Microelectrode Studies on Retrograde Concealment of Multiple Premature Ventricular Responses

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ABSTRACT

Retrograde concealment of serially evoked premature ventricular responses was studied in an isolated preparation of rabbit atria and ventricles. Transmembrane potentials were recorded simultaneously from single cells within the atrioventricular (A-V) node and ventricular specialized conduction system, together with atrial and ventricular bipolar electrograms. In these experiments, retrograde conduction delays and block occurred within the ventricles, between ventricular muscle and the Purkinje-bundle branch system, between the bundle branch and His bundle, and within the A-V node.

The ventricular specialized conduction system was found to be a major location of retrograde conduction delays and block. Many examples of retrograde concealment and block, previously interpreted as due to A-V nodal mechanisms, almost certainly result instead from mechanisms operating within the ventricular specialized conduction system.

Antegrade and retrograde block of sequentially evoked premature responses occurred within the A-V node. Reentry "echo" responses occurred in many experiments following premature stimulation of the atria, the ventricles, or both.

ADDITIONAL KEY WORDS ventricular specialized conduction system cardiac arrhythmias ventricular extrasystoles A-V node retrograde delay reentrant echo responses retrograde block

Langendorf introduced the term "concealed conduction" to describe the effects of partial penetration of premature atrial and ventricular impulses into the atrioventricular (A-V) junction upon subsequent A-V conduction (1). The concealment of single and multiple premature atrial response has been extensively investigated in both clinical (2-4) and experimental (5-9) studies. Although ventricular premature systoles occur more frequently in electrocardiograms than do atrial (2), relatively few experimental investigations on retrograde concealment of premature ventricular responses have been reported. Langendorf and Pick (3) have observed that a premature ventricular response can prolong A-V conduction of a subsequent atrial beat, and Langendorf and co-workers (4) found that a spontaneous A-V junctional pacemaker was disturbed by retrograde conduction of ventricular impulses. These observations constitute indirect evidence for retrograde concealment. In the elaborate experimental studies of Moe and associates (5) on retrograde conduction of ventricular premature responses, it was possible in only a few instances to obtain direct evidence for retrograde concealment. Since only atrial and ventricular electrograms were recorded in these experiments (5), it was impossible except by inference to define the site of retrograde delay or block.

In many experimental and most clinical investigations, the A-V node has been thought to be the location for both antegrade and retrograde concealment. Premature His bundle
Concealed retrograde conduction

Responses have been shown to be concealed within the A-V node in studies employing intracellular (10) and extracellular (11) recording techniques. However, microelectrode experiments demonstrating concealment within the A-V node of premature responses originating in ventricular myocardium are lacking. That retrograde concealment probably occurs also within the ventricular specialized conduction system has been indicated by microelectrode experiments on isolated canine ventricular muscle-Purkinje fiber preparations where conduction delays and block occurred between the ventricular muscle and Purkinje fibers (12, 13). The importance of the ventricular specialized conduction system in concealment of premature atrial responses has already been reported (9).

The present experiments were undertaken to study retrograde concealment of serially evoked premature ventricular responses in isolated preparations of rabbit heart. Transmembrane potentials were recorded simultaneously from cells within the A-V node and ventricular specialized conduction system together with atrial and ventricular bipolar electrograms. In these studies, retrograde conduction delays and block have been observed within the ventricles, between ventricular muscle and the Purkinje-bundle branch system, between the bundle branch and His bundle, and within the A-V node.

Methods

Rabbits weighing 2 to 4 lb were anesthetized intravenously with pentobarbital sodium (30 mg/kg), and the hearts rapidly removed and placed in oxygenated Tyrode's solution. The left atrium, left ventricular free wall, and right ventricular free wall were removed. The right atrium was opened from the atrioventricular groove through the cranial vena cava. The sinoatrial nodal region was always removed to permit slow heart rates and to prevent interference by spontaneous atrial responses. The preparation containing the right atrioventricular conduction system was pinned in a 40-ml muscle chamber and perfused with Tyrode's solution maintained at 37°C and equilibrated with 5 per cent carbon dioxide in oxygen. The left ventricular specialized conduction system was anoxic and probably nonfunctional, since the preparation was mounted with the left septum on the paraffin block. Conventional microelectrodes with resistances between 20 and 30 megohms were employed to record from single cardiac cells, using cathode followers without capacity neutralization. Voltage was electronically differentiated with respect to time, using a Tektronix model O operational amplifier. Bipolar surface electrograms were recorded from atrium and ventricles using Grass P-5 amplifiers. In a few cases, bipolar silver electrodes were inserted through the myocardial wall and used for recording or stimulating. Signals were displayed on a Tektronix 565 oscilloscope and photographed with a Grass C-4 camera. In a few experiments, signals were recorded on an Ampex FR 1300 tape recorder and played back for photography. All preparations were stimulated through bipolar silver electrodes at various frequencies, and premature stimuli were evoked through the same electrodes at regular intervals following six or more basic stimuli. The stimulator used digital circuitry that permitted up to 13 pulses to be programmed in variable sequences with intervals between premature stimuli variable in 0.1-msec steps (14). Functional refractory period was measured as the shortest interval between two propagated responses (13).

Results

Figure 1 was recorded while the ventricles were stimulated through bipolar electrodes. The basic ventricular cycle length (V1-V1 interval) was 400 msec. Retrograde conduction

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

RA = right atrial electrogram; BH = bundle of His transmembrane potentials; RBB = septal right bundle branch transmembrane potentials; RV = right ventricular electrogram; T = time intervals of 100 msec.

The preparation was driven from a right ventricular electrode. Basic ventricular and atrial responses are denoted as V1 and A1; premature responses are designated as V2, V3 and A2. Rapid deflections re-touched in all figures.
proceeded from the right ventricle (RV), through the Purkinje system (not shown) to the impaled right bundle branch (RBB) fiber, to the His bundle (BH) fiber, and then was conducted with considerable delay through the A-V node to the right atrium (RA). The conduction time through the A-V node is indicated by the interval between the responses at the His bundle and the right atrial recording site. This interval, of course, is longer than the actual A-V nodal conduction time by an amount equal to the conduction time between the His bundle recording site to A-V node plus that between the A-V node to the atrial recording site. Basic retrograde conduction time between the right ventricular and right atrial recording electrodes (V1-A1) at this heart rate was 104 msec. A premature ventricular response, V2, was evoked 138 msec after the eighth ventricular response. This V2 response was conducted with increased retrograde delay through the Purkinje system to the impaled RBB fiber, where it arrived while the RBB was still partially refractory because of incomplete repolarization from the previous basic (RBB1) response. Consequently, the rate of depolarization, duration of the action potential, and amplitude of the RBB2 response were decreased. Additional delays in retrograde conduction of the V2 response occurred between the RBB and bundle of His (RBB2-BH2 interval) and within the A-V node (BH2-RA2 interval).

In Figure 1, it also can be observed that block of retrograde conduction can occur between the ventricles and the Purkinje-bundle branch system. A second premature ventricular response, Vn, was evoked 121 msec after the V2 response. The Vn response, however, was blocked between the ventricles and the impaled RBB fiber because of the disparity in refractory periods of muscle and Purkinje tissue (12, 13). This is demonstrated in Figure 2 for an isolated rabbit preparation driven at a basic cycle length of 445 msec. The V1 and V2 responses were conducted to the impaled Purkinje fiber. The difference in action potential duration between the Purkinje fiber and ventricular muscle fiber in Figure 2 is greater than observed in most isolated canine preparations. The V2 response excited the Purkinje fiber during its relative refractory period; i.e., before repolarization was completed. The V3 response was blocked, however, because of the long functional refractory period of Purkinje fibers (12, 13). In the absence of the initial V2 responses in Figures 1 and 2, V3 was conducted to the Purkinje system with only a slight increase in conduction time.

In Figure 3, the V1 response was conducted...
over the Purkinje system to the impaled RBB fiber, to the His bundle, and through the A-V node to the right atrium. The V2 response, elicited 129 msec after the V1 response, was conducted with increased retrograde conduction time between the ventricles and RBB and between the RBB and His bundle. A further delay of 12 msec occurred within the A-V node, as indicated by the prolonged BH2-RA2 interval. The V3 response, evoked 125 msec after the V2 response, was conducted from the ventricle over the Purkinje system and reached the RBB while it was still partially refractory from the preceding RBB2 response. The action potential duration and functional refractory period of the bundle branch fiber shortened considerably with decrease in preceding cycle length; i.e., the RBB3 response is shorter than the RBB2, which in turn is shorter than the RBB1 response. The action potential duration and functional refractory period of the bundle of His fiber, however, did not shorten as much following decreases in preceding cycle length as did bundle branch and Purkinje fibers. This difference between RBB and His bundle fibers in the effect of preceding cycle length on functional refractory period enabled the RBB3 response in Figure 3 to be conducted to the impaled His bundle fiber while it was still refractory. Since at physiological heart rates, action potential durations of His bundle fibers are shorter than those of bundle branch fibers, one might not expect retrograde conduction block to occur in this region.

Figure 4 demonstrates retrograde block of a premature ventricular response within the A-V node. The ventricles were stimulated at a basic cycle length of 298 msec (V1-V1). The V1 response was conducted over the Purkinje system to the impaled RBB fiber, through the His bundle to the impaled A-V nodal fiber, and then to the right atrium. Activity from the V2 response, evoked 142 msec after the V1 response, reached the impaled RBB fiber during its relative refractory period. Consequently, the rate of rise (Bdv/dt), amplitude and duration of the premature RBB2 action potential were decreased. The RBB2 action potential still was capable of exciting the His bundle, and retrograde conduction occurred through the A-V node to the right atrium. In these experiments, the V2 response did not become blocked within the A-V node when the ventricles were driven at constant basic cycle lengths between 250 and 400 msec; retrograde block of V2 responses routinely occurred within the ventricular specialized conduction system or between the ventricles and Purkinje system. In Figure 4, a V3 response was evoked 154 msec after the V2 response. Activity from the V3 response reached the impaled RBB fiber at the end of its relative refractory period, the rate of rise (Bdv/dt) of the RBB3 response was nearly equal to that of the RBB2 response. However, activity from this RBB3 response, when conducted over the His bundle, elicited only a local nonpropagated response in the impaled A-V nodal fiber. This concealed response within the A-V node caused no increase in retrograde conduction time of the subsequent V4 response, which was evoked quite late and was clearly outside the relative refractory period of the ventricles and RBB.

In Figure 5, the right ventricle was driven at a basic cycle length of 298 msec (V1-V1) followed by three premature ventricular re-
FIGURE 5

RA = right atrial electrogram; AVN and RBB, AV nodal and septal right bundle branch transmembrane potentials. NdV/dt and BdV/dt = first derivatives of A-V nodal and right bundle branch transmembrane potentials, respectively. RV = right ventricular electrogram; V1, V2, V3, and A1 = premature responses.

FIGURE 6

RA = right atrial electrogram; BH = bundle of His transmembrane potentials; RBB = right bundle branch transmembrane potentials; RV = right ventricular electrogram; T = time intervals of 100 msec. Vt and A1 = basic responses; V2, V3, V4, V5, and A4 = premature responses.

responses, V2, V3, and V4. The V2 response was elicited 138 msec after the last V1 response. Activity from the V2 response arrived at the impaled RBB fiber during its relative refractory period and resulted in a double depolarization complex that arrived at the RBB fiber. The first RBB depolarization complex had a faster rate of depolarization than the second; the second depolarization occurred at a lower membrane potential and 36 msec after the initial RBB2 depolarization. The V3 response, which occurred 151 msec after the V2 response, arrived at the RBB fiber at the end of its relative refractory period; the RBB3 action potential exhibited nearly the control rate of depolarization (BdV/dt). However, activity from RBB3 failed to excite the impaled A-V nodal fiber. The subsequent premature V4 response was conducted back to the atria with a small increase in conduction time over the basic V-A transmission time. The small delay occurred between the RBB and atria since the V3-V4 interval equaled the RBB-V-A interval.

In Figure 6, 2:1 retrograde V-A block is demonstrated. The V2 response arrived at the RBB fiber during its relative refractory period and resulted in an RBB response of decreased amplitude, duration and rate of depolarization. This RBB2 response failed to be conducted to the His bundle fiber. The subsequent V3 response was conducted retrograde over the ventricular specialized conduction system to the atrium. The V4 response was blocked between the bundle branch and His bundle. The amplitude and rate of rise of the RBB4 response was nearly equal to that of the RBB2 response. Retrograde block in this instance probably occurred nearer the His bundle than did block of the RBB2 response. V2 was conducted back to the atrium, giving an instance of 2:1 retrograde block. Most of the retrograde conduction delay of the V3 response occurred between the ventricle and RBB. The blocked V4 response caused the retrograde conduction time of the V5 response to be prolonged by 18 msec over the V1-V2 retrograde conduction time.

Figure 7 is a recording made during a spontaneous ventricular tachycardia that resulted in 2:1 retrograde block. It is not possible in this figure to determine the precise origin or mechanism of the ventricular tachycardia. The first right ventricular potential traveled to the right atrium. The second right ventricular potential resulted in only a local response in the RBB fiber and was not conducted to the A-V nodal fiber; conduction...
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RA = right atrial electrogram; AVN = A-V nodal transmembrane potentials; Ndv/dt = first derivative of A-V nodal transmembrane potentials; RBB = right bundle branch transmembrane potentials; Bdv/dt = first derivative of bundle branch transmembrane potentials; RV = right ventricular electrogram.

FIGURE 7

block developed near the impaled RBB fiber. The last right ventricular complex in the RV electrogram can be observed to differ from the other RV responses. At this instant, spontaneous ventricular tachycardia, which was initiated by a series of premature ventricular stimuli, reverted back to the previous slow rhythm. Notice that the last local RBB response was smaller in amplitude than the three previous local RBB responses; also, unlike the previous RBB potentials, the final local RBB response occurred before the ventricular depolarization complex.

Figure 8 is from an experiment in which both antegrade and retrograde block of premature responses were demonstrated. The atria were first driven at a constant rate for nine responses; the A1 response was conducted from the right atrium to the right ventricle. An A2 was then produced; it resulted in an attenuated atrial response. The A2 response was conducted to the impaled A-V nodal fiber with some increase in atrium-node conduction time. The AVN2 action potential failed to be conducted to the impaled RBB fiber. Concealment of the A2 response occurred between the His bundle and bundle branch-Purkinje system as reported previously (9). A premature ventricular response, V2, was then elicited 258 msec after the premature A2 atrial response. This premature ventricular response was propagated to the RBB fiber and then, with considerable retrograde conduction delay, through the His bundle back to the impaled A-V nodal fiber. However, only a local response occurred in the A-V nodal fiber with retrograde conduction of the premature V2 response. Therefore, in this experiment, antegrade concealment below the A-V node was followed by retrograde concealment within the A-V node.

In Figure 9, recorded in the same experiment as Figure 8, the interval A2-V1 between the premature atrial response, A2, and premature ventricular response, V1, was increased. Delaying the premature ventricular response, V1, by 4 msec permitted retrograde conduction of V1 back to the atrium (A2*). Some local activity preceded the upstroke of the AVN* action potential.

In Figure 10, recorded in the same experiment as Figs. 8 and 9, two atrial premature responses, A2 and A3, and a single ventricular premature response, V4, were blocked sequentially. The preparation was driven at a basic cycle length for nine atrial responses before premature responses were elicited. The A2 response was conducted through the A-V node but blocked above the impaled RBB fiber. The A3 response resulted in only a local response in the impaled A-V nodal fiber and
was blocked within the A-V node. Following the A₃ response, a premature ventricular response, Vₓ, was evoked in the right ventricle. The Vₓ response was conducted over the ventricular specialized conduction system to the impaled A-V nodal fiber, but caused only a small nonpropagated response in the impaled A-V nodal fiber (second local AVN response). The amplitude of the local response in the impaled A-V nodal fiber increased in amplitude when the Vₓ response was evoked progressively later and decreased with prematurity. Thus it was possible to conceal both antegrade and retrograde responses consecutively within the A-V node. Note that duration and configuration of the local AVN nonpropagated response recorded from the same A-V nodal fiber during antegrade and retrograde concealment differed.

In Figure 11, the heart was driven at a basic rate from an electrode located on the right atrium. Following the ninth A₁ response, two premature atrial responses, A₂ and A₃, were evoked. A₂ was conducted through the A-V node but was blocked above the impaled RBB fiber. The A₃ response was blocked above this A-V nodal fiber; in the absence of a premature ventricular response (Vₓ), A₃ failed to cause even a local response in the AVN potential at this A₂-A₃ interval. A premature ventricular response, Vₓ, was evoked at the right ventricular stimulating electrode 72 msec after the premature atrial response A₃. Vₓ was conducted through the Purkinje system to the impaled RBB fiber, to the His bundle (not shown), through the A-V nodal fiber and to the right atrium. The change in the complex of the right atrial electrogram during retrograde activation can be noted. In this figure, the A₄ response reentered or
“echoed” back to the right ventricle and caused the ventricular complex labeled V₈. When both antegrade and retrograde impulses were evoked in close succession, at least two premature atrial responses usually were necessary to cause an echo. For example, in this experiment, when A₃ was eliminated and the V₁-V₈ interval was maintained, increased or decreased, “echo” responses were not observed. In the presence of A₃, echo responses could be elicited consistently with variations in the RV₁-V₈ interval of about 10 msec. Multiple spontaneous echoes reentering back and forth between atrium and ventricle were not observed when the A-V transmission system was intact between the right atrium and right ventricle. However, when the isolated preparation was limited to the atrium, A-V node and His bundle, it was possible by introducing two or more premature His bundle responses to initiate multiple echoes that would reecho between atria and ventricles for four or more responses.

Discussion

These experiments have demonstrated that, during retrograde conduction of premature ventricular responses, there are multiple sites in the ventricular specialized conduction system (VSCS) and A-V node where conduction delays and block can occur. Retrograde conduction delays and blocks were observed between the ventricles and Purkinje system, between the Purkinje-bundle branch system and His bundle, and at various sites within the A-V node. Although most clinical and experimental studies on concealment of premature ventricular responses usually fail to consider the possibility of conduction delays and block within the VSCS, these investigations on isolated rabbit A-V preparations suggest that the VSCS is of major importance in retrograde conduction delays and concealment of ventricular premature responses. In studies on concealment of antegrade responses, A-V conduction delays and concealment were observed to occur within the A-V node and within the VSCS (8, 9). However, in the studies on antegrade concealment, the A-V node was found to be the region where most premature atrial responses were delayed or blocked; the VSCS played a minor role in antegrade concealment.

In these microelectrode studies on retrograde concealment in isolated right A-V preparations, clear evidence of retrograde block without nodal penetration was repeatedly observed. Lack of any subsequent prolongation in retrograde conduction time of a second premature ventricular (V₈) response following a blocked V₂ response was observed when V₂ was blocked between ventricular muscle and the Purkinje system as well as in experiments where V₂ was blocked above the Purkinje system but below the A-V node. When block of the V₂ response occurred between ventricular muscle and the Purkinje system, one would not anticipate any subsequent delay in retrograde conduction of an early V₈ response; the VSCS and A-V node would both be excitable when activity from the V₂ response arrived. When the V₂ response was blocked above the bundle branch but below the A-V node, a propagated subsequent V₈ response traveled over a Purkinje-bundle branch system whose functional refractory period was considerably shortened by the preceding short cycle produced by the V₂ response. Conduction of a subsequent V₈ response through the A-V node occurred over recovered nodal tissue.

In in-vivo experiments on retrograde concealed conduction, Moe and associates (5) thought that retrograde conduction delays and block occurred within the A-V node. However, since only atrial and ventricular electrograms were recorded in their studies (5), it is quite possible that retrograde delays and block occurred within both the VSCS and the A-V node; i.e., functionally, it would be difficult to distinguish effects of A-V nodal delays and block from those occurring within the VSCS. Several major differences exist between experiments on intact animals and the present studies. The site of origin of a premature ventricular response plays a definite role in determining where conduction delays and block occur. In these studies on isolated A-V preparations, premature ven-
tricular responses usually were evoked near the anterior papillary muscle. Consequently, premature ventricular responses had early access to the Purkinje-bundle branch system, and retrograde block between ventricular muscle and the Purkinje system was easily demonstrated. When the stimulating electrode was moved to the base of the right ventricular septum, it was difficult and sometimes impossible to demonstrate retrograde block between ventricular fibers and Purkinje fibers, since propagation occurred within ventricular tissue until excitable Purkinje fibers were encountered. The fact that the left ventricular specialized conduction system, part of the right Purkinje system and the left and right ventricular free walls were removed in our isolated preparations must account for some differences between the present studies and those performed on intact hearts. For example, if the left VSCS were functional, then retrograde conduction might still occur, even in the presence of block within the right ventricular specialized conduction system. Nevertheless, in the intact heart, retrograde conduction delays and block undoubtedly do occur within ventricular muscle, between ventricular muscle and the Purkinje-bundle branch system, between bundle branches and His bundle as well as within the A-V node.

The occurrence of retrograde block and conduction delays between the Purkinje-bundle branch system and bundle of His was unexpected. The action potential duration and functional refractory period of bundle of His fibers are shorter than those of the bundle branch or Purkinje fibers at basic physiological heart rates. However, when the preceding cycle length was rapidly shortened by evoking a series of premature ventricular responses with successively decreasing preceding cycle lengths, it was possible in some cases to have the functional refractory period of RBB fibers shorter than that of bundle of His fibers. Also, preliminary experiments (Moore, unpublished) suggest that His bundle fibers have a higher current threshold for excitation at the end of their functional refractory period than do Purkinje fibers. Thus, block between the right bundle branch and His bundle may have resulted in some instances from propagation of excitation into less excitable His bundle fibers. Whether or not block occurred within the ventricular specialized conduction system depended not only on the difference in the functional refractory period of His bundle and bundle branch fibers, but also on the amount that conduction velocity was slowed because propagation occurred in partially refractory tissue. If conduction velocity from the ventricles to His bundle was sufficiently decreased, then adequate time was provided in some instances for the bundle of His to recover excitability.

The possibility of depressed conduction caused by enhanced pacemaker activity cannot be ruled out when delays or block occur anywhere within the ventricular specialized conduction system. As shown first by Weidmann and more recently by Hoffman (15) and associates, conduction delays and block can result from conduction of an impulse into cells partially depolarized by diastolic depolarization. In these experiments, prominent diastolic depolarization was not observed in the impaled Purkinje, RBB, or bundle of His fibers.

In confirmation of previous concepts, retrograde conduction delays and block of premature ventricular responses did occur within the A-V node in these experiments. However, the frequent occurrence of retrograde conduction delays and block within the ventricular specialized conduction system demonstrates the difficulty in determining the location and mechanisms of conduction delays and block only from information obtained from atrial and ventricular electrograms, or from electrocardiograms. When block occurred within the A-V node, local responses could be recorded from A-V nodal fibers located in the region of block. When atrial premature responses were blocked within the A-V node, the local non-propagated A-V nodal response recorded from the same A-V nodal fiber often had a different configuration than the local response resulting from retrograde block. Paes de Carvalho and de Almeida (16) previously dem-
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Demonstrated that the configuration of the upstroke of antegrade and retrograde A-V nodal responses differed. The reason for these differences cannot be determined at the present time because of a lack of information concerning the anatomical structure and electrical properties of the A-V node. In some experiments, retrograde all-or-none A-V nodal action potentials occurred at the end of a local response; i.e., a step appeared on the upstroke of the A-V nodal action potential. This was due to dissociation within the A-V node. Some A-V nodal fibers exhibited all-or-none action potentials synchronous with the upstroke of A-V nodal action potential developing out of the local step potential; other A-V nodal fibers exhibited only a local response.

A single ventricular premature response was not blocked within the A-V node when the ventricles were driven at physiological heart rates. It was possible, however, by direct stimulation of the His bundle, to block a single premature bundle of His response within the A-V node. When the premature His bundle response was conducted through the A-V node to the atrium, retrograde conduction time was increased. To demonstrate marked retrograde A-V nodal delay, however, it was necessary to evoke a second premature bundle of His response. Echo responses often were observed following two consecutive early premature His bundle responses; in many instances, reentrant echo responses reciprocated between atrium and His bundle for four or more beats. In isolated preparations in which the entire right A-V conduction system was functional, single atrial and single ventricular echoes were not uncommon; multiple echoes, however, were not observed. The rare occurrence of multiple echoes in an intact A-V conduction system agrees with the results obtained by Moe and associates in intact dogs (17). The fact that echoes are more readily evoked by premature stimulation of the His bundle than by eliciting premature ventricular beats is probably related to the ability of premature bundle of His responses to invade the A-V node earlier during its relative refractory period than ventricular premature responses. Echo responses were also observed following excitation of the A-V node by consecutively evoking premature atrial and ventricular premature responses.

The studies of Van Dam and associates (18) indicate that transmembrane potentials having “double” depolarization complexes as observed in Figure 5 are usually caused by a shift of the site of origin of the propagated action potential when stimuli occur during the relative refractory period. Activity originating at a more distant part of the conduction system can spread bidirectionally; i.e., toward the A-V node and backwards toward the impaled RBB fiber, leading to a second depolarization complex in the RBB fiber. A similar phenomenon has been demonstrated in intracellular recordings from A-V nodal fibers (7, 9, 19). The occurrence of these double depolarization complexes on action potentials may be significant from several standpoints. They indicate the difficulty of graphing the progress of A-V conduction with intracellular recording techniques, since an impaled fiber may not accurately represent the overall time course of propagation. The double spikes that Van Dam and associates (20), using extracellular bipolar recording techniques, recorded during the relative refractory period of different parts of the ventricular specialized conduction system probably resulted from cardiac fibers exhibiting this type of double action potential. In plotting graphs of A-V conduction, selecting one of the two spikes to represent the spread of cardiac conduction may lead to errors. A problem may also arise in using high and low pass filter setting on amplifiers for recording extracellular potentials. These could alter the recorded electrogram sufficiently that only one of the spikes would be manifest.

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