Functional Block in the Intraventricular Conduction System

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WHEN a normal impulse of supraventricular origin arrives at the His bundle, the entire specialized conduction system has had ample time to recover from the preceding response; the activation sequence of the ventricles and the resulting QRS complex are "normal." When a lesion permanently damages one of the major divisions of the common bundle, the ventricular activation sequence is permanently abnormal. When the electrocardiographic pattern of bundle-branch block is intermittent or occasional, and is dependent upon the preceding cycle length, the cause is clearly not a destructive lesion of the conducting system; the block is "functional." Perhaps the most common situation in which functional block occurs is in atrial fibrillation; the pattern of irregular transmission across the atrioventricular (A-V) node provides the occasional opportunity for early activation of the His bundle after a relatively long preceding cycle. This combination of events often results in aberrant ventricular conduction, for reasons which we shall demonstrate.

Like any biologic system, cardiac tissue is nonhomogeneous. It is heterogeneous in the sense that some cells are specialized for impulse generation, some for impulse conduction, and the bulk of the tissue for contractility. Within any of these separate categories, the tissues are nonhomogeneous; variations in excitability, conductivity, refractory period duration, geometry, membrane potential, ionic environment, contractility, and even proximity to nerve endings, occur from cell to cell and region to region.

Variations of action potential duration (APD) and the related property of refractory period (RP), are of primary importance in any consideration of functional block. In a tissue which is absolutely homogeneous with respect to these properties an impulse must invade the whole tissue no matter how early in the relatively refractory period it is generated. In a nonhomogeneous conducting system, an early impulse may encounter some areas which are still refractory while others have recovered sufficiently to support a propagated response; functional block is the result.

Cardiac muscle cells are connected through low resistance junctions with their functional neighbors. Variations in APD between immediate neighbors are therefore minimal; the electrotonic current flow between functionally connected units tends to synchronize their repolarization. Minimal variations between immediate neighbors in a functionally-connected cable do not, however, guarantee synchronization from one cable to another; the right and left bundle branches, once these have diverged anatomically from the common bundle, are electrotonically remote from each other. At a few millimeters beyond the separation, the electrotonic interactions are insignificant. As a result, the APD and RP of contiguous cells in the right bundle branch may be nearly identical, but they may be quite significantly different from the functionally remote cells of the left bundle. The functional differences are often remarkably gross even in normal cardiac tissue. A premature impulse of supraventricular origin which arrives at the bundle of His before one of its divisions has recovered will be delayed in transit, or may be totally blocked in that division.

Functional block in man occurs more commonly in the right than in the left bundle;
it can be demonstrated almost at will in the normal dog heart. To verify that functional block is in fact the result of differences in the duration of the RP, it is necessary to assess the RP of the successive tissues in the A-V transmission system. This can be done by determining, at each of several levels, the minimal attainable interval between a basic response (resulting from rhythmic driving stimuli) and a premature response.

For the A-V node itself, the functional RP can be estimated, in dog and in man, by recording His bundle responses to impulses propagated from the atrium. If premature atrial contractions (A₂) are induced at various intervals after a basic response (A₁), a curve relating "output" intervals (H₁-H₂) to "input" intervals will define the functional RP of the A-V node. The briefest attainable interval between two responses of the His bundle, both propagated from the atrium, is the functional refractory period of the A-V node.

The RP of the His bundle can be assessed in the experimental animal by determining the earliest premature stimulus applied directly to the bundle, which will evoke a response. The earliest premature response of the His bundle which can enter the bundle branches provides, in turn, an estimate of the functional RP of these components of the conduction system.

As in all cardiac tissues, the refractory periods of the A-V node, the His bundle, and the bundle branches are frequency-dependent: the RP diminishes as the cycle length decreases (i.e., as the rate increases). As illustrated in figure 1, derived from an experiment on the dog heart, the curves relating RP to basic cycle length are neither identical nor parallel. At the slowest driving rate the RP of the right bundle branch (RBB) in this experiment was conspicuously longer than that of the His bundle, and also longer than that of the A-V node. Accordingly, when the basic cycle length was greater than 500 msec, the response to a premature atrial stimulus could traverse the A-V node and the bundle of His, arriving at the right bundle before the expiration of its RP. The pattern of RBBB would therefore be manifest. At shorter basic cycle lengths (less than 500 msec), the effective RP of the right bundle was less than that of the A-V node. The earliest premature atrial response that could pass through the node would now fail to reach the His bundle before the expiration of refractoriness in the right bundle; at the higher basic frequency, the activation sequence of the ventricles would be normal.

The diagrams of figure 2 illustrate how functional block in the right bundle would occur in a heart exhibiting the relationships plotted in figure 1. In the uppermost schema, the shaded areas represent the duration of the RP in the A-V node (AVN), the His bundle, and the right bundle (RBB), following a basic cycle of 700 msec. A premature response initiated in the atrium at A₂ could arrive at the His bundle 230 msec after A₁, but would be blocked by refractoriness in the RBB. The activation of the ventricles would be accomplished by way of the LBB, and the electrical response recorded from electrodes attached to the epicardial surface of the right and left ventricles would be inverted, as indicated by the schematic "electrogram."

The effect of acceleration of the basic heart rate is illustrated in the lowermost diagram of

![Figure 1](http://circ.ahajournals.org/)

*Figure 1*

Refractory periods of A-V node, His bundle, and RBB as a function of basic cycle length. (Reprinted from Circ Res 16: 261, 1965, by permission.)
FUNCTIONAL BLOCK

figure 2. Although the RP's of all three layers are abbreviated, the RBB is most affected; accordingly, the earliest premature atrial response could not reach the His bundle earlier than 217 msec after A1, or about 14 msec after recovery of conductivity in the RBB. This is in accordance with clinical experience: “aberrant” intraventricular conduction is more likely to occur when an early premature atrial beat follows a long preceding cycle.¹ ⁴

At basic cycle lengths below 500 msec, the RP of the right bundle, although briefer than that of the A-V node, still exceeded that of the His bundle and the left bundle branch. A premature response initiated by stimulation of the His bundle would still evoke the pattern of right bundle-branch block. At a cycle length of 200 msec, however, the curves relating RP to cycle length in the His bundle and its right branch converge. Any premature response initiated in the His bundle at a basic cycle length of 200 msec or less would again be normally distributed to both bundle branches.

In any heart exhibiting the relationships shown in figure 1, there are three phases related to the basic frequency: at slow frequencies, an early premature atrial response will yield the bundle-branch pattern; at intermediate frequencies, a premature response in the His bundle, but not in the atria, will expose functional block; at high basic frequencies, the pattern of block will not occur even with a premature response of the His bundle.

The results exemplified by figures 1 and 2 were commonly observed in experiments on the dog heart, and there is little reason to doubt that similar relationships occur in the normal human heart. Occasionally, however, the left bundle branch, or one of its divisions,

Figure 2

Diagrams illustrating the effect of heart rate and of vagal stimulation on refractory periods in the A-V transmission system. The sketch in the right upper corner indicates the placement of stimulating electrodes on the right atrium (S), and recording electrodes on right (R) and left (L) ventricular surfaces. The shaded areas in the diagram represent the functional refractory periods of A-V node (AVN), His bundle (HIS), and right bundle branch (RBB).

In the upper diagram, a premature atrial response (A2) is blocked at the origin of the RBB; the pattern of bundle-branch block is schematically represented below. In the middle diagram, at the same basic cycle length, vagal stimulation prolongs the FRP of the node, but does not alter that of the RBB. In the lower diagram at a faster basic heart rate, the FRP of the node is abbreviated, but it exceeds that of the RBB.
has a longer RP than the right. Sometimes the RP of the His bundle is briefer than either of its branches, in which case functional bilateral bundle-branch block can be demonstrated. His bundle responses which occasionally fail to propagate to the ventricles have been recorded in man. The differential effect of frequency upon the RPs of the A-V transmission system suggest that functional block might be exposed by vagal slowing of the heart. The reverse is true. Cholinergic influences on the contractility of the ventricles have been demonstrated in a number of situations, but the electrical properties of the intraventricular conduction system appear to be essentially immune to vagal effects. In the dog, at least, no modification of the RP, APD, excitability, or conduction velocity of the His bundle or its branches accompanies vagal stimulation. The A-V node, on the other hand, is profoundly responsive to cholinergic influence. Vagal stimulation greatly depresses nodal conductivity, and prolongs the A-V nodal RP. In the diagram of figure 1, this would be equivalent to displacing the A-V node curve upward, without altering the position of the other curves. Because of these effects, a premature atrial response would be delayed in transit by vagal stimulation. It would no longer arrive in the His bundle before the end of the RP of the right bundle, and the pattern of bundle-branch block would not occur. Premature responses initiated in the His bundle, unaffected by vagal stimulation, would still encounter a refractory right bundle; this relationship has been clearly defined in the experimental preparation and is illustrated in the middle diagram of figure 2. At a slow driving rate (basic cycle 700 msec), vagal stimulation prolonged the RP of the A-V node from 230 to 250 msec, but did not influence the His bundle or its branches.

Adrenergic stimulation, unlike vagal stimulation, exerts clearly defined effects on the intraventricular conduction system. Sympathetic nerve stimulation, or administration of catecholamines, abbreviates the RP of the A-V node, but also affects the His-Purkinje system. In the experimental preparation, the results depend upon the relative effects of the adrenergic mediator on the several components of the A-V transmission system. If the effect on the A-V node exceeds the effect on the right bundle (i.e., if the A-V node curve of figure 1 is displaced downwards relative to the other curves), then an atrial premature beat will expose the RBBB pattern at a briefer basic cycle length.

If, on the other hand, the adrenergic influence on the right bundle is relatively greater, the frequency range within which atrial premature beats can result in functional block will be shifted to the right. In the experimental animal, infusion of epinephrine shifts the range to the right, i.e., functional block is still demonstrable, but only at slower frequencies; but because adrenergic stimulation also increases the frequency of the sinus node, it may no longer be possible to achieve the lower frequency. Because the RP of the bundle branches is abbreviated more than that of the His bundle, block in the RBB may no longer be demonstrable even with premature excitation of the common bundle. With substantial adrenergic influence, no phase of functional block can be demonstrated at all.

Block in the right bundle branch, if due to a relatively longer RP, should be unidirectional. An impulse which reaches the His bundle at a time when its branches are dissociated (with respect to refractoriness) must engage the left bundle system, and will eventually enter the right bundle through its peripheral ramifications. Retrograde excitation of the right bundle will occur. If the left bundle branch should have a longer refractory period than the right, a similar sequence of events would occur, but in the converse direction. Both of these possibilities have been demonstrated in the dog; and Wellens and Durrer have described a clinical case in which retrograde activation of the left bundle was probably responsible for aberrant conduction during an episode of supraventricular tachycardia.

There are two important consequences of retrograde activation of the previously blocked bundle. First, if the time required for
FUNCTIONAL BLOCK

Figure 3

Retrograde activation of the right bundle branch. The diagrams represent the sequence of events in the His bundle and its branches. Shaded areas represent refractory periods of His bundle (H) and right bundle branch (B). Block of the second response occurs because of refractoriness in the RBB; propagation to the ventricles succeeds over the LBB (broken arrow), and retrograde activation of the RBB follows (solid arrow). In the upper diagram, the retrograde response causes re-excitation of the His bundle, which can proceed to the atria as an echo, and can also re-enter the LBB. In the lower diagram, retrograde activation of the RBB establishes the conditions for repetitive block. This would be self-sustained as long as the rhythm is uninterrupted, but it could be terminated by a ventricular premature stimulus (S), which would pre-excite the RBB and permit its recovery prior to arrival of the next supraventricular response.

The electrocardiogram was recorded in a patient with atrial flutter and variable A-V block (lead V1). Following a relatively long R-R interval, an episode of repetitive bundle-branch block occurs; the conditions are similar to those in the diagram above. (Reproduced from Arch Intern Med [Chicago] 124: 101, 1969, by permission.)

an excitation wave to pass down the left bundle, cross over to the right ventricle, and return to the His bundle over the RBB exceeds the duration of refractoriness in the His bundle, reentry can occur, as illustrated in the upper part of figure 3. Here, we have diagrammed the events as they would occur within the His-Purkinje system (sketch in the right upper corner). The second impulse, arriving at the His bundle before expiration of the RP in the RBB, would proceed to the ventricles over the LBB (broken line), cross over to the RBB and activate it from below (solid line and arrows). The total time for this passage, as indicated, clears the RP of the His bundle. Reactivation of the His bundle could lead to retrograde activation of the atria (an atrial “echo”). If the LBB were also recovered, the reentrant circuit could be maintained as one possible mechanism of ventricular flutter. Single “echoes” of this form have been demonstrated. Attempts to initiate self-sustained flutter in the dog heart have not been successful, but the larger human heart might well sustain such a rhythm. Digitalis, which depresses conduction in the His-Purkinje system without prolonging its RP, could provide the necessary conditions.

A second consequence of retrograde activation of the RBB is illustrated in the lower schema of figure 3. In this diagram, the second response of the His bundle is shown to encounter a refractory RBB. Activation of the ventricles over the LBB (broken line) again causes retrograde excitation of the RBB (solid arrow). Because the two bundle branches have been “dephased,” the third response of the His bundle will also be blocked in the RBB. The pattern of bundle-branch block will then be maintained as long as the appropriate frequency persists. In the clinical situation, a paroxysm of supraventricular tachycardia, suddenly interrupting a basically slow sinus rhythm, could yield this pattern and lead to the attendant diagnostic problem of differentiating ventricular tachycardia from supraventricular tachycardia with block. The electrocardiogram at the bottom of figure 3, obtained from a patient with atrial flutter, illustrates this phenomenon.

Conclusions

Functional block may be exposed when a premature response of supraventricular or junctional origin encounters a refractory bundle branch. The disparity between the RP of the bundle branch (usually the right) and that of the A-V node is increased when the
basic heart rate is slow, but is diminished by both vagal and sympathetic influences. Retrograde activation of the blocked ramus may lead to reentrant excitation of the His bundle, may initiate an intraventricular circus-movement flutter, and can set up the conditions for repetitive bundle branch block.

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