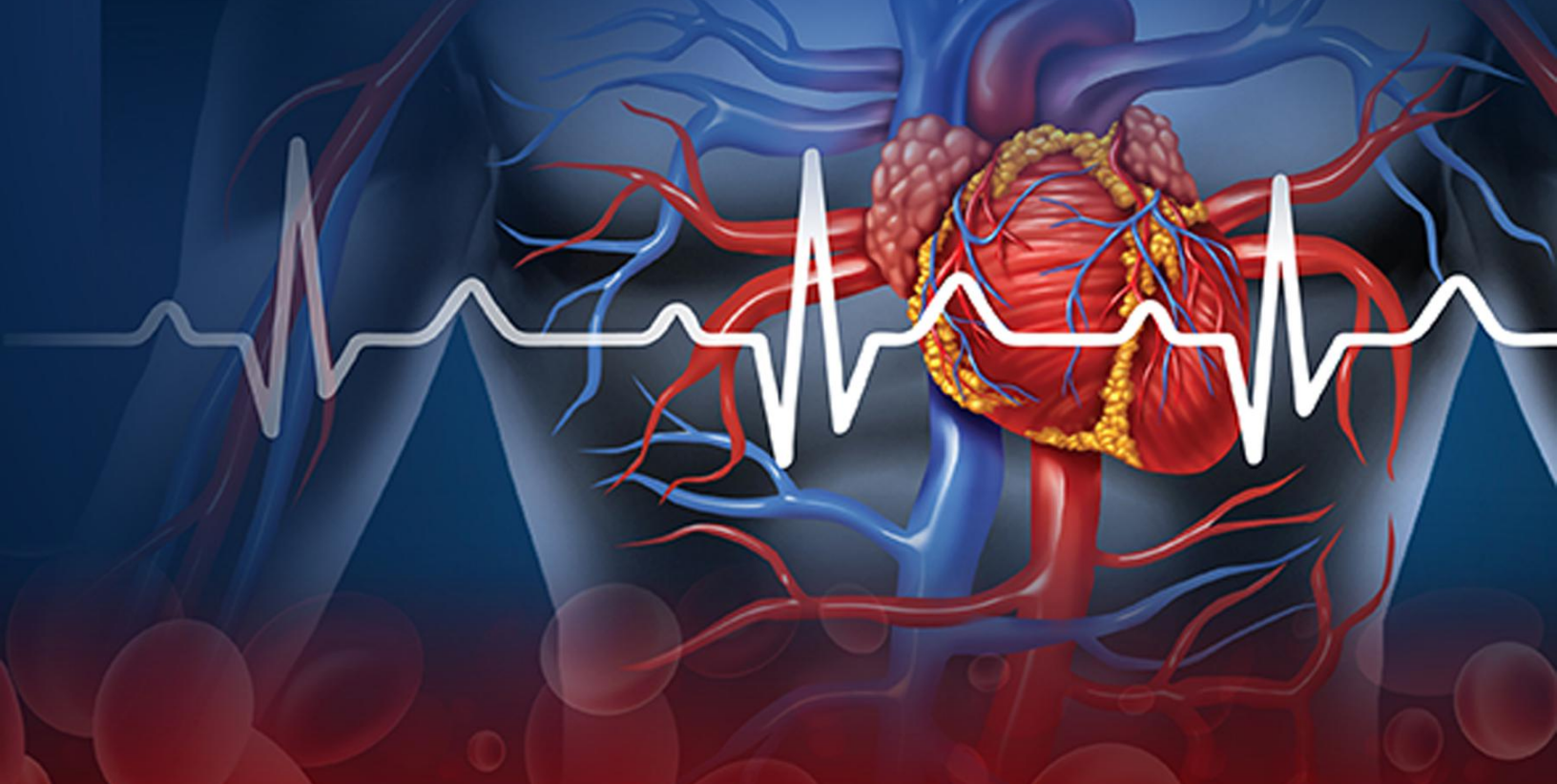
HUSZAR'S

ECG AND 12-LEAD INTERPRETATION

SIXTH EDITION



KEITH WESLEY, MD, FACEP, FAEMS

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KEITH WESLEY, MD, FACEP, FAEMS

Medical Director
United Emergency Medical Response
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To all the emergency medical technicians, paramedics, physicians,
and nurses I've had the honor to work with during my career.
You are the tireless heroes of medicine.

To my wife, Karen.
You are the light of my life.

To my three sons: JT, Austin, and Camden.
Thank you for being such incredible men.

And to their wives: Nikki, Jen, and Brenna.
Thank you for loving them as much as I do.

To all you: without your love, confidence, and patience,
I could not have pursued my dream.

I love you.

Keith Wesley, MD, FACEP, FAEMS

ABOUT THE AUTHOR

Keith Wesley is board certified in emergency medicine and holds subspecialty certification in emergency medical service (EMS) medicine. Originally from Tyler, Texas, he graduated from Brigham Young University in 1982 and Baylor College of Medicine in Houston, Texas, in 1986. He completed an emergency medicine residency at Methodist Hospital in Indianapolis, Indiana, where he gained his first exposure to EMS flying air medical missions.

Dr. Wesley has been involved in EMS since 1989, working with many services in Wisconsin. In 1992, he was selected by the governor as a founding member of the Wisconsin State Physician Advisory Committee and served for 12 years, the last 4 years as chair.

From 1992 to 2004, Dr. Wesley was a clinical assistant professor at the University of Wisconsin Family Practice Residency in Eau Claire, Wisconsin. There he was responsible for the training and education of family practice residents rotating through the emergency department.

In 2006, Dr. Wesley was selected as the Wisconsin State EMS medical director. He held that position until 2008, when he moved his practice to Minnesota and accepted the position of Minnesota State EMS medical director, which he held until 2010. From 2008 to 2020, he worked for M Health Fairview EMS, formally HealthEast Medical Transportation, in St. Paul, Minnesota, as the EMS medical director for Emergency Medical Services. From 2007 to the present, he has been the medical director for United Emergency Medical Response in Wisconsin Rapids, Wisconsin.

Dr. Wesley is a former chair of the National Council of State EMS Medical Directors and is active in the National Association of EMS Physicians. He has coauthored four textbooks and numerous articles and papers and is a frequent speaker at state and national conferences. He is currently on the editorial board of *JEMS* magazine.

An active member of the American College of Emergency Physicians and the National Association of EMS Physicians, Dr. Wesley has been actively involved in creating educational programs for medical and nursing students, emergency medical technicians (EMTs), and physicians.

When not engaged in EMS duties, Dr. Wesley enjoys spending time with his wife, Karen, who is a retired police officer and tactical paramedic. They live in Eau Claire, Wisconsin, with their golden retriever, Charly, and shih tzu, Sammie.

FOREWORD TO THE SIXTH EDITION

Dr. Keith Wesley's *Huszar's ECG and 12-Lead Interpretation* has been a mainstay of emergency medical services (EMS) and allied health education for decades. It has always provided a pragmatic approach to the interpretation and utilization of the electrocardiogram (ECG) in the clinical setting. The sixth edition is an important update for this essential textbook and reflects the evolving science and practice of acute cardiac care.

I had the honor of meeting Dr. Robert J. Huszar many years ago when this book was released by a different publisher. He was passionate about cardiology from an emergency standpoint. He was witty and knowledgeable and had a great amount of respect for those learning ECGs and ECG monitoring. When I was instructing paramedics in the late 1970s in Texas, we always required Dr. Huszar's book (then called *Emergency Cardiac Care*) for our students for ECG interpretation instruction. Dr. Keith Wesley is a fellow emergency physician and longtime friend and colleague. He has taken Dr. Huszar's book to a new level, which is why it remains popular decades later. He has continued the great tradition of Dr. Huszar.

The Centers for Disease Control and Prevention reports that one person dies every 37 seconds in the United States from cardiovascular disease. As with many medical conditions, early diagnosis and treatment of certain cardiovascular conditions are essential. Electrocardiography, the science of the ECG, is one of the most sensitive tools was have for rapidly detecting heart attacks and other life-threatening conditions. Because of this, EMS providers and other healthcare practitioners must be adept at ECG recognition and interpretation. Drs. Huszar and Wesley have recognized this, and using the book you are holding, they will help you understand ECGs and help ensure that your patients with acute cardiac conditions receive the prompt and efficient emergency care they need.

The sixth edition of *Huszar's ECG and 12-Lead Interpretation* has been extensively updated and provides a concise and comprehensive discussion of the science of electrocardiography. It is an excellent educational tool and reference for any provider who deals with ECG monitoring and interpretation.

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PUBLISHER'S NOTE

The author and publisher have made every attempt to check dosages and advanced life support content for accuracy. The care procedures presented here represent accepted practices in the United States. They are not offered as a standard of care. Advanced life support–level emergency care is performed under the authority of a licensed physician. It is the student's responsibility to know and follow local care protocols as provided by his or her medical advisors. It is also the student's responsibility to stay informed of changes in emergency care procedures, including the most recent guidelines set forth by the American Heart Association and printed in their textbooks.

PREFACE

This text was written to teach medical, nursing, and emergency medical service (EMS) providers basic skills in cardiac rhythm interpretation. It also provides a wealth of advanced instruction in the clinical signs, symptoms, and management of patients presenting with cardiac dysrhythmias.

With the advent of electrocardiogram (ECG) monitoring has come readily accessible 12-lead electrocardiography, an essential tool in the detection and management of acute coronary syndromes. Accordingly, this edition has several chapters dedicated to 12-lead ECG interpretation. The book also offers in-depth coverage of the pathophysiology, clinical signs and symptoms, and management of acute coronary syndromes.

Each rhythm is first presented in its classic form, with a quick-reference box outlining its unique characteristics. The accompanying text contains a detailed explanation of these features and a discussion of the range of variability and the possible exceptions to each pattern. Many of the rhythm strips are from real patients. Thus they do not necessarily have all of the classic characteristics described in the text. That's the challenge of ECG rhythm interpretation.

The treatment algorithms are based on the latest resuscitation guidelines issued by the American Heart Association and the American College of Cardiology. However, because the science continues to evolve and local policy and protocol may vary, it's important to stay abreast of new treatments as they evolve.

This edition of the text offers information in a more visual format that allows quick reference and review.

ECG KEYS BOXES

Skillful diagnosis and treatment of rhythm disorders are based on mastery of a body of foundational knowledge. ECG Keys boxes highlight this core information, such as clinical indications and end points for the administration of certain agents.

AUTHOR'S NOTES

It's critical to be aware of the broad range of variables that might make a patient's rhythm look less than classic. In Author's Notes, I point out some of these considerations. Many of the notes pertain to diagnosis or treatment and not strictly interpretation. For example, I advise asking patients about discomfort, not just pain, when taking a history. Author's Notes also point out ways in which my recommendations may differ from the course of action suggested in other texts or required by local protocols.

KEY DEFINITIONS

This text contains a full glossary. Key Definitions call attention to the most relevant terms, making them easily accessible while you're reviewing the surrounding topics. These on-page definitions often elaborate on the information given in the glossary.

TAKE-HOME POINTS

When you don't have time to read or reread an entire chapter, Take-Home Points hit the highlights. This bulleted summary gives you need-to-know information about the most important topics covered.

CHAPTER REVIEW QUESTIONS

Appendix A provides answers to the Chapter Review Questions sections to check your knowledge of the main points presented. Appendix B contains 290 ECG rhythms for interpretation, along with case scenarios, with answers provided in Appendix C.

Each chapter in this book builds on the skills and principles explored earlier. By moving sequentially through the chapters, you'll have all the tools you need for accurate rhythm interpretation, diagnosis, and clinical management. If you're a seasoned veteran, you already know how rewarding such competence can be. If you're a student, welcome to this exciting, critical, and sometimes challenging subject.

Keith Wesley, MD, FACEP, FAEMS

ACKNOWLEDGMENTS

First, I would like to thank the dedicated men and women of M Health Fairview EMS, formally HealthEast Medical Transportation, and United Emergency Medical Response, who provided me encouragement, criticism, and reams of rhythm strips and 12 leads.

Next, I must acknowledge the successful foundation upon which this book is based. Dr. Huszar expertly crafted this text in its first three editions. To be given the opportunity to carry on where this great man left off is an honor.

Keith Wesley, MD, FACEP, FAEMS

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CONTENTS

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HUSZAR'S

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1 Anatomy and Physiology of the Heart

OUTLINE

CARDIAC ANATOMY AND PHYSIOLOGY

The primary purpose of the heart is to pump blood through the circulatory system. The heart is a muscle made up of four chambers (Fig. 1.1) arranged in pairs: the upper chambers are called *atria,* and the lower chambers are called *ventricles.* The two upper chambers, the *right and left atria,* have thin walls. The two lower chambers, the *right and left ventricles,* have thick, muscular walls. The point at which the two atria connect to the vascular system is referred to as the *base of the heart.* The ventricles form a rounded cone attached at the *apex of the heart.* This arrangement may seem a bit backward when you look at a diagram of the heart because the base is at the top and the apex at the bottom (see Fig. 1.1).

AUTHOR'S NOTE The heart is also part of the endocrine system. It secretes hormones that help regulate blood pressure and kidney function. In this text, we will limit our discussion to the heart's role in pumping blood.

Composition

The walls of the atria and ventricles are composed of three layers of tissue (Fig. 1.2):

1. **Endocardium.** The innermost layer, called the *endocardium,* is thin and smooth. The endocardium reduces friction between the blood and the inner walls of the atria and ventricles.
2. **Myocardium.** The middle layer is called the *myocardium.* It contains the muscle cells that contract and relax. In the ventricles, where the muscular walls are much thicker than in the atria, the myocardium is divided into the *subendocardial area,* which is the inner half of the myocardium, and the *subepicardial area,* the outer half of the myocardium.
3. **Epicardium.** The outermost tissue layer, the *epicardium,* is a thin layer of smooth connective tissue similar to the endocardium. The epicardium reduces friction between the heart and the pericardial sac that envelops it.

The walls of the left ventricle are more muscular and about three times thicker than the walls of the right ventricle. The atrial walls, like those of the ventricles, are also composed of three layers of tissue, but the middle muscular layer of the atrial walls is much thinner than that of the ventricles.

AUTHOR'S NOTE The epicardium is also referred to as the *visceral pericardium.*

Protection

The heart is enclosed in a protective dual-layered membrane called the *pericardium,* or *pericardial sac.* Its tough outer layer, the parietal pericardium (see Fig. 1.2), comes into direct contact with the lungs and the diaphragm. The inner layer of the pericardium is called the *visceral pericardium.* It is in contact with the outer surface of the heart. The space between the visceral pericardium and the parietal pericardium contains a small amount of pericardial fluid. This fluid reduces friction between the beating heart and the pericardial sac.

Inferiorly (at the bottom), the pericardium is attached to the center of the diaphragm. Anteriorly (at the front), it's attached to the sternum; posteriorly (at the back), to the esophagus,

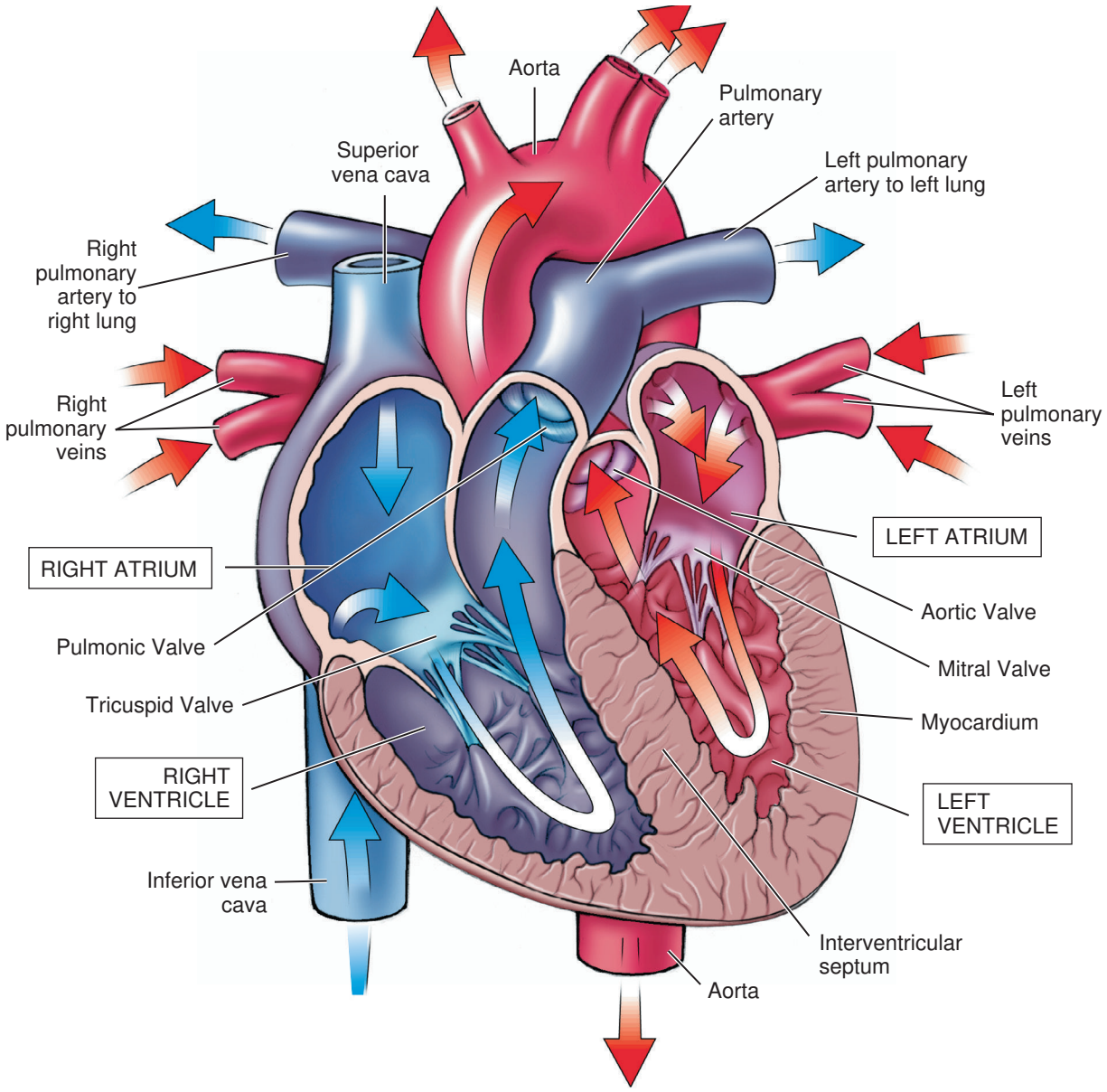


FIG. 1.1 Anatomy and circulation of blood through the heart. (Modified from Herlihy, B. [2011]. *The human body in health and illness* [4th ed.]. Saunders.)

trachea, and main bronchi; and at the base (top) of the heart, to the aorta, the superior and inferior vena cava (collectively referred to as the *venae cavae*), and the right and left pulmonary veins. In this way, the pericardium anchors the heart to the chest and limits its movement within the mediastinum.

Two Pumps

The interatrial septum (a thin, membranous wall) separates the two atria. A thicker, more muscular wall, the interventricular septum, separates the two ventricles. These two septa, in effect, divide the heart lengthwise into two pumping systems: the right heart and the left heart. Each consists of an atrium and a ventricle.

RIGHT HEART

The right heart pumps blood into the pulmonary circulation. The left heart pumps blood into the systemic circulation. The systemic circulation includes the arteries that supply blood to the body and the arteries of the coronary circulation, which supply blood to the heart.

The right atrium receives deoxygenated blood from the body from two sources:

1. Two of the body's largest veins, the superior vena cava and inferior vena cava (together called the *venae cavae*).
2. The coronary sinus, a large vein located on the back side of the heart. This vein receives venous blood from the coronary circulation.

The blood in the right atrium is then delivered to the right ventricle through the tricuspid valve. Next, the right ventricle pumps the deoxygenated blood through the pulmonary valve and into the lungs through the right and left pulmonary arteries. In the lungs, the blood picks up oxygen and releases carbon dioxide.

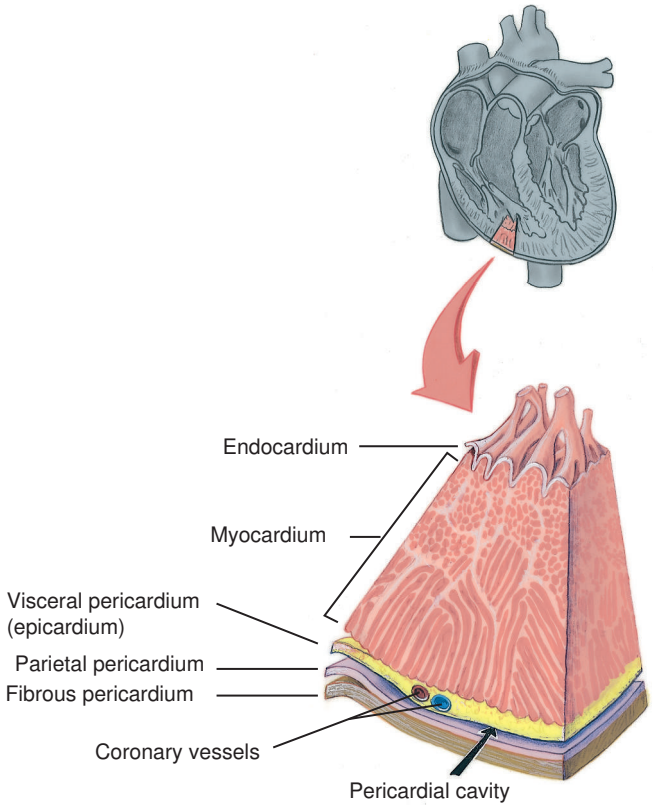


FIG. 1.2 Pericardium and pleura. (Applegate, E. [2011]. *The anatomy and physiology learning system* [4th ed.]. Saunders.)

LEFT HEART

The left atrium receives the newly oxygenated blood from the lungs via the right and left pulmonary veins and delivers it to the left ventricle through the mitral valve. The left ventricle then pumps the oxygenated blood out through the aortic valve and into the aorta, the largest artery in the body. From there, the blood is distributed throughout the body, including the heart, where the blood supplies oxygen to the cells.

ATRIAL AND VENTRICULAR DIASTOLE AND SYSTOLE

The heart performs its pumping action repeatedly in a rhythmic sequence, as follows (Fig. 1.3):

- First, the atria relax (atrial diastole), allowing the blood to pour in from the venae cavae and pulmonary veins. The pressure of blood in the venae cavae and pulmonary veins is referred to as *preload.*
- As the atria fill with blood, the ventricles begin to relax. As the atrial pressure rises above that in the ventricles, the tricuspid and mitral valves (collectively called the *atrioventricular valves*) open. Blood empties rapidly through the open valves into the relaxed ventricles.
- Then the atria contract (atrial systole) to maintain the preload pressure required to keep the atrioventricular valves open. Blood continues to fill the ventricles to capacity. Toward the end of atrial contraction, the pressure in the atria and ventricles equalizes, and the tricuspid and mitral valves begin to close.
- Next, the ventricles contract vigorously (ventricular systole). This causes a sharp spike in ventricular pressure. As the tricuspid and mitral valves close completely, the aortic and pulmonary valves snap open. This allows the forceful ejection of blood into the pulmonary and systemic circulation.
- Meanwhile, the atria have again relaxed and begun filling with blood. As soon as the ventricles empty and begin to relax (ventricular diastole), the ventricular pressure falls, the aortic and pulmonary valves shut tightly, the tricuspid and mitral valves open, and the rhythmic cardiac sequence begins anew.

AUTHOR'S NOTE Coronary perfusion occurs during ventricular diastole, when the aortic valve is closed.

The sequence of one ventricular systole followed by ventricular diastole is called a *cardiac cycle.* The cardiac cycle extends from the initiation of atrial contraction to ventricular relaxation.

ELECTRICAL CONDUCTION SYSTEM OF THE HEART

The conduction system of the heart (Fig. 1.4) is composed of the following structures:

- The sinoatrial (SA) node
- The conduction tracts between the SA and AV nodes and the conduction tract between the atria (Bachmann's bundle)
- The atrioventricular (AV) junction, consisting of the AV node and bundle of His
- The right bundle branch and the left bundle branch and its anterior and posterior small conduction tracts, called *fascicles*
- The Purkinje network

The SA node lies in the wall of the right atrium near the inlet of the superior vena cava. It consists of pacemaker cells that generate electrical impulses automatically and regularly. These impulses travel to the atria and ventricles, causing them to contract (Fig. 1.5).

Three conduction tracts run through the walls of the right atrium between the SA node and the AV node: the anterior, middle, and posterior internodal tracts. These tracts conduct the electrical impulse from the SA node to the AV node in 0.03 seconds. The interatrial conduction tract (Bachmann's bundle), a branch of the anterior internodal tract, extends across the atria, conducting the electrical impulses from the SA node to the left atrium.

The AV node, the proximal part of the AV junction, lies partly in the right side of the interatrial septum in front of the opening of the coronary sinus and partly in the upper part of the interventricular septum above the base of the tricuspid valve.

The AV node consists of three regions:

1. **Atrionodal region.** The small upper region, located between the lower part of the atria and the nodal region, is called the *atrionodal region.*

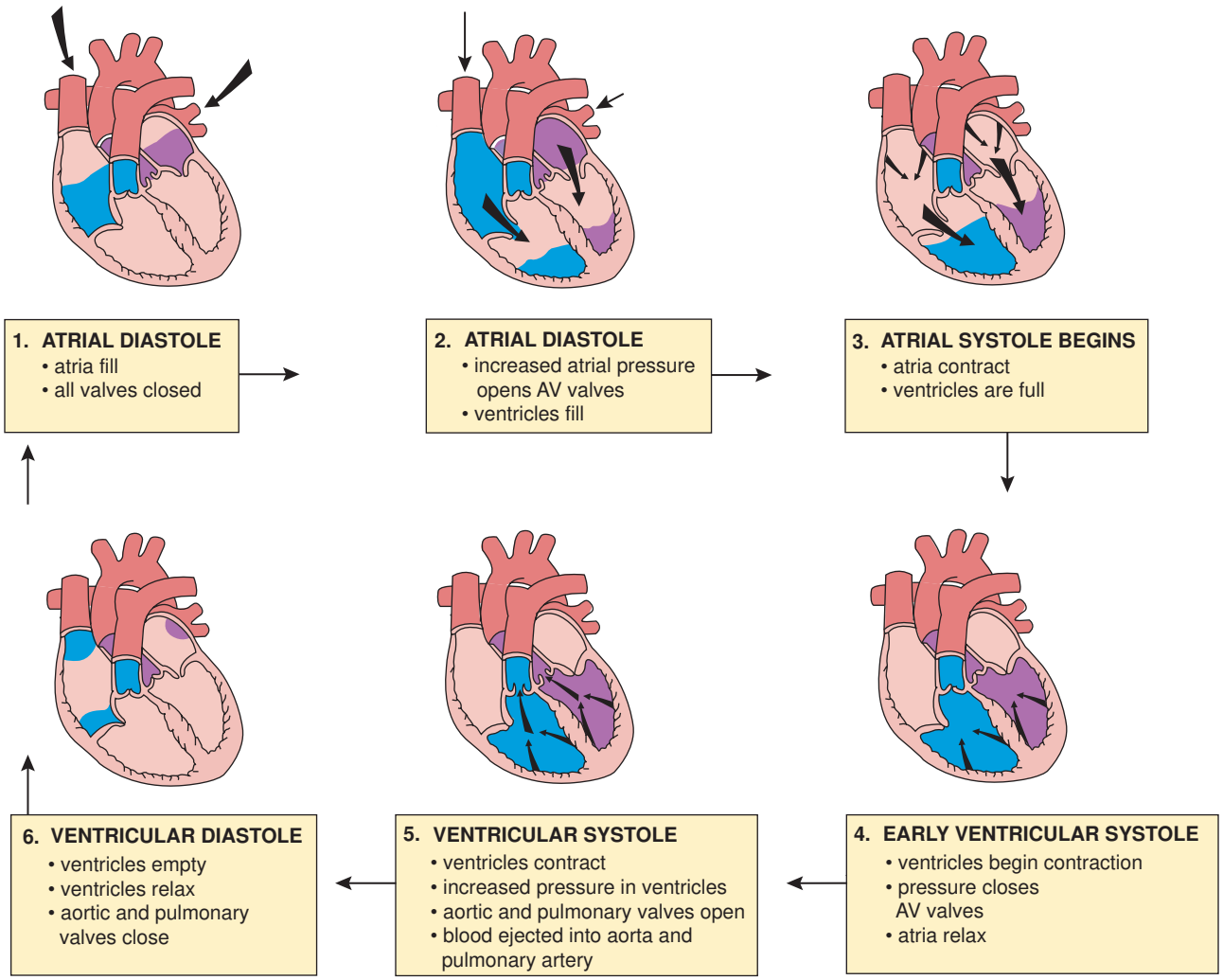


FIG. 1.3 Ventricular diastole and systole. (VanMeter, K. C., & Hubert, R. J. [2014]. *Gould's pathophysiology for the health professions* [5th ed.]. Saunders.)

2. **Middle nodal region.** The large, central area of the AV node is called the *middle nodal region.* In this area, the progression of electrical impulses from the atria to the ventricles is slowed.
3. **Nodal-His region.** The small, lower nodal-His region is located between the nodal region and the bundle of His. The atrionodal and nodal-His regions contain pacemaker cells (described later in the chapter), whereas the nodal region does not.

The primary function of the AV node is to ensure that electrical impulses from the atria to the bundle of His follow the most efficient pathway and to slow their progression so that they arrive at the ventricles after the ventricles have filled with blood. A ring of fibrous tissue insulates the remainder of the atria from the ventricles, preventing electrical impulses from entering the ventricles except through the AV node, unless there are accessory conduction pathways, as described later.

The electrical impulses slow as they travel through the AV node, taking about 0.06 to 0.12 seconds to reach the bundle of His. This delay allows the atria time to contract and empty and the ventricles to fill completely before they (the ventricles) are stimulated to contract.

The bundle of His, the distal part of the AV junction, lies in the upper part of the interventricular septum. It connects the AV node to the two bundle branches. Once the electrical impulses enter the bundle of His, they travel rapidly through the fibrous tissue that electrically separates the atria from the ventricles and enter the bundle branches.

The right and left bundle branches arise from the bundle of His. The bundle of His, the right and left bundle branches, and the Purkinje network are also known as the *His–Purkinje system of the ventricles*. Pacemaker cells are located throughout the His–Purkinje system.

The bundle branches and their fascicles subdivide into smaller and smaller branches, with the smallest ones connecting with the Purkinje network. This intricate web of tiny fibers, distributed widely throughout the ventricles beneath the endocardium, conducts the electrical impulses. The ends of the Purkinje fibers terminate at the myocardial cells.

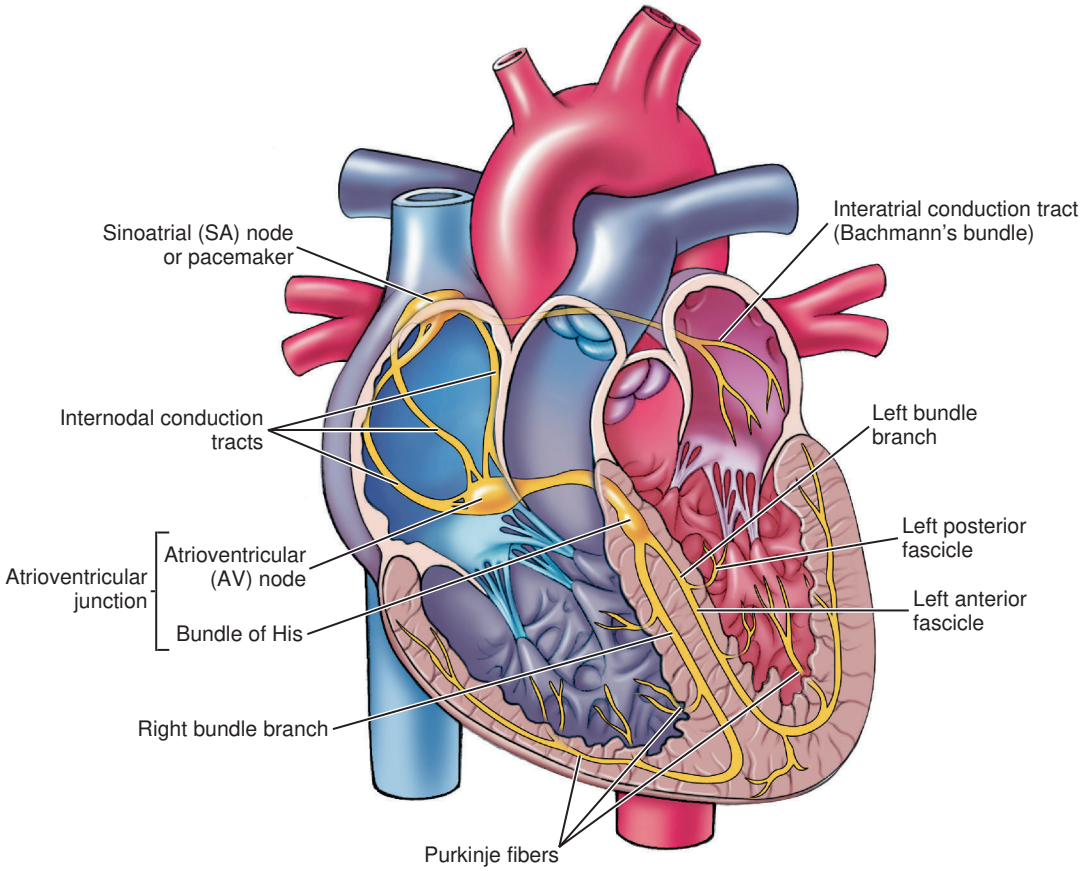


FIG. 1.4 Electrical conduction system. (Modified from Herlihy, B. [2011]. *The human body in health and illness* [4th ed.]. Saunders.)

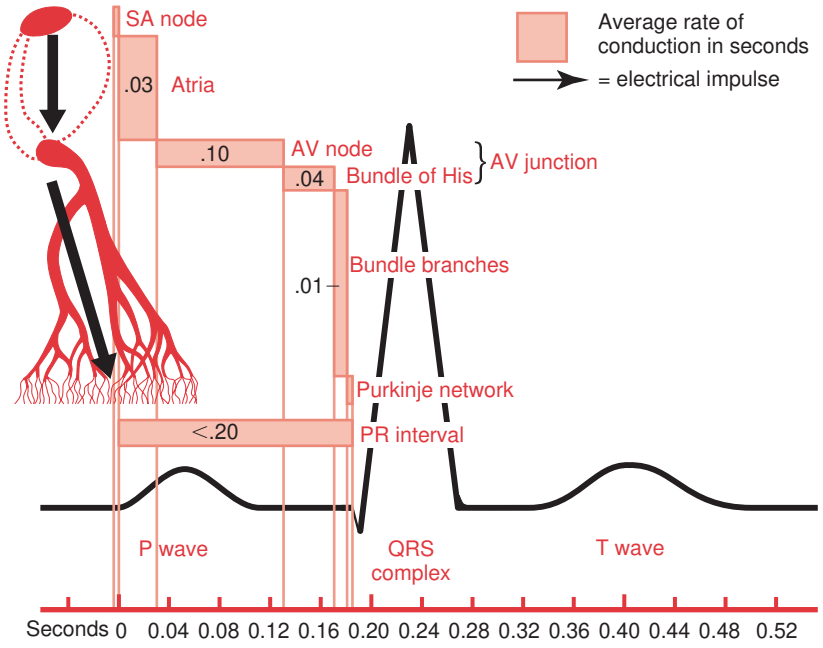


FIG. 1.5 The average rate of conduction of the electrical impulse through various parts of the electrical conduction system.

The electrical impulses travel very rapidly (in less than 0.01 second) through the bundle branches to the Purkinje network. Overall, it normally takes an electrical impulse less than 0.02 seconds to travel from the SA node to the Purkinje network in the ventricles.

CARDIAC CELLS

There are two basic kinds of cardiac cells—the myocardial, or "working," cells and the pacemaker cells of the electrical conduction system (Fig. 1.6 and Table 1.1).

Myocardial Cells

Myocardial cells are cylindrical. At their ends, they partially divide into two or more branches. These branches connect to the ends of adjacent cells, forming a network of cells called a *syncytium* (pronounced *syn-SIE-shee-um,* from *syn-,* meaning "together," and *cyto-,* meaning "cell"). At the junctions where the branches intersect, there are specialized cell membranes not found in any other cells. These membranes are called the *intercalated disks.* They contain areas of low electrical resistance called *gap junctions.* They permit very rapid conduction of electrical impulses from one cell to another. The ability of cardiac cells to conduct electrical impulses is called the *property of conductivity.*

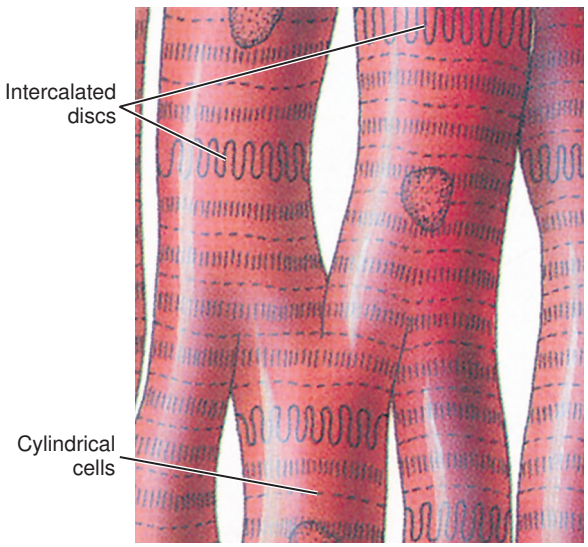


FIG. 1.6 Cardiac cells. (Modified from McCance, K. L., & Huether, S. E. [2015]. *Pathophysiology: The biologic basis for disease in adults and children* (5th ed.). Mosby.)

TABLE 1.1 Cardiac Cells and Their Function

Type	Primary Function
Myocardial cells	Contraction and relaxation
Pacemaker cells	Generation and conduction of electrical impulses

Each myocardial cell is enclosed in a semipermeable membrane. This membrane allows charged chemical particles, called *ions,* to flow in and out of the cells. The ability of sodium, potassium, and calcium ions to enter and leave myocardial cells allows the heart to contract and relax.

AUTHOR'S NOTE Recall that a permeable cell membrane allows ions to flow freely through the cell wall. A cell membrane that is impermeable, on the other hand, does not permit the flow of ions across it. A semipermeable cell membrane is selective, allowing only certain ions to enter and leave the cell.

The myocardial cells form the thin muscular layer of the atrial wall and the much thicker muscular layer of the ventricular wall (myocardium). These cells contain many thin muscle fibers, or *myofibrils,* made up of protein filaments called *actin* and *myosin.* Myofibrils give myocardial cells the property of contractility, that is, the unique ability to shorten when stimulated by an electrical impulse and then return to their original length.

AUTHOR'S NOTE The force of myocardial contractility increases in response to certain drugs (for example, digitalis and stimulants) and physiologic conditions such as increased venous return to the heart, exercise, emotion, hypovolemia, and anemia. In contrast, drugs such as procainamide, quinidine, beta blockers, and potassium, as well as conditions such as hypocalcemia and hypothyroidism, decrease the force of myocardial contractility.

Pacemaker Cells

The pacemaker cells of the heart's electrical conduction system contain no myofibrils and therefore cannot contract. They do, however, contain more gap junctions than do myocardial cells. Thus they can conduct electrical impulses very rapidly—at least six times faster than myocardial cells. The pacemaker cells are also capable of generating electrical impulses spontaneously. Myocardial cells, on the other hand, cannot normally do so. This capability, known as the *property of automaticity,* will be discussed in detail later in this chapter.

ELECTROPHYSIOLOGY OF THE HEART

The human heart is regulated by electrical impulses. Pacemaker cells in the heart are capable of generating and conducting the electrical impulses. The myocardial cells are capable of contracting but are also capable of conducting electrical impulses to adjacent myocardial cells, although they do so less efficiently and at a slower rate than do the specialized pacemaker cells. These electrical impulses are conducted because of the brief but rapid flow of positively charged ions (primarily sodium and potassium and, to a lesser extent, calcium) back and forth across the pacemaker cell membrane. The difference in the concentration of these ions inside compared with outside the cell membrane at any given instant produces an electrical

potential. This potential energy, or charge, is measured in millivolts (mV).

AUTHOR'S NOTE Because myocardial cells and pacemaker cells can both conduct electrical impulses, we will refer to them collectively as *cardiac cells.*

Resting State of the Cardiac Cell

At rest, a cardiac cell has a layer of positive ions surrounding its cell membrane. It has an equal number of negative ions lining the inside of the cell membrane directly opposite each positive ion. When the positive and negative ions are aligned this way, like rival football teams at the 50-yard line, the resting cell is said to be *polarized* (Fig. 1.7).

When a cardiac cell is in the resting state, there is a high concentration of positively charged sodium (Na^+) ions called *cations* outside the cell. At the same time, inside the cell, there is a high concentration of negatively charged ions called *anions* (especially organic phosphate, organic sulfate, and proteins), mixed with a lower concentration of potassium cations (K^+). Cations carry a positive charge, whereas anions carry a negative charge. This makes the interior of the cell electrically negative compared with the outside of the cell. Under these conditions, a negative electrical potential exists across the cell membrane. This is possible because the cell membrane is not permeable to either the positively charged sodium cations outside the cell membrane or the negatively charged phosphate, sulfate, and protein anions inside the cell.

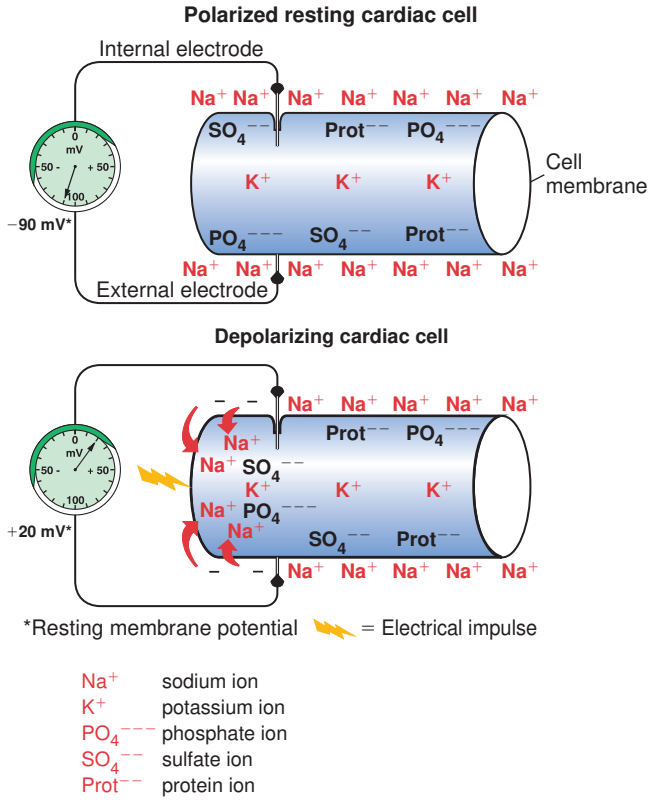


FIG. 1.7 Membrane potentials of polarized and depolarized cardiac cells.

The electrical potential maintained across the membrane of a resting cardiac cell is called the *resting membrane potential.* The resting membrane potential in atrial and ventricular cardiac cells and in the pacemaker cells of the electrical conduction system is normally −90 mV. Remember, a negative (−) membrane potential indicates that the concentration of positive ions, or cations, outside the cell is greater than the concentration inside the cell. A positive (+) membrane potential indicates the opposite—that there are more cations inside the cell than outside.

Depolarization and Repolarization

When stimulated by an electrical impulse, the membrane of a polarized cardiac cell becomes permeable to sodium cations, allowing sodium to flow into the cell. This causes the interior of the cell to become less negative compared with its exterior.

The process by which the cell's resting, polarized state is reversed is called *depolarization* (Fig. 1.8). When the membrane potential drops from its resting potential of −90 mV to about −65 mV, large pores in the membrane momentarily open. These pores are called *fast sodium channels.* They facilitate the rapid, free flow of sodium across the cell membrane, resulting in a sudden large influx of positively charged sodium cations into the cell. This quickly causes the interior of the cell to become more positively charged. The moment the concentration of sodium cations within the cell reaches the concentration outside the cell, the membrane potential drops to zero, and the cardiac cell is depolarized. The influx of sodium cations continues, however, causing a temporary rise in the membrane potential to about +20 to +30 mV—the so-called *overshoot.*

The fast sodium channels are found in the myocardial cells but not in the pacemaker cells. The pacemaker cells have slow calcium–sodium channels that open when the membrane potential drops to about −50 mV. During depolarization, these channels permit calcium and sodium cations to enter the cardiac cells at a slow and gradual rate. The result is a slower rate of depolarization compared with the depolarization rate of myocardial cells that contain fast sodium channels.

As soon as a cardiac cell depolarizes, potassium cations flow out of the cell. This movement across the cell membrane initiates a process by which the cell returns to its resting polarized state. This process, called *repolarization* (see Fig. 1.8), involves a complex exchange of sodium, calcium, and potassium ions across the cell membrane.

AUTHOR'S NOTE The electrical potential outside a polarized cardiac cell is less negative than the potential outside a depolarized cardiac cell. This difference in electrical potential provides the energy to generate the electrical current that depolarizes and repolarizes the cell.

Depolarization of one cardiac cell acts as an electrical impulse that stimulates and depolarizes adjacent cells. Depolarization of the myocardial cells results in contraction of the muscle

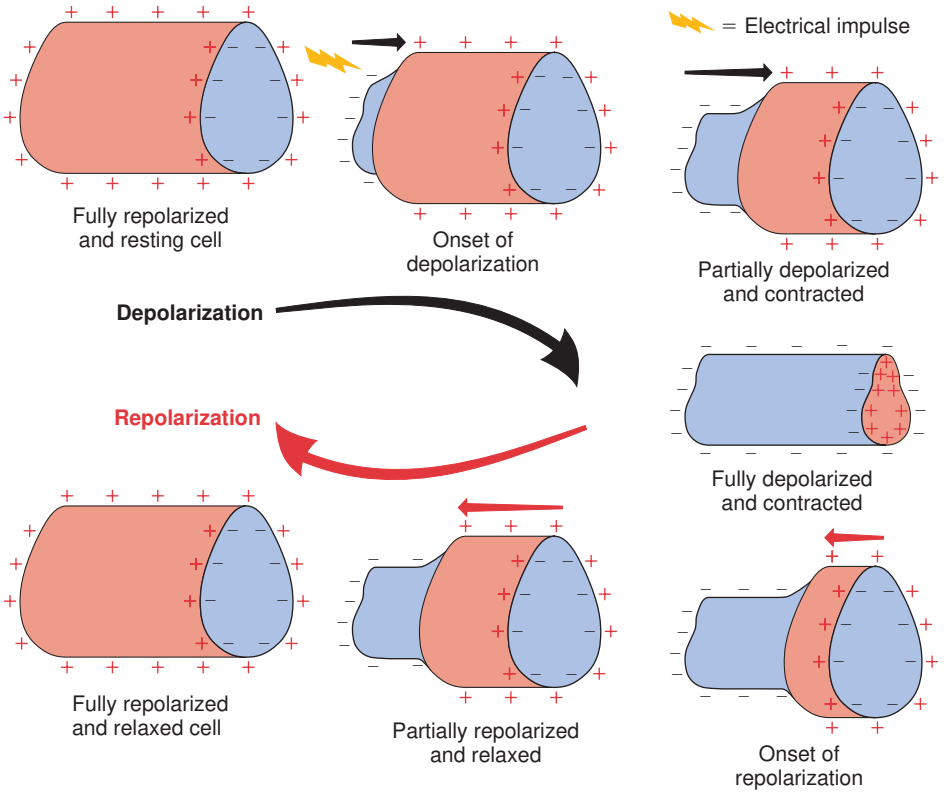


FIG. 1.8 Depolarization and repolarization of a myocardial cell.

and propagation (generation) of an impulse. Depolarization of the pacemaker cells propagates an impulse to adjacent pacemaker cells. This wave of depolarization from cell to cell produces a wave of electrical energy that can be measured as an electrical current flowing through the heart.

AUTHOR'S NOTE The electrocardiogram (ECG) machine detects only the electrical current of the myocardial cells because their size and number far exceed the size and number of the pacemaker cells.

Threshold Potential

A cardiac cell need not be repolarized completely to its resting polarized state (−90 mV) before it can be stimulated to depolarize again. The cells of the SA and AV nodes can be depolarized when they have been repolarized to about −30 to −40 mV. The remaining cells within the electrical conduction system of the heart and the myocardial cells can be depolarized when they have been repolarized to about −60 to −70 mV. The level to which a cell must be repolarized before it can be depolarized again is known as its *threshold potential.*

AUTHOR'S NOTE The fact that the myocardial cells have a higher (more negative) threshold potential than the pacemaker cells helps to ensure that the dedicated electrical pathways are the primary and most efficient means of electrical conduction through the heart.

Action Potential

The *action potential* refers to the change in membrane potential (from a positive to a negative state) during depolarization and repolarization. This change can be represented by a diagram in which the action potential is divided into five phases: phase 0 to phase 4 (Fig. 1.9):

- **Phase 0.** Phase 0 (depolarization phase) is the sharp, tall upstroke of the action potential, during which the cell membrane reaches its threshold potential. This triggers the fast sodium channels to open momentarily, permitting the rapid entry of sodium into the cell. As the cations flow into the cell, the interior of the cell becomes electrically positive. During the upstroke, the cell depolarizes and begins to contract.
- **Phase 1.** During phase 1 (early rapid repolarization phase), the fast sodium channels close, terminating the rapid flow of sodium into the cell, followed by a loss of potassium from the cell. The net result is a decrease in the number of positive electrical charges within the cell and a drop in the membrane potential to zero.
- **Phase 2.** Phase 2 is the prolonged plateau phase, during which the myocardial cell is slowly repolarized. The gradual completion of this phase allows the myocardial cell to finish contracting and begin relaxing. During phase 2, the membrane potential remains near zero. In a complicated exchange of ions across the cell membrane, calcium gradually enters the cell through the slow calcium channels. Sodium enters gradually as well, whereas potassium continues to leave the cell.

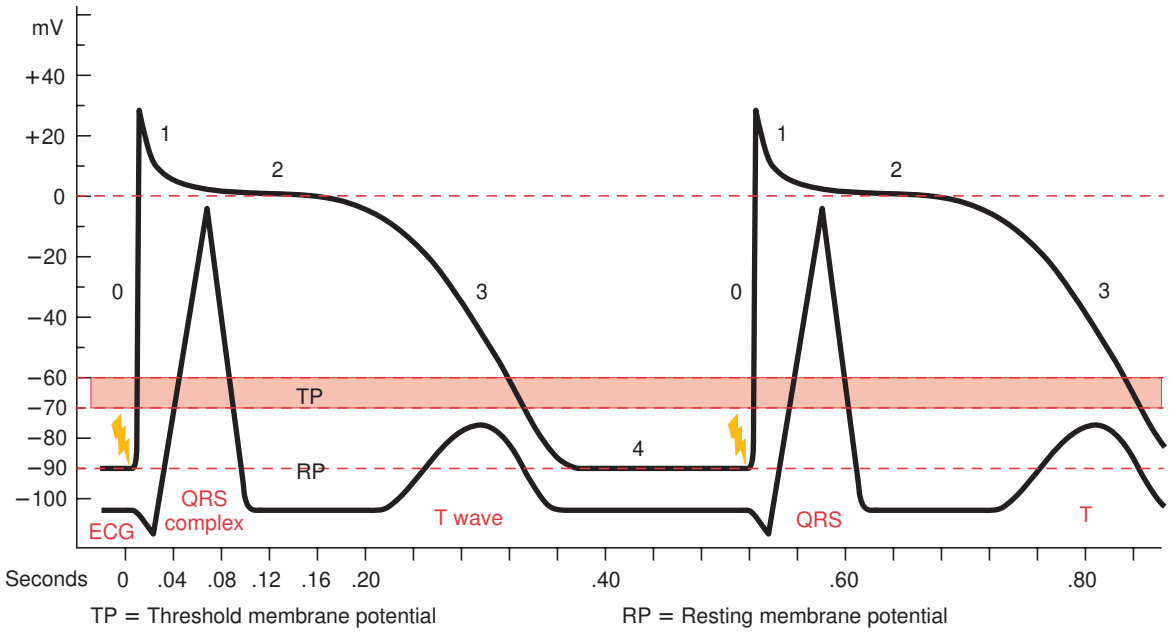


FIG. 1.9 Action potential of myocardial cells.

- **Phase 3.** Phase 3 is the final or terminal phase of rapid repolarization. During this phase, the inside of the cell becomes markedly negative, and the membrane potential once again returns to its resting level of about −90 mV. This change is caused primarily by the flow of potassium from the cell. Repolarization is complete by the end of phase 3.
- **Phase 4.** At the onset of phase 4 (the period between action potentials), the membrane has returned to its resting potential, and the inside of the cell is once again maximally negative (−90 mV) compared with the outside. But there is still an excess of sodium in the cell and an excess of potassium outside. At this point, a physiologic mechanism known as the *sodium–potassium pump* is activated, transporting the excess sodium out of the cell and ushering potassium back in. Because of this mechanism and the impermeability of the cell membrane to sodium during this phase, the myocardial cell normally maintains a stable membrane potential between action potentials.

REFRACTORY PERIODS

The refractory period of a cardiac cell begins with the onset of phase 0 of the cardiac action potential and ends just before the end of phase 3. On the ECG, this period extends from the onset of the QRS complex to about the end of the T wave.

The refractory period is divided into absolute and relative refractory periods (Fig. 1.10). The absolute refractory period (ARP) constitutes the first two-thirds of the refractory period. It begins with the onset of phase 0 and ends midway through phase 3, at about the peak of the T wave. During this period, the cardiac cells—having completely depolarized—are in the process of repolarizing. Because they have not repolarized to their threshold potential, the cardiac cells cannot be stimulated to depolarize. In other words, the myocardial cells cannot contract, and

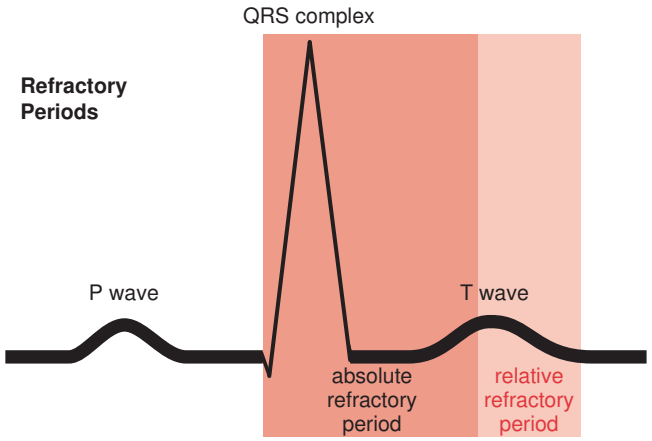


FIG. 1.10 Refractory periods.

the cells of the electrical conduction system cannot depolarize during the ARP.

The relative refractory period (RRP) occupies the remaining one-third of the refractory period. The RRP extends through most of the second half of phase 3, corresponding to the downslope of the T wave. During this period, the cardiac cells can be stimulated to depolarize if the stimulus is strong enough because they have been repolarized to their threshold potential. This period is also called the *vulnerable period of repolarization.*

refractory period

The period between the onset of depolarization and the end of repolarization of a cardiac cell, during which it cannot be stimulated to repolarize.

AUTOMATICITY

The ability of a cardiac cell to depolarize spontaneously during phase 4 is called the *property of automaticity.* To depolarize spontaneously, the cell membrane must become permeable to sodium during phase 4, thus allowing a steady leakage of sodium ions into the cell. This causes the resting membrane potential to become progressively less negative. As soon as its threshold potential is reached, the cell rapidly depolarizes (phase 0). The rate of spontaneous depolarization depends on the slope of phase 4 depolarization (Fig. 1.11). The steeper the slope of phase 4 depolarization, the faster the rate of spontaneous depolarization and impulse formation (the firing rate). The flatter the slope is, the slower the firing rate.

automaticity (auto-ma-TISS-ity)

A property of cardiac cells that allows them to reach threshold potential and then depolarize spontaneously and completely, without external stimulation.

AUTHOR'S NOTE Increased sympathetic activity and administration of catecholamines increase the slope of phase 4 depolarization, which increases the automaticity of the pacemaker cells and their firing rate. On the other hand, increased parasympathetic activity or administration of drugs that decrease the slope of phase 4 depolarization reduces the automaticity and firing rate of the pacemaker cells.

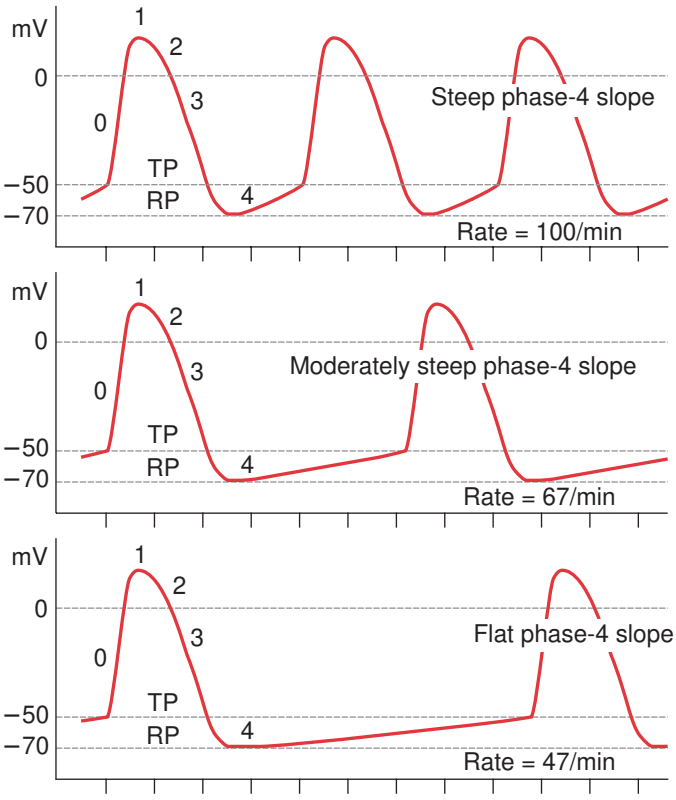


FIG. 1.11 Action potential of pacemaker cells. Rate of spontaneous depolarization is dependent on the slope of phase 4 depolarization. *RP,* Resting membrane potential; *TP,* threshold potential.

Dominant and Escape Pacemakers of the Heart

As we have described, pacemaker cells are specialized cells in the electrical conduction system that normally have the property of automaticity. These cells are located in the SA node; in some areas of the internodal atrial conduction tracts and AV node; and throughout the bundle of His, bundle branches, and Purkinje network. The pacemaker cells of the SA node have the fastest spontaneous firing rate (60–100 times per minute). As a result, they are normally the dominant (or primary) pacemaker cells of the heart (Fig. 1.12). The pacemaker cells in the rest of the electrical conduction system have a lower rate of automaticity and are normally called on to depolarize only if the SA node fails to function properly or if electrical impulses fail to reach them. For this reason, these pacemaker cells are called *escape pacemaker cells.*

Normally, the heart rate is controlled by the pacemaker cells with the highest level of automaticity. Each time these pacemaker cells generate an electrical impulse, the more slowly firing escape pacemaker cells are depolarized before they can do so spontaneously. This phenomenon is called *overdrive suppression.*

The SA node is normally the dominant and primary pacemaker of the heart (see Fig. 1.11) because it possesses the highest level of automaticity; that is, its spontaneous rate of automatic firing (60–100 times per minute) is normally greater than that of the other pacemaker cells.

If the SA node fails to depolarize at its normal rate or stops functioning entirely, or if the conduction of the electrical impulse is blocked for any reason (for example, in the AV node), escape pacemaker cells in the AV junction will usually assume the role of pacemaker of the heart but at a slower rate (40–60 times per minute). If the AV junction is unable to take over as the pacemaker, an escape pacemaker in the electrical conduction system below the AV junction or in the ventricles (in the bundle branches or Purkinje network) may take over at an even slower rate (fewer than 40 times per minute).

The rate at which the SA node or an escape pacemaker normally generates electrical impulses is called the *pacemaker's inherent firing rate.* A beat or a series of beats arising from an escape pacemaker is called an *escape beat* or *rhythm* and is identified according to its origin (for example, junctional, ventricular).

Mechanisms of Ectopic Electrical Impulse Formation

Under certain circumstances, cardiac cells in any part of the heart, whether they are escape pacemaker cells or myocardial cells, are capable of generating additional electrical impulses. Such activity within the heart is referred to as *ectopic* because it originates outside the normal conduction pathway. The result can be ectopic rhythms. These rhythms are identified according to the location of the ectopic pacemaker (for example, atrial, junctional, or ventricular). The three basic mechanisms that are responsible for ectopic beats and rhythms are (1) enhanced automaticity, (2) reentry, and (3) triggered activity.

ectopy

Origination of electrical impulses in a site outside the heart's normal conduction pathway, resulting in abnormal (ectopic) rhythms or beats.

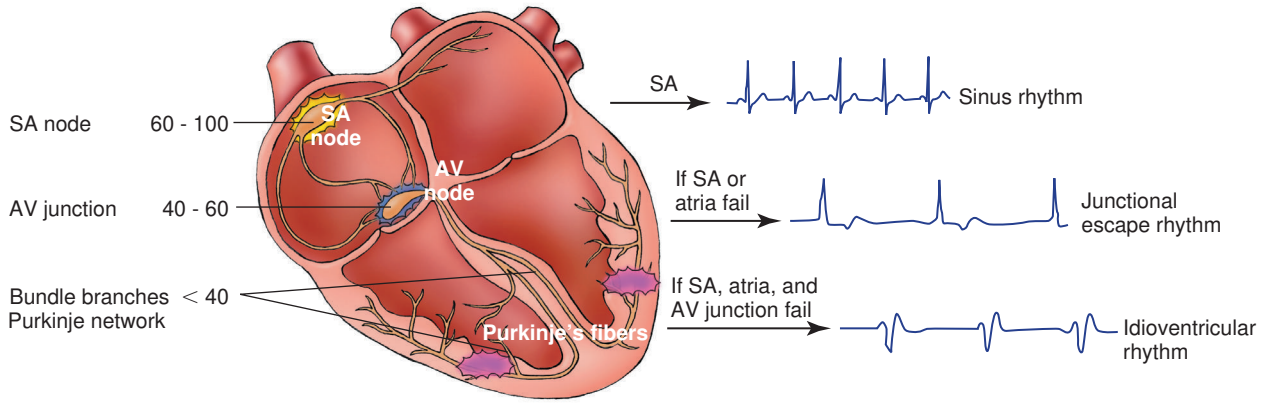


FIG. 1.12 Dominant and escape pacemakers.

ENHANCED AUTOMATICITY

Enhanced automaticity is a condition in which the cell's firing rate is increased beyond its inherent rate. This occurs when the cell membrane becomes abnormally permeable to sodium during phase 4. The result is an abnormally high leakage of sodium ions into the cells and, consequently, a sharp rise in the phase 4 slope of spontaneous depolarization. Even myocardial cells that do not ordinarily possess automaticity may acquire this property and depolarize spontaneously. Enhanced automaticity can cause atrial, junctional, and ventricular ectopic rhythms.

Common causes of enhanced automaticity include elevated levels of catecholamines (stimulants), digitalis toxicity, and administration of atropine. In addition, hypoxia, hypercapnia, myocardial ischemia or infarction, stretching of the heart muscle, hypokalemia, hypocalcemia, and heating or cooling of the heart may also cause enhanced automaticity.

REENTRY

Reentry is a condition in which the progression of a wave of depolarization is delayed or blocked (or both) (Fig. 1.13, *A* and *B*) in one or more segments of the electrical conduction system while being conducted normally through the rest of the conduction system. This delays antegrade (forward) or retrograde (backward) conduction of electrical impulses into adjacent cardiac cells that have just been depolarized by the normally conducted electrical impulse. If these cardiac cells have repolarized sufficiently, the delayed electrical impulse depolarizes them prematurely, producing ectopic rhythms. Myocardial ischemia and hyperkalemia are the two most common causes of a delay or block in the conduction of an electrical impulse through the electrical conduction system responsible for the reentry mechanism. Another cause of the reentry mechanism is the presence of an accessory conduction pathway (see Fig. 1.13, *C* and *D*), such as the accessory AV pathways located between the atria and ventricles described earlier in this chapter.

After normal antegrade progression of a wave of depolarization through the electrical conduction system and depolarization of the cardiac cells, the electrical impulse enters the accessory conduction pathway and progresses in a retrograde fashion to reenter the proximal end of the electrical conduction system much sooner than the next expected normal electrical impulse. The electrical impulse is then conducted antegrade as before, causing depolarization of the cardiac cells prematurely. Thus a reentry circuit is a condition that can result in the conduction of a rapid series of electrical impulses through the electrical conduction system. The electrical impulse can also progress in an antegrade direction through the accessory conduction pathway and retrogradely through the electrical conduction system.

This reentry mechanism can result in the abnormal generation of single or repetitive electrical impulses in the atria, AV junction, bundle branches, and Purkinje network. It produces atrial, junctional, or ventricular ectopic rhythms, such as atrial, junctional, and ventricular tachycardias. Such reentry tachycardias typically start and stop abruptly.

TRIGGERED ACTIVITY

Triggered activity is an abnormal condition of myocardial cells in which the cells may depolarize more than once after stimulation by a single electrical impulse. The cellular membrane action potential spontaneously increases after the first depolarization until it reaches threshold potential, causing the cells to depolarize, once or repeatedly. This phenomenon, called *afterdepolarization,* can occur almost immediately after depolarization in phase 3 or later in phase 4. Triggered activity can result in atrial or ventricular ectopic complexes occurring singly, in groups of two (paired or coupled), or in bursts of three or more complexes (paroxysms of tachycardia).

AUTHOR'S NOTE Common causes of triggered activity, like those of enhanced automaticity, include an increase in catecholamines, digitalis toxicity, hypoxia, myocardial ischemia or injury, and stretching or cooling of the heart.

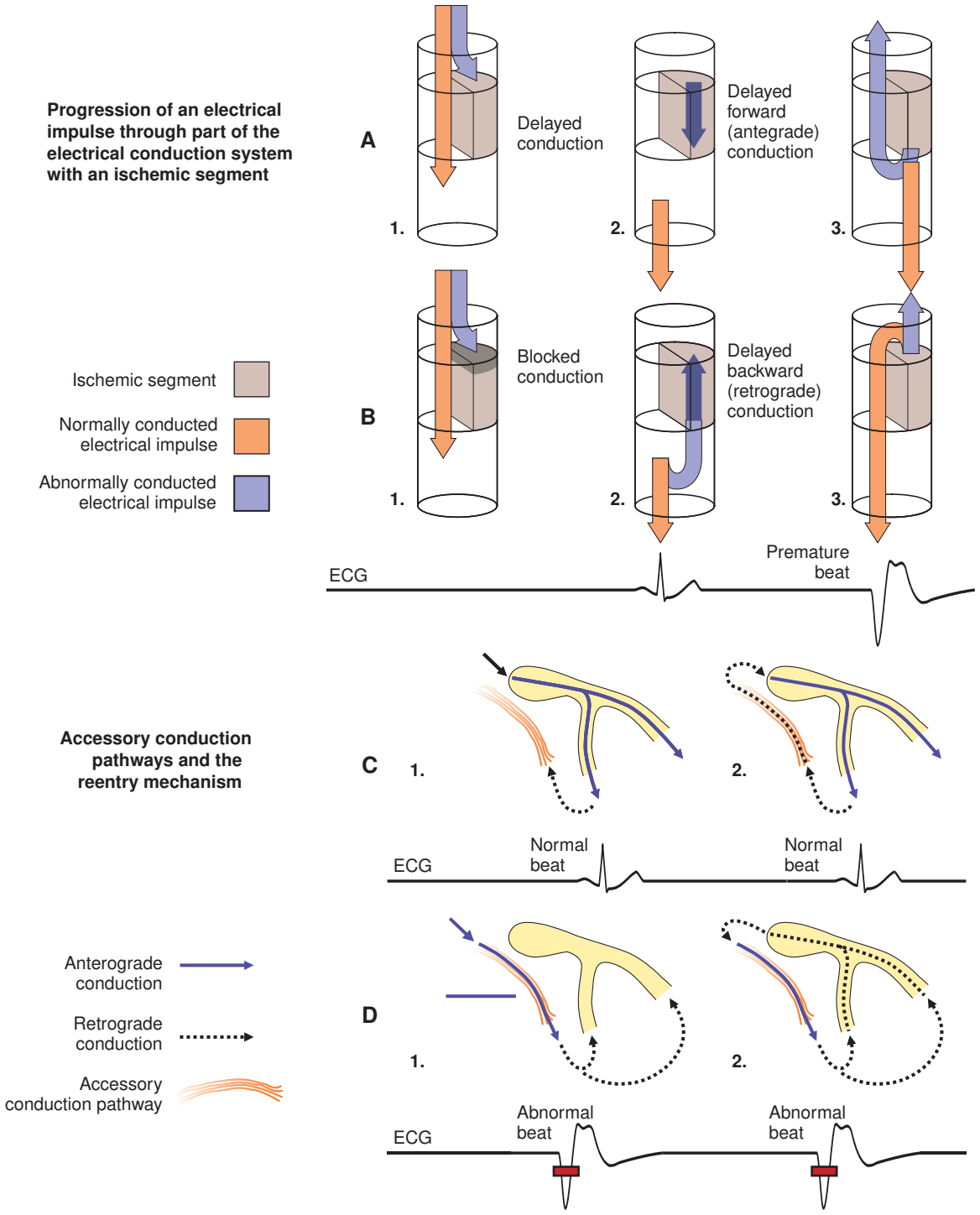


FIG. 1.13 Examples of reentry mechanisms. (A) Delayed conduction. (B) Blocked and delayed conduction. (C) Antegrade conduction through the conduction system. (D) Retrograde conduction through the conduction system.

AUTONOMIC NERVOUS SYSTEM CONTROL OF THE HEART

The heart is under constant control of the autonomic nervous system (ANS), which includes the sympathetic and parasympathetic divisions (Fig. 1.14). By producing opposite effects, these divisions work together to regulate cardiac output and blood pressure.

ANS control of the heart originates in two separate nerve centers in the medulla oblongata, a part of the brainstem:

1. **Cardioaccelerator center.** This center is part of the sympathetic nervous system. Impulses from the cardioaccelerator center reach the electrical conduction system of the

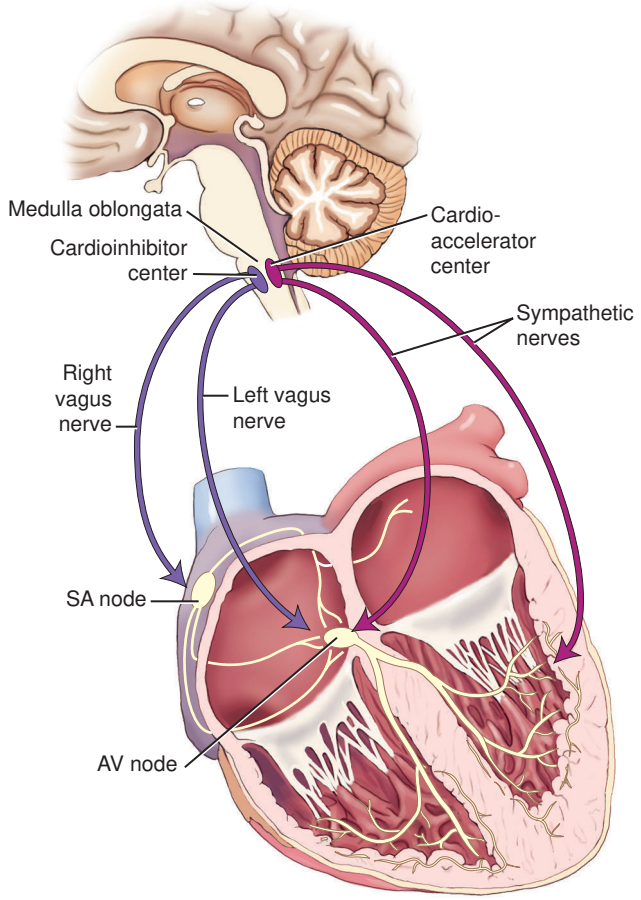


FIG. 1.14 Sympathetic and parasympathetic regulation of the heart.

heart and the atria and ventricles by way of the sympathetic nerves.

Stimulation of the sympathetic nervous system produces the following effects:

- An increase in the firing rate of the SA node and escape and ectopic pacemakers throughout the heart
- An increase in the conductivity of electrical impulses through the atria and ventricles, especially through the AV node
- An increase in the force of atrial and ventricular contractions
- The result is an increase in heart rate, cardiac output, and blood pressure.

2. **Cardioinhibitor center.** This center is part of the parasympathetic nervous system. Impulses from the cardioinhibitor center travel to the SA node, atria, and AV junction and, to a small extent, the ventricles by way of the right and left vagus nerves. When the vagus nerve fires, the heart rate slows. When it fires less, the effects of the sympathetic nervous system dominate, and the heart rate increases. The rate at which the vagus nerve fires is referred to as *vagal tone.* Another important cardioinhibitor (parasympathetic) nerve center is the carotid sinus. The carotid sinus is a slight dilation of the common carotid artery, located at the point where it branches into the internal and external carotid arteries. Sensory nerve endings in the carotid sinus help regulate blood pressure and heart rate.

Stimulation of the parasympathetic nervous system produces the following effects:

- A decrease in the firing rate of the SA node and escape and ectopic pacemakers in the atria and AV junction
- A slowing of conduction of electrical impulses through the AV node

The result is a decrease in heart rate, cardiac output, and blood pressure and, sometimes, a complete block of the electrical impulse through the AV node.

As the blood pressure requirements of the body change, multiple sensors in the body relay impulses to the cardioinhibitor and cardioaccelerator centers for analysis. From there, the sympathetic and parasympathetic nerves transmit the appropriate impulses to the electrical conduction system of the heart and to the atrial and ventricular myocardium, where they influence the automaticity, conductivity, and contractility of the cardiac cells.

AUTHOR'S NOTE The parasympathetic nervous system can be stimulated by putting pressure on the carotid sinus, performing the Valsalva maneuver (the action of straining against a closed glottis [airway]), straining to move the bowels, or distention of the urinary bladder. Nausea, vomiting, bronchial spasm, sweating, faintness, and hypersalivation are manifestations of excessive parasympathetic activity. The drug atropine effectively blocks the parasympathetic nervous system.

TAKE-HOME POINTS

- The heart lies in the center of the chest, with its base at the atria and its apex at the ventricles. It is surrounded by the pericardium, enveloped by the epicardium, and lined by the endocardium.
- The walls of the ventricles are composed of three layers: the innermost layer is the smooth tissue of the endocardium, the middle layer is the muscular myocardium, and the outermost is a thin layer of connective tissue called the *epicardium.*
- Blood from the venae cavae and lungs enters the atria and then moves into the ventricles, where it is pumped to the lungs and body during the cardiac cycle.
- The cardiac cycle has two phases: Diastole is the phase during which the atria and ventricles relax and fill with blood. Systole is the phase during which the atria and ventricles contract to pump blood out.
- The heart has specific pathways that allow electrical impulses to be transmitted in order to stimulate contraction of the atria and ventricles. The primary pathway consists of the SA node, the internodal/interatrial tract, the AV junction, the right and left bundle branches, and the Purkinje network. Signals may also travel along accessory pathways, causing various rhythm disturbances.
- The specific properties of conductivity, automaticity, and contractility allow the cardiac muscle to perform its unique duty to ensure coordinated contraction and relaxation

of the heart in order to generate blood flow through the body.
- The heart has three layers. The innermost layer is the smooth tissue of the endocardium. The middle layer is the muscular myocardium. The outermost layer is a thin layer of connective tissue.
- The electrophysiology of the heart is regulated by the movement of positive and negative ions across cell membranes. The unique properties of these ions change the permeability of the membrane.
- This process of ion movement charges the cell with an action potential that allows it to cause adjacent cells to depolarize and transmit the electrical impulse forward.
- The action potential has five phases: phase 0, depolarization; phase 1, early rapid repolarization; phase 2, the prolonged plateau phase; phase 3, the terminal phase of rapid repolarization; and phase 4, the period between action potentials.
- The presence of dominant and escape pacemakers ensures that the heart has backup mechanisms in place to generate a pulse in the event that one of the higher pacemakers fails.
- The process of depolarization and repolarization causes electrical impulses to move down the conduction pathway. This process can be influenced by the autonomic nervous system and other environmental stimuli and pathologic conditions.

CHAPTER REVIEW QUESTIONS

1. Which term is commonly used to describe the inner layer of the pericardium, which covers the heart itself?
   A. Endocardium
   B. Epicardium
   C. Myocardium
   D. Pericardium

2. The _____ side of the heart pumps blood into the _____ circulation, and the _____ side of the heart pumps blood into the _____ circulation.
   A. left; pulmonary; right; systemic
   B. left; ventricular; right; atrial
   C. right; pulmonary; left; systemic
   D. right; systemic; left; pulmonary

3. The right ventricle pumps deoxygenated blood through the _____ valve and into the lungs through the _____ artery.
   A. aortic; mitral
   B. mitral; tricuspid
   C. pulmonary; pulmonary
   D. tricuspid; pulmonary

4. Which term best describes the period during which the heart relaxes and the ventricles fill with blood?
   A. Atrial diastole
   B. Atrial systole
   C. Ventricular diastole
   D. Ventricular systole

5. Which structure is a normal component of the heart's electrical conduction system?
   A. Atrial septa
   B. Coronary sinus
   C. Right bundle branch
   D. Vagus nerve

6. Which term best describes the ability of cardiac cells to depolarize spontaneously?
   A. Automaticity
   B. Conductivity
   C. Contractility
   D. Self-excitation

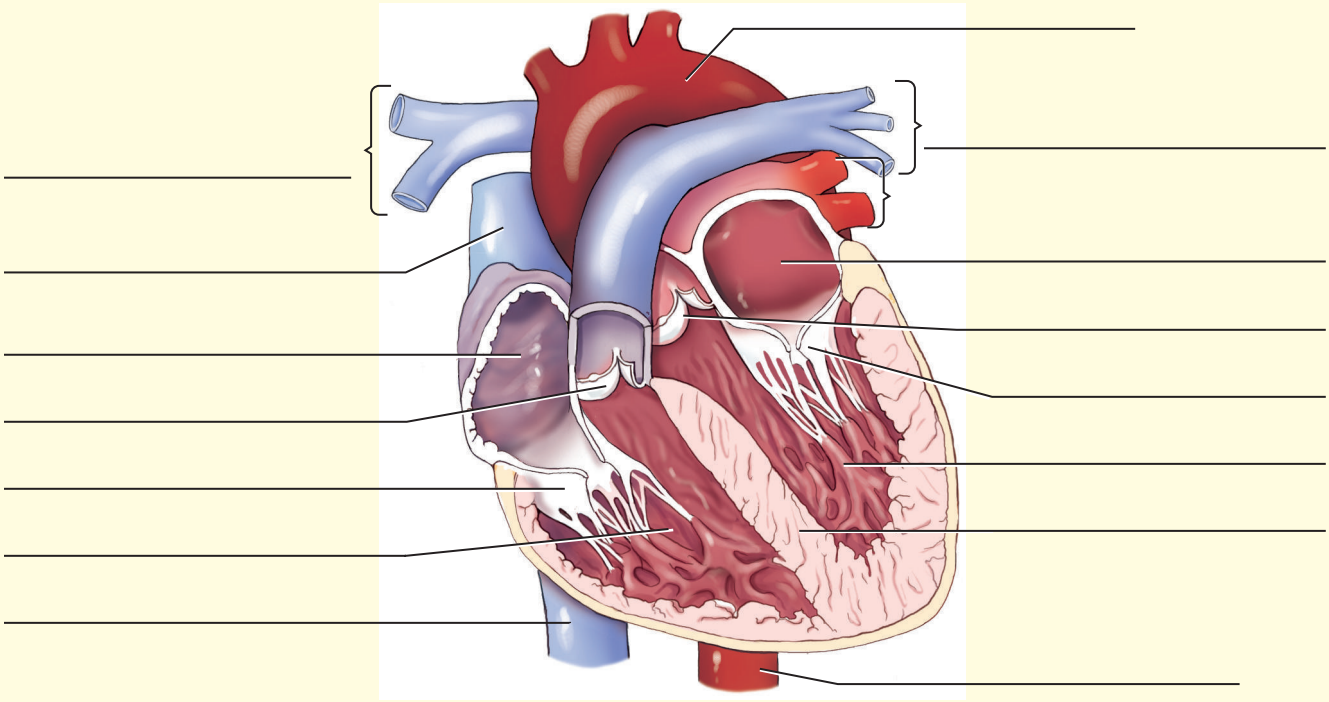
7. In the resting state, a myocardial cell has a high concentration of the _____ charged _____ ions present outside the cell.
   A. negatively; potassium
   B. negatively; sodium
   C. positively; potassium
   D. positively; sodium

8. Cardiac cells cannot be stimulated to depolarize during which part of the cardiac cycle?
   A. Absolute refractory period
   B. Ectopic period
   C. Relative refractory period
   D. Resting state

9. Which term best describes the normal and dominant pacemaker of the heart?
   A. AV node
   B. Bundle of His
   C. Purkinje fibers
   D. SA node

10. Under what conditions does the vagus nerve, part of the parasympathetic nervous system, slow the heart?
    A. When the vagus nerve fires
    B. When the vagus nerve is blocked by atropine
    C. When the vagus nerve is severed
    D. When the patient is given a stimulant

11. Label the figure.



2 ECG Leads and Cardiac Monitoring

OUTLINE

BASIC ECG CONCEPTS

AUTHOR'S NOTE Throughout this text, we will use the term *ECG* for *electrocardiogram*. Some texts use the abbreviation *EKG,* which is derived from the German word *Elektrokardiogramm.* Also, *EKG* is often the abbreviation used verbally because *ECG* can be confused with *EEG,* which refers to an electroencephalogram that measures electrical activity in the brain.

Electrical Basis of the ECG

The ECG is a graphic record of changes in the magnitude and direction of the heart's electrical activity. The ECG does not detect the depolarization of individual cells. In fact, it does not detect the combined depolarization of the pacemaker cells because the resulting impulse is too small to detect with standard electrodes. What the ECG does detect is the combined electrical impulse generated by the wave of depolarization and repolarization that progresses through the myocardial cells of the atria and ventricles during each cardiac cycle. This electrical activity is sufficient enough to be detected by electrodes attached to the skin.

ECG Paper

ECGs are printed on grid paper that shows time in seconds (sec) along the horizontal axis. Voltage or strength (amplitude) in millimeters (mm) appears along the vertical axis (Fig. 2.1).

These intersecting dark and light vertical and horizontal lines form a grid of large and small squares. The distance between the vertical lines depends on the rate of paper output when the ECG is recorded. For example, the grid will look different at an output rate of 25 millimeters per second (mm/sec) compared with 50 mm/sec. The standard recording speed is 25 mm/sec. Other speeds are used only for specialized purposes.

AUTHOR'S NOTE All ECGs in this text will be based on the standard recording speed of 25 mm/sec unless otherwise noted.

When the ECG is recorded at the standard speed of 25 mm/sec, the measurements between the vertical lines are as follows:

- The dark vertical lines are 5 mm apart.
- If 1 second = 25 mm, then 5 mm = ⅕ of a second, or 0.20 second.
- The light vertical lines are 1 mm apart.
- If 5 mm = ⅕ of a second, then 1 mm = ⅕₂₅ of a second, or 0.04 second.

Regardless of the speed of the recording, the measurements between the horizontal lines are as follows:

- The dark horizontal lines are 5 mm apart.
- The light horizontal lines are 1 mm apart.

Therefore at the standard paper speed of 25 mm/sec, the large and small squares have the following characteristics:

- One large square is 5 mm tall and 0.2 second long.
- One small square is 1 mm tall and 0.04 second long.

The sensitivity of an ECG machine must be calibrated. Conventionally, a 1-millivolt (mV) electrical signal should produce a 10-mm (2 large squares or 10 small squares) deflection on the ECG.

Short vertical lines (or small arrowheads) are printed along the top or bottom edge of the ECG paper at regular intervals. These lines denote intervals of time and thus are called *time lines.* They are spaced 15 large squares apart (75 mm, or about 3 inches apart). When the ECG is recorded at the standard paper speed of 25 mm/sec, the distance between the time lines represents 3 seconds. Every third vertical line, then, represents the passage of 6 seconds.

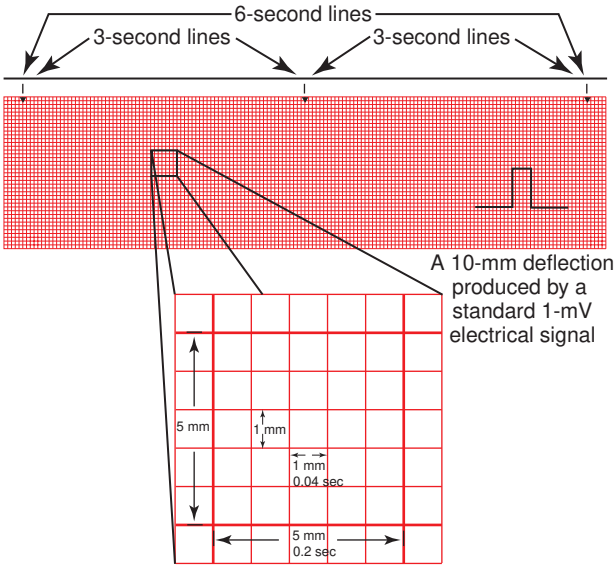


FIG. 2.1 ECG paper.

time lines

Short vertical lines (or small arrowheads) that denote intervals of time on an ECG. At the standard paper speed, the distance between the lines represents a period of 3 seconds.

BASIC COMPONENTS OF A NORMAL ECG

It is vital to understand the relationship between the various components of the ECG and the electrical activity occurring in the heart. The current generated by depolarization and repolarization of the atria and ventricles is detected by electrodes. It is then amplified and displayed on a screen. It is also recorded on the ECG paper as a series of waves and complexes. This combination of waves and complexes is referred to as the *ECG waveform* (Fig. 2.2). Between the ECG waveforms, the ECG tracing returns to a nearly flat line, called the *baseline* or *isoelectric line*. During this period, no electrical activity occurs. Generally speaking, when we examine the ECG waveform, we will be evaluating the waves and complexes based on their shape, duration, and timing; the intervals based on their length; and the segments based on their relationship to the baseline.

baseline

A nearly flat line on the ECG that reflects a period during which no electrical activity occurs in the heart. The baseline serves as a point of reference for measuring and interpreting waves, complexes, intervals, and segments.

The next chapter will discuss each component in greater detail, but let's take a look at the basics:

- The electric impulse generated by atrial depolarization is recorded as the P wave.
- The impulse generated by ventricular depolarization is recorded as the Q, R, and S waves. Together they are called the *QRS complex*.
- Ventricular repolarization is manifested by the *T wave*. Because atrial repolarization normally occurs during ventricular depolarization, it is hidden in the QRS complex.

In a normal ECG waveform, the P wave occurs first. It is followed by the QRS complex and then the T wave. The sections of the ECG between waves and complexes are called *segments* and *intervals*. Their shape and length reveal the speed of electrical conduction through the heart:

- The PR segment starts at the beginning of the P wave and ends at the start of the QRS complex.
- The ST segment starts at the end of the QRS complex and ends at the start of the T wave.
- The start of the ST segment, where the QRS complex ends, is the J point.
- The TP segment starts at the end of the T wave and stops at the start of the next P wave.

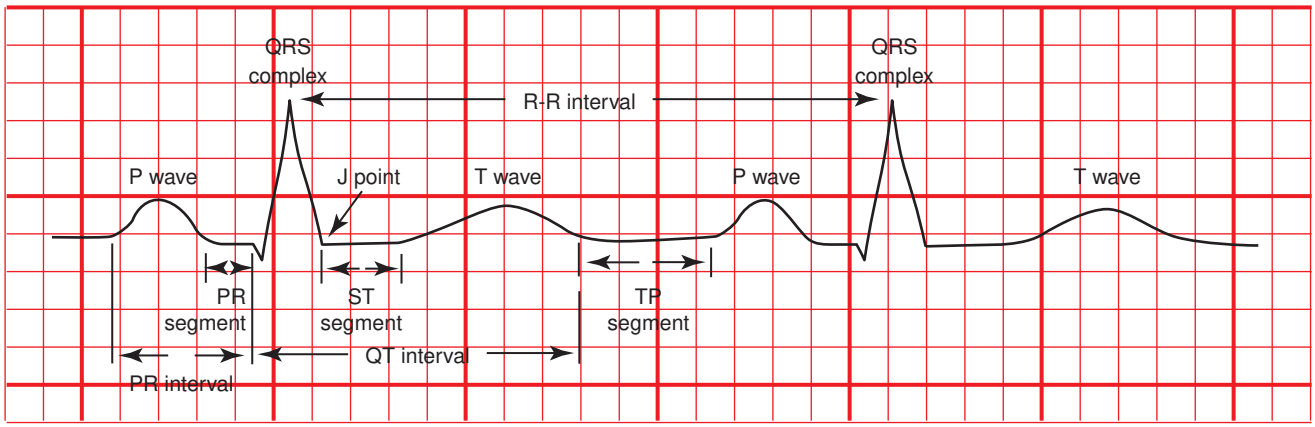


FIG. 2.2 Components of the ECG.

- The PR interval starts at the beginning of the P wave and ends at the beginning of the QRS complex.
- The QT interval starts at the beginning of the QRS complex and stops at the end of the T wave.
- And finally, the R-R, or "R to R," interval is measured from the tips of two consecutive QRS complexes.

ECG LEADS

Lead Basics

Electrodes, attached to the skin, detect the electrical impulse generated by the depolarization and repolarization of the myocardial cells. Each electrode is designated as either negative or positive and is placed on a specified area of the body, such as the right or left arm, the left leg, or one of several locations on the chest wall. The positive and corresponding negative electrodes are referred to as *leads*.

electrode

A sensing device attached to the skin that detects positive or negative electrical activity in the heart.

The ECG represents the movement of the negatively charged electrical impulse toward and away from the positive electrode. Therefore the orientation of the lead around the heart determines its "view" of the heart's electrical activity.

lead

The view of the heart's electrical activity from the perspective of the positive electrode.

To obtain an ECG, self-adhesive electrodes are affixed to the patient's skin and then connected to the ECG machine with wires. The machine then designates each electrode as positive or negative and changes its polarity depending on the lead selected. Two types of leads are used in ECG analysis: bipolar and unipolar.

Bipolar Leads

A lead with both a positive and negative electrode is a bipolar lead. Bipolar leads are referred to as *standard limb leads* because the electrodes are usually attached to the arms and legs of the patient. The standard limb leads are leads I, II, and III.

bipolar lead

A view of the electrical impulse between a positive and negative electrode from the positive electrode's perspective.

When monitoring the heart solely for rhythms, a single bipolar ECG lead, such as lead II (Fig. 2.3), is usually used. MCL_1 is another bipolar monitoring lead that's often used. *MCL* stands for *modified chest lead.* This lead is especially useful in monitoring heart rhythms in the hospital. Bipolar leads used less often for monitoring include leads I, III, and MCL.

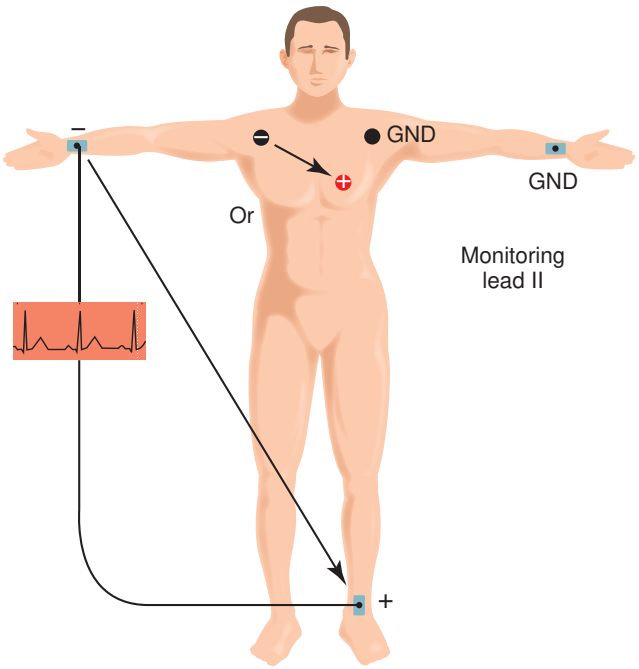


FIG. 2.3 Monitoring lead II.

MONITORING LEAD II

Lead II is usually obtained by attaching the negative electrode to the right arm and the positive electrode to the left leg. Electrodes sometimes detect respiratory chest movement rather than electrical activity of the heart. This electrical interference, or noise, can move the baseline or leave other traces on the ECG. To reduce this distortion when using lead II for monitoring, we attach a third, electrically neutral electrode, or ground electrode. It can be attached to the upper left chest; to an extremity (the left arm or right leg); or, for that matter, to any part of the body.

When an electrical impulse flows toward the positive electrode of a lead, a positive (upward) deflection is recorded on the ECG. Conversely, a negative (downward) deflection is recorded when the electrical impulse flows away from the positive electrode. If the positive electrode is attached to the left leg, all of the electric impulses generated in the heart that flow toward the left leg will be recorded as positive (upward) deflections. Those that flow away from the left leg will be recorded as negative (downward) deflections (Fig. 2.4).

Normal depolarization of the atria and ventricles progresses from the right upper chest downward toward the left leg. As a result, the electrical impulses generated during depolarization will also flow toward the left leg. They will be recorded as two positive (upward) deflections—a positive P wave (atrial depolarization) and a large positive R wave (ventricular depolarization)—in lead II.

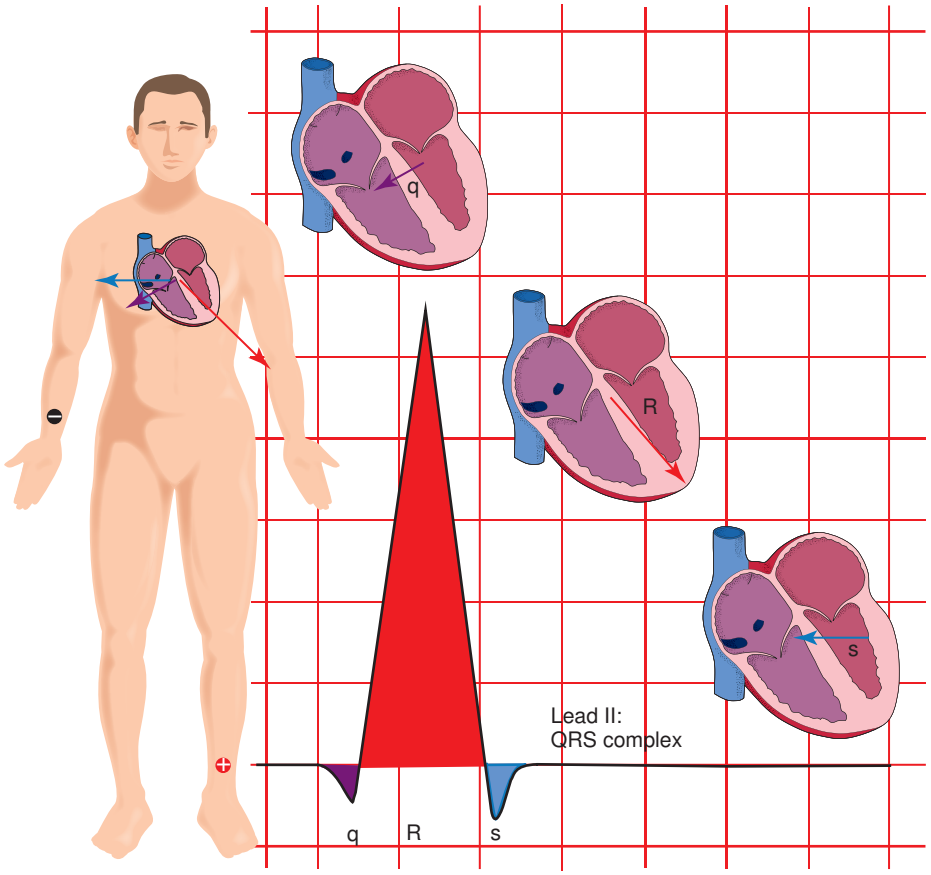


FIG. 2.4 Sequence and direction of normal depolarization.

Depolarization and repolarization of the atria and ventricles appear in the P wave, QRS complex, and T wave (Fig. 2.5) in predictable ways:

- **P wave.** Depolarization of the atria normally begins near the sinoatrial (SA) node and proceeds downward and to the left, producing a positive P wave.
- **QRS complex.** Depolarization of the ventricles usually starts with the depolarization of the relatively thin interventricular septum from left to right, resulting in a small negative deflection—the Q wave. This is immediately followed by the depolarization of the large left ventricle from right to left, which overshadows the almost simultaneous left-to-right depolarization of the smaller right ventricle, resulting in a large R wave. In addition, depending on the position of the heart in the chest, the size of the ventricles, and the rotation of the heart, depolarization of the base of the left ventricle from left to right usually produces a small negative (inverted) deflection after the R wave—the S wave.
- **T wave.** Finally, the T wave is produced as the ventricles repolarize from left to right.

AUTHOR'S NOTE The ECG components and strips shown in this book are depicted as they would appear in lead II unless otherwise noted.

MONITORING LEADS I AND III

The two other bipolar leads, leads I and III, are also used for ECG monitoring (Fig. 2.6):

- **Lead I.** Lead I is obtained by attaching the negative electrode to the right arm, the positive electrode to the left arm, and the ground electrode to the right leg. Lead I can also be obtained by attaching the negative electrode to the upper right anterior chest wall below the right clavicle and the positive electrode to the upper left anterior chest wall below the left clavicle. The ground electrode is attached to the right or left lower chest wall.
- **Lead III.** Lead III is obtained by attaching the negative electrode to the left arm, the positive electrode to the left leg, and the ground electrode to the right leg. Lead III can also be obtained by attaching the negative electrode to the upper left anterior chest wall below the left clavicle and the positive electrode to the lower-left anterior chest wall at the intersection of the fifth intercostal space and the midclavicular line. The ground electrode is attached to the right lower chest wall.

MODIFIED CHEST LEADS

MCLs are similar to the unipolar chest leads used in 12-lead ECGs but have less sensitivity. They can, however, be used to monitor certain rhythms.

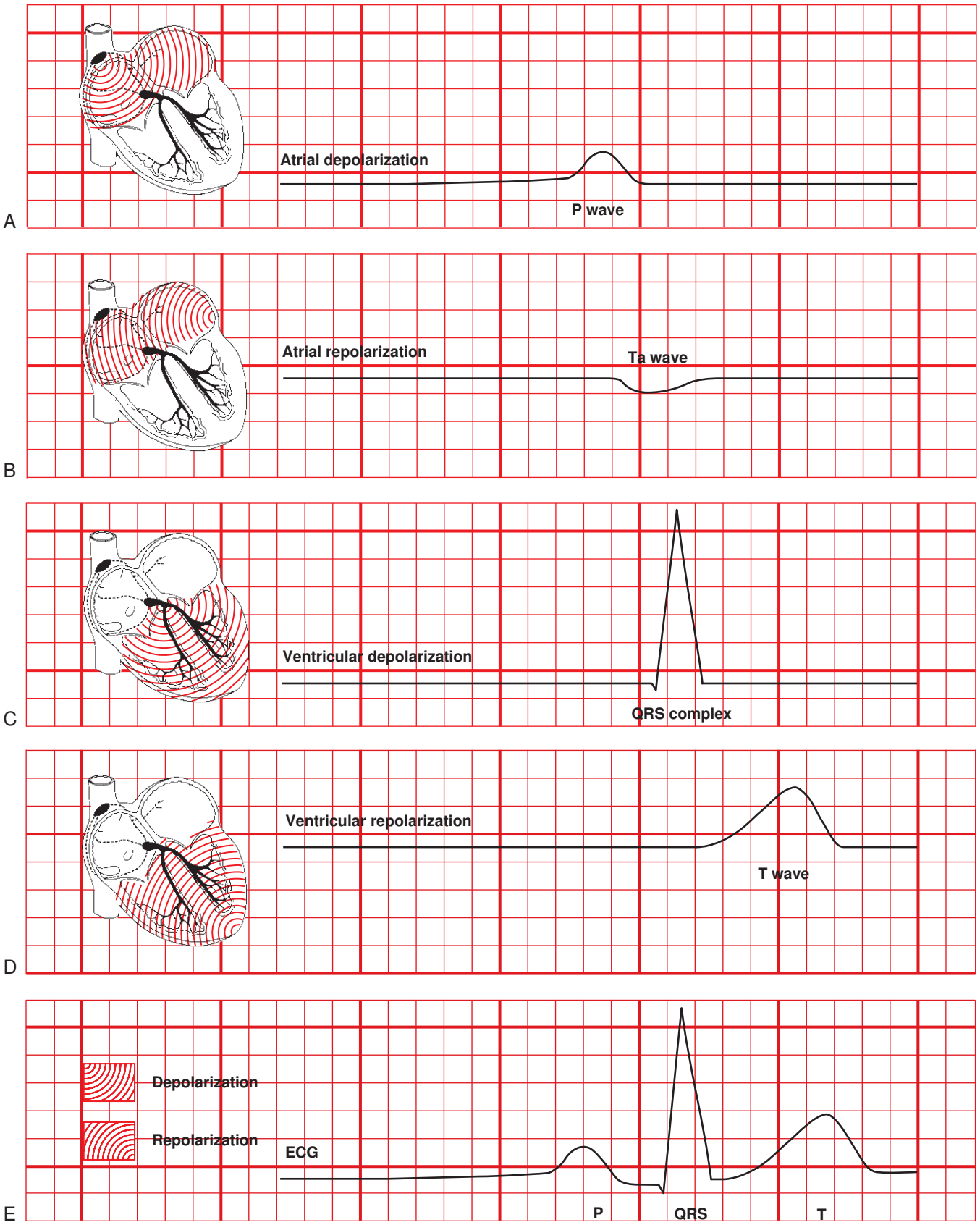


FIG. 2.5 Depolarization and repolarization of the atria and ventricles and the ECG. (A) P wave. (B) T_a wave. (C) QRS complex. (D) T wave. (E) ECG in lead II.

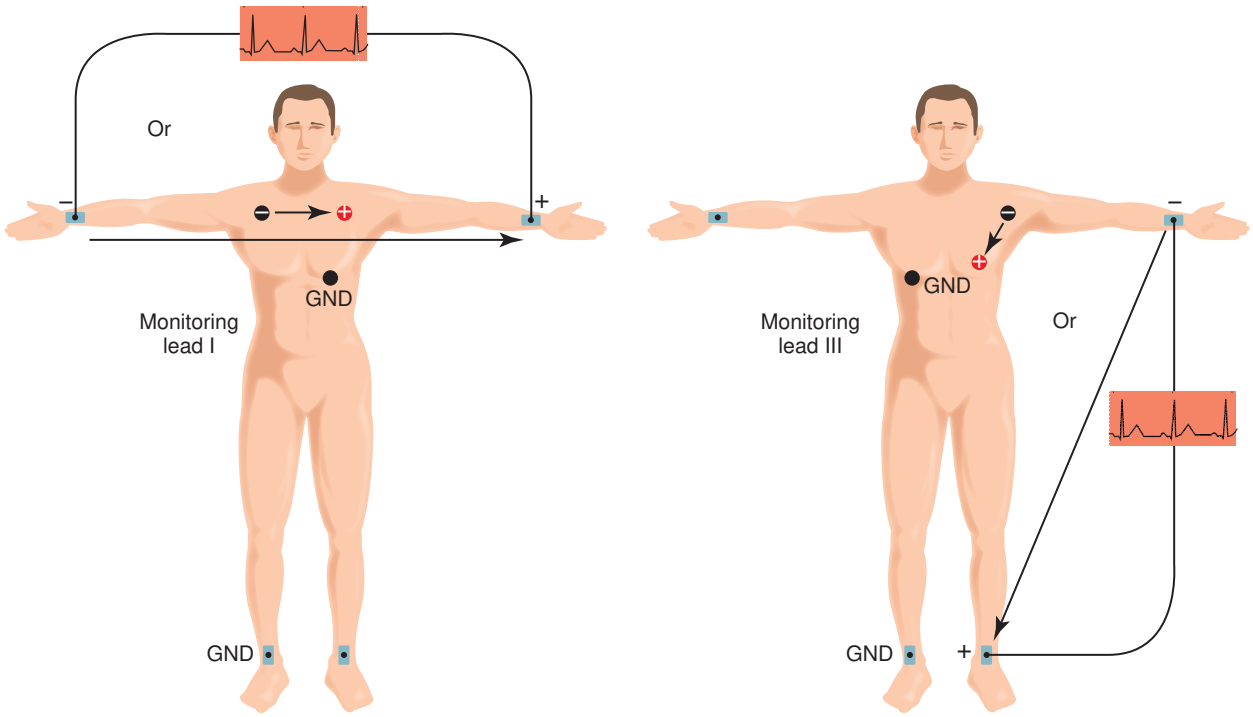


FIG. 2.6 Monitoring leads I and III.

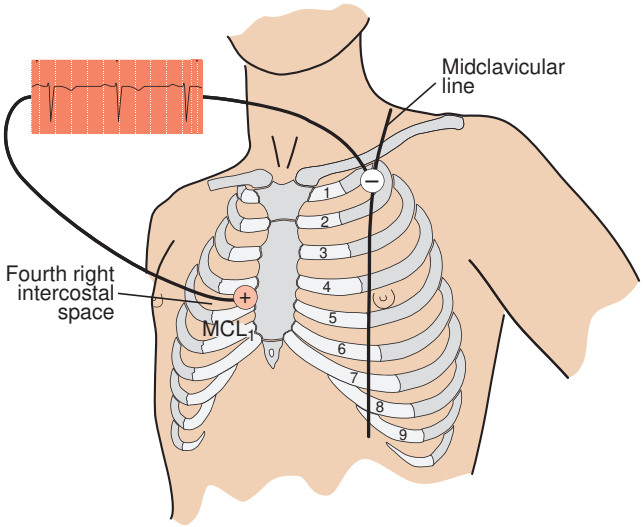


FIG. 2.7 Monitoring lead MCL_1.

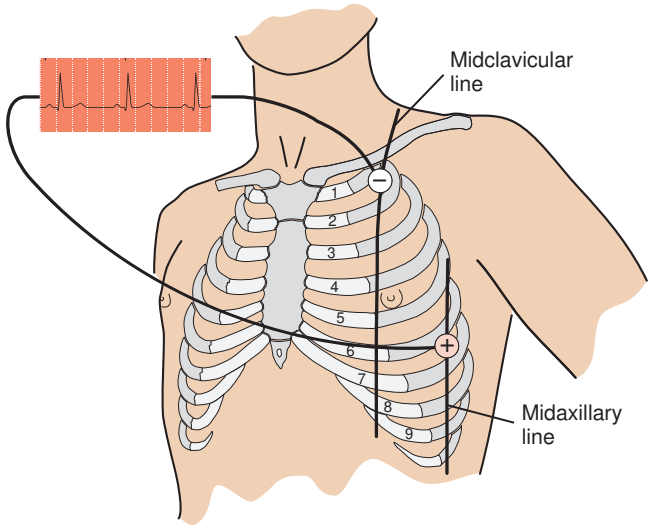


FIG. 2.8 Monitoring lead MCL_6.

- **Monitoring lead MCL_1.** Lead MCL_1 is a bipolar lead similar to lead V_1 of the 12-lead ECG (Fig. 2.7). It is obtained by attaching the positive electrode from lead III to the right side of the anterior chest in the fourth intercostal space just right of the sternum and the negative electrode to the left chest in the midclavicular line below the clavicle. Lead MCL_1 is helpful in identifying the origin of certain rhythms with wide QRS complexes, particularly when a full 12-lead ECG can't be obtained.
- **Monitoring lead MCL_6.** Lead MCL_6, a bipolar lead that resembles the unipolar lead V_6 of the 12-lead ECG, is obtained by attaching the positive electrode of lead III to the left chest in the fifth intercostal space in the midaxillary line and the negative electrode in the midclavicular line below the clavicle on the same side (Fig. 2.8). The P waves, QRS complexes, and T waves are similar to those in lead II. MCL_6, like MCL_1, is often used when a full 12-lead ECG can't be obtained.

Unipolar Leads

A lead that has only one electrode (which is positive) is called a *unipolar lead.* It does not have a corresponding negative lead. Instead, a theoretical electrode is created by the ECG machine to represent the center of the heart's electrical field. Unipolar leads are used extensively in 12-lead ECGs. As with bipolar leads, the view of the heart is from the perspective of the positive electrode.

unipolar lead

A view of the heart's electrical impulses from a single positive electrode.

There are 12 different leads in a standard ECG (Fig. 2.9), providing a detailed analysis of the heart's electrical activity. We will discuss 12-lead ECGs in detail in later chapters, but let's take a quick look now.

A 12-lead ECG consists of the following:

- Three standard (bipolar) limb leads (leads I, II, and III)
- Three augmented (unipolar) leads (leads aVR, aVL, and aVF)
- Six precordial (unipolar) leads (V_1, V_2, V_3, V_4, V_5, and V_6)

Augmented and precordial leads will be fully explained in Chapter 12.

The 12-lead ECG is used to diagnose changes associated with acute coronary syndrome (ACS), or "heart attack," and bundle-branch block. It also helps us differentiate between certain kinds of tachycardias (for instance, supraventricular versus ventricular). Clinicians frequently rely on the 12-lead ECG in the hospital, and it is the standard of care in prehospital medicine because of its accuracy in identifying patients with ACS so that they can be delivered efficiently to the most appropriate facility for definitive care.

ACQUIRING A QUALITY ECG

Artifacts

Artifacts are abnormal waves and spikes in an ECG that come from sources other than the heart's electrical activity. These traces of other activity or movement can interfere with or distort the components of the ECG, making interpretation difficult. The causes of artifacts include muscle tremor, alternating current (AC) interference, poor electrode contact with the skin, and external chest compression.

artifacts

Traces of activity or movement other than the heart's electrical activity that can distort the components of the ECG.

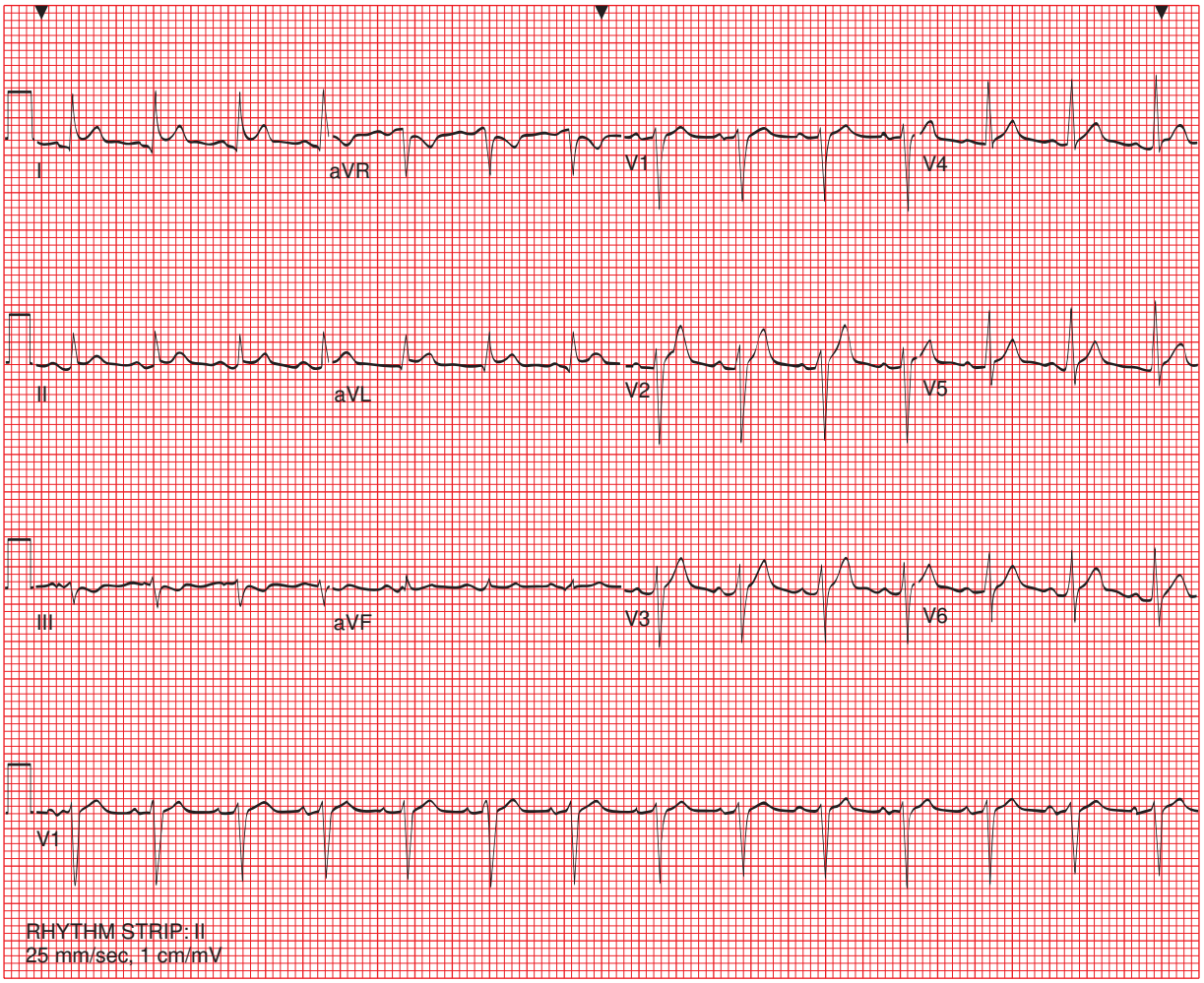


FIG. 2.9 Sample of a 12-lead ECG.

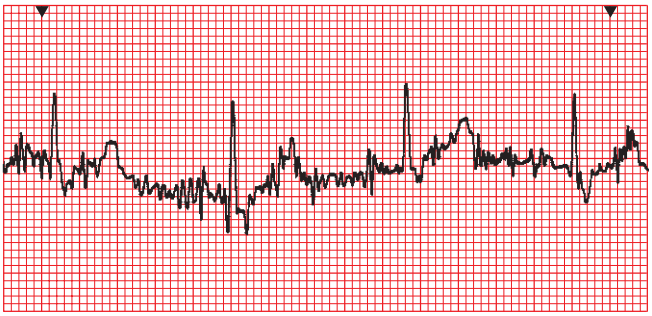


FIG. 2.10 Muscle tremor.

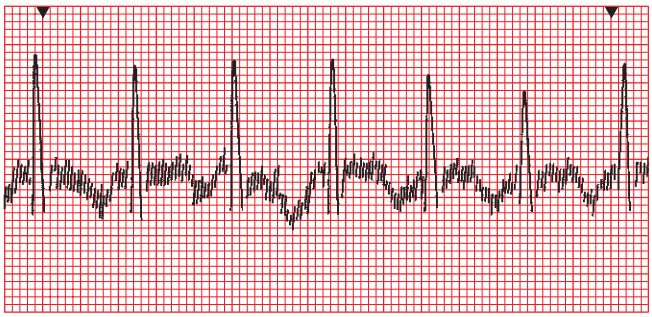


FIG. 2.11 Alternating current interference.

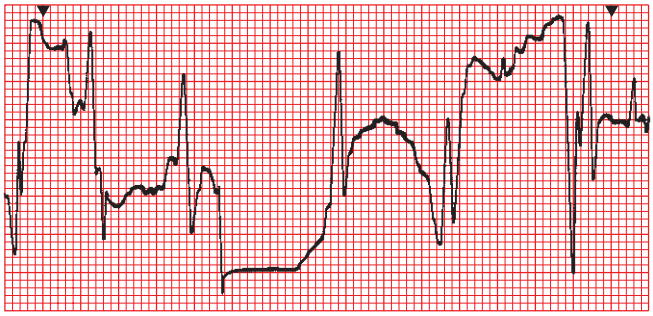


FIG. 2.12 Loose electrodes.

Muscle tremor (Fig. 2.10), for example, can occur in tense, nervous, or shivering patients, giving the ECG a jagged appearance that can be either fine or coarse. An artifact can also occur as a result of the patient breathing. This can cause the baseline of the ECG rhythm to wander up and down, making the determination of various abnormalities difficult.

AC electrical interference (Fig. 2.11) can occur when an improperly grounded AC-operated ECG machine is used or when an ECG is obtained near high-tension wires, transformers, or electrical appliances. This results in a thick baseline composed of 60-cycle waves.

Loose electrodes, or electrodes that are in poor contact with the skin (Fig. 2.12) (because of insufficient or dried electrode paste or jelly) can cause multiple sharp spikes and waves in the ECG. This is the most common cause of artifacts. Loose connecting wires can cause similar artifacts. In addition, any extraneous matter on the skin, such as blood, vomit, sweat, or hair, can result in poor electrode contact and the appearance of artifacts.

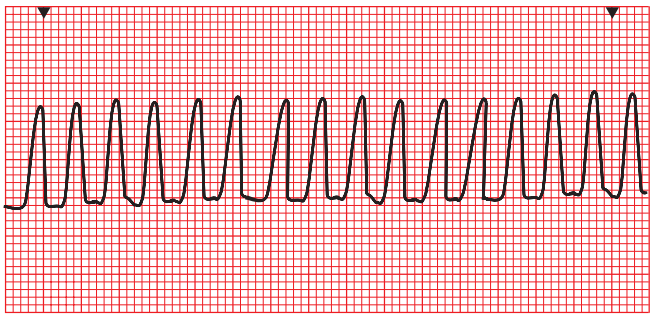


FIG. 2.13 External chest compression.

External chest compression (Fig. 2.13) during cardiopulmonary resuscitation (CPR) causes regularly spaced, wide, upright waves. The waves occur in sync with the rhythmic downward compressions.

AUTHOR'S NOTE Unfortunately, the appearance of waves on the ECG during CPR does not necessarily indicate that the chest compression is producing adequate cardiac output and circulation.

QRS Size and Wandering Baseline

The ECG machine can amplify the signal it receives and display it on the monitor. If the signal strength is low, most machines have a control that allows you to increase the amplitude. This control is called the *gain*. Increasing the gain will increase the size of the ECG waveform printed on the ECG graph paper and can be very helpful when you are using only the monitor to interpret the rhythm.

gain

An adjustable control on an ECG machine that allows the operator to amplify its electrical signal.

Dense tissue increases resistance to the signal as it passes through the chest. Causes of low-amplitude waveforms on the ECG include large barrel chests and/or obesity.

TAKE-HOME POINTS

- An electrocardiogram or ECG is a graphical representation of the electrical impulses generated during the depolarization and repolarization of the atria and ventricles. This signal is detected by electrodes attached to the body.
- The resulting ECG waveform is displayed on the monitor and printed on ECG graph paper for analysis.
- The ECG graph paper is designed to allow accurate measurement of both the strength (amplitude) and duration or timing of the various components of the waveform.
- The ECG waveform is detected by multiple leads, each of which provides a different view of the electrical activity of the heart.
- Waves and complexes are evaluated by shape and duration, intervals are analyzed by length, and segments are measured in relation to the baseline.

- The basic components of the ECG are the P wave; Q, R, and S waves; the T wave; the PR, ST, and TP segments; the PR, QT, and R-R intervals; and the J point.
- There are bipolar and unipolar leads. The bipolar leads (leads I, II, and III) have two electrodes attached to the body, whereas the unipolar leads have one. Lead II is the most common lead used in ECG rhythm interpretation.
- MCL1 and MCL6 are bipolar leads that can be used when a full 12-lead ECG cannot be obtained. They mimic the unipolar leads V1 and V6.
- Unipolar leads have only one electrode attached to the body and are used only in 12-lead ECGs.
- Rhythm interpretation generally relies on bipolar leads, whereas unipolar leads are commonly used to identify various ACSs.
- Artifacts on an ECG are traces of activity or movement other than the heart's electrical activity that can distort the components of the ECG. Artifacts can be caused by muscle tremors, AC interference, poor electrode contact with the skin, or external chest compression.
- Adjusting the gain on an ECG machine to amplify the signal can be helpful when using only the monitor for rhythm interpretation.

CHAPTER REVIEW QUESTIONS

1. What kind of electrical activity does an ECG record?
   A. The depolarization and repolarization of the atria and ventricles
   B. The flow of blood through the heart
   C. The mechanical contraction and relaxation of the atria and ventricles
   D. The transmission of electrical impulses responsible for initiating depolarization of the atria and ventricles

2. When an ECG is recorded at the standard paper speed of 25 mm/sec, how many seconds apart are the dark vertical lines and the light vertical lines?
   A. 5 sec and 1 sec
   B. 20 sec and 4 sec
   C. 0.20 sec and 0.4 sec
   D. 0.20 sec and 0.04 sec

3. The sensitivity of the ECG machine is calibrated so that a(n) _____ electrical signal produces a(n) _____ deflection on the ECG.
   A. 0.5-mV; 1-mm
   B. 1-mV; 10-mm
   C. 5-mV; 10-mm
   D. 10-mV; 5-mm

4. Which part of the waveform shows the electrical impulses generated by ventricular depolarization?
   A. P wave
   B. QRS complex
   C. Atrial T wave (T_a)
   D. T wave

5. Which part of the waveform shows the electrical impulses generated by ventricular repolarization?
   A. P wave
   B. QRS complex
   C. Atrial T wave (T_a)
   D. T wave

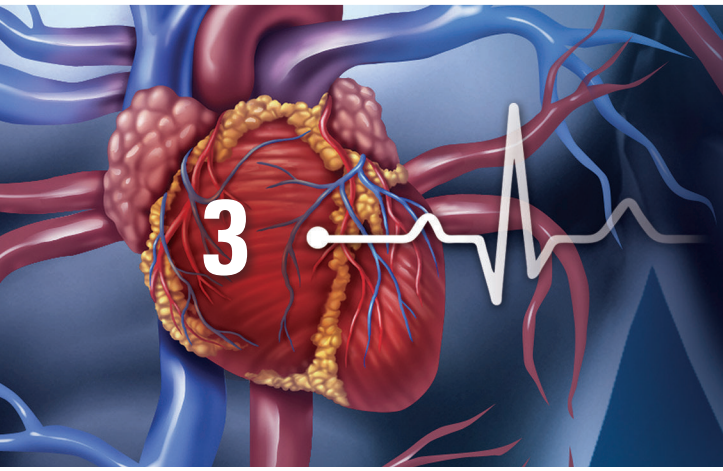
6. Which of the following is the most common cause of an ECG artifact?
   A. External chest compression
   B. Muscle tremor
   C. Poor electrode contact with the skin
   D. Turning up the gain

7. Which ECG lead is composed of a single positive electrode and a reference point located at the center of the heart?
   A. Bipolar lead
   B. MCL_1 lead
   C. Multifocal lead
   D. Unipolar lead

8. Monitoring lead II is obtained by attaching the negative electrode and positive electrode to what?
   A. Left arm and left leg
   B. Right arm and left arm
   C. Right arm and left leg
   D. Right arm and left upper chest

9. If the positive electrode is attached to the left leg or lower left anterior chest, in which direction will the electrical impulses that flow toward the positive electrode be deflected on the ECG?
   A. Negative (downward)
   B. Negative (upward)
   C. Positive (downward)
   D. Positive (upward)

10. Monitoring lead MCL_1 is obtained by attaching the positive electrode from lead III to which part of the body?
    A. Left chest below the clavicle
    B. Middle of the sternum at the level of the fourth intercostal space
    C. Left side of the sternum in the fourth intercostal space
    D. Right side of the anterior chest in the fourth intercostal space next to the sternum



3 Components of the ECG Waveform

OUTLINE

In 1887, Professor A. D. Waller was the first to record the electrical activity of the human heart. Using a device called the *Lippmann capillary electrometer,* Waller initially detected only two waves. He labeled them V_1 and V_2, to indicate ventricular events in the heart. Using the same device, Dr. William Einthoven detected four waves, which he initially labeled A, B, C, and D. Further refinement of the device revealed more wave patterns. Einthoven chose to relabel these waveforms P, Q, R, S, and T, in recognition of the letters used by the analytical mathematician René Descartes to describe points along a curve. In 1903, Einthoven invented a more sophisticated ECG machine that could record a detailed tracing of the heart's electrical activity. For that accomplishment, he was awarded the Nobel Prize in Medicine in 1924. As a result, Einthoven is sometimes called "the father of modern electrocardiography."

AUTHOR'S NOTE Lead II is the most common lead used to analyze the heart's electrical activity. For that reason, the shape (morphology) of the ECG waveforms presented in this and subsequent chapters will be described as they are seen in lead II unless other leads provide a better "view."

As noted in Chapter 2, the ECG waveform is composed of waves, intervals, and segments (see Fig. 2.2).

WAVES

P WAVE

P wave

A P wave represents depolarization of the right and left atria.

Normal Sinus P Wave

CHARACTERISTICS

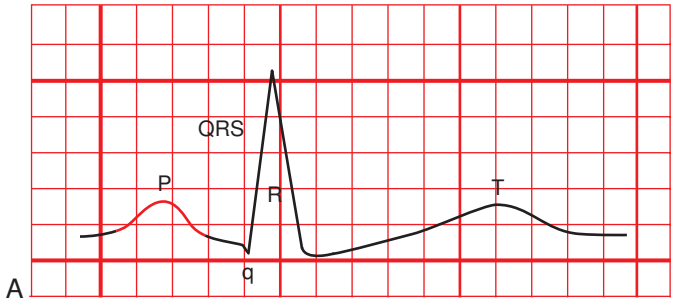
Origin	Sinoatrial (SA) node
Physiology	Atrial depolarization
Onset/End	TP segment/ PR segment
Direction	Upward
Duration	0.08–0.10 sec
Amplitude	0.5–2.5 mm
Shape	Smooth and rounded
Sequence	Precedes QRS complex unless a block is present

Origin

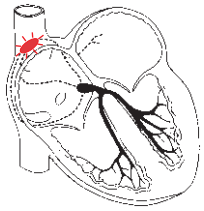
The normal P wave originates in the SA node and is called a *sinus P wave.*

Relationship to Cardiac Anatomy and Physiology

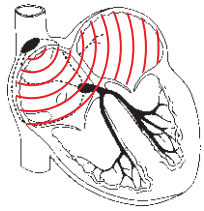
A normal sinus P wave (Fig. 3.1) represents normal depolarization of the myocardial cells of the atrial wall. Depolarization of the atria begins near the SA node and progresses from right to left and downward. The first part of the sinus P wave represents depolarization of the right atrium; the second part represents depolarization of the left atrium. During the P wave, the electrical impulse progresses from the SA node through the internodal atrial conduction tracts and most of the atrioventricular (AV) node.



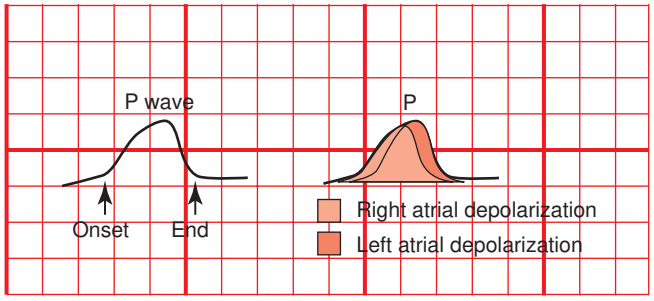
A Normal sinus P wave



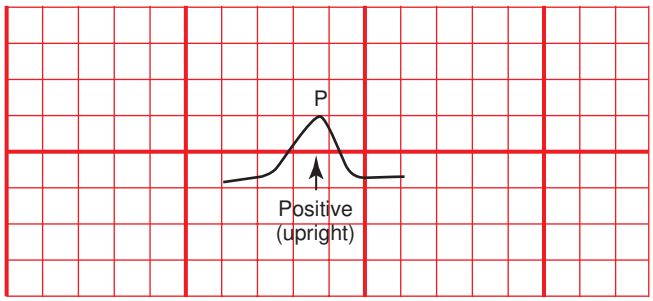
B Pacemaker site



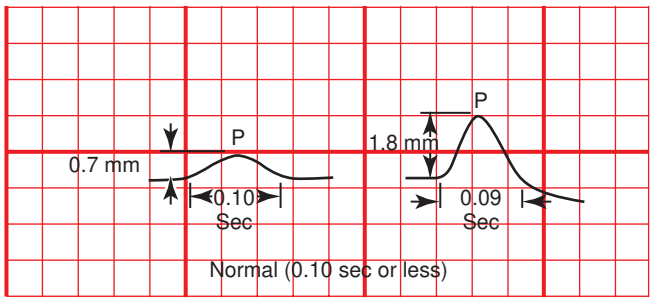
Significance



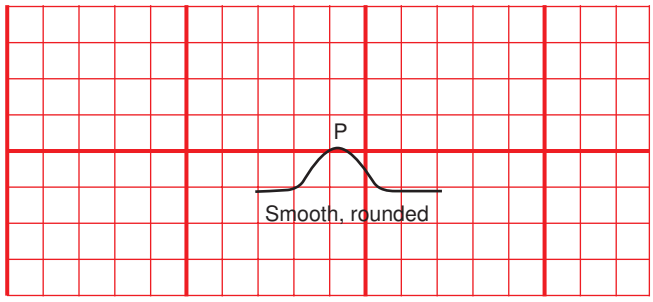
C Onset and end



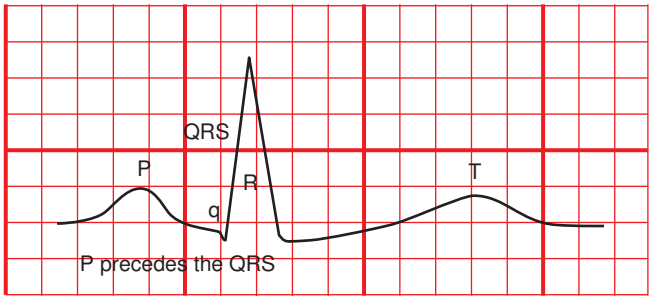
D Direction



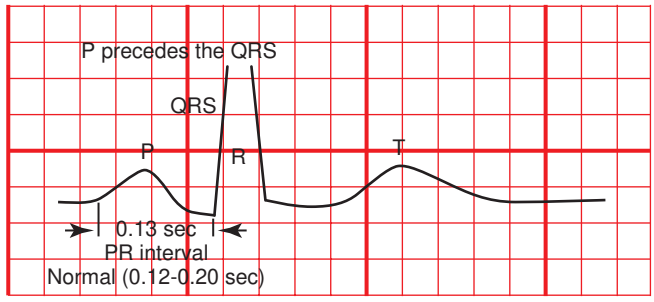
E Duration amplitude



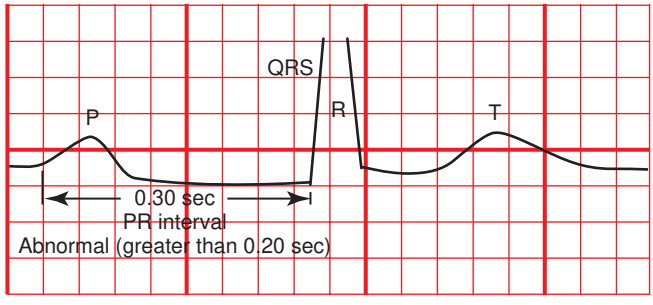
F Shape



G P-QRS relationship



H PR interval



I PR interval

FIG. 3.1 Normal sinus P wave.

DESCRIPTION

Onset and End

The onset of the normal P wave is identified as the first gradual deviation from the baseline. The point at which the wave flattens out to return to the baseline, joining with the PR segment, marks the end of the P wave.

Direction

The direction is positive (upward). This is because most of the impulse is directed toward the positive electrode of lead II, which is on the right upper chest or right arm.

Duration

From 0.08 and 0.10 second.

Amplitude

Ranges from 0.5 to 2.5 mm, although it rarely exceeds 2 mm in height.

Shape

Smooth and rounded.

Sequence

A QRS complex follows each sinus P wave except in certain rhythms, such as AV blocks (see Chapter 9).

SIGNIFICANCE

A normal sinus P wave indicates that the electrical impulse responsible for the P wave originated in the SA node and that normal depolarization of the right and left atria has occurred.

Abnormal Sinus P Wave

CHARACTERISTICS

Origin

The abnormal sinus P wave originates in the SA node.

Relationship to Cardiac Anatomy and Physiology

An abnormal sinus P wave (Fig. 3.2) represents depolarization of damaged or abnormal atria. Increased right atrial pressure and right atrial dilatation and hypertrophy may result in P waves that are abnormally tall or wide or that deflect in abnormal directions.

P waves that occur in two phases are called *biphasic P waves.* This pattern occurs in both right and left atrial dilation and hypertrophy. Biphasic P waves are best detected in leads V_1 and V_2 because these two unipolar leads have direct views of the SA node from the front of the chest. They will show an initial positive deflection during right atrial depolarization. Then a negative deflection will appear as the left atrium is depolarized. Conditions with biphasic P waves are described in Chapter 14.

DESCRIPTION

Onset and End

Same as for those of a normal P wave.

Direction

Positive (upright) in lead II; may be biphasic (initially positive, then negative) in leads V_1 and V_2.

Duration

Usually normal (0.08–0.10 sec) and rarely greater than 0.16 second.

Amplitude

Either normal (0.5–2.5 mm) or greater than normal. When the amplitude of the P wave is greater than 2.5 mm, it is referred to as *P pulmonale.*

Shape

Tall and symmetrically peaked or wide and notched. By definition, notched P waves equal to or greater than 0.12 second with the top of each mound greater than 0.04 second apart is called *P mitrale* and may be biphasic in leads V_1 and V_2.

Sequence

Same as that of a normal sinus P wave.

SIGNIFICANCE

The presence of an abnormal sinus P wave indicates that although the electrical impulse responsible for the P wave originated in the SA node, changes in the atrial walls altered depolarization of the atrial muscle.

Ectopic P Wave: P Prime, or P′

ectopic P wave

A P wave produced by depolarization of the atria in an abnormal direction.

CHARACTERISTICS

Origin

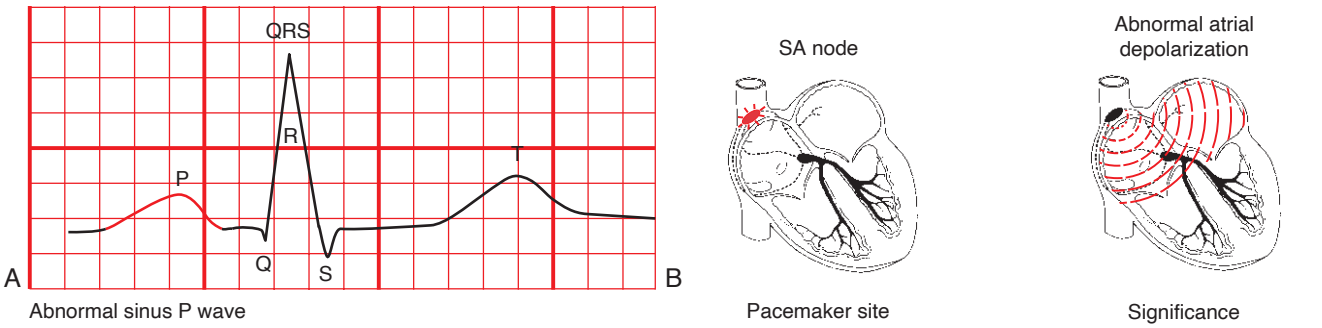
An ectopic P wave is called *P prime,* denoted as P′. It can originate in the AV junction or anywhere in the atrium other than the SA node.

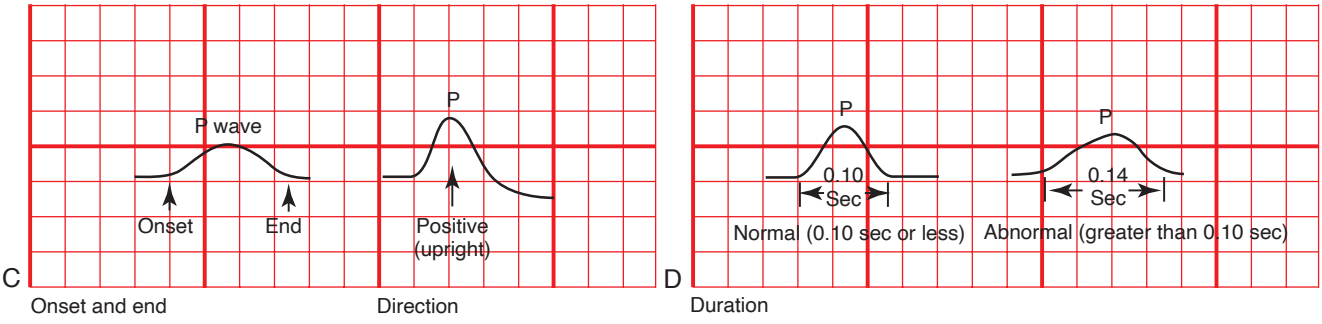
AUTHOR'S NOTE The word *ectopic* comes from the Greek *ek-,* meaning "outside of," and *topos,* meaning "place or location." An ectopic P wave is one that originates outside the SA node.

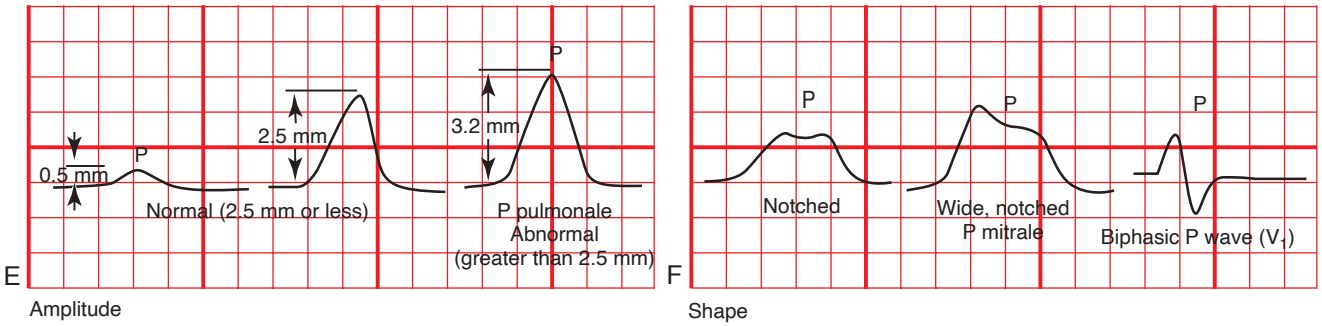
Relationship to Cardiac Anatomy and Physiology

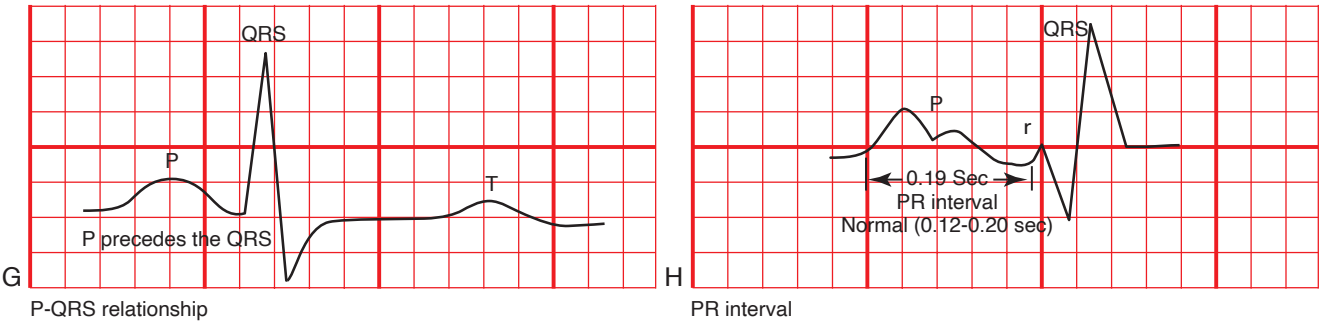
An ectopic P wave (P′) (Fig. 3.3) represents atrial depolarization originating somewhere in the atrium other than the SA node and proceeding in an abnormal direction, sequence, or both, depending on where the impulse originates. We call this location an *ectopic pacemaker* because the site sets the pace of the rhythm.

- If the ectopic pacemaker is in the upper or middle right atrium, depolarization of the atria occurs in a normal, antegrade direction (right to left and downward).









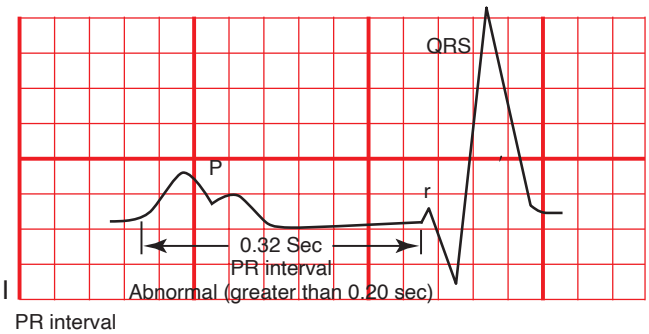


FIG. 3.2 Abnormal sinus P wave.

- If the ectopic pacemaker is in the lower right atrium near the AV node or in the left atrium, depolarization of the atria occurs in a retrograde direction (left to right and upward).
- If the ectopic pacemaker is in the AV junction, the electrical impulse travels upward through the AV junction into the atria (retrograde conduction), causing retrograde atrial depolarization. Ectopic P waves occur in the following ECG rhythms:
  - Wandering atrial pacemaker
  - Premature atrial complexes
  - Atrial tachycardia

DESCRIPTION

Onset and End

Same as those of a normal P wave.

Direction

Either positive (upright) or negative (inverted) if the ectopic pacemaker is in the atria. Generally, if the ectopic pacemaker is in the upper part of the right atrium, the P′ wave is positive, resembling a normal sinus P wave.

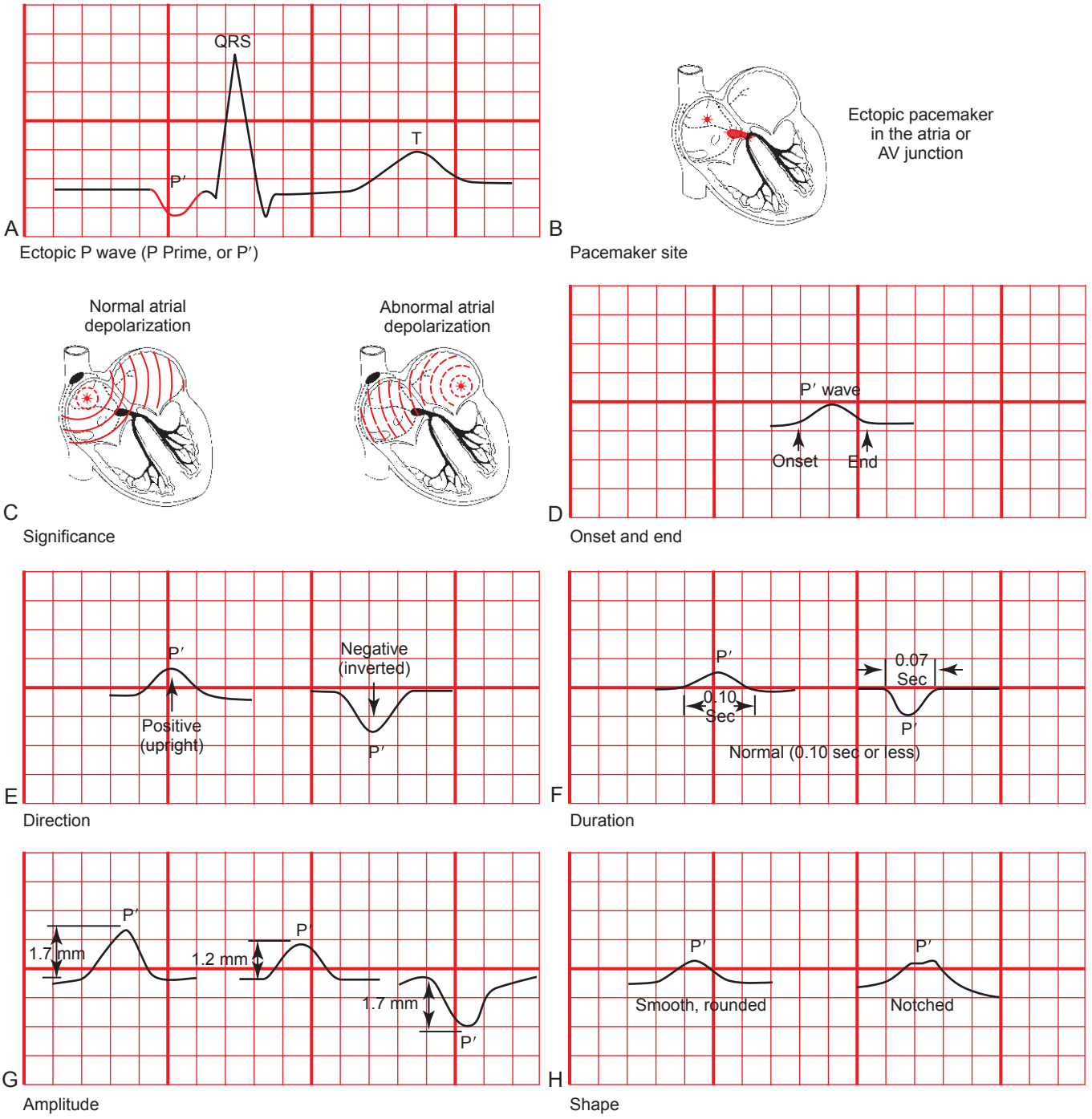


FIG. 3.3 Ectopic P wave (P prime, or P′).

Continued

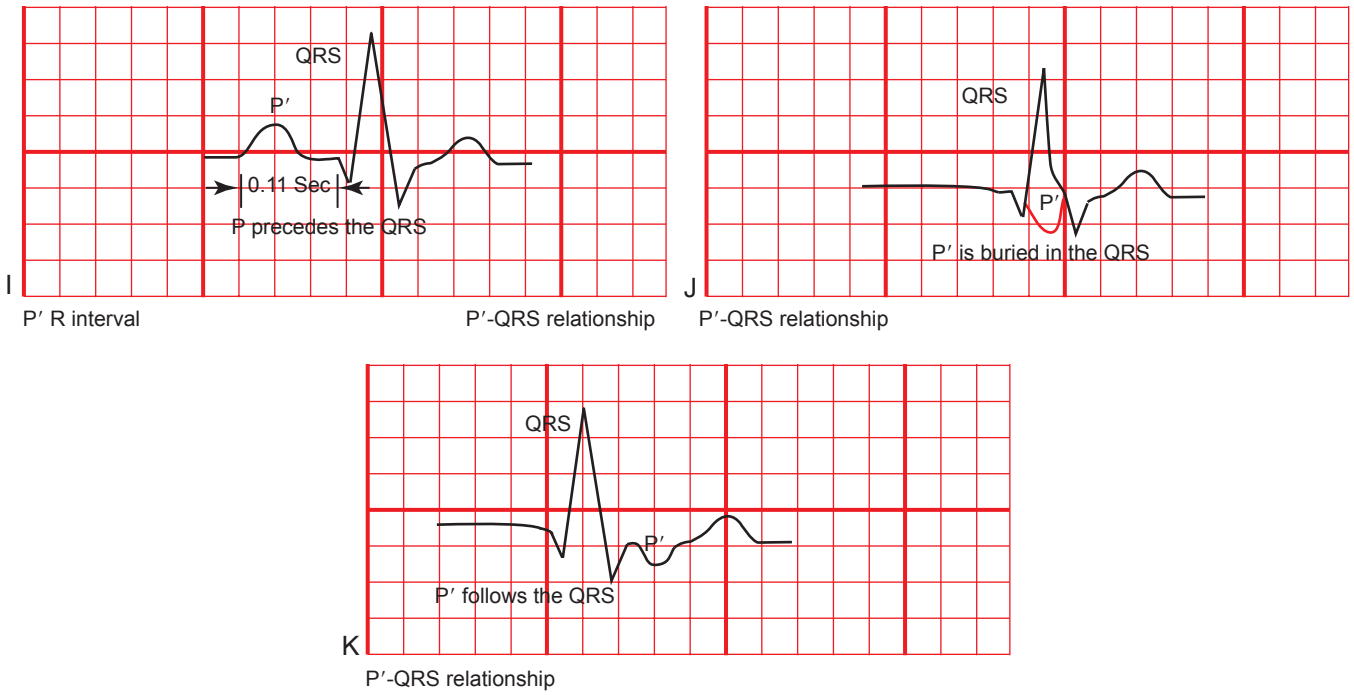


FIG. 3.3 cont'd

If the ectopic pacemaker is in the middle of the right atrium, the P′ wave is less positive (upright) than one arising from the upper right atrium. If the ectopic pacemaker is in the lower right atrium near the AV node or in the left atrium or in the AV junction, the P′ wave is negative (inverted).

Duration

Normal or prolonged, depending on the site of origin.

Amplitude

Usually less than 2.5 mm.

Shape

May be smooth and rounded, peaked, or slightly notched.

Sequence

The ectopic P wave may precede, be embedded in, or follow the QRS complex with which it is associated.

- If the ectopic pacemaker is in any part of the atria or in the upper part of the AV junction, the P′ wave generally precedes the QRS complex.
- If the ectopic pacemaker is in the lower part of the AV junction, the P′ wave can occur during or even after the QRS complex. This is because the electrical impulses travel more quickly through the ventricles than they do backward through the AV junction, causing them to appear on the ECG embedded in or after the QRS complex.

If the P′ wave occurs during the QRS complex, it is embedded within the QRS complex and is said to be hidden or invisible. If it follows the QRS complex, it becomes superimposed on (laid on top of) the ST segment and/or T wave, distorting them.

SIGNIFICANCE

An ectopic P wave indicates that the electrical impulse originated in part of the atria outside the SA node and that depolarization of the right and left atria has occurred in an abnormal direction, sequence, or both.

AUTHOR'S NOTE Another type of ectopic atrial depolarization is seen in atrial fibrillation and atrial flutter. In atrial fibrillation, multiple randomly located ectopic impulses result in the absence of a single P wave. These are called *f waves*. In atrial flutter, a single ectopic atrial depolarization is spontaneously firing at a high rate. This results in regular sawtooth-shaped waves called *F waves*. This will be explored in detail in Chapters 4 and 6.

QRS COMPLEX

QRS complex

The QRS complex represents depolarization of the right and left ventricles.

Normal QRS Complex

CHARACTERISTICS

Origin	Interventricular septum below AV junction
Physiology	Depolarization of ventricles
Onset/End	Deviation from PR interval/beginning of ST segment

Duration	0.06–0.12 sec
Direction	
Q wave	First negative deflection
R wave	First positive deflection
S wave	First negative deflection after an R wave
QS wave	Single negative deflection
RS wave	Single positive deflection
Amplitude	2–15 mm
Shape	Narrow and sharply pointed
Sequence	Follows the P wave and precedes the T wave

Origin

Interventricular septum just below the AV junction.

Relationship to Cardiac Anatomy and Physiology

A normal QRS complex (Fig. 3.4) represents normal depolarization of the ventricles. Depolarization begins in the left side of the interventricular septum near the AV junction and progresses across the interventricular septum from left to right. Then, beginning at the endocardial surface of the ventricles, depolarization progresses through the ventricular walls to the epicardial surface.

The first short part of the QRS complex, usually the Q wave, represents depolarization of the interventricular septum; the rest of the QRS complex represents the simultaneous depolarization of the right and left ventricles. Because the left ventricle is larger than the right ventricle and has more muscle mass, the QRS complex represents, for the most part, depolarization of the left ventricle.

DESCRIPTION

Onset and End

The onset of the QRS complex is identified as the point where the first wave of the complex just begins to deviate, usually abruptly, from the baseline of the PR interval following the end of the P wave. The end of the QRS complex is the point where the last wave of the complex sharply flattens to meet the ST segment. This point, the junction between the QRS complex and the ST segment, is called the *junction* or *J point.*

Components

The QRS complex consists of one or more of the following: positive (upright) deflections called *R waves* and negative (inverted) deflections called *Q, S,* and *QS waves.* The characteristics of the waves that make up the QRS complex are as follows:

- **Q wave:** The Q wave is the first negative deflection in the QRS complex not preceded by an R wave.
- **R wave:** The R wave is the first positive deflection in the QRS complex. Subsequent positive deflections are called *R prime (R′), R double prime (R″),* and so forth.
- **S wave:** The S wave is the first negative deflection in the QRS complex after an R wave. Subsequent negative deflections are called *S prime (S′), S double prime (S″),* and so forth.
- **QS wave:** A QS wave is a QRS complex that consists entirely of a single, large negative deflection without an intervening positive deflection.

Whereas there is only one Q wave, there can be more than one R and S wave in the QRS complex.

The waves comprising the QRS complex are usually identified by uppercase (capital) or lowercase (small) letters, depending on the relative size of the waves. In other words, the major deflections—the large waves—are identified by the letters Q, R, and S. The smaller waves, which are less than half the amplitude of the major deflections, are identified by the letters q, r, and s. Thus the ventricular depolarization complex can be described more accurately by using the specific letters assigned to the waves (for example, qR, Rs, or qRs). However, we still refer to it as a QRS complex when discussing this complex in general.

Direction

The QRS complex is described as being mostly positive (upright), mostly negative (inverted or downward), or biphasic (positive and negative). In a mostly positive QRS complex, for example, the R wave—the major deflection—covers more area than the Q and S waves do. Usually this is easy to see simply by looking at the QRS complex; however, if you are unsure, place a ruler at the baseline and estimate the number of small squares covered by the height and depth of the QRS complex above and below the baseline.

Duration

The duration of the QRS complex is measured from the onset of the Q or R wave to the end of the last wave of the complex, called the *J point,* and is normally 0.06 to 0.12 second in adults and 0.08 second or less in children. The duration of the Q wave is normally 0.04 second or less.

> **AUTHOR'S NOTE** The time between the onset of the Q wave and the peak of the R wave is the ventricular activation time (VAT). The VAT is the time it takes for depolarization of the interventricular septum and depolarization of the ventricle from the endocardium to the epicardium under the facing lead. The upper limit of the normal VAT is 0.05 second. Some texts refer to the VAT as the *intrinsicoid deflection.*

Amplitude

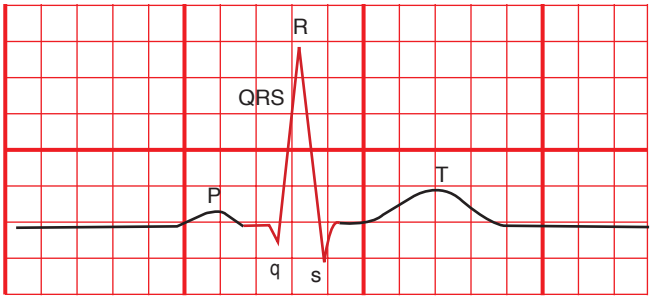
The amplitude of the R or S wave in the QRS complex varies from 2 to 15 mm or more. The normal Q wave is less than 25% of the height of the R wave that follows it.

Shape

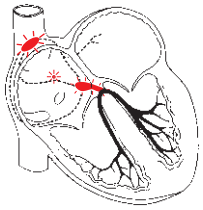
The normal QRS complex is generally narrow and sharply pointed.

Sequence

The normal QRS complex follows the P wave and precedes the T wave.

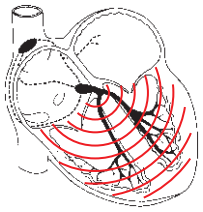


A Normal QRS complex



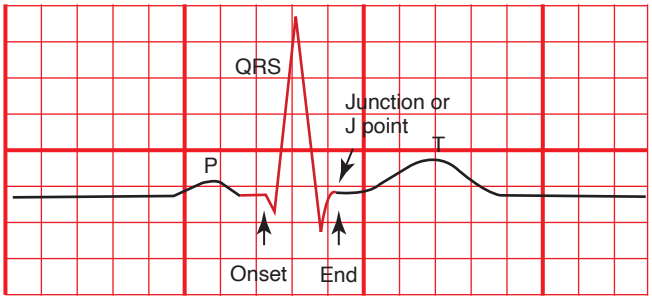
SA node or ectopic pacemaker in the atria or AV junction

B Pacemaker site

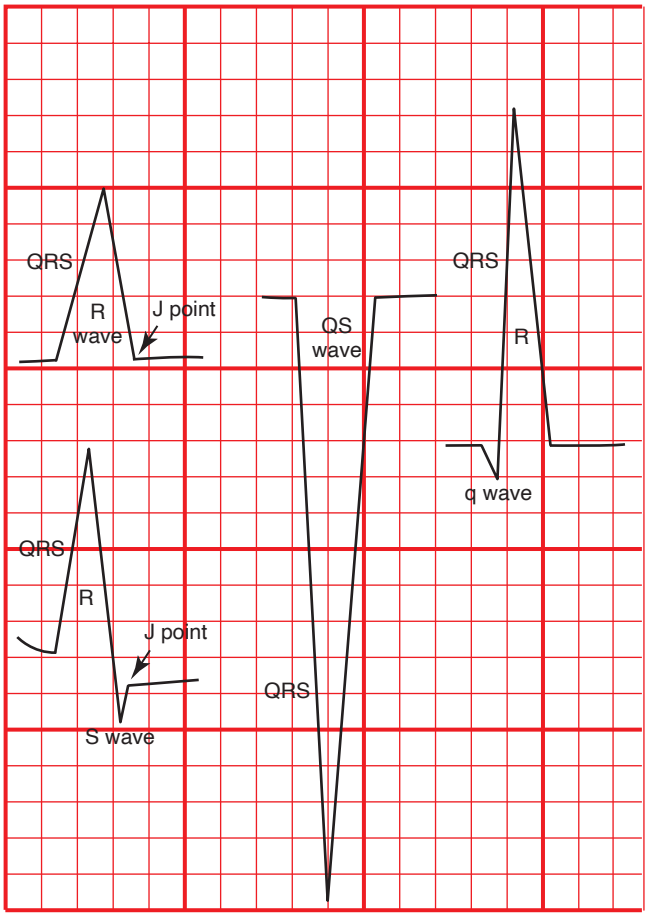


Normal ventricular depolarization

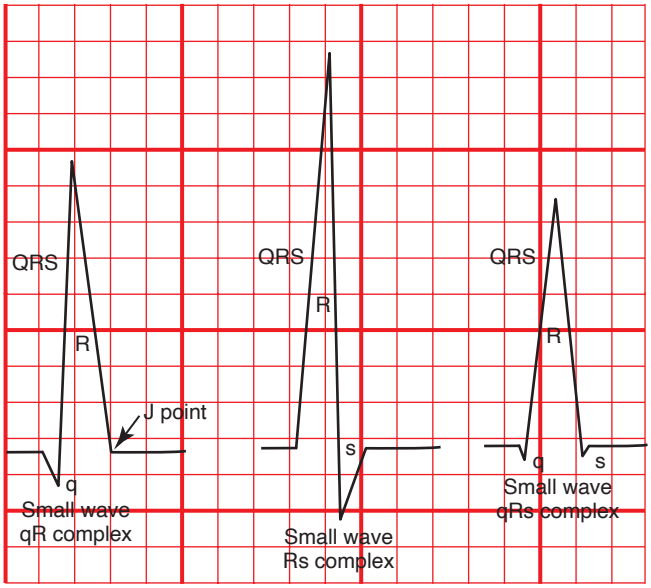
C Significance



D Onset and end



E Components



F Large wave major deflection

FIG. 3.4 Normal QRS complex.

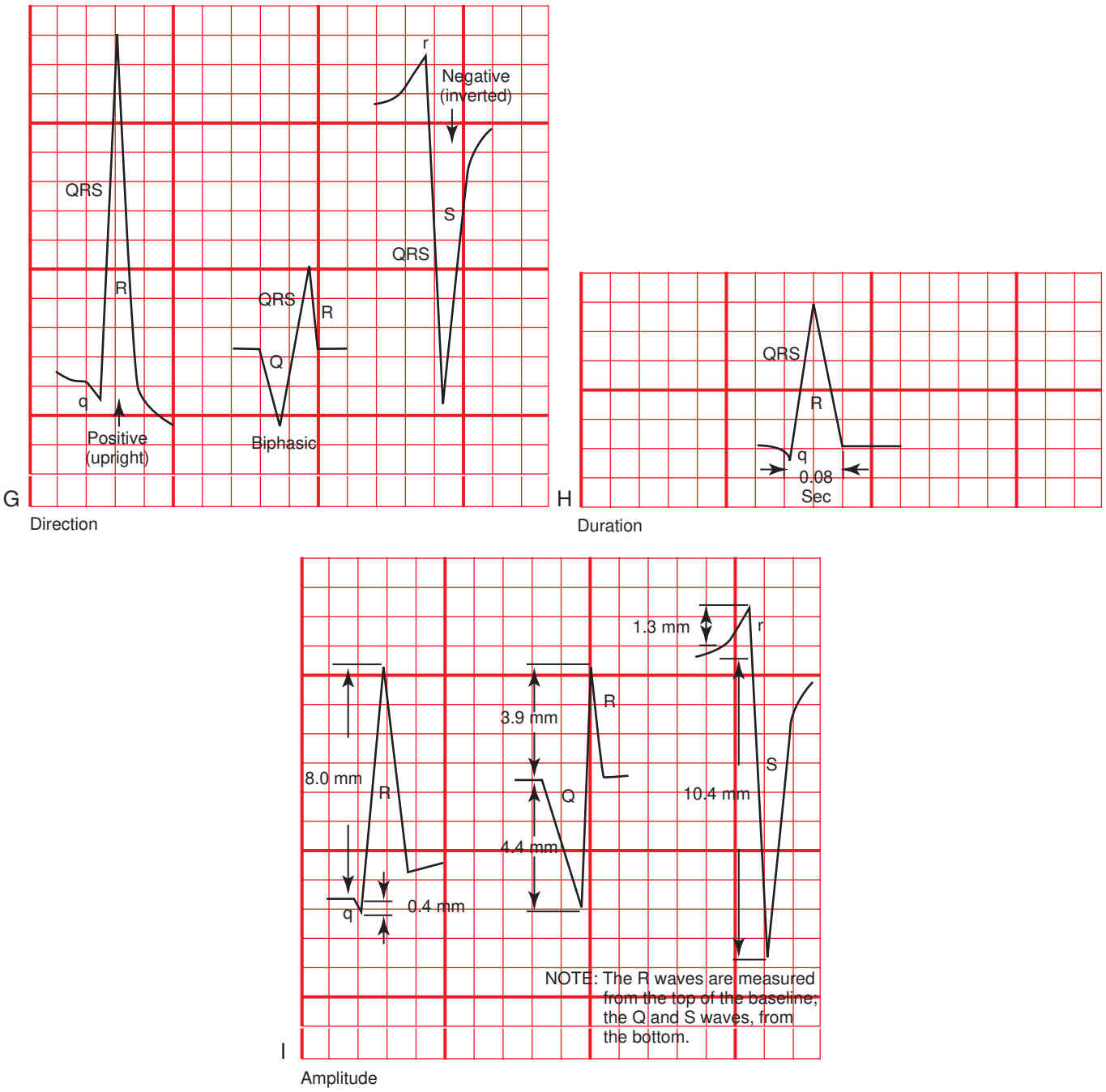


FIG. 3.4 cont'd

SIGNIFICANCE

A normal QRS complex indicates that the electrical impulse has progressed normally from the AV junction down the bundle of His to the Purkinje network and through the right and left bundle branches to depolarize the right and left ventricles normally.

Abnormal QRS Complex

CHARACTERISTICS

Origin

Within or below the AV junction or from the bundle branches, Purkinje network, or ventricular myocardium.

Relationship to Cardiac Anatomy and Physiology

An abnormal QRS complex (Fig. 3.5) represents abnormal depolarization of the ventricles. It is referred to as *aberrant (abnormal) ventricular conduction.* The cause may be any of the following:

- Intraventricular conduction disturbance (such as a bundle-branch block)
- Ventricular preexcitation
- An ectopic electrical impulse
- Ventricular pacing by an implanted cardiac pacemaker

Intraventricular conduction disturbance is most often caused by right or left bundle-branch block. It can also be caused by a nonspecific, widespread electrical conduction defect associated

with specific types of heart diseases, electrolyte imbalances, or excessive administration of certain cardiac drugs. Bundle-branch block is caused by the blockage or partial blockage of electrical impulses from the bundle of His to the Purkinje network through the right or left bundle branch. Conduction through the unaffected bundle branch is unimpeded (see Chapter 13). A block in one bundle branch causes depolarization of the ventricle on that side to occur later than on the unaffected side because the affected ventricle must wait until the electrical impulse travels through a longer and less efficient route to depolarize the myocardial cells. This delay results in a widened QRS that exceeds 0.12 seconds in duration.

In partial or incomplete bundle-branch block, conduction of the electrical impulse is only partially blocked, resulting in less of a delay in depolarization of the ventricle on the side of the block than in complete bundle-branch block. Consequently, the QRS complex is greater than 0.10 but less than 0.12 seconds in duration and often appears normal.

Complete and incomplete bundle-branch block may be present in normal sinus rhythm and in any supraventricular arrhythmia (that is, any arrhythmia arising above the ventricles in the SA node, atria, or AV junction).

Ventricular preexcitation is a transient inability of the right or left bundle branch to conduct an electrical impulse normally. This may occur when an electrical impulse arrives at the bundle branch that has just conducted an electrical impulse while it is still refractory to conducting another. This occurs with premature atrial complexes and some tachycardias. It results in an abnormal QRS complex that often resembles an incomplete or complete bundle-branch block.

Abnormal ventricular conduction may occur in the following supraventricular rhythms that mimic ventricular rhythms (see Chapter 8):

- Premature atrial and junctional complexes
- Atrial tachycardia
- Atrial flutter and fibrillation
- Nonparoxysmal junctional tachycardia
- Paroxysmal supraventricular tachycardia

An electrical impulse originating in an ectopic or escape pacemaker in the bundle branches, Purkinje network, or myocardium of one of the ventricles depolarizes that ventricle earlier than the other. The result is an abnormal QRS complex that is greater than 0.12 second in duration and appears bizarre. Such QRS complexes typically occur in ventricular rhythms such as

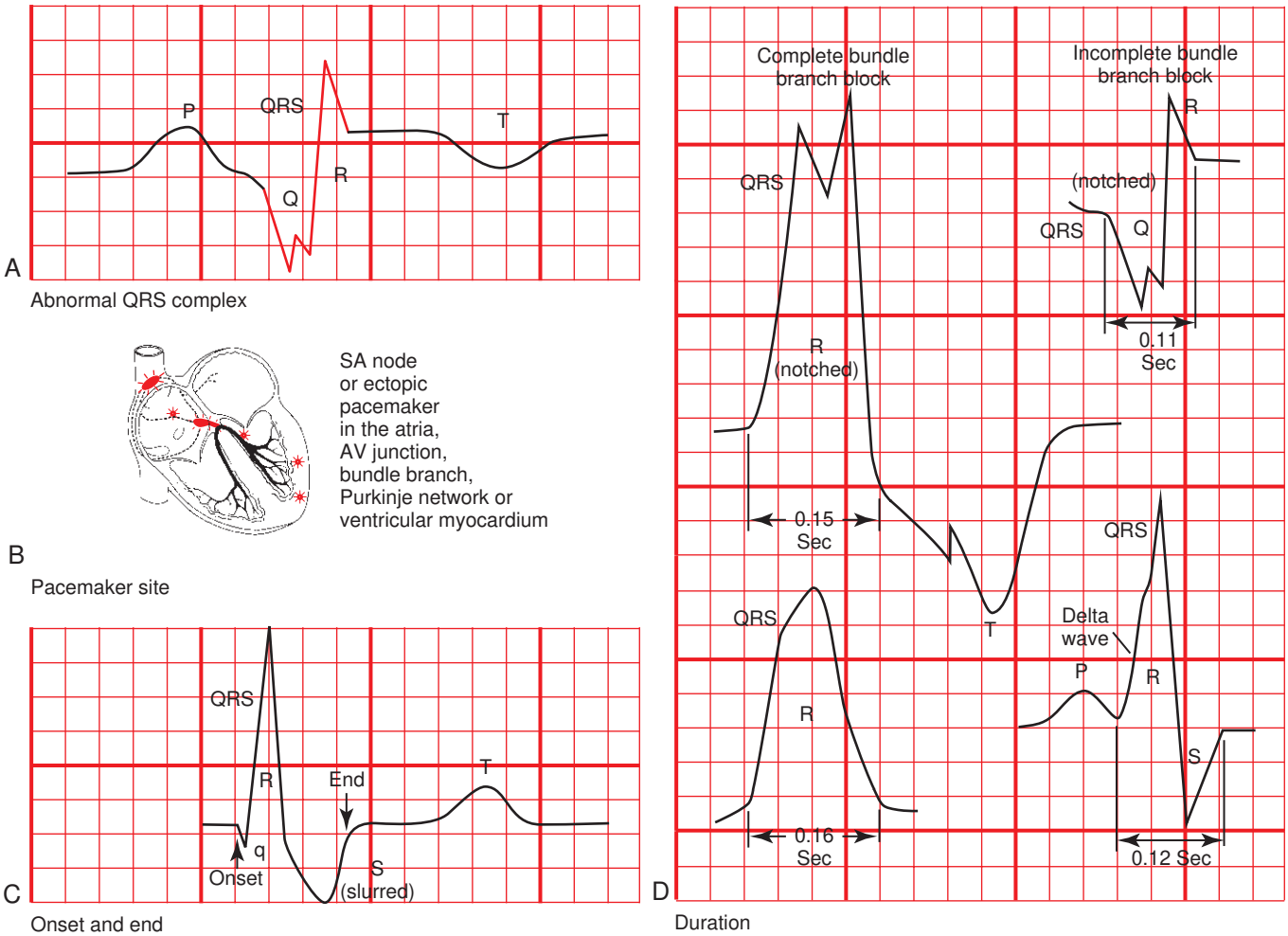
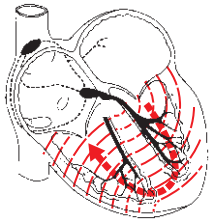
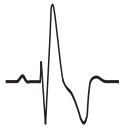


FIG. 3.5 Abnormal QRS complex.

1. Blockage of conduction of the electrical impulse through a bundle branch

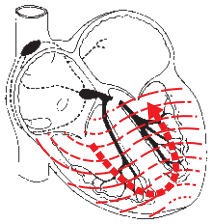
Block in right bundle branch





Right bundle branch block

Block in left bundle branch



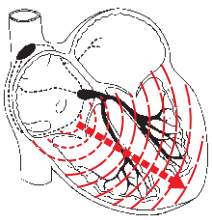
Direction of ventricular depolarization

Left bundle branch block

Bundle branch block and aberrant ventricular conduction

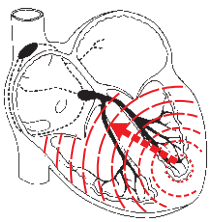
2. Conduction of the electrical impulse through accessory conduction pathways

Ventricular preexcitation



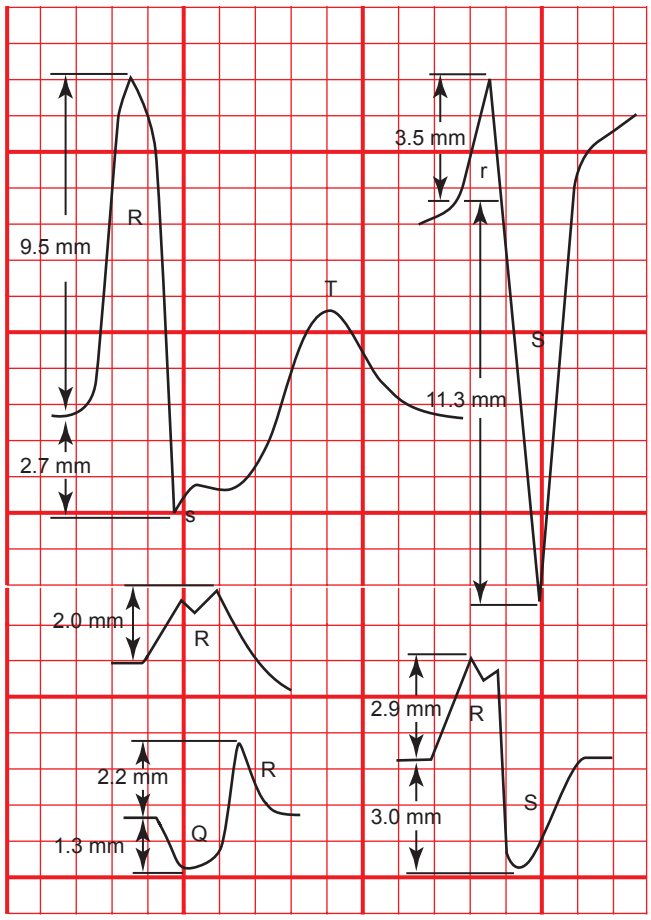
3. Ventricular ectopic or artificial cardiac pacemaker

Abnormal ventricular depolarization

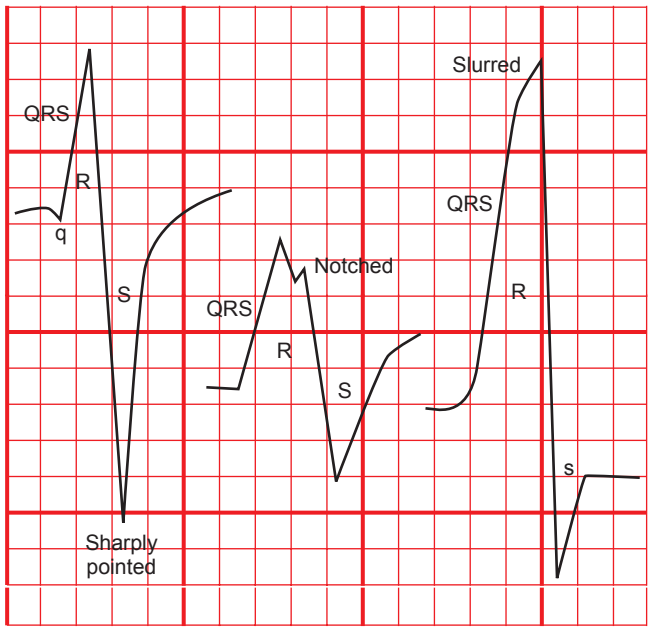


Ventricular pacemaker

E



F Amplitude



G Shape

FIG. 3.5 cont'd

accelerated idioventricular rhythm, ventricular escape rhythm, ventricular tachycardia, and premature ventricular complexes (see Chapter 8). The occurrence of ventricular ectopic beats or rhythms is often referred to as *ventricular ectopy.*

Implanted cardiac pacemaker–induced QRS complexes are generated by the electrical stimulation from a wire embedded in the inner wall of the right ventricle. The resulting QRS complexes are generally 0.12 second or greater in width, similar to a left bundle-branch block because the impulse must travel through a slower pathway from the right to the left ventricle. However, because the depolarization occurs in the endocardium of the ventricle, the cardiac pacemaker–induced QRS complex has a similar shape as a ventricular ectopic complex. A common indicator of a pacemaker-induced QRS complex is a narrow deflection, often biphasic, preceding the QRS complex called the *pacemaker spike,* which is the electrical stimulus from the pacemaker wire. (see Chapter 10).

DESCRIPTION

Onset and End

Same as those of a normal QRS complex.

Direction

Mostly positive (upright), mostly negative (inverted), or biphasic (positive and negative).

Duration

Greater than 0.12 second. If a bundle-branch block is present and the duration of the QRS complex is between 0.10 and 0.12 second, the bundle-branch block is called *incomplete.* If the duration of the QRS complex is greater than 0.12 second, the bundle-branch block is called *complete.* In ventricular preexcitation, the duration of the QRS complex is greater than 0.12 second.

The duration of a QRS complex caused by an electrical impulse originating in an ectopic or escape pacemaker in the Purkinje network or ventricular myocardium is always greater than 0.12 second; typically, it is 0.16 second or greater. However, if the electrical impulse originates in a bundle branch, the duration of the QRS complex may be only slightly greater than 0.10 second and appear normal.

Amplitude

The amplitude of the waves in the abnormal QRS complex varies from 1 to 2 mm to 20 mm or more.

Shape

Varies widely in shape, from one that appears quite normal—narrow and sharply pointed (as in incomplete bundle-branch block)—to one that is wide and bizarre, slurred, and notched (as in complete bundle-branch block and ventricular rhythms). In ventricular preexcitation, the QRS complex is wider than normal at the base because of an initial slurring or bulging of the upstroke of the R wave (or of the downstroke of the S wave, as the case may be) known as the *delta wave.*

Sequence

The abnormal QRS may or may not follow a P wave but will be preceded by a T wave.

SIGNIFICANCE

Indicates that abnormal depolarization of the ventricles has occurred because of one of the following:

- A block in the progression of the electrical impulse from the bundle of His to the Purkinje network through the right or left bundle branch (bundle-branch block and aberrant ventricular conduction)
- The progression of the electrical impulse from the atria to the ventricles through an abnormal accessory conduction pathway (ventricular preexcitation)
- The origination of the electrical impulse responsible for the ventricular depolarization in a ventricular ectopic or escape pacemaker
- The depolarization of the ventricles initiated by an implanted cardiac pacemaker

T WAVE

T wave

A T wave represents ventricular repolarization. T waves are characterized as normal or abnormal.

Normal T Wave

CHARACTERISTICS

Origin	Epicardial surface of the ventricles
Physiology	Repolarization of ventricles
Onset/End	Deviation from ST segment/TP segment
Direction	Positive
Duration	0.10–0.25 sec
Amplitude	Less than 5 mm
Shape	Bluntly rounded and asymmetrical
Sequence	Always follows a QRS

Origin

Epicardial surface of the ventricles.

Relationship to Cardiac Anatomy and Physiology

A normal T wave (Fig. 3.6) represents normal repolarization of the ventricles. Normal repolarization begins at the epicardial surface of the ventricles and progresses inwardly through the ventricular walls to the endocardial surface. The T wave occurs during the last part of ventricular systole.