SYMPOSIUM
Cardiac Arrhythmias
(Part 1)

Anatomy of the Conduction System

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SUMMARY
Some, but not all, cardiac arrhythmias are related to pathologic lesions of the cardiac conduction system. Common atrial dysrhythmias and first-degree atrioventricular (A-V) block rarely are explained on the basis of anatomic lesions in specific sites of the conduction system or its blood supply. Second-degree A-V block of Mobitz type II, which may be a precursor of complete (third-degree) heart block, commonly is associated with fibrotic lesions of uncertain etiology in the branching part of the bundle of His or the bundle branches. Ischemic lesions are found less often, and other pathologic processes rarely are present. Chronic complete heart block most often results from nonspecific, fibrotic interruption of the distal bundle of His, or of the first parts of the bundle branches after their origins. Ischemic lesions are uncommonly the cause of chronic block. High-grade A-V block complicating acute myocardial infarction may be associated with infarction of the A-V conduction system, but often morphologic evidence of ischemia cannot be identified. Congenital variants in anatomy of the conduction system are responsible for some relatively uncommon arrhythmias.

Additional Indexing Words:
Heart block, complete Heart block, incomplete Sinus node Atrioventricular node
Bundle of His Left bundle branch Right bundle branch

Correlations between abnormal cardiac electrophysiology and anatomic findings in the heart and its conduction system have been sought for many years. Some dysrhythmic states result from specific pathologic lesions, but many have not been correlated definitely with an anatomic abnormality. Additionally, different dysrhythmias, particularly of atrioventricular conduction, seem to be associated with similar pathologic lesions. The purpose of this report is to review those conditions in which a pathologic lesion of the conduction system appears to be the basis for an arrhythmia. Traumatic lesions, such as injuries to the conduction system during surgical procedures, with the production of permanent or transient electrocardiographic abnormalities, will not be discussed.

Congenital Abnormalities
The findings in the conduction system in congenitally malformed hearts will not be considered except for those instances in which reasonably specific electrocardiographic abnormalities usually are present and likely explicable by the anatomy of the conduction system.

Persistent common atrioventricular canal has reasonably characteristic abnormal electrocardiographic and vectorcardiographic features related to relatively early excitation of the posterior parts of the left ventricle. Morphologically, 1 the atrioventricular conduction system is deformed by the large deficiency of the septal tissue in such a fashion that, compared to normal hearts, the posterior left bundle-branch fascicles have a relatively early origin from the common bundle, and the anterior fascicles appear relatively, perhaps absolutely, deficient. Additionally, the right bundle branch appears to have an abnormally long course relative to that of normal hearts.

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In **tricuspid atresia**, the electrocardiographic finding of a counterclockwise loop with left-axis deviation also may be related to an early origin of the left bundle branches and a markedly elongated course of the right bundle.²

**Congenital complete heart block** usually results from congenital interruption of one or more parts of the major atrioventricular conduction system, most commonly involving the bundle of His or the origins of the bundle branches.³

In contrast to the foregoing situations, in most instances of tetralogy of Fallot, ventricular septal defects of different types, origin of both great arteries from the right ventricle, and corrected transposition of the great arteries (levotransposition with ventricular inversion), classic electrocardiographic and vectorcardiographic features do not appear to reflect primarily the anatomy of the conduction system.⁴

**Acquired Abnormalities**

**Atrial Dysrhythmias.** In most instances, common abnormalities such as atrial fibrillation, atrial flutter, and atrial tachycardias cannot be related to lesions in specific sites of the conduction system, although they often are related to atrial pathology. Atrial dysrhythmias present in coronary heart disease, hypertensive cardiac disease, and posttraumatic valvular deformities usually cannot be ascribed specifically to morphologic abnormalities of known atrial conduction tissues. When functional-morphologic correlations have been established, the pathologic lesions most often have been nonspecific inflammation, degenerative and fibrotic processes, or ischemic states secondary to disease of the sinus node artery or its parent vessel.⁵⁻⁶

**Abnormalities of Atrioventricular Conduction.** For many years, anatomic studies of the atrioventricular conduction system have searched for pathologic lesions to explain the pathogenesis of abnormal atrioventricular conduction of various types and have attempted to define etiologic factors responsible for the lesions. In general, anatomic correlations have been most successful when the atrioventricular conduction disturbance was a relatively stable, permanent one. Electrophysiologic information and definitions, particularly since the advent of intracardiac electrophysiologic recordings, especially His bundle electrograms, have led to reasonably precise localization and comprehension of many atrioventricular dysrythmias, but morphologic correlates with many of these studies are imprecise or have not been made with certainty. Problems in making such correlations include: (1) an electrophysiologic abnormality that may not have a clearly demonstrable anatomic basis but may be the result of physiologic or biochemical factors such as drug effects and alterations in electrolyte concentrations; (2) the inability as yet to recognize and predict the effects of pathologic lesions that do not completely interrupt a defined part of the conduction system; (3) the possibility that the morphologic lesions are within the terminal ramifications of the conduction system, that is, the distal parts of the bundle branches, which are difficult to study morphologically; and (4) the possibility that the morphologic lesions are within the myocardium apart from the conduction system.

The discussion that follows is considered in terms of electrophysiologic abnormalities with indication of the site(s) of morphologic lesions responsible for the arrhythmia and comments on etiologic and pathogenetic factors.

**Atrioventricular Dissociation**

Atrioventricular dissociation without atrioventricular block theoretically may result from functional abnormality in the atrium, especially the sinus node, the atrial approaches to the A-V node, the A-V node itself, the common bundle (His bundle), or the origins of the bundle branches. Definitive morphologic correlations have not yet been made. In some instances, no morphologic abnormality has been found. In other instances, pathologic lesions of one or more parts of the atrioventricular conduction system have been identified, but the types of lesions and their locations are not different from those associated with various atrioventricular blocks, discussed in the following paragraphs. Although correlative data are not complete, many instances of atrioventricular dissociation without block appear to have a functional rather than an anatomic basis.

**Incomplete Atrioventricular Block**

Incomplete atrioventricular block includes both first-degree atrioventricular block with a prolonged P-R interval and second-degree block with dropped beats.

**First-degree atrioventricular block** appears most often to be “functional” in origin rather than having a definitive morphologic explanation. Morphologically, nondestructive inflammatory changes of cellular exudation and apparent edema may be found in the approaches to the A-V node, the node itself, the common bundle, or the origins of the...
bundle branches in some cases, particularly those associated with an inflammatory disease such as acute rheumatic fever. In other instances, small foci of nonspecific fibrosis have been found in one or more of these parts of the atrioventricular conduction system. As with atrioventricular dissociation, anatomic-electrophysiologic correlations are imprecise in most cases since the lesions observed usually differ only in extent, if at all, from those associated with other, more advanced types of atrioventricular block. The etiologic and pathogenetic factors responsible for foci of fibrosis, which apparently never completely interrupt the affected area, are not firmly defined. Speculative considerations include previous inflammation, ischemic heart disease, and idiopathic degenerative change. At this time, degenerative change probably is the most frequent explanation.

Second-degree A-V block also may result from abnormality in any of the components of the A-V conduction system. Those types characterized by the Wenckebach phenomenon (Mobitz type I second-degree A-V block) often do not have a morphologic lesion that explains satisfactorily the functional abnormality. This, and the fact that this form of incomplete A-V block often is not permanent, support the conclusion that the arrhythmia more often is related to physiologic than to anatomic factors. Like A-V dissociation and first-degree block, morphologic lesions observed generally have been nonspecific and have not had specific localizations that allowed precise correlations.

In contrast to the foregoing situations, second-degree A-V block without the Wenckebach phenomenon (Mobitz type II) appears to have a more definite morphologic basis, although relatively few cases have been studied. It is difficult to find autopsied cases in which this arrhythmia has existed for a reasonably long time, possibly because this abnormality often is a precursor of complete (third-degree) A-V block. Extrapolation from the morphologic findings in instances in which third-degree block followed a period of second-degree block and clinical and experimental electrophysiologic studies are the bases for many of the anatomic correlations. The block may be related to abnormality in any part of the atrioventricular conduction system, but morphologic lesions most often are found in the branching part of the bundle of His, or in the bundle branches themselves (fig. 1), or in both areas. It is uncommon for the lesion, whatever its nature, to replace all fibers of the involved area, but the relative percentages of conduction fibers that can be lost and still maintain some atrioventricular conduction have not been defined. Morphologically, the extent of the abnormality and its location appear to determine whether second- or third-degree block will be present, but prediction of the type of atrioventricular block is hazardous unless complete interruption of the fibers is found. Any combination of localizations and extent of lesions may be found. Depending on the combination in a given heart, the patient may have had Mobitz type II second-degree block, intermittent bundle-branch block, or block of the anterior or posterior fascicles of the left bundle branch (anterior and posterior hemiblocks), with or without right bundle block. Incomplete forms also could occur. Which functional abnormality may be present with a given anatomic lesion apparently is dependent not only on the lesion itself but also on the many physiologic factors that influence transmission of the impulse at any given moment. Further complicating the interpretation of pathologic findings is the possibility that fibrous septae in the bundle of His of some hearts may reflect specific longitudinal pathways of transmission of the impulse from the A-V node to the ventricles so that a lesion in the His bundle might be related to an arrhythmia that is generally ascribed to lesions more distal in the A-V conduction system.

Despite the rather formidable problems in establishing anatomic correlates with incomplete forms of A-V block, morphologic lesions usually are found in the A-V conduction systems when an arrhythmia other than first-degree block was a reasonably permanent phenomenon. The location and extent of the lesions have been considered. Anatomically, the lesions rarely may be congenital interruption or lack of development of one or more areas. Specific and nonspecific inflammatory processes may be found, including rheumatic fever, rheumatoid arthritis, syphilis, tuberculosis, fungal infections, myocarditides of various etiologies, and granulomatous processes of unknown etiology. Rarely, a neoplastic lesion may be so situated as to involve one of the anatomic areas leading to incomplete block. Uncommonly, infiltrations in the conduction system such as amyloid and hemochromatosis have been found. The common lesions associated with some form of incomplete A-V block are idiopathic degenerative processes and ischemic conditions. Degenerative conditions seem to be more common than clearly ischemic lesions, even when the block appears to be a complication of myocardial infarction. Both conditions have the
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Figure 1

Lesions of bundle branches associated with atrioventricular block. (Left) Severe fibrosis of intramyocardial portion of right bundle branch (seen in center). (Right) Fibrosis of left bundle branch (L). (Mallory-Heidenhain stain, ×50.)

same general morphologic features, apart from extent, whether associated with incomplete or with complete A-V block discussed in the following.

Complete Atrioventricular Block

Complete atrioventricular block (third-degree heart block) that is a permanent, chronic condition virtually always can be related to morphologic interruption of one or more parts of the A-V conduction system.\(^7,12,13,16-22\) The interruptive pathologic alteration could be in the approaches to the A-V node, the node itself, any part of the His bundle, or the bundle branches before their terminal ramifications in the subendocardium of the right and left ventricles. Destruction of the atrial connections (approaches) to the node as an isolated event occurs rarely, if ever. Lesions that destroy or replace the A-V node, with the exception of a few rare neoplasms, usually also involve the bundle of His and the origins of the bundle branches. For these reasons, it is appropriate to focus on morphologic lesions of the common bundle and its branches in consideration of permanent complete (third-degree) heart block. A-V blocks complicating acute myocardial infarction are somewhat specialized situations to be discussed separately.

Although any type of lesion may be found, including inflammatory, neoplastic, infiltrative, degenerative, and ischemic types, the most common morphologic lesions in chronic complete A-V block are nonspecific fibrotic or fibrocalcific processes involving the distal common bundle, or the origins of the bundle branches, or both.\(^7,19\) Lenègre\(^7\) proposed that the fibrotic or fibrocalcific lesions might be related to a sort of compression of the conducting tissues between fibrosis of the summit of the ventricular septum and adjacent endocardial fibrous tissue or fibrous tissue of the atrioventricular junction. Levy\(^19,20\) suggested that the fibrotic process was related to "wear and tear" on the fibrous supporting tissues of the heart and termed the condition "sclerosis of the left side of the cardiac skeleton." Yater and Cornell\(^16,17\) previously referred to similar findings as a result of "stress and strain." Morphologically, the lesions are usually composed of collagen fibers that replace the normal myogenous conduction fibers (fig. 2). In many
Figure 2

Chronic complete heart block due to fibrotic replacement in distal common bundle and origins of bundle branches. (Top) His bundle (B) is slightly fibrotic but all fascicles of left bundle branch were interrupted at their origins by fibrous tissue (arrow). (Bottom) Distal, branching bundle (B) was markedly fibrotic, as were both left (L) and right (R) bundle branches. (Mallory-Heidenhain stain, × 22.)

cases, the fibrosis is peculiarly limited to the conduction tissue but, often, increase of the normal fibrotic tissue of the region is apparent; collagenous structures in the region are the fibrous atrioventricular ring, the central fibrous body (right fibrous trigone), the membranous septum, and the normally thin collagenous components of the endocardium. In most cases, there also is some degree of nonspecific fibrosis of the muscular septum in the form of increased interstitial connective tissue and fibrous replacement of muscle fibers. Inflammation and infiltrations of abnormal substances such as amyloid are uncommon. Vascular disease of epicardial coronary arteries, or of small intramyocardial muscular arteries, or of arterioles cannot be implicated in most instances. At this time, the etiology and pathogenesis of the fibrous degenerative lesions that are the most common morphologic abnormality associated with chronic complete heart block are uncertain, so that idiopathic sclerosis or similar terminology is a suitable designation for the problem.

When chronic complete block is present in conjunction with calcific aortic valvular disease, or rarely calcification of the mitral annulus, lesions of the conduction system are found in similar sites to
those just described. The fibrocalcific lesions appear to be extensions of the fibrocalcific valvular lesion into the His bundle and bundle branches. Rarely, specific or nonspecific inflammatory or infiltration processes may destroy the same tissues and be the cause of heart block.

Vascular disease may lead to ischemic destruction of the conduction tissues and produce heart block. While infarction of these tissues may result from disease of major epicardial coronary arteries or from occlusive lesions of smaller vessels, true ischemic infarction of the A-V conduction system is uncommon at autopsy, even when heart block complicates acute myocardial infarction. When found, infarction most often is identified in the bundle branches (fig. 3) rather than in the His bundle. In many instances, the severity and extent of myocardial infarction all around the morphologically intact A-V conduction system suggest a relative immunity of the conducting tissues to ischemia, at least in a morphologic sense. These morphologic findings are supported by the temporary nature of many instances of complete heart block complicating acute myocardial infarction. Sometimes, lesions of the conduction system are present that morphologically antedate the infarction; in this situation, the conduction system may have been compromised already and the physiologic, biochemical, or morphologic alterations of the infarction completed functional interruption.

Any of the morphologic abnormalities mentioned might be located in the right and left bundle branches prior to their terminal divisions and be responsible for complete heart block because of production of "trifascicular block." It is uncommon for lesions responsible for complete block to have only these specific localizations. When a process involves the bundle branches distal to their immediate origins, consideration of normal anatomy indicates that it must be an extensive one in the basilar part of the ventricular septum in order to interrupt all fibers of the fascicular divisions of the left bundle branch. Most cases have been related to ischemic myocardial lesions, usually large infarcts or scars that involved the subendocardial portions of the basilar half of the ventricular septum.

**Intraventricular Conduction Abnormalities**

These usually refer to complete or incomplete bundle-branch blocks or delays that may reflect abnormality of the right bundle branch, one or both fascicles of the left bundle branch, or the regions of myocardium supplied by these conduction fibers. In hearts that manifested these problems during life, morphologic lesions may not be present in the A-V conduction system or those found may not be correlated clearly with the electrophysiologic abnormality. Lesions that can be related most often to delayed intraventricular conduction usually involve the ventricular septum and are ischemic. In many cases, the morphologic lesions are located in areas in which specific conducting pathways are not known or which should have only distal ramifications of a bundle branch.

*Figure 3*

*Infarction of left bundle branch (L) in association with acute myocardial infarction. Darker fibers to the right are infarcted ordinary ventricular myocardium. (Hematoxylin and eosin, x 120.)*

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Anomalous Atrioventricular Excitation

Anomalous atrioventricular excitation ("preexcitation"), particularly when associated with an atrial arrhythmia constituting the Wolff-Parkinson-White (W-P-W) syndrome, may be due, in some cases, to anomalous muscle bundles (Kent fibers) connecting atrial and ventricular muscle and forming a possible pathway for conduction in addition to the normal common bundle of His.26, 27 Such accessory bundles are usually present about the right A-V junction, but apparently may be located also in the left A-V junction. Theoretically, the paraspecific fibers of Mahaim28 that are present in some hearts and connect the common bundle or the bundle branches near their origins to the immediately subjacent working ventricular septal fibers might serve as a means for anomalous excitation, but this is difficult to ascertain because such fibers commonly are found in hearts in which electrocardiograms have been normal. Additionally, atrial fibers described by James14, 29 that "bypass" the bulk of the A-V node and may insert into the bundle of His possibly contribute to the genesis of the W-P-W syndrome in some instances;30 the existence of such fibers has not been confirmed definitely.31 In rare instances, detailed morphologic studies have failed to demonstrate any accessory bundle apart from the normal A-V conduction system in the W-P-W syndrome (Pongpanich B, Burchell HB, Titus JL: Unpublished data). Only a relatively small number of instances of W-P-W syndrome have been adequately studied both morphologically and electrophysiologically, so complete anatomic conclusions are uncertain.

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