Experimental Studies on the Facilitation of AV Conduction by Ectopic Beats in Dogs and Rabbits

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ABSTRACT

Experiments were performed on the hearts of open-chest anesthetized dogs and on isolated rabbit hearts. Intracellular microelectrodes and bipolar surface electrodes were used for recording from and for stimulating the atria, AV node, bundle of His, bundle branches and ventricles. Atrial or ventricular premature beats which were usually blocked or delayed within the AV node were facilitated when ectopic activity caused preexcitation and early recovery of the site of block or delay. Ectopic atrial, ventricular, nodal, and bundle of His responses allowed previously blocked or delayed antegrade and retrograde beats to be conducted only when the ectopic activity arrived at an appropriate time at the site of block within the AV node. Therefore, apparent supernormal AV conduction may occur in the human heart by this mechanism, and some reported cases of supernormal conduction may be due to the preexcitation and early recovery of a refractory barrier by ectopic activity.

KEY WORDS

supernormal conduction

arrhythmias microelectrode experiments premature impulses

Supernormal conduction is a term applied to atrioventricular (AV) conduction that is either more rapid than expected or occurs during a time when block is expected. It should be emphasized that AV transmission is never faster than normal and that the possibility of supernormal conduction is considered only when AV conduction is depressed. Clinical cases of apparent supernormal conduction during AV dissociation and first- and second-degree heart block have been reported (1–5). Moe et al. (6) have suggested the concept of preexcitation and early recovery of a site of block as an explanation of many clinical cases in which facilitation of AV transmission occurred. They introduced the term “peeling back” of a refractory barrier to describe this phenomenon. The present studies were undertaken to investigate apparent supernormal conduction resulting from facilitation of conduction due to preexcitation of the AV node by ectopic activity.

MATERIALS AND METHODS

WHOLE ANIMAL PREPARATION

Experiments were performed on dogs anesthetized by pentobarbital sodium (30 mg/kg iv) and maintained on controlled positive ventilation. The hearts were exposed by right thoracotomy (fifth intercostal space). Bipolar recordings were made from atrium, His bundle, bundle branches, and ventricular muscle with plunge electrodes (7). The electrograms were displayed on an eight-channel oscilloscope and photographed on 35-mm film. Electrical stimuli were delivered to the atria, bundle of His, and ventricles by a digital stimulator which could be programmed.

ISOLATED HEART PREPARATION

Hearts were rapidly excised from anesthetized rabbits (pentobarbital sodium, 30 mg/kg) weighing 1.5–3.0 kg. The hearts were dissected in Tyrode’s solution and the right atrial and
An ectopic ventricular beat causing the conduction of a previously blocked atrial premature beat in an open-chest dog. Bipolar electrograms were recorded from the right atrium (RA), bundle of His (BH) and left Purkinje fiber (LPF), together with the lead II ECG. In the bundle of His electrogram, h indicates the His potential. In the left Purkinje fiber electrogram, p indicates the Purkinje potential. The timing signal (T) denotes 100 msec. The basic cycle length was 290 msec and the premature atrial cycle length was 230 msec. The preexcitation of the ventricles occurred 55 msec after the basic atrial beat. See text for discussion.

Results

Figure 1 shows an example of an ectopic ventricular response resulting in the conduction of a previously blocked premature atrial beat. The experiment was performed on an anesthetized open-chest dog, and bipolar electrograms were recorded from the right atrium, bundle of His, and left Purkinje fiber. A lead II electrocardiogram was recorded simultaneously. In A, the heart was paced from the right atrium at a basic cycle length of 290 msec. The conduction time from right atrium to His bundle was 108 msec and from His bundle to Purkinje fiber was 24 msec. After every twelfth beat, the atrial stimulus was delivered 60 msec prematurely (arrow). This small degree of prematurity was sufficient to cause complete AV block, as shown by the absence of a QRS complex following the third P wave. AV block probably occurred within the AV node since depolarization complexes were not recorded in either the bundle of His or left Purkinje fiber electrograms. The records in B demonstrate that a properly timed ventricular premature beat can result in conduction of a previously blocked atrial response. The pattern of atrial stimulation was identical to that in A. However, just before the arrow in B, the ventricles were activated 175 msec before the premature atrial beat and 55 msec after the last basic atrial beat. Note that the beginning of the ectopic QRS complex of the premature ventricular contraction preceded any activity in either the His bundle or left Purkinje fiber electrograms. This ventricular premature beat was conducted retrogradely back to the left Purkinje fiber and to the bundle of His. Retrograde activation of the Purkinje system and the bundle of His was confirmed by the change in configuration and the time of onset of depolarization in these specialized conduction tissues, i.e., the bundle of His and left Purkinje.
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Fiber electrograms occur at 65 msec and 84 msec, respectively, following the atrial electrogram, and they therefore could not have been conducted antegradely. The fact that the His bundle and Purkinje spikes resulted from retrograde activation was also confirmed by deleting the premature atrial beat (arrow) and making the ventricular premature beat slightly later. Retrograde conduction back to the atrium resulted, and the accompanying His bundle and Purkinje spikes were identical to those recorded in this illustration. In B, retrograde conduction back to the atrium did not occur. The fact, however, that retrograde invasion of the AV node was present was confirmed by delaying the ventricular beat by about 10 msec, resulting in retrograde conduction back to the atrium. Therefore, in B, the occurrence of a premature ventricular contraction resulted in collision of retrograde and antegrade activation presumably within the AV node, thereby permitting antegrade conduction of the atrial premature beat. This is an example of AV conduction at a time when otherwise there was complete AV block, i.e., apparent supernormal conduction.

Several possible mechanisms could explain how a premature ventricular response might cause the AV node to have a shorter absolute refractory period as experimentally observed in Figure 1. Figure 2 is a schematic representation of two possibilities. The hatched area represents the duration of the absolute refractory period which follows each conducted response. The AV node is presented as having two regions with different absolute refractory periods. In A, the A2 response was propagated through the upper region of the AV node but failed to be conducted to the ventricles because it was blocked by the longer refractory period of the lower AV nodal system. In B, which is a schematic representation of the experiment of Figure 1B, a premature ventricular response occurred prior to the expected arrival of the antegradely propagated ventricular V1 action potential. As in Figure 1B, the premature ventricular response was not conducted retrogradely to the atria. However, by exciting the AV node earlier than the normal antegrade conducted A1 response, a briefer preceding cycle length resulted for the lower AV nodal tissues in Figure 2B than in Figure 2A. Since the preceding cycle length usually governs the absolute refractory periods of

![Figure 2](image)

Schematic representations using ladder diagrams to present mechanisms that might explain supernormal conduction following preexcitation. Shaded areas represent the absolute refractory period of the AV node following atrial excitation (A). Ventricular premature beats (VS) are shown as being conducted retrogradely to the AV node. See text for discussion.

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An ectopic ventricular beat causing conduction of a previously blocked atrial premature beat in an isolated rabbit heart. Bipolar electrograms were recorded from the right atrium (RA) and right ventricle (RV), together with transmembrane potentials recorded from the AV node (AVN) and right bundle branch (RBB).

The timing signal (T) denotes 100 msec. The basic cycle length was 500 msec and the premature atrial cycle length was 310 msec. See text for discussion.

cardiac fibers, it is possible that the $A_2$ atrial response in Figure 1B was conducted because of an abbreviation of the refractory period of the AV nodal tissues at the site of previous block resulting from a shortening of cycle length as schematically diagramed. A second possible explanation of how a premature ventricular response might result in a shortening of the absolute refractory period of the AV node is shown in Figure 2C. The absolute refractory period of both the upper and lower AV node are assumed to undergo no shortening following retrograde conduction of the premature ventricular response. However, the premature ventricular response is presumed to be conducted retrogradely through the previous site of AV nodal block of $A_2$. Since the lower AV nodal tissue is excited earlier, it would be expected to recover excitability earlier. When the relative timing of the premature ventricular response and the atrial response occur within certain time periods, as shown in Figure 2C, then the premature ventricular response could allow complete repolarization and disappearance of refractoriness within the AV node to occur by the time the premature $A_2$ impulse arrives. Thus premature excitation by the premature ventricular response effectively removes the previously existing AV nodal barrier of refractoriness by permitting earlier recovery at the site of AV nodal block. Moe et al. have referred to this latter possibility as peeling back of a refractory barrier.

Experiments similar to those shown in Figure 1 were done on the isolated rabbit heart to define more precisely the site and mechanism of this phenomenon. Figure 3 is an example of one such experiment. Bipolar surface electrodes were used to record from the right atrium and right ventricle, together with simultaneous transmembrane recordings from the AV node and septal right bundle branch. In A, the preparation was paced from the atrium at a basic cycle length of 500 msec. Every twelfth atrial beat was made premature by 190 msec. The premature beat with a cycle length of 310 msec is seen as the third atrial complex. Notice that the premature beat failed to traverse the AV node with only a subthreshold local response being elicited in the impaled nodal cell. The anatomical location of the impaled cell, as well as its action potential configuration and timing, indicates that it represents a cell from the lower N region or upper NH region of the node. The occurrence of a subthreshold AV node potential at 170 msec following the premature atrial complex suggests that the site of block was not far above the impaled cell and was most probably in the N region of the node. In B of Figure 3, the pattern of atrial stimulation was exactly the same as in A. At the arrow the atrial premature beat was preceded at an interval of 350 msec by ventricular stimulation. The ventricular activity was conducted retrogradely through the region of the impaled nodal cell but was blocked before reaching the atrium. The second atrial beat in the record was then...
An ectopic junctional beat caused conduction of a previously blocked atrial premature beat. Bipolar electrograms were recorded from the right atrium (RA), bundle of His (BH) and left Purkinje fiber (LPF) in an open-chest dog together with a lead II ECG. In the bundle of His electrograms, h indicates the His potential and s the stimulus artifact during His bundle stimulation. The timing signal (T) denotes 100 msec. The basic cycle length was 290 msec and the premature atrial cycle length was 224 msec. See text for discussion.

Junctional escape beats can also cause conduction of previously blocked activity. Figure 4 shows an experiment similar to that shown in Figure 1, performed on an open-chest anesthetized dog, which demonstrates this phenomenon. The heart was paced from the right atrium at a basic cycle length of 290 msec. The premature atrial impulse at the arrow in A followed the basic beat by 224 msec and failed to reach the recording site of the bundle of His electrogram. The right atrium-to-His bundle conduction time and the His bundle-to-Purkinje conduction time for the normal beats were 106 msec and 24 msec, respectively. In B, the same pattern of atrial stimulation was continued, with the bundle of His stimulated 130 msec before the premature atrial beat occurred. The stimulated His bundle activity was conducted to the left Purkinje system with the same conduction time as the normally conducted activity (stimulus-to-Purkinje time was equal to the His bundle-to-Purkinje time). The QRS complex associated with the bundle of His stimulation is of the same configuration as the normally conducted QRS complex, and His bundle stimulation shortened the P-R interval of the normally conducted beat by 20 msec. A 20-msec change in the P-R interval (0.5 mm) is just at the limits of the resolution of the clinical ECG recorded at 25 mm/sec. Notice that following the bundle of His stimulation, the previously blocked atrial impulse (arrow) was now conducted to the ventricles with a right atrium-to-His bundle interval of 130 msec and a His bundle-to-Purkinje interval of 24 msec. Thus conduction time between the previously blocked atrial beat and the bundle
FIGURE 5

An ectopic functional beat causing conduction of a previously blocked atrial premature beat in an isolated rabbit heart. Bipolar electrograms were recorded from the right atrium (RA) and right ventricle (RV), together with transmembrane potentials from the NH region of the AV node and the right bundle branch (RBB). The timing signal (T) denotes 100 msec intervals. The basic cycle length was 345 msec and the premature atrial cycle length was 265 msec. See text for discussion.

An experiment in an isolated rabbit heart in which an ectopic beat in the lower AV node or upper bundle of His resulted in conduction of a previously blocked atrial beat is presented in Figure 5. Electrograms were recorded from the right atrium and right ventricle simultaneously with transmembrane action potentials recorded from an AV nodal fiber at the bundle of His-AV nodal junction (NH) and from a right bundle branch fiber. The first two atrial responses are at a basic cycle length of 345 msec and were propagated through the impaled NH and right bundle branch fibers to the right ventricle. The third atrial response, which was evoked prematurely at an interval of 265 msec, was blocked above the impaled NH fiber. In Figure 5B, the recording microelectrode was used to prematurely excite (arrow) the impaled NH fiber 160 msec before the premature atrial beat occurred and 38 msec before the normally conducted activity would have arrived at the NH recording site. The intracellularly evoked premature NH action potential was conducted antegradely to the impaled right bundle branch fiber and to the ventricles, but

FIGURE 6

An ectopic junctional beat causing acceleration in conduction of a previously delayed atrial premature beat in an open-chest dog. Bipolar electrograms were recorded from the right atrium (RA), bundle of His (BH), and left Purkinje fiber (LPF), together with the lead II ECG. In the bundle of His electrograms, h indicates the His potential and s the stimulus artifact during His bundle stimulation. The timing signal (T) denotes 100 msec. The figure shows two superimposed records with and without His preexcitation. The His potential indicated by 1 occurred without prior His preexcitation. The His potential indicated by 2 occurred following His preexcitation. The basic cycle length was 300 msec and the premature atrial cycle length was 237 msec. See text for discussion.

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Retrograde conduction to the right atrium did not occur. However, this ectopic premature beat within the lower AV node did conduct retrogradely to the site of antegrade conduction block, as clearly demonstrated by the previously blocked atrial response then conducted to the ventricles in the presence of this ectopic beat. Therefore, preexcitation of a site of nodal block by an ectopic AV junctional beat can allow a previously blocked premature beat to be conducted.

Figures 1 through 5 demonstrate how ventricular and AV junctional ectopic activity can result in AV conduction of previously blocked beats. Figures 6 and 7 show examples of how depressed conduction can be accelerated by junctional ectopic beats. In Figure 6, electrograms were recorded from the right atrium, bundle of His, and left Purkinje fibers simultaneously with a lead II ECG. The superimposed records with and without His bundle stimulation are shown. The records are on an expanded time scale (time marks denote 100 msec intervals) and show only the last normally conducted atrial beat of the stimulation series and the premature atrial beat. The first complex in the right atrial record is composed of two superimposed atrial activations. Without prior preexcitation of the His bundle, this atrial activity was conducted to the bundle, producing the His potential in the bundle of His electrogram and was also conducted antegrade through the bundle branches and Purkinje system to the ventricles producing the second QRS complex of the superimposed group. The premature atrial response at the arrow (second atrial complex in the right atrial electrogram) was conducted with an increased AV nodal conduction time to the bundle of His (larger His complex) as indicated by the number 1 above the His bundle complex and by the upper broken line. Upon conduction to the ventricles, this activity produced the corresponding late QRS complex in the superimposed tracings (indicated by the number 1). With preexcitation of the bundle of His as indicated by the stimulus artifact, activity from the basic atrial response (first right atrial complex) was not conducted through the AV node to the bundle of His. However, premature stimulation of the bundle of His did result in antegrade conduction to the Purkinje system (note Purkinje spike p) and to the ventricles. Preexcitation of the His bundle shortened the P-R interval by 71 msec, as can be seen by the first QRS complex in the superimposed group, but the ventricles were normally activated as demonstrated by the resulting normal QRS complex. After preexcitation of the His bundle, the following premature atrial beat (arrow) was conducted to the bundle of His with a shorter AV conduction time, as indicated by the number 2 above the bundle of His complex and also by the lower dotted line. AV conduction to the ventricles produced the correspondingly early QRS indicated by the number 2 in the record below, i.e., shortened P-R interval. Therefore, a premature beat originating within the bundle of His resulted in shortening of the P-R interval by 18 msec.

Acceleration of AV conduction produced by ectopic activity was also demonstrated in the isolated rabbit heart; an example is shown in Figure 7. A and B are records taken before preexcitation of the impaled NH cell; C shows records taken during preexcitation. The three records are time-aligned in the figure so that the relative times of activation of all complexes can be compared. Transmembrane action potentials were recorded from an AV nodal fiber at the AV node-His bundle junction and from a fiber within the right bundle branch, along with right atrial and ventricular electrograms. Time marks indicate 10 msec and 100 msec. The preparation was driven from right atrial bipolar electrodes, and the pattern of atrial stimulation was identical in A, B, and C. Notice that the third atrial response was premature so that conduction to the ventricle was delayed by 40 msec compared to the first two basic atrial beats (the normal right atrium-to-NH time was 150 msec and the premature time was 190 msec). The greater part of the AV delay occurred above the site of the impaled nodal cell and the increased AV delay occurring with the premature atrial response resulted from an
An ectopic junctional beat causing acceleration in conduction of a previously delayed atrial premature beat in an isolated rabbit heart. Bipolar electrograms were recorded from the right atrium (RA) and right ventricle (RV), together with transmembrane potentials from the NH region of the AV node (NH) and the right bundle branch (RBB). The timing of a stimulus artifact of a subthreshold stimulus of 4 msec duration through the recording microelectrode can be noted in the AV node transmembrane potential record. The timing signal (T) denotes 100 msec. The broken line aligns the beginning of the nodal potentials in A and B with the corresponding record in C. The basic cycle length was 344 msec and the premature atrial cycle length was 242 msec. See text for discussion.

FIGURE 7

An ectopic atrial beat causing retrograde conduction of a previously blocked ventricular premature beat in an isolated rabbit heart. Bipolar electrograms were recorded from the right atrium (RA) and right ventricle (RV), together with transmembrane potentials from the N region of the AV node (N) and the right bundle branch (RBB). The timing signal (T) denotes 100 msec. The basic retrograde cycle length was 500 msec and the premature ventricular cycle length was 260 msec. See text for discussion.

FIGURE 8

increase in the atrial to lower node conduction time. Therefore, the atrial impulse was being conducted through the AV node during its refractory period. In B, at the time indicated by the arrow, a subthreshold current for 4 msec was passed through the recording microelectrode impaling the His-AV node fiber. Notice that the subthreshold current pulse had no effect on this beat nor on the conduction times of the following conducted beats. In C, the current at the arrow was at threshold and resulted in a prematurely evoked action potential in the impaled nodal cell. Notice that although this prematurely evoked NH action potential was not conducted retrogradely to the atrium, the concealed retrograde activity blocked the antegrade conduction of the usually conducted second.
atrial beat. Notice also that the activity evoked in the single cell of the NH region of the node was conducted antegradely to the impaled right bundle branch fiber and the ventricle. The broken lines in A, B, and C align the depolarization phases of the AV nodal action potentials resulting from conduction of the premature atrial response (third right atrial complex). Notice that the 108-msec preexcitation of the NH fiber in C caused the premature atrial beat in this instance to be conducted to the NH fiber 35 msec faster than in A and B. This decrease in AV nodal conduction time is reflected in the decreased AV conduction time for the premature atrial response in C of Figure 7.

The same mechanisms demonstrated in the previous figures can be shown to occur during retrograde conduction. Figure 8 shows an example in the isolated rabbit heart of an ectopic atrial beat causing retrograde conduction of a previously blocked ventricular premature beat. In A, the preparation was paced from the right ventricle at a basic rate of 120 per/min with resultant 1:1 retrograde conduction to the atrium. Every twelfth ventricular beat was made premature by 240 msec (arrow). This degree of prematurity permitted retrograde conduction up to the region of the impaled right bundle branch fiber. However, the premature ventricular beat was blocked below the impaled fiber in the upper NH region of the AV node (N). Previous studies on retrograde concealment of premature ventricular responses suggest that, in this experiment, block very likely occurred within the His-Purkinje system, rather than within the AV node (9). In B, the pattern of ventricular stimulation was precisely the same as in A except that the right atrium was preexcited 320 msec before the premature ventricular beat was evoked. The premature atrial beat in B, which is the second atrial complex, was conducted to the impaled N fiber but not to the right bundle branch fiber. The fact that antegrade conduction through the N fiber occurred is supported by the presence of a foot on the second antegradely excited AV nodal potential (N) plus the brief interval between the right bundle branch and AV nodal action potentials following the premature atrial response. Therefore, collision of antegrade and retrograde activity resulted somewhere below the impaled AV nodal fiber but above the right bundle branch system, i.e., within the ventricular specialized conduction system (His-Purkinje system) rather than within the AV node. The effect of atrial preexcitation and the resulting collision was to allow the previously blocked premature ventricular response to be propagated retrogradely to the atrium. It should be noted that a small degree of diastolic depolarization is present in the impaled nodal fiber. As pointed out by Moe et al. (6), diastolic depolarization may account for some instances of true supernormal conduction. For example, if an impulse arrives early before diastolic depolarization has changed the membrane potential to a low potential then its conduction velocity will be faster than at a later time. However, in the present experiment, the premature right bundle branch action potential (arrow) would have arrived at the impaled AV nodal fiber at an earlier time in A than in B. Therefore, if diastolic depolarization in the impaled AV nodal fiber was a factor in causing supernormal conduction in this instance, one might expect the conduction velocity in A to be faster than in B, i.e., time lapse would have permitted diastolic depolarization to progress to a lower membrane potential in B than in A, and therefore conduction should have been more rapid in A. In summary, we feel that Figure 8 is very probably an example in which premature atrial excitation caused early recovery of a refractory barrier within the ventricular specialized conduction system, thereby permitting conduction of a previously blocked ventricular premature response.

Discussion

Experimental studies in isolated hearts have shown that the site of conduction delay and block of premature beats is usually within the AV node, but above the NH region (10, 11). The present studies also demonstrate this and
have verified the concept that preexcitation of a refractory barrier within the N region of the node permits early recovery, thereby facilitating conduction of premature beats. Moe et al. have suggested the term peeling back to describe this phenomenon. However, it is possible that changes in cycle length accompanying preexcitation may be a contributing factor to facilitated conduction by ectopic activity. The microelectrode experiments point out that concealed activity must invade the site of conduction delay or block in order to facilitate conduction. The mechanism of facilitation is preexcitation with associated earlier recovery of the site of AV nodal block or delay, i.e., a temporal peeling back of a refractory AV nodal barrier. Of course, preexcitation and associated early recovery cannot cause AV conduction to be more rapid than normal. The effect of junctional or ventricular ectopic activity was to improve depressed AV conduction to times closer to normal. Therefore, conduction could be said to be supernormal only relative to the depressed state.

For facilitated conduction to occur, it was necessary for the junctional or ventricular ectopic response to occur during a critical period of time; the ectopic response had to occur sufficiently early to preexcite the AV node, thereby resulting in sufficient shortening of the relative refractory period to permit facilitated conduction. The lower limit of this interval was quite precise, however, and variations in the timing of the facilitating beat of 1 msec relative to the premature beat could determine if the premature beat was blocked or conducted. The interval for the ectopic beat to be effective in facilitating conduction was dependent on the normal basic driving rate and the degree of prematurity of the premature beat. In some cases, during very rapid driving rates when nodal activations were close together, ectopic activity could not invade the nodal site of block to peel back the refractory barrier and to facilitate conduction.

In most of the experiments described here, the ectopic activity could be discerned from the electrocardiographic leads or from atrial or ventricular electrograms. In these cases, the phenomenon of preexcitation and associated early recovery of the refractory barrier can be defined in terms of the R-P, P-R rule. That is, conduction of the premature atrial beat was facilitated more when the ventricular activity preceding it occurred with a longer R-P interval. However, it is possible for junctional ectopic activity to be completely concealed in both the antegrade and retrograde directions but still facilitate conduction. In such a case the cause of the facilitation would not be apparent and might be attributed to true supernormal conduction. Also, as can be seen in Figure 4, the change in P-R interval due to a junctional escape beat could go undetected in the clinical ECG and the accompanying facilitated conduction could be interpreted as true supernormal conduction.

The retrograde experiment shown in Figure 8 is the converse of the experiment of Figure 3. The site and mechanism involved with facilitation during the retrograde experiment in Figure 8 was probably associated with preexcitation and early recovery of a refractory barrier within the ventricular specialized conduction system, rather than within the AV node. The same mechanism as suggested in Figure 2C is thought to have occurred in this instance: the longer absolute refractory period of the tissue at the site of block within the ventricular specialized conduction system was abbreviated by preexcitation and associated early recovery. As pointed out diagrammatically by Moe et al. (their Fig. 3), atrial preexcitation may also peel back a refractory barrier within the AV node. Therefore, these experiments point out the possibilities for complex interaction between antegrade and retrograde conduction with facilitation of sites of delay and block occurring within the ventricular specialized conduction system (His-Purkinje system) as well as within the AV node.

The experiments described in Figure 7 demonstrate an interesting corollary of AV conduction in the rabbit heart. In these
experiments, threshold stimulation was attained in a single cell of the lower region of the AV node or upper His bundle by gradually increasing the amount of current passed through the recording microelectrode (B and C). In Figure 6C, the evoked all-or-none action potential must have been initiated in a very small number of fibers and most probably was initiated within the impaled single cell. This activity arising in a single cell must have been rapidly conducted to all of the adjacent conduction cells, since the ectopic beat which arose in a single cell within the lower AV node was conducted through the impaled right bundle branch to the ventricles with a conduction velocity comparable to the normally conducted beats. Also, multiple impalements of fibers within the right bundle branch failed to exhibit any nonuniform activation of various bundle branch fibers. These data suggest an extensive functional lateral communication for conduction in the AV junctional tissues.

The present experiments were designed to help understand the mechanism involved during the facilitation of conduction through a site of previous block or delay. Block or delay was consistently produced within the AV node by premature atrial activation. The site of block or delay was eliminated by preexcitation with retrograde invasion into the left bundle branch. Circulation 38:474-479, 1968.

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