every QRS there will be a P wave." In some rare cases of AV node reentry, the His bundle will have 2:1 block so there will be more P waves than QRS complexes.
4. B. The patient has a supraventricular tachycardia start abruptly with a premature ventricular complex. This type of initiation makes reentry the most likely underlying mechanism.
5. C. The patient probably has orthodromic AV reentry with deflections in the ST segment that represent retrograde P waves. Atrial tachycardia is very unlikely since the tachycardia started with a premature ventricular contraction. The same logic applies for atrial fibrillation and, in addition, the tachycardia is regular. Ventricular tachycardia would have been associated with a wide QRS complex. Not given as a choice is AV node reentry, which would be the second most likely cause for this arrhythmia, given the way it is initiated.

Chapter 12
Wide complex tachycardia

Case Study: Mr. Jack Ramsey is a 68-year-old retired police officer who sustained an anterior wall myocardial infarction one year ago. He has done well, but earlier this morning he developed a sudden onset of dizziness. There was no accompanying chest pain or shortness of breath. His ECG is shown in Figure 1.

As described in the last chapter, there are four anatomic causes for rapid heart rates (atrial tachycardia, junctional tachycardia, ventricular tachycardia, and accessory pathway mediated tachycardia). If a tachycardia is associated with a wide QRS complex, ventricular activation is abnormal. As described in the next section, all four of the anatomic types of tachycardia can present as wide complex tachycardia. However from a clinical standpoint, it is imperative to distinguish ventricular tachycardia from the other forms of wide complex tachycardia, since ventricular tachycardia can be unstable and cause hemodynamic compromise and often portends a worse prognosis.

The ECG is used as a tool to help separate ventricular tachycardia from supraventricular tachycardias with abnormal or aberrant ventricular activation.

Pathophysiology

Abnormal ventricular activation can occur due to an arrhythmogenic site within ventricular tissue or any supraventricular tachycardia associated with aberrant ventricular conduction (Figure 2). Aberrant ventricular activation from a supraventricular rhythm can occur due to block in the right or left bundle or via an accessory pathway.

Ventricular tachycardias are more common in the presence of structural heart disease. The most common cause for ventricular tachycardia is a prior myocardial infarction. Within the myocardial infarction zone, myocyte damage and subsequent fibrosis does not form a solid scar but instead forms patches separated by areas of relatively normal tissue. Although the myocytes are intact, conduction through these narrow and twisting channels can be slow and can provide the substrate for reentry (Figure 3). In Figure 3, a premature ventricular contraction depolarizes the myocardium, but a relatively slow activation wave travels through the scar (dotted

F. Kusumoto, *ECG Interpretation: From Pathophysiology to Clinical Application*,
DOI 10.1007/978-0-387-88880-4_12,

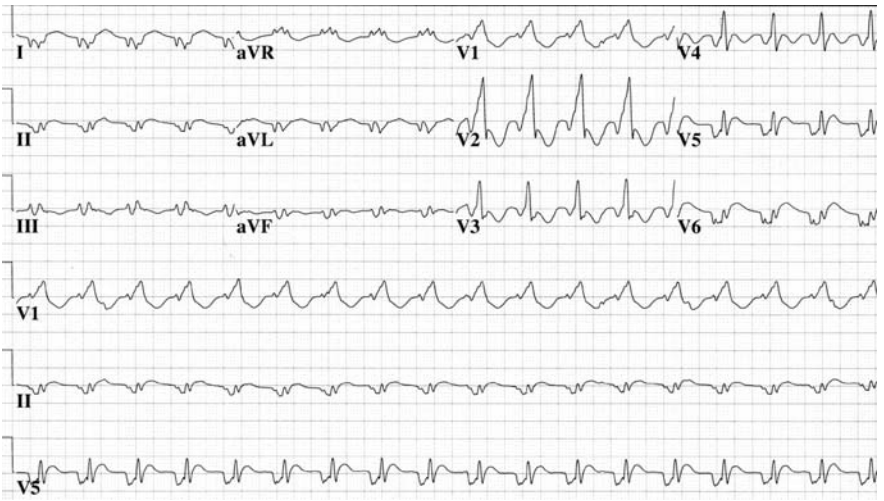


Figure 1: Mr. Ramsey's ECG.

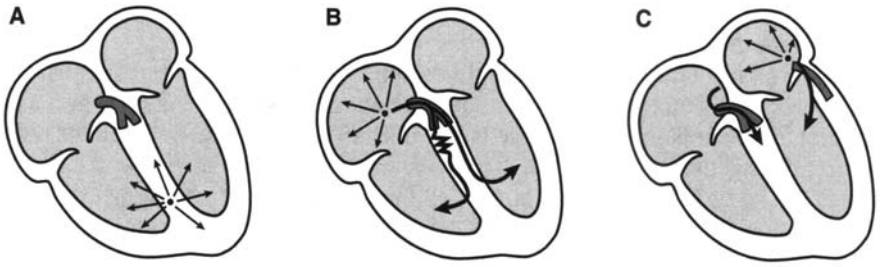


Figure 2: Differential diagnosis of wide complex tachycardia: **A**. ventricular tachycardia in which a reentrant or automatic site within the ventricles fires rapidly. **B**. Any type of supraventricular tachycardia associated with bundle branch block. Focal atrial tachycardia with right bundle branch delay is shown. **C**. Atrial tachycardia with activation of the ventricles in part over an accessory pathway. (reprinted with permission from Kusumoto FM, *Cardiovascular Pathophysiology*, Hayes Barton Press, Raleigh, NC, 1999).

line). If the slowly activating wave emerges from the scar and finds ventricular tissue that is no longer refractory, a reentrant circuit can be initiated. Sometimes the reentrant circuit is able to self-perpetuate for three or four beats, perhaps because of refractoriness within the scar, or perhaps the wave of depolarization "short-circuits" itself by traveling through another channel. In this case the ventricular tachycardia is called unsustained. However, in some cases the reentrant circuit continues and results in sustained ventricular tachycardia.

Although reentry is the most common mechanism for ventricular tachycardia, the other cellular mechanisms for tachycardia can be present in ventricular tissue

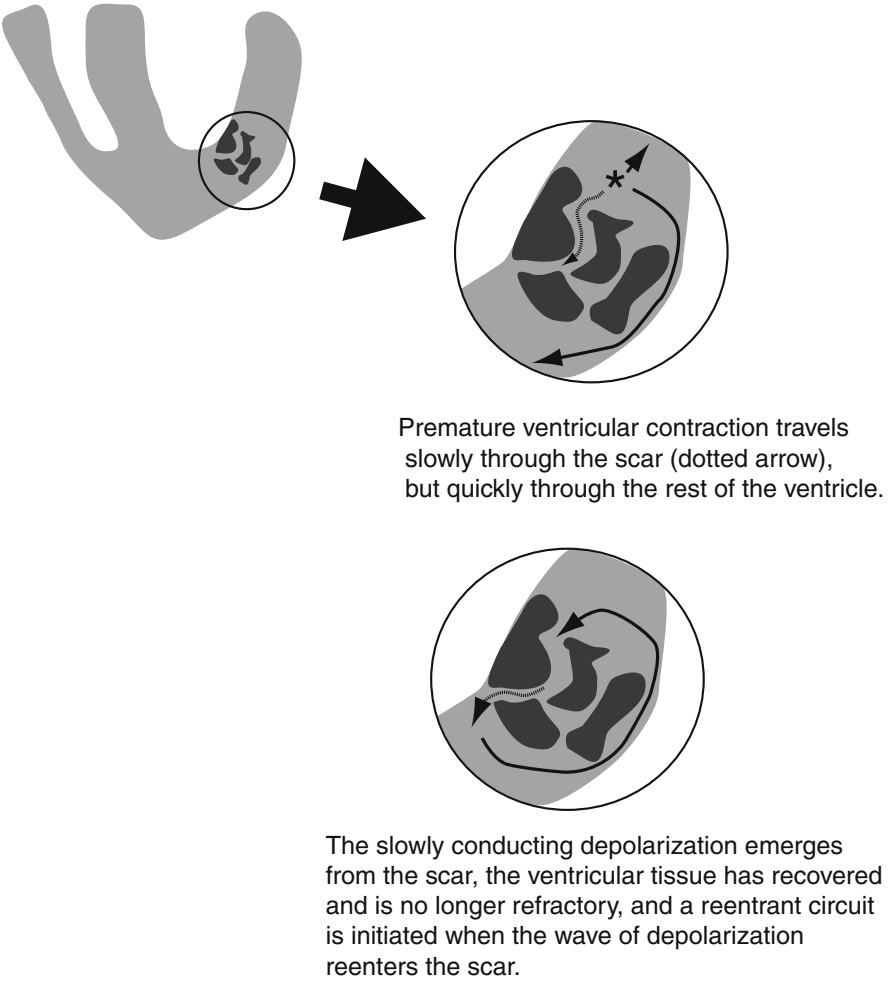


Figure 3: Development of reentry in a patient that has had a myocardial infarction. The myocardial infarction results in patchy scar formation at the site of the infarction. The patchy scar forms the substrate for ventricular tachycardia that is initiated with a premature ventricular contraction.

and cause ventricular tachycardia. A single site within the ventricles can develop abnormal automaticity and if repetitive firing develops, ventricular tachycardia can ensue. Triggered activity is the cause of torsade de pointes, the ventricular arrhythmia associated with QT interval prolongation that was introduced in Chapter 6.

Pathophysiologic causes for ventricular tachycardia:

1. *Reentry due to myocardial scar*
2. *Increased automaticity*
3. *Triggered arrhythmias*

ECG analysis

Although the ECG is the most important tool for evaluating the patient with wide complex tachycardia and for differentiating between ventricular tachycardia and supraventricular tachycardia with aberrant conduction, it is important to acknowledge its limitations. Experts in electrocardiography will not be able to characterize approximately 10% of wide complex tachycardias. A guide for the evaluation of a patient with wide complex tachycardia is provided in Table 1.

"You are not really having fun until your heart rate is twice the normal rate."
—Keith Oken (medical resident, now Assistant Professor of Medicine, Mayo Clinic)

Clinical clues

Although this is a book on ECG analysis, it is important to review the relative importance of clinical clues in the patient with wide complex tachycardia. In one study, a history of myocardial infarction, congestive heart failure, and recent angina pectoris all had positive predictive values $> 95\%$ for ventricular tachycardia. In another study of conscious patients with wide complex tachycardia, if the patients answered "yes" to two questions ("Have you had a heart attack in the past?" and "Did symptoms start after your heart attack?"), patients had ventricular tachycardia in 28 of 29 cases. Both of these studies emphasize that when a patient has some form of structural heart disease, and therefore has the substrate for the development of reentry, ventricular tachycardia becomes a much more likely diagnosis. Even

Table 1: Evaluation of the patient with regular wide QRS tachycardia

Clinical situation/ECG finding	ECG criteria for VT
Does the patient have a history of heart disease?	If "Yes" VT is more likely
Is AV dissociation present?	Presence of capture or fusion beats "unexpected" deflections
QRS morphology	
• Frontal axis?	"Northwest axis": VT
• QRS width:	The wider the QRS, the more likely the patient has structural heart diseases and the more likely the patient has ventricular tachycardia. > 0.16 seconds: more likely VT
• Negative or positive concordance?	If either present: VT
• Negative QRS in V_1: LBBB morphology	Figure 15
• Positive QRS in V_1: RBBB morphology	Figure 15

in studies evaluating all patients that present to the emergency room with wide complex tachycardia, ventricular tachycardia will be the cause in 80–90% of cases. Since ventricular tachycardia potentially has such dire clinical consequences, when in doubt, ventricular tachycardia should always be the "fallback" diagnosis.

As a final point, the clinician should remember that hemodynamic stability is not useful for differentiating ventricular tachycardia from supraventricular tachycardia with aberrancy. Although ventricular tachycardia is often associated with hemodynamic compromise, in patients with wide complex tachycardia and "stable" hemodynamics (mean systolic blood pressure 110 mm Hg), ventricular tachycardia was still present in almost all of the cases.

Ventricular tachycardia should always be considered as the cause of a wide complex tachycardia "unless proven otherwise."

Atrial-ventricular relationship

One of the most important ECG clues for determining the etiology of wide complex tachycardia is to evaluate the relationship between atrial and ventricular activity. If the arrhythmogenic source is within ventricular tissue, the atrial and ventricular relationship may not be relevant (Figure 4). Conversely, if the patient

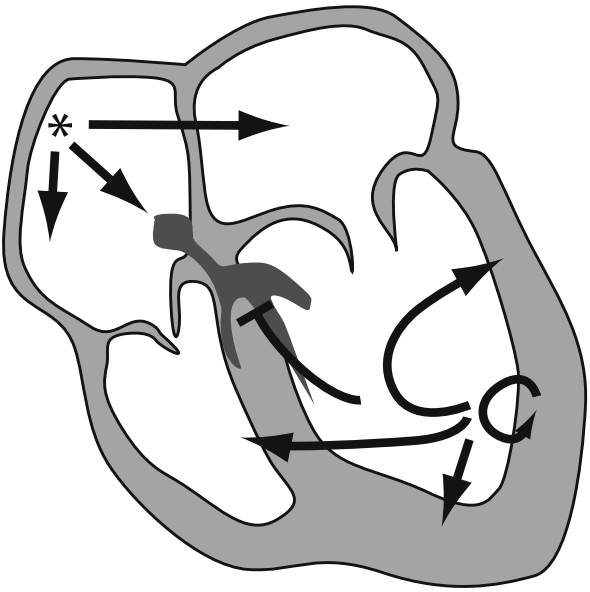


Figure 4: Schematic showing AV dissociation in ventricular tachycardia. A reentrant circuit develops at the site of the scar, and leads to repetitive abnormal depolarization of the ventricles. The sinus node is unaffected and continues to activate the atria (it does not "care" about ventricular activation).

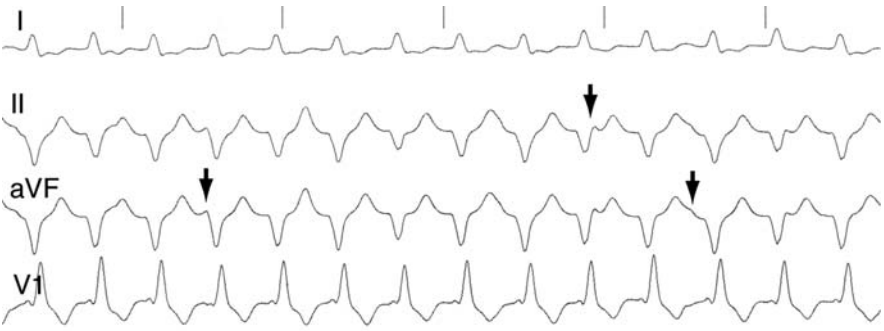


Figure 5: ECG showing AV dissociation. Notice the "unexpected" deflections that must represent atrial activity and AV dissociation (*arrows*). Evidence for atrial activity can be subtle; it is useful to scan the entire ECG for evidence of AV dissociation.

has supraventricular tachycardia with aberrant conduction, rapid activity (whether automatic or reentry) from atrial tissue or junctional tissue will be "driving" ventricular activation, and atrial activity will usually be related to ventricular activity. Unrelated or dissociated atrial activity can be observed as "unexpected" deflections in the ECG and is usually termed "AV dissociation" (Figure 5). On rare occasions the atrial activation will bring about partial or complete activation of ventricular tissue, leading to "fusion complexes" in which the QRS is a combination of ventricular activation from the His-Purkinje tissue and the ventricular focus and capture beats in which a QRS complex from His-Purkinje tissue occurs. In Figure 6 a fusion beat is shown. The patient has a wide complex tachycardia with a single beat with a different morphology (left bundle branch block). This beat is due to a perfectly timed sinus beat that is able to "sneak in" by activating a portion of the ventricles via the AV node and His bundle. Notice that partial ventricular capture is present because the QRS is different in V_1 but similar in V_2 and V_3. One of the nice features of modern ECG machines is that the recordings are acquired simultaneously so that the clinician can often evaluate the QRS morphology in several leads. This single beat does not affect the reentrant circuit, so the QRS morphology due to ventricular tachycardia returns immediately after the capture beat. The presence of the capture beat, and by definition AV dissociation, confirms the diagnosis of ventricular tachycardia for this rhythm. Fusion and capture beats are more commonly seen if the ventricular tachycardia rate is slower, since there is a higher likelihood that activation via the AV node and the His Bundle will occur.

It is much easier to look for AV dissociation rather than AV association. There is a tendency to look for P waves in the "expected" place, which leads to difficulty deciding whether a deflection is a T wave or a P wave. It is much more productive to look at the ECG with a "wide-angle" lens. Instead of looking for recurring deflections that "might" be atrial activity but could also be T waves, the clinician should evaluate for any unexpected deflections that represent A-V dissociation (reexamine Figure 4). A-V dissociation is most easily seen in the inferior leads (II, III, and aVF)

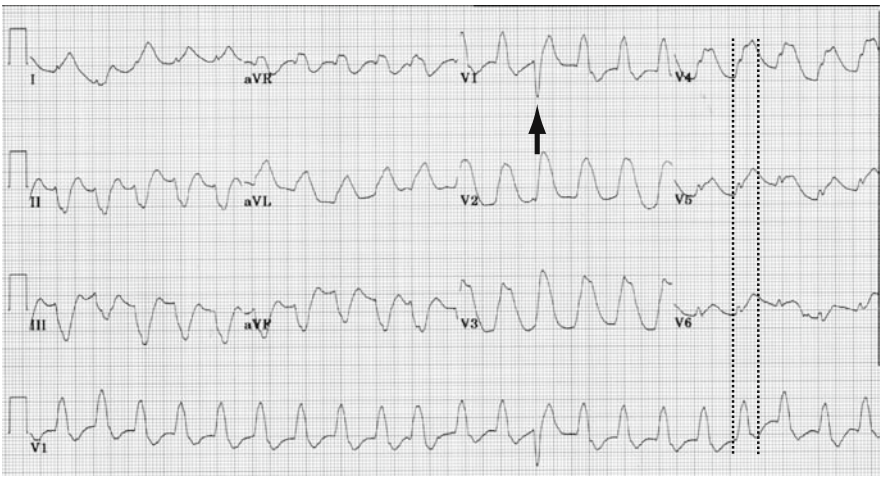


Figure 6: ECG showing fusion. A relatively narrow QRS complex is noted in lead V_1 (*arrow*). This represents partial ventricular activation from a preceding perfectly timed sinus beat activating a portion of the ventricle via the His-Purkinje system. Notice that this would be classified as a "fusion" beat since ventricular activation in V_2 and V_3 are unchanged, but if only lead V_1 were being recorded it would be considered a "capture" beat. The distinction between a capture beat and a fusion beat has no clinical utility, since the presence of either one confirms the presence of AV dissociation, and makes the diagnosis of ventricular tachycardia. It is often difficult to determine where a QRS begins and ends in some leads. Since leads are acquired simultaneously, it is useful to draw a vertical line from a lead where the beginning and end of the QRS are easier to determine and transpose this information to more difficult to interpret leads.

and V_1. Although very specific for ventricular tachycardia, unfortunately, AV dissociation and its manifestations (fusion beats and capture beats) can only be identified in approximately 25–40% of cases.

Case Study (continued): Examine the ECG from Mr. Ramsey. Are there any unexpected deflections? Remember that the inferior leads and V_1 are usually the most fruitful leads for finding "wayward" P waves.

Initiation and termination

The initiation and termination of a tachycardia can provide important clues to the diagnosis. Initiation of the tachycardia with a premature atrial beat suggests supraventricular tachycardia. Initiation of the tachycardia with a ventricular premature beat makes ventricular tachycardia more likely, but some forms of supraventricular tachycardia (AV node reentrant tachycardia and orthodromic AV reentrant

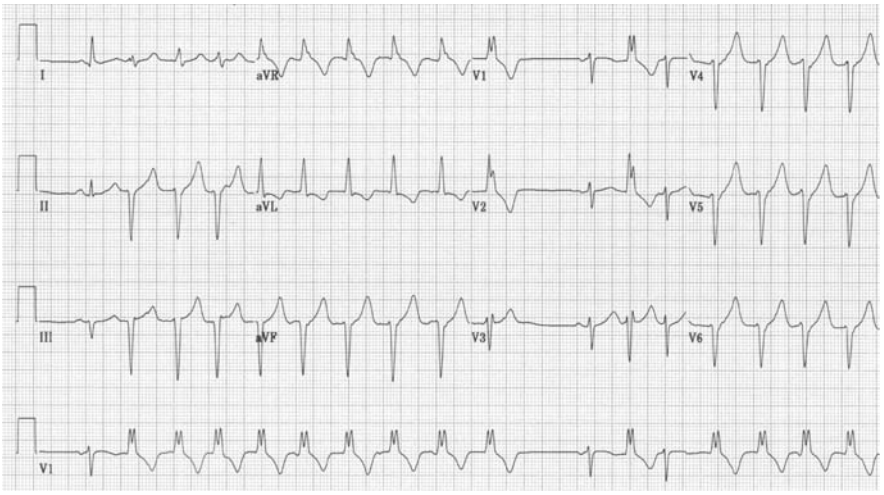


Figure 7: An ECG from a woman with salvos of wide complex tachycardias. Notice that the wide QRS tachycardia starts without a preceding premature atrial complex, making ventricular tachycardia the most likely possibility. In addition, the mechanism is probably automaticity, since the first and subsequent beats have exactly the same morphology.

tachycardia) can be initiated with a premature ventricular tachycardia. Figure 7 shows initiation of a wide complex rhythm.

Rate, axis, and QRS width

Unfortunately, rate is not a useful discriminator for differentiating tachycardia. Both ventricular tachycardia and supraventricular tachycardia with aberrancy can present with almost any heart rate.

The frontal plane axis can be used for distinguishing ventricular tachycardia from supraventricular tachycardia with aberrancy. If the frontal QRS is in the right upward quadrant (also referred to colloquially as the “northwest” axis), the arrhythmia is more likely to be ventricular tachycardia. This makes anatomic sense, since this axis would imply initial ventricular activation from near the apex of the left ventricle. It would be hard to imagine any type of aberrant activation of the bundles leading to initial activation in this area. Unfortunately, a frontal plane axis in any other quadrant is not helpful for making the correct diagnosis, and even the presence of a “northwest” axis is not absolute (Figure 8). It is worth emphasizing that most of the ECG criteria that use QRS morphology provide supporting evidence rather than absolute proof that ventricular tachycardia is present.

The QRS duration can provide some clues on whether ventricular tachycardia or supraventricular tachycardia with aberrancy is present. In general the QRS

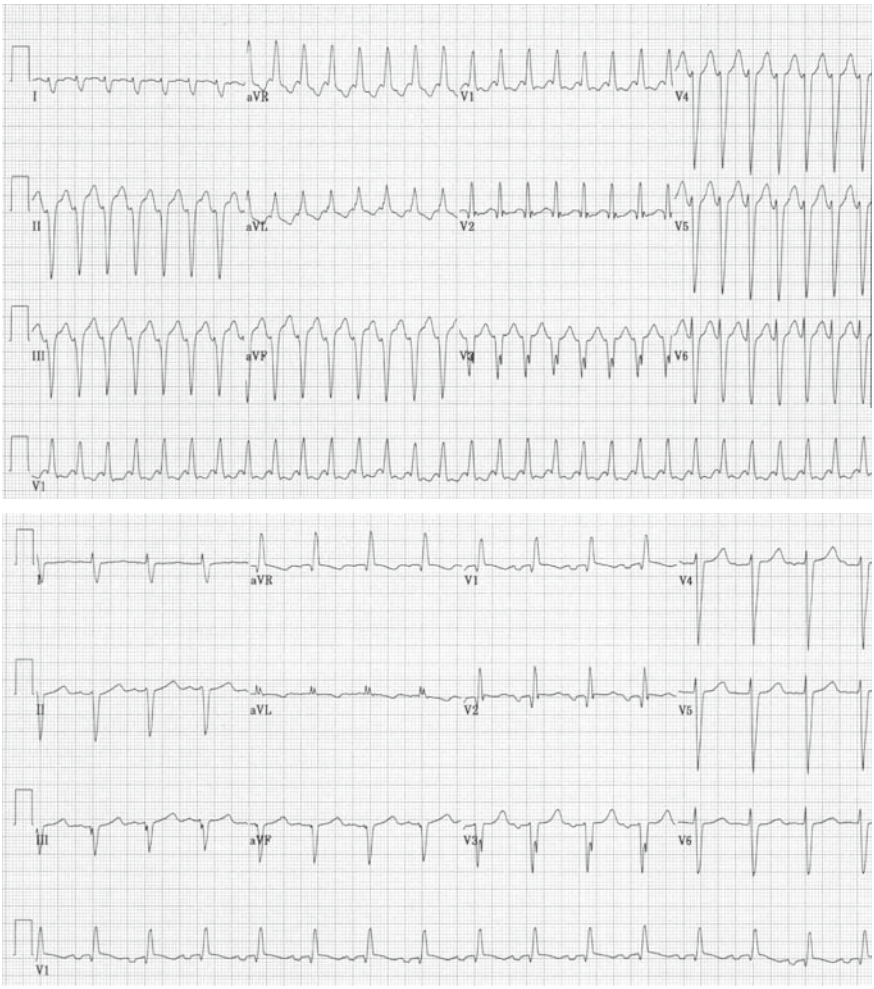


Figure 8: *Top*. ECG from a patient that presents with a wide QRS tachycardia. The largest positive QRS complex in the frontal leads is aVR and the QRS in lead I is predominantly negative, which yields a calculated axis of –95° to –105°. The patient was later found to have AV node reentry with right bundle branch block. *Bottom*: ECG after termination of the tachycardia. Notice that in sinus rhythm the patient has a baseline right bundle branch block with significant left axis deviation. This example illustrates the usefulness of having a baseline ECG.

complex is narrower with aberrant conduction when compared to ventricular tachycardia, since it is more likely that a portion of the ventricles are being activated with the His-Purkinje system with aberrant conduction. In addition, the wider the QRS complex, the more likely that structural heart disease is present and that the patient has ventricular tachycardia. A QRS width > 0.16 s has been traditionally

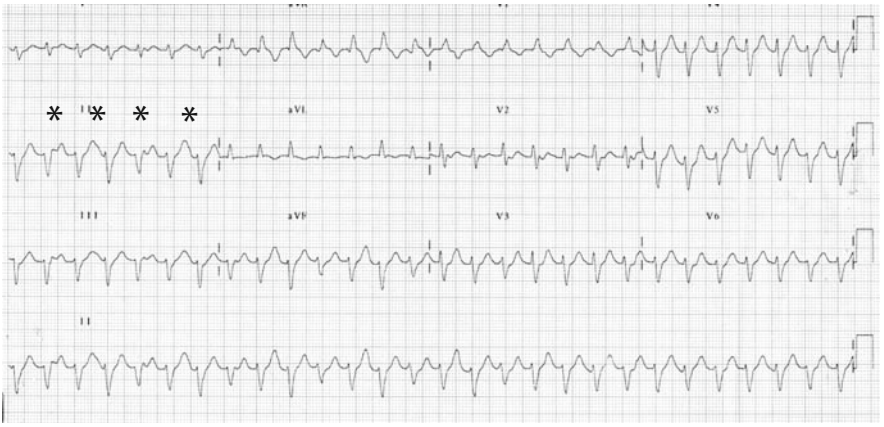


Figure 9: Patient with ventricular tachycardia. Although the QRS complex is relatively narrow (< 0.16 s), atrial activity (*) separate from ventricular activity can be observed as subtle and not so subtle changes in the ST segment and T wave.

used as the best cut-off for identifying ventricular tachycardia, but even this "best value" has limited sensitivity and specificity (60–80%). Ventricular tachycardia can be associated with a narrow QRS tachycardia if the reentrant circuit or region of abnormal automaticity is near His-Purkinje tissue. In practice, the author has found that evaluation of QRS width has limited clinical utility. An example of a patient with ventricular tachycardia with a relatively narrow QRS complex is shown in Figure 9. Although the QRS complex is less than 0.16 s (4 "little" boxes), notice that there is obvious AV dissociation.

Precordial QRS morphology

Precordial concordance

The ventricular activation pattern in the precordial leads should be evaluated. If all of the QRS complexes in the precordial leads are negative (negative concordance), the ventricles must be activated from an apical source. Since it is almost impossible for any type of aberrant conduction to initiate ventricular activation from the apex, the presence of negative concordance is an excellent sign for ventricular trachycardia. If all of the QRS complexes in the precordial lead are positive (positive concordance), ventricular activation is being initiated from a site at the most posterior portion of the left ventricle (Figure 10). Although it is possible that a left-sided accessory pathway can be associated with positive concordance (see Case 5 in Chapter 15), because accessory pathways are so uncommon the presence of positive concordance is an excellent clue that ventricular tachycardia is present.

Sometimes it is difficult in the precordial leads to decide where the QRS begins and ends, and thus whether a QRS is positive or negative in all of the precordial

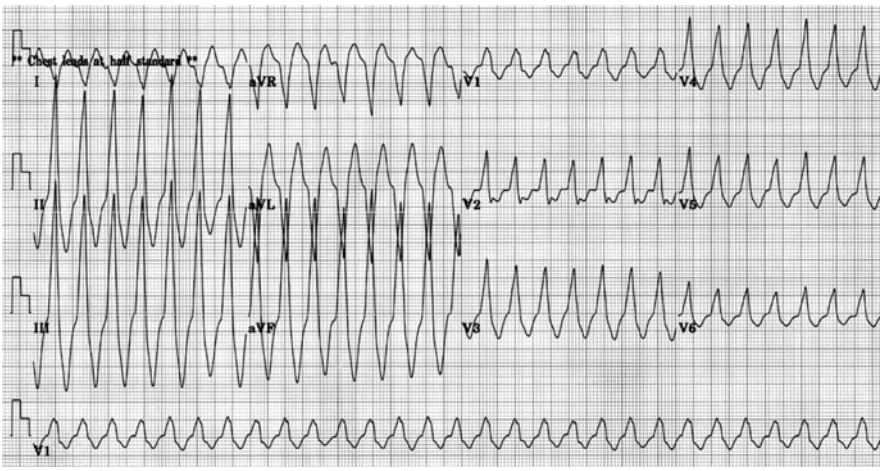


Figure 10: Patient with ventricular tachycardia. Although AV dissociation cannot be seen, positive concordance in the precordial leads is present.

leads. Simultaneous recording of the leads can be very helpful in determining the precordial QRS morphology. Reexamine Figure 6. The beginning and end of the positive QRS complex in lead V_1 is relatively easy to identify, but it becomes much more difficult to decide what the QRS complex is in V_4 through V_6. Where does the QRS begin and end, and is the QRS positive or negative? By drawing lines upward from the beginning and end of the QRS complex from the bottom tracing in V_1 under the V_4 through V_6 column (dotted lines), it becomes apparent that the QRS complexes in V_4 through V_6 are positive and that positive concordance is present.

Most experts in elctrocardiography use "absence of an RS complex in the precordial leads" as their initial tool for assessing whether ventricular tachycardia is present. The astute student will realize that this "rule" will encompass both negative and positive concordance and a few additional tachycardias (those with both positive and negative QRS complexes but no biphasic QRS complexes) (Figure 11). In the author's experience, the absence of an RS complex is the best of all of the QRS morphology criteria for identifying ventricular tachycardia.

Negative QRS complex in V_1 (Left bundle branch block morphology)

If the QRS complex is negative in lead V_1 the tachycardia is classified as having a left bundle branch block morphology. In general, a left bundle block morphology suggests that the right ventricle or interventricular septum is the first portion of the ventricle to be activated. There are several clues that have been described in the QRS morphology that can be useful for differentiating ventricular tachycardia from supraventricular tachycardia with left bundle branch block. In true left bundle branch block, the initial septal activation results in a relatively narrow R wave and a

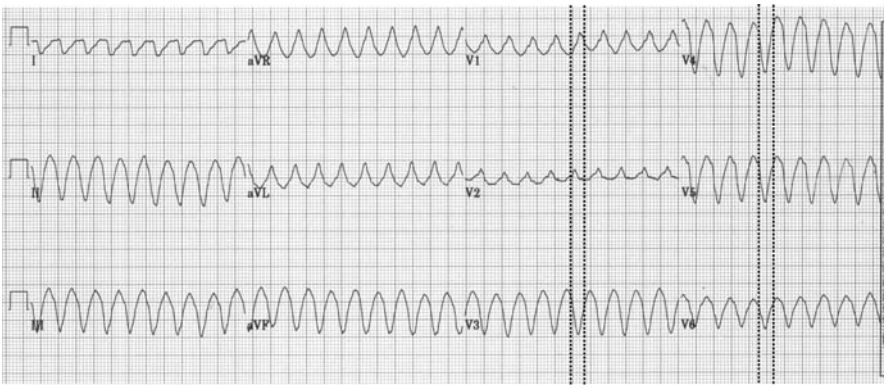


Figure 11: ECG from a patient with ventricular tachycardia with no biphasic RS complexes in the precordial leads (positive in V_1 and V_2, negative in V_3–V_5).

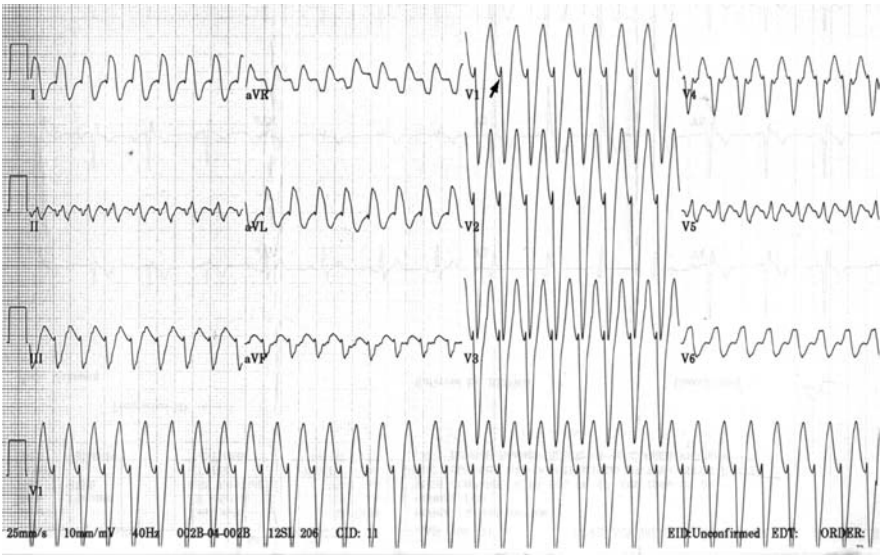


Figure 12: An ECG from a patient with AV node reentry and left bundle branch block. Notice that the downstroke in V_1 is quite steep and the septal R wave in V_1 is narrow (*arrow*).

steep downstroke (Figure 12). A broad R wave (> 0.03 s, or approximately one "little" box) or a slower downstroke (interval of the beginning of the QRS complex to the nadir of the S wave > 0.07 s, or approximately two "little" boxes) suggests that ventricular tachycardia is present. Examine the ECG in Figure 13. The wide complex tachycardia has a left bundle branch block morphology. The downstroke in V_1 is slow. In addition, a notch on the downstroke is a sign of ventricular tachycardia.

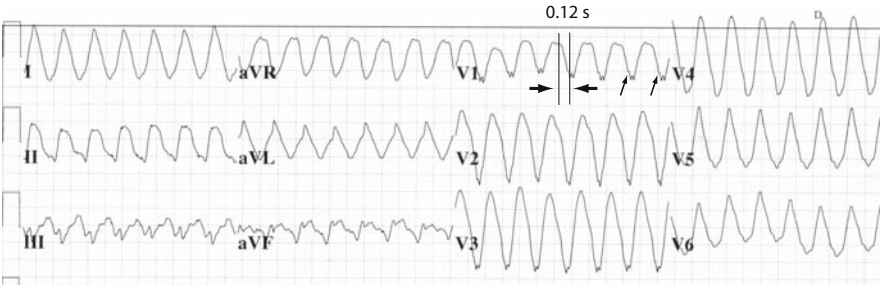


Figure 13: Ventricular tachycardia with a LBBB morphology. Notice that the downstroke in V_1 is slower than that in Figure 12, leading to a 0.12 s interval from the beginning of the QRS to the initial S wave. A notch in the downstroke in lead V_1 is also present (small *arrows*)

All three of these signs are manifestations of extremely abnormal septal activation and the presence of ventricular tachycardia and underlying structural heart disease. In fact, the notch on the QRS probably represents a Q wave from a previous myocardial infarction. As a final point, the presence of Q waves in any set of leads suggestive of a prior myocardial infarction is an excellent sign for ventricular tachycardia. Since most ventricular tachycardias are due to reentrant circuits developing at the site of a prior myocardial infarction, the morphology of ventricular activation will reflect this site of ventricular activation. For example, in the patient with an inferior wall myocardial infarction and a reentrant circuit emanating from this region, Q waves will be observed in the inferior leads, since the general direction of ventricular activation will initially be away from this site.

Case Study (continued): Reexamine Mr. Ramsey's ECG. Notice that there are prominent anterior and lateral Q waves from his prior myocardial infarction. Although the QRS complexes are very bizarre, the beginning of the QRS complex can be determined by drawing a vertical line up from the initial small deflection in V_1 (This cannot be a P wave because the PR interval would be too short; and remember, you found the "real" dissociated P waves earlier in this lead). His ventricular tachycardia is due to a reentrant circuit at this scar site.

Positive QRS complex in V_1 (right bundle branch block morphology)

If a predominantly positive QRS complex is observed in lead V_1, then the patient either has ventricular tachycardia with initial activation in the left ventricle or supraventricular tachycardia with right bundle branch block. Three patterns in V_1 are more likely to be associated with ventricular tachycardia: monophasic R, qR, or an Rsr'. An example of a patient with ventricular tachycardia and a monophasic R in V_1 is shown in Figure 14. If the patient has an rsR' complex with a prominent terminal positive complex, supraventricular tachycardia with right bundle branch block is more likely (Figure 15). It can sometimes be useful to evaluate V_6 in a

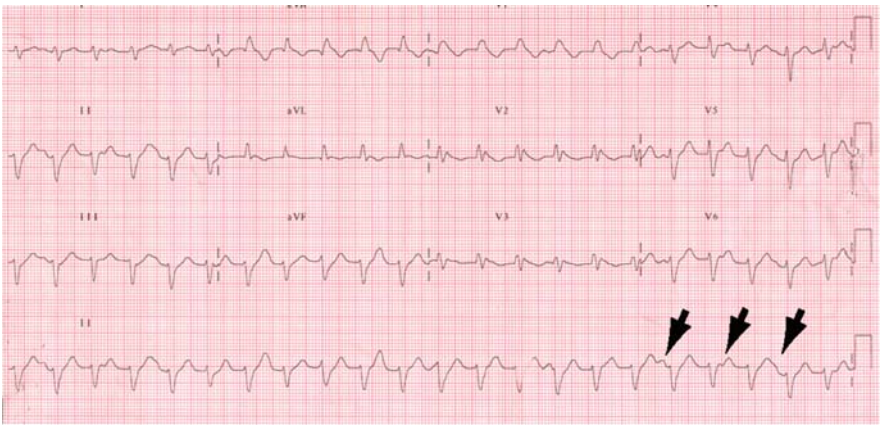


Figure 14: A patient with ventricular tachycardia. A monophasic R wave is seen in V_1. AV dissociation (*arrows* mark atrial activity) is the main finding that confirms the diagnosis as ventricular tachycardia.

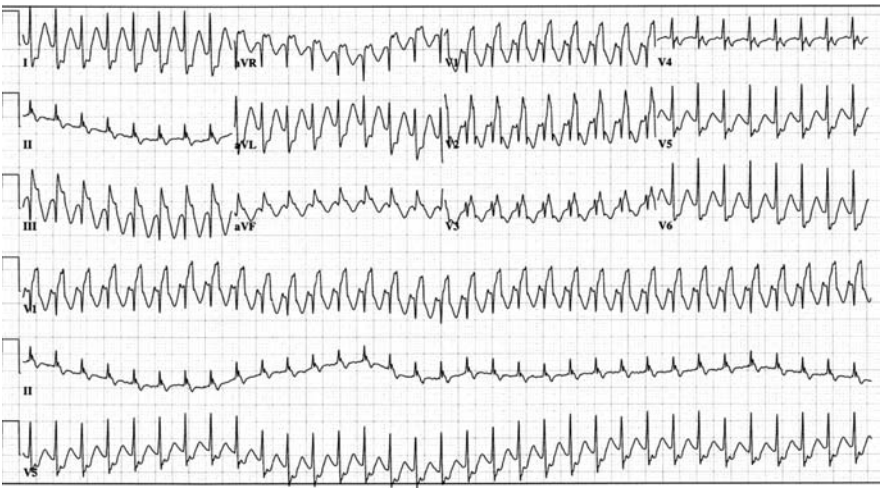


Figure 15: A patient with a wide complex tachycardia due to orthodromic AV reentry using an accessory pathway. Although AV node conduction is normal, the patient has right bundle branch block.

patient with a right bundle branch block morphology tachycardia. In typical right bundle branch block, V_6 will usually have an R wave with a terminal S wave. A V_6 pattern that is completely negative or positive would not be produced by right bundle branch block and would be diagnostic for the presence of ventricular tachycardia.

The morphology clues for differentiating ventricular tachycardia from supraventricular tachycardia with aberrant conduction are summarized in Figure 16. It is important to note that while helpful, QRS morphology characteristics are the

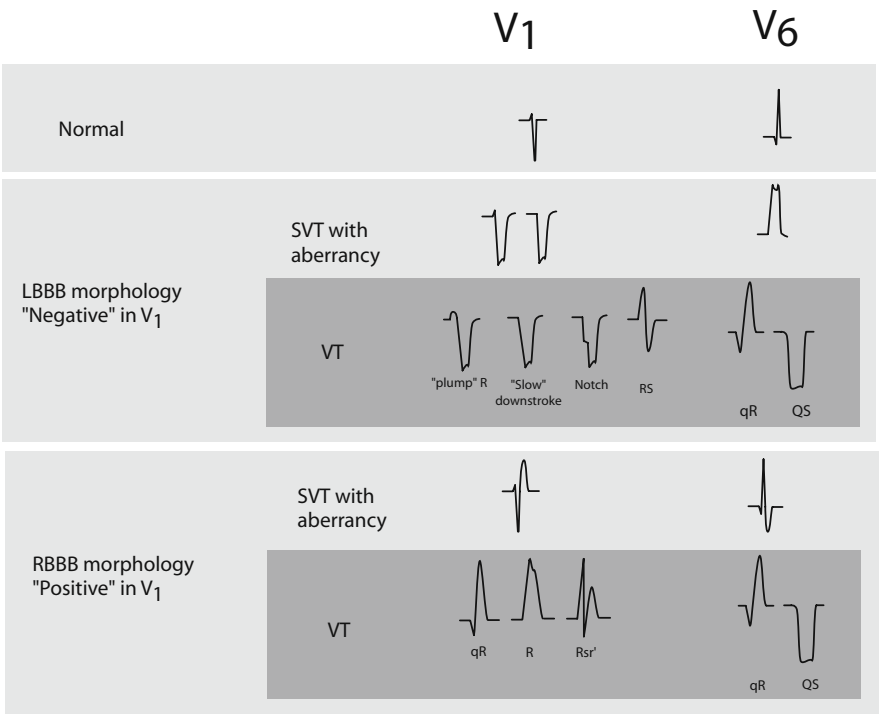


Figure 16: Summary of QRS morphology clues in LBBB and RBBB morphology tachycardias.

most difficult and least reliable ECG tools for evaluating wide complex tachycardia. So how does the student remember this apparent myriad of precordial patterns? The best technique is always to keep in mind what aberrancy ought to look like, and if the morphology is different "from expected," make a diagnosis of ventricular tachycardia. For example, right bundle branch block typically has an rSR' complex in V_1 with a large terminal R wave, and V_6 usually has an RS pattern, while left bundle branch block usually has a completely negative QS complex in V_1 and a monophasic positive R wave in V_6. If a tachycardia has any deviation from these basic forms, the diagnosis will often be ventricular tachycardia.

Relative value of ECG criteria for differentiating ventricular tachycardia from supraventricular tachycardia with aberrant conduction:

1. *AV dissociation (and its manifestations: fusion, capture): "Gold standard." Presence is diagnostic for ventricular tachycardia.*
2. *Absence of a precordial RS (negative and positive concordance): Very useful. If present, ventricular tachycardia is extremely likely.*
3. *Other QRS morphology criteria, including "northwest" axis and QRS width: Provide clues but use with caution.*
4. *"Fallback" diagnosis is always ventricular tachycardia.*

Irregular wide complex tachycardia

Clinically, the most common form of irregular tachycardia is atrial fibrillation with bundle branch block aberrancy (Figure 17). However, it is important for the reader to remember that irregular ventricular tachycardia can be observed and that the morphology of the QRS complexes should be specifically evaluated. For example, the ECG in Figure 18 was diagnosed as atrial fibrillation for several days on the hospital floor. However, careful examination of the ECG makes aberrant conduction unlikely. Notice that in the precordium, all of the QRS complexes are positive. Ultimately, an electrode catheter was placed in the right atrium. In this case, more accurate recordings of cardiac depolarization can be obtained because the recording system is immediately adjacent to cardiac tissue, rather than recording the ECG, through the skin and other tissue, as is done with the 12-lead ECG. With the catheter placed in the right atrium, organized atrial activation can be observed, confirming the presence of AV dissociation and proving the mechanism to be ventricular tachycardia.

Rapid ventricular activation can lead to disorganized ventricular activation. This is frequently called ventricular fibrillation or polymorphic ventricular tachycardia (Figure 19). If rapid ventricular depolarization leads to large deflections from baseline, the rhythm is called polymorphic ventricular tachycardia; and if only small deflections are observed, the term ventricular fibrillation is used. There are no specific amplitude criteria to differentiate between ventricular fibrillation and polymorphic ventricular tachycardia. The only clinical distinction is that polymorphic

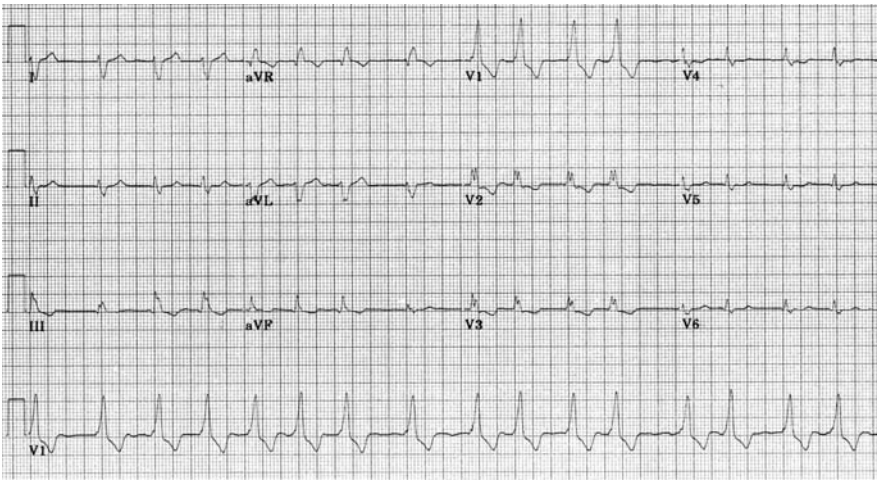


Figure 17: ECG from a patient with atrial fibrillation and right bundle branch block. A monophasic R is seen in V_1, showing the limitations of QRS morphology clues for differentiating ventricular tachycardia from supraventricular tachycardia with aberrant conduction.

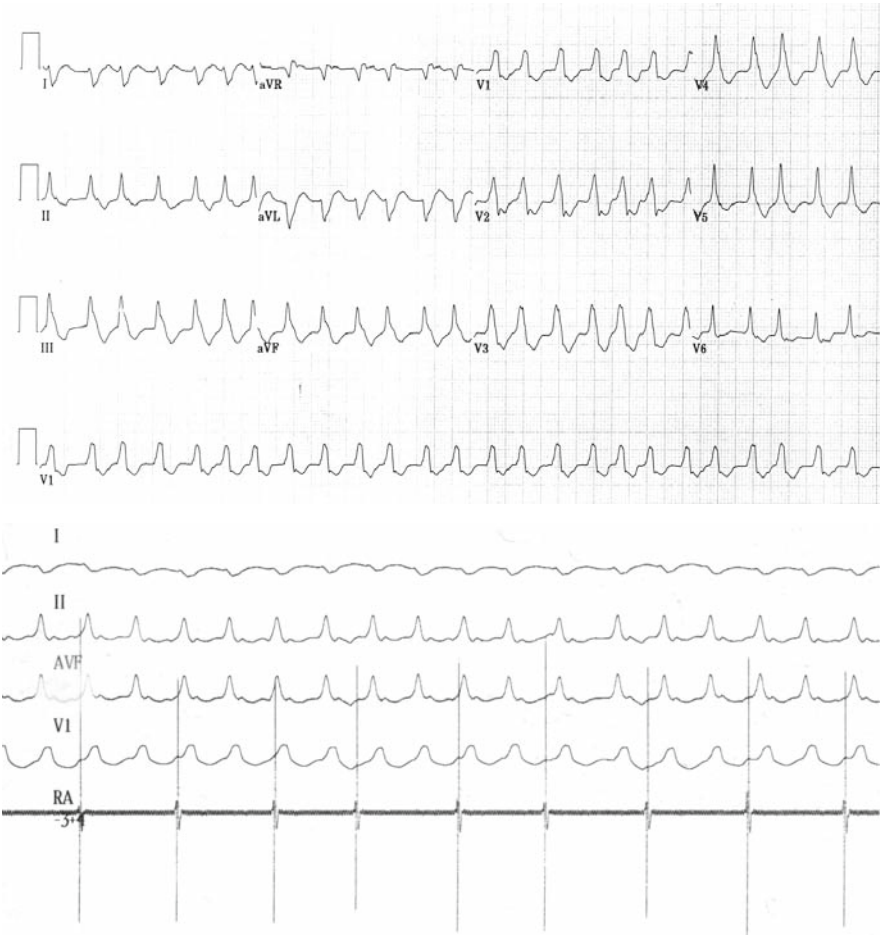


Figure 18: *Top*. ECG from a patient that was thought to have atrial fibrillation with right bundle branch block. Compare this ECG to Figure 16. Although in both ECGs a monophasic R wave in V_1 is present, this ECG has positive concordance (absence of an RS complex). *Bottom*. The patient had an electrode catheter placed in the right atrium. Notice that atrial activity is organized (atrial fibrillation would have displayed rapid activity) and dissociated from ventricular activity, confirming the presence of ventricular tachycardia.

ventricular tachycardia can potentially self-terminate (often called "nonsustained"), but otherwise there is very little difference clinically; both represent very dangerous and potentially lethal arrhythmias. In general, the only effective treatment of ventricular fibrillation or sustained polymorphic ventricular tachycardia is defibrillation, in which electrical current is delivered through the heart to extinguish the fibrillatory waves (Figure 20). After a pause, the patient's heart begins to beat normally. The

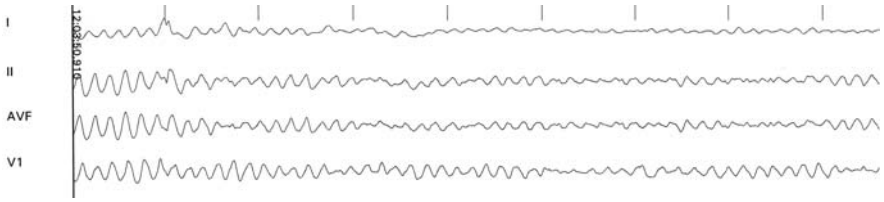


Figure 19: Ventricular fibrillation. Rapid irregular ventricular activity that is hemodynamically unstable.

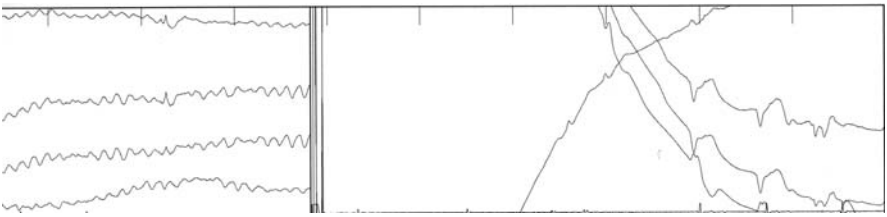


Figure 20: Defibrillation for the treatment of ventricular fibrillation. An external shock of 360 J is delivered from pads placed on the anterior and posterior chest that extinguishes the multiple reentrant circuits of ventricular fibrillation and restores normal sinus rhythm. Transient ST segment elevation is observed after the cardioversion. Defibrillation leads to significant artifact on the ECG due to the energy from the shock.

specific form of polymorphic ventricular tachycardia called torsades de pointes was discussed in detail in Chapter 6. The diagnosis of torsade de pointes is made if the patient has an associated prolonged QT interval noted during normal beats before and after the epidsode of polymorphic ventricular tachycardia.

A final specific form of irregular wide complex tachycardia should be mentioned here. In patients with the Wolff-Parkinson-White Syndrome, development of atrial fibrillation will lead to a very rapid and irregular wide complex tachycardia due to rapid ventricular activation via the AV node and an accessory pathway. A complete discussion of this arrhythmia in the Wolff-Parkinson-White Syndrome is provided in Chapter 15.

Key points

1. Wide complex tachycardia can be due to ventricular tachycardia or supraventricular tachycardia with abnormal ventricular activation.
2. Clinically, a history of prior cardiac disease makes ventricular tachycardia much more likely.

3. There are several findings on the ECG that can help determine if ventricular tachycardia is present, including AV dissociation, concordance of the precordial leads, and specific morphology clues.
4. When examining the ECG, the first step is to scan the entire ECG as a whole and look for any unexpected deflections that suggest the presence of AV dissociation.

Review questions

1. The ECG in Figure 21 would be considered:
   A. Left bundle branch block
   B. Right bundle branch block morphology
   C. Indeterminant
2. The ECG in Figure 21 is:
   A. Atrial fibrillation with aberrant conduction
   B. Ventricular tachycardia
   C. Supraventricular tachycardia with aberrant conduction
3. The ECG in Figure 22 would be classified as:
   A. Left bundle branch block morphology
   B. Right bundle branch block morphology
4. The ECG in Figure 22 is:
   A. Atrial fibrillation with aberrant conduction
   B. Ventricular tachycardia
   C. Supraventricular tachycardia with aberrant conduction

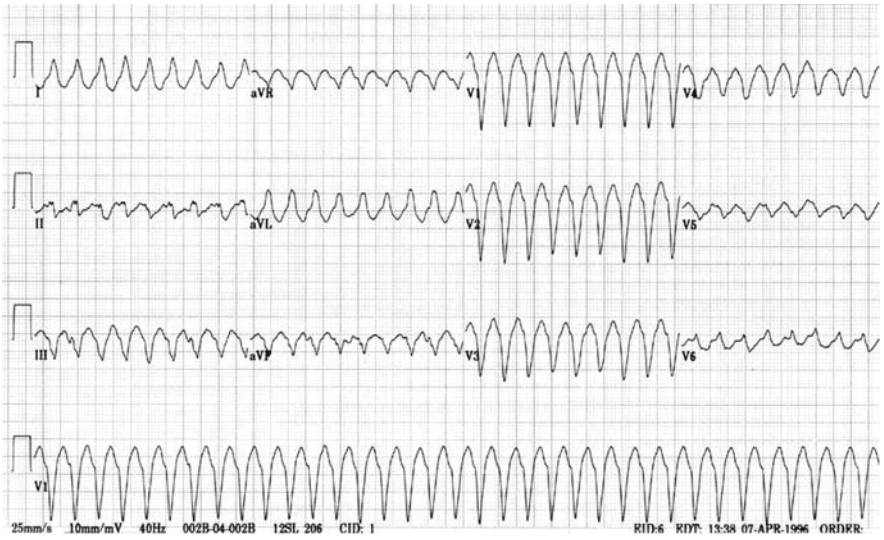


Figure 21: ECG for Questions 1 and 2.

5. All of the following ECG criteria suggest ventricular tachycardia except:

   A. Fusion beats.
   B. "Northwest axis."
   C. Presence of an RS in the precordial leads.
   D. A QRS duration > 0.16 s.

6. Describe the nonsustained wide QRS tachycardias in Figure 23.

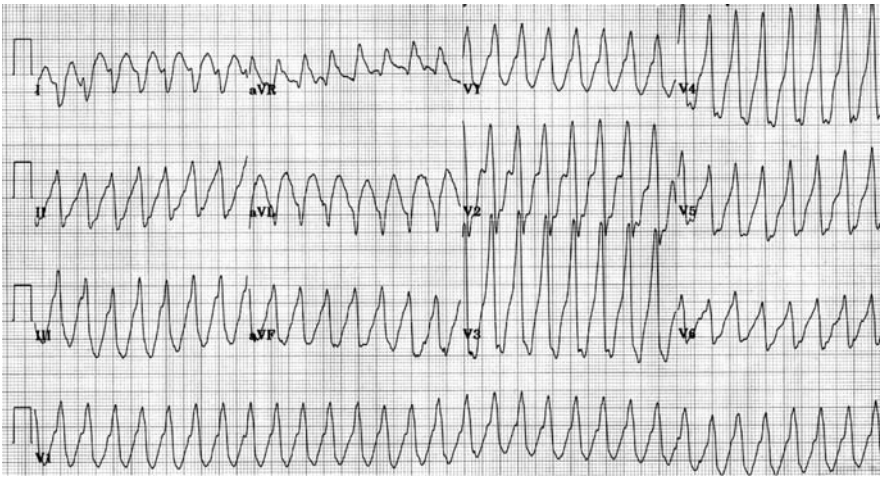


Figure 22: ECG for Questions 3 and 4.

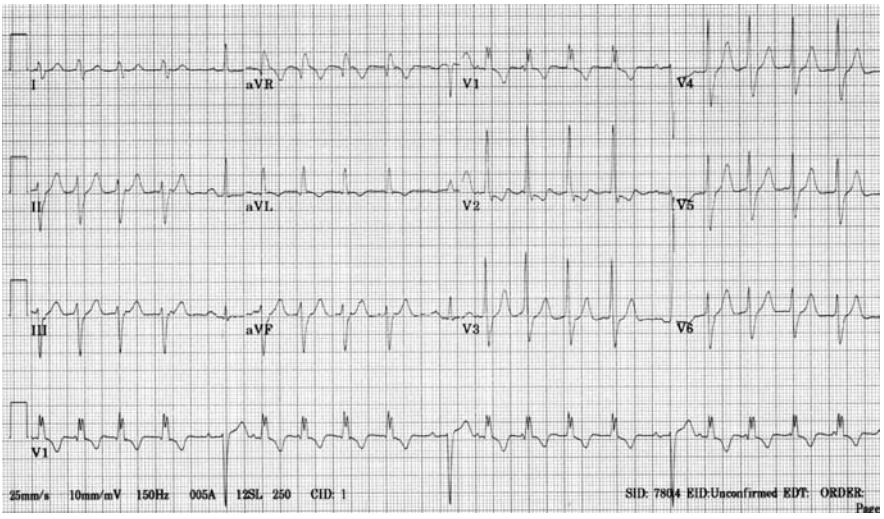


Figure 23: ECG for Question 6.

Answers

1. A. The ECG would be considered to have left bundle branch block morphology since the QRS complex in V_1 is entirely negative.
2. B. The patient has ventricular tachycardia. AV dissociation is present and can best be seen in the inferior leads.
3. B Since the V_1 morphology is positive, the tachycardia would be classified as having a right bundle branch block morphology.
4. B. This ECG is much more difficult to interpret than Figure 21. Generally there is positive concordance, with only a slight negative S wave in V_2. There is a monophasic R in V_1. There is a suggestion of AV dissociation with a negative deflection present just before the second QRS in I.
5. C. It is the absence of an RS complex in the precordial leads that suggests the presence of ventricular tachycardia. AV dissociation and its manifestations (fusion and capture beats) are the best ECG criteria for ventricular tachycardia. A wide QRS and "northwest" axis are far less useful, but the presence of either finding would make ventricular tachycardia more likely.
6. Figure 23 shows a patient with salvos of nonsustained ventricular tachycardia. Notice that although a P wave precedes the first QRS complex of the tachycardia, it occurs at the expected time and the PR interval is short. This rules out intermittent right bundle branch block aberrancy, since in this case the PR interval would be the same as the narrow QRS beat. The rate of the wide QRS rhythm is faster than the sinus rate (AV dissociation), so that a P wave does not precede the third or fourth beats of the wide QRS salvos. Finally, the mechanism is probably an automatic site, since the first and subsequent beats of the salvos have the same morphology. By evaluating the QRS morphlogy, the general location of the automatic site can be determined. The right bundle branch block morphology suggests that the site is within the left ventricle, the relative narrowness of the QRS complex suggests that at least a portion of the Purkinje system is used (site must be septal), and finally the superior axis suggests that the ventricle is activated from inferior to superior. Taken together, analysis of the QRS morphology suggests that the automatic site arises near the inferior and midportion of the left side of the interventricular septum. Specialized cardiologists called electrophysiologists use this information to ablate or destroy the abnormal focus if clinically indicated.

Chapter 13
Pacemakers

Pacemakers are implantable devices designed to ameliorate symptomatic slow heart rates. The first implanted pacemakers were developed fifty years ago and were large, unreliable devices with short lifespans. Pacemakers have evolved from devices that only paced the ventricle from a single site to sophisticated devices that utilize one, two, or three leads and that maintain atrioventricular synchrony, attempt to mimic normal ventricular activation, and continuously monitor the heart rhythm and other hemodynamic parameters.

Case study

Ms. Fischer is an eighty-five-year-old woman who comes to your office for a routine physical examination. She had a permanent pacemaker implanted 3 years ago. Her ECG is shown in Figure 1. How are the ventricles being activated? Is the atrial-ventricular relationship normal?

Single chamber pacemaker

The simplest type of pacemakers use one lead placed in the heart. Most commonly a single pacing lead is placed through the subclavian vein, superior vena cava, into the right ventricle (Figure 2). The pacing lead allows transmission of electricity produced by the pulse generator (usually placed in the anterior chest/shoulder region) to the endocardium. If sufficient energy is used, the ventricle can be depolarized from this site. The pacing lead can also receive signals from the heart (sensing).

The pacemaker system functions by a series of timers that can be individually programmed. When ventricular activity is sensed or the ventricle is paced, a low rate timer is started. If the timer expires without a sensed ventricular event, the pacemaker will deliver a ventricular stimulus and the low rate timer is restarted (Figure 2). The pacemaker can be programmed to different pacing rates by changing the low rate timer. In this way pacing is provided only when there is significant bradycardia; if the patient has a sufficiently high heart rate the low rate timer will continue to reset and not deliver a pacing stimulus (inhibited). This type of pacing is sometimes called demand pacing, since pacing is provided only when required. In contrast, a pacemaker can be programmed to pace continuously (usually called asynchronous pacing). When programmed in this manner the low rate timer will not

F. Kusumoto, *ECG Interpretation: From Pathophysiology to Clinical Application*,
DOI 10.1007/978-0-387-88880-4_13,

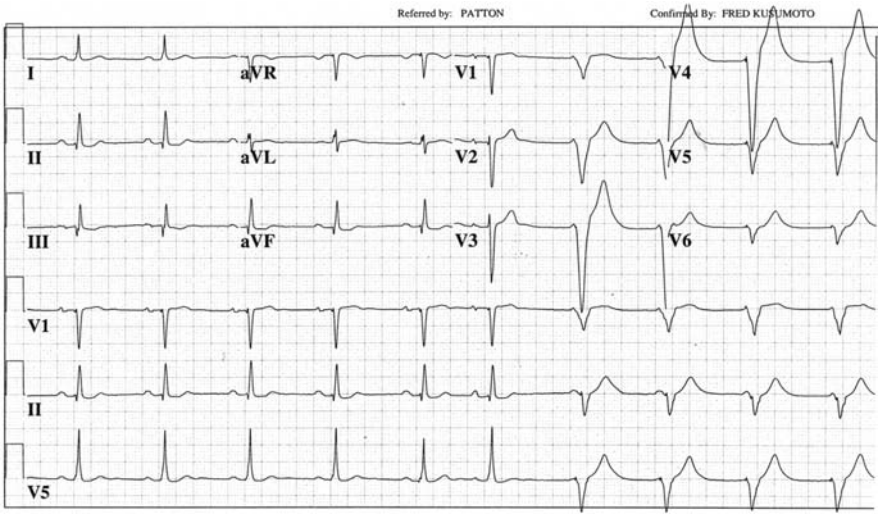


Figure 1: ECG from Ms. Fischer.

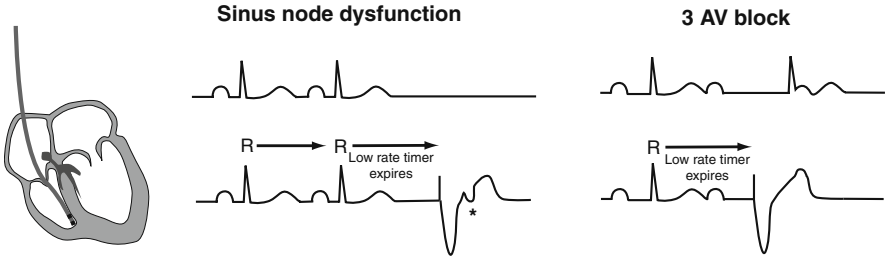


Figure 2: Timing cycles for a ventricular pacemaker programmed to a "demand" mode in the setting of sinus node dysfunction and AV block. A sensed R wave or a pacing stimulus will reset the low rate timer. If the low rate timer expires without a sensed QRS complex, a pacing stimulus will be delivered. In a patient with sinus node dysfunction, since AV conduction is intact, the His bundle and AV node are a "two-way street" and a retrograde P wave will often be observed (*).

be reset when an intrinsic QRS complex is sensed; instead it will pace continuously every time the low rate timer expires. An example of asynchronous pacing is shown in Figure 3. Every time the low rate timer expires, a pacing stimulus is delivered. The patient's intrinsic QRS complexes do not reset the low rate timer. Some pacing stimuli do not produce QRS complexes because they are delivered at a time the ventricles are refractory.

With single chamber ventricular pacemakers, atrioventricular synchrony is not preserved. While single chamber ventricular pacemakers prevent slow heart rates, atrioventricular synchrony is absent and ventricular depolarization is abnormal.

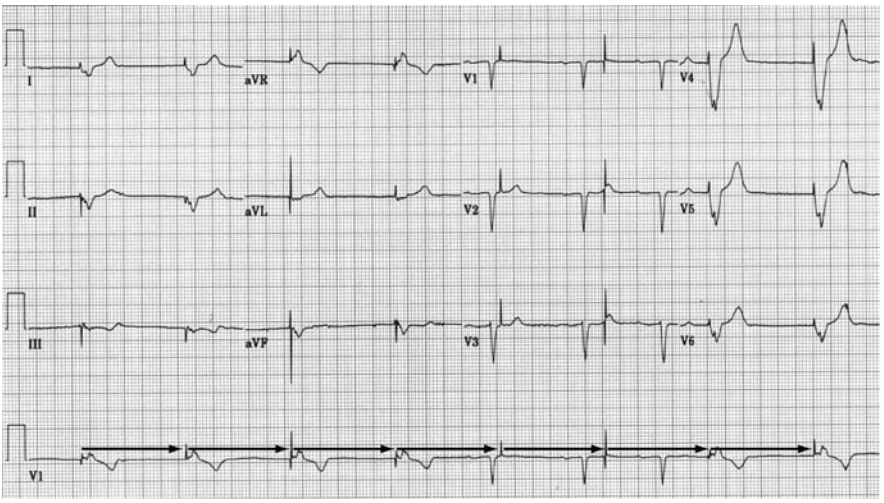


Figure 3: 12-lead of a patient with asynchronous ventricular pacing. In this case a sensed R wave will not reset the low rate timer, so the pacemaker continuously delivers impulses each time the low rate timer expires. If the pacing stimulus is delivered at a time when the ventricles are refractory, no QRS due to the pacing stimulus will be produced.

Remember that the ventricle is normally activated over the His-Purkinje system and ventricular depolarization is completed within 90–110 ms (< 3 little boxes). If the ventricles are activated from a single lead within the right ventricle, depolarization of the ventricles occurs in a "cell-to-cell" fashion and the resulting QRS complex is broad and usually greater than 120 ms. The site of pacing can be determined from the morphology of the QRS complex. Most commonly ventricular leads are placed in the inferoapical portion of the right ventricle, and the paced QRS complex has a left bundle branch block morphology because the right ventricle is activated before the left ventricle. In addition, the precordial QRS complexes are predominantly negative and the axis in the frontal plane is superior (Figure 3). If the pacing lead were placed in the right ventricular outflow tract, the paced QRS complex would still have a left bundle branch block morphology, but would have an inferior axis in the frontal plane (Figure 4). For patients that require pacing after surgery, pacing leads are frequently placed on the epicardial surface of the left ventricle. In this case, since the left ventricle is activated before the right ventricle, the QRS will often have a right bundle branch block morphology (Figure 5).

Case Study (continued): Ms. Fischer has a single chamber pacemaker. After the premature atrial contraction, there is a slight pause in sinus node automaticity (due to resetting of the sinus node), but because the patient has a single chamber ventricular pacemaker, a ventricular stimulus and a ventricular paced beat is observed. After the ventricular stimulus the QRS is wide with a left bundle branch block morphology.

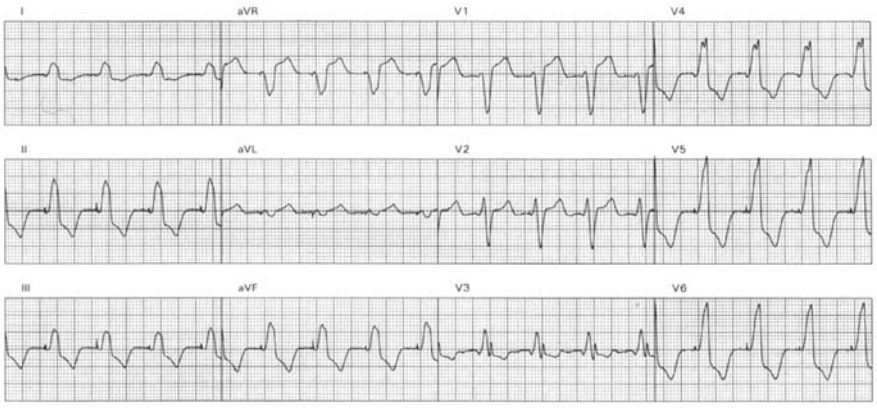


Figure 4: Ventricular pacing from the right ventricular outflow tract. Since the pacing lead is in the right ventricle, a QRS with a LBBB morphology is produced. The QRS axis is directed inferiorly since the ventricles are activated from the superiorly located pacing lead (reprinted with permission from Kusumoto F, Goldschlager N (Eds.) *Cardiac Pacing for the Clinician*, Springer, New York NY, 2008).

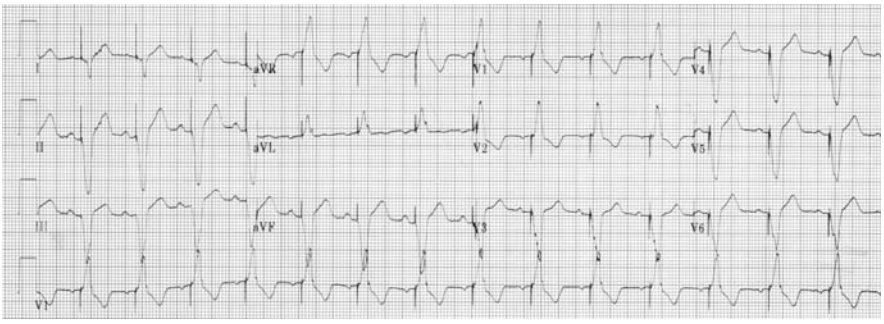


Figure 5: Ventricular pacing from the inferolateral wall of the left ventricle using an epicardial pacing lead. Notice that the QRS complex is negative in the inferior leads and V_3–V_6. Activation of the left ventricle proceeds upward and rightward, leading to positive QRS complexes in V_1 and V_2, aVL and aVR, with aVR > aVL.

Since the ventricular lead is normally placed in the right ventricle, most commonly the paced QRS has a left bundle branch block morphology because the right ventricle is activated before the left ventricle. It is hard to know the reason for Ms. Fischer's initial implant: Sinus rate slowing leads to ventricular pacing, but the ECG shows first degree AV block and mild QRS widening (without a specific fascicular block morphology).

On questioning her in the clinic she notices that she gets short of breath when she walks up the stairs in her house.

Dual chamber pacemakers

As the name suggests, in dual chamber pacemakers two leads are used: one lead is placed in the right atrium and the other lead is in the right ventricle. For dual chamber pacemakers several additional timing cycles are required; the most important new timing cycle is the AV interval that attempts to mimic the PR interval (Figure 6). After an atrial sensed event (the patient's P wave) or atrial stimulus, the AV interval is started and the low rate timer is started. If the AV interval expires without sensed ventricular activity, a ventricular stimulus is delivered. If a ventricular signal is sensed the pacemaker will not deliver a ventricular stimulus (Figure 6). If the low rate timer expires without a sensed atrial event, an atrial stimulus will be delivered and the process repeats. Using the combination of the low rate timer and the AV interval, the dual chamber pacemaker prevents bradycardia and maintains atrioventricular synchrony if the patient develops atrioventricular block or sinus node dysfunction. In patients with bradycardia due to sinus node dysfunction, since the problem is sinus node automaticity, atrial pacing will often be observed. Since the patient has intact AV node conduction, an R wave will be sensed before the AV interval timer expires and a ventricular stimulus will not be observed. In patients with bradycardia due to AV block, sinus node automaticity is normal, so atrial pacing is usually not observed. However, because of the AV block, after the P wave the AV interval timer will usually expire without a sensed R wave and a ventricular stimulus will be delivered.

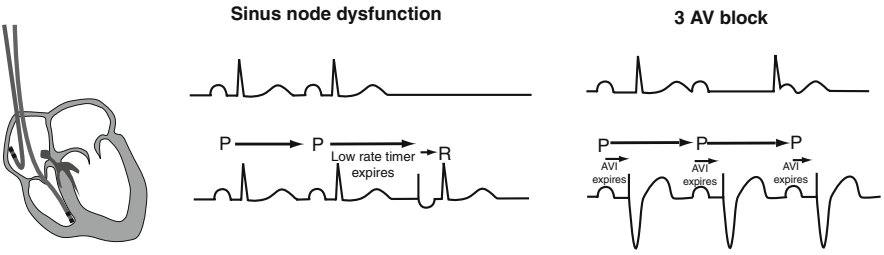


Figure 6: Timing cycles for a dual chamber pacemaker. In sinus node dysfunction, the absence of appropriate sinus node automaticity leads to expiration of the low rate timer, and in this case an atrial stimulus is delivered and the AV interval timer is started. Since the patient has intact AV conduction, the paced atrial beat leads to an intrinsic QRS complex. The QRS is sensed by the pacemaker before the AV interval expires and a ventricular stimulus is not provided. In contrast, in AV block, sinus node automaticity is normal, but after a sensed P wave no intrinsic QRS is sensed, so when the AV interval timer expires a ventricular stimulus is delivered.

Although dual chamber pacemakers are better able to maintain atrioventricular synchrony for both sinus node dysfunction and AV block, an additional timing cycle called the upper rate limit has to be introduced. The pacemaker cannot differentiate whether an atrial rate is normal or abnormal. Think back to the chapter on supraventricular tachycardia. Since the AV node has decremental conduction properties, it will limit the ventricular rate in the setting of atrial tachycardias. For example, in atrial flutter, the AV node will often only conduct every other or every third atrial flutter depolarization. It is important to place a set limit on the upper rate at which a pacemaker will deliver ventricular stimuli.

An example of the importance of the upper rate limit is shown in Figure 7. In the top ECG, notice the rapid ventricular paced rate. When the pacemaker is inhibited, it reveals that the patient is in an atrial tachycardia with an atrial rate of approximately 240 beats per minute. If each of these atrial beats was "tracked" by the pacemaker, the patient would end up with a ventricular rate of 240 beats per minute. Fortunately, the pacemaker has an upper rate limit of 120 beats per minute, so that no matter how fast the atrial rate, ventricular pacing will not occur faster than this specific value.

In the bottom portion of Figure 7, the pacemaker has been reprogrammed to a "single chamber" ventricular pacemaker with a programmed low rate timer for 40 beats per minute. Since the patient's own native rate is 60 beats per minute, the pacemaker does not deliver pacing stimuli. It is important to realize that pacing systems can be programmed to different pacing modes, so dual chamber pacemakers can be programmed to function as single chamber pacemakers.

The patient has a regular heart rate of 60 beats per minute. There are two possibilities for the patient's native rhythm. In response to the atrial tachycardia the AV node could be conducting every fourth beat (since the atrial rate is approximately 240 beats per minute, conduction of every fourth beat would lead to a heart rate of 60 beats per minute), or the patient could have complete heart block with a ventricular escape rate of 60 beats per minute. If AV conduction were present, often there would be some irregularity due to different degrees of block (3:1, 2:1, and 5:1), and the relationship between the P waves and QRS complexes would be constant. In this case there is no relationship between the P waves and QRS complexes (varying "PR intervals"), and the ventricular rhythm is constant. This ECG illustrates the importance of trying to evaluate the native heart rhythm in patients with pacemakers.

Biventricular pacing

In patients with left bundle branch block the septal and lateral ventricular walls are activated sequentially rather than simultaneously. If the delay is severe enough, the lateral wall may contract at a time when the septum has already completed contraction or is even relaxing. Poor timing of ventricular depolarization is often referred to as cardiac dyssynchrony. In an effort to provide more efficient contraction patterns in these patients, pacemakers have been developed that activate the

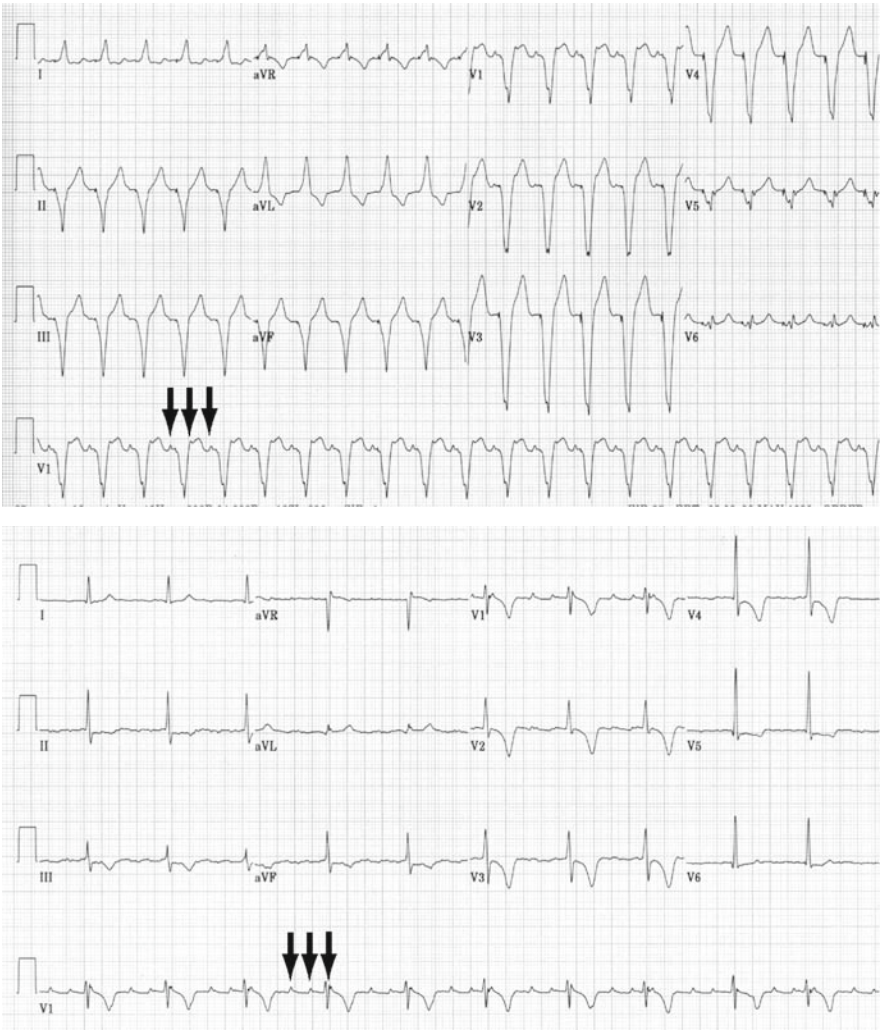


Figure 7: *Top*. Patient with a rapid wide complex rhythm of approximately 120 beats per minute. Since the initial factory setting for the upper rate limit is 120 beats per minute for most manufacturers, and since many physicians (unfortunately) do not change this value, a wide complex rhythm with this rate and left bundle branch block morphology should raise suspicion of the presence of a pacemaker tracking a rapid atrial rate. In this case the patient has an atrial tachycardia at a rate of approximately 240 beats per minute (*arrows*). *Bottom*. The pacemaker is reprogrammed to a slow ventricular rate (so now the pacemaker does not deliver pacing stimuli unless a slow ventricular rate is present). The P waves from the atrial tachycardia are now evident (*arrows*). The patient has underlying complete heart block with a ventricular rate of approximately 60 beats per minute.

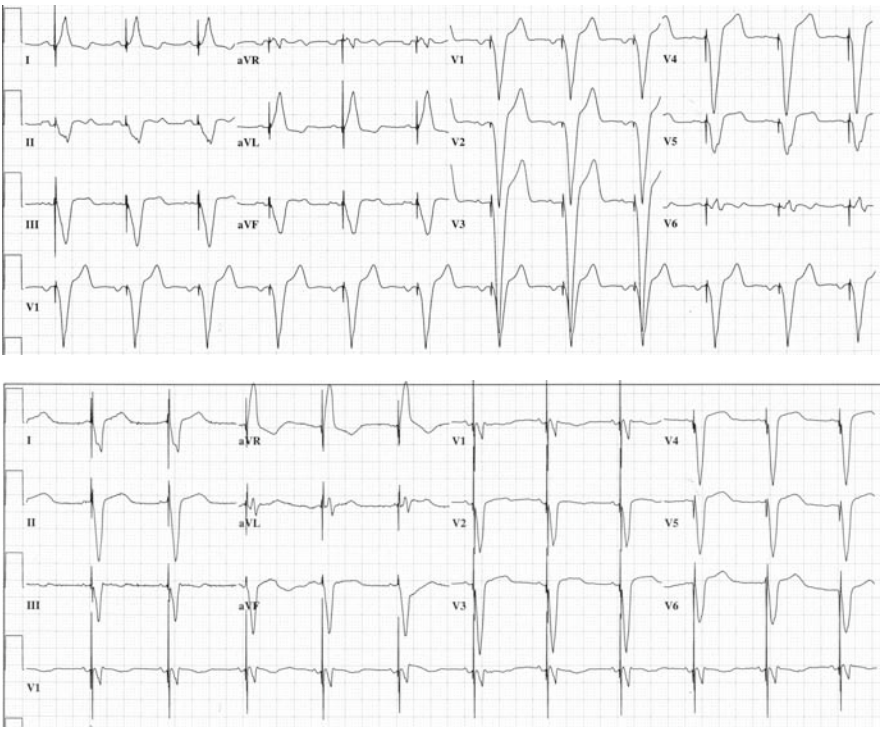


Figure 8: *Top*. ECG with pacing from the right ventricular apex shows left bundle branch block morphology with a superior axis. *Bottom*. Biventricular pacing from both the right ventricle and the lateral wall in the same patient is associated with a narrower QRS complex that simulates more closely normal ventricular activation.

ventricles from two sites, usually the right ventricular septum and the lateral wall of the left ventricle (biventricular pacing). In the presence of biventricular pacing the QRS complex is often narrower (Figure 8) and can improve hemodynamic function in some cases due to more coordinated ventricular contraction.

While single-site ventricular pacemakers prevent bradycardia, the ventricle is activated abnormally and cardiac dyssynchrony is present.

Case Study (continued): The clinician has several options, depending on the cause of Ms. Fischer's shortness of breath. Although both sinus node dysfunction and AV block are possible, the most likely cause of her symptoms would be insufficient increase in her sinus rate with exercise. Pacemakers can often be programmed to increase ventricular rates in response to activity by employing a motion sensor. Although this would provide increased heart rates with exercise, atrioventricular synchrony is lost and dyssynchronous ventricular activation is introduced. In some cases implantation of an additional atrial pacing lead to allow atrial pacing is required. To help elucidate the cause of her symptoms, the clinician could consider

a treadmill test to simulate walking upstairs, or place a continuously recording ECG monitor on the patient to evaluate her heart rhythm when she has symptoms.

Key points

1. Single chamber pacemakers usually have a single lead in the right ventricle.
2. Dual chamber pacemakers usually use a lead in the right atrium and a second lead in the right ventricle. Since both atrial and ventricular activity are monitored, atrioventricular synchrony can be maintained.
3. By evaluating the paced QRS complex, the location of the ventricular lead can be determined.
4. Pacing systems that stimulate both the left and right ventricle can be used for treatment of patients with heart failure who have dyssynchronous ventricular activation due to bundle branch block.

Questions

1. Examine the ECG in Figure 9. Is pacemaker function normal or abnormal? What programming changes should be considered?
2. Examine the ECG in Figure 10. Is pacing function normal?

Answers

1. Interestingly, this pacemaker is functioning normally as programmed. Notice the ventricular pacing stimuli delivered during the QRS complex. This is an example of "pseudofusion." The patient has a dual chamber device with a lead placed in the right ventricular apex. The patient has intact AV conduction,

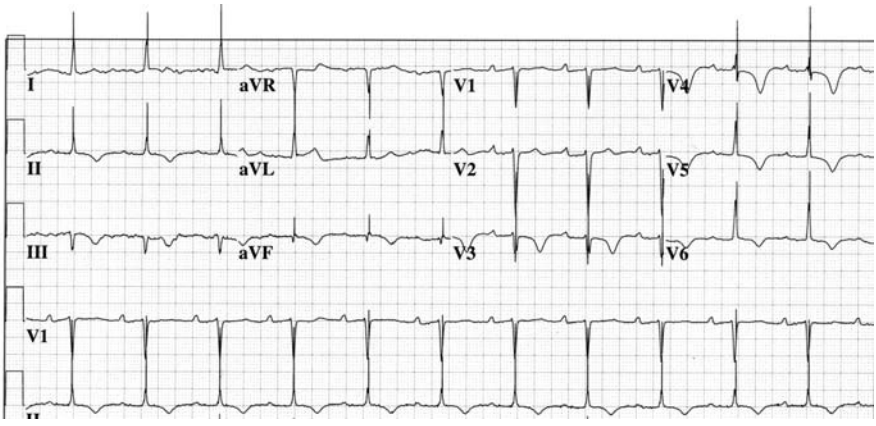


Figure 9: ECG for Question #1.

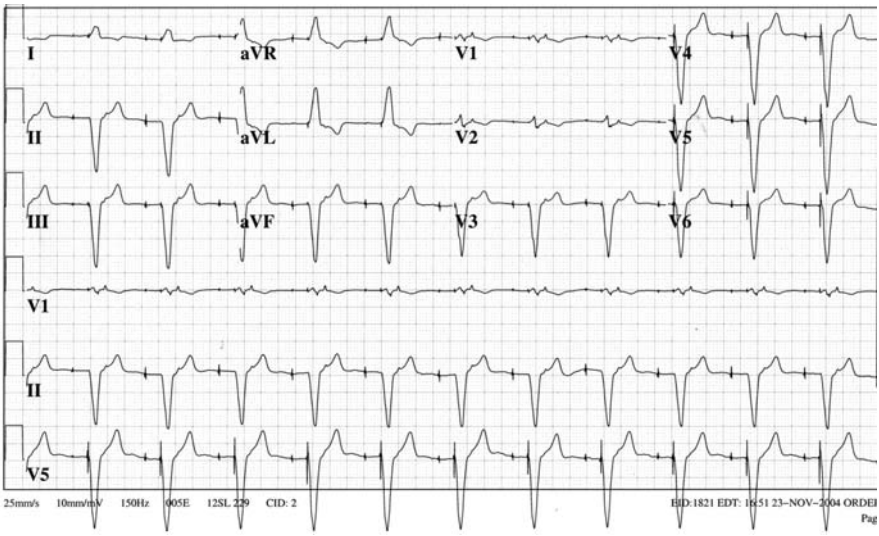


Figure 10: ECG for Question #2.

so the intrinsic P wave leads to an intrinsic QRS complex. However, in this case the AVI expires even as the ventricle is being activated, since the right ventricle is relatively late, and so a pacing stimulus is delivered. Fortunately, this is of little consequence, but it tends to deplete the battery, since it takes far more energy to deliver a stimulus than to sense an R wave. The AVI should be extended to allow ventricular sensing.

2. Pacemaker function is abnormal. Both atrial and ventricular stimuli can be observed. A QRS complex follows the ventricular stimulus, but no P wave follows the atrial stimulus. In fact, a retrograde P wave can be seen in the ST segment of the inferior leads. In this case the pacemaker should be programmed to higher voltages to capture the atria, and in some cases surgery is required to replace or reposition the atrial lead.

Part V

"Putting it all together"

Chapter 14

Analyzing ECGs: Methods, techniques, and identifying abnormalities

There are multiple techniques and systems for analyzing the ECG. In the beginning it is important for the student to develop a systematic and methodical approach to ECG reading (Table 1). Eventually everyone develops a personal style, although most experienced ECG readers evaluate the ECG with an initial "glance" to determine the extent of abnormalities and determine whether any urgent problems are immediately apparent on the ECG, and then go from "big" picture to "little" picture, focusing on specific findings at the end of their evaluation.

Computer-based analysis is now available for all commercially available ECG machines. Overall, the accuracy of these algorithms is excellent and ranges from 85 to 92%. However, several studies have shown that computerized analysis is less accurate than interpretation by experienced ECG readers. However, computerized interpretation has become an important adjunctive tool for ECG interpretation, and even the experienced reader will always review the computer diagnoses and compare them to his or her interpretation.

Systematic analysis

Assess quality

Are all 12 leads recorded, and is the ECG recorded at standard speed and gain? Significant baseline artifact due to patient movement or poor electrode contact will preclude accurate evaluation. If the patient is "hooked up" to the ECG machine, are the electrodes in the appropriate place (limb leads on the limbs and connected correctly)? The specific ECG findings with lead misplacement are covered in the last chapter.

Extremity electrode locations: Right arm: white; Left arm: black; Right leg: green; Left leg: red.
"Christmas tree on the bottom, green and white on the right."
"When I am driving my right foot is on the gas (green), my left foot is on the brake (red), and my left arm is getting a tan (black).

F. Kusumoto, *ECG Interpretation: From Pathophysiology to Clinical Application*,
DOI 10.1007/978-0-387-88880-4_14, © Springer Science+Business Media, LLC 2009

Table 1: Systematic evaluation of the ECG

Criteria	Considerations
Rate	• Is the heart rate within the normal range (60–100 bpm)?
Rhythm	• Is normal sinus rhythm present with normal atrioventricular conduction (a P wave in front of every QRS complex)?
Intervals	• Are the intervals normal?
PR	• PR $<$ 0.12: Is WPW present? PR $>$ 0.22: first degree AV block
QRS	• QRS $>$ 0.12: Bundle branch block? Electrolytes? WPW?
QT	• QT prolonged? Electrolytes, drugs, genetic
Axis	• Is right axis deviation or left axis deviation present?
P wave morphology	• Is the P wave normal duration or abnormal shape?
QRS morphology	• Q waves? • High or low voltages? • Prominent R wave in V_1? • Delta wave? • Terminal waves in the QRS?
ST segment	• Is the ST isoelectric? • What leads have ST segment elevation present? • What leads have ST segment depression?
T wave	• Are T waves in the expected direction?

WPW: Wolff-Parkinson-White Syndrome

Assess rate and rhythm

Slow heart rates can be due to decreased automaticity or blocked atrioventricular conduction. Rapid heart rates are classified as rates $>$ 100 beats per minute and are generally evaluated by determining whether the QRS is wide or narrow. Please see the specific chapters on bradycardia and tachycardia for specific discussion of these conditions, and also the Appendix, Tables 6 and 7.

The majority of ECGs will have “normal” heart rates between 60 and 100 beats. In addition most of these ECGs will have normal sinus rhythms. Normal sinus rhythm is typically defined by three components: (1) The ventricular rate will be between 60 and 100 beats per minute. (2) The P wave will be upright in lead II and negative in aVR (the sinus node is initiating the beat). (3) Every P wave will be followed by a QRS complex within 0.20 seconds (atrioventricular conduction through the AV node and His-Purkinje system is normal) and the ventricular and atrial rate will be between 60 and 100 beats per minute.

Identify P waves/atrial activity/shape

Atrial activity should be analyzed. The shape of the P wave will provide clues to the initiation site of atrial depolarization. The height of the P wave will provide clues to whether atrial enlargement is present, and the width of the P wave provides clues to the presence of atrial chamber enlargement or slower atrial depolarization.

Assess ventricular depolarization

Wide or narrow?

If the QRS complex is less than 0.12 s, ventricular depolarization is more or less occurring normally over the His-Purkinje system. There are several causes for a wide QRS complex, which are summarized in Table 2. In each of these cases the ventricle is being abnormally activated. Most commonly, a wide QRS complex is due to abnormal conduction over the right or left bundle due to block or delay of ventricular activation. Rarely, a wide QRS complex is observed because the ventricles are being activated via both the His-Purkinje system and an accessory pathway. With greater use of cardiac devices, a wide QRS complex will be observed in patients with pacemakers where the ventricle is being activated artificially from an electrical impulse delivered via a ventricular pacing lead.

QRS axis?

The main axis of ventricular depolarization in the frontal plane should be evaluated. The normal QRS axis lies within −30° and 100°. An axis more leftward than −30° is defined as left axis deviation and simply means that ventricular activation is directed more superiorly than normal. Common causes for left axis deviation are summarized in Table 3. Left axis deviation can be observed in the presence of left anterior fascicular block, left ventricular hypertrophy, and inferior wall myocardial infarction. A more comprehensive list of causes for left axis deviation is given in the Appendix, Table 1.

In right axis deviation, ventricular activation has shifted in the clockwise direction and is greater than 100°. The common causes for right axis deviation are summarized in Table 4. A rightward shift can be due to left posterior fascicular block (late activation of the inferior left ventricle), an increase in right ventricular size or thickness, or a shift of the heart within the body (a more vertically positioned heart due to the hyperinflated lungs and flattened diaphragm observed in some patients with chronic obstructive lung disease). A more comprehensive list of right axis deviations is given in the Appendix, Table 1.

QRS voltage?

Prominent QRS voltages should arouse suspicion of ventricular hypertrophy. Since the left ventricle is generally larger than the right ventricle, increased QRS voltage is more noticeable in left ventricular hypertrophy. Reduced voltages can

Table 2: Wide QRS complex with a normal rate: abnormal ventricular activation

Condition	Pathophysiology	ECG findings
Bundle branch block	Block in the right or left bundle leads to sequential activation of the right and left ventricles	In right bundle branch block an rSR' will be seen in lead V_1, and in left bundle branch block a wide predominantly negative QRS complex will be seen
Accessory pathway	The ventricles are activated by both the AV node and an accessory pathway	The PR interval will be short due to early ventricular activation from the accessory pathway and the QRS complex will be abnormal because of cell-to-cell activation of ventricular tissue
Hyperkalemia	High levels of potassium lead to slow ventricular depolarization	Prominent T waves will often be observed
Ventricular rhythm	A site within the ventricles drives ventricular depolarization	P waves will not precede the QRS complexes
Ventricular paced rhythm	Similar to the ventricular rhythm	A pacing artifact will be observed

be observed in any condition that leads to increased space between the heart and the electrode (increased lungs due to chronic obstructive lung disease, or fat due to obesity), or any condition that leads to loss of cardiac myocytes (replacement of myocytes associated with amyloidosis).

Q waves?

An initial negative deflection in the QRS complex (Q wave) is normally seen in several leads. The QRS complex in aVR is usually negative because ventricular depolarization travels away from this lead. Small Q waves due to septal depolarization are often seen in the lateral precordial leads. Finally, small q waves (less than < 0.04 s wide and less than 0.1 mV deep) should generally not be considered abnormal.

Table 3: Common causes for left axis deviation

Condition	Pathophysiologic Basis	ECG findings
Left anterior fascicular block	Block of the left anterior fascicle leads to initial ventricular activation via the left posterior fascicle and then late activation of the ventricles	Small q waves in I and aVL lead to qR complexes
Left ventricular hypertrophy	Mass effect of the left ventricle leads to predominant leftward activation	Prominent voltages in the precordial leads. Associated STT changes
Inferior wall myocardial infarction	Relative loss of ventricular depolarization in the inferior wall leads to more prominent leftward and lateral ventricular depolarization	Pathologic Q waves will be seen in the inferior leads

Table 4: Common causes for right axis deviation

Condition	Pathophysiologic Basis	ECG findings
Left posterior fascicular block	Block of the left posterior fascicle leads to initial ventricular activation via the left anterior fascicle and then late activation of the inferior and posterior left ventricle	Small q waves in II, III, and aVF lead to qR complexes
Right ventricular hypertrophy	Mass effect of the right ventricle leads to predominant rightward activation	Prominent voltages in the right precordial leads, particularly lead V_1, Associated STT changes
Lateral wall myocardial infarction	Relative loss of ventricular depolarization in the lateral wall leads to more prominent rightward and inferior ventricular depolarization	Pathologic Q waves will be seen in the lateral leads

With these exceptions in mind, Q waves represent a larger than expected initial negative deflection. By far the most common cause of an abnormal Q wave is an old myocardial infarction. A Q wave is seen on the ECG because the scar related to the myocardial infarction leads to a relative loss of electrical force in the affected area. However, Q waves can be seen in a number of conditions and are often a relatively nonspecific finding. In addition, with the advent of more aggressive treatment of myocardial infarction focused on restoring flow, the absence of a dense scar is making Q waves less commonly observed.

Assess ventricular repolarization

The ST segment and T waves represent ventricular repolarization. The ST segment should be isoelectric and the T wave should generally be "in the same direction" as the QRS complex. Any deviation of the ST segment should arouse clinical suspicion, particularly in the symptomatic patient (chest pain, shortness of breath). Although changes in T waves can be observed in a great number of conditions (Appendix, Table 4), the presence of abnormal T waves in the symptomatic patient always warrants careful clinical evaluation.

In addition to the morphology of the ST segment and T wave, the QT interval as a measure of the duration of the ventricular action potential should be assessed. A prolonged QT interval suggests that there is a delay in ventricular repolarization—a prolonged plateau phase. Causes for a prolonged QT interval were discussed in Chapter 6, but in general can be due to genetic or acquired (metabolic, drug) causes.

ECG reading "Zen"

Components of the preceding section can be found in just about any ECG book and are the fundamentals that all beginning ECG students should assess. However, experienced clinicians know that the ECG is simply one tool for taking care of an individual patient, to be taken in context with history and physical examination findings and other initial diagnostic tests. As ECG readers become more experienced, a more practical "urgent vs. less urgent" approach emerges.

"Urgent"

Some findings on the ECG allow the clinician to quickly triage a patient and identify some potentially life-threatening conditions that will have an immediate impact on patient management. The first critical finding is an accurate assessment of the heart rate. The cause of a slow heart rate (sinus node dysfunction or atrioventricular block) can be immediately identified. When a fast heart rate is present, the ECG identifies whether ventricular activation is normal or abnormal. Wide complex tachycardia is due to rapid abnormal ventricular activation and can be life-threatening.

Other than heart rate, the other urgent finding is the presence of ST segment deviation from baseline. In particular, ST segment elevation suggests the presence

of a myocardial infarction due to a completely occluded coronary artery. Immediate restoration of blood flow by removing the occlusion through mechanical means (angioplasty) or chemical means (thrombolysis) is critical to minimize the amount of damage sustained by the heart.

"Less urgent"

In this situation, although an important problem could be present, the ECG is not "screaming" for attention. Although the patient can be critically ill from sepsis or other life-threatening diseases, the ECG findings alone do not identify conditions that need to be immediately addressed. The ECG remains an important supplementary diagnostic tool and several questions should be asked in the ECG analysis.

Rhythm

1. Is the sinus node "driving the heart?" As discussed above, are the P waves upright in II, and is every P wave followed by a QRS complex?
2. Are any irregular heart beats present? Are premature ventricular or premature supraventricular complexes present? Are there any unexplained pauses or other rhythm irregularities?

QRS and T wave morphology

1. Is ventricular activation normal? Is the QRS complex narrow or wide? Is the QRS axis in the normal range (downward and to the left), and are normal QRS complexes noted (S wave in V_1, R wave in V_6, no Q waves are present)?
2. Are there subtle deviations from baseline in the ST segment?
3. Are the T waves normally shaped and in the "expected" direction (the same direction as the QRS complex)?
4. Is the repolarization duration normal (QT interval less than 0.450 s)?
5. Is left ventricular (prominent QRS complexes, STT changes, left atrial abnormality) or right ventricular (prominent R in V_1, right axis deviation) hypertrophy present?

Principle rule: Always consider the ECG in the context of the clinical situation. How do the ECG findings fit into the overall clinical picture and complaints of this patient?

Summary

It is important to develop a systematic approach to ECG reading, with the understanding that this approach may evolve and become partially subconscious with time. The ECG is an important initial diagnostic cardiac test that is easily obtained and can quickly identify certain life-threatening conditions.

Chapter 15
Analyzing ECGs: Putting it together with case studies

This chapter provides a series of cases to help integrate the principles from the preceding chapters and illustrate how ECG interpretation can be used in clinical management. The differential diagnosis for specific ECG findings will be emphasized.

Case 1

Introduction

John Martini is a 34-year-old man who has noted worsening shortness of breath with exertion. His ECG is shown in Figure 1.

Discussion:

Using a "first glance, identify any urgent problems" approach, the heart rate is normal: approximately 60 beats per minute with five large boxes between the QRS complexes, every P wave appears to be followed by a QRS complex. Inspection of the ST segments demonstrates 1 mm ST segment elevation in the anterior leads with T wave inversion. However, there are no reciprocal ST segment changes. Q waves are present in the inferior leads, but there is no accompanying ST segment elevation in these leads. Since the patient is not complaining of chest pain, taking all the clinical information together, the ECG is identified as "less urgent" and can be carefully analyzed without initiating any specific therapeutic interventions.

Although the sinus node is initiating activation of the atria, leading to a negative P wave in aVR and a positive P wave in II, the P wave in V_1 is completely negative—with the negative component greater than 1 "little box" wide and deep. This finding is diagnostic for left atrial abnormality. Remember that the left atrium is activated after the right atrium, so the latter part of the P wave generally reflects left atrial depolarization.

The QRS complex has several abnormal findings, including left axis deviation, abnormal Q waves in the inferior and lateral leads, and a prominent R wave in lead V_1. The differential diagnoses for left axis deviation, Q waves, and a prominent

F. Kusumoto, *ECG Interpretation: From Pathophysiology to Clinical Application*,
DOI 10.1007/978-0-387-88880-4_15, © Springer Science+Business Media, LLC 2009

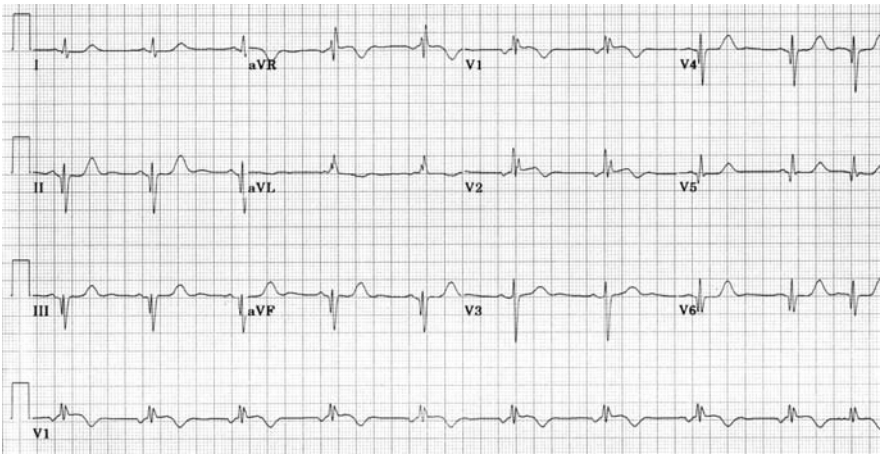


Figure 1: ECG for Case 1.

R wave in lead V_1 are provided in Table 1 and Tables 1, 2, and 3 in the Appendix. The ECG reader must integrate these abnormal findings to develop the most likely unifying clinical diagnosis and additional less likely possibilities. For the sake of this discussion we will focus first on the prominent R wave in V_1. (Table 3 in the Appendix)

A prominent R wave in lead V_1 indicates an abnormal left to right direction of depolarization in the precordial plane. In newborns, the right and left ventricles are similar in size, but once the lungs expand, pulmonary circulation increases and

Table 1: Common Causes for Q waves

Cause	ECG characteristics/clues
Normal variant	Small Qs can sometimes be observed in the inferior leads and the lateral leads.
Myocardial infarction	Q waves will be present in the region of the myocardial infarction.
Fascicular block	Anterior fascicular block: Qs in I, aVL. Posterior fascicular block: Qs in III, aVF.
Hypertrophic cardiomyopathy	Qs noted inferiorly and laterally (large septum).
Dilated cardiomyopathy	Precordial Q waves can be observed.
Infiltrative disease (amyloid, sarcoid)	Usually with anterior Q waves and low voltages, particularly in the frontal leads.
Wolff-Parkinson-White Syndrome	Q waves will be observed in the location where the accessory pathway inserts into ventricular tissue. The PR interval will be short.

pulmonary pressures decrease significantly compared to systemic pressures. For this reason, as we mature the left ventricle becomes thicker and larger relative to the right ventricle; and the relatively prominent R wave in the newborn gives way to the more normal adult pattern, with a small r wave due to septal activation and a deep S wave due to greater left ventricular depolarization compared to right ventricular depolarization. Figure 2 shows an ECG from a newborn. Notice the prominent R wave in V_1. Several other characteristic ECG findings are present. The normal heart rate for a newborn can exceed 200 bpm. Since the newborn heart is smaller, the QRS duration is significantly shorter.

In adults the most common cause of a prominent R wave in lead V_1 is right bundle branch block. In right bundle branch block, delayed and unopposed right ventricular activation leads to the prominent R wave in the terminal portion of the QRS. In this case, the QRS complex is slightly wide (0.12 s), but the initial R wave is more prominent than the terminal R wave. This would be atypical for right bundle branch block; and remember that since initial septal activation is normal in isolated right bundle branch block, Q waves are generally not observed.

Another cause for a prominent R wave in V_1 would be right ventricular hypertrophy. Right ventricular hypertrophy will often be associated with right axis deviation. In this case, accompanying left axis deviation makes right ventricular hypertrophy extremely unlikely.

Dextrocardia, in which the heart is located in the right chest, can also be associated with a prominent R wave in V_1. An example of a patient with dextrocardia is shown in Figure 3. In dextrocardia a normally positioned lead V_6 will be almost completely negative. Dextrocardia is most commonly associated with situs inversus

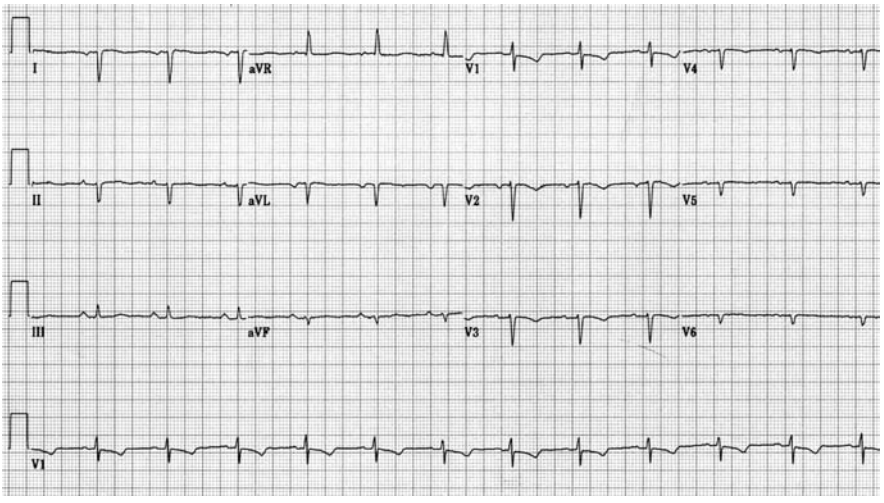


Figure 2: A normal ECG from a 2-day old boy. The prominent R wave in V_1 is a normal finding.

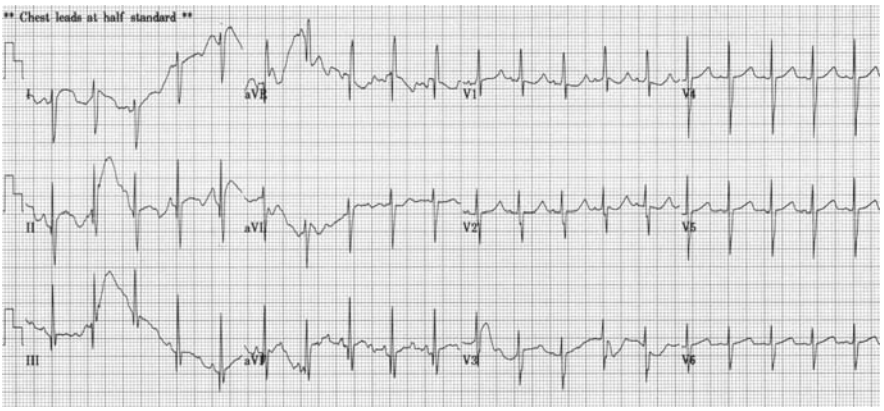


Figure 3: ECG from a patient with dextrocardia. A prominent R wave is noted in V_1 but since the heart is in the right chest, V_6 has a QS configuration and the frontal QRS axis is directed to the right. The P wave depolarization is directed from left to right since the sinus node is on the left.

(visceral organs are also on opposite sides, liver and vena cava on the left) and the sinus node is located in the left chest and atrial activation occurs from left to right. Again taken together, the ECG findings suggest that dextrocardia is not present.

A left-sided atrioventricular accessory pathway in the Wolff-Parkinson-White Syndrome can also be associated with a prominent R wave in V_1 (see case #7). Since the left ventricle is located "behind" the right ventricle, a left-sided accessory pathway leads to early left to right depolarization of the ventricles. In the Wolff-Parkinson-White Syndrome inferior Q waves can be seen in the inferior or lateral leads due to abnormal initial left ventricular activation. However, in the Wolff-Parkinson-White Syndrome, an abnormally short PR interval will be observed; the normal PR interval in this case makes Wolff-Parkinson-White Syndrome very unlikely.

A "posterior" myocardial infarction can also be associated with a prominent R wave in V_1. Remember from Chapter 7 that the prominent R wave actually represents an abnormal Q wave because of abnormal depolarization. "Posterior " myocardial infarctions are generally associated with Q waves in the inferior leads, due to an accompanying inferior myocardial infarction. In addition to a prominent R wave, the T wave is usually upright in V_1 in a "posterior" infarction; the inverted T wave in this case would not be typical but can be seen in some cases of "posterior" myocardial infarction. Similarly, left atrial abnormality can be observed in some patients with a "posterior" myocardial infarction, but it does not sway the diagnosis in any direction.

A genetic condition due to mutations of the sarcomere (filaments or associated proteins) can lead to abnormal hypertrophy of the heart and a condition called hypertrophic cardiomyopathy. In some cases of hypertrophic cardiomyopathy, thickening

is most prominent in the left ventricular septum and a prominent R wave in V_1 and Q waves in the inferolateral leads can be observed due to the abnormally large depolarization of the septum. Left atrial abnormality is commonly observed due to left atrial enlargement and thickening because of left atrial contraction into a thick and stiff left ventricle. T wave inversion and ST depression can be observed due to abnormal left ventricular repolarization.

To summarize, by ECG analysis, hypertrophic cardiomyopathy would be the most likely diagnosis that unifies most of the ECG findings, but atypical presentations of a "posterior" myocardial infarction or right bundle branch block would also have to be "in the running."

Conclusion

The simplest tool for differentiating between the diagnoses suggested by the ECG is transthoracic echocardiography. With this technique, ultrasound signals are used to reconstruct a realtime image of the heart. In this case the echocardiogram confirmed the presence of hypertrophic cardiomyopathy with severe thickening of the interventricular septum, but with all ventricular walls thickening and contracting normally. If simple right bundle branch block was responsible for the ECG findings, normal left ventricular function would have been observed; and if a posterior wall myocardial infarction was present, abnormal thickening and movement of specific walls of the left ventricle would have been observed.

Case 2

Introduction

Wynn Muse is a 54-year-old man who comes to the emergency room because of chest pain for the past two hours. His ECG is shown in Figure 4.

Discussion

In this case, using a first glance method, the heart rate is borderline low at 50 beats per minute, and atrioventricular conduction is not normal, with P waves not associated with QRS complexes. This can best be appreciated by noting that there are "unexpected" deflections in the QRS complexes, ST segment, and T wave that represent P waves that are not associated with the QRS complexes. Finally, prominent ST segment elevation in the inferior leads suggestive of an acute inferior wall myocardial infarction is present. The ECG provides immediate information that an "urgent" condition is present based on heart rhythm and the presence of ST segment changes. Immediate preparations should be made to transport the patient

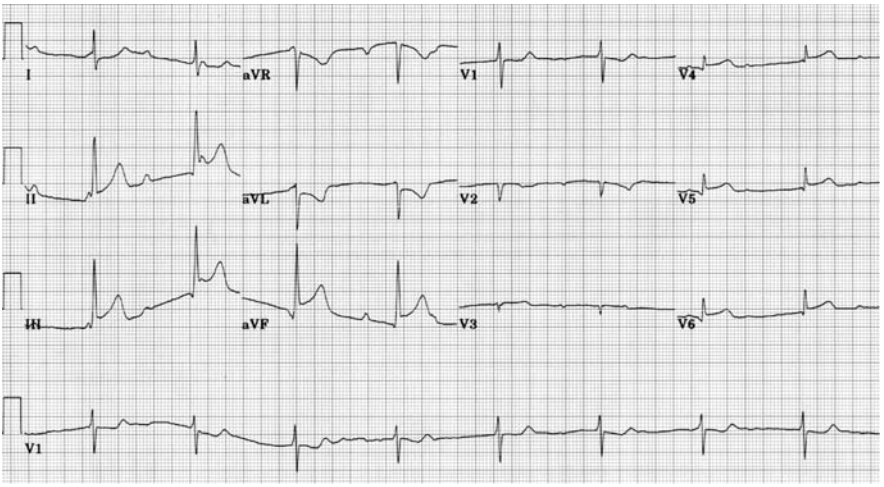


Figure 4: ECG for Case 2.

to the cardiac catheterization laboratory so that a definitive diagnosis can be made and, if possible and appropriate, blood flow restored to the region at risk.

Once the clinical issues have been addressed the ECG should be carefully evaluated. Since the P waves are negative in aVR and positive in II, the sinus node is initiating atrial depolarization. However, in this case the P waves are dissociated from the QRS complex and 3° AV block is present. AV block is observed in approximately 3–7% of patients with an inferior wall myocardial infarction. The artery that supplies the AV node is the first branch of the posterior descending artery. In most people the posterior descending artery is a branch of the right coronary artery (right dominant). The incidence of AV block in the setting of an inferior wall myocardial infarction has a bimodal distribution, due to two underlying mechanisms. In the first hour of an inferior wall myocardial infarction, an initial peak in the incidence of AV block appears to be due to increased parasympathetic tone. The incidence of AV block has a second rise approximately 24 h after the onset of symptoms that appears to be due to tissue edema. AV block in the setting of an inferior wall myocardial infarction can persist for several days and even weeks, but generally resolves without requiring pacing therapy. In this case AV block does not signify a worse prognosis. In contrast, AV block is very uncommonly observed in an anterior wall myocardial infarction, and if present suggests extensive myocardial damage and a far worse prognosis.

In this case ST segment elevation is observed in the inferior and lateral leads, suggesting that a large area of myocardium is at risk. In addition, notice that reciprocal ST segment depression is not observed in leads V_1 and V_2, suggesting the presence of an accompanying right ventricular myocardial infarction. The reciprocal ST depression in aVL also suggests that the right coronary artery is the "culprit" artery.

Taken together, the ECG findings suggest that the patient has an occlusion in the proximal portion of the right coronary artery, before the acute marginal artery that supplies the right ventricle. The right coronary artery probably has extensive branches on the posterior and lateral walls (posterolateral branches). Clinically, the ECG suggests that a large area of myocardium is at risk, and that coronary artery flow must be reestablished emergently, either mechanically (angioplasty) or chemically (thrombolysis).

Causes for ST segment elevation other than myocardial infarction were reviewed in Chapter 8. The distinction between pericarditis and myocardial infarction bears special emphasis (Chapter 8, Tables 1 and 2). Although inferolateral ST segment elevation can be observed in pericarditis, the presence of reciprocal ST segment depression in leads I and aVL essentially rules out pericarditis as a diagnosis in this case. Pericarditis is not associated with AV block.

Conclusion

The patient was given aspirin and taken immediately to the cardiac catheterization laboratory and underwent angioplasty and stenting of a totally occluded right coronary artery. His AV block resolved while he was in the cardiac catheterization laboratory. He recovered with a follow-up echocardiogram showing only mildly abnormal contraction (hypokinesis) of the inferior wall but otherwise normal left ventricular function.

Case 3

Introduction

Henry Knox is a 64-year-old man who has complained of chest pain and shortness of breath for the past several hours. His initial ECG is shown in Figure 5.

Discussion

The ECG shows a heart rate within the normal range (60–100 bpm) with 5 "large boxes" between the QRS complexes (heart rate of approximately 60 bpm). Although there is one irregular beat, every P wave appears to be followed by a QRS complex. Prominent ST segment elevation is noted in leads V_1 to V_4, and the ECG is classified as "urgent" due to the presence of an acute anterior wall myocardial infarction. As in the previous case, treatment aimed at reestablishing flow through the obstructed coronary artery should be initiated.

Careful inspection of the ECG reveals that the single irregular beat is due to a premature atrial complex that probably arises from an area near the sinus node,

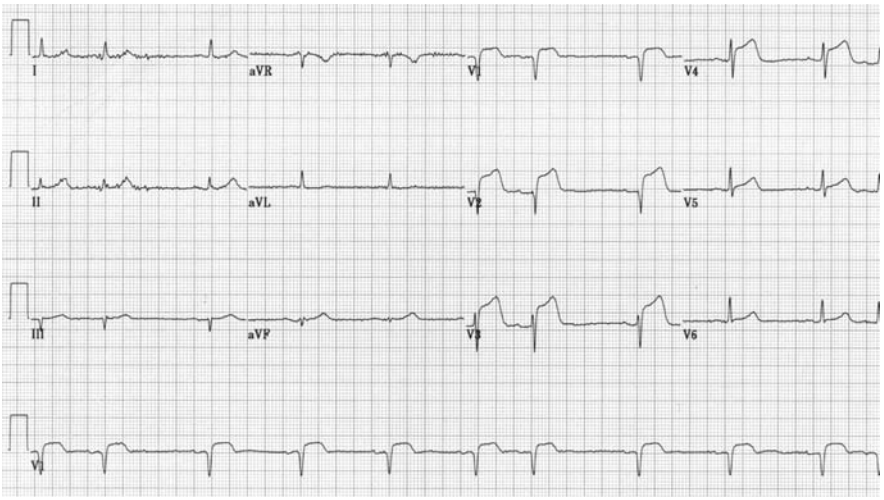


Figure 5: ECG for Case 3.

since the P wave of the premature atrial complex is similar in morphology to the P wave from normal sinus rhythm. In this case atrioventricular conduction is normal, with each P wave (even the premature atrial complex) followed by a QRS complex. Notice that the PR interval for the premature beat is slightly longer due to partial refractoriness and slower conduction in the AV node.

Prominent ST segment elevation in V_1 through V_4 is consistent with an occluded left anterior descending artery. The absence of ST segment elevation in leads I and aVL suggest that the occlusion is just distal to the first diagonal artery.

ST segment elevation in a specific coronary artery distribution (anterior: left anterior descending; lateral: circumflex; inferior: right coronary) should always be considered a very worrisome sign for myocardial infarction. As in the last case, several important points differentiating myocardial infarction from pericarditis will be emphasized. Pericarditis is usually associated with diffuse anterior, inferior, and lateral ST segment elevation, with ST segment depression in aVR. Pericarditis is not associated with abnormal Q waves; in this case, loss of the septal r wave in V_1 probably represents an abnormal Q wave.

Conclusion

The patient was given a thrombolytic drug, a tissue plasminogen activator that acts to dissolve the occlusive thrombus. One hour after the drug is initiated, the patient complains of less pain and the ECG in Figure 6 is obtained. Notice that the ST segment elevation remains and is even more prominent. Notice too that a P wave does not precede the QRS complexes. In this case an accelerated

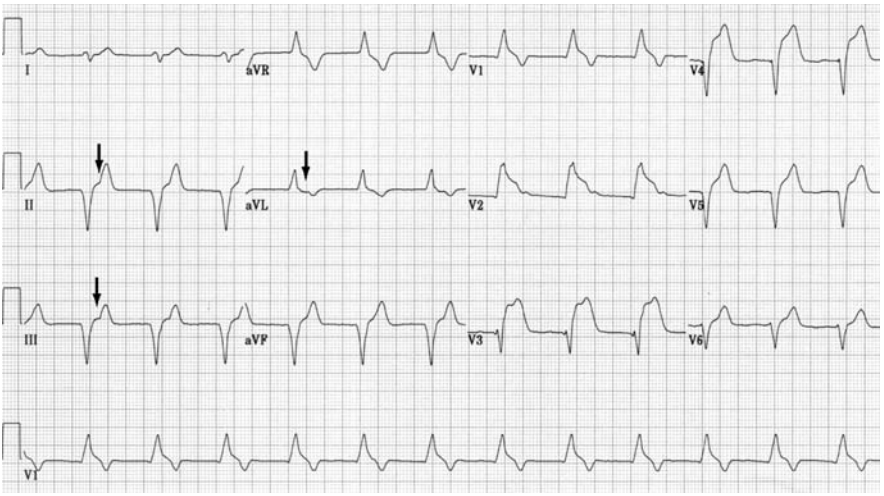


Figure 6: The same patient when his chest pain suddenly improves. The patient is no longer in sinus rhythm. Ventricular activity is not preceded by a P wave. In fact, retrograde P waves are observed within the ST segment (*arrows*). This is an accelerated idioventricular rhythm that usually arises from automaticity from the infracted area that results in inferior and anterior Q waves.

idioventricular rhythm is present. Accelerated idioventricular rhythm is usually due to automaticity in ventricular tissue. It is called accelerated because the normal ventricular pacemaker rate is usually less than 30–40 bpm. Acclerated idioventricular rhythm is often observed when coronary artery reperfusion is established during an ST segment elevation myocardial infarction. It can also be seen with digoxin use. In general, patients remain hemodynamically stable and no specific therapy is required. After several minutes the accelerated idioventricular rhythm resolves and the ECG in Figure 7 is recorded. The ST segment elevation has decreased substantially, although Q waves are now present in leads V_1 through V_3. Sinus rhythm has returned, but a single ventricular beat is observed (notice that the first QRS complex is wide and not preceded by a P wave) and a single junctional beat is observed (the second QRS complex is slightly wider than the following sinus rhythm associated QRS complexes, and the PR interval is too short for the P wave to be responsible for ventricular activation).

Case 4

Introduction

Ashley Lewis is a 59-year-old woman who has had episodes of intermittent lightheadedness for the past two weeks. Her ECG is shown in Figure 8.

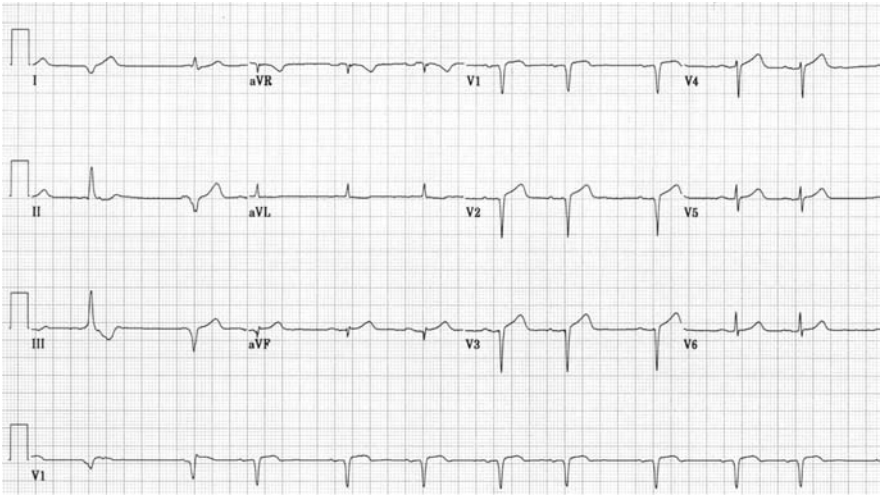


Figure 7: ECG after the accelerated idioventricular rhythm resolves. The ST segment is less elevated in the anterior leads, and Q waves have developed in V_1 through V_3.

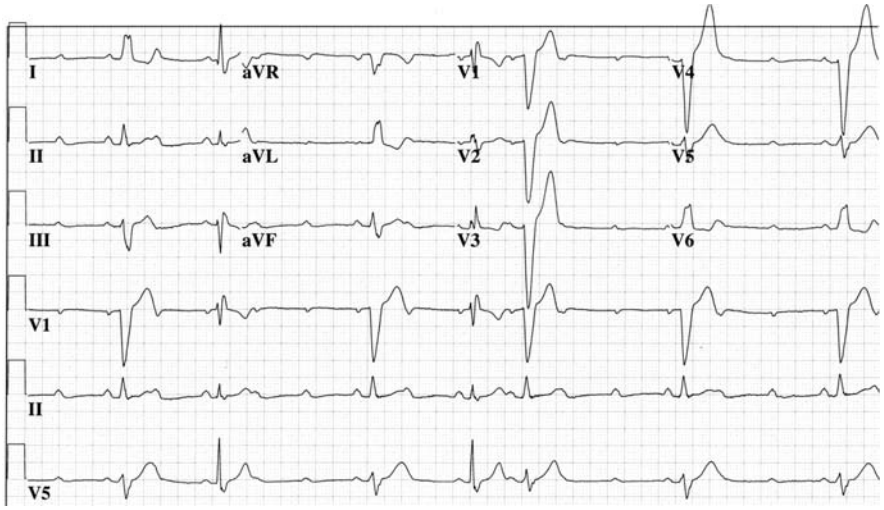


Figure 8: ECG for Case 4.

Discussion

The ECG shows high grade atrioventricular block with alternating block in both the right bundle and left bundle. Figure 9 shows a rhythm strip with a diagram of conduction. Notice that some P waves are not associated with a QRS due to

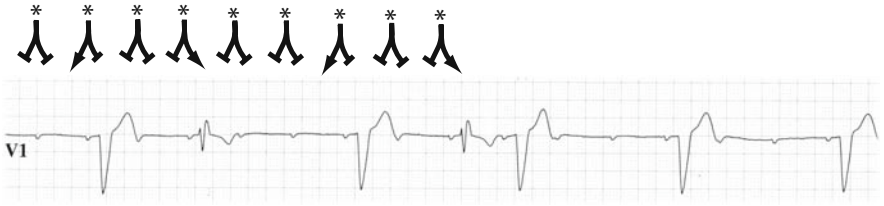


Figure 9: Rhythm strip with the conduction diagram for Case 4. Notice that some P waves show block in both the right and left bundle, some P waves in the right bundle, and other P waves in the left bundle.

AV block, some P waves conduct only down the left bundle (QRS with a right bundle branch block pattern), and other P waves conduct only down the right bundle (QRS with a left bundle branch block pattern). This is a situation where patients can suddenly develop asystole due to prolonged episodes of heart block. Since the block is within the bundles, the patient is dependent on the pacemaker activity of ventricular cells, which is very unreliable and often not present.

Conclusion

The patient received a dual chamber permanent pacemaker with complete resolution of her symptoms.

Case 5

Introduction

Mary Booth is a 66-year-old woman who underwent abdominal surgery several days ago. She has been doing well after her surgery, but this morning she developed an irregular heart rate that has been associated with mild shortness of breath. A rhythm strip is shown in Figure 10.

Discussion

The ECG shows an irregular rhythm with a relatively normal ventricular rate: QRS complexes separated by 3–5 boxes with an average heart rate in the 70s. P waves are not seen and the QRS complexes are separated by a "sawtooth" pattern with no isoelectric period. The ST segments and T waves are not significantly abnormal, so despite the abnormal heart rhythm the ECG can be classified as "less urgent," and once it is confirmed that the patient is in stable clinical condition the ECG can be carefully evaluated.

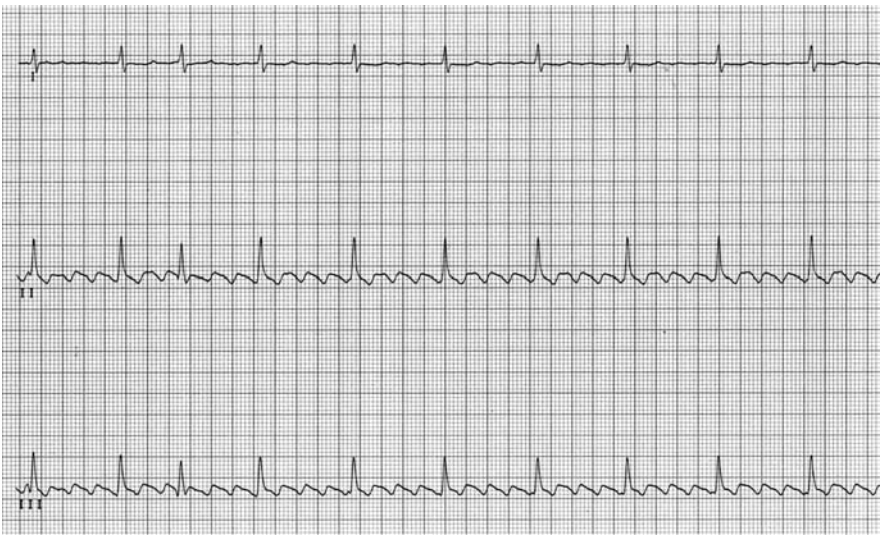


Figure 10: ECG for Case 5.

The patient is not in sinus rhythm. Rather the atria are activated continuously at a rate of approximately 300 bpm—approximately one "large box" between successive peaks. This is an example of "typical" atrial flutter. The term atrial flutter is generally used to describe any rapid atrial rhythm due to a reentrant circuit (a rapid atrial rhythm due to abnormal automaticity is often called atrial tachycardia; in practice it is very difficult to differentiate the mechanism of an arrhythmia by ECG). Although in theory there could be a number of possible circuits within the atria, a reentrant circuit that circulates around the tricuspid valve is by far the most common and is called "typical" atrial flutter. "Typical" atrial flutter is also often called isthmus dependent atrial flutter because the "slow zone" of the reentrant circuit is formed by atrial tissue between the tricuspid valve and the inferior vena cava (Figure 11). Remember from Chapter 11 that reentry requires a protected channel that is bounded on either side by tissue or structures that do not allow conduction across the side boundaries of the channel. As "holes," the tricuspid valve and the vena cava form perfect boundaries to facilitate the development of reentry. The usual atrial flutter circuit travels through the isthmus from the lateral wall of the right atrium to the interatrial septum, then travels "low-high" up the interatrial septum and "high-low" down the lateral wall of the right atrium. The flutter waves are generally negative in the inferior leads because the left atrium (which has larger mass than the right atrium) is activated passively "low-high." However, since the circuit of isthmus dependent atrial flutter is quite large, no quiescent period of atrial activation is observed, and consequently no true isoelectric period is present.

Notice that although the atria are activated 300 times per minute, ventricular depolarization is significantly slower. The AV node has slow conduction properties

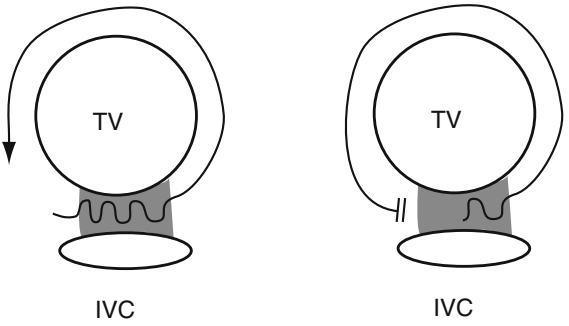


Figure 11: In typical atrial flutter a reentrant circuit travels around the tricuspid valve. The critical isthmus is bounded by the tricuspid valve and the inferior vena cava. Atrial flutter can terminate if the reentrant circuit encounters unexcitable tissue anywhere along the circuit. The most likely place for block to occur is somewhere within or at the entrance of the tricuspid-inferior vena cava isthmus.

and a relatively long refractory period. In this case, usually every fourth atrial depolarization conducts through the AV node and leads to ventricular depolarization: 4:1 "block," although sometimes conduction occurs in a 3:1 ratio or a 5:1 ratio. It is apparent that the AV node acts as a regulator that limits the ventricular response during atrial arrhythmias.

Conclusion

Atrial flutter is not a life-threatening arrhythmia. However, it can lead to hemodynamic inefficiency due to rapid ventricular rates or loss of normal atrioventricular synchrony.

In some cases medications are given in an attempt to terminate the abnormal rhythm. Antiarrhythmic medications target ion channels. Changing ion channel permeability will change the action potential shape. Medications that block Na^+ channels will cause a decrease in the phase 0 slope (Figure 12). Na^+ channel blockers can terminate reentrant arrhythmias by increasing the likelihood that the reentrant wave of depolarization will encounter a region of unexcitable tissue. Medications that block K^+ channels increase the action potential duration. The reentrant circuit will terminate if the wave of depolarization encounters tissue that is still in the absolute refractory period.

In this case the patient is given ibutilide, a medication that blocks K^+ channels and increases atrial refractoriness. A subsequent ECG is shown in Figure 13. Notice that the atrial flutter cycle lengths have increased. This is probably due to increased refractoriness causing a decrease in conduction velocity (by encroaching on the relative refractory period). The patient converts to sinus rhythm and the final ECG is shown in Figure 14. Notice that the QT interval is prolonged. The drug

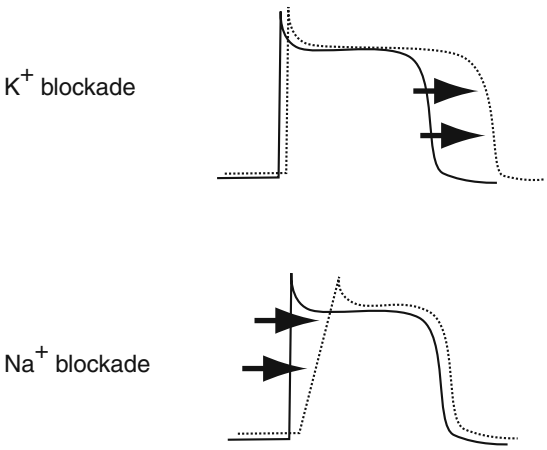


Figure 12: The effects of antiarrhythmic medications on the action potential. Na^+ channel blockers make the phase 0 upstroke less steep. K^+ channel blockers prolong the action potential duration.

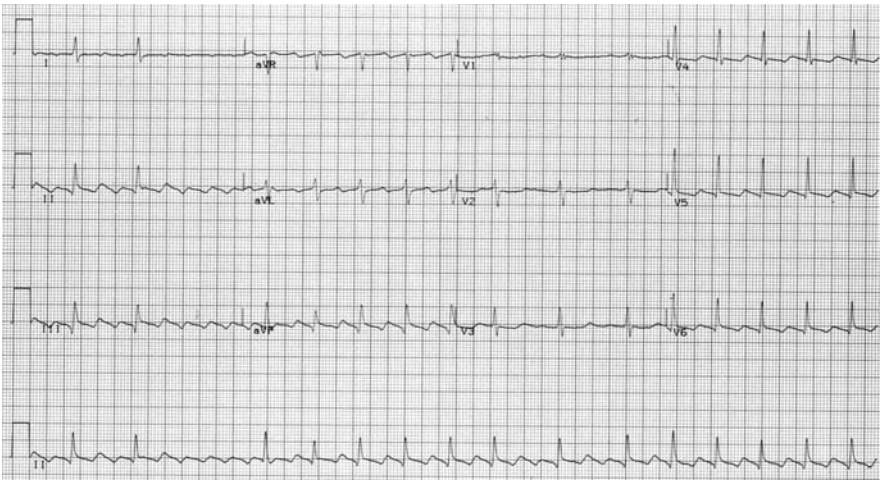


Figure 13: Treatment with the K^+ channel blocker causes slowing of the atrial flutter rate (Flutter waves are farther a part). The Ventricular rate (distance between QRS complexes) may be faster.

exerts its beneficial effects on atrial tissue, but its effects are not tissue selective, and concomitant ventricular action potential prolongation leads to a prolonged QT interval. Drugs that act to block K^+ channels all pose a risk for torsade de pointes (Chapter 6).

Antiarrhythmic drugs that block K^+ channels increase the phase 2 plateau duration and prolong the QT interval. In contrast, antiarrhythmic medications that

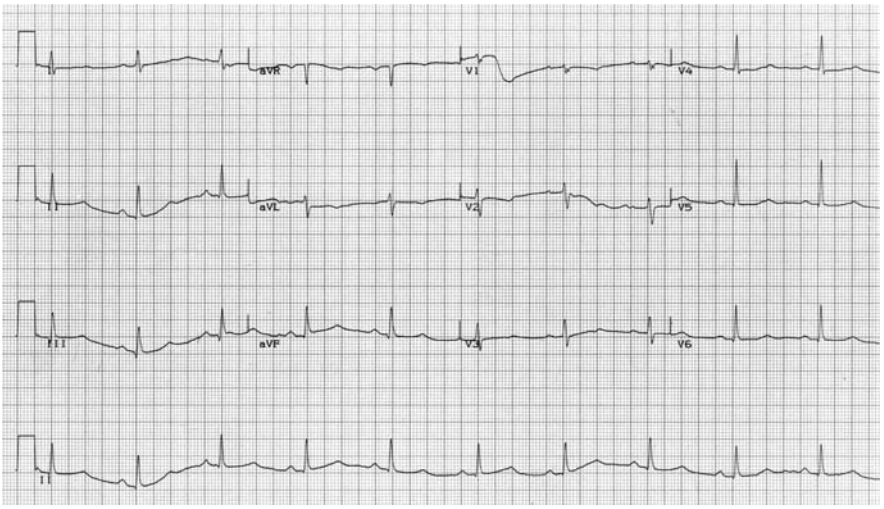


Figure 14: Once the atrial flutter has resolved, prolongation of the QT interval is noted. Antiarrhythmic medications do not have tissue selective effects. Changes in ion permeability will be observed in both atrial and ventricular tissue.

block Na^+ channels decrease the phase 0 slope and prolong the QRS duration. In Figure 15, the ECGs from a patient that receives the Na^+ channel blocker flecainide are shown. When the patient is given flecainide the QRS duration increases significantly. As a general rule, if an antiarrhythmic medication causes significant changes in the ECG-significant prolongation of the QRS interval or the QT interval, the patient will have a higher risk for the development of additional abnormal arrhythmias and the medication should be stopped.

Case 6

Introduction

Mike Martin is a 76-year-old man who comes to the emergency room with sudden onset of lightheadedness 2 hours ago. He tells you that he had a heart attack 4 years ago. His ECG is shown in Figure 16.

Discussion

The ECG shows a rapid wide complex rhythm and would be classified as “urgent". The axis is normal and does not help in distinguishing ventricular tachycardia from supraventricular tachycardia with aberrancy. Lead V_1 has a mainly

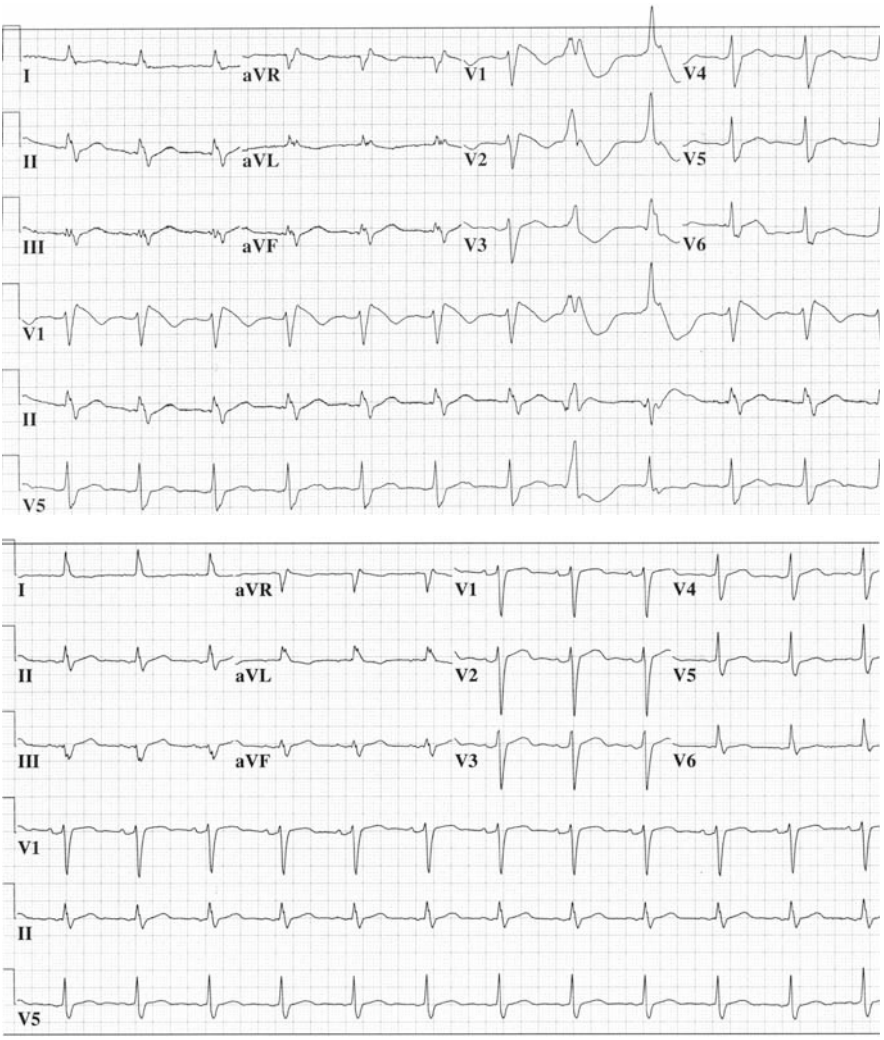


Figure 15: *Top*: ECG from a patient receiving the Na^+ channel blocker flecainide. Notice that the QRS interval is significantly prolonged. The drug is stopped and a follow-up ECG is shown (*bottom*). The QRS interval has normalized after the medication has been stopped.

positive complex with small negative "s" wave. An Rs pattern in V_1 is consistent with ventricular tachycardia. Hopefully, the student has already noted that the patient has obvious AV dissociation noted in V_1, thus confirming the diagnosis of ventricular tachycardia.

He undergoes synchronized cardioversion. His ECG after cardioversion is shown in Figure 17. He has obvious inferior Q waves and a prominent R wave with

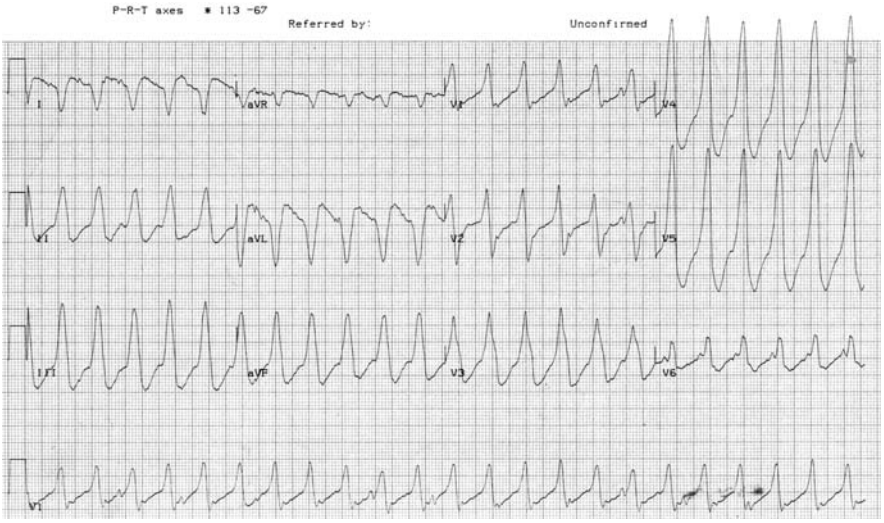


Figure 16: Case 6.

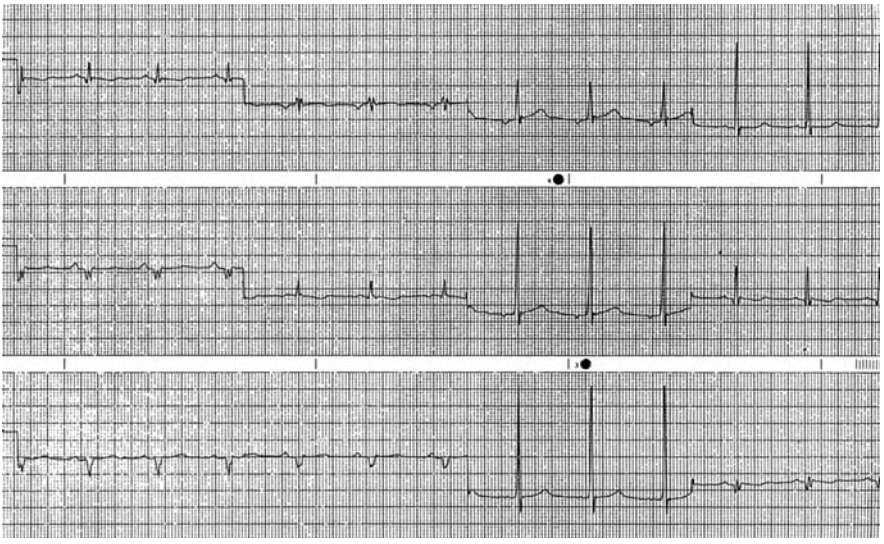


Figure 17: ECG in the patient after cardioversion. The patient has evidence of an old inferoposterior myocardial infarction with Q waves in the inferior leads and a prominent R wave in V_1. Notice that there are no acute ST changes. Patients with ventricular tachycardia most commonly do not have associated acute ischemia as a cause for ventricular tachycardia.

an upright T wave in lead V_1. These findings are consistent with an old inferior and posterior wall myocardial infarction. Patients with prior myocardial infarction have a higher likelihood of the development of ventricular tachycardia. The resultant scar can have channels of viable tissue that allow the development of reentrant circuits.

Conclusion

The patient receives an implantable cardiac defibrillator (ICD) that is designed to automatically detect a ventricular arrhythmia and deliver a shock between a specialized coil in the right ventricle and the defibrillator "can." (Chapter 17, Figure 11).

Case 7

Introduction

Jonathan Malcom is a 22-year-old man who comes into the emergency room complaining of lightheadedness and a rapid heart beat for the past hour. His ECG is shown in Figure 18.

Discussion

The ECG shows a very rapid and irregular wide complex rhythm. This triad of findings is associated with atrial fibrillation developing in a patient with the

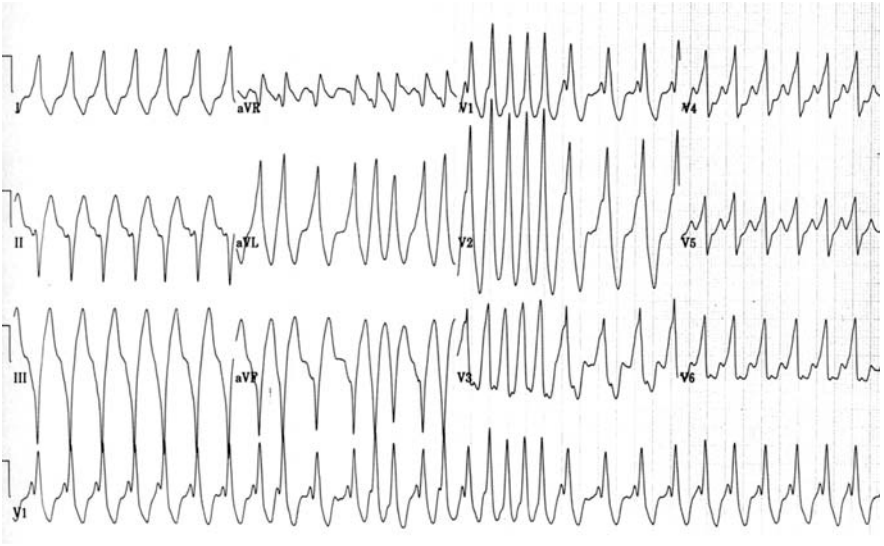


Figure 18: ECG for Case 7.

Wolff-Parkinson-White Syndrome. In the Wolff-Parkinson-White Syndrome a patient has an accessory pathway that provides an additional connection between atrial and ventricular tissue (Figure 19). The presence of an accessory pathway means that patients can develop a number of arrhythmias. The first and most common possibility is orthodromic atrioventricular reentrant tachycardia. In this case a reentrant circuit develops in which the depolarization wave travels down the AV node ("ortho" or correct direction), activates the ventricles, travels retrogradely up

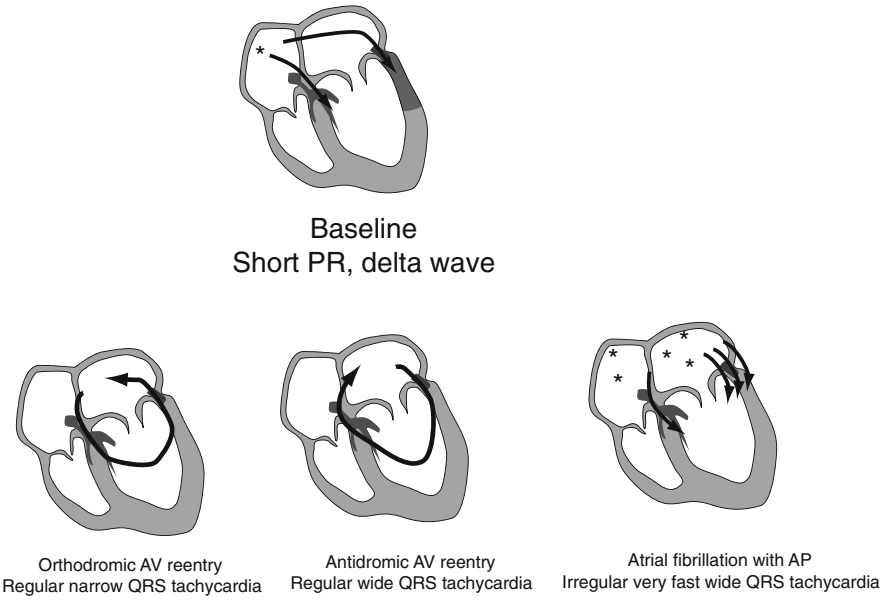


Figure 19: Schematic of the ECG findings in the Wolff-Parkinson-White Syndrome. At baseline the presence of an accessory pathway (AP) means that a portion of the ventricle is activated or preexcited by the accessory pathway. On ECG a short PR interval and a delta wave is observed. Patients with an AP can have three different types of tachycardias. In orthodromic atrioventricular (AV) reentry, the ventricles are activated via the AV node and the atria are activated by retrograde activation via the accessory pathway. This produces a regular tachycardia with a narrow QRS complex. In antidromic tachycardia, the reentrant circuit is reversed, the ventricles are activated via the AP and the atria by retrograde activation of the AV node. This produces a regular tachycardia with a wide QRS. Finally, if the patient develops an atrial arrhythmia (usually atrial fibrillation), the ventricles can be activated very rapidly via the AP and also in part by the AV node. Ventricular rates can be very rapid because the AP has fast conduction properties (the AP is usually composed of normal atrial or ventricular tissue). This leads to an irregular and very rapid wide QRS tachycardia.

the accessory pathway to activate the atria, and the circuit repeats. Since the ventricles are activated normally via the AV node, the patient has a narrow QRS tachycardia (supraventricular tachycardia). The second reentrant arrhythmia is antidromic atrioventricular reentrant tachycardia. In this case the reentrant circuit is reversed, and the ventricles are activated by the accessory pathway and retrograde activation of the atria occurs via the AV node. This tachycardia is called antidromic, for "anti," or the wrong way. In this case the patient develops a regular wide complex tachycardia. The third tachyarrhythmia associated with Wolff-Parkinson-White Syndrome is the development of atrial fibrillation or atrial flutter and rapid activation of the ventricles via both the AV node and the accessory pathway. Remember that the slow conduction properties of the AV node limit the ventricular rate in response to rapid atrial rates. However, in patients with an accessory pathway, rapid atrial activity can be quickly transmitted to the ventricle, leading to a fast irregular ventricular rate with a wide QRS complex (since the ventricles are not being activated by the His-Purkinje system). Of the three tachycardias, orthodromic tachycardia is by far the most common, followed by atrial tachycardia with anterograde activation via the accessory pathway. Antidromic tachycardia is very rare, accounting for less than 5% of arrhythmias.

The location of the accessory pathway in a patient with the Wolff-Parkinson-White Syndrome can usually be determined by examining the baseline ECG. The initial slurring of the QRS complex (usually called a delta wave) will be due to ventricular activation from the accessory pathway. If the accessory pathway connects the left atrium and left ventricle, a prominent R wave is usually noted in lead V_1. Remember that the left ventricle is behind the right ventricle so the presence of a left-sided accessory pathway means that a portion of the ventricles is activated from "back-to-front." In contrast, in a patient with a right-sided accessory pathway, Q waves will be observed in the anterior leads and the R wave will develop in lead V_5 rather than V_3 or V_4 (late precordial transition). Since the sinus node is located in the right atrium, the PR interval is shorter, and preexcitation is usually more obvious in patients with right-sided accessory pathways. Compare the ECGs from Figures 20 and 21. In Figure 20, the ECG from Mr. Malcom is shown. Notice that there is a prominent R wave in V_1 due to the presence of a left-sided accessory pathway that activates the left ventricle earlier (think of the ventricular activation pattern in right bundle branch block). In contrast, the ECG shown in Figure 21 is from a patient with a right-sided accessory pathway. In this case activation of the ventricles from right to left leads to a later precordial progression with the R wave becoming greater than the S wave in V_5 (think of the ventricular activation pattern in left bundle branch block, where the right ventricle is activated before the left ventricle). In addition, the PR interval is shorter and the QRS is wider in patients with a right-sided accessory pathway, since the accessory pathway is activated before the AV node. In contrast, in patients with a left-sided accessory pathway the AV node is activated before the accessory pathway. Since the AV node has slow conduction, the PR interval is still short due to preexcitation from the left-sided accessory pathway, but some leads do show an isoelectric portion of the PR interval.

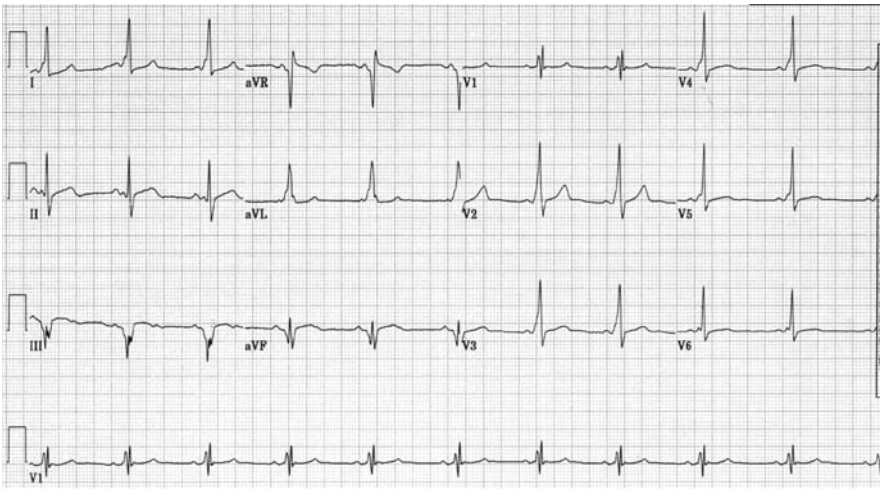


Figure 20: Mr. Malcom's ECG after his atrial fibrillation is terminated. The patient is now in sinus rhythm (the P waves are negative in aVR and positive in II). The short PR interval and the delta wave are obvious. Notice the very prominent R wave in V_1.

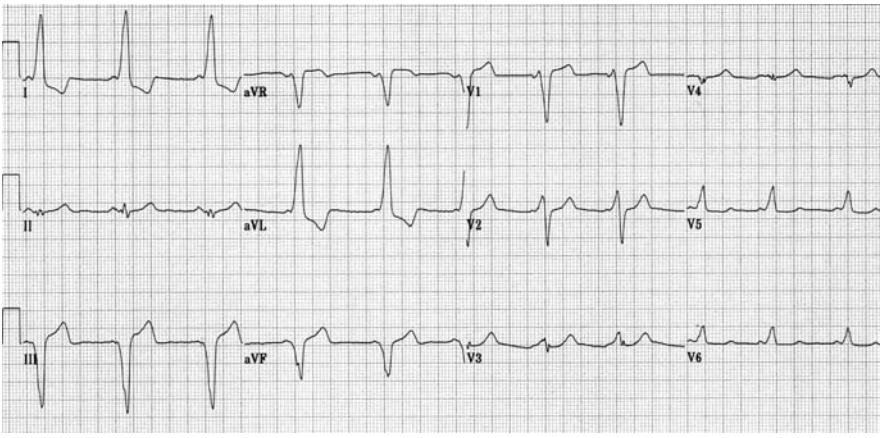


Figure 21: Patient with Wolff-Parkinson-White Syndrome with a right-sided accessory pathway. Since the right ventricle is more anterior, a new right to left force leads to late progression in the precordial plane, with the R becoming greater than the S in V_5. In addition, since the sinus node is in the right atrium, the PR interval is shorter and preexcitation is more obvious in right-sided leads.

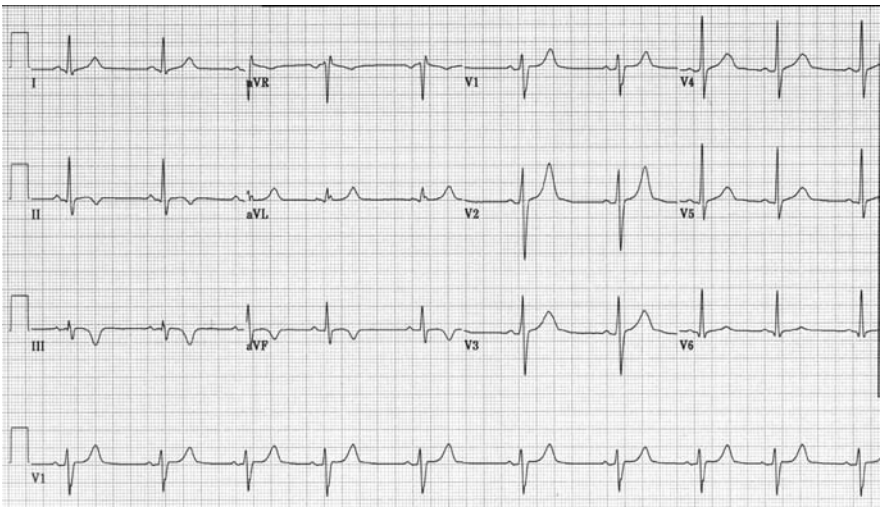


Figure 22: Mr. Malcom's ECG after an ablation procedure was performed to cauterize the accessory pathway. Notice that the PR interval is normal and the QRS complex has a normal pattern of activation.

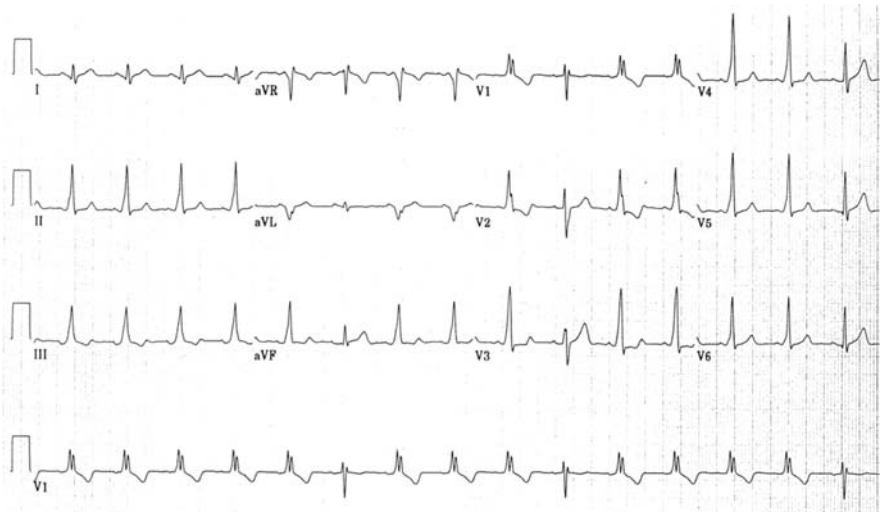


Figure 23: An ECG from a patient with intermittent preexcitation. Intermittently the accessory pathway blocks and the QRS complex normalizes and a normal PR interval is present. This suggests that the accessory pathway cannot conduct very well (longer refractoriness) and the patient is not at risk for sudden cardiac death.

Conclusion

In this case the patient received a Na^+ channel blocker that slows conduction in the accessory pathway. This leads to a slower ventricular rate and improved hemodynamic status. The atrial fibrillation terminates, and his baseline ECG is shown in Figure 20. At baseline, the presence of "preexcitation" where a portion of the ventricles is activated via an accessory pathway is obvious. As discussed above, the patient has a left-sided accessory pathway. In addition, notice that Q waves are present in leads III and aVF. Since initial activation of the left ventricle is occurring in the inferior leads, it can be inferred that the accessory pathway is located in the inferior portion of the left ventricle. By identifying the location of the accessory pathway it can be cauterized and destroyed (ablated) by a specialized catheter. Mr. Malcom's ECG after an ablation procedure is shown in Figure 22. The PR interval has normalized and the QRS now has a normal morphology.

Patients with the Wolff-Parkinson-White Syndrome are at risk for sudden cardiac death. The presence of an accessory pathway that conducts rapidly means that the ventricles can be activated rapidly, and in some cases deterioration to ventricular fibrillation can result. In patients with an accessory pathway the ECG can sometimes be useful for determining whether a patient is at risk. An ECG from another patient with an accessory pathway is shown in Figure 23. In this case some QRS complexes are narrow. This is due to intermittent block at baseline in the accessory pathway. In this patient the accessory pathway does not conduct very well—a dirt road rather than a freeway—so the likelihood of rapid ventricular activation if atrial fibrillation were to develop is very unlikely. (There is a limit to the number and speed of the cars that can travel over the dirt road.)

Chapter 16
Electrolyte disorders

The effects of some electrolyte disorders have been discussed in prior chapters: the effect of hypokalemia on the QT interval and hyperkalemia as a nonischemic cause for ST segment elevation. However, it is worthwhile to review the spectrum of ECG changes that can occur with electrolyte disorders, particularly those involving potassium and calcium.

Potassium

Hypokalemia

The main ECG finding is that hypokalemia is the development of a bifid T wave (Figure 1). ECG findings are evident in approximately 80% of patients with K^+ concentrations less than 2.7 mEq/L; ECG findings are usually not observed when the K^+ concentrations are > 3.0 mEq/L (Table 1). The second component of the T wave can be quite large and is often mistakenly identified as a U wave. In hypokalemia, the phase 3 repolarization slope becomes less steep (Figure 2). The beginning of the T wave coincides with separation of action potentials within the ventricle, as epicardial cells begin to repolarize while M cells and endocardial cells remain at the plateau phase. The first peak of the T wave appears to arise from separation between the endocardial action potential and the M cell action potential. The second peak of the T wave is reached when the epicardial layer has fully repolarized, and the T wave ends with complete repolarization of the M-cell. Thus in hypokalemia, the beginning of the T wave, the second peak of the T wave, and the end of the T wave correlate with the same cellular events under normal conditions. The only difference is a first T wave peak that becomes apparent due to separation of endocardial repolarization from M cell repolarization, since all of the action potentials are lengthened.

With severe hypokalemia, T wave inversion and ST segment depression can be observed. Similar to any condition that prolongs the QT interval, hypokalemia can be associated with development of triggered activity and torsade de pointes.

F. Kusumoto, *ECG Interpretation: From Pathophysiology to Clinical Application*,
DOI 10.1007/978-0-387-88880-4_16, © Springer Science+Business Media, LLC 2009

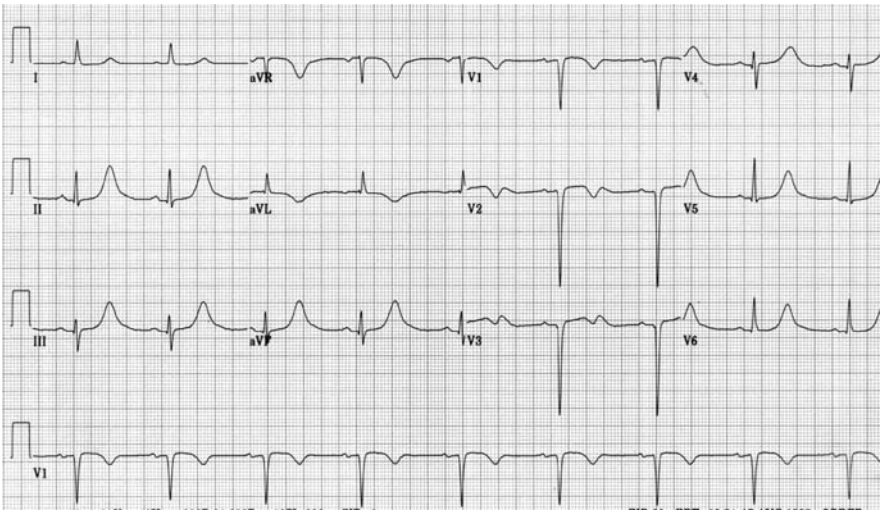


Figure 1: Hypokalemia ECG.

Table 1: ECG changes with potassium disturbances

Condition	Level (mmol/L)	ECG findings
Hypokalemia	< 2.5–3.0	• Prolongation of the QT interval • Development of bifid T waves ("pathologic U waves") • Torsade de pointes
Hyperkalemia	5.5–6.5	• T wave peaking
	> 6.5	• QRS widening • PR interval prolongation
	> 8.0	• Loss of P waves (sinoventricular rhythm) • Very wide QRS complexes • Slow ventricular rhythms • Asystole

Hyperkalemia

Hyperkalemia is associated with T wave peaking and other ECG findings. Seminal research by Antzelevitch and colleagues suggests that T wave peaking is mediated by a steeper phase 3 and decreased differences in repolarization among epicardial, endocardial, and M cells (Figures 2 and 3). T wave peaking will usually become evident when the K^+ is greater than 5.5 mEq/L. Interestingly, it has been reported that patients with left ventricular hypertrophy and inverted T waves due to abnormal repolarization can develop upright T waves with hyperkalemia ("pseudonormalization").

Hyperkalemia can cause ST segment elevation, particularly in the presence of accompanying diabetic ketoacidosis. An example of this situation was presented in the preceding chapter on noninfarct-related causes for ST segment elevation.

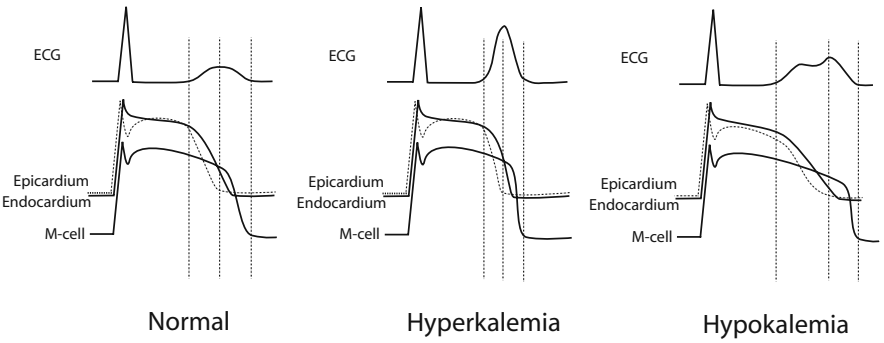


Figure 2: Cellular explanations for the genesis of the ECG findings associated with hyperkalemia and hypokalemia. In normal conditions the peak of the T wave corresponds with full repolarization of epicardial tissue, and the end of the T wave corresponds with repolarization of the M cell which has the longest action potential duration. In hyperkalemia the phase 3 repolarization becomes steeper for all three cell types and the differences in action potential duration between the cell types decreases. These changes lead to a narrower based peaked T wave. In hypokalemia, phase 3 repolarization becomes less steep, and the differences in repolarization duration among the three cell types is magnified, leading to a prolonged QT interval with a bifid T wave. The first peak coincides with the separation between endocardial and M cell repolarization. The second peak occurs when the epicardium is fully repolarized; and the third peak comes at the end of the T wave when the M cells have repolarized and all ventricular cells are at the resting state.

Hyperkalemia leads to depolarization of the membrane. Remember that at the resting state K^+ ions are at equilibrium. The actual value of the membrane potential at K^+ equilibrium is calculated by the Nernst equation (Chapter 1). In hyperkalemia extracellular K^+ is elevated, and mathematically the natural logarithm of a larger fraction is a less negative value. More simplistically, the higher K^+ concentration outside means that K^+ tends to flow into the cell against the electrical gradient until a new less negative membrane potential is reached. Partial membrane depolarization means that some cells cannot depolarize normally since there are fewer Na^+ channels in the resting state and fewer Na^+ channels available for opening. In severe cases of hyperkalemia the phase 0 upstroke becomes less steep and the QRS complex begins to widen. QRS changes can be noted when the serum K^+ concentration is greater than 6.5 mEq/L (Figure 4). The wide QRS can sometimes be differentiated from the QRS complex in left bundle branch block because deep S waves are often seen in the lateral precordial leads or I and aVL (Figure 3). With extremely high K^+ levels the QRS and T wave can merge to form a "sine wave" complex on the ECG.

Atrial tissue is more sensitive to hyperkalemia than ventricular tissue, so as K^+ concentrations become very high ($>$ 8.0 mEq/L), P waves are no longer visible. Since the sinus node still "drives" the heart, this rhythm is often called

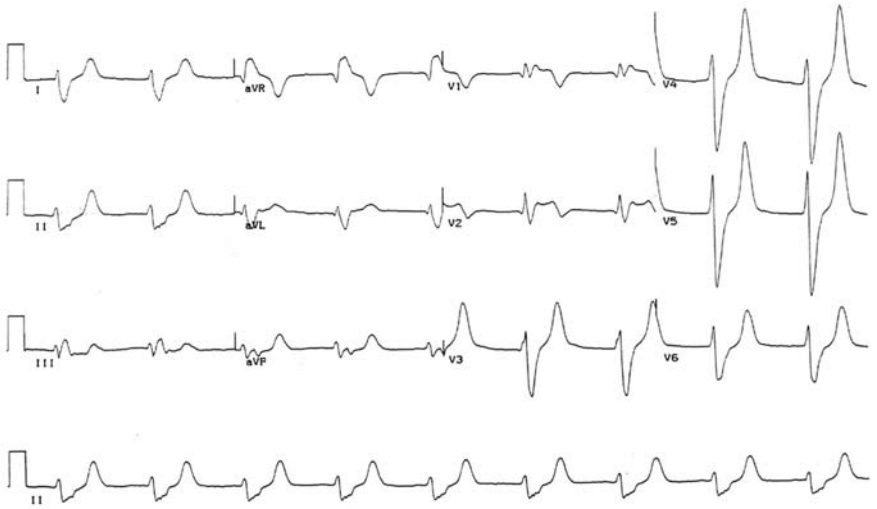


Figure 3: ECG in a patient with hyperkalemia. The QRS is wide and the T wave is prominent and relatively narrow, based in the precordial leads. Notice that the QRS complex has a deep S wave in I and aVL, which would not be seen in left bundle branch block. There are no P waves before the QRS complexes. This rhythm is called a sinoventricular rhythm.

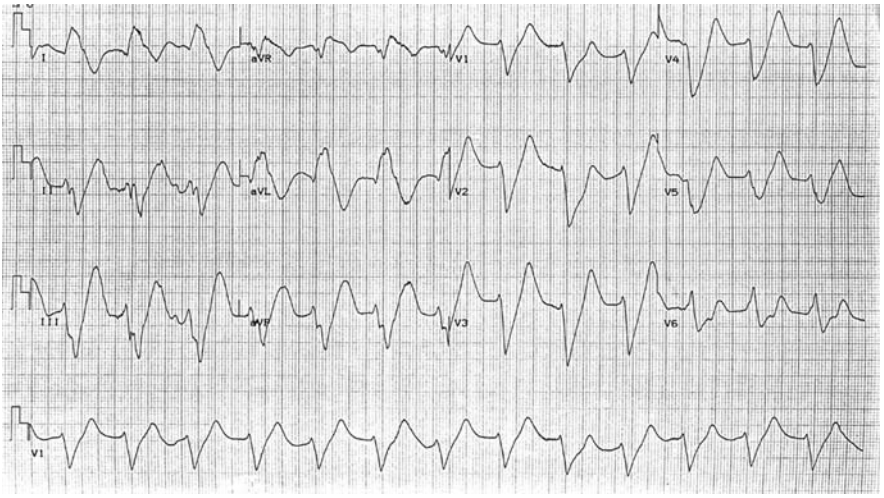


Figure 4: ECG in a patient with hyperkalemia. The QRS is wide and the T waves are prominent. In this case the rhythm is ventricular. A single dissociated P wave can be observed between the second and third QRS complexes in lead III.

sinoventricular rhythm and is observed only in hyperkalemia (Figure 3). As K^+ becomes higher, the ventricular rate slows and asystole develops.

Calcium

Hypocalcemia

In hypocalcemia, the phase 2 plateau is prolonged, leading to prolongation of the ST segment and QT interval. Interestingly, phase 3 repolarization is relatively unaffected, so a normal T wave morphology is often observed, although inverted T waves have been reported. The QT interval prolongation roughly correlates with the severity of hypocalcemia: greater QT prolongation with more severe hypocalcemia. In some cases ST segment elevation has been associated with hypocalcemia. An example of a patient with hypocalcemia is shown in Figure 5. The QT interval is prolonged, with significant ST segment prolongation and a relatively normal T wave. In addition, subtle ST segment elevation is present in the anterior leads.

Hypercalcemia

Hypercalcemia shortens the plateau phase, so a short QT interval is observed, with the T wave starting just after the QRS complex and no intervening ST segment.

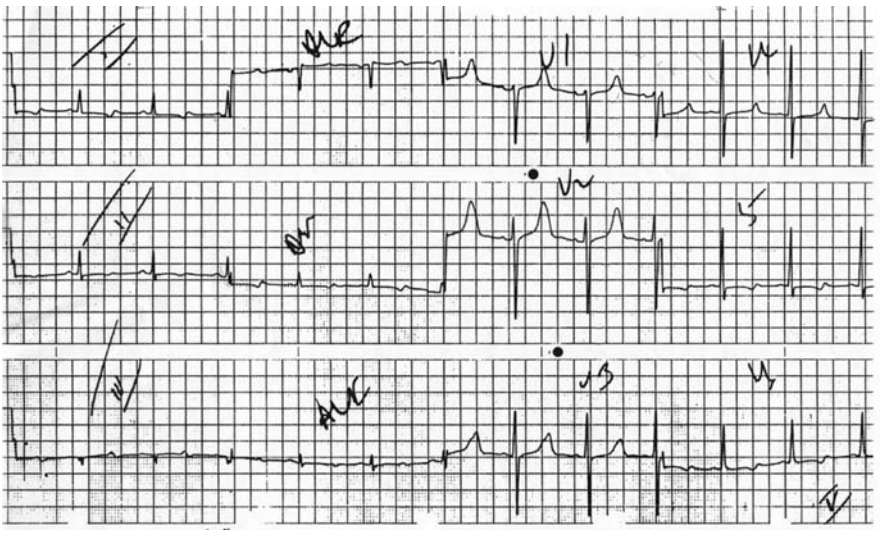


Figure 5: ECG in hypocalcemia. The QT is prolonged due mainly to prolongation of the ST segment. The T wave is relatively normal. Anterior ST segment elevation is present (courtesy Tom Evans).

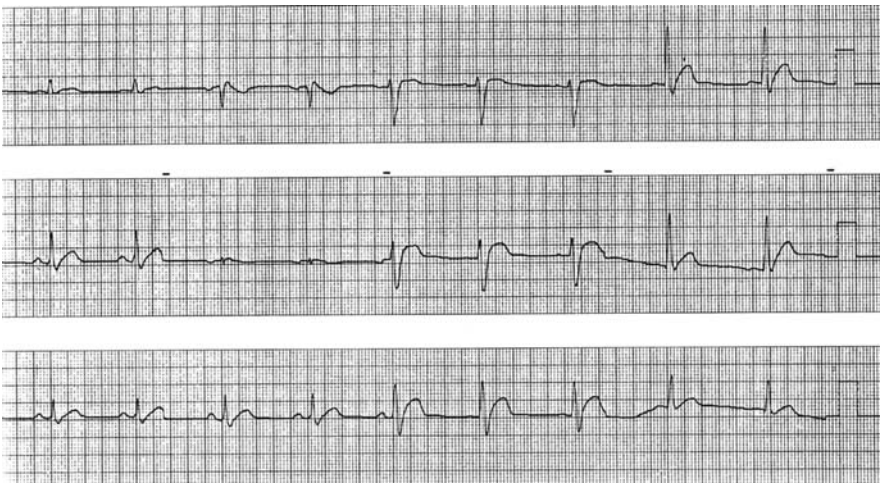


Figure 6: ECG in hypercalcemia. The isoelectric portion of the ST segment is no longer apparent. The T wave appears to start immediately after the QRS, resulting in a short QT interval (courtesy Tom Evans).

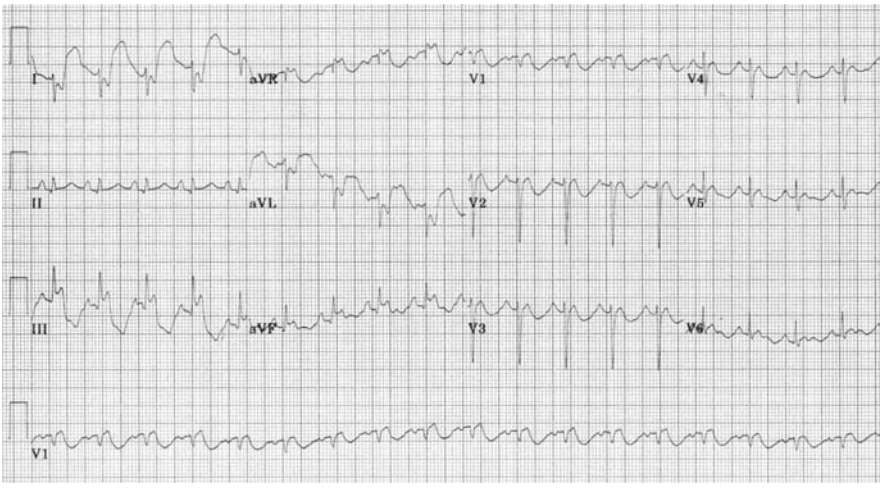


Figure 7: Another ECG in hypercalcemia. In this case the serum Ca^{2+} is 16.5 mg/dL. The patient has developed QRS widening due to development of a prominent J wave at the terminal portion of the QRS complex. The T wave still starts immediately after the J wave, but the QRS widening leads to a relatively normal QT interval.

The T wave will often begin before the QRS returns to baseline, so that "ST segment" elevation similar to that in myocardial infarction can be observed. The loss of the ST segment leads to a short QT interval, usually less than 0.4 s, and often less than 0.35 s. Figure 6 shows a patient with hypercalcemia. No isoelectric ST segment is present; the T wave appears to develop immediately after the QRS.

When the serum Ca^{2+} is extremely elevated (> 16 mg/dL), in some cases extreme QRS widening due to the development of prominent J waves (also called Osborn waves) can lead to normalization of the QT interval. An ECG from a patient with a Ca^{2+} level of 16.5 mg/dL (normal: 8.9–10.1 mg/dL) and QRS widening due to a J wave is shown in Figure 7. This deflection is often also associated with hypothermia and appears to be mediated by accentuation of the phase 1 notch in epicardial cells and M cells. In hypercalcemia, the accentuation of the notch appears to be mediated by an increase in the I_{to}. Additional discussion of the Osborn wave is provided in the final chapter, in the section on hypothermia.

In severe hypercalcemia, atrioventricular delays with PR interval prolongation and atrioventricular block have been reported.

Other electrolyte disorders

Hypomagnesemia can be associated with QT interval prolongation, although there are no specific abnormalities that are characteristic of hypomagnesemia. Figure 8 shows an ECG from a man with isolated hypomagnesemia (K^+ and Ca^{2+} were normal and the patient was taking no medications associated with QT

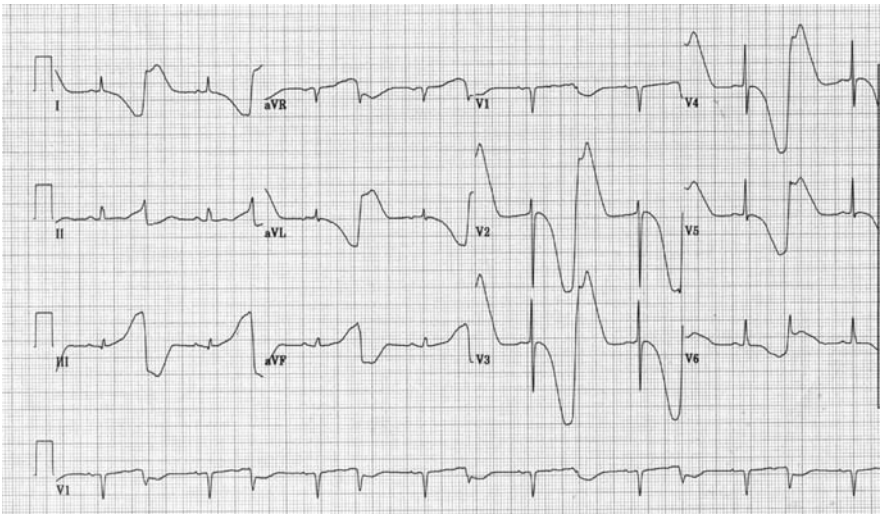


Figure 8: An ECG from a patient with isolated hypomagnesemia. QT interval prolongation due to the development of bizarre biphasic T waves is present in all leads.

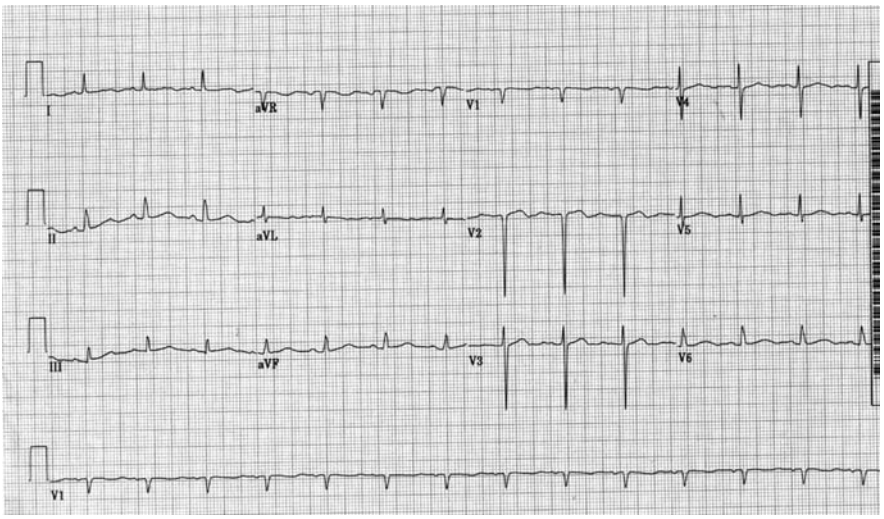


Figure 9: An ECG from the same patient as in Figure 8 after serum magnesium has returned to normal.

prolongation). Bizarre biphasic T waves with QT prolongation are seen. All of the ECG changes resolve several hours later after the hypomagnesemia is corrected (Figure 9). Hypomagnesemia is often associated with hypokalemia. Hypomagnesemia has also been associated with premature atrial and ventricular contractions. Sodium disturbances do not appear to have an effect on the ECG.

Key points

1. Hypokalemia is associated with the development of a bifid T wave. The second hump is often incorrectly called a U wave
2. Hyperkalemia is associated with a spectrum of ECG findings: most often, T wave peaking, but also. ST segment elevation and QRS widening
3. Hypocalcemia causes prolongation of the QT interval
4. Hypercalcemia causes shortening of the ST segment

Review questions

1. Hyperkalemia can be associated with each of the following except:

   A. Peaked T waves
   B. Bradycardia
   C. Bifid T waves
   D. QRS widening

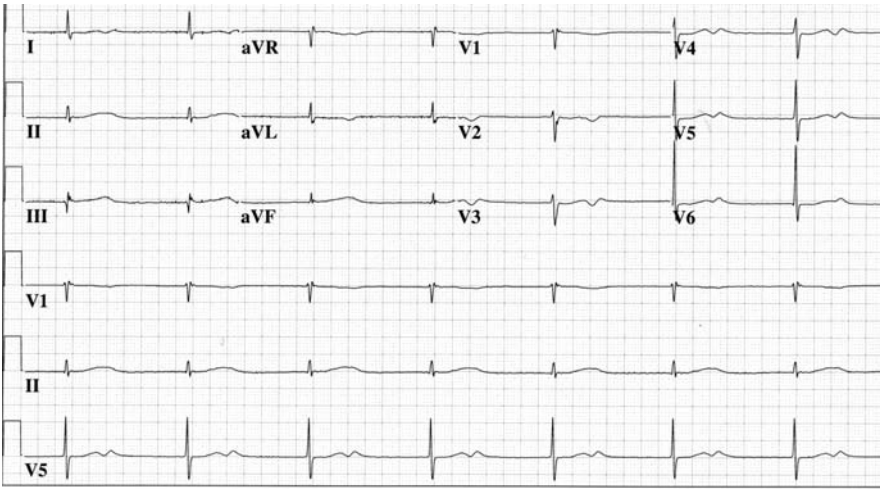


Figure 10: ECG for Questions 4 and 5.

2. The major ECG finding associated with hypocalcemia is:

   A. Prominent U waves
   B. Bifid T waves
   C. ST interval prolongation
   D. QRS widening

3. Which statement is true?

   A. Hypercalcemia is associated with QT prolongation
   B. Sinoventricular rhythms are observed with hyperkalemia
   C. Peaked T waves are observed in patients with hypercalcemia
   D. Ventricular arrhythmias are not associated with hypokalemia

4. What is the rhythm in Figure 10?

   A. Sinus rhythm
   B. Junctional rhythm
   C. AV block
   D. Ventricular automaticity

5. The ECG in Figure 10 is most consistent with:

   A. Hypokalemia
   B. Hypocalcemia
   C. Hyperkalemia
   D. Hypercalcemia

Answers

1. The correct answer is C. Hyperkalemia is associated with peaked T waves, QRS widening, and bradycardia. Bifid T waves are characteristic of hypokalemia.
2. The correct answer is C. Hypocalcemia is associated with QT prolongation due to prolongation of the ST segment.
3. The correct answer is B. Hyperkalemia can cause sinoventricular rhythm due to increased sensitivity of atrial tissue.
4. The correct answer is B. The patient is in junctional rhythm since no P waves precede the QRS complexes.
5. The correct answer is A. The patient has hypokalemia with bifid T waves observed in the anterior leads (often incorrectly called U waves), and QT interval prolongation. Although hypocalcemia can also cause QT interval prolongation, the ST segment itself would be prolonged, and the T wave would have a normal morphology.

Chapter 17
Orphans

There are a few miscellaneous conditions that are associated with characteristic ECG changes that warrant discussion in this last "mop-up" chapter.

Pulmonary embolus

The best use for an ECG in a patient with chest pain is identifying changes suggestive of myocardial injury and ischemia. Pulmonary embolus, in which a blood clot obstructs flow within one of the branches of the pulmonary artery, is another important cause for chest pain. However, generally, the ECG provides very little diagnostic value for the diagnosis of pulmonary embolus.

The ECG usually has nonspecific findings in the setting of a pulmonary embolus, including sinus tachycardia and nonspecific STT changes. In some cases right axis deviation is present. One ECG pattern that has been described for pulmonary embolus is the S1Q3T3 pattern, in which a deep S wave will be observed in lead I and a Q wave and inverted T wave will be seen in lead III (Figure 1). This pattern was first described in the 1930s and may be due to clockwise rotation of the heart from acute dilation of the right ventricle. In older studies, the S1Q3T3 pattern was observed in approximately 25–30% of cases, although more recent studies suggest a lower incidence (7–12%). The specificity of the S1Q3T3 pattern is not known. Despite these shortcomings, presence of an S1Q3T3 in a patient with shortness of breath or chest pain should increase clinical suspicion of the presence of an acute pulmonary embolus, although the diagnosis will be made with other more definitive tests.

Infiltrative diseases: amyloidosis

Amyloidosis is a disease associated with deposition of extracellular proteins. When amyloidosis affects the heart, deposition of protein and loss of myocytes results in congestive heart failure. Patients with cardiac amyloidosis have a normal ECG or isolated left anterior fascicular block. However, approximately 50–60% of

F. Kusumoto, *ECG Interpretation: From Pathophysiology to Clinical Application*,
DOI 10.1007/978-0-387-88880-4_17, © Springer Science+Business Media, LLC 2009

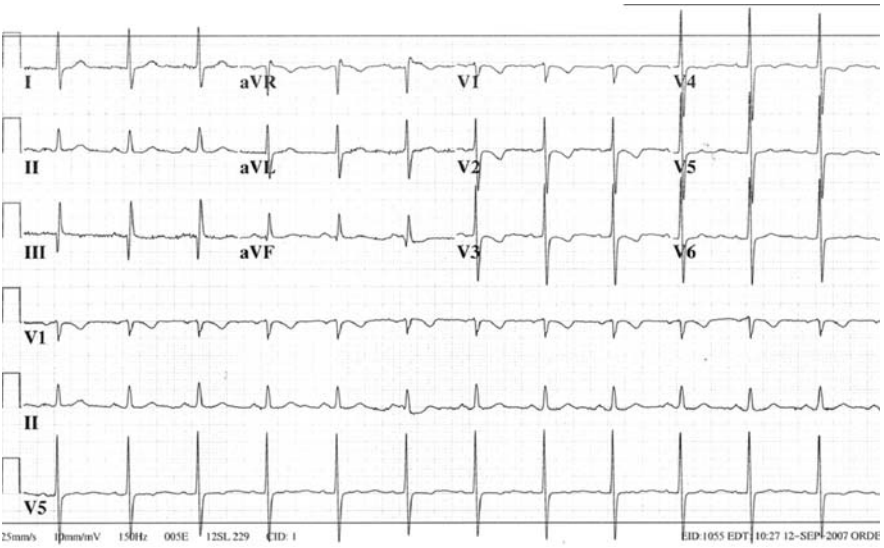


Figure 1: ECG from a patient with a pulmonary embolism with the S1Q3T3 pattern. The ECG shows an S wave in I and a Q wave with an inverted T wave in lead III.

patients will have low ECG voltages, usually defined as < 5 mm in all frontal leads or < 10 mm in the precordial leads. An ECG from a patient with cardiac amyloidosis is shown in Figure 2. Although the precordial lead voltages are relatively normal, the voltages in all of the frontal leads are markedly attenuated (< 5 mm). In addition, anterior Q waves are noted in V_1 through V_3; abnormal Q waves are seen in approximately 40–60% of patients with cardiac amyloidosis, most commonly in the anterior leads. Conduction disturbances (both atrioventricular and intraventricular) can be seen. In Figure 2, first degree AV block is present in addition to borderline QRS prolongation with significant right axis deviation.

Other infiltrative diseases such as sarcoidosis and hemachromatosis, can be associated with low voltage and development of Q waves. A patient with cardiac sarcoidosis is shown in Figure 3. Sarcoidosis is a systemic disease of unknown etiology that is associated with formation of noncaseating granulomas in affected tissues. The ECG manifestations of sarcoid are diverse, depending on the affected organs and the extent of disease. In this case, a patient with cardiac sarcoidosis, anterior and inferior Q waves are observed. Formation of cardiac granulomas can form the substrate for ventricular arrhythmias and atrial arrhythmias.

Although Q waves are usually discussed in the context of myocardial infarction, it is obvious from the current discussion that Q waves can be observed in any infiltrative disease because of regional loss of cardiac myocytes. In fact, the differential diagnosis for Q waves is quite diverse, since the presence of a Q wave simply means that the average ventricular depolarization is directed “away” from that lead. Q waves can be observed in a variety of conditions, including the

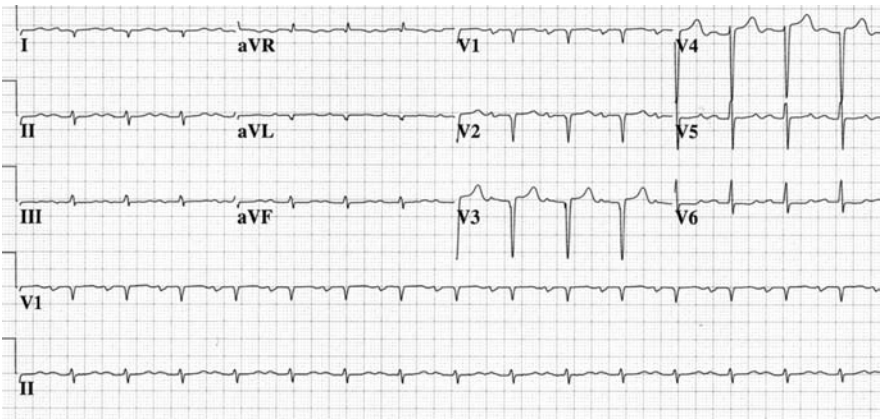


Figure 2: ECG from a patient with cardiac amyloidosis. Notice the reduced voltages in the frontal leads and the Q waves in V_1 through V_3. Anterior ST segment elevation is present in addition to subtle ST depression in V_5 and V_6, with T wave flattening in the lateral leads. First degree AV block is present.

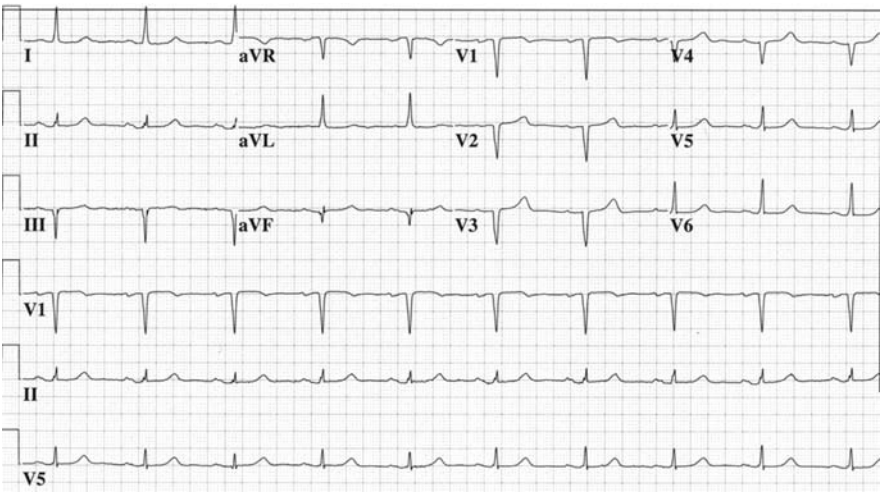


Figure 3: ECG from a patient with cardiac sarcoidosis. Q waves in the anterior leads (V_1–V_4) and inferior leads (III, aVF) are present.

Wolff-Parkinson-White Syndrome, pulmonary embolism, spontaneous pneumothorax, hyperkalemia, myocardial contusion, left bundle branch block, left anterior fascicular block, left ventricular hypertrophy, and chronic obstructive lung disease (Appendix, Table 2). The presence of a Q wave in a lead overlying an area where there has not been a previous myocardial infarction has traditionally been called a "pseudoinfarction" pattern by ECG readers. An example of a "pseudoinfarction"

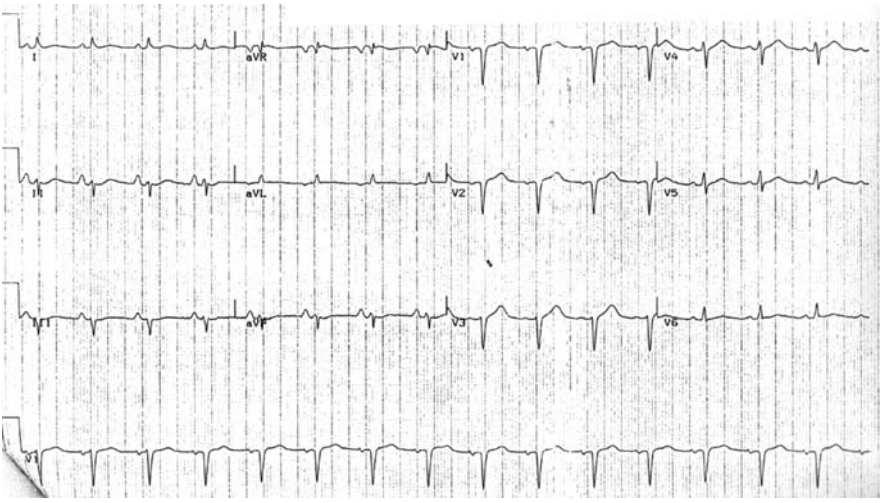


Figure 4: ECG from a patient with chronic obstructive lung disease showing a "pseudoinfarction" pattern with Q waves in the anterior leads (V_1–V_3). Notice the tall P wave, suggestive of right atrial enlargement (courtesy Irwin Hoffman).

pattern in a patient with chronic obstructive lung disease is shown in Figure 4. Vertical positioning of the heart due to hyperinflation and flattening of the diaphragm causes the standard precordial positions to be "too high," leading to poor R wave progression and anterior Q waves in some cases (see discussion on lead misplacement).

Another finding in infiltrative diseases that is often encountered in ECG interpretation is "poor R wave progression." The R wave in the precordial leads becomes more prominent as the electrode orientation changes from lead V_1 to V_6. This finding is not surprising, since the R wave represents septal activation in V_1, and as one moves laterally it represents the brunt of left ventricular depolarization. The QRS will become predominantly positive between V_2 and V_4. The R wave transition is usually defined as the lead in which the R wave becomes greater than the S wave. Early R wave transition leading to a prominent R wave in V_1 was reviewed in the first case of Chapter 15. Poor R wave progression is usually described when the transition is relatively late (V_4 or V_5). A late transition simply means that the average forces of depolarization are occurring away from the anterior precordial leads. Consequently "poor R wave progression" can be seen in a number of circumstances, including any condition that causes loss of myocytes (infiltrative disease, myocardial infarction), posterior rotation of the direction of precordial depolarization (left bundle branch block, left ventricular hypertrophy, right-sided accessory pathway, pacing from the right ventricle), superior misplacement of the precordial leads, and even as a normal variant. An extreme example of "poor R wave progression" is shown in Figure 5. All of the precordial leads have a QS complex, since the ventricle is being depolarized from a pacing lead located in the right ventricular apex

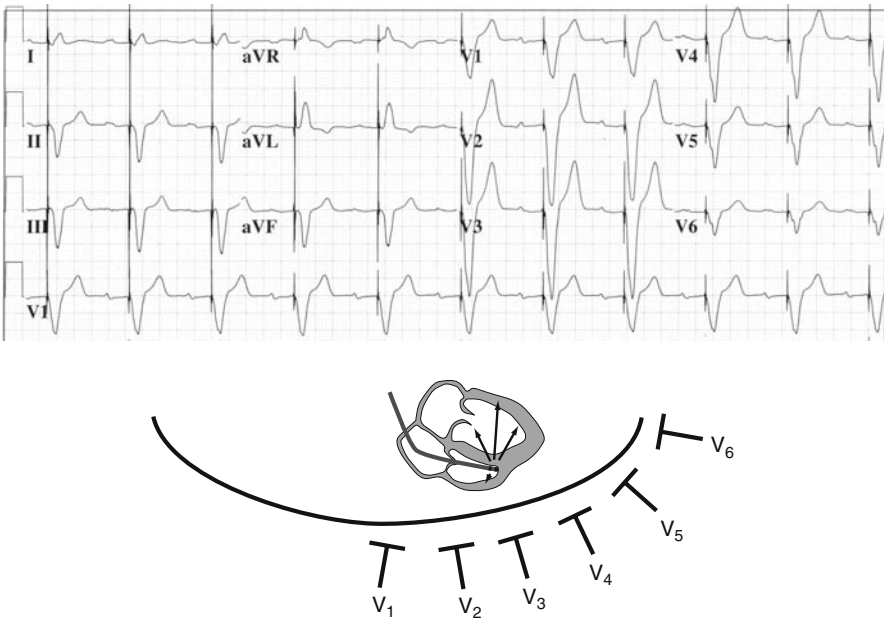


Figure 5: *Top*: Extreme "poor R wave progression" and anterior Q waves due to pacing from the right ventricle. *Bottom*: Since the pacing lead is located in the more anterior right ventricle, depolarization of the posterior left ventricle leads to QS complexes in the precordial leads.

that is anteriorly positioned relative to the posterior left ventricle. The differential diagnosis for "poor R wave progression" is the same as for the presence of anterior Q waves given in Table 2 of the Appendix. Although the presence of poor R wave progression should always "raise an eyebrow," generally the author has found very little clinical utility in poor R wave progression as an isolated ECG finding.

Lightning

A lightning strike is one of the environmental causes of sudden cardiac death, with a mortality rate of approximately 30%. Lightning can have multiple cardiac effects, including direct cardiac damage such as myocardial stunning, cardiomyopathy, or development of Takotsubo Syndrome and development of arrhythmias. Multiple ECG changes have been reported with lightning strikes, including ST segment elevation, Q wave formation, nonspecific STT changes, and QT interval prolongation. Figure 6 shows an example of a patient struck by lightning. Initially the ECG shows ST segment elevation in the anterior leads. The ECG from the next day shows partial resolution of the ST segment elevation, widening of the QRS complex, and development of anterior Q waves (the patient survived with no significant long-term cardiac consequences).

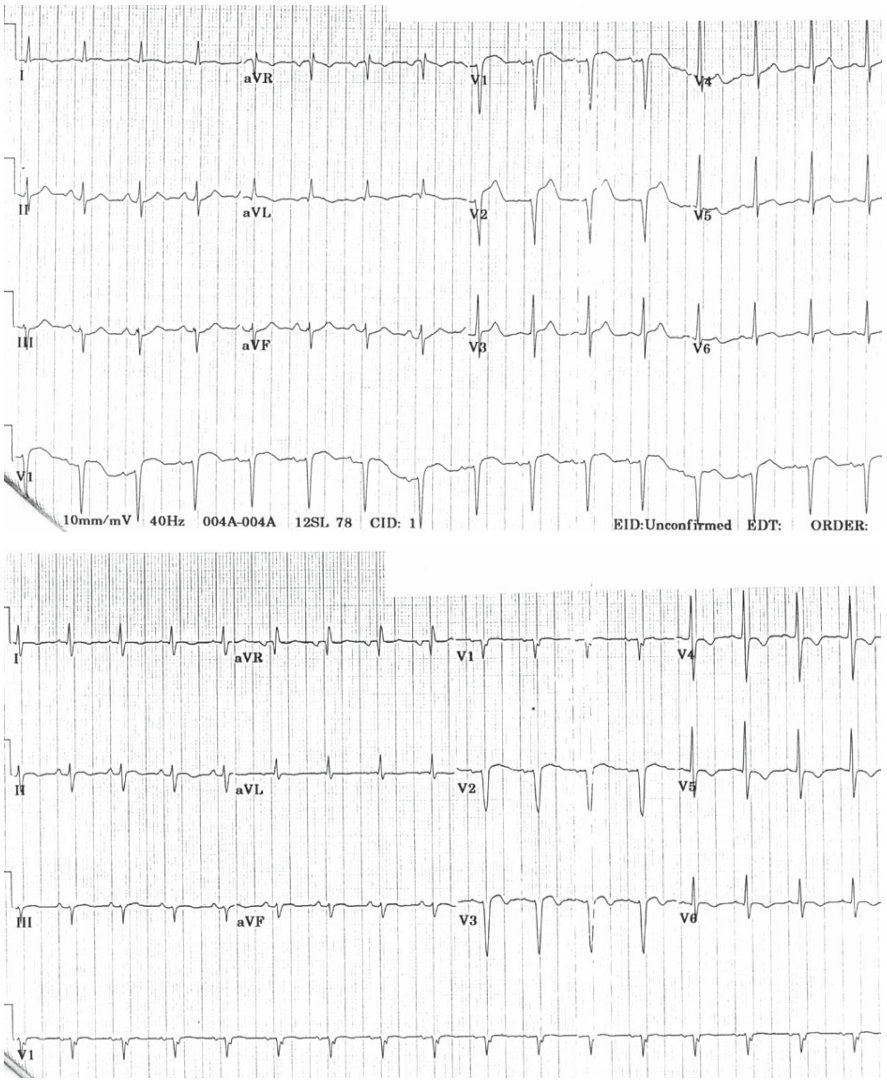


Figure 6: *Top*. Initial ECG from a patient that was struck by lightning, showing anterior ST segment elevation. *Bottom*. ECG on the next day shows partial resolution of the ST segment elevation, QRS widening, and development of anterior Q waves (courtesy Irwin Hoffman).

Hypothermia

John Osborn described the ECG effects of hypothermia in 1953. The most characteristic finding is the development of prominent positive waves just after the QRS complex, called J waves or Osborn waves. The J waves become more

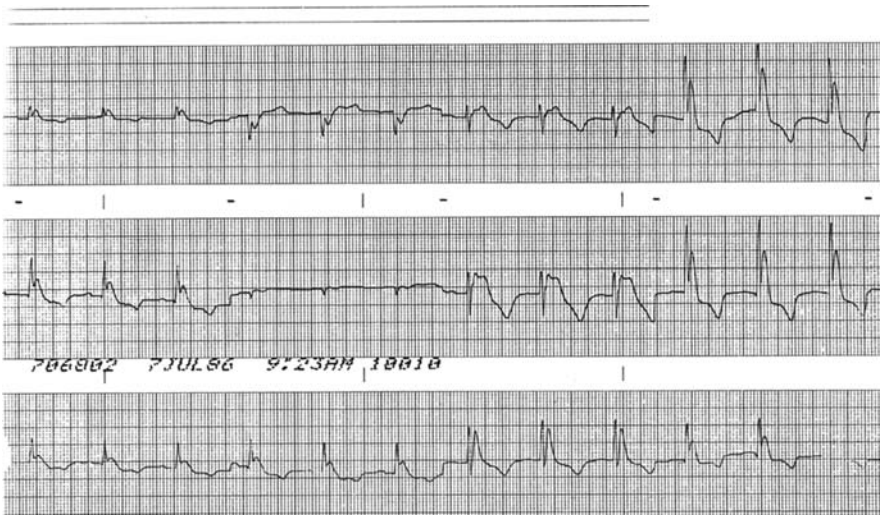


Figure 7: ECG from a patient with severe hypothermia. All of the leads are characterized by a QRS with a terminal Osborn wave that is in the same general direction as the main QRS complex (courtesy Tom Evans).

prominent with worsening hypothermia. The presence of J waves has also been reported in hypercalcemia, Brugada syndrome, and myocardial injury. It is thought that the J waves are a result of potentiation of the "spike and dome" morphology of epicardial cells due to a relative increase in the transient outward current. Experimental preparations have confirmed that hypothermia is associated with accentuation of the phase 1 notch in epicardial tissue. An ECG from a patient with hypothermia is shown in Figure 7. Notice the bizarre QRS complex with the prominent deflections or Osborn waves at the terminal portion of the QRS in all twelve ECG leads. Other findings associated with hypothermia include QT prolongation and T wave inversion.

Muscular dystrophy

Muscular dystrophy due to mutation of the genes responsible can lead to cardiac effects. In Duchenne's muscular dystrophy, mutation of the dystrophin gene leads to a prominent R wave in V_1 due to selective myocyte loss and fibrosis in the posterior wall (think back to the mechanism of a prominent R wave in the setting of a posterior wall myocardial infarction). An ECG from a patient with Duchenne's muscular dystrophy is shown in Figure 8. A prominent R wave in V_1 is noted. Notice that the QRS complex in V_1 has a complex morphology with several sharp deflections. This finding can be observed in Duchenne's muscular dystrophy and is probably due to late depolarization of portions of the scarred posterior wall.

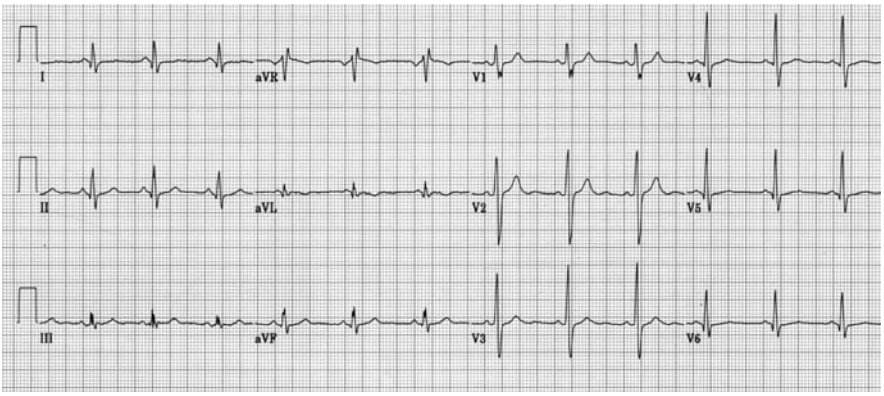


Figure 8: ECG from a patient with Duchenne's muscular dystrophy. A relatively prominent R wave in V1 is noted (the R and S waves are equal). In addition, notice that the terminal portion of the QRS complex has several "notches."

Arrhythmogenic right ventricular cardiomyopathy

Another interesting disease that produces characteristic ECG findings is arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/ARVD). In this inherited disease, mutations of desmosomal proteins responsible for cell adhesion lead to fatty and fibrous tissue deposition within the heart and loss of myocytes. Although histological alterations can be observed in both ventricles, the right ventricle is generally the most affected, particularly in the epicardial portions around the tricuspid valve. At baseline, the tissue infiltration leads to wide QRS complexes with atypical right bundle branch block morphology, often with multiple components. In addition, discrete waves at the terminal portion of the QRS or even within the ST segment, called epsilon waves, can sometimes be observed. An ECG from a patient with ARVC is shown in Figure 9. Notice that a multicomponent QRS (RSr's') with an atypical right bundle branch block morphology is observed in lead V_1. The small negative wave (s') observed in V_1 could potentially be called an epsilon wave, although strictly speaking the epsilon wave should be within the ST segment, separated from the QRS by an isoelectric portion of the ST segment (Appendix, Table 8). In addition, T wave inversion in V_1–V_3 is observed in 50–70% of patients with ARVC.

Patients with ARVC are particularly susceptible to the development of ventricular arrhythmias, because the scar tissue can form a substrate for the development of ventricular reentry. An ECG showing ventricular tachycardia from the patient with the baseline ECG in Figure 9 is shown in Figure 10. The mainstay of treatment for them in patients at high risk for ventricular arrhythmias is an implantable cardiac defibrillator or ICD. One of the functions of an ICD is to deliver a shock from a specialized electrode located on a lead placed in the right ventricle to the ICD "can" (Figure 11). The shock extinguishes the ventricular fibrillation and the patient

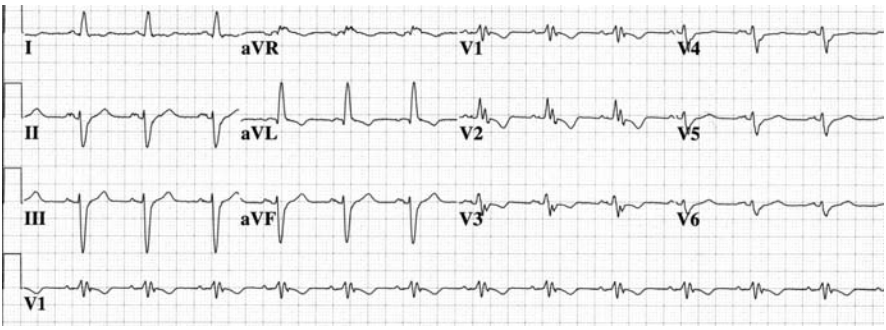


Figure 9: Baseline ECG from a patient with ARVC. A multicomponent RBBB pattern QRS is noted in V_1 and V_2. The terminal negative component of the QRS complex is often called an epsilon wave. Anterior T wave inversion in the right precordial leads is also a common finding in patients with ARVC.

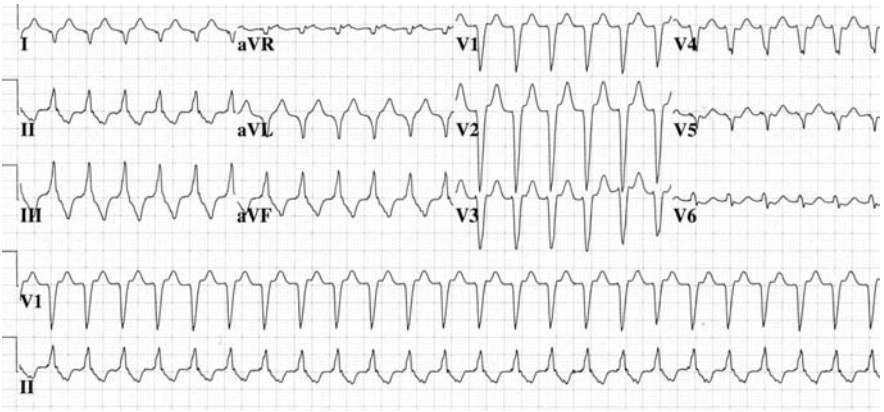


Figure 10: Wide complex tachycardia from the same patient shown in Figure 9. The ECG shows a relatively narrow LBBB pattern. Unfortunately, no evidence of AV dissociation is present. The only clues that this represents ventricular tachycardia are the QS complexes in I and aVL. Remember that typical LBBB is usually characterized by monophasic R waves in I and aVL.

returns to normal rhythm. However, ICD shocks are painful and poorly tolerated. The newest generation of ICDs is designed to pace the heart rapidly to terminate reentrant ventricular arrhythmias. Rapid pacing is designed to enter the slow pathway of the reentrant circuit so that when pacing is stopped the reentrant circuit terminates because it encounters refractory tissue. An example of pace termination of ventricular tachycardia in our patient with ARVC is shown in Figure 12.

We are now in a position to think about the mechanisms for terminal deflections in the QRS (Table 8, Appendix). Additional deflections at the end of the QRS

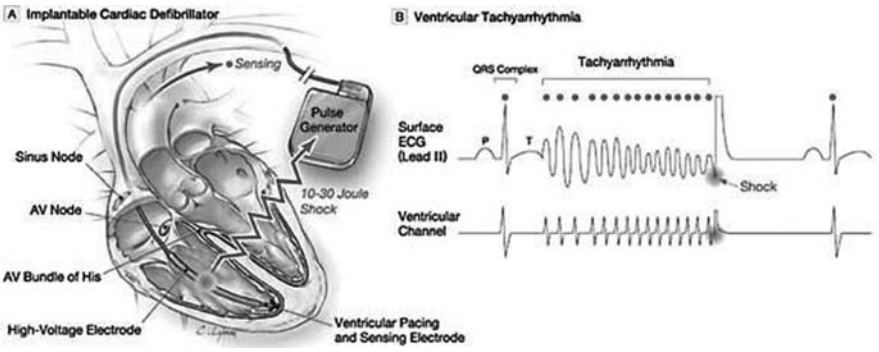


Figure 11: Normal ICD function. **A**. Normally an ICD uses a special lead placed in the right ventricle, attached to an ICD generator placed in the upper left chest. **B**. A ventricular arrhythmia is sensed by an electrode in the tip of the right ventricle as rapid activity. If the rapid ventricular activity exceeds a programmable period, a shock is delivered from a more proximal coil electrode within the right ventricle to the ICD "can" (reproduced with permission from Kusumoto FM, Goldschlager N. Device therapy for cardiac arrhythmias. JAMA 2002;287:1848–52).

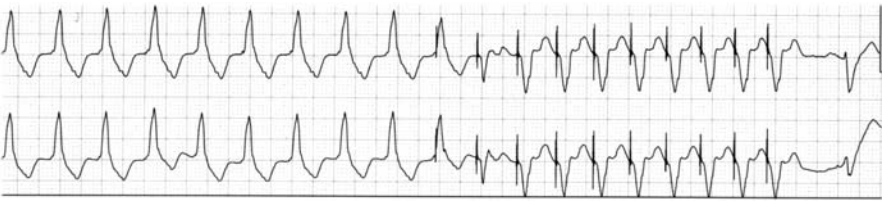


Figure 12: This patient has an ICD to treat ventricular arrhythmias. In ARVC, ventricular tachycardia develops because of scarring within the right ventricle. Rapid pacing of the ventricular tachycardia can terminate the reentrant circuit by causing refractory tissue to develop within the critical portions of the reentrant circuit.

can be due to either delayed depolarization of a region or unusual repolarization. Although we do not usually think of the terminal R' in right bundle branch block as an unusual terminal QRS deflection, it is useful to remember that the final portion of the QRS complex is due to late depolarization of the right ventricle. Similarly, high frequency notches at the terminal portion of the QRS in Duchenne's muscular dystrophy appears to be due to late activation of the posterior wall of the left ventricle, and the atypical right bundle branch block and epsilon waves in arrhythmogenic right ventricular cardiomyopathy is due to late activation of the right ventricle. Abnormal repolarization due to changes in the phase 1 downstroke also can cause deflections in the terminal portion of the QRS. This appears to be the mechanism for J waves in hypothermia and hypercalcemia, the notch in early repolarization, and the terminal R in Brugada Syndrome.

Terminal deflections in the QRS complex:

Delayed depolarization: right bundle branch block, Duchenne's muscular dystrophy, arrhythmogenic right ventricular cardiomyopathy.

Abnormal repolarization: Brugada Syndrome, early repolarization, J waves (hypothermia, hypercalcemia).

Electrode misplacement

Misplacement of electrodes can cause a number of findings that can lead to erroneous interpretations. The most easily identified electrode misplacement of the frontal leads is a right arm-right leg switch. In this case, since lead II measures the

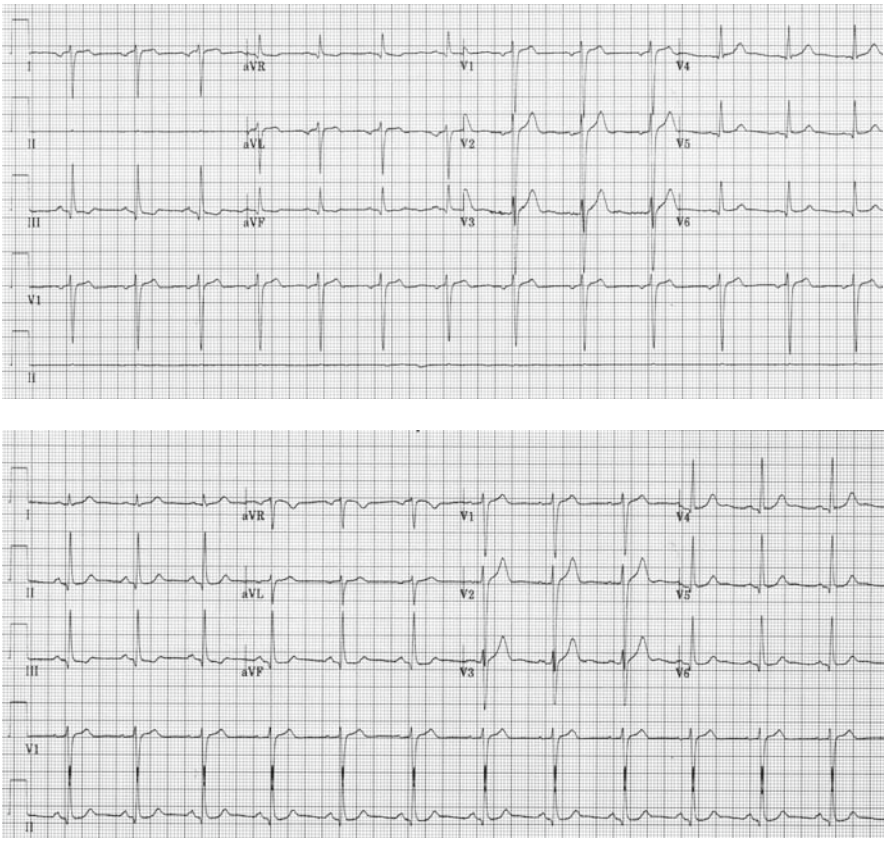


Figure 13: *Top*. Right leg-right arm switch. Since lead II records the difference between the right arm and the left leg, this particular electrode switch causes attenuation of the signals in lead II, since the voltage changes "observed" from the right leg and left leg are very similar. *Bottom*: Electrode switch corrected.

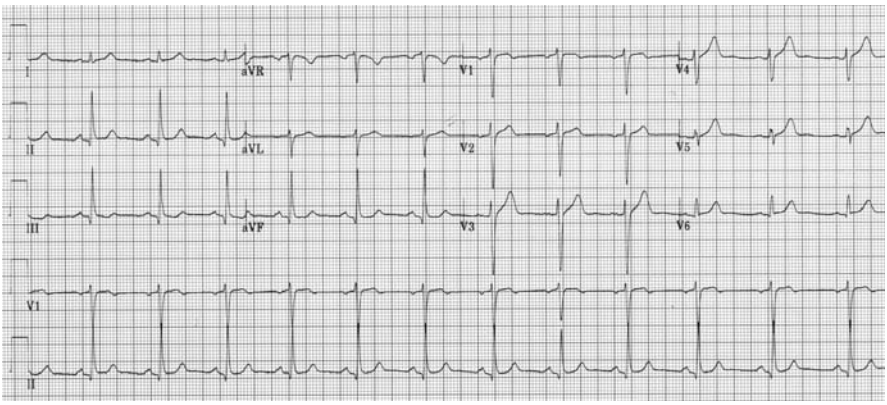


Figure 14: The same patient as in Figure 13 but with the precordial leads placed one interspace too high. Notice that this results in the development of "poor R wave progression" and ST segment elevation and T wave inversion in V_1.

difference between the right arm and left leg electrode, a vey attenuated and often "flatline" signal is recorded in lead II because of the very small voltage differences between the right leg and the left leg. An example of a right leg-right arm switch is shown in Figure 13.

The most common electrode misplacement problem for the precordial leads is placing the electrodes too high. An example of the effects on the ECG of placing the precordial leads one interspace too high is shown in Figure 14. Notice that the T wave has become inverted in V_1 and the R wave progression is attenuated. This is another reason that poor R wave progression is such a nonspecific finding that often can give misleading results.

Artifact

Body movement can affect ECG recording. An example of an apparent wide complex tachycardia or atrial flutter (depending on which lead is chosen) due to a loose lead is shown in Figure 15. The easiest way to evaluate whether artifact is present is to note whether QRS complexes can be seen to "march through" (after checking to make sure the patient is okay), and fortunately in this case lead III still has a technically satisfactory recording. This is another example of the usefulness of simultaneous recording of multiple leads.

Another form of artifact is due to digital signal processing techniques. In most cases an ECG can be set to detect pacing stimuli by enhancing any sudden changes in voltage. The digital processing will then accentuate this signal so that it is more easily seen on a monitor. This can lead to unusual artifacts that suggest pacemaker malfunction when in fact normal pacemaker function is present. Figure 16 shows digital artifacts due to digital enhancement algorithms used to detect pacing.

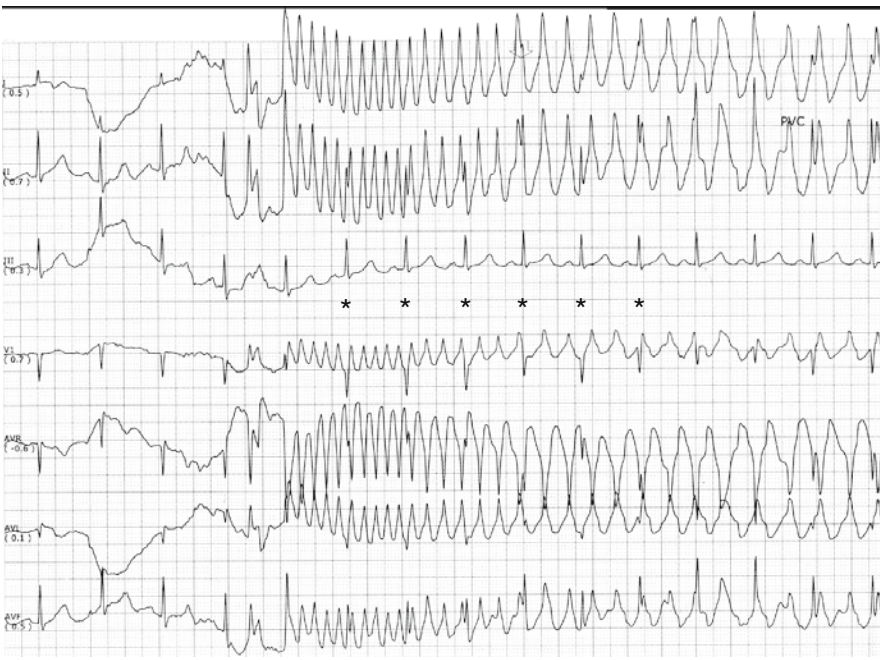


Figure 15: Artifact that could potentially lead to the erroneous diagnosis of wide complex tachycardia in leads I, II, aVF, aVR, and aVL, or atrial flutter in lead V_1. The QRS complexes (*) "march through" regularly in the frontal leads, making sudden initiation of atrial flutter or ventricular tachycardia unlikely. Of course in this case lead III reveals normal sinus rhythm.

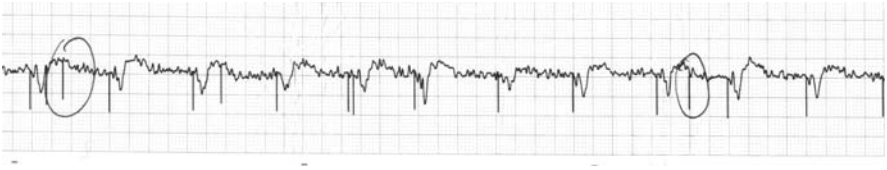


Figure 16: Digital artifact. In this ECG monitoring system, a "detect pacemaker" algorithm was turned "on." The algorithm is designed to identify any sharp voltage changes that probably represent pacing and artificially add a pacing artifact. The additional pacing artifact suggests pacemaker malfunction with delivery of pacing stimuli during the ST segment.

A final form of artifact is incorrect acquisition. The standard ECG is acquired with 1 mm equal to 0.1 mV. If the ECG is acquired at double or half standard, an incorrect interpretation of left ventricular hypertrophy or low voltage can be made (Figure 17).

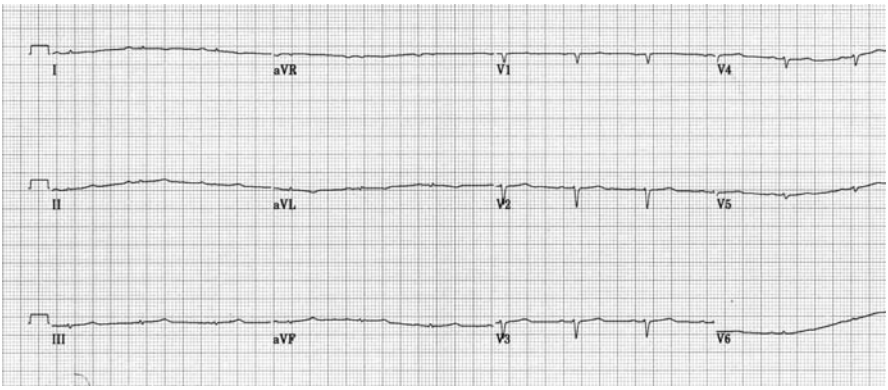


Figure 17: ECG acquired at half-standard that could potentially lead to the incorrect diagnosis of low voltages.

Parasystole

Parasytole is an unusual arrhythmia for which the traditional definition was a "protected" focus that is not affected by the patient's normal rhythm. It is now clear that parasystolic foci are not fully protected and are modulated to some extent by the patient's normal rhythm. Ventricular parasystole is the most commonly described form of parasystole. In this case a ventricular focus will depolarize the ventricles if the ventricles are not refractory. This is analogous to a "natural version" of an asynchronous pacemaker. An example of ventricular parasystole is shown in Figure 18. In this case, a ventricular QRS with a right bundle branch block morphology is observed at regular intervals interspersed between normally conducted beats. The most important characteristic that differentiates parasystole from simple premature ventricular contractions is its variable relationship to the intrinsic QRS complexes and a regular rate for the parasytolic QRS complexes that is relatively constant.

The incidence of parasytole is unknown. More importantly, although it is an interesting phenomenon, there are no specific clinical consequences that have been identified with the presence of parasystole, and it is generally considered an ECG oddity with very little clinical relevance.

Digoxin toxicity

Twenty years ago, digoxin was an extensively used medication for many cardiac disorders, including atrial fibrillation and congestive heart failure. Although the use of digoxin has decreased substantially, it is important for the student of ECGs to remember a few patterns associated with digoxin toxicity. As mentioned in Chapter 6, digoxin affects the ST segment and T wave of ventricular myocytes.

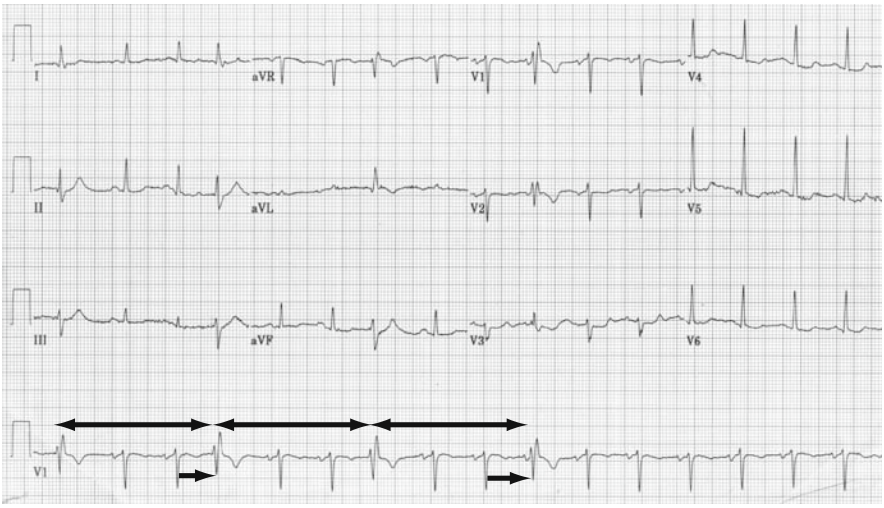


Figure 18: A patient with ventricular parasystole. Four ventricular QRS complexes with a right bundle branch block pattern that have a rate independent to the QRS complexes due to sinus rhythm are present. Parasystole is present because the RBBB QRS complexes have a constant rate, and the relationship between the sinus rhythm QRS complexes and the RBBB QRS complexes do not have a constant coupling interval (in other words, the two rhythms appear to be independent).

Since digoxin is still often used in patients with atrial fibrillation, one of the common manifestations of digoxin toxicity is the development of complete heart block. An example is shown in Figure 19. Atrial fibrillation should be associated with an irregular ventricular rhythm. The development of a regular slow rhythm should arouse suspicion that complete AV block has developed. The ST segments have the typical downsloping associated with digoxin (Chapter 6, Figure 5). Baseline undulation is also noted and should not be misidentified as a premature ventricular contraction.

Ashman's phenomenon

In patients with rapid heart rates due to atrial arrhythmias, bursts of transient wide complex tachycardia due to sustained aberrant ventricular activation can sometimes be observed (Figure 20). These bursts are called Ashman's phenomenon in honor of the physician that originally described it. The mechanism for Ashman's phenomenon is shown in Figure 21. The refractory period for the bundles is determined by the preceding R-R intervals. With slower rates refractory periods increase, and with faster rates refractory periods shorten. With the first QRS after a relatively long RR interval, conduction occurs normally over both bundles. However, if this is

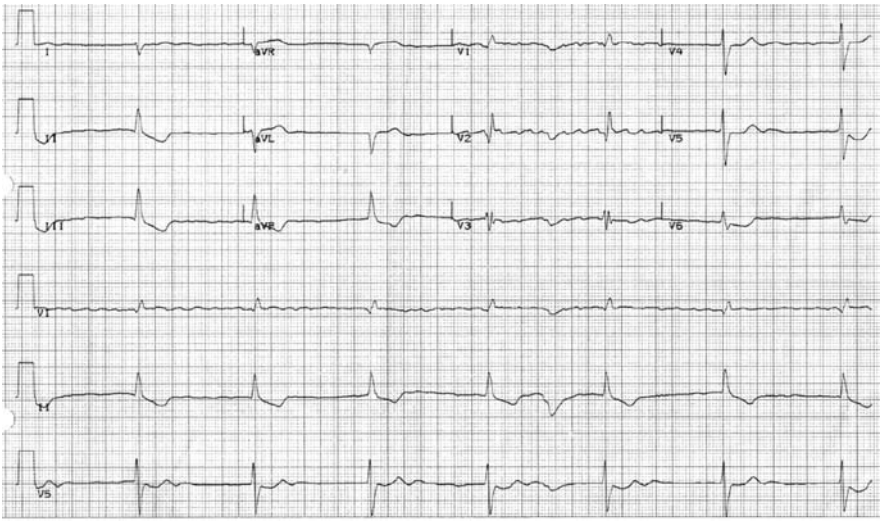


Figure 19: Patient in atrial fibrillation where rapid irregular atrial activity normally leads to a fast and irregular ventricular rhythm. In atrial fibrillation, no discrete P waves are seen. Rather there is continuous fibrillatory activity due to continuous, irregular, and rapid depolarization of atrial tissue. However, in this case 3° AV block is present, so the QRS complexes are regular even though the atria are fibrillating.

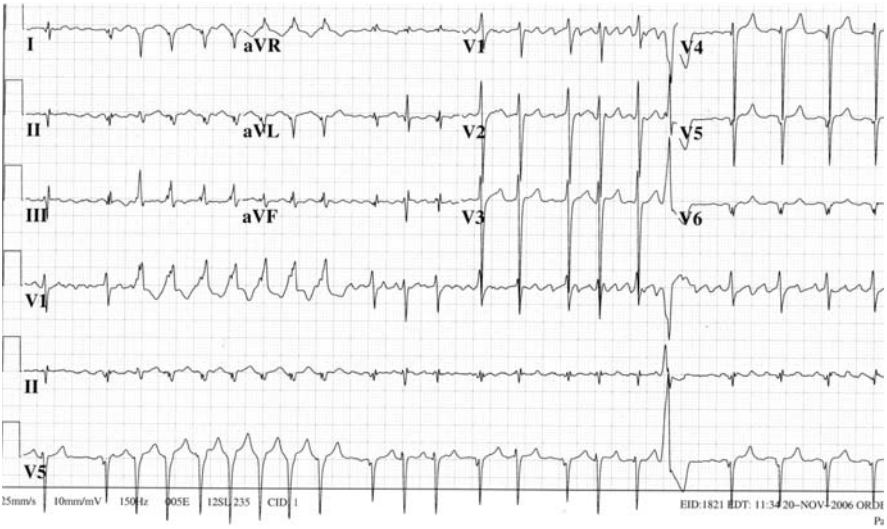


Figure 20: Burst of wide complex rhythm with a RBBB morphology in a patient with atrial fibrillation. This most likely represents Ashman's phenomenon, since it was preceded by a "long-short" sequence. In contrast, the patient has a single wide complex beat later in the ECG that has a LBBB morphology. This is most likely a premature ventricular contraction because it is not initiated by a "long-short" sequence and has a LBBB morphology.

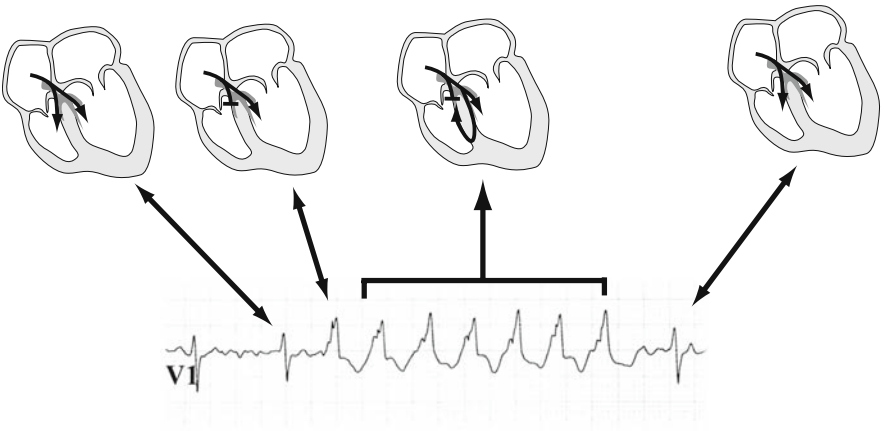


Figure 21: Mechanism of Ashman's phenomenon. For the first beat after the pause, conduction occurs over both the right and left bundle. When the next earlier beat occurs, the right bundle is refractory and a QRS with a RBBB pattern is observed. Continued RBBB is present because of retrograde penetration into the right bundle from the preceding beat. In this case, conduction returns to normal after a relatively long R-R interval.

followed by an early beat ("long-short coupling"), the right bundle will be refractory and the next QRS will have a RBBB morphology. Sustained RBBB occurs because of continued retrograde activation of the right bundle from the preceding beat. Once another pause occurs, or if the refractory period of the right bundle shortens enough, the QRS complex becomes narrow again. It can often be difficult to differentiate Ashman's phenomenon from nonsustained ventricular tachycardia. Ashman's phenomenon is more likely if the wide complex tachycardia is initiated by a "long-short" sequence, and it most commonly has a RBBB morphology because the right bundle generally has the longest refractory period of any of the His-Purkinje system.

Appendix

Table 1: Causes for abnormal axis deviation

Left axis deviation

- Normal variant (2–5%)
- Left anterior fascicular block
- Left ventricular hypertrophy
- Inferior wall myocardial infarction
- Primum atrial septal defect
- Hyperkalemia
- Left bundle branch block

Right axis deviation

- Normal variant
- Lead misplacement
- Left posterior fascicular block
- Right ventricular hypertrophy
- Lateral wall myocardial infarction
- Dextrocardia
- Pulmonary embolus
- Chronic obstructive lung disease
- Secundum atrial septal defect

Table 2: Differential diagnosis for Q waves

Anterior

- Anterior wall myocardial infarction
- Left ventricular aneurysm
- Left ventricular hypertrophy
- Left bundle branch block
- Infiltrative diseases (amyloid, sarcoid)
- Right-sided accessory pathway
- Chronic obstructive lung disease
- Pneumothorax
- Dilated cardiomyopathy
- Intracranial hemorrhage
- Hyperkalemia
- Pacing

Inferior

- Inferior wall myocardial infarction
- Left posterior fascicular block
- Inferior accessory pathway
- Hypertrophic cardiomyopathy
- Pacing

Lateral

- Lateral wall myocardial infarction
- Left anterior fascicular block
- Left lateral accessory pathway

Table 3: Prominent R wave in V_1

Cause	Reason for	ECG characteristics
Right bundle branch block	Delay in right ventricle activation allows unopposed right ventricular activation to be observed	• QRS is wide • Usually with an rsR' morphology
Left-sided accessory pathway	Abnormal activation of the left ventricle leads to a new early force pointed anteriorly	• QRS wide • PR interval is short
Right ventricular hypertrophy	Thickened right ventricle	• Right axis deviation • Inverted T wave in V_1
Posterior myocardial infarction	The R wave actually represents a Q wave from the posterior region of the heart	• Usually an accompanying inferior wall myocardial infarction with inferior Q waves • Upright T wave in V_1
Dextrocardia	Heart is in the right chest	• Right axis deviation • No precordial R wave progression • Atrial activation usually left to right
Duchenne's muscular dystrophy	Scarring in the posterior wall leads to more prominent and unopposed anterior forces in V_1	• Terminal "notches" in V_1

Table 4: T wave changes and possible causes

Nonspecific T wave changes

- Heart disease
- Drugs
- Electrolyte abnormalities
- Hyperventilation
- Pericarditis
- Normal variant
- Left ventricular hypertrophy
- Bundle branch block
- Pancreatitis, cholecystitis, esophageal spasm
- Hypothyroid

T wave inversion

- Normal variant
- Myocardial infarction/ischemia
- Digoxin, antiarrhythmic medications
- After ventricular pacing or radiofrequency catheter ablation (cardiac memory)
- Left ventricular hypertrophy
- Bundle branch block
- Central nervous system problems

Peaked T waves

- Hyperkalemia
- Myocardial infarction/injury
- Normal variant (early repolarization)
- Intracranial hemorrhage
- Left bundle branch block
- Left ventricular hypertrophy

Table 5: Low voltage

"Stuff between the surface and the heart"
• Obesity • Chronic obstructive lung disease • Pleural effusion • Pericardial effusion
"Less heart"
• Infiltrative cardiomyopathies (amyloidosis, hemachromatosis) • Dilated cardiomyopathy • Ischemic cardiomyopathy
Miscellaneous
• Myxedema • Artifact (incorrect voltage standardization)

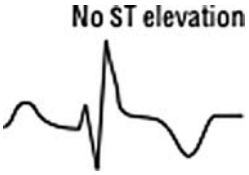
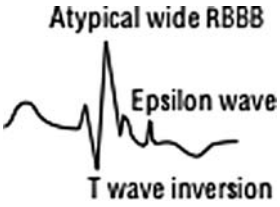
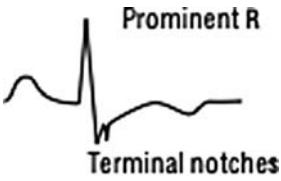
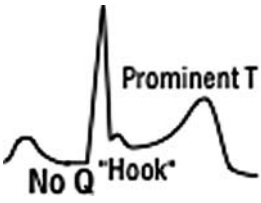
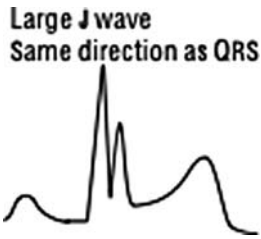
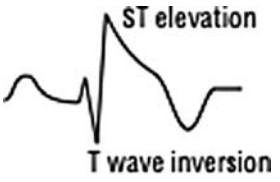
Table 6: Slow heart rates

Cause	ECG findings
Sinus node dysfunction	Very few or no P waves. When a P wave is present, conduction to the ventricle is usually observed.
Atrioventricular block	More P waves than QRS complexes

Table 7: Narrow complex tachycardia

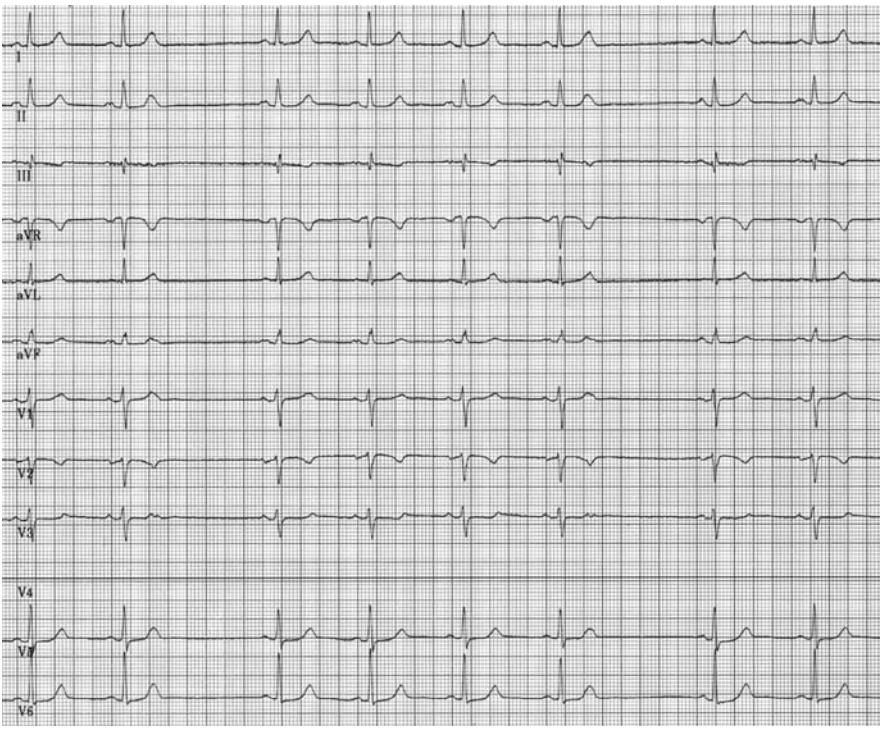
Cause	ECG characteristics
Irregular	
Multifocal atrial tachycardia	Discrete P waves with several different shapes
Atrial fibrillation	No discrete atrial activity observed
Regular	
Sinus tachycardia	Discrete P waves that are similar in shape to the P waves t baseline
Atrial tachycardia	Discrete P waves that are shaped differently from the baseline P wave
Atrial flutter	Sawtooth activation
AV node reentrant tachycardia	P waves are often not seen since they are "buried" within the QRS complex
Accessory pathway mediated tachycardia	P waves are usually seen in the ST segment

Table 8: "Funny waves" at the end of the QRS complex

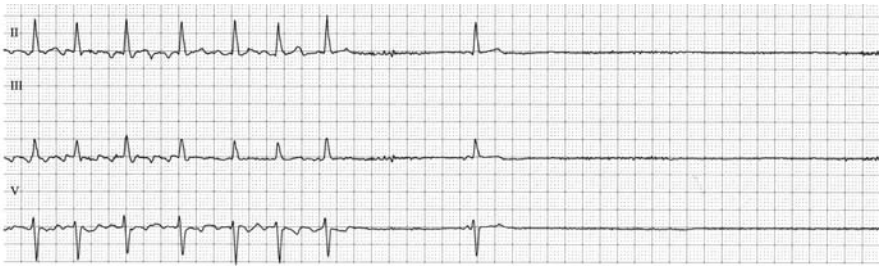
Delayed depolarization		
Right bundle branch block	• R' in V_1 • Larger and wider the R' the greater the delay/block • No ST elevation	
Arrhythmogenic RV cardiomyopathy	• R' in V_1 with atypical RBBB • anterior T wave inversion. • Epsilon waves	
Duchenne's muscular dystrophy	• High frequency "notches" in V_1 • Prominent R in V_1	
Repolarization abnormalities		
Early repolarization	• "Hook" at the end of the QRS • ST elevation	
Osborn waves • Hypothermia • Hypercalcemia	• J wave in the same direction as the QRS. • Seen in all leads.	
Brugada Syndrome	• R' in V_1 • Anterior ST segment elevation • Anterior T wave inversion	

Extra practice
“So you’re a glutton for punishment”

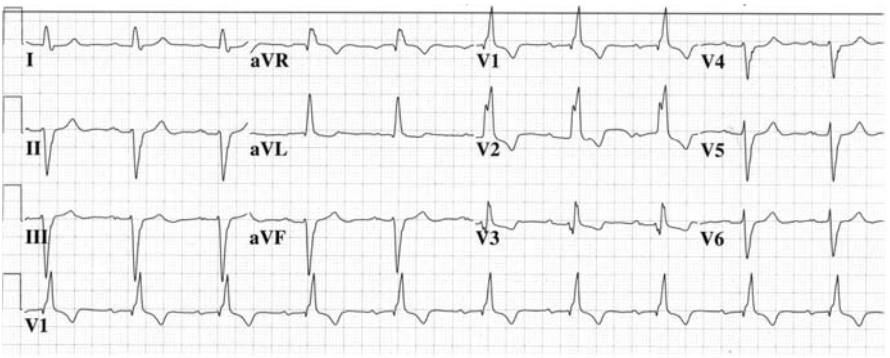
Unknowns



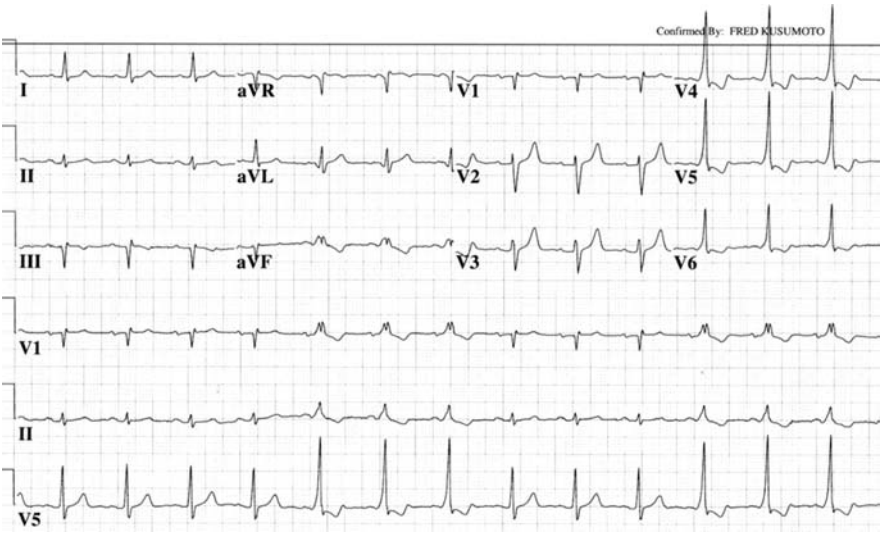
Problem 1:



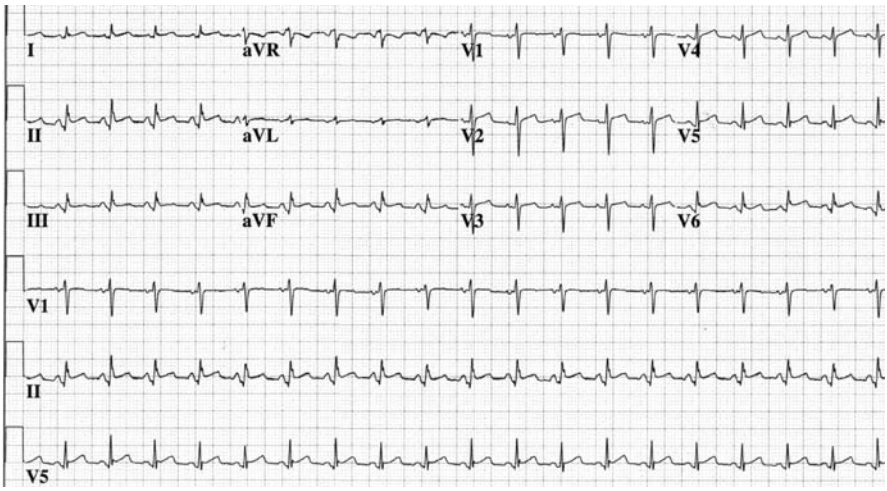
Problem 2:



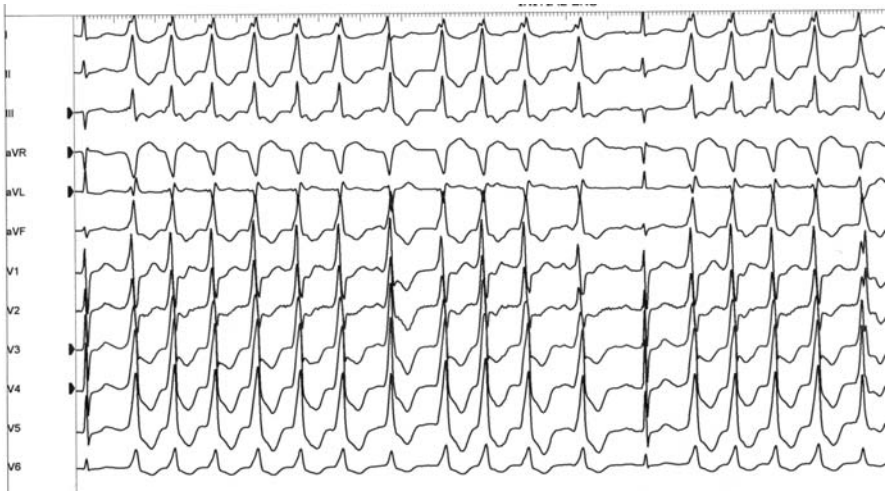
Problem 3:



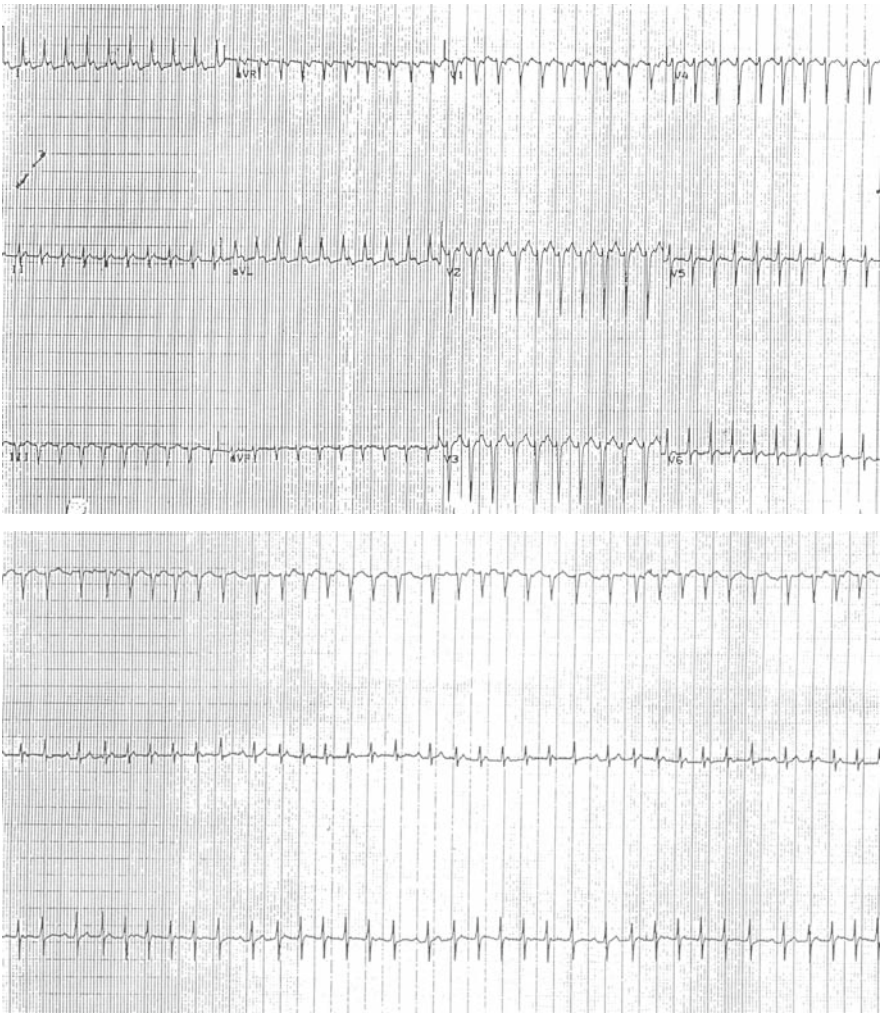
Problem 4:



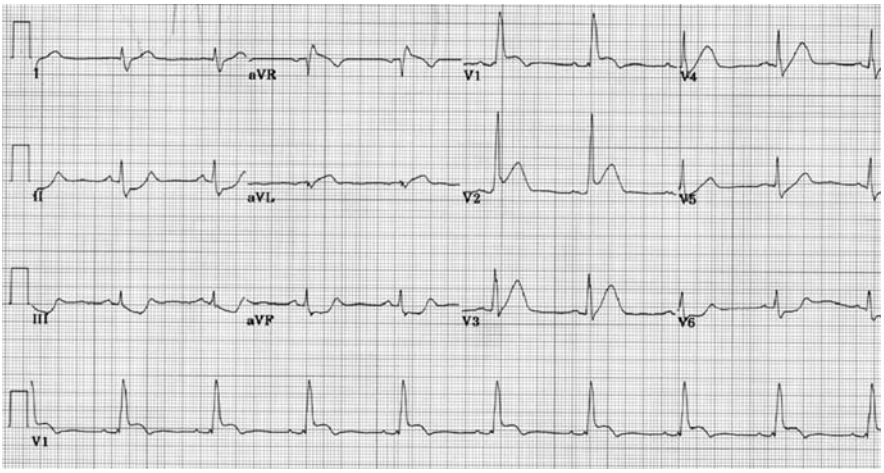
Problem 5:



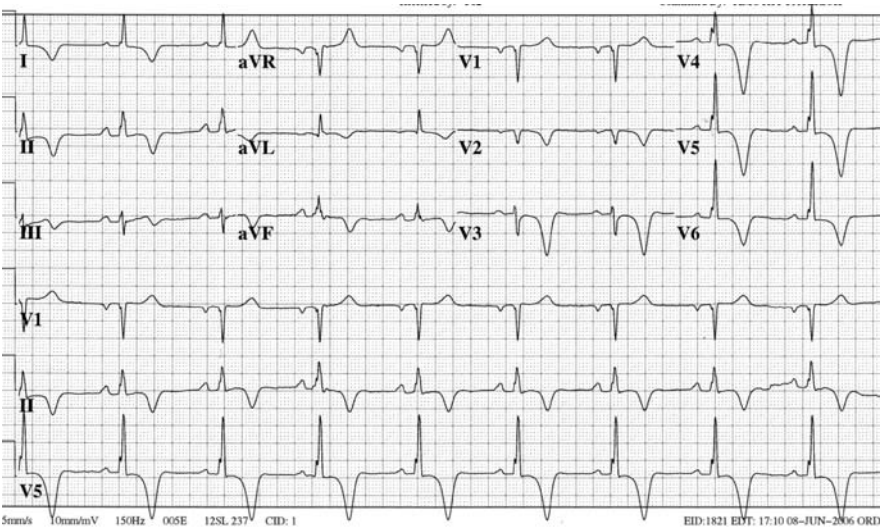
Problem 6:



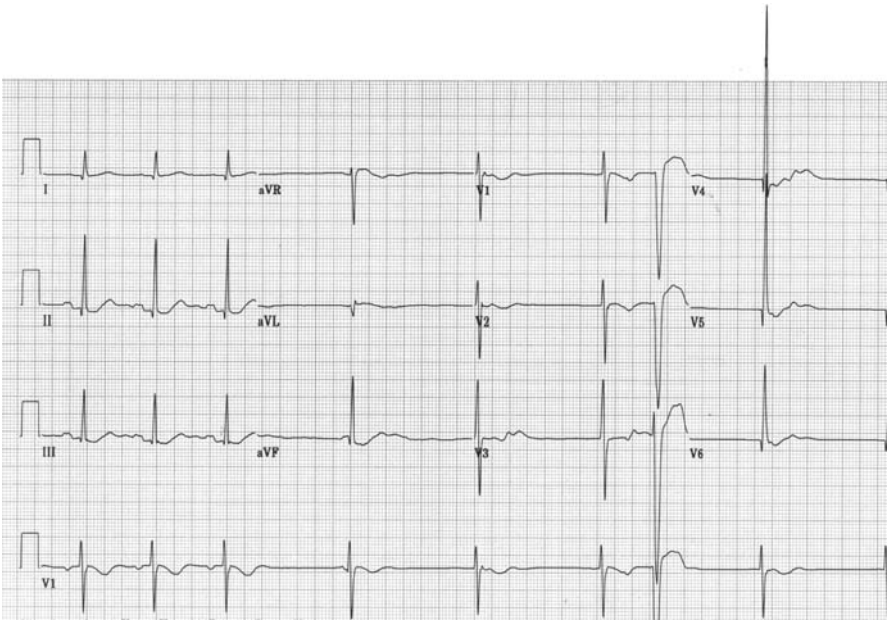
Problem 7:



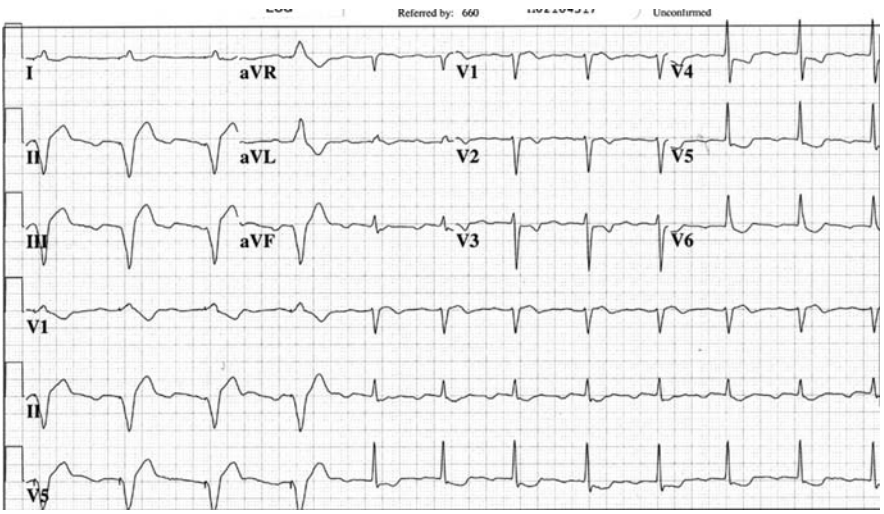
Problem 8:



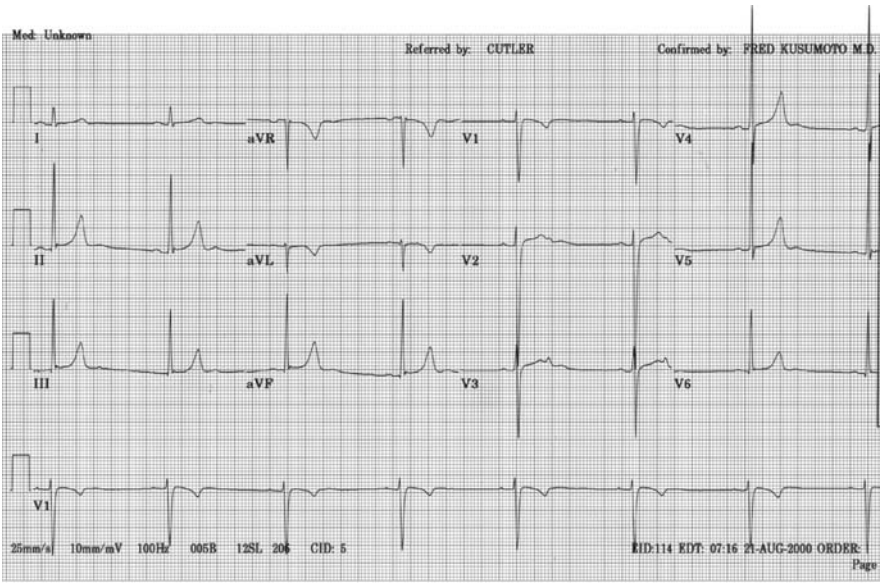
Problem 9:



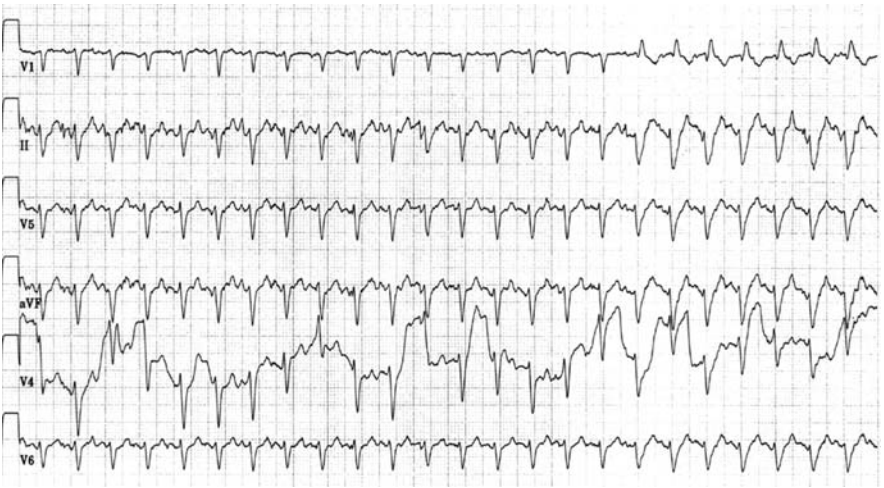
Problem 10:



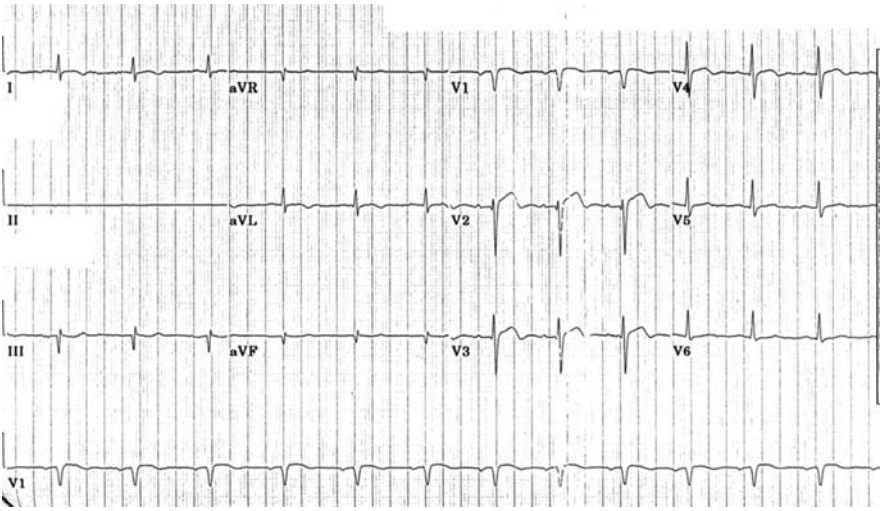
Problem 11:



Problem 12:



Problem 13:



Problem 14:

Answers

1. The ECG shows an irregular but reasonably normal heart rate. Since all of the QRS complexes are the same (ruling out an irregular rhythm due to premature ventricular complexes), the most important thing to evaluate is whether the P waves are regular. In this case nonconducted premature atrial complexes can be seen to distort the T waves just before the pauses. The premature atrial complexes are best seen in V_3.
2. The ECG shows atrial fibrillation that spontaneously terminates and leads first to an ectopic atrial beat (probably near the AV node, based on P wave morphology and the short PR interval) and then to a long sinus pause. Pauses after atrial fibrillation terminates are relatively common, particularly in the elderly, where it is often called “brady-tachy” syndrome because rapid rates (due to atrial fibrillation) are interspersed with slow rates due to sinus bradycardia or sinus node arrest. It appears that after constant bombardment from atrial fibrillation the sinus node can sometimes take a while to “wake up.”
3. The patient has first degree AV block, right bundle branch block, and left anterior fascicular block. The presence of block of two of the three fascicles (right bundle branch block and left anterior fascicular block, right bundle branch block and left posterior fascicular block, or left bundle branch block) and PR interval prolongation has traditionally been called trifascicular block because delay in the third fascicle could potentially produce this ECG pattern. It is very hard to differentiate between this possibility and bifascicular block with accompanying AV nodal block from the surface ECG. Sometimes direct

measurement of activation within the heart (electrophysiologic testing) is required to distinguish between these two possible explanations for the ECG finding.

4. The patient has intermittent preexcitation due to conduction over a left-sided accessory pathway. Notice that the wide QRS complex beats are positive in lead V_1 and are associated with a short PR interval.
5. The patient has pericarditis. There is diffuse ST segment elevation with reciprocal ST segment depression in aVR. Conversely, the PR segment is depressed in the inferolateral leads and elevated in aVR.
6. The patient has an irregular nonsustained ventricular tachycardia. Notice that there is an intervening sinus beat. There is no obvious evidence for AV dissociation, but the presence of an RS complex in V_1 makes ventricular tachycardia the most likely diagnosis.
7. The patient has a short RP tachycardia with the P wave in the ST segment. This pattern is most consistent with an accessory pathway mediated tachycardia. However, the P wave has a "high-low" morphology with positive P waves noted in the inferior leads. This morphology would be unusual for an accessory pathway mediated orthodromic atrioventricular tachycardia, where the atria are activated in retrograde fashion via an accessory pathway. The patient spontaneously develops AV Wenckebach due to refractoriness of the AV node. Since the tachycardia continues in the presence of AV block (AV node independent), the diagnosis must be atrial tachycardia. The atrial tachycardia focus is near the sinus node—this is why the P wave has a "high-low" pattern and is negative in aVR.
8. The patient has an anterior wall myocardial infarction due to a proximal occlusion in the left anterior descending artery, leading to ST segment elevation in aVL, V_1–V_4. The prominent R wave in V_1 is due to accompanying right bundle branch block.
9. Diffuse T wave inversion is present in the inferior, lateral, and anterior leads. The QT interval is normal. The QRS is abnormal with abnormal Q waves present in V_1 and V_2. This ECG pattern is not specific and would be worrisome for ischemia if the patient is complaining of chest pain. However, this ECG is from a patient with hypertrophic cardiomyopathy (genetic disease of sarcomere proteins) that primarily affects the apex. It is interesting to note that even with this diagnosis and significant left ventricular hypertrophy documented by echocardiography, the patient does not have any ECG voltage criteria for left ventricular hypertrophy.
10. The patient has sinus node arrest followed by junctional rhythm. The single wide QRS beat I is probably due to conduction of a P wave with a prolonged PR interval (AV node was partially refractory from the prior junctional beat) and left bundle branch block aberrancy due to the "long-short" coupling. A premature ventricular contraction cannot be ruled out, although the septal R wave in V_1 is quite narrow (one can apply QRS morphology clues for differentiating ventricular tachycardia from supraventricular tachycardia with aberrant conduction to single wide QRS beats). In this case the patient does have

voltage criteria for left ventricular hypertrophy: S V_1 + R V_5 > 35 mm. Nonspecific ST changes are also present.

11. The patient has a ventricular pacemaker that inhibits appropriately when sensed intrinsic QRS complexes are present. The patient has ST changes during the intrinsic beats that suggest the use of digoxin. When evaluating a patient with a pacemaker it is important to attempt to evaluate the underlying atrial rhythm. In this case the patient is probably in atrial flutter with 2:1 conduction to the ventricles, with every other flutter wave “concealed” by the ST segment. Notice the inverted flutter waves in the inferior leads. An ectopic atrial rhythm with associated first degree AV block cannot be ruled out from this ECG. A longer rhythm strip with the development of spontaneous higher grade AV block (3:1, 4:1, etc.), or AV block induced by vagal maneuver or adenosine might help, but the pacemaker would have to be reprogrammed to a slower rate.
12. This is an ECG from a young man with no medical complaints. Notice the notched QRS and ST segment changes associated with early repolarization. Unusual notched T waves are observed in V_2 and V_3. While these could represent nonconducted premature atrial complexes, there is no evidence of atrial activity in the other leads. In this case the notched T waves remained even with changes in sinus rates, suggesting that the notches were coupled to ventricular activity rather than atrial activity.
13. In this ECG the patient is exercising (this is why baseline artifact is present). The patient has sinus tachycardia with left anterior fascicular block. As the sinus rate increases, the patient develops right bundle branch block due to refractoriness within the right bundle.
14. The patient has a right arm-right leg switch leading to the “flatline” signal in II. Fortunately, the precordial leads are not affected that show an anterior wall myocardial infarction with ST segment elevation and T wave inversion in V_1–V_4 and Q waves in V_1 and V_2. Lead III is unaffected by this switch (left arm-left leg), but the Q wave in lead aVF resolves once the leads are placed correctly.

Index

A

Accessory pathway (AP), 168–169, 171, 174
  See also Tachycardia
Action potentials (AP)
  fast response, 5
    ion permeabilities at rest and during, 6
  slow response, 7
    ion channel opening and closing in, 7–8
  See also Heart
Acute myocardial infarction
  anterior infarction, 94–96
  bundle branch block, 104–106
  cellular changes associated with, 81–83
  ECG changes in
    Q waves, 88–89
    ST segment changes, 85–93
    T wave peaking, 84–85, 89–93
  inferior myocardial infarction, 96–98
  and ischemia, 83–84
  lateral myocardial infarction, 98–100
  left main coronary artery, 102–103
  right ventricular infarction, 100–102
Amyloidosis, 259–263
Anterior infarction, 94–96
Arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/ARVD), 266–269
Ashman's phenomenon, 273–275
Atria, 8–9
  activation, 38
  depolarization, 21
  enlargement, 37
  fibrillation, 171
    atria activation of, 162
    ECG, 162–163, 198
    mechanisms of, 163
  flutter
    atypical atrial flutter, 165
    AV nodal block, ventricular rate, 164
    mitral valve and, 164
Atrial tachycardia (AT), 161, 174
  and AV nodal block, 171
  narrow QRS complex, sinus node rate, 158
Atrioventricular (AV), 174
  AV junctional tachycardia
    automatic junctional tachycardia, 167–168
    node reentry, 166–167
  block
    artifact mimicking, 144
    atria, 140
    case study, 152
    degree of, 143–146
    ECG clues for, 152
    pathologic study of people, 140–141
    PR interval and, 142–143, 145–146
    QRS complex, heart block, site of, 151
    types of, 146–151
    and Valsalva maneuver, 177
    ventricular depolarization, 153
    Wenckebach block for Karl, 145–146
  conduction, 21–23
  node, 134
Axis deviation, causes for abnormal, 277

B

Bazett's correction, 31
  See also Electrocardiography (ECG)
Bradycardia
  atrioventricular block, 142
    case study, 152–153
    ECG for, 147
    with first and third degree, 146, 149, 151
    high grade, 146, 150
    identification of site of, 152
    P waves within QRS complex and, 143
    QRS pattern, 151

Bradycardia *(Cont.)*
second degree, 143–144, 148, 151
Type I and II 2^0, 145, 149–150
vagally induced, 147–148, 150
case study, 139
and ECG manifestations, 141
sinus node
abnormal automaticity and, 139–140
dysfunction of, 141–142
Brugada syndrome, 119–120
and early repolarization, 112
ECG from patient with, 120
possible electrophysiologic mechanisms for, 121

C

Ca^{2+} ATPase and the Na^{+}-Ca^{2+} exchanger, 4
Cornell product, 43
See also Ventricular enlargement
Coronary artery spasm, 117

D

Defetilide, antiarrhythmic drugs, 74–75
Dextrocardia, 227
See also Heart
Digoxin toxicity, 272–273
and ST segments downsloping, 68
Duchenne's muscular dystrophy, *see* Muscular dystrophy

E

Early afterdepolarization (EAD), triggered activity, 156
Early repolarization
and Brugada syndrome, 112
ST segment elevation, 111
Einthoven's triangle, 13–14
See also Electrode recording system
Electrocardiography (ECG)
with abnormal repolarization with inverted T waves, 31
accessory pathway, tachycardia, 173
after cardioversion, 241
anterior myocardial infarction, 94–95
arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/ARVD), 266–269
artifact, 270–272
asynchronous ventricular pacing, 207
atrial depolarization, 21
P wave representation, 22
atrial enlargement, 37–38
atrial flutter, 239
atrioventricular conduction, 21–22
AV node reentry with, 172
blockage in left main coronary artery, 103
with cardiac amyloidosis, 261
case of
abdominal surgery and irregular heart rate, 235–239
chest pain with, 229–231
episodes of intermittent lightheadedness, 233–235
lightheadedness and rapid heart beat, 242–247
shortness of breath and chest pain, 231–233
shortness of breath with exertion, 225–229
sudden onset of lightheadedness, 239–242
chest/precordial leads, 16–17
chronic obstructive lung disease, 262
2-day old boy, 227
depolarization, 11, 13
derived 12-lead ECG
EASI system, 17
with dextrocardia, 228
with Duchenne's muscular dystrophy, 266
electrode misplacement and, 269–270
electrolyte disorders and
calcium, 253–255
magnesium, 255–256
potassium, 249–253
format in columns, 18
in frontal plane, 25–26
gold standard and, 44
heart rate and PR interval, 30
with hyperkalemia, 252
in hypocalcemia, 253–254
hypomagnesemia, 255
hypothermia, 264–265
inferior wall myocardial infarction, 97, 99

intervals, 29
irregular tachycardias, examples of, 170
junctional tachycardia of, 167
large inferior and lateral wall myocardial infarction, 93
in left bundle branch block, 53, 105
with right bundle branch block, 52
limb lead systems, 13
location of standard positions for electrodes, 13–15
low voltage, 281
muscular dystrophy, 265–266
Na^+ channel blocker flecainide with, 240
nomenclature for ventricular activation, 24–25
with pacing from right ventricular apex, 212
posterior wall myocardial infarction, 98
in precordial plane
ventricular repolarization, 27–29
precordial QRS morphology, 192–195
pulmonary embolism with S1Q3T3 pattern, 260
QRS interval, 30
QT interval, 30–31
readings, 222–223, 284–292
recording systems, 15
repolarization, 13
right bundle branch block, 106
severe hypothermia, 265
slow heart rates and narrow complex tachycardia, 281
during ST elevation myocardial infarction, 92, 102
struck by lightning, 264
systematic analysis, assess
quality, 217–218
rate and rhythm, 218
ventricular depolarization and repolarization, 219–222
three-cell model for measuring signals, 12
unipolar/augmented leads, 15–16
ventricular depolarization, 23–24
ventricular enlargement
left ventricle, 39–44
right ventricle, 44–45
ventricular parasystole, 273
wide complex tachycardia, LBBB pattern, 267
in Wolff-Parkinson-White syndrome, 243
Electrode recording system
chest/precordial leads, 16–17
derived 12-lead ECG, 17
limb lead
bipolar leads, 13, 15
unipolar/augmented leads, 15–16
Electrolyte disorders
hypercalcemia, 253–255
hyperkalemia, 121–122, 250–253
hypocalcemia, 253
hypokalemia, 249–250
hypomagnesemia, 255–256
hypothermia, 264–265

F

Focal atrial tachycardia, 158
P wave morphology in, 159–161

H

Heart
cardiac anatomy
septal region of atria, 9
sinus node, 8
cellular membrane, 3
action potentials, 5
protein pumps and exchangers, 4
ICD function and, 268
and interpretation of ECGs, 4–9
irregular heart rhythm and premature atrial complexes, 130
normal activation of, 8
Hemachromatosis, infiltrative diseases, 260
His-Purkinje system, 9, 132, 134
His bundle
anatomy of, 49
ECG for, 50
left anterior fascicular block, 55–57
left bundle branch block, 53–55
left hand as model for bundles, 51
left posterior fascicular block, 57–58
right bundle branch block, case study, 50–52
His-Purkinje tissue, 169

I
Ibutilide, antiarrhythmic drugs, 74
Inferior myocardial infarction, 96–98
Ion channel
ion permeabilities at rest and during cardiac action potential (AP), 6
opening and closing in slow response cells, 7

J
J point elevation, 111–112
See also ST segment
Junctional tachycardia
ECG of, 167

L
Lateral myocardial infarction, 99–100
Left anterior fascicular block (LAFB), 55
ECG in, 57
hand model for, 56
Left bundle branch block, 112–114
Left posterior fascicular block (LPFB), 57
case study, 58
ECG in, 58
Left ventricular aneurysms
ECG from patient with, 118
Left ventricular hypertrophy, 112–113
Lightning strike and sudden cardiac death, 263–264
Long QT syndrome, 70–71

M
Mason-Likar limb leads, 15
M-Cells and ion channels, 67
Multifocal atrial tachycardia, 161–162, 171
12-lead ECG from, 162
Muscular dystrophy, 265–266

P
Pacemakers, implantable devices
biventricular pacing, 212
case study, 212–213
dual chamber
case study, 210
rapid wide complex rhythm, 211
in right ventricular apex, 212
timing cycles for, 209
inferolateral wall of left ventricle, 208
single chamber, 205–209
ventricular pacing from right ventricular outflow tract, 208
Parasytole, 272
Pericarditis
diffuse ST segment elevation, 116
myocardial infarction, 117
ECG in patient with, 115
vs. myocardial infarction, 116
positive electrode of VR in, 115
PR segment elevation, 116
Permanent junctional reciprocating tachycardia (PJRT), 175–176
Premature atrial contractions, 132
AV node and ventricular activation, response, 129
early P wave, 129
irregular heart rhythm, 130
nonconducted premature atrial complexes, 131
Premature beats
sinus node and intrinsic rate of automaticity
case study, 129
Premature junctional complex
early normal-appearing QRS, 134–135
Premature ventricular contractions (PVCs)
His bundle tissue and AV node, 132–133
and QRS complex, 132–133
significance in heart disease and, 133–134
Pulmonary embolism
ECG findings in, 120
Osborn waves observed in, 121
Pulmonary embolus, 255–256
P wave
ectopic atrial focus, 129
See also Premature atrial contractions

Q
QRS complexes, 112–114
atria, retrograde activation of, 135
cause of, 134
deep S waves, 111
funny waves, 282
with left bundle branch block, 133
morphology of, 132
P wave, 134
location of, 171
short R-P tachycardia, 171–172

Q waves, 88–89
causes for, 226
differential diagnosis for, 278
genesis of, 89

R
Right coronary artery occlusion, 101
Right ventricular infarction, 100–102
Romhilt-Estes scoring system for left ventricular hypertrophy, 43–44
R waves, 40, 279
poor progression, 263

S
Sarcoidosis, infiltrative diseases, 260–261
Sawtooth flutter waves, 164
Sinus node
bradycardia, 139
diastolic depolarization, 139–140
dysfunction, ECG manifestation of, 141–142
ectopic atrial rhythm, 141–142
premature beats, 129
rate and atrial tachycardia, 158
structure, 8–9
timing cycles for, 206
Sinus rhythm, case study, 134
Sinus tachycardia, 158
ventricular rhythms, accelerated, 155–156
Sokolow criteria for left ventricular hypertrophy, 40
Sotalol, antiarrhythmic drugs, 74–75
ST segment, 81
elevation
Brugada Syndrome, 119–120
cause of, ECG clues, 113
coronary artery spasm, 117
and early repolarization, 112, 114
hyperkalemia, 121–122
J point and T wave, 111
left bundle branch block, 112–114
left ventricular aneurysms, 118
left ventricular hypertrophy, 112–114
notch, cellular mechanism of, 112
pericarditis seldom, 115–117
pulmonary embolism, 120
transient outward current, 112
transthoracic cardioversion, 117
Supraventricular tachycardia, 155
anatomic classification
atrial tachycardia, 158–165
AV junctional tachycardia, 166–168
AV node, accessory pathway mediated, 168–169
case study, 177–178
ECG with adenosine, 179
causes of, 178
diagnostic algorithms for evaluation, 178
electrocardiographic diagnosis, 169–170
AV node dependent *vs.* independent, 176–179
P wave location and morphology, 171–176
regular *vs.* irregular, 170–171
mechanistic causes for fast heart rate
increased automaticity, 155–156
reentry, 156–158
triggered activity, 156
termination of, 177

T
Tachycardia
accessory pathway mediated, 168–169, 171
adenosine, effect of, 177
atrial tachycardia, 158–159
cellular/tissue mechanisms for, 157
ECG examples of, 170
flow diagram for, 169–170
increased automaticity, 155
long R-P tachycardias
differential diagnosis, 175
termination of, 178
P wave, 175–176
reentry, 156, 158
short R-P tachycardias, differential diagnosis, 174
sodium/calcium channels, reactivation of, 156
Takotsubo syndrome, 118
ECG from patient with, 119
Three-cell model for measuring ECG signals, 12
Transient outward current (Ito), 112
Transthoracic cardioversion, 117

T wave
changes and causes, 280
peaking, 84–85, 92

V
Ventricles, 9–10
depolarization, 23–24
repolarization, 27–29
T waves, 65–77
U waves, 77–78
Ventricular arrhythmias and ICD, 268
Ventricular enlargement
left ventricle, hypertrophy and, 39
Cornell product, 43
ECG depolarization forces in, 40–41
repolarization changes, 41–43
Romhilt-Estes scoring system for, 43–44
sensitivity and specificity of ECG criteria for, 44
right ventricle, 44
ECG in hypertrophy of, 45
Ventricular tachycardias, 183–184, 192, 196
pathophysiologic causes for, 185

W
Wenckebach block for Karl, 145–146
Wide complex tachycardia
case study, 183
differential diagnosis of, 184
ECG analysis
atrial-ventricular relationship, 187–189
clinical clues, 186–187
initiation and termination, 189–190
precordial QRS morphology, 192–197
rate and axis, 190–192
irregular wide complex, 198–200
pathophysiology, 183–185
Wolff-Parkinson-White syndrome, 228
accessory pathway in, 244
with right-sided accessory pathway, 245