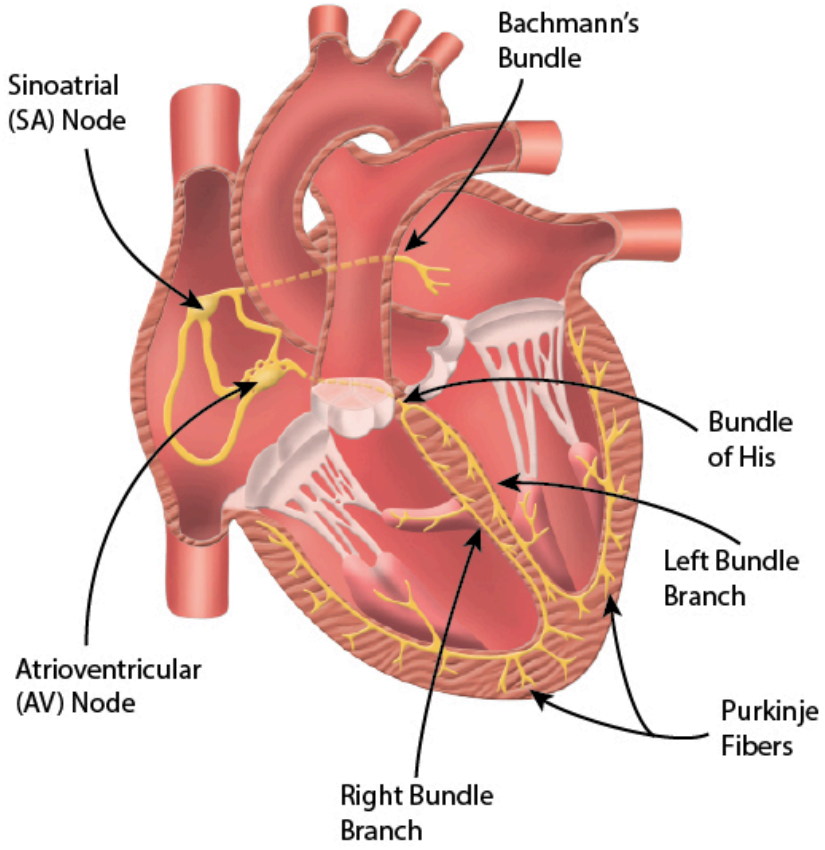
3.3.1

Heart Conduction System

Cardiac muscle cells have the ability to conduct action potentials from cell to cell through the gap junctions of the intercalated disks. This conduction, however, cannot account for the ordered, synchronous contractions observed in th heart. To ensure the proper sequence of contraction and to speed the conduction of the action potentials through the heart muscle, the heart is equipped with a specialized conduction system composed of non-contractile cardiac muscl cells that are modified for the task of generating and conducting action potentials. The autorhythmic cells discussed i the previous section are part of this system.



Electrical Conduction System of the Heart.

Image was drawn by BYU-Idaho student Nate Shoemaker Spring 2016

Located on the posterior wall of the right atrium near the site of connection of the superior vena cava is the **sinoatrial node (SA node)**. The SA node is composed of autorhythmic cells and is the **"pacemaker"** for the heart, generating the action potentials that initiate contraction. These action potentials then spread through the right and left atria, causing them to contract. Although cardiac muscle cells are perfectly capable of conducting action potentials from cell to cell, to achieve a synchronous contraction, the spread of depolarization must be tightly controlled. To aid the two atria in

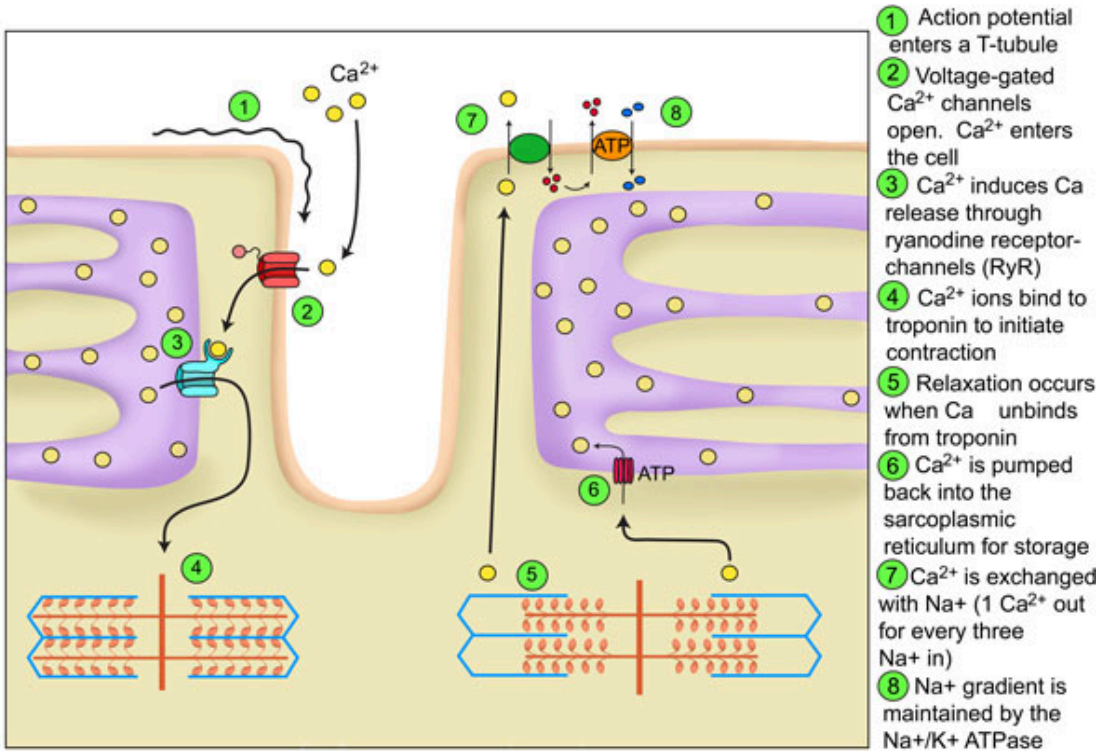
contracting simultaneously there is thought to be a special band of rapidly conducting tissue called **Bachman's bundle** that quickly spreads the action potential to the left atrium.

Although physically connected by a ring of connective tissue called the **cardiac skeleton**, the atria and the ventricles a electrically isolated, such that the action potential cannot spread directly from the atria to the ventricles. Instead, the action potential is detected by the **atrioventricular node (AV node)** which is located in the floor of the right atrium nea the **interatrial septum**. Again, there are specialized conduction pathways called the **internodal pathways** that quickly conduct the action potentials from the SA node directly to the AV node. The AV node then "delays" the action potential for approximately 0.15 seconds, allowing the atria to contract before the ventricles. This delay is due to the very slow speed of conduction in the AV node, ~0.05 m/sec. From the AV node, the action potential is conducted via the **atrioventricular bundle (AV bundle or bundle of His)** into the interventricular septum. In the septum the AV bundle splits into the **right and left bundle branches** that descend through the **interventricular septum** to the apex of the hea

At the apex, the bundle branches split into numerous **Purkinje fibers** that then ascend the walls of the ventricles. The AV bundle, bundle branches, and Purkinje fibers conduct the action potentials much faster than the cardiac muscle fibers, 1-4 m/sec vs 0.3-0.4 m/sec, respectfully. This rapid conduction creates a more coordinated contraction of the ventricular muscle. Also, since the action potentials, and hence contractions, spread from the apex toward the base of the heart the blood is pushed up toward the large arteries exiting the ventricles.

It should be noted that even though the SA node is the pacemaker, the other components of the conducting system ar also capable of spontaneously generating action potentials. Each has its own intrinsic rate of generating action potentials, the SA node has a rate of 60-80/minute, the AV node a rate of ~40/minute and the AV bundle and Purkinje fibers a rate of ~20/minute. The reason the SA node is the "pacemaker" is simply because it has the fastest rate and reaches threshold before the other areas. If the SA node becomes damaged or stops functioning the AV node can take over and the heart will continue to beat, albeit at a slower rate.

Sometimes excitable cells (pacemaker cells) can grow in the heart in other places besides the nodes. When this happens, we call the location an ectopic focus. This is usually not life-threatening, but over time can disrupt the norma conductance of the heart and alter the heartbeat, making it beat faster than normal or slower than normal. In most cases, the faster rate of the SA node will mask the other cells, but if the SA node becomes compromised, ectopic foci can begin to control the heart rate, a scary situation because the ectopic foci is not modulated by the nervous system. Ectopic foci can also alter electrocardiogram readings (ECG), appearing as extra deflections and causing misdiagnosis



Cardiac Excitation Coupling

BYU-Idaho image by Becky T: Created Fall 2018

Now that you know how action potentials are generated and conducted in the heart, we need to study how these actio potentials lead to contraction of the cardiac muscle cells. We have already explained that cardiac muscle is very much like skeletal muscle and that the mechanism of contraction is the same in both muscle types. Therefore, the key to cardiac muscle contraction is also calcium. As with skeletal muscle, calcium is stored intracellularly in the sarcoplasmic reticulum. However, unlike skeletal muscle, cardiac muscle also relies on extracellular calcium for prope functioning. This means that the extracellular level of calcium is very important for heart muscle function.

Intracellular stores of calcium in the well-developed sarcoplasmic reticulum of skeletal muscle can help buffer against blood calcium level changes, but heart muscle does not have this advantage. The heart muscle sarcoplasmic reticulu is much less developed and heart muscle fibers are more sensitive to extracellular calcium level oscillations. In fact, small changes in blood calcium levels can cause heart arrhythmias.

As might be expected, there are some important ion channels that mediate the entry of calcium. The first is a voltage-gated calcium channel found in the membranes of the T-tubules (L-Type Calcium Channels). These are the same channels discussed earlier that are responsible for the plateau phase of the action potential. When the action potentia descends into the T-tubules these channels open, allowing calcium to diffuse into the cell. This calcium then binds to calcium release channels found in the membranes of the SR (these channels are also known as Ryanodine receptor, RyR, channels). The binding of calcium to these channels causes them to open allowing calcium to diffuse out of the S and bind to troponin initiating contraction. Thus, extracellular calcium triggers the release of sarcoplasmic reticular calcium. This process is referred to as calcium-induced, calcium release. Once the signal ends, a calcium ATPase (SERCA) in the membranes of the SR pumps the calcium back into the SR and contraction ends. Since some of the calcium that is now in the cell came from the extracellular fluid, a Na^+-Ca^{2+} exchanger (secondary active transport 3 Na^+ to 1 Ca^{2+}) moves calcium from the cytoplasm back to the extracellular fluid. The strength of the muscular contraction in the heart is dependent on the amount of calcium that enters; hence, there are mechanisms to regulate how much calcium enters the cells.