Abstract

By the end of this section, you will be able to:

- Describe the structure of cardiac muscle
- Identify and describe the components of the conducting system that distributes electrical impulses through the heart
- Compare the effect of ion movement on membrane potential of cardiac conductive and contractile cells
- Relate characteristics of an electrocardiogram to events in the cardiac cycle
- Identify blocks that can interrupt the cardiac cycle

Recall that cardiac muscle shares a few characteristics with both skeletal muscle and smooth muscle, but it has some unique properties of its own. Not the least of these exceptional properties is its ability to initiate an electrical potential at a fixed rate that spreads rapidly from cell to cell to trigger the contractile mechanism. This property is known as autorhythmicity. Neither smooth nor skeletal muscle can do this. Even though cardiac muscle has autorhythmicity, heart rate is modulated by the endocrine and nervous systems.

There are two major types of cardiac muscle cells: myocardial contractile cells and myocardial conducting cells. The **myocardial contractile cells** constitute the bulk (99 percent) of the cells in the atria and ventricles. Contractile cells conduct impulses and are responsible for contractions that pump blood through the body. The **myocardial conducting cells** (1 percent of the cells) form the conduction system of the heart. Except for Purkinje cells, they are generally much smaller than the contractile cells and have few of the myofibrils or filaments needed for contraction. Their function is similar in many respects to neurons, although they are specialized muscle cells. Myocardial conduction cells initiate and propagate the action potential (the electrical impulse) that travels throughout the heart and triggers the contractions that propel the blood.
1 Structure of Cardiac Muscle

Compared to the giant cylinders of skeletal muscle, cardiac muscle cells, or cardiomyocytes, are considerably shorter with much smaller diameters. Cardiac muscle also demonstrates striations, the alternating pattern of dark A bands and light I bands attributed to the precise arrangement of the myofilaments and fibrils that are organized in sarcomeres along the length of the cell (Figure 1 (Cardiac Muscle )a). These contractile elements are virtually identical to skeletal muscle. T (transverse) tubules penetrate from the surface plasma membrane, the sarcolemma, to the interior of the cell, allowing the electrical impulse to reach the interior. The T tubules are only found at the Z discs, whereas in skeletal muscle, they are found at the junction of the A and I bands. Therefore, there are one-half as many T tubules in cardiac muscle as in skeletal muscle. In addition, the sarcoplasmic reticulum stores few calcium ions, so most of the calcium ions must come from outside the cells. The result is a slower onset of contraction. Mitochondria are plentiful, providing energy for the contractions of the heart. Typically, cardiomyocytes have a single, central nucleus, but two or more nuclei may be found in some cells.

Cardiac muscle cells branch freely. A junction between two adjoining cells is marked by a critical structure called an intercalated disc, which helps support the synchronized contraction of the muscle (Figure 1 (Cardiac Muscle )b). The sarcolemmas from adjacent cells bind together at the intercalated discs. They consist of desmosomes, specialized linking proteoglycans, tight junctions, and large numbers of gap junctions that allow the passage of ions between the cells and help to synchronize the contraction (Figure 1 (Cardiac Muscle )c). Intercellular connective tissue also helps to bind the cells together. The importance of strongly binding these cells together is necessitated by the forces exerted by contraction.
Cardiac muscle cells have myofibrils composed of myofilaments arranged in sarcomeres, T tubules to transmit the impulse from the sarcolemma to the interior of the cell, numerous mitochondria for energy, and intercalated discs that are found at the junction of different cardiac muscle cells. (b) A photomicrograph of cardiac muscle cells shows the nuclei and intercalated discs. (c) An intercalated disc connects cardiac muscle cells and consists of desmosomes and gap junctions. LM × 1600. (Micrograph provided by the Regents of the University of Michigan Medical School ©2012)

Cardiac muscle undergoes aerobic respiration patterns, primarily metabolizing lipids and carbohydrates.
Myoglobin, lipids, and glycogen are all stored within the cytoplasm. Cardiac muscle cells undergo twitch-type contractions with long refractory periods followed by brief relaxation periods. The relaxation is essential so the heart can fill with blood for the next cycle. The refractory period is very long to prevent the possibility of tetany, a condition in which muscle remains involuntarily contracted. In the heart, tetany is not compatible with life, since it would prevent the heart from pumping blood.

**Repair and Replacement**

Damaged cardiac muscle cells have extremely limited abilities to repair themselves or to replace dead cells via mitosis. Recent evidence indicates that at least some stem cells remain within the heart that continue to divide and at least potentially replace these dead cells. However, newly formed or repaired cells are rarely as functional as the original cells, and cardiac function is reduced. In the event of a heart attack or MI, dead cells are often replaced by patches of scar tissue. Autopsies performed on individuals who had successfully received heart transplants show some proliferation of original cells. If researchers can unlock the mechanism that generates new cells and restore full mitotic capabilities to heart muscle, the prognosis for heart attack survivors will be greatly enhanced. To date, myocardial cells produced within the patient (in situ) by cardiac stem cells seem to be nonfunctional, although those grown in Petri dishes (in vitro) do beat. Perhaps soon this mystery will be solved, and new advances in treatment will be commonplace.

2 Conduction System of the Heart

If embryonic heart cells are separated into a Petri dish and kept alive, each is capable of generating its own electrical impulse followed by contraction. When two independently beating embryonic cardiac muscle cells are placed together, the cell with the higher inherent rate sets the pace, and the impulse spreads from the faster to the slower cell to trigger a contraction. As more cells are joined together, the fastest cell continues to assume control of the rate. A fully developed adult heart maintains the capability of generating its own electrical impulse, triggered by the fastest cells, as part of the cardiac conduction system. The components of the cardiac conduction system include the sinoatrial node, the atrioventricular node, the atrioventricular bundle, the atrioventricular bundle branches, and the Purkinje cells (Figure 2 (Conduction System of the Heart)).
2.1 Sinoatrial (SA) Node

Normal cardiac rhythm is established by the **sinoatrial (SA) node**, a specialized clump of myocardial conducting cells located in the superior and posterior walls of the right atrium in close proximity to the orifice of the superior vena cava. The SA node has the highest inherent rate of depolarization and is known as the **pacemaker** of the heart. It initiates the **sinus rhythm**, or normal electrical pattern followed by contraction of the heart.

This impulse spreads from its initiation in the SA node throughout the atria through specialized **internodal pathways**, to the atrial myocardial contractile cells and the atrioventricular node. The internodal pathways consist of three bands (anterior, middle, and posterior) that lead directly from the SA node to the next node in the conduction system, the atrioventricular node (see Figure 2 (Conduction System of the Heart)). The impulse takes approximately 50 ms (milliseconds) to travel between these two nodes. The relative importance of this pathway has been debated since the impulse would reach the atrioventricular node simply following the cell-by-cell pathway through the contractile cells of the myocardium in the atria. In addition, there is a specialized pathway called **Bachmann’s bundle** or the **interatrial band** that conducts the im-
pulse directly from the right atrium to the left atrium. Regardless of the pathway, as the impulse reaches the atrioventricular septum, the connective tissue of the cardiac skeleton prevents the impulse from spreading into the myocardial cells in the ventricles except at the atrioventricular node. Figure 3 (Cardiac Conduction) illustrates the initiation of the impulse in the SA node that then spreads the impulse throughout the atria to the atrioventricular node.
Cardiac Conduction

Figure 3: (1) The sinoatrial (SA) node and the remainder of the conduction system are at rest. (2) The SA node initiates the action potential, which sweeps across the atria. (3) After reaching the atrioventricular node, there is a delay of approximately 100 ms that allows the atria to complete pumping blood before the impulse is transmitted to the atrioventricular bundle. (4) Following the delay, the impulse travels through the atrioventricular bundle and bundle branches to the Purkinje fibers, and also reaches the right papillary muscle via the moderator band. (5) The impulse spreads to the contractile fibers of the ventricle. (6) Ventricular contraction begins.
The electrical event, the wave of depolarization, is the trigger for muscular contraction. The wave of depolarization begins in the right atrium, and the impulse spreads across the superior portions of both atria and then down through the contractile cells. The contractile cells then begin contraction from the superior to the inferior portions of the atria, efficiently pumping blood into the ventricles.

2.2 Atrioventricular (AV) Node

The atrioventricular (AV) node is a second clump of specialized myocardial conductive cells, located in the inferior portion of the right atrium within the atrioventricular septum. The septum prevents the impulse from spreading directly to the ventricles without passing through the AV node. There is a critical pause before the AV node depolarizes and transmits the impulse to the atrioventricular bundle (see Figure 3 (Cardiac Conduction), step 3). This delay in transmission is partially attributable to the small diameter of the cells of the node, which slow the impulse. Also, conduction between nodal cells is less efficient than between conducting cells. These factors mean that it takes the impulse approximately 100 ms to pass through the node. This pause is critical to heart function, as it allows the atrial cardiomyocytes to complete their contraction that pumps blood into the ventricles before the impulse is transmitted to the cells of the ventricles itself. With extreme stimulation by the SA node, the AV node can transmit impulses maximally at 220 per minute. This establishes the typical maximum heart rate in a healthy young individual. Damaged hearts or those stimulated by drugs can contract at higher rates, but at these rates, the heart can no longer effectively pump blood.

2.3 Atrioventricular Bundle (Bundle of His), Bundle Branches, and Purkinje Fibers

Arising from the AV node, the atrioventricular bundle, or bundle of His, proceeds through the interventricular septum before dividing into two atrioventricular bundle branches, commonly called the left and right bundle branches. The left bundle branch has two fascicles. The left bundle branch supplies the left ventricle, and the right bundle branch the right ventricle. Since the left ventricle is much larger than the right, the left bundle branch is also considerably larger than the right. Portions of the right bundle branch are found in the moderator band and supply the right papillary muscles. Because of this connection, each papillary muscle receives the impulse at approximately the same time, so they begin to contract simultaneously just prior to the remainder of the myocardial contractile cells of the ventricles. This is believed to allow tension to develop on the chordae tendineae prior to right ventricular contraction. There is no corresponding moderator band on the left. Both bundle branches descend and reach the apex of the heart where they connect with the Purkinje fibers (see Figure 3 (Cardiac Conduction), step 4). This passage takes approximately 25 ms.

The Purkinje fibers are additional myocardial conductive fibers that spread the impulse to the myocardial contractile cells in the ventricles. They extend throughout the myocardium from the apex of the heart toward the atrioventricular septum and the base of the heart. The Purkinje fibers have a fast inherent conduction rate, and the electrical impulse reaches all of the ventricular muscle cells in about 75 ms (see Figure 3 (Cardiac Conduction), step 5). Since the electrical stimulus begins at the apex, the contraction also begins at the apex and travels toward the base of the heart, similar to squeezing a tube of toothpaste from the bottom. This allows the blood to be pumped out of the ventricles and into the aorta and pulmonary trunk. The total time elapsed from the initiation of the impulse in the SA node until depolarization of the ventricles is approximately 225 ms.

3 Electrocardiogram

By careful placement of surface electrodes on the body, it is possible to record the complex, compound electrical signal of the heart. This tracing of the electrical signal is the electrocardiogram (ECG), also commonly abbreviated EKG (K coming cardiology, from the German term for cardiology). Careful analysis of the ECG reveals a detailed picture of both normal and abnormal heart function, and is an indispensable clinical diagnostic tool. The standard electrocardiograph (the instrument that generates an ECG) uses 3,
5, or 12 leads. The greater the number of leads an electrocardiograph uses, the more information the ECG provides. The term “lead” may be used to refer to the cable from the electrode to the electrical recorder, but it typically describes the voltage difference between two of the electrodes. The 12-lead electrocardiograph uses 10 electrodes placed in standard locations on the patient’s skin (Figure 4 (Standard Placement of ECG Leads)). In continuous ambulatory electrocardiographs, the patient wears a small, portable, battery-operated device known as a Holter monitor, or simply a Holter, that continuously monitors heart electrical activity, typically for a period of 24 hours during the patient’s normal routine.
Standard Placement of ECG Leads

Figure 4: In a 12-lead ECG, six electrodes are placed on the chest, and four electrodes are placed on the limbs.
A normal ECG tracing is presented in Figure 5 (Electrocardiogram). Each component, segment, and interval is labeled and corresponds to important electrical events, demonstrating the relationship between these events and contraction in the heart.

There are five prominent points on the ECG: the P wave, the QRS complex, and the T wave. The small **P wave** represents the depolarization of the atria. The atria begin contracting approximately 25 ms after the start of the P wave. The large **QRS complex** represents the depolarization of the ventricles, which requires a much stronger electrical signal because of the larger size of the ventricular cardiac muscle. The ventricles begin to contract as the QRS reaches the peak of the R wave. Lastly, the **T wave** represents the repolarization of the ventricles. The repolarization of the atria occurs during the QRS complex, which masks it on an ECG.

The major segments and intervals of an ECG tracing are indicated in Figure 5 (Electrocardiogram). Segments are defined as the regions between two waves. Intervals include one segment plus one or more waves. For example, the PR segment begins at the end of the P wave and ends at the beginning of the QRS complex. The PR interval starts at the beginning of the P wave and ends with the beginning of the QRS complex. The PR interval is more clinically relevant, as it measures the duration from the beginning of atrial depolarization (the P wave) to the initiation of the QRS complex. Since the Q wave may be difficult to view in some tracings, the measurement is often extended to the R that is more easily visible. Should there be a delay in passage of the impulse from the SA node to the AV node, it would be visible in the PR interval. Figure 6 (ECG Tracing Correlated to the Cardiac Cycle) correlates events of heart contraction to the corresponding segments and intervals of an ECG.
Visit this site\(^1\) for a more detailed analysis of ECGs.

\(^1\)http://openstaxcollege.org/I/ECG
**Figure 5**: A normal tracing shows the P wave, QRS complex, and T wave. Also indicated are the PR, QT, QRS, and ST intervals, plus the P-R and S-T segments.
ECG Abnormalities

Occasional o ccurances are generally transitory and nonlife threatening, but if the condition becomes chronic, it may lead to either an arrhythmia, a deviation from the normal pattern of impulse conduction and contraction, or to fibrillation, an uncoordinated beating of the heart.

While interpretation of an ECG is possible and extremely valuable after some training, a full understanding of the complexities and intricacies generally requires several years of experience. In general, the size of the electrical variations, the duration of the events, and detailed vector analysis
provide the most comprehensive picture of cardiac function. For example, an amplified P wave may indicate enlargement of the atria, an enlarged Q wave may indicate a MI, and an enlarged suppressed or inverted Q wave often indicates enlarged ventricles. T waves often appear flatter when insufficient oxygen is being delivered to the myocardium. An elevation of the ST segment above baseline is often seen in patients with an acute MI, and may appear depressed below the baseline when hypoxia is occurring.

As useful as analyzing these electrical recordings may be, there are limitations. For example, not all areas suffering a MI may be obvious on the ECG. Additionally, it will not reveal the effectiveness of the pumping, which requires further testing, such as an ultrasound test called an echocardiogram or nuclear medicine imaging. It is also possible for there to be pulseless electrical activity, which will show up on an ECG tracing, although there is no corresponding pumping action. Common abnormalities that may be detected by the ECGs are shown in Figure 7 (Common ECG Abnormalities ).
Common ECG Abnormalities

(a) Second-degree (partial) block

(b) Atrial fibrillation

(c) Ventricular tachycardia

(d) Ventricular fibrillation

(e) Third-degree block

Note how half of the P waves are not followed by the QRS complex and T waves while the other half are. **Question:** What would you expect to happen to heart rate (pulse)?

Note the abnormal electrical pattern prior to the QRS complexes. Also note how the frequency between the QRS complexes has increased. **Question:** What would you expect to happen to heart rate (pulse)?

Note the unusual shape of the QRS complex, focusing on the “S” component. **Question:** What would you expect to happen to heart rate (pulse)?

Note the total lack of normal electrical activity. **Question:** What would you expect to happen to heart rate (pulse)?

Note that in a third-degree block some of the impulses initiated by the SA node do not reach the AV node while others do. Also note that the P waves are not followed by the QRS complex. **Question:** What would you expect to happen to heart rate (pulse)?

**Figure 7:** (a) In a second-degree or partial block, one-half of the P waves are not followed by the QRS complex and T waves while the other half are. (b) In atrial fibrillation, the electrical pattern is abnormal prior to the QRS complex, and the frequency between the QRS complexes has increased. (c) In ventricular tachycardia, the shape of the QRS complex is abnormal. (d) In ventricular fibrillation, there is no normal electrical activity. (e) In a third-degree block, there is no correlation between atrial activity (the P wave) and ventricular activity (the QRS complex).
Chapter Review

The heart is regulated by both neural and endocrine control, yet it is capable of initiating its own action potential followed by muscular contraction. The conductive cells within the heart establish the heart rate and transmit it through the myocardium. The contractile cells contract and propel the blood. The normal path of transmission for the conductive cells is the sinoatrial (SA) node, internodal pathways, atrioventricular (AV) node, atrioventricular (AV) bundle of His, bundle branches, and Purkinje fibers. Recognizable points on the ECG include the P wave that corresponds to atrial depolarization, the QRS complex that corresponds to ventricular depolarization, and the T wave that corresponds to ventricular repolarization.

Visit this site for a more complete library of abnormal ECGs.

http://op openstaxcollege.org/l/abnormalECG
5 Review Questions

Exercise 1  (Solution on p. 19.)
Which of the following is unique to cardiac muscle cells?

a. Only cardiac muscle contains a sarcoplasmic reticulum.
b. Only cardiac muscle has gap junctions.
c. Only cardiac muscle is capable of autorhythmicity
d. Only cardiac muscle has a high concentration of mitochondria.

Exercise 2  (Solution on p. 19.)
Which portion of the ECG corresponds to repolarization of the atria?

a. P wave  
b. QRS complex  
c. T wave  
d. none of the above; atrial repolarization is masked by ventricular depolarization

Exercise 3  (Solution on p. 19.)
Which component of the heart conduction system would have the slowest rate of firing?

a. atrioventricular node  
b. atrioventricular bundle  
c. bundle branches  
d. Purkinje fibers

6 Critical Thinking Questions

Exercise 4  (Solution on p. 19.)
How does the delay of the impulse at the atrioventricular node contribute to cardiac function?

Exercise 5  (Solution on p. 19.)
How do gap junctions and intercalated disks aid contraction of the heart?
Solutions to Exercises in this Module

to Exercise (p. 18)
C
to Exercise (p. 18)
D
to Exercise (p. 18)
D
to Exercise (p. 18)

It ensures sufficient time for the atrial muscle to contract and pump blood into the ventricles prior to the impulse being conducted into the lower chambers.

to Exercise (p. 18)

Gap junctions within the intercalated disks allow impulses to spread from one cardiac muscle cell to another, allowing sodium, potassium, and calcium ions to flow between adjacent cells, propagating the action potential, and ensuring coordinated contractions.

Glossary

**Definition 7: artificial pacemaker**
medical device that transmits electrical signals to the heart to ensure that it contracts and pumps blood to the body

**Definition 7: atrioventricular bundle**
(also, bundle of His) group of specialized myocardial conductile cells that transmit the impulse from the AV node through the interventricular septum; form the left and right atrioventricular bundle branches

**Definition 7: atrioventricular bundle branches**
(also, left or right bundle branches) specialized myocardial conductile cells that arise from the bifurcation of the atrioventricular bundle and pass through the interventricular septum; lead to the Purkinje fibers and also to the right papillary muscle via the moderator band

**Definition 7: atrioventricular (AV) node**
clump of myocardial cells located in the inferior portion of the right atrium within the atrioventricular septum; receives the impulse from the SA node, pauses, and then transmits it into specialized conducting cells within the interventricular septum

**Definition 7: autorhythmicity**
ability of cardiac muscle to initiate its own electrical impulse that triggers the mechanical contraction that pumps blood at a fixed pace without nervous or endocrine control

**Definition 7: Bachmann’s bundle**
(also, interatrial band) group of specialized conducting cells that transmit the impulse directly from the SA node in the right atrium to the left atrium

**Definition 7: bundle of His**
(also, atrioventricular bundle) group of specialized myocardial conductile cells that transmit the impulse from the AV node through the interventricular septum; form the left and right atrioventricular bundle branches

**Definition 7: electrocardiogram (ECG)**
surface recording of the electrical activity of the heart that can be used for diagnosis of irregular heart function; also abbreviated as EKG

**Definition 7: heart block**
interruption in the normal conduction pathway

http://cnx.org/content/m47785/1.1/
Definition 7: interatrial band
(also, Bachmann’s bundle) group of specialized conducting cells that transmit the impulse directly from the SA node in the right atrium to the left atrium

Definition 7: intercalated disc
physical junction between adjacent cardiac muscle cells; consisting of desmosomes, specialized linking proteoglycans, and gap junctions that allow passage of ions between the two cells

Definition 7: internodal pathways
specialized conductile cells within the atria that transmit the impulse from the SA node throughout the myocardial cells of the atrium and to the AV node

Definition 7: myocardial conducting cells
specialized cells that transmit electrical impulses throughout the heart and trigger contraction by the myocardial contractile cells

Definition 7: myocardial contractile cells
bulk of the cardiac muscle cells in the atria and ventricles that conduct impulses and contract to propel blood

Definition 7: P wave
component of the electrocardiogram that represents the depolarization of the atria

Definition 7: pacemaker
cluster of specialized myocardial cells known as the SA node that initiates the sinus rhythm

Definition 7: prepotential depolarization
(also, spontaneous depolarization) mechanism that accounts for the autorhythmic property of cardiac muscle; the membrane potential increases as sodium ions diffuse through the always-open sodium ion channels and causes the electrical potential to rise

Definition 7: Purkinje fibers
specialized myocardial conduction fibers that arise from the bundle branches and spread the impulse to the myocardial contraction fibers of the ventricles

Definition 7: QRS complex
component of the electrocardiogram that represents the depolarization of the ventricles and includes, as a component, the repolarization of the atria

Definition 7: sinoatrial (SA) node
known as the pacemaker, a specialized clump of myocardial conducting cells located in the superior portion of the right atrium that has the highest inherent rate of depolarization that then spreads throughout the heart

Definition 7: sinus rhythm
normal contractile pattern of the heart

Definition 7: spontaneous depolarization
(also, prepotential depolarization) the mechanism that accounts for the autorhythmic property of cardiac muscle; the membrane potential increases as sodium ions diffuse through the always-open sodium ion channels and causes the electrical potential to rise

Definition 7: T wave
component of the electrocardiogram that represents the repolarization of the ventricles