Introduction to the Congenital Heart Defects
Anatomy of the Conduction System

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KEYWORDS

• Congenital heart disease • Conduction system • AV node • His-Purkinje system • Heart block
• Supraventricular tachycardia • Atrioventricular reciprocating tachycardia • Heterotaxy syndrome

KEY POINTS

- Knowledge of the location of the conduction tissue is essential for safe and effective arrhythmia management for patients with congenital heart disease.
- The sinus node complex is located in the usual position for most forms of congenital heart disease with the notable exception of the heterotaxy syndromes.
- The location of the compact atrioventricular node and His bundle vary significantly according to the type of congenital heart malformation.
- There are scarce histologic data on the location of transitional cell inputs to the atrioventricular node in congenital heart disease.
- Knowledge of both histologic and clinical data of the location of the conduction system should improve the safety and efficacy of ablation procedures in this population.

INTRODUCTION

The conduction system in congenital heart disease has been studied extensively with notable publications emerging in the early 20th century.[1] The nature of the conduction system in congenital heart disease is intricately related to the underlying lesion, and no description can occur without reference to the unique structural anatomy of this population. Although only subtle differences between simple congenital heart lesions and normal may exist, almost every patient with congenital heart disease harbors an important variation in the conduction system anatomy.

Studies of the conduction system based on histopathology techniques are generally limited to identification of the sinus node complex, the compact atrioventricular (AV) node, and the His-Purkinje system in relation to the identifiable gross anatomic structures as visualized by the pathologist or cardiac surgeon. As a consequence, application of this information to the electrophysiology laboratory is not necessarily straightforward. Perhaps more importantly, because of the focused nature of these studies, there are limited data on the location of the transitional cell inputs to the compact AV node. This finding may be clinically relevant, for instance, when attempting to modify the slow pathway for AV node reentry tachycardia in patients with complex congenital heart disease.

This article summarizes the vast body of literature by keeping to the original classical concepts and by attempting to translate the available

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knowledge into useful points for the practicing interventionalist and electrophysiologist. Greater focus is placed on those lesions with the most significant departure from normal, where an in-depth understanding of the conduction system is essential for safe and effective treatment of cardiac arrhythmia. This discussion spans the entire spectrum ranging from simple to complex congenital heart disease.

SIMPLE DEFECTS

Atrial Septal Defect

The embryologic location of the conduction tissue in atrial septal defect is generally not altered by the congenital heart disease, except for the primum atrial septal defect, which is described extensively in the section on atrioventricular septal defect. With right heart enlargement caused by augmented pulmonary blood flow, however, there may be significant right atrial and ventricular enlargement with distortion of the landmarks for the conduction system anatomy.

Ventricular Septal Defect

There are multiple anatomic types of ventral septal defect (VSD), all of which have important implications for the anatomy of the conduction system. In general, the compact AV node together with the transitional cell inputs are expected to be located in their normal location at the apex of the triangle of Koch for all of the various subtypes, whereas the AV bundle and bundle branches are located variably. These issues are generally of greatest importance to the cardiac surgeon, who must repair these defect without incurring AV block. With the increasing frequency of transcatheter interventions for VSD closure, however, this anatomy must also be increasingly understood by the interventional cardiologist.

There are various classification schemes for VSD, generally derived for their surgical significance.[2,3] The most common type of VSD is the perimembranous (also referred to as *membranous*, *conoventricular*, or *subaortic*) defect, which involves a deficiency in the membranous portion of the interventricular septum and the surrounding muscular tissue.[4] The defect may extend into the inlet, trabecular, or infundibular portions of the ventricular septum, and is named according to the type and degree of extension. Although the precise relationship between the AV bundle and the defect varies with the type of extension, all perimembranous defects share the quality that the AV bundle passes posterior and inferior to them (or rightward as viewed by the surgeon). The portion of the bundle that is at highest risk for surgically-induced AV block is located at the posteroinferior edge of the defect where it is encased in a thin rim of fibrous tissue (**Fig. 1**).[5] In general, defects with extension into the inlet septum are most closely related to the AV bundle whereas defects with outlet extension are the most remote. Defects with muscular trabecular extension are intermediate with respect to their proximity to the conduction tissue but are generally considered to be at low risk for surgically-induced AV block.

Of the other types of VSD, isolated muscular inlet defects are important to recognize. These are the only VSDs in which the conduction tissue is located in an anterosuperior position relative to the defect (or leftward as viewed by the surgeon). Fortunately, these defects are typically remote from the AV bundle.[6] The final category of defects, the muscular outlet defects and the so-called doubly committed or subarterial defects, are also remote from the conduction tissue. In these cases, the AV bundle and bundle branches travel posterior and inferior to the defect, but at a distance.

MODERATELY COMPLEX DEFECTS

Atrioventricular Septal Defect

The gross and histologic anatomy of the AV conduction system in atrioventricular septal defect

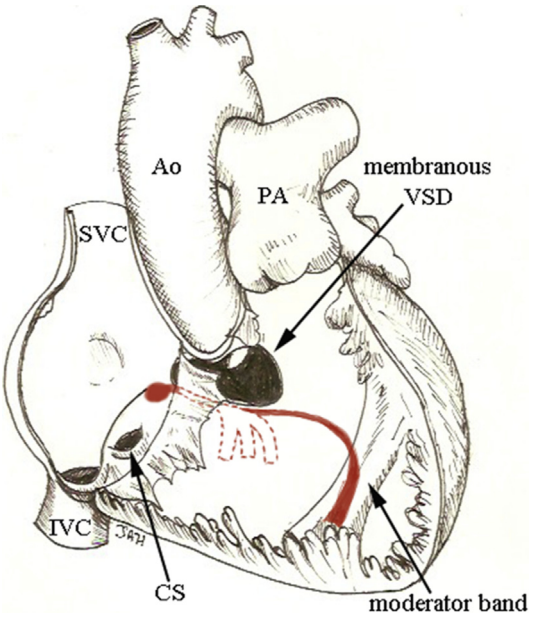

Fig. 1. Perimembranous septal defect. Ao, aorta; CS, coronary sinus; IVC, inferior vena cava; PA, pulmonary artery; SVC, superior vena cava; VSD, ventricular septal defect.

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