

Electrophysiology of Concealed Conduction*

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The phenomenon of cardiac excitation penetrating conducting tissue but failing to traverse it completely was called "concealed conduction" by Langendorf in 1948 (1). Earlier electrocardiographers had clearly demonstrated its occurrence. Since the introduction of this term in 1948, the concept of concealed atrioventricular (A-V) conduction has been invoked to explain many complex arrhythmias and has found wide application in clinical electrocardiography. In the clinical electrocardiogram, penetration of impulses that do not emerge from the A-V conduction system (concealed conduction) can be inferred from their influence on subsequent events such as 1) delay of conduction of a succeeding propagated response, 2) block of a succeeding atrial impulse which occurs at a time when the transmission system should have been excitable, 3) delay of the expected discharge of a junctional pacemaker, or 4) in some cases, facilitation or acceleration of a succeeding impulse.

In many early experimental, and most clinical investigations, the A-V node was thought to be the location in which antegrade as well as retrograde incomplete penetration of an impulse resulted in an effect on subsequent events. This was largely based upon the fact that the ECG only permits analysis of atrial and ventricular activity; electrocardiographers, therefore, considered the A-V node as the location where the atrial input was modulated to produce the resultant ventricular response pattern. Further refinements in microelectrode tech-

niques have allowed more precise localization of the area of concealed conduction as well as some insight into the mechanisms by which a nonpropagated impulse can block, delay, or accelerate subsequent conduction within the A-V node or His-Purkinje conduction systems, or both.

Several figures are presented here to demonstrate the different mechanisms of concealed conduction in which microelectrodes were used to record from the A-V node and His-Purkinje system together with simultaneously recorded atrial and ventricular electrograms. Of course, in a standard ECG, one would not be able to determine what was occurring within the specialized A-V conduction system, since only the information provided by the atrial and ventricular electrograms (P wave and QRS complex) would be available for interpretation.

Delays and block of antegrade conduction by premature beats are the most common examples of concealed conduction in man. The first illustration, recorded in an isolated rabbit atrioventricular preparation, shows an example of a premature ventricular contraction (PVC) causing complete A-V block of the subsequent sinus beat. This is due to the PVC partially penetrating the A-V conduction system. In figure 1, the upper trace is an electrogram recorded from the right atrium (RA), and the lower trace (RV) is an electrogram recorded from the right ventricular muscle. Simultaneously, transmembrane action potentials were recorded from a single A-V nodal fiber (AVN, second trace) and a single right bundle branch fiber (RBB, third trace). Time dots denote 100 msec intervals. The first atrial response was propagated from the atrium, through the A-V node, bundle of His (not recorded from), right bundle branch fibers and then to the

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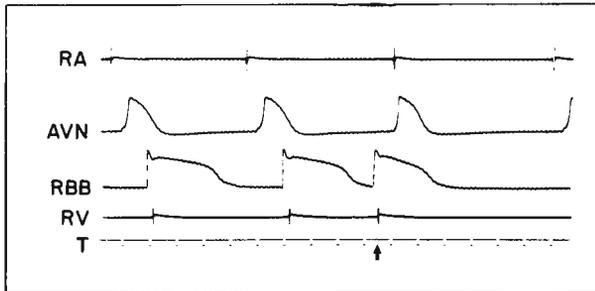


Fig. 1—A concealed ventricular premature response causing block of a normally conducted atrial activation. Bipolar electrograms were recorded in the isolated rabbit heart from the right atrium (RA) and right ventricles (RV), together with transmembrane potentials from the A-V node (AVN) and right bundle branch (RBB). The timing pulses (T) denote 100 msec. At the arrow, the preparation was prematurely activated by electrical stimulation through electrodes placed over the region of the right bundle branch. The third sinus response was blocked as a result of the premature ventricular response prematurely invading the A-V node. (Reproduced, with modifications, by permission of the *American Journal of Cardiology*, 28:409, 1971.)

ventricular myocardium. Following the first two normally conducted atrial beats, a premature ventricular contraction develops (third ventricular electrogram in the RV tracing). Retrograde conduction of this PVC is blocked as clearly shown by the failure of the third atrial response resulting in a ventricular response at the expected time. In this instance, the PVC was conducted retrograde and excited the A-V nodal fiber earlier than conduction from the atrium would have been expected to excite the impaled A-V nodal fiber. Note that the time between the third atrial to A-V nodal response is clearly shorter than either the normally conducted first or second beats.

Another important and commonly observed clinical example of the role that atrial rate plays in determining the occurrence and frequency of concealment is the fact that the ventricular response is more rapid during atrial flutter than during atrial fibrillation. In figure 2, extracellular atrial (RA) and ventricular electrograms (RV) were recorded simultaneously with transmembrane action potentials from a bundle of His fiber (BH) and right bundle branch fiber (RBB) in an isolated rabbit atrioventricular preparation during atrial flutter (fig. 2A) and during atrial fibrillation (fig. 2B). During atrial flutter, every other atrial response is blocked within the A-V node, as demonstrated by the absence of all-or-none bundle of His, right bundle branch, and

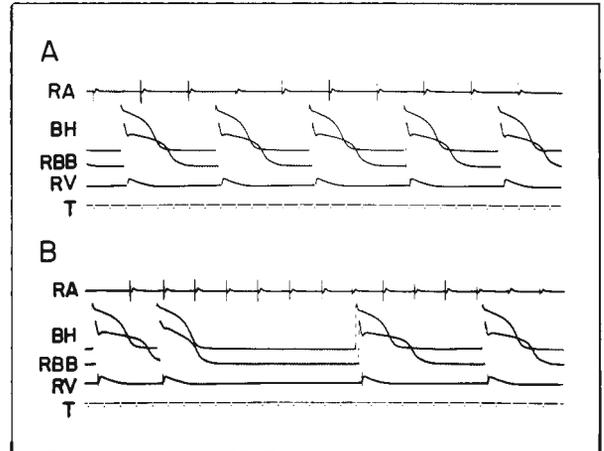


Fig. 2—Atrioventricular conduction during atrial flutter and fibrillation in the isolated rabbit heart. Bipolar electrograms were recorded from the right atrium (RA) and right ventricle (RV) together with transmembrane potentials from the bundle of His (BH) and right bundle branch (RBB). The timing pulses (T) denote 100 msec. A: the isolated atrioventricular preparation is driven from the atria at a cycle length of 300 msec. B: the atria are stimulated rapidly to simulate atrial fibrillation. (Reproduced, with modifications, by permission of the *American Journal of Cardiology*, 28:408, 1971.)

ventricular responses. During atrial fibrillation provoked by very rapid electrical stimulation of the atria, the ventricular response interval in the RV electrogram is slower and more irregular than during atrial flutter. During the long interval between the second and third ventricular responses (RV-RV), 5 atrial responses occur in the atrial electrogram which are not conducted to the bundle of His and the right bundle branch. All of the ventricular responses are delayed to various degrees within the A-V node. The degree of consecutive concealment governs the irregularity of the ventricular rate during atrial fibrillation.

Occasionally during atrial fibrillation, aberrant QRS complexes are recorded. This has been thought to result in some instances from escape of subsidiary pacemaker. However, we have observed that aberrant QRS complexes may result from block or concealed conduction within the right bundle branch. Similar findings have been reported in man during His bundle recordings. We believe, therefore, that variation in degree of concealed conduction within the A-V node is the most common cause of the irregular, slow R-R periods observed during atrial fibrillation.

A third manifestation of concealed conduction is that a partially conducted impulse may not only affect subsequent conduction, but may also disturb impulse formation in a subsidiary pacemaker. In figure 3, a subsidiary pacemaker was present either in the lower A-V node or bundle of His fiber (BH, second trace). Pacemaker activity, also referred to as diastolic depolarization or phase 4 depolarization, can be noted in the pacemaker fiber labelled BH by the fact that upon repolarization, the membrane potential progressively depolarizes until the threshold potential is reached and an action potential develops. The fact that the BH fiber drives both the atrium and ventricles can be noted by the evocation of its action potential prior to atrial, right bundle branch, or ventricular activity. Thus retrograde conduction to the atria and antegrade conduction to the ventricles occurs in the first two beats. Following the second atrial response, a premature atrial beat develops (third RA electrograms). This atrial response is conducted antegrade to the impaled BH fiber and prematurely fires a subsidiary pacemaker. Concealment is complete since the premature atrial beat fails to be propagated beyond the subsidiary pacemaker, that is, no action potentials occur at either the RBB or RV recording sites. In this instance, the development of the next expected discharge of the subsidiary BH pacemaker was delayed by 58 msec due to the premature discharge of the BH pacemaker.

The fourth example of concealed conduction is one where partial penetration of the A-V con-

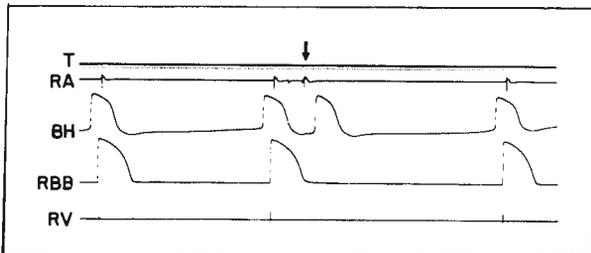


Fig. 3—Premature atrial activation causing “resetting” of a junctional pacemaker. Bipolar electrograms were recorded in the isolated rabbit heart from the right atrium (RA) and right ventricle (RV), together with transmembrane potentials from the bundle of His (BH) and right bundle branch (RBB). The timing pulses (T) denote 10 msec. At the arrow, the preparation was prematurely activated by electrical stimulation through electrodes placed on the right atrium. (Reproduced, with modifications, by permission of the *American Journal of Cardiology*, 28:410, 1971.)

duction system by a cardiac impulse results in the facilitation or acceleration of a succeeding impulse. Figure 4 is an example, also recorded in the isolated rabbit atrioventricular preparation, in which facilitation of conduction occurs; this figure could also be considered an example of supernormal conduction. Electrograms were recorded from the right atrium and ventricles (RA and RV) simultaneously with transmembrane action potentials recorded from the A-V node (AVN) and right bundle branch (RBB). In figure 4A, the atria were driven at a constant rate, and basic atrial responses were conducted antegrade to the ventricles as shown by the first 2 atrial beats. The third atrial response was evoked prematurely, and it can be seen that the

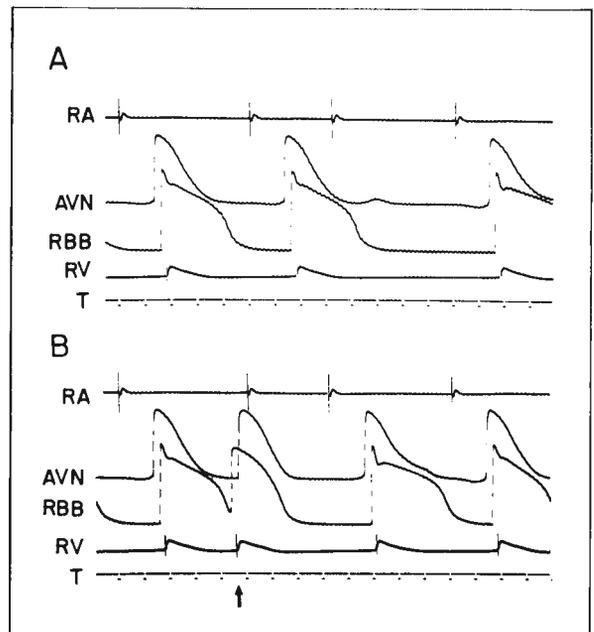


Fig. 4—A concealed ventricular premature activation causing conduction of a previously blocked atrial activation. Bipolar electrograms were recorded in the isolated rabbit heart from the right atrium (RA) and right ventricle (RV), together with transmembrane potentials from the A-V node (AVN) and right bundle branch (RBB). The timing pulses (T) denote 100 msec. The sequence of atrial activation was identical in A and B. In A, note that the third atrial response was not conducted to the ventricles but was blocked within the A-V node with only a local nonpropagated response occurring (unpublished data, Moore and Spear). At the arrow in B, the preparation was prematurely activated by electrical stimulation through electrodes placed over the region of the right bundle branch. (Reproduced, with modifications, by permission of the *American Journal of Cardiology*, 28:412, 1971.)

impulse caused a nonpropagated local response in the impaled A-V nodal cell which failed to be conducted to the bundle branch and ventricles. Therefore, it was concealed somewhere within the A-V node. In figure 4B, the atria were driven at the identical cycle lengths as those in figure 4A, but a premature ventricular response (at the arrow) was evoked prior to the expected arrival of the ventricular response which would have resulted from antegrade conduction of the second atrial response. This ectopic ventricular response was conducted retrograde to the impaled A-V nodal cell. The second atrial complex was evoked at precisely the same time as in figure 4A; however, conduction of this atrial response to the impaled A-V nodal cell was blocked since the impaled A-V nodal fiber was excited by the premature ventricular response. Therefore, bidirectional collision occurred somewhere above the impaled A-V nodal fiber. The premature atrial response (third atrial RA response) occurred at identical times in panels A and B. However, the response in figure 4B, unlike that in 4A, was conducted to the ventricles because the concealed premature ventricular response facilitated conduction of the premature atrial response by peeling back a refractory barrier within the A-V node. Figure 4B, then, is an example of concealed conduction which resulted in the development of facilitated or "supernormal" conduction.

In summary, I have tried to present examples and mechanisms for concealed conduction. Illustrations recorded in the isolated rabbit A-V preparation

were selected since this preparation permits one to obtain not only the information available in the ECG (atrial and ventricular activity), but also to see what the A-V node, bundle of His, bundle branches, and Purkinje system are doing during concealment. The development of the catheter electrode technique for recording from the bundle of His now permits validation of phenomena occurring within different regions of the A-V conduction system in man.

Thus, concealed conduction of atrial, junctional, and ventricular impulses can result in delay or block in the conduction of a subsequent impulse, delay in the expected discharge of a junctional pacemaker or, occasionally, facilitation or acceleration in conduction of a subsequent impulse. Concealed conduction is also involved in producing the irregular ventricular response during atrial fibrillation.

REFERENCES

1. LANGENDORF, R. Concealed A-V conduction: the effect of blocked impulses on the formation and conduction of subsequent impulses. *Amer. Heart J.* 35:542-552, 1948.
2. LANGENDORF, R. AND PICK, A. Concealed conduction: further evaluation of a fundamental aspect of propagation of the cardiac impulse. *Circulation* 13:381-399, 1956.
3. MOORE, E. N., KNOEBEL, S. B., AND SPEAR, J. F. Concealed conduction. *Amer. J. Cardiol.* 28:406-413, 1971.