3-D Physiology: Cardiac Conduction

The circulation of blood throughout our body is the result of the rhythmic contraction of the heart. The origination of each beat occurs within the heart itself.

The contraction of the heart is the result of two types of cardiac muscle cells, autorhythmic fibers and contractile fibers. Autorhythmic fibers do not contract but create and transmit the action potential, which stimulates the contractile fibers to shorten. Contractile fibers make up the bulk of the heart and are responsible for the movement of blood.

There are bundles of autorhythmic fibers that establish the rate at which the heart contracts. The bundle that creates the action potential most rapidly is found in the right atrium and is known as the Sinoatrial Node, or SA node.

The SA Node not only stimulates the atrial muscle to contract but also initiates an action potential in the ventricles. The Atrioventricular Node, or AV Node, which is connected to the SA Node by internodal bundles, carries the action potential from the atria to the ventricles.

The transmission of the action potential from the atria to the ventricles passes through the AV node at a slower rate, allowing the atria time to contract, maximizing the filling of the ventricles.

After leaving the AV Node, the autorhythmic fibers form the AV Bundle or Bundle of HIS, which splits to carry the action potential to each ventricle via the bundle branches.

From the Bundle of HIS, each branch rapidly transmits the action potential through Purkinje fibers to stimulate ventricular muscle contraction.

Although autorhythmic fibers each have the ability to create a rhythmic action potential they do not do so at the same rate. Since all other autorhythmic fibers follow the lead of the fastest depolarizing area, the SA Node is commonly referred to as the pacemaker of the heart. The SA node depolarizes, during rest, at about 70 to 80 beats per minute.

If the SA node becomes nonfunctional or its transmission to the ventricles is interrupted, the AV node can become the ventricular pacemaker at 40 to 60 beats per minute.

If the AV Node is unable to pace the heart, the Bundle of HIS and Purkinje fibers can pace the ventricular myocardium at 20 to 40 beats per minute.

We will begin the description of the generation of the autorhythmic cell action potential as the
membrane is repolarizing from the previous stimulating wave. Autorhythmic fibers do not have a resting potential because the membrane potential is continuously changing. As positive potassium ions diffuse out of the membrane, the inside of the membrane becomes more negative. We will chart the changes in electrical potential, in millivolts, that occur over time on the inside of the membrane.

The generation of the next action potential begins when the electrical potential on the inside of the membrane reverses direction. This is caused by F type sodium channels. They have the unique property of opening wider as the voltage inside the membrane becomes more negative, below –50 mv. As the F-type channels open, the increasing influx of positively charged sodium ions causes the membrane potential to become more positive.

As the membrane potential heads in the positive direction, F-Type channels close. A special type of calcium channel, known as a T-type channel, temporarily opens to allow positive calcium ions to diffuse in. This causes the membrane potential to reach a more positive value of negative 40 mv, the threshold which stimulates the generation of an action potential.

Once the threshold of negative 40 mv has been reached T-type calcium channels close. Then, another type of calcium channel, known as an L-type channel, opens to allow the rapid inward diffusion of calcium ions. This causes the membrane of the autorhythmic cells to depolarize to 0 mv as negative charges inside the membrane are cancelled by the addition of positively charged calcium ions.

At the same time, potassium channels are also stimulated, though they open slowly. As the potassium channels open and potassium ions diffuse out of the cell, the L-Type calcium channels close, blocking the entrance of any more calcium ions into the cell. The loss of positively charged potassium ions on the inside of the cell membrane causes the membrane to become negatively charged again, or repolarized.

This same sequence of events occurs beat by beat throughout our life. It is the depolarization and repolarization of the autorhythmic fibers that creates the action potential to stimulate contraction of the contractile fibers.

The stimulus from the autorhythmic fibers spreads through gap junctions to the next cardiac muscle cell, transmitting the action potential throughout the heart.

Connections between cardiac muscle cells, known as intercalated discs, bind muscle cells end to end. These connections contain both desmosomes, to provide a strong bond between cells, and gap junctions, to conduct the action potential from one muscle cell to the next.

The autorhythmic stimulus causes four voltage-gated channels in the contractile muscle cell membrane to open. It is the speed at which the channels open that determines the sequence of
events involved in muscle contraction

The first voltage-gated channel to open is the fast sodium channel. Sodium ions rapidly diffuse in resulting in the depolarization of the contractile fiber membrane to stimulate muscle contraction.

As the inside of the membrane depolarizes, fast sodium channels close and fast potassium channels open, allowing potassium ions to diffuse out, causing the membrane to begin to move in the negative direction.

Very shortly after the fast potassium channels open, L-Type Calcium channels open. Calcium ions diffuse in to balance the outward movement of potassium ions and keep the membrane depolarized at 0 mv for an extended period of time. Elevated intracellular calcium causes the cardiac muscle to remain contracted long enough for blood to move out of each chamber.

Finally, slow potassium channels open as the calcium channels close, resulting in the complete repolarization of the membrane to – 90 mv, and allowing the cardiac muscle to relax.

Stimulated by the action potential from the autorhythmic fibers, the depolarization and repolarization of the contractile fiber’s membrane results in well timed cardiac muscle contractions capable of moving blood through the cardiovascular system.

Monitoring the electrical stimulation of cardiac muscle on the surface of the body is known as the electrocardiogram, or ECG. It is important to note that the ECG indicates the summed depolarization and repolarization of cardiac muscle and not its contraction.

The ECG contains three basic waves. The P-wave occurs as the atria depolarize. Once the stimulation reaches all of the atria, the wave returns to its baseline.

The second wave is the QRS complex which indicates the depolarization of the ventricles. This is a larger wave than the P wave due to the greater mass of depolarizing muscle in the ventricles than in the atria. During the QRS complex the atria also repolarize, but the bulk of the ventricular muscle depolarizing creates a wave so large it overshadows the atrial repolarization.

The final wave is the T-wave which indicates the repolarization of the ventricles, allowing the ventricles to relax. Physicians look for abnormalities in the shape or timing of the electrocardiogram as a means of diagnosing the condition of the heart.

If we just consider the individual at a resting heart rate of 75 beats per minute, their heart beats
4500 times in an hour or 108,000 times every 24 hours. The process begins only a few weeks after conception and continues, nonstop throughout our life.