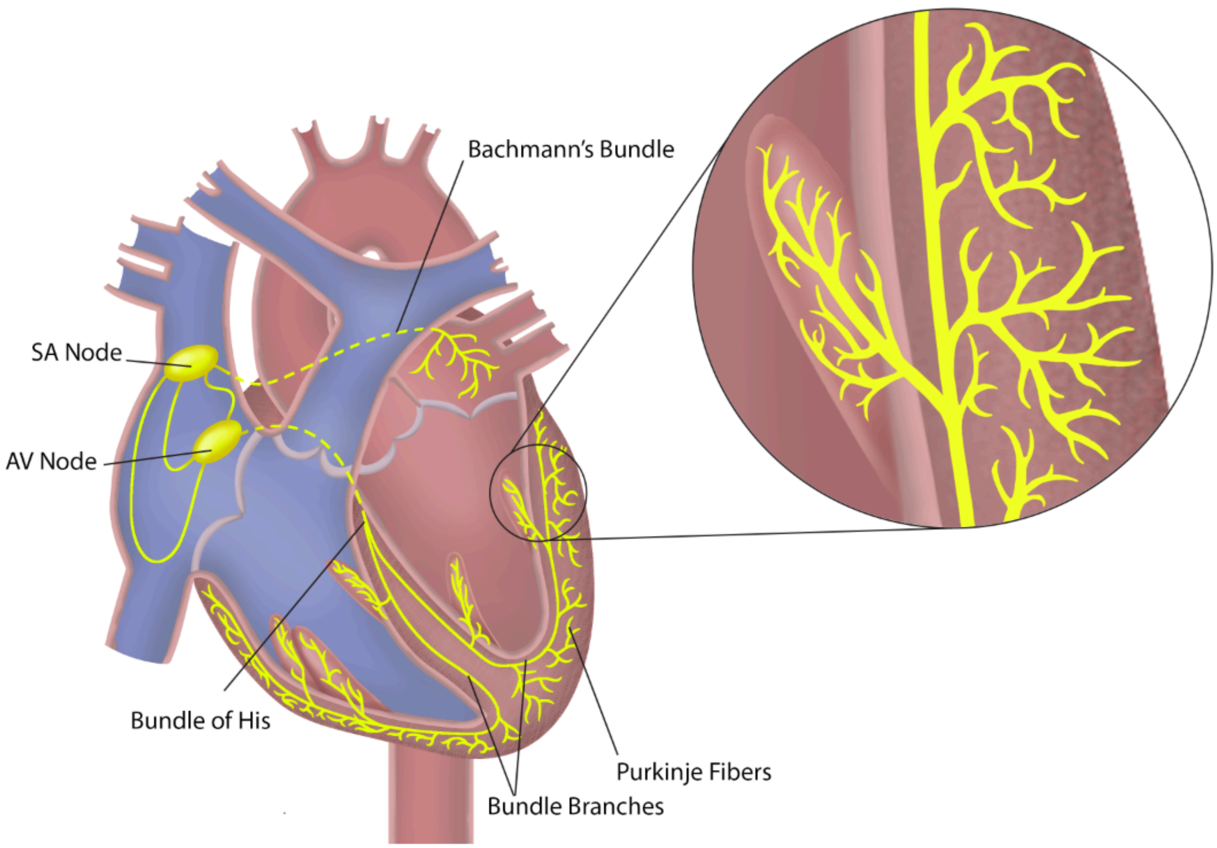
1.3.1

Heart Conduction System

As described earlier, 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, is relatively slow and cannot account for the ordered, synchronous contractions observed in the 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 muscle cells that are modified for the task of generating and conducting action potentials. The autorhythmic cells discussed in the previous section are part of this system.



Electrical Conduction System of the Heart. *Image by Becky T. S20*

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, the rate of conduction is relatively slow. 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 are 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 near 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 heart.

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, respectively. 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 are 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 they can disrupt the normal 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.