Demonstration of Entrance Block into the Atrioventricular Node of Man

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SUMMARY

Entrance block of an atrial premature beat (APB) into the atrioventricular node is demonstrated by its lack of effect on the A-V nodal conduction of a subsequent beat which is introduced before the full recovery time of the A-V node. This phenomenon was demonstrated in five patients by using programmed atrial stimulation and His bundle recordings. Entrance block into the A-V node occurred in a narrow range (28–33%) of the basic cycle length and concealed conduction occurred at coupling intervals longer than this range. It appears that entrance block of these early APBs is due to a “functional block” between the atrium and the A-V node. The concept of an entrance block into the A-V node is useful in explaining some forms of the supernormal phase of A-V conduction as well as in the interpretation of complex arrhythmias.

Additional Indexing Words:
Concealed conduction
Full recovery time of A-V node
His bundle recordings

Supernormal phase of A-V conduction
Premature atrial stimulation

IMPULSES which fail to propagate along the entire atrioventricular (A-V) conducting system are called “concealed.” “Concealed conduction” is an electrocardiographic term that refers to the effect which these concealed impulses have on the conduction of subsequent impulses. A premature atrial impulse which is concealed (blocked) within the A-V node may cause delayed A-V nodal conduction of a subsequent atrial beat providing the latter follows the concealed atrial premature beat at a close coupling interval. If the subsequent beat occurs sufficiently late after the concealed atrial premature beat, the A-V node will have recovered and A-V nodal conduction time of the subsequent beat will be normal. Thus, one of the determinants of seeing electrocardiographic evidence of concealed conduction is the occurrence of the subsequent beat before the full recovery time of the A-V node. A second determinant of concealed conduction is that the nonpropagated premature atrial beat must enter the A-V node. If the premature atrial impulse does not enter the A-V node, refractoriness of this structure is not altered, and consequently no conduction delay of subsequent beats would be expected.

This study was undertaken to demonstrate the phenomenon of entrance block into the A-V node by means of His bundle recordings and programmed atrial stimulation. The relationship of this phenomenon to clinical cardiac arrhythmias will be discussed.

Methods

Right heart catheterization was performed on a total of 15 patients in the postabsorptive, nonsedated state. All subjects were normal except
Concealed conduction of an interposed atrial beat \( A_2 \). (Panel A) An \( A_2 \) was introduced at a coupling interval of 290 msec and was not followed by a His bundle electrogram. Due to concealed conduction of \( A_2 \) the subsequent \( A_3 \) had a long \( A_3-H_2 \) interval (370 msec). (Panel B) The influence of concealed conduction was further proved in this panel by omitting \( A_2 \) and demonstrating marked shortening of the \( A_2-H_2 \) interval (160 msec). The illustration shows from top to bottom standard electrocardiographic leads I, II, and III, high right atrial electrogram (HRA), His bundle electrogram (HBE), and time lines \( T \) at intervals of 10 and 100 msec. \( A \) = atrial depolarization; \( H \) = His bundle depolarization; \( V \) = ventricular depolarization; \( S \) = stimulus artifact. This sequence and abbreviations are used also in the subsequent figures.

Figure 1

one who had evidence of left anterior fascicular block in the electrocardiogram. None of the subjects was taking cardiac medications. The nature of the procedure was explained in detail and signed consent obtained from each subject. Bundle of His electrograms were recorded as previously described. Also, previously described, a quadripolar electrode catheter was positioned high in the right atrium for simultaneous bipolar atrial stimulation and recording. The right atrium was stimulated with a programmed digital stimulator*, which delivered rectangular impulses of 1.5 msec at twice diastolic threshold. All equipment was properly grounded.

In all patients the sequence of atrial stimulation was as follows: The atrium was paced at basic cycle length \( (A_1-A_1) \) which was slightly faster than the sinus rate. After every eighth basic beat \( (A_1) \) a premature atrial beat \( (A_2) \) was introduced. The coupling interval for \( A_2 \) (i.e., the

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A1-A2 interval) was the shortest interval producing atrial depolarization which was not followed by a His deflection. This A1-A2 interval was very close to the absolute or effective refractory period of the atrium. The A1-A2 interval was held constant and a third atrial depolarization (A3) was introduced after A2. The A1-A3 interval was then progressively shortened. The conduction time of A3 to the ventricles was measured with and without A2 at progressively shorter A1-A3 intervals. The scan of A1-A3 intervals was limited by that point at which A3 was blocked proximal to the bundle of His.

Three simultaneous ECG leads and time marks at 10 and 100 msec were simultaneously recorded on magnetic tape and subsequently transferred to photographic paper.

The A1-A1, A1-A2, and A1-A3 intervals were measured from the high-right atrial electrograms. The A-H intervals were used as a measure of A-V nodal conduction time.

**Figure 2**

Entrance block into the A-V node for an interposed atrial premature depolarization. (Upper panel) An A3 was introduced at a coupling interval of 270 msec and was not followed by a His depolarization. The subsequent beat introduced at a coupling interval of 620 msec (shorter than the full recovery time of the A-V node at that cycle length showed an A1-A3 interval of 170 msec. (Lower panel) The A2-H2 interval remained the same in the lower panel even though A3 was omitted. This lack of influence of A3 on the subsequent A2-H2 interval suggests that A2 did not enter the A-V node.

**Full Recovery Time of the A-V Node**

The end of the full recovery time of the A-V node for a sinus or paced cycle length is defined as that point in time at which a premature atrial beat conducts with a longer A-H interval than the basic beat. The end of the full recovery time marks the beginning of the relative refractory period of the A-V node.

**Results**

For the purposes of this report, A2 is the concealed or blocked beat, and A3 is the subsequent beat.

The phenomenon of concealed conduction, as illustrated in figure 1, was demonstrated in all patients. In panel A, a premature atrial impulse (A2) was blocked proximal to the bundle of His and the subsequent beat (A3) was conducted with an A3-H3 interval of 370
msec. That A₂ affected the conduction of A₃ is shown in panel B, where A₂ was omitted and the A₃-H₃ interval significantly decreased.

Entrance block into the A-V node was demonstrated in only five of 15 studies. In 10 patients the atrium was refractory to the A₂ stimulus at relatively long A₁-A₂ intervals. The phenomenon of entrance block into the A-V node is illustrated in figure 2 and is reflected in the fact that neither the presence nor absence of A₂ affected the conduction of A₃.

In both figures 1 and 2 the A₃ occurs before the full recovery time of the A-V node, that is, within its relative refractory period, as evidenced by the fact that the A₃-H₃ interval is longer than the A₁-H₁ interval. A₃ was made to occur before the A-V node fully recovered from the basic driving cycle by making the A₁-A₃ interval sufficiently shorter than the A₁-A₁ interval.

Figure 3 is a representative curve demonstrating entrance block into the A-V node for the premature atrial beat (A₂) for various A₁-A₃ coupling intervals which are less than the full recovery time of the A-V node. Basic cycle length is 750 msec. The full recovery time of the A-V node ends at 660 msec as shown by a prolonged A₂-H₃ interval. The curve demonstrates that the premature atrial beat (A₂) has no effect on the conduction of the subsequent beat (A₃) irrespective of whether A₃ is introduced before the full recovery time of the A-V node i.e., A₁-A₃ is less than 660 msec or after the full recovery time of the A-V node (A₁-A₃ greater than 660 msec). The lack of effect of A₂ on the A₃-H₃ interval at longer coupling intervals than 660 msec does not necessarily demonstrate entrance block because at these long coupling intervals the A-V node may have recovered completely from partial penetration of A₂. On the other hand, the fact that the A₃-H₃ interval for shorter coupling intervals than the full recovery period is unaltered by the presence or absence of A₂ does confirm entrance block for A₂.

The A₁-A₂ coupling intervals at which entrance block into the A-V node occurred varied from 210 msec to 290 msec with a mean

![Figure 3](image_url)

*Figure 3*  
Entrance block into the A-V node of A₂ at varying coupling intervals of A₁-A₃. On the abscissa A₁-A₃ intervals and on the ordinate A₃-H₃ intervals are plotted. The circles indicate A₃-H₃ with A₂ and the X's indicate A₃-H₃ without A₂. The arrow indicates the full recovery period of the A-V node at that basic cycle length. The circles and the X's follow the same course. This indicates that A₂ has no influence on the subsequent A₃-H₃ interval even at shorter coupling intervals than the full recovery period of the A-V node and confirms entrance block of A₂ into the A-V node.
value of 257 msec. The percentage of the basic cycle length at which entrance block of A₂ occurred varied from 28 to 33%.

Discussion

Concealed conduction has been extensively studied and has proven a valuable concept in the understanding of simple and complex cardiac arrhythmias.4-10 Less well studied has been the concept of entrance block which has heretofore been applied to certain characteristics of parasystolic foci11 and the sinus node.12 In the present study, the requisite conditions for entrance block of premature impulses into the A-V node have been studied. These include premature atrial impulses (A₂) which occur at 28 to 33% of the preceding cycle length (A₁-A₁) and lack of A-H prolongation of a subsequent beat (A₃) which occurs prior to the full recovery time of the A-V node.

One could argue that the interposed beat (A₂) might have entered only the upper part of the A-V node and by the time the subsequent beat (A₃) had reached the A-V node the upper part had recovered completely. To support this hypothesis one would have to assume that the full recovery time is different for various portions of the A-V node. Moreover, this hypothesis seems unlikely since, as shown in Figure 3, we studied the A-V conduction of the subsequent beat at varying coupling intervals with and without an interposed beat, and even at the shortest coupling interval the interposed atrial premature beat had no effect on A-V conduction. A small degree of penetration cannot be entirely excluded without mapping the whole of the upper part of the A-V node with microelectrodes.

The atrial premature depolarizations that have demonstrated entrance block into the A-V node were introduced at 28 to 33% of the basic cycle length. One could speculate that these early stimuli would produce only a local response and that entrance block is due to the inability of the atrial activation front to reach the A-V node. However, the atrial premature depolarizations that demonstrated entrance block activated the whole atrium as shown by the high and low atrial electrograms and the P wave on the surface ECG. Entrance block of these atrial premature depolarizations is not the result of a local response but is due to a "functional block" at the junction of the atrium and the A-V node.

An alternate explanation for the lack of influence of A₂ on the A-V conduction of a closely coupled subsequent beat (A₃) can be offered by suggesting that A₂ had entered only one pathway or a portion of the A-V node and the other was free and unaffected to conduct A₃ to the ventricle. But A₂ showed concealed conduction at long coupling intervals, and to explain this on the basis of the above hypothesis, one has to assume that at long coupling intervals A₂ engaged both pathways or portions of the A-V node while at short coupling intervals A₂ entered only one pathway or a portion of the A-V node. This theory has not been proved, and these events can be more easily explained on the basis of an entrance block into the A-V node for A₂.

Supernormal conduction has come to denote conduction which is "better" than anticipated under the circumstances and not to conduction which is faster than normal.13 If the concept of entrance block is not applied to explain some of our figures, one might label them as examples of supernormal A-V conduction. A typical example is shown in Figure 2. If one assumes that A₂ did enter the A-V node, then for such a short A₂-A₃ interval A₃ should have a longer A₂-H₃ interval than it did. The absence of a prolonged A₂-H₃ interval might tempt one to designate supernormal A-V conduction for A₃. The mechanism was demonstrated by omitting A₂ in the subsequent panel and A₂-H₃ was unaffected indicating that A₂ never entered the A-V node. The concept of entrance block would help to explain some forms of the so-called supernormal phase of A-V conduction.

In rapid atrial tachyarrhythmias concealment and entrance block may both be occurring. In atrial tachycardia with block and in atrial flutter with varying block some of the blocked beats may not be entering the A-V node. The occasional failure of a single atrial
premature beat to affect a reentrant A-V nodal tachycardia may relate to entrance block into the A-V node of that premature beat. The concept of entrance block might be useful in elucidating some of the complex arrhythmias.

References

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