Cardiac Conduction System

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In order to understand the pediatric electrocardiogram, one must first understand the normal path of electrical conduction in the heart. In the structurally normal heart, the cardiac impulse is automatically generated by the pacemaker of the heart, the sinus node, and terminates in the ventricular myocardium. This chapter will discuss the anatomy and physiology of everything in between.

Sinus Node

The cardiac impulse begins in the sinus node, which historically, was the last component of the conduction system to be discovered. Discoveries actually began in 1839 with Jan Evangelista Purkinje, who found his famous fibers but believed that they were nonconducting cartilaginous structures. By the 1880s, Walter Gaskell knew that the cardiac impulse was generated at the sinus venosus but did not identify the specific source. Wilhelm His discovered his bundle in 1893. Sunao Tawara found the "complex knoten" known as the atrioventricular node in 1906. Finally, Arthur Keith and Martin Flack, an anatomist and medical student, respectively, identified the sinus node in 1907, thus completing the cardiac conduction system. Thomas Lewis later proved, using primitive electrocardiography, that the sinus node was the pacemaker of the heart [1].

The sinus node is a 10–20 mm by 5 mm mass located at the lateral aspect of the superior vena cava-right atrial junction just beneath the epicardium. Embryologically, it begins as a horseshoe-shaped structure in fetal life but morphs into a crescent structure by birth. In 90 % of people, it is located just inferior to the crest of the right atrial appendage, whereas in 10 % it extends across the terminal crest. The so-called head of the sinus node courses toward the interatrial groove, while its tail extends toward the inferior vena cava [2].

There are three distinct sinus node cell types identified by histology: nodal cells, transitional cells, and atrial muscle cells. Nodal cells are small, ovoid, and pale when stained with hematoxylin and eosin. They are poorly striated and have fewer mitochondria than contractile myocytes, which makes sense from a functional standpoint, given that they are not responsible for contraction and therefore require less energy. Nodal cells are grouped together in a complex fibrous meshwork of interconnecting fascicles. Transitional cells, as the name implies, share properties of nodal and atrial myocytes. They bridge the gap between the nodal and atrial myocytes, allowing impulse propagation between the two [3].

Internodal and Interatrial Conduction

Internodal and interatrial depolarization travels from the sinus node to the left atrium and atrioventricular node (AV node) via internodal tracts. Heated debate has occurred over the years as to whether these tracts contain specialized myocytes [4]. The current evidence indicates that they are not histologically different but are the preferred route of conduction due to optimal muscle fiber orientation, thickness, and geometry. A study of cardiac specimens conducted by James in 1963 showed that the preferred pathways contained an abundance of Purkinje fibers, mixed with other cells including fat cells. In the end, there are three distinct pathways of conduction between the sinus and AV node and between the right and left atrium [3].

James described the course of the internodal tracts in great detail. The anterior internodal tract courses anteriorly from the sinus node along the superior vena cava and anterior wall of the right atrium to Bachmann's bundle, where it

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divides into an anterior and posterior branch. The posterior branch conducts the cardiac impulse from the right to the left atrium. The anterior branch curves back toward the anterior portion of the interatrial septum, eventually terminating at the anterior superior margin of the AV node. The middle internodal tract, also known as Wenckebach's bundle, curves behind the superior vena cava, courses through the sinus intercavarum into the dorsal portion of the interatrial septum, and terminates in the superior margin of the AV node. The posterior internodal tract, or Thorel's bundle, courses posteriorly through the entirety of the crista terminalis and continues to the AV node via the Eustachian ridge [5].

Atrioventricular Junction

The atrial components of the atrioventricular axis are confined within three anatomic landmarks in the right atrial wall, which include the continuation of the Eustachian valve into the atrial myocardium, the tendon of Todaro (absent in two-thirds of people), and the hinge of the septal leaflet of the tricuspid valve. Collectively these boundaries are referred to as the triangle of Koch.

The AV node causes a delay in the cardiac impulse before it is propagated to the ventricular pathways. There are three distinct components of the AV junctional area: the atrial myocardium, the transitional cell zone, and the compact AV node. The transitional cell zone is a connection between the working atrial myocardium and the outer layer of the compact AV node. The compact AV node is located at the apex of the triangle of Koch, just beneath the right atrial posterior epicardium. At the base of the triangle is the coronary sinus. Below that is the cavo-tricuspid isthmus, a component of the most common form of atrial flutter. Between the coronary sinus and the hinge of the tricuspid valve is the septal isthmus, which contains the slow pathway into the AV node. The AV node lies directly next to the central fibrous body of the heart, which is formed by the fusion of the membranous septum with the rightward end of the area of fibrous continuity between the leaflets of the aortic and mitral valves in the roof of the left ventricle [4]. The distal continuation of the compact AV node is the penetrating portion of the AV bundle, or the bundle of His. In this region, the irregularly arranged fibers of the AV node become more organized and parallel, thereby forming the His bundle. This bundle subsequently dives into the ventricle through the fibrous central body, where it surfaces and then branches at the interface between the membranous and muscular ventricular septum in the left ventricular outflow tract [4, 6].

In the normal heart, the atrioventricular axis is the only myocardial structure that crosses the insulated plane of the AV junction. In patients with Wolff-Parkinson-White syndrome, there are other muscular connections between the right atrium and right ventricle, known as bundles of Kent, that allow for the electrical impulse to bypass the normal conduction pathway and preexcite the right ventricle before the impulse has had a chance to transmit through the AV junction. There are other accessory pathways that can exist between the His bundle and the crest of the ventricular septum. These are called Mahaim fibers and can also cause preexcitation. These accessory pathways are discussed further in later chapters.

Ventricular Conduction System

The ventricular conduction system is made up of the bundle branches and the Purkinje fibers. Conduction through this system is rapid due to specialized connexin proteins with high conductance properties [2].

Bundle Branches

The bundle of His divides into the right and left bundle branches at the crest of the muscular interventricular septum. The left bundle branch originates first from the common AV bundle and spreads like a web over the left ventricle, dividing into three branches, the anterior, middle, and posterior branches. The right bundle branch arises more distally from the common bundle and courses in the moderator band toward the right ventricular free wall. Its course from the common bundle along the right side of the ventricular septum begins subendocardially, then continues through the septal myocardium, and finishes subendocardially as it fans out into fascicles over the right ventricular myocardium [7]. From the fascicles in both ventricles, the impulse reaches the working myocytes via the Purkinje network.

Purkinje Fibers

First discovered by Jan Purkinje and described as nonconducting cartilaginous structures, these fibers are the final component of the cardiac conduction system. They are located in the subendocardium and extend transmurally. In human hearts, they are histologically not much different than adjacent ventricular myocytes. Interestingly, there are myocytes in the atrial myocardium and the pulmonary venous sleeves that resemble Purkinje cells and may be a nidus for ectopy [2].

Although their primary function is to conduct the cardiac impulse, Purkinje fibers can group together as false tendons and contribute, albeit minimally, to contraction.

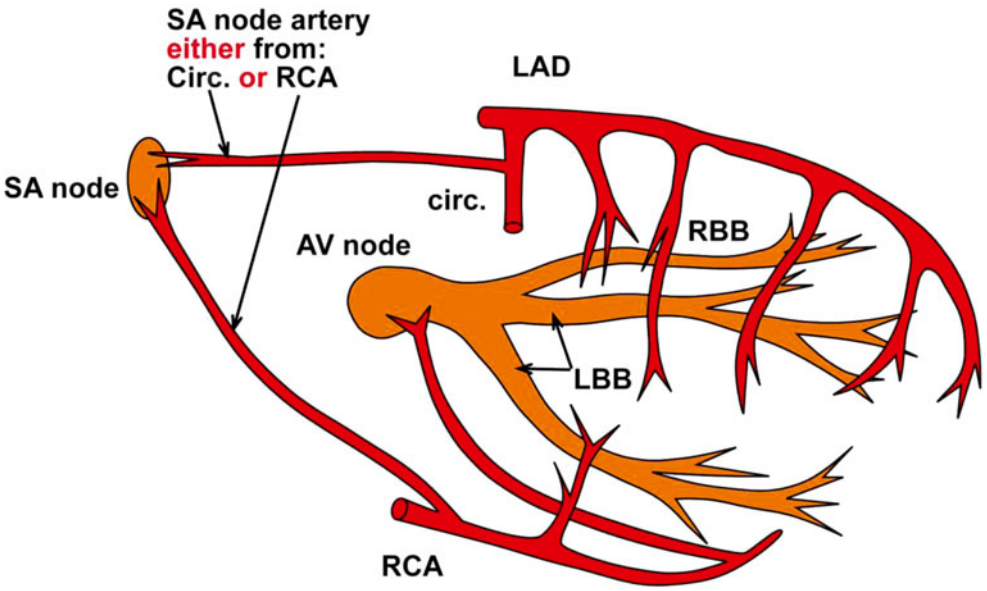

Fig. 4.1 Coronary blood supply to the cardiac conduction system. SA node is supplied either by a branch from the left circumflex artery or the right coronary artery, *not both as the illustration may imply*. AV node is supplied by the right coronary artery in most instances. *AV node* atrioventricular node, *Circ.* circumflex artery, *LBB* left bundle branch, *LCA* left coronary artery, *RBB* right bundle branch, *RCA* right coronary artery, *SA node* sinoatrial (sinus) node

Blood Supply

The sinus node receives its blood supply from the right coronary artery in 55–60 % of cases and the left circumflex coronary artery in 40–45 % of cases. The AV node is supplied by the AV nodal branch of the right coronary artery in about 80–85 % of people. In the other 10–15 %, it is supplied from the left circumflex artery. Due to its location in the superior muscular ventricular septum, the His bundle has a rich blood supply from the left anterior and posterior descending coronary arteries, making it well protected against ischemia [6] (Fig. 4.1).

Autonomic Control of Conduction

Autonomic input to the cardiac conduction system controls heart rate, also called chronotropy. An increase in heart rate is called positive chronotropy and a decrease is called negative chronotropy. It is important to understand that autonomic control of conduction is not merely the net sum of two opposing actions working independently at different parts of the conduction system, but rather it is an interactive process in which vagal and sympathetic nerves antagonize each other's ability to function. In terms of chronotropy, the autonomic nervous system exerts its main effects on the sinus and AV nodes. The sinus node is richly innervated with postganglionic adrenergic and cholinergic nerve terminals, containing more than a threefold greater density of beta-adrenergic and muscarinic cholinergic receptors than adjacent atrial tissue. Stimulation of beta-adrenergic receptors results in positive chronotropy. Stimulation of muscarinic receptors in the sinus node by acetylcholine produces the opposite effect, negative chronotropy. Acetylcholine also produces negative chronotropy by prolonging intranodal conduction time. Similar effects are seen at the AV node [8].

It took many years to fully define the elusive cardiac conduction system. Each component plays a unique role in the transmission of the cardiac impulse. Knowing the anatomy and path of conduction will allow one to fully understand the components of the pediatric electrocardiogram.

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