Dual Atrioventricular Nodal Pathways Associated With a Gap Phenomenon in Atrioventricular Nodal Conduction

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A 67 year old man underwent electrophysiologic study for evaluation of syncope. During atrial pacing at a basic cycle length of 600 ms, atrial premature stimuli were introduced at progressively shorter coupling intervals. The graph of atrial coupling intervals versus corresponding His bundle responses revealed an abrupt increase in progressively premature atrial beats are usually propagated to the ventricle with a gradual increase in the conduction time across the atrioventricular (AV) node until the effective refractory period of some part of the AV conducting system is reached. The smooth progression of times may be distorted by dual pathways within the AV node. The presence of a fast pathway with a long effective refractory period and a slow pathway with a shorter effective refractory period is manifested by a sudden increase in AH intervals as the coupling time of atrial premature beats is shortened.

Another phenomenon that may disrupt the usual sequence of AV conduction times is a so-called gap phenomenon. A gap phenomenon is identified if premature beats at longer and shorter coupling intervals are transmitted normally, whereas beats within the narrow range of the gap are blocked. There are two requirements for a gap to be demonstrated. First, the effective refractory period of a distal structure must exceed the functional refractory period of a proximal structure; second, there must be sufficient delay in the proximal tissue to allow the distal site to recover from its refractoriness (1).

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Case Report

A 67 year old white man was referred for electrophysiologic study to evaluate recurrent syncope in the absence of identifiable cardiac or neurologic causes. Cardiac catheterization 2 years before admission revealed normal coronary arteries and findings consistent with congestive cardiomyopathy. At the time of the study, he was receiving only alpha methyl dopa for hypertension. The electrocardiogram showed sinus rhythm, a normal PR interval and left bundle branch block. Twenty-four hour ambulatory monitoring demonstrated only rare atrial and ventricular premature beats.

Methods. After informed consent was obtained, an electrophysiologic study was performed with the patient in the nonsedated, postabsorptive state. Intracardiac electrograms were recorded at a paper speed of 100 mm using 40 to 500 Hz filtering. The high right atrium was paced at a basic cycle length of 600 ms. After eight drive beats, atrial premature stimuli were introduced at coupling intervals of 490 to 200 ms in 10 ms decrements. The A1A2 interval was measured from the onset of the atrial deflection on the His bundle electrogram after the last basic drive stimulus and the premature stimulus, respectively (2). With the use of the onset of the His bundle deflection, the H1H2 interval was measured in a similar manner.

Results. Increasingly premature atrial stimuli initially resulted in a gradual increase in A2H2 intervals and a smooth decrease in H1H2 intervals. An example of the response to a long coupling interval is shown in Figure IA with an A1A2 interval of 470 ms and a resultant H1H2 interval of 495 ms. As A1A2 decreased to 350 ms, H1H2 diminished to 450 ms.
An abrupt increase in $H_1H_2$ to 670 ms occurred in response to an $A_1A_2$ of 330 ms (Fig. 1C). Further shortening of $A_1A_2$ to 290 ms resulted in a marked decrease in $H_1H_2$ to 450 ms (Fig. 1D). The effective refractory period of the atrium was 280 ms. The results of AV conduction of all atrial premature beats are shown in Figure 2.

**Discussion**

The findings in this patient suggest that two alterations in atrioventricular (AV) nodal conduction are operative: dual AV nodal pathways and a gap phenomenon involving AV nodal conduction.

**Evidence for dual AV conduction pathways and gap phenomenon.** The sudden prolongation in $H_1H_2$ intervals with $A_1A_2$ times of 340 to 320 ms suggests that conduction switched from a fast to a slow pathway. This can occur if the refractory period of the fast pathway is longer than the refractory period of the slow pathway. This change in conduction is generally attributed to dual AV nodal pathways. Usually, conduction over the slow pathway would continue until the prematurity of the $A_1A_2$ intervals became less than the effective refractory period of the slow pathway. Beyond this point, no AV conduction would occur because the effective refractory period of both the fast and slow pathways would have been surpassed. This did not occur in our patient as further decrements in the $A_1A_2$ coupling intervals below 320 ms resulted in the return of fast pathway conduction. This gave rise to an apparent gap in the fast pathway conduction at the $A_1A_2$ intervals from 330 to 310 ms.

The overlap of fast and slow conduction at 330 and 340 ms probably reflects the very critical balance between the refractory periods of the two pathways. Perhaps the most plausible but not the only explanation of this observation is that further slowing in conduction in the proximal AV node allowed for premature impulses of less than 320 ms to arrive at the fast pathway late enough to find it now excitable. Premature impulses at less than 290 ms were completely blocked in the atrium, preventing access to either pathway. Thus, the gap in conduction within the fast pathway may have resulted from differences in refractory periods of the proximal and distal AV node.

**Previous reports.** AV nodal pathways were originally identified by Moe et al. (3) in animal studies and were later verified in human subjects by Denes et al. (4). This lon-
Figure 2. Graph of coupling intervals of all atrial premature beats ($A_1A_2$) versus the His bundle intervals ($H_1H_2$) after atrial premature beats. A small gap is noted between $A_1A_2$ coupling times of 310 to 330 ms. A, B, C and D refer to Figure 1A, B, C and D, respectively.

The coexistence of dual pathways and gap phenomenon. The coexistence of dual AV nodal pathways and gaps in the AV conduction appears to be quite rare. Mirvis and Bandura (8) reported on a patient with dual AV nodal pathways and a gap in conduction between the AV node and distal His-Purkinje system (type I gap). In that case, it was suggested that the dual pathways actually allowed the gap to be manifested. Normal conduction resumed after the gap at shorter coupling intervals, because a proximal delay in the slow pathway allowed recovery to occur in the distal His-Purkinje system. In contrast, our patient demonstrated an AV conduction gap only in the fast pathway. Conduction returned at shorter coupling intervals in the fast pathway because of greater conduction delay at a point proximal to the dual pathways rather than delay within the slow pathway.

Implications. This patient has findings of both dual AV nodal pathways and a gap phenomenon in the fast pathway. It is suggested that the gap may be secondary to differences in the refractory periods of the proximal and distal AV node. To our knowledge, this unique case represents the first report of this type of gap to be associated with dual AV nodal pathways. The observations illustrate the complexities of AV nodal conduction and the possible relation of these complexities to differences in refractoriness and conduction at various sites within the node. However, these findings did not explain the syncopal episodes in this patient.

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References