The Gating Mechanism in the Distal Atrioventricular Conducting System

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An impulse propagating from the atrioventricular (A-V) node to ventricular muscle must traverse a length of conducting tissue consisting of the bundle of His, bundle branches, peripheral ramifications of the branches, and subendocardial Purkinje network. Electrophysiologic studies of the properties of these tissues have provided a body of information on the nature of normal and abnormal conduction, functional block, and the genesis of the arrhythmias.

Recording of transmembrane action potentials from single cells with intracellular microelectrodes has shown that the refractory period of individual cells after a depolarization is primarily related to the duration of the action potential—that is, to the time required to achieve a certain level of repolarization of the membrane¹ (fig. 1). The refractory period is prolonged as the duration of the action potential increases. It has further been demonstrated that action potential durations in the conduction system increase from the bundle branches distally.² An area of maximum action potential duration occurs about 2 mm proximal to the termination of Purkinje cells in muscle,³ and then the durations progressively decrease through a sequence of Purkinje cells, transitional cells, and muscle cells⁴ (fig. 2).

Figure 3 demonstrates the significance of the pattern of these changes. The drawing shown at the top represents a preparation of canine tissue composed of a single free-running false tendon between the right bundle branch and a segment of free-wall muscle. The graph demonstrates the direct relationship between action potential durations (plotted as the solid line) and local refractory periods (plotted as the dashed line). Thus, the area of maximum action potential duration is also the area having the longest local refractory period between the bundle branch and ventricular muscle. Premature impulses having coupling intervals (S₁-S₂ intervals) longer than the refractory period at the area of maximum action potential duration can be conducted unimpeded from the bundle branch to ventricular muscle or from ventricular muscle retrogradely to the bundle branch. In the example shown in figure 3, the refractory period at the area of maximum action potential duration was 290 msec. A premature impulse (S₂) applied 300 msec after the driven impulse (S₁) could therefore be conducted in either direction. However, when the S₁-S₂ interval was shorter than the refractory period at the area of maximum action potential duration, the premature impulse remained confined. That is, if it was applied to the bundle branch, it was conducted only through the tissue proximal to the area of maximum action potential duration. Or if it was applied to the free wall, it was conducted only through tissue distal to this area. Because of these functional properties, the area of maximum action potential duration has been referred to as a gate,⁵ with the intended implication that the gate is responsible for determining the minimum possible coupling interval between a driven impulse and a premature impulse conducted across these false tendons.
Transmembrane action potentials and refractory periods. (A) The resting potential of the quiescent cell, recorded when the membrane is impaled by the microelectrode, is \(-92\) mV. (B) Stimulation of tissue evokes an action potential. (C) A premature stimulus applied just before the membrane has repolarized sufficiently to respond evokes only a graded response. (D) A premature stimulus applied 10 msec later results in a regenerative response. The ability to achieve a regenerative response, i.e., absence of refractoriness is related primarily to the membrane potential at the time of arrival of the premature impulse. Thus, refractory period is a function of action potential duration. Vertical calibration = 20 mV; horizontal calibration = 50 msec.

The distal end of the canine A-V conducting system is composed of multiple branching false tendons and subendocardial tracts of Purkinje fibers. Mapping experiments have demonstrated that each of these distal bundles of conducting tissue has an area of maximum action potential duration which functions as a gate. Thus, the peripheral conducting system contains multiple distal gates (fig. 4).

In a preparation of the right bundle branch and its peripheral ramifications, the durations of the refractory periods at the multiple gates are identical, or nearly so, when the tissue is physiologically normal. This is also true for a preparation composed of a division of the left bundle branch and its peripheral ramifications. However, when the durations are compared in tissue from the right and left sides of the same heart, the refractory periods at the gates are usually longer in tissue obtained from the right than from the left.

Conduction of premature impulses can be studied in preparations of a bundle branch and its multiple ramifications terminating in large blocks of free wall muscle (fig. 5). When the bundle branch in such preparations is stimulated prematurely at a coupling interval less than the duration of the refractory periods at the distal gates, excitation of the proximal tissue by the premature impulse will occur, but the impulse will remain confined proximal to the gates. When the tissue distal to the gates is stimulated at a similar coupling interval, confinement of the premature impulse in the distal tissue will occur, as long as conduction velocities are not depressed. It has been demonstrated in tissue blocks up to approximately 9 cm² in surface area that appropriately timed premature impulses may remain confined in the distal tissue and not be able to find a route back into the proximal conducting system through any of the multiple false tendons.

The gating mechanism in preparations composed of multiple false tendons (as in fig. 5) may be modified, or broken down, by certain interventions. The effect of local application of potassium chloride to the region

![Figure 1](http://circ.ahajournals.org/attachment/doi:10.1161/01.CIR.43.2.956/fig-1)

![Figure 2](http://circ.ahajournals.org/attachment/doi:10.1161/01.CIR.43.2.956/fig-2)
GATING MECHANISM

Figure 3

The gate. The preparation (top) consists of a single free-running false tendon from the right bundle branch (RBB) to the free wall of the right ventricle (FWRV). The graph demonstrates that the changes in action potential duration (APD) along the length of the preparation are paralleled by changes in local refractory period (RP). There is a progressive increase in APD and RP to maximum of 290 msec and a sharp fall as the conduction fibers approach the free wall. The functional refractory period of the preparation is the minimum coupling interval ($S_1 - S_2$) which can result in excitation of the FW by the premature impulse applied to the RBB. The duration of the functional refractory period is determined by the duration of the local refractory period at the area of maximum action potential duration, or gate. (See text for further details.)

of the gate of one of the false tendons is particularly dramatic. The potassium ions, in appropriate concentration, will shorten the action potential duration and refractory period at the gate of the one false tendon, without affecting the others. This causes a "short-circuit," resulting in a shortened functional refractory period of the preparation. That is, the minimum coupling interval for conduction of a premature impulse across the conducting system to ventricular muscle is shortened. In addition, since the premature impulse reaches the ventricular muscle by way of a single branch of conducting tissue rather than the normal multiple branches, aberrant conduction of the impulse through the muscle must occur. In some preparations, "short-circuiting" of this type was seen spontaneously. It was sometimes possible to identify single abnormal false tendons, having shortened refractory periods at their gates, in such preparations. Cutting the abnormal false tendons restored the gating mechanism in these preparations.

The response of the distal gates to rapid rates of stimulation is somewhat different from the response to premature stimulation. While
parts of the conducting system were obliterated completely at very rapid rates, and thus the gating mechanism was obliterated.

Clinical Considerations

The assignment of clinical significance to a gating mechanism in the intact human heart is quite a different matter from the demonstration of the gating mechanism in isolated segments of the canine conducting system. This gap has not yet been bridged. However, a large body of information from both clinical and animal observations suggests that a mechanism of this type is indeed operative and significant in human physiology. The following speculations are offered for consideration.

Aberrant Ventricular Conduction of Premature Supraventricular Impulses

Aberrant conduction of premature impulses arising above the ventricles usually assumes a right bundle-branch block configuration, with or without associated left hemiblock. This clinical observation had led to the assumption, later verified experimentally, that the refractory period of the right system is longer than that of the left system. The lack of the ability to identify comparable areas in tissue from the two sides prevented the identification of a cellular basis for these apparent differences. The region of the gate now seems to permit valid comparisons, however, and these comparisons show that refractoriness at the gates of the right system usually outlasts that of the left.

Aberrant Ventricular Conduction During Supraventricular Tachycardia

Aberrant conduction in this setting follows no fixed pattern from patient to patient. The degree of aberration is variable, as is the sequence of depolarization. This is consistent with the observations of the response of canine tissue preparations to rapid rates of stimulation. At appropriate rates of stimulation, some false tendons may develop delay of conduction across the gates, while normal conduction occurs across others. Thus, the sequence of depolarization of the muscle mass

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would be altered. Extrapolating this to human electrocardiography, patterns of altered QRS morphology would occur.

**Bidirectional Tachycardia**

The occurrence of 2:1 conduction across the gates of some false tendons, concomitantly with 1:1 conduction across others, at rapid rates of stimulation suggests a possible mechanism for bidirectional tachycardia. In such a setting, the sequence of ventricular muscle depolarization would alternate and thus QRS morphology would alternate. This seems to be a reasonable mechanism by which bidirectional tachycardia may occur, although it clearly may not be the only mechanism.\(^{10}\)

**Concealed Extrasystoles**

This is an unusual disturbance in which premature impulses arise distal to the A-V node and presumably proximal to the gates. The coupling interval between the premature impulse and the preceding sinus beat is such that the premature impulse is blocked both proximally and distally—thus it is concealed in the A-V junction. Its presence is usually indicated by partial or complete block of the next normal impulse due to refractoriness caused by the premature impulse. Concealed extrasystoles may be considered confined impulses in the sense discussed earlier.

**Site of Origin of “Premature Ventricular Beats”**

Electrocardiographic complexes having the morphology of premature ventricular beats are assumed to arise distal to the bifurcation of the bundle of His. When their site of origin is proximal to the gates, the coupling interval should be limited by the duration of the refractory period at the gates. In normal hearts, this limit will preclude the premature impulse falling in the vulnerable period of the ventricular muscle because the refractory periods at the gates are much longer than the repolarization time of ventricular muscle. Focal disease, however, may shorten the refractory periods at some gates, and permit coupling intervals of much shorter duration—as might also be possible when impulses arise distal to the gates. Furthermore, premature impulses arising at the A-V node or bundle of His, and having short coupling intervals which would cause them to be concealed extrasystoles in normal tissue, may find pathways of conduction into ventricular muscle in the presence of focal disease. Such impulses would necessarily be aberrantly conducted, and the sequence of ventricular depolarization would depend upon the site(s) at which the impulses entered the myocardium. Despite the fact that they arise proximal to the bifurcation of the bundle of His, the electrocardiographic

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**Figure 5**

Preparation composed of multiple false tendons for studying confinement of impulses. The local refractory periods at the multiple distal gates are in the range of 220 to 222 msec at a basic cycle length of 600 msec. Premature impulses applied to the free wall or to the bundle branch at coupling intervals less than this value (e.g., 210 msec) remain confined to one side of the multiple gates or the other. Interventions which decrease the refractory period at one gate, while not affecting the others (see text), can cause a breakdown of confinement of premature impulses.
features of these impulses would be indistinguishable from premature ventricular beats and the coupling intervals could be quite short.

**References**

1. **Hoffman BF, Kao CY, Suckling EE**: Refractoriness in cardiac muscle. Amer J Physiol 190: 473, 1957
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