Pseudosimultaneous Fast and Slow Pathway Conduction: A Common Electrophysiologic Finding in Patients With Dual Atrioventricular Nodal Pathways

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Two ventricular responses following termination of rapid atrial pacing were noted in 24 of 87 patients with dual atrioventricular (AV) nodal pathways and supraventricular tachycardia. In all 24 patients, the AH intervals of the first and second ventricular responses were comparable with those of the fast and slow pathways, respectively. Careful analysis of the whole pacing sequence revealed that, in 21 patients, this phenomenon resulted from sustained slow pathway conduction with long AH intervals. In these patients, as the AH interval of each paced beat was progressively lengthened during pacing, the corresponding His bundle and ventricular responses were pushed one cycle behind the current atrial paced beat, so that the last paced beat was followed by two His bundle and ventricular responses. In only three patients did double ventricular responses result from simultaneous fast and slow pathway conduction. One of these three patients also showed two ventricular responses resulting from sustained slow pathway conduction.

Several factors predispose to the occurrence of this phenomenon in patients with dual AV nodal pathways. These include an ability to sustain slow pathway conduction, a longer slow pathway AH interval, a shorter sinus AH interval (fast pathway) and a shorter atrial paced cycle length that sustains slow pathway conduction. In conclusion, sustained slow pathway conduction with resultant long AH intervals is the mechanism of two ventricular responses following termination of atrial pacing in most patients with dual AV nodal pathways. This phenomenon should be distinguished from the rare occurrence of double ventricular responses to an atrial impulse due to simultaneous fast and slow pathway conduction.

In patients with dual atrioventricular (AV) nodal pathways, an atrial impulse may occasionally conduct simultaneously through both the fast and the slow pathway resulting in double His bundle and ventricular responses (1–5). In such patients, simultaneous fast and slow pathway conduction was usually noted during rapid atrial pacing (1–3). In only three reported patients was this phenomenon noted during sinus rhythm (4,5). Because of the rarity of this phenomenon, its electrophysiologic mechanisms have not been completely elucidated. On the other hand, two His bundle and ventricular responses after termination of rapid atrial pacing are frequently noted during electrophysiologic studies. The electrophysiologic manifestation of this condition and its differentiation from true simultaneous fast and slow pathway conduction have not been previously described. In this study, we evaluated a large group of patients with dual AV nodal pathways and paroxysmal supraventricular tachycardia, and examined the incidence and the mechanism of two His bundle and ventricular responses after termination of atrial pacing.

Methods

Patients. Selection criteria for inclusion of patients in this study were electrocardiographic documentation of supraventricular tachycardia and electrophysiologic demonstration of discontinuous A1A2, A2H2, curves suggesting anterograde dual AV nodal pathways. There were 87 patients, 36 male and 51 female, with ages ranging between 16 and 70 years (mean ± SD 42 ± 15).

Electrophysiologic studies. These studies were performed in a postabsorptive, nonsedated state after informed consent was obtained. A 7 quadripolar electrocath-
eter was percutaneously introduced into the right femoral vein, advanced to the right atrium and positioned across the tricuspid valve. The distal two electrodes were utilized for right ventricular stimulation, while the proximal two electrodes were used for His bundle recording. A second 7 hexapolar electroadie was introduced into the right antecubital vein by a small incision, and was advanced to the right atrium and then into the coronary sinus. The distal two electrodes recorded the left atrial electrogram from the coronary sinus, the middle two electrodes recorded the right atrial electrogram and the proximal two electrodes were used for right atrial stimulation. Multiple surface electrocardiograms as well as intracardiac electrograms were simultaneously recorded on a multichannel oscilloscopic photographic recorder (Electronics for Medicine, VR-16) at a paper speed of 100 mm/s. Electrical stimuli of 2 ms duration and approximately twice diastolic threshold were provided by a programmable digital stimulator (DTU PG 100, M. Bloom).

Anterograde and retrograde conduction properties were evaluated with atrial and ventricular incremental and extrastimulus testing techniques. The atrial extrastimulus was coupled to a sinus cycle length as well as to driven cycle lengths, and the ventricular extrastimulus was coupled to ventricular driven cycle lengths. In each patient, atrial pacing was repetitively performed with decremental cycle lengths until a cycle length was reached that induced second degree AV block proximal to the His bundle recording site. In those patients in whom sustained supraventricular tachycardia was not induced during control study, the study was repeated after intravenous administration of atropine, 0.5 to 1 mg.

Conduction intervals, the refractory period and the echo zone were defined and measured as previously described (6,7). The diagnosis of dual AV nodal pathways and AV nodal reentrant tachycardia was made using the previously described criteria (6–12).

Statistics. Student's t test for unpaired data was used for statistical analyses. All values are expressed as mean ± SD.

Results

In 24 of the 87 patients, the last atrial paced beat was followed by two His bundle and ventricular responses; this phenomenon occurred during the control study in 17 patients, after atropine administration in 5 and both during the control study and after atropine administration in 2. In all 24 patients, the AH interval of the first ventricular response was comparable with the AH interval during fast pathway conduction and was longer than that during sinus rhythm. The AH interval of the second ventricular response was comparable with the AH interval during slow pathway conduction. In 21 of the 24 patients, the second ventricular response was followed by initiation of supraventricular tachycardia.

Study of atrial pacing recordings in the 24 patients elucidated two mechanisms responsible for two His bundle and ventricular responses after the last atrial paced beat; one is sustained slow pathway conduction and the other is simultaneous fast and slow pathway conduction.

Two His bundle and ventricular responses due to sustained slow pathway conduction. This phenomenon was observed in 21 patients. An example is shown in Figure 1; as the atria were paced at a cycle length of 330 ms, the AH interval increased progressively from 105 to 150 ms. The last atrial paced beat was followed by two His bundle and ventricular responses. The AH interval of the first ventricular response was 155 ms and that of the second was 485 ms. The second ventricular response was followed by initiation of supraventricular tachycardia. The ladder diagram in Figure 1 suggests a possible explanation for this phenomenon: Pacing-induced conduction delay in both the fast

Figure 1. Two His bundle and ventricular responses at termination of rapid atrial pacing. Shown are electrocardiographic leads I, aVF and V1, high right atrial electrogram (HRA), left atrial electrogram recorded from the coronary sinus (CS) and His bundle electrogram (HBE). A and H, respectively, refer to low septal right atrial and His bundle responses to the stimulus (S). A and H, respectively, refer to atrial and His bundle responses during induced supraventricular tachycardia. The atrial paced cycle length (CL) was 330 ms. In the ladder diagram, A, AVN and H, respectively, represent the atrium, the atrioventricular node and the His bundle. The solid line represents conduction through the fast pathway and the dashed line conduction through the slow pathway (see text).
and the slow pathway put these two pathways out of phase, so that the last atrial paced beat was conducted simultaneously through both pathways resulting in two His bundle and ventricular responses. Nevertheless, when recordings of the entire pacing sequence were critically analyzed, this explanation proved incorrect.

Figure 2 is a continuous recording of atrial pacing at an identical cycle length in the same patient as in Figure 1. At the onset of atrial pacing, the AH interval increased from 85 to 170 ms, consistent with the fast pathway conduction. The AH interval then jumped to 330 ms and increased further to 485 ms, consistent with a shift of conduction to the slow pathway. As the AH interval of each paced beat was markedly lengthened, the corresponding His bundle and ventricular responses were pushed one cycle behind the current atrial paced beat. Therefore, the last atrial paced beat was followed by two His bundle and ventricular responses, resembling simultaneous fast and slow pathway conduction.

Two His bundle and ventricular responses due to simultaneous fast and slow pathway conduction. In only 3 of the 24 patients was true simultaneous fast and slow pathway conduction with resultant two His bundle and ventricular responses noted during rapid atrial pacing. In one of these three patients, both true and pseudosimultaneous fast and slow pathway conduction were noted after atropine administration (Fig. 3). In Figure 3A, the atria were paced at a cycle length of 360 ms during the control study; the conduction shifted to the slow pathway at the third paced beat resulting in long AH intervals (indicated by an arrow), and the last atrial paced beat was followed by two His bundle and ventricular responses, one in response to the second last paced beat and the other to the last paced beat. Figure 3B to D illustrate recordings of the study performed after atropine administration. In Figure 3B, the atria were paced at a cycle length of 270 ms; the last three paced beats were conducted through the slow pathway (the arrow indicates shift of the conduction pathway) and the last paced beat was followed by two His bundle and ventricular responses, as a result of a long slow pathway conduction. In Figure 3C, the atria were paced at a cycle length of 260 ms; the paced beats were conducted through the fast pathway and the last paced beat was conducted simultaneously through both fast and slow pathways resulting in two His bundle and ventricular responses followed by initiation of supraventricular tachycardia. In Figure 3D, an atrial extrastimulus was coupled to an atrial driven cycle length of 430 ms at a coupling interval of 230 ms and the premature atrial beat was conducted simultaneously through both fast and slow pathways resulting in two His bundle and ventricular responses followed by initiation of supraventricular tachycardia.

Comparison of patients with and without two ventricular responses due to sustained slow pathway conduction. The 19 patients in whom two ventricular responses resulting from sustained slow pathway conduction were demonstrated during the control study were compared...
Figure 3. Recordings from a patient showing two His bundle and ventricular responses due both to sustained slow pathway conduction and to simultaneous fast and slow pathway conduction. HRAe and CSle, respectively, refer to the high right atrial and the coronary sinus response during induced supraventricular tachycardia. HRA1, CS1, A1 and H1, respectively, refer to the high right atrial, the coronary sinus, the low septal right atrial and the His bundle response to the basic atrial driven beat (S1). HRA2, CS2, A2 and H2, respectively, refer to the high right atrial, the coronary sinus, the low septal right atrial and the His bundle response to the atrial extrastimulus (S2). H* and H** refer to the slow pathway His bundle responses during simultaneous fast and slow pathway conduction, respectively, to rapid atrial pacing and to the atrial extrastimulus.

Panel A is the study during the control period. Two His bundle and ventricular responses with initiation of supraventricular tachycardia due to sustained slow pathway conduction are noted at termination of atrial pacing at a paced cycle length (CL) of 360 ms. Panels B, C and D are studies after atropine. In B, two His bundle and ventricular responses with initiation of supraventricular tachycardia due to sustained slow pathway conduction are noted at termination of atrial pacing at a paced cycle length of 270 ms; the last two QRS complexes show right bundle branch block due to a long-short preceding cycle length. In C, two His bundle and ventricular responses with initiation of supraventricular tachycardia due to simultaneous fast and slow pathway conduction are noted at termination of atrial pacing at a paced cycle length of 260 ms. In D, two His bundle and ventricular responses with initiation of supraventricular tachycardia are noted during atrial extrastimulus testing. The atrial extrastimulus is coupled to an atrial driven cycle length of 430 ms at a coupling interval of 230 ms (see text).
with the remaining 68 patients without this finding. The AH interval during sinus rhythm (fast pathway) was significantly shorter in the former than in the latter patients (70 ± 14 versus 84 ± 25 ms, p < 0.05). In contrast to all 19 patients with two ventricular responses, only 42 of the 68 patients without these responses were able to sustain 1:1 slow pathway conduction during rapid atrial pacing. The shortest atrial paced cycle length that sustained 1:1 slow pathway conduction was significantly shorter in the 19 patients with two ventricular responses than in the subgroup of 42 patients without these responses (328 ± 58 versus 387 ± 76 ms, p < 0.01). The AH interval at the shortest atrial paced cycle length that sustained 1:1 slow pathway conduction was significantly longer in the patients with two ventricular responses than in the subgroup of 42 patients without these responses (424 ± 57 versus 337 ± 76 ms, p < 0.001). Similarly, the difference between the slow pathway AH interval and the atrial paced cycle length that sustained 1:1 slow pathway conduction was significantly longer in the former than in the latter patients (97 ± 73 versus 51 ± 76 ms, p < 0.001). The age, sex, cycle length of supraventricular tachycardia and the shortest atrial paced cycle length that maintained 1:1 fast pathway conduction were not significantly different between the two groups.

Discussion

Mechanisms of two ventricular responses at termination of atrial pacing. Double His bundle and ventricular responses to a single atrial impulse due to simultaneous fast and slow pathway conduction has been reported in patients with dual AV nodal pathways (1-5). The occurrence of this phenomenon depends on a critical conduction delay in the slow pathway. In this situation the difference in conduction time between the fast and the slow pathways exceeds the effective refractory period of the distal conduction system, thus allowing the distal conduction system to respond to anterograde impulses from both the fast and the slow pathway. This phenomenon has exclusively been demonstrated during rapid atrial pacing when differential conduction delay in the two pathways put them out of phase (1-3). However, sustained slow pathway conduction occurs frequently during rapid atrial pacing (9). Under such circumstances, the corresponding His bundle and ventricular responses to each atrial paced beat may lag behind the next paced atrial beat, so that, at termination of atrial pacing, the last paced beat is followed by two His bundle and ventricular responses, resembling simultaneous fast and slow pathway conduction. Thus, a misinterpretation of data is made unless the whole pacing sequence is critically analyzed. In patients with simultaneous fast and slow pathway conduction, the atrial paced beats are conducted through the fast pathway with a shorter AH interval; and simultaneous fast and slow pathway conduction with double ventricular responses occurs during pacing or at termination of pacing, resulting in false shortening of the AH interval (during pacing) or in one additional ventricular response at the end of pacing (1-3). In patients with sustained slow pathway conduction and a prolonged AH interval, atrial paced beats are initially conducted through the fast pathway with a short AH interval, and then their conduction shifts to the slow pathway with gradual prolongation of AH intervals. Throughout pacing, a 1:1 AV relation is maintained without an additional ventricular response (9).

Factors predisposing to the occurrence of two ventricular responses due to sustained slow pathway conduction. In patients with dual AV nodal pathways, several factors predispose to the occurrence of two ventricular responses due to sustained slow pathway conduction. As was shown in this study, these patients were able to sustain slow pathway conduction during rapid atrial pacing, resulting in longer slow pathway AH intervals. The shortest atrial paced cycle length that maintained 1:1 slow pathway conduction was also significantly shorter. This is understandable, because a shorter atrial paced cycle length facilitates achievement of a longer slow pathway AH interval, and thus, the difference between the slow pathway AH interval and the atrial paced cycle length is more likely to exceed the fast pathway AH interval (a prerequisite of this phenomenon).

Conclusions. In patients with dual AV nodal pathways, two ventricular beats frequently follow the last atrial paced beat at termination of rapid atrial pacing. In most instances, this phenomenon results from sustained slow pathway conduction with long AH intervals. Only rarely does this phenomenon result from double ventricular responses to a single atrial impulse due to simultaneous fast and slow pathway conduction. Careful analysis of the whole pacing sequence enables one to make a distinction between the two.

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References


