Rhythmic Excitation of the Heart

The heart is endowed with a specialized system (1) for generating rhythmical impulses to cause rhythmical contraction of the heart muscle and (2) for conducting these impulses rapidly throughout the heart. When this system functions normally, the atria contract about one sixth of a second ahead of ventricular contraction, which allows extra filling of the ventricles before they pump the blood through the lungs and peripheral circulation. Another special importance of the system is that it allows all portions of the ventricles to contract almost simultaneously, which is essential for effective pressure generation in the ventricular chambers.

Unfortunately, though, this rhythmical and conduction system of the heart is very susceptible to damage by heart disease, especially by ischemia of the heart tissues resulting from poor coronary blood flow. The consequence is often a very bizarre heart rhythm, or abnormal sequence of contraction of the heart chambers, and the pumping effectiveness of the heart is often affected severely, even to the extent of causing death.

The Sinus Node

The sinus node is a small, flattened, ellipsoid strip of specialized muscle approximately 3 millimeters wide, 15 millimeters long, and 1 millimeter thick; it is located in the superior lateral wall of the right atrium immediately below and lateral to the opening of the superior vena cava. The fibers of this node have almost no contractile filaments and are each 3 to 5 micrometers in diameter, in contrast to a diameter of 10 to 15 micrometers for the surrounding atrial muscle fibers. However, the sinus fibers are continuous with the atrial fibers, so that any action potential that begins in the sinus node spreads immediately into the atria.

Automatic Rhythmicity of the Sinus Fibers

Many cardiac fibers have the capability of self-excitation, a process that can cause automatic rhythmical contraction. This is especially true of the fibers of the heart's specialized conducting system; the portion of this system that displays self-excitation to the greatest extent is the fibers of the sinus node. For this reason, the sinus node ordinarily controls the rate of beat of the entire heart, as is discussed in detail later in this chapter. First, however, let us describe this automatic rhythmicity.

Mechanism of Sinus Nodal Rhythmicity. Figure 10-1 illustrates action potentials recorded from a sinus nodal fiber for three heartbeats and, by comparison, a single ventricular muscle fiber action potential, shown to the right. Note that the potential of the sinus nodal fiber between discharges has a negativity of only -55 to -60 millivolts in comparison with -85 to -90 millivolts for the ventricular fiber. The cause of this reduced negativity is that the cell membranes of the sinus fibers are naturally leaky to sodium ions.

Before attempting to explain the rhythmicity of the sinus nodal fibers, first recall from the discussions of Chapters 5 and 9 that in cardiac muscle three different types of membrane ion channels play important roles in causing the voltage changes of the action potential. These are (1) the fast sodium channels, (2) the
slow calcium-sodium channels, and (3) the potassium channels. The opening of the fast sodium channels for a few 10,000ths of a second is responsible for the very rapid spikelike onset of the action potential observed in ventricular muscle because of rapid influx of positive sodium ions to the interior of the fiber. Then the plateau of the ventricular action potential is caused primarily by slower opening of the slow calcium-sodium channels, which lasts for a few tenths of a second. Finally, increased opening of the potassium channels and diffusion of large amounts of positive potassium ions out of the fiber return the membrane potential to its resting level.

But there is a difference in the function of these channels in the sinus nodal fiber because of the much lesser negativity of the “resting” potential — only — 55 millivolts. At this level of negativity, the fast sodium channels have mainly become “inactivated,” which means that they have become blocked. The cause of this is that any time the membrane potential remains less negative than about — 60 millivolts for more than a few milliseconds, the inactivation gates on the inside of the cell membrane that close these channels become closed and remain so. Therefore, only the slow calcium-sodium channels can open (that is, can become “activated”) and thereby cause the action potential. Therefore, the action potential is slower to develop than that of the ventricular muscle and also recovers with a slow decrement of the potential rather than the abrupt recovery that occurs for the ventricular fiber.

**Self-Excitation of Sinus Nodal Fibers.** Sodium ions naturally tend to leak to the interior of the sinus nodal fibers through multiple membrane channels, and this influx of positive charges also causes a rising membrane potential. Thus, as illustrated in Figure 10-2, the “resting” potential gradually rises between each two heartbeats. When it reaches a threshold voltage of about — 40 millivolts, the calcium-sodium channels become activated, leading to very rapid entry of both calcium and sodium ions, thus causing the action potential. Therefore, basically, the inherent leakiness of the sinus nodal fibers to sodium ions causes their self-excitation.

Why does this leakiness to sodium ions not cause the sinus nodal fibers to remain depolarized all the time? The answer is that two events occur during the course of the action potential. First, the calcium-sodium channels become inactivated (that is, they close) within about 100 to 150 milliseconds after opening, and second, at about the same time greatly increased numbers of potassium channels open. Therefore, now the influx of calcium and sodium ions through the calcium-sodium channels ceases simultaneously, while large quantities of positive potassium ions diffuse out of the fiber, thus terminating the action potential. Furthermore, the potassium channels remain open for another few tenths of a second, carrying a great excess of positive potassium charges out of the cell, which temporarily causes considerable excess negativity inside the fiber; this is called hyperpolarization. This hyperpolarization initially carries the “resting” membrane potential down to about — 55 to — 60 millivolts at the termination of the action potential.

Last, we must explain why the state of hyperpolarization also is not maintained forever. The reason is that during the next few tenths of a second after the action potential is over, progressively more and more of the potassium channels begin to close. Now the inward-leaking sodium ions once again overbalance the outward flux of potassium ions, which causes the “resting” potential to drift upward, finally reaching the threshold level for discharge at a potential of about — 40 millivolts. Then the entire process begins again: self-excitation, recovery from the action potential, hyperpolarization after the action potential is over, upward drift of the “resting” potential, then re-excitation still again to elicit another cycle. This process continues indefinitely throughout the life of the person.
INTERNODAL PATHWAYS AND TRANSMISSION OF THE CARDIAC IMPULSE THROUGH THE ATRIA

The ends of the sinus nodal fibers fuse with the surrounding atrial muscle fibers, and action potentials originating in the sinus node travel outward into these fibers. In this way, the action potential spreads through the entire atrial muscle mass and eventually also to the A-V node. The velocity of conduction in the atrial muscle is approximately 0.3 m/sec. However, conduction is somewhat more rapid in several small bundles of atrial muscle fibers. One of these, called the anterior interatrial band, passes through the anterior walls of the atria to the left atrium and conducts the cardiac impulse at a velocity of about 1 m/sec. In addition, three other small bundles curve through the atrial walls and terminate in the A-V node, also conducting the cardiac impulse at this rapid velocity. These three small bundles, illustrated in Figure 10-1, are called, respectively, the anterior, middle, and posterior internodal pathways. The cause of the more rapid velocity of conduction in these bundles is the presence of a number of specialized conduction fibers mixed with the atrial muscle. These fibers are similar to the very rapidly conducting Purkinje fibers of the ventricles, which are discussed subsequently.

THE A-V NODE AND DELAY IN IMPULSE CONDUCTION

Fortunately, the conductive system is organized so that the cardiac impulse will not travel from the atria into the ventricles too rapidly; this allows time for the atria to empty their contents into the ventricles before ventricular contraction begins. It is primarily the A-V node and its associated conductive fibers that delay this transmission of the cardiac impulse from the atria into the ventricles.

The A-V node is located in the posterior septal wall of the right atrium immediately behind the tricuspid valve and adjacent to the opening of the coronary sinus, as illustrated in Figure 10-1. Figure 10-3 shows diagrammatically the different parts of this node and its connections with the atrial internodal pathway fibers and the A-V bundle. The figure also shows the approximate intervals of time in fractions of a second between the genesis of the cardiac impulse in the sinus node and its appearance at different points in the A-V nodal system. Note that the impulse, after traveling through the internodal pathway, reaches the A-V node approximately 0.03 second after its origin in the sinus node. Then there is a further delay of 0.09 second in the A-V node itself before the impulse enters the penetrating portion of the A-V bundle. A final delay of another 0.04 second occurs mainly in this penetrating A-V bundle, which is composed of multiple small fascicles passing through the fibrous tissue separating the atria from the ventricles.

Thus, the total delay in the A-V nodal and A-V bundle system is approximately 0.13 second. About a quarter of this time lapse occurs in the transitional fibers, which are very small fibers that connect the fibers of the atrial internodal pathways with the A-V node (see Fig. 10-3). The velocity of conduction in these fibers is as little as 0.02 to 0.05 m/sec (about 1/4 that in normal cardiac muscle), which greatly delays entrance of the impulse into the A-V node. After entering the node proper, the velocity of conduction in the nodal fibers is still quite low, only 0.05 m/sec, about one-eighth the conduction velocity in normal cardiac muscle. This low velocity of conduction is also approximately true for the penetrating portion of the A-V bundle.

Cause of the Slow Conduction. The cause of the extremely slow conduction in the transitional as well as the nodal and penetrating A-V bundle fibers is partly that their sizes are considerably smaller than the sizes of the normal atrial muscle fibers. However, most of the slow conduction is probably caused by two entirely different factors. First, all these fibers have resting membrane potentials that are much less negative than the normal resting potential of other cardiac muscle. Second, very few gap junctions connect the successive fibers in the pathway, so that there is great resistance to the conduction of excitatory ions from one fiber to the next. Thus, with both low voltage to drive the ions and great resistance to the movement of the ions, it is easy to see why each succeeding fiber is slow to be excited.

TRANSMISSION IN THE PURKINJE SYSTEM

The Purkinje fibers lead from the A-V node through the A-V bundle into the ventricles. Except for the
initial portion of these fibers where they penetrate the atrioventricular fibrous barrier, they have functional characteristics quite the opposite of those of the A-V nodal fibers; they are very large fibers, even larger than the normal ventricular muscle fibers, and they transmit action potentials at a velocity of 1.5 to 4.0 m/sec, a velocity about 6 times that in the usual cardiac muscle and 150 times that in some transitional fibers. This allows almost immediate transmission of the cardiac impulse throughout the entire ventricular system.

The very rapid transmission of action potentials by Purkinje fibers is believed to be caused by increased permeability of the gap junctions at the intercalated discs between the successive cardiac cells that make up the Purkinje fibers. At these discs, ions are transmitted easily from one cell to the next, thus enhancing the velocity of transmission. The Purkinje fibers also have very few myofibrils, which means that they barely contract during the course of impulse transmission.

**One-Way Conduction Through the A-V Bundle.** A special characteristic of the A-V bundle is the inability except in abnormal states of action potentials to travel backward in the bundle from the ventricles to the atria. This prevents re-entry of cardiac impulses by this route from the ventricles into the atria, allowing only forward conduction from the atria to the ventricles.

Furthermore, it should be recalled that the atrial muscle is separated from the ventricular muscle by a continuous fibrous barrier, a portion of which is illustrated in Figure 10–3. This barrier normally acts as an insulator to prevent passage of the cardiac impulse between the atria and the ventricles through any other route besides forward conduction through the A-V bundle itself. (However, in rare instances an abnormal muscle bridge does penetrate through the fibrous barrier elsewhere besides at the A-V bundle. Under such conditions, the cardiac impulse can then re-enter the atria from the ventricles and cause serious cardiac arrhythmia.)

**Distribution of the Purkinje Fibers in the Ventricles.** After penetrating through the atrioventricular fibrous tissue, the distal portion of the A-V bundle passes downward in the ventricular septum for 5 to 15 millimeters toward the apex of the heart, as shown in Figure 10–1 and 10–3. Then the bundle divides into the left and right bundle branches that lie beneath the endocardium of the two respective sides of the septum. Each branch spreads downward to the apex of the ventricle, dividing into smaller branches that course around each ventricular chamber and back toward the base of the heart. The terminal Purkinje fibers penetrate about one third of the way into the muscle mass and then become continuous with the cardiac muscle fibers.

From the time that the cardiac impulse first enters the bundle branches until it reaches the terminations of the Purkinje fibers, the total time that elapses averages only 0.03 second; therefore, once the cardiac impulse enters the Purkinje system, it spreads almost immediately to the entire endocardial surface of the ventricular muscle.

**TRANSMISSION OF THE CARDIAC IMPULSE IN THE VENTRICULAR MUSCLE**

Once the impulse has reached the ends of the Purkinje fibers, it is then transmitted through the ventricular muscle mass by the ventricular muscle fibers themselves. The velocity of transmission is now only 0.3 to 0.5 m/sec, one sixth that in the Purkinje fibers.

The cardiac muscle wraps around the heart in a double spiral with fibrous septa between the spiraling layers; therefore, the cardiac impulse does not necessarily travel directly outward toward the surface of the heart but instead angles toward the surface along the directions of the spirals. Because of this, transmission from the endocardial surface to the epicardial surface of the ventricle requires as much as another 0.03 second, approximately equal to the time required for transmission through the entire Purkinje system. Thus, the total time for transmission of the cardiac impulse from the initial bundle branches to the last of the ventricular muscle fibers in the normal heart is about 0.06 second.

**SUMMARY OF THE SPREAD OF THE CARDIAC IMPULSE THROUGH THE HEART**

Figure 10–4 illustrates in summary form the transmission of the cardiac impulse through the human heart. The numbers on the figure represent the intervals of time in hundredths of a second that lapse be-
between the origin of the cardiac impulse in the sinus node and its appearance at each respective point in the heart. Note that the impulse spreads at moderate velocity through the atria but is delayed more than 0.1 second in the A-V nodal region before appearing in the ventricular septal A-V bundle. Once it has entered this bundle, it spreads rapidly through the Purkinje fibers to the entire endocardial surfaces of the ventricles. Then the impulse once again spreads slowly through the ventricular muscle to the epicardial surfaces.

It is extremely important that the reader learn in detail the course of the cardiac impulse through the heart and the times of its appearance in each separate part of the heart, for a quantitative knowledge of this process is essential to the understanding of electrocardiography, which is discussed in the following three chapters.

CONTROL OF EXCITATION AND CONDUCTION IN THE HEART

THE SINUS NODE AS THE PACEMAKER OF THE HEART

In the previous discussion of the genesis and transmission of the cardiac impulse through the heart, we have noted that the impulse normally arises in the sinus node. However, this often is not the case under abnormal conditions, for other parts of the heart can exhibit rhythmic contraction in the same way that the sinus nodal fibers can; this is particularly true of the A-V nodal and Purkinje fibers.

The A-V nodal fibers, when not stimulated from some outside source, discharge at an intrinsic rhythmic rate of 40 to 60 times per minute, and the Purkinje fibers discharge at a rate of somewhere between 15 and 40 times per minute. These rates are in contrast to the normal rate of the sinus node of 70 to 80 times per minute.

Therefore, the question that we must ask is: Why does the sinus node control the heart's rhythmicity rather than the A-V node or the Purkinje fibers? The answer to this derives from the fact that the discharge rate of the sinus node is considerably greater than that of either the A-V node or the Purkinje fibers. Each time the sinus node discharges, its impulse is conducted into both the A-V node and the Purkinje fibers, discharging their excitable membranes. Then these tissues as well as the sinus node recover from the action potential and become hyperpolarized. But the sinus node loses its hyperpolarization much more rapidly than does either of the other two and emits a new impulse before either one of them can reach its own threshold for self-excitation. The new impulse again discharges both the A-V node and Purkinje fibers. This process continues on and on, the sinus node always exciting these other potentially self-excitatory tissues before self-excitation can actually occur.

Thus, the sinus node controls the beat of the heart because its rate of rhythmic discharge is greater than that of any other part of the heart. Therefore, the sinus node is the normal pacemaker of the heart.

Abnormal Pacemakers — The Ectopic Pacemaker. Occasionally some other part of the heart develops a rhythmic discharge rate that is more rapid than that of the sinus node. For instance, this often occurs in the A-V node or in the Purkinje fibers. In either of these cases, the pacemaker of the heart shifts from the sinus node to the A-V node or to the excitable Purkinje fibers. Under more rare conditions, a point in the atrial or ventricular muscle develops excessive excitability and becomes the pacemaker.

A pacemaker elsewhere than the sinus node is called an ectopic pacemaker. Obviously, an ectopic pacemaker causes an abnormal sequence of contraction of the different parts of the heart.

Another cause of shift of the pacemaker is blockage of transmission of the impulses from the sinus node to the other parts of the heart, this occurring most frequently at the A-V node or in the penetrating portion of the A-V bundle on the way to the ventricles. When A-V block occurs, the atria continue to beat at the normal rate of rhythm of the sinus node, while a new pacemaker develops in the Purkinje system of the ventricles and drives the ventricular muscle at a new rate somewhere between 15 and 40 beats per minute. However, after a sudden block, the Purkinje system does not begin to emit its rhythmic impulses until 5 to 30 seconds later because up to that point it has been "overdriven" by the rapid sinus impulses and consequently is in a suppressed state. During this 5 to 30 seconds, the ventricles fail to pump any blood, and the person faints after the first 4 to 5 seconds because of lack of blood flow to the brain. This delayed pickup of the heart beat is called Stokes-Adams syndrome. If the period is too long, it can lead to death.

ROLE OF THE PURKINJE SYSTEM IN CAUSING SYNCHRONOUS CONTRACTION OF THE VENTRICULAR MUSCLE

It is clear from the previous description of the Purkinje system that the cardiac impulse arrives at almost all portions of the ventricles within a very narrow span of time, exciting the first ventricular muscle fiber only 0.06 second ahead of excitation of the last ventricular muscle fiber. This causes all portions of the ventricular muscle in both ventricles to begin contracting at almost exactly the same time. Effective pumping by the two ventricular chambers requires this synchronous type of contraction. If the cardiac impulse traveled through the ventricular muscle very slowly, much of the ventricular mass would contract prior to contraction of the remainder, in which case the overall pumping effect would be greatly depressed. Indeed, in some types of cardiac debilities, some of which are discussed in Chapters 12 and 13, slow transmission does occur, and the pumping effectiveness of the ventricles is decreased perhaps as much as 20 to 30 per cent.
CONTROL OF HEART RHYTHMICITY AND CONDUCTION BY THE SYMPATHETIC AND PARASYMPATHETIC NERVES

The heart is supplied with both sympathetic and parasympathetic nerves, as illustrated in Figure 9–11 of the previous chapter. The parasympathetic nerves (the vagi) are distributed mainly to the sinus and A-V nodes, to a lesser extent to the muscle of the two atria, and even less to the ventricular muscle. The sympathetic nerves, on the other hand, are distributed to all parts of the heart, with a strong representation to the ventricular muscle as well as to all the other areas.

Effect of Parasympathetic (Vagal) Stimulation on Cardiac Rhythm and Conduction—Ventricular Escape. Stimulation of the parasympathetic nerves to the heart (the vagi) causes the hormone acetylcholine to be released at the vagal endings. This hormone has two major effects on the heart. First, it decreases the rate of rhythm of the sinus node, and, second, it decreases the excitability of the A-V junctional fibers between the atrial musculature and the A-V node, thereby slowing transmission of the cardiac impulse into the ventricles. Very strong stimulation of the vagi can completely stop the rhythmic contraction of the sinus node or completely block transmission of the cardiac impulse through the A-V junction. In either case, rhythmic impulses are no longer transmitted into the ventricles. The ventricles stop beating for 4 to 10 seconds, but then some point in the Purkinje fibers, usually in the ventricular septal portion of the A-V bundle, develops a rhythm of its own and causes ventricular contraction at a rate of 15 to 40 beats per minute. This phenomenon is called ventricular escape.

Mechanism of the Vagal Effects. The acetylcholine released at the vagal nerve endings greatly increases the permeability of the fiber membranes to potassium, which allows rapid leakage of potassium to the exterior. This causes increased negativity inside the fibers, an effect called hyperpolarization, which makes excitable tissue much less excitable, as was explained in Chapter 5.

In the sinus node, the state of hyperpolarization decreases the “resting” membrane potential of the sinus nodal fibers to a level considerably more negative than the normal value, to a level as low as -65 to -75 millivolts rather than the normal level of -55 to -60 millivolts. Therefore, the upward drift of the resting membrane potential caused by sodium leakage requires much longer to reach the threshold potential for excitation. Obviously, this greatly slows the rate of rhythmicity of these nodal fibers. And, if the vagal stimulation is strong enough, it is possible to stop completely the rhythmic self-excitation of this node.

In the A-V node, the state of hyperpolarization makes it difficult for the minute junctional fibers, which can generate only small quantities of current during the action potential, to excite the nodal fibers. Therefore, the safety factor for transmission of the cardiac impulse through the junctional fibers and into the nodal fibers decreases. A moderate decrease in this simply delays conduction of the impulse, but a decrease in safety factor below unity (which means so that the action potential of a fiber cannot cause an action potential in the successive portion of the fiber) completely blocks conduction.

Effect of Sympathetic Stimulation on Cardiac Rhythm and Conduction. Sympathetic stimulation causes essentially the opposite effects on the heart to those caused by vagal stimulation as follows: First, it increases the rate of sinus nodal discharge. Second, it increases the rate of conduction as well as the level of excitability in all portions of the heart. Third, it increases greatly the force of contraction of all the cardiac musculature, both atrial and ventricular, as discussed in the previous chapter.

In short, sympathetic stimulation increases the overall activity of the heart. Maximal stimulation can almost triple the rate of heart rate and can increase the strength of heart contraction as much as twofold.

Mechanism of the Sympathetic Effects. Stimulation of the sympathetic nerves releases the hormone norepinephrine at the sympathetic nerve endings. The precise mechanism by which this hormone acts on cardiac muscle fibers is still somewhat doubtful, but the present belief is that it increases the permeability of the fiber membrane to sodium and calcium. In the sinus node, an increase of sodium permeability causes a more positive resting potential and an increased rate of upward drift of the membrane potential to the threshold level for self-excitation, both of which obviously accelerate the onset of self-excitation and therefore increase the heart rate.

In the A-V node, increased sodium permeability makes it easier for the action potential to excite the succeeding portion of the conducting fiber, thereby decreasing the conduction time from the atria to the ventricles.

The increase in permeability to calcium ions is at least partially responsible for the increase in contractile strength of the cardiac muscle under the influence of sympathetic stimulation because calcium ions play a powerful role in exciting the contractile process of the myofilaments.

REFERENCES


(See also Chapter 9.)