Double Atrial Responses to a Single Ventricular Impulse Due to Simultaneous Conduction Via Two Retrograde Pathways

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Electrophysiologic studies were performed in two patients. In one patient (Case 1) with ventricular pre-excitation and paroxysmal supraventricular tachycardia, studies after diltiazem administration showed two QRS responses to a single atrial stimulus during atrial pacing at a cycle length of 300 ms. The first QRS response with full pre-excitation and short PR interval was consistent with accessory pathway conduction, while the second QRS response with a normal duration and an atrio-His bundle interval of 350 ms was consistent with normal pathway conduction. The second QRS response was followed by initiation of supraventricular tachycardia.

Studies after verapamil administration on a separate day disclosed two atrial responses to a single QRS complex during ventricular pacing at cycle lengths between 330 and 280 ms, suggesting simultaneous retrograde accessory and normal pathway conduction.

In Case 2 with a supraventricular tachycardia using a fast atrioventricular nodal pathway for anterograde Simultaneous conduction of an atrial impulse through a fast and a slow atrioventricular (AV) pathway resulting in double ventricular responses has been described previously in patients with ventricular pre-excitation (1–5), with a James tract (6) and with dual AV nodal pathways (7–11). Wu et al. (7) predicted that double atrial responses to a single QRS complex during ventricular pacing at cycle lengths between 330 and 280 ms, suggesting simultaneous retrograde accessory and normal pathway conduction.

In Case 2 with a supraventricular tachycardia using a fast atrioventricular nodal pathway for anterograde conduction, two atrial responses to a single QRS complex were observed during ventricular pacing at cycle lengths between 500 and 400 ms. The first atrial response showed a stimulus to atrial interval of 120 ms and an atrial activation sequence with the low septal right atrium being earlier than other atrial sites, suggesting retrograde fast pathway conduction. The second atrial response showed a stimulus to atrial interval of 505 ms and an atrial activation sequence with low septal right atrium being simultaneous with the proximal coronary sinus, suggesting retrograde slow pathway conduction. Thus, double atrial responses to a single QRS complex may result from simultaneous conduction through two retrograde pathways when pacing induced conduction delay in the slower pathway is sufficient to allow recovery of the atria to respond to the second slower retrograde impulse.

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Anterograde and retrograde conduction properties were evaluated by incremental pacing and extrastimulus testing techniques. The conduction intervals and refractory periods were measured and defined as previously described (12). According to previously described criteria, diagnoses were made of 1) the usual form (slow-fast) of AV nodal reentry using a slow AV nodal pathway for anterograde and a fast AV nodal pathway for retrograde conduction (12,13), 2) orthodromic AV reentry using a retrogradely conducting accessory pathway (12), 3) the unusual form (fast-slow) of AV nodal reentry using a fast AV nodal pathway for anterograde and a slow AV nodal pathway for retrograde conduction (12,13), and 4) the unusual form of AV reentry using a slowly conducting retrograde paraseptal accessory pathway (14,15). The usual form of AV nodal reentry or orthodromic AV reentry is characterized by tachycardia with a retrograde P wave occurring either simultaneously with or slightly after the QRS complex. The unusual form of AV nodal reentry or AV reentry is characterized by tachycardia with a retrograde P wave occurring before the QRS complex.

In one patient (Case 1), anterograde and retrograde refractory periods were measured at a basic driven cycle length of 500 ms; in the other (Case 2), the anterograde refractory periods were measured during sinus rhythm and the retrograde refractory periods were measured at a basic driven cycle length of 500 ms. In both patients, the quadripolar electrocatheter was removed after the initial study, and the hexapolar electrocatheter was repositioned at the right ventricular apex and secured for subsequent serial drug studies.

Definitions. HRA, CS, A, H and V, respectively, refer to high right atrial, left atrial, low septal right atrial, His bundle and ventricular responses to the driven stimulus (S). HRA₁, CS₁, A₁, H₁ and V₁, respectively, refer to high right atrial, left atrial, low septal right atrial. His bundle and ventricular responses to sinus impulse or basic driven stimulus (S₁). HRA₂, CS₂, A₂, H₂ and V₂, respectively, refer to high right atrial, left atrial, low septal right atrial. His bundle and ventricular responses to the first extrastimulus (S₂). HRA₃, CS₃, A₃, H₃ and V₃, respectively, refer to high right atrial, left atrial, low septal right atrial. His bundle and ventricular responses to the second extrastimulus (S₃). HRA₄, CS₄, A₄, H₄ and V₄, respectively, refer to high right atrial, left atrial, low septal right atrial. His bundle and ventricular responses during induced echoes or paroxysmal supraventricular tachycardia.

Results

Case 1

This was a 32 year old woman with a 20 year history of episodes of palpitation. The electrocardiogram at rest showed type B ventricular pre-excitation. The initial electrophysiologic study showed a ratio of 1:1 accessory pathway conduction to an atrial paced cycle length of 330 ms. The anterograde effective refractory period of the accessory pathway was 280 ms; the atrial functional refractory period was 250 ms, which limited AV conduction. Supraventricular tachycardia was inducible at A₁-A₂ intervals between 290 and 250 ms when anterograde conduction failed in the accessory pathway. Retrograde study showed 1:1 ventriculoatrial (VA) conduction to a ventricular paced cycle length of 280 ms. The VA internal (measured from stimulus to the high right atrial electrogram, S-HRA) was 140 ms. Supraventricular tachycardia was inducible at ventricular paced cycle lengths between 330 and 280 ms. The effective refractory period of the ventricle was 220 ms, which limited VA conduction.

Double ventricular responses to a single atrial impulse. After the initial study, 14 mg of diltiazem was administered intravenously over 1 minute. Repeat study showed a 1:1 ratio for accessory pathway conduction to an atrial paced cycle length of 280 ms. An atrial paced cycle length of 300 ms, the atrial stimulus was frequently followed by two QRS complexes. Figure 1A shows termination of rapid atrial pacing at a cycle length of 300 ms. The last atrial stimulus was followed by two QRS complexes. The first QRS complex showed full ventricular pre-excitation consistent with accessory pathway conduction. The second QRS complex had a normal QRS duration and was preceded by a His bundle response with an AH interval of 350 ms consistent with normal pathway conduction. The second QRS complex was followed by initiation of supraventricular tachycardia. The anterograde effective refractory periods of the accessory pathway and the AV node were 270 and 250 ms, respectively. Retrograde study showed 1:1 VA conduction to a ventricular paced cycle length of 280 ms. The ventricular effective refractory period of 210 ms limited VA conduction.

Double atrial responses to a single ventricular impulse. The patient was then given oral verapamil, 80 mg every 6 hours, and a repeat electrophysiologic study was performed 5 hours after the fourth dose. A 1:1 accessory pathway conduction was noted to an atrial paced cycle length of 280 ms. The effective refractory period of the accessory pathway was 260 ms; the atrial functional refractory period was 260 ms, which limited AV conduction. Retrograde study showed that at ventricular paced cycle lengths between 330 and 280 ms, the ventricular stimulus was frequently followed by two atrial responses. Figures 1B and C, respectively, show double atrial responses to a single ventricular stimulus (beats with an asterisk) at ventricular paced cycle lengths of 330 and 280 ms. The retrograde conduction interval of the first atrial response (S-HRA) remained fixed at 140 ms. However, the retrograde conduction interval of the second atrial response (S-HRA') shortened from 380 to 350 ms as the ventricular paced cycle length shortened from 330 to 280 ms. The ladder diagram in panel C explains this phenomenon. The ventricular effective refractory period was 250 ms, which limited VA conduction.
Figure 1. Case 1. Double ventricular responses to a single atrial impulse and double atrial responses to a single ventricular impulse. Electrocardiographic lead \( V_{10} \), high right atrial electrogram (HRA) and His bundle electrogram (HBE) are shown. HRA, A and H, respectively, refer to high right atrial, 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 paroxysmal supraventricular tachycardia. HRA' refers to the second high right atrial response to a single ventricular impulse. In the ladder diagram at the bottom, A = atria, AV = atrioventricular conducting system and V = ventricles. The solid line represents conduction through the fast pathway and the interrupted line represents conduction through the slow pathway. Panel A, Double ventricular responses to a single atrial impulse during cessation of rapid atrial pacing at a paced cycle length (CL) of 300 ms. The last paced stimulus was followed by two QRS complexes. The second QRS complex was followed by initiation of supraventricular tachycardia. Note that the configuration of the second QRS response was identical to the subsequent QRS complexes during supraventricular tachycardia. Panel B, Double atrial responses to a single ventricular impulse during rapid ventricular pacing at cycle lengths of 330 and 280 ms, respectively. The beats marked with an asterisk were followed by two atrial electrograms. The beats without an asterisk were blocked in the retrograde direction. This phenomenon is illustrated in the ladder diagram.

Case 2

This patient was a 67 year old man with a 2 year history of episodes of palpitation. The electrophysiologic study showed an AH interval of 65 ms and an HV interval of 40 ms. The AH interval increased slightly from 65 to 85 ms as the atrial paced cycle length decreased from 600 to 360 ms. Second degree AV block proximal to the His bundle recording site was noted at an atrial paced cycle length of 330 ms. An unusual form of supraventricular tachycardia was inducible at atrial paced cycle lengths between 460 and 360 ms (Fig. 2). The tachycardia was characterized by a cycle length of 415 ms, an A-H interval of 79 ms and an H-A interval of 336 ms. A single ventricular extrastimulus delivered during tachycardia failed to reset the atria. Atrial extrastimuli were coupled to sinus rhythm at a cycle length of 700 ms; the A-H interval increased slightly from 65 to 85 ms as the A-A interval decreased from 600 to 265 ms. The unusual form of supraventricular tachycardia was inducible at A-A interval between 320 and 265 ms (Fig. 3A). A sudden jump of A-A interval to 390 ms was noted at the A-A interval of 255 ms; this was associated with the induction of a usual form of slow-fast AV nodal reentrant atrial echo (Fig. 3B). An A-A interval was occasionally blocked in the AV node at an A-A interval of 255 ms, which also defined the atrial functional refractory period (Fig. 3C).

Incremental ventricular pacing showed a sudden prolongation of VA conduction time as the paced cycle length decreased from 400 to 360 ms; this was associated with change of the retrograde atrial activation sequence. The low septal right atrium was activated earlier than other atrial recording sites at ventricular paced cycle lengths between 500 and 400 ms. The interval between ventricle and low septal right atrium (VA) was 120 ms, between ventricle and coronary sinus (V-CS) was 165 ms and between ventricle and high right atrium (V-HRA) was 165 ms. The atrial
Figure 2. Case 2. Induction of an unusual form of supraventricular tachycardia with rapid atrial pacing. Electrocardiographic leads I, aVF, and V₁ and left atrial electrogram recorded from the coronary sinus (CS) are also shown. The atrial paced cycle length was 460 ms in panel A and 400 ms in panel B. Supraventricular tachycardia was induced at an AH interval of 65 ms and was characterized by an Aₑ-Hₑ-Hₑ-Aₑ ratio of less than 1.

The atrial electrogram recorded from the proximal coronary sinus was activated simultaneously with that recorded from the low septal right atrium at a ventricular paced cycle length of 360 ms. The VA interval was 450 ms, V-CS was 450 ms and V-HRA was 480 ms. Second degree VA block occurred at a ventricular paced cycle length of 300 ms. The unusual form of supraventricular tachycardia was induced at ventricular paced cycle lengths between 360 and 300 ms. Ventricular extrastimuli were coupled to a ventricular driven cycle length of 500 ms. The V₂-A₂ interval increased from 120 to 180 ms as the V₁-V₂ interval decreased from 460 to 310 ms. A sudden jump of the V₂-A₂ interval from 180 to 300 ms was noted when the V₁-V₂ interval decreased from 310 to 300 ms. The ventricular functional refractory period of 250 ms limited VA conduction.

Double atrial responses to a single ventricular impulse. A single ventricular stimulus that gave rise to double atrial responses was observed during ventricular pacing at cycle lengths between 500 and 400 ms (Fig. 4). The first three ventricular stimulating beats in Figure 4A and B were the basic driven beats at a cycle length of 500 ms. The VA conduction time of the first two beats showed a stimulus to low septal right atrium interval (S₁-A₁) of 120 ms, a stimulus to coronary sinus interval (S₁-CS₁) of 165 ms and a stimulus to high right atrium interval (S₁-HRA₁) of 165 ms; the A₁ interval was activated earlier than CS₁ and HRA₁. The third ventricular stimulating beat had a shorter VA conduction time and with an S₁-CS₁' of 50 ms and an S₁-HRA₁' of 80 ms; CS₁' was activated earlier than HRA₁'.

This phenomenon is explained in the ladder diagram at the bottom of Figure 4. The first ventricular stimulus was conducted to the atria through the fast AV nodal pathway with a normal retrograde atrial activation sequence and a shorter conduction time. The second ventricular stimulus was conducted to the atria through both the fast AV nodal and the slowly conducting retrograde VA pathway (either a slow AV nodal pathway or a septal accessory pathway), resulting in double atrial responses. The first atrial response with a normal retrograde atrial activation sequence and a shorter VA conduction time was consistent with conduction through the fast AV nodal pathway, whereas the second atrial response with an abnormal retrograde atrial activation sequence and a longer VA conduction time was consistent with conduction through the slow VA pathway. Superimposition of the atrial response and the QRS complex of the third ventricular stimulating beat resulted in a pseudoshortening of VA conduction time. In fact, the third ventricular stimulating beat was blocked in the retrograde direction.

In Figure 4A, S₂ was delivered at the S₁-S₂ interval of 300 ms, and was conducted to the atria through the slow VA pathway with the S₂-A₂ interval of 300 ms. Two ventricular repetitive responses (V₃ and V₄) were provoked;
Figure 3. Case 2. Induction of the unusual form of supraventricular tachycardia with an atrial extrastimulus and demonstration of dual AV nodal pathways. A₁ and H₁ refer to low septal right atrial and His bundle responses to the basic sinus beat. A₂ and H₂ refer to the low septal right atrial and His bundle responses to the extrastimulus (S₂). The sinus cycle length was 700 ms, and the A₁-H₁ interval was 65 ms. Panel A, Induction of the unusual form of paroxysmal supraventricular tachycardia at an A₁-A₂ coupling interval of 300 ms that achieved an A₂-H₂ interval of 85 ms. Panel B, Induction of the usual slow-fast form of AV nodal reentrant echo at an A₁-A₂ interval of 255 ms when an A₂-H₂ interval suddenly increased to 390 ms. Panel C, A₂ was occasionally blocked proximal to H at an A₁-A₂ interval of 255 ms.

Subsequent electrophysiologic study showed that lidocaine had no effect on the reentrant circuit and supraventricular tachycardia, although digoxin, propranolol, verapamil and procainamide depressed the retrograde slow pathway conduction and prevented induction of sustained supraventricular tachycardia.

Discussion

Double ventricular responses to a single atrial impulse. The major determinant of double ventricular responses to a single atrial impulse is the presence of a fast and a slow AV conducting pathway. The fast pathway can be an accessory pathway, a James tract or a fast AV nodal pathway, while the slow pathway can either be a normal AV node-His bundle pathway or a slow AV nodal pathway (1–11). Under such circumstances, conduction delay in the slow pathway induced by rapid atrial pacing or atrial premature stimulation may put the conduction in both pathways out of phase so the distal conduction system and ventricles are able to respond to both anterograde fast and slow pathway impulses. If the conduction delay in the slow pathway is prolonged enough, the fast pathway may recover suffi-
Figure 4. Case 2. Double atrial responses to a single ventricular impulse, and induction of both the unusual form of supraventricular tachycardia and the usual slow-fast AV nodal reentrant echo with ventricular stimulation. CS₂ refers to the left atrial response to S₁; CS₃ refers to the left atrial response to S₂; HRA₁, CS₁, and A₁, respectively, refer to high right atrial, left atrial and low septal right atrial responses to a spontaneous premature ventricular beat after S₁; or a second extrastimulus (S₂); HRA₂, CS₂, and A₂, respectively, refer to high right atrial, left atrial and low septal right atrial responses to a second spontaneous premature ventricular beat after S₁ or a third extrastimulus (S₃). The conduction intervals are listed. The basic driven cycle length was 500 ms. In both panels A and B, the first beat was conducted to the atria through the fast pathway; the second beat (with an asterisk) was conducted to the atria through both the fast and slow pathways resulting in double atrial responses; and the third beat was blocked in both pathways. Panel A, S₁ was coupled at an S₁-S₂ interval of 300 ms and conducted to the atria through the slow pathway. Two premature ventricular beats, V₁ and V₄, were provoked, and both were conducted to the atria through the slow pathway. The unusual form of supraventricular tachycardia was induced after beat V₄. Panel B, S₂ was coupled at an S₂-S₃ interval of 320 ms and was conducted to the atria through the fast pathway. An S₃ was coupled to S₄ at an S₃-S₄ interval of 310 ms and conducted to the atria through the slow pathway. The S₄ induced the usual slow-fast form of AV nodal reentrant echo. Note that the retrograde atrial activation sequence during fast pathway conduction and during the usual slow-fast form AV nodal reentrant echo was identical, but was different from that during slow pathway conduction. The retrograde atrial activation sequence during slow pathway conduction and during the unusual form of paroxysmal supraventricular tachycardia was identical. The phenomena observed in panel B are illustrated in the ladder diagram below.

Double ventricular responses with initiation of sustained supraventricular tachycardia occurred in our patient with type B ventricular pre-excitation after administration of diltiazem (Case 1). Prolongation of AV nodal conduction time with diltiazem provided the necessary condition for simultaneous anterograde accessory and normal pathway conduction in this patient. Initiation of sustained supraventricular tachycardia after the second ventricular response has not been noted in patients with ventricular pre-excitation.

With the exception of one patient reported by Pimenta et al. (5), the QRS configuration of the second ventricular response in all reported cases with ventricular pre-excitation had a pattern of left bundle branch block. Thus, the possibility of the second ventricular response being reentry within the bundle branch system or local intraventricular
reentry could not be excluded absolutely. In our patient, double ventricular responses to a single atrial impulse occurred during rapid atrial pacing rather than during premature atrial stimulation. Reentry within the bundle branch system is not likely to occur during rapid atrial pacing. In addition, the QRS duration of the second ventricular response was normal and its configuration was identical to that of subsequent QRS complexes during supraventricular tachycardia. These findings strongly suggest that simultaneous anterograde accessory and normal pathway conduction was responsible for the double ventricular responses seen in this patient.

**Double atrial responses to a single ventricular impulse.** Double atrial responses to a single ventricular impulse due to simultaneous retrograde fast and slow pathway conduction has not been previously described. In our patient with type B ventricular pre-excitation (Case 1), the retrograde fast pathway was the accessory pathway, while the retrograde slow pathway was the normal pathway. Prolongation of retrograde AV nodal conduction by verapamil could have provided sufficient time for the atria to respond to the second retrograde impulse from the normal pathway. As anterograde AV nodal block to the reentrant atrial impulse (a concealed event) initiated by the retrograde accessory pathway impulse is required for successful retrograde normal pathway conduction, verapamil may have facilitated simultaneous accessory and normal pathway conduction by increasing the anterograde AV nodal refractoriness. The finding that the retrograde normal pathway conduction interval during simultaneous accessory and normal pathway conduction (S-HRA') was shorter at a faster ventricular paced rate is consistent with this hypothesis. Therefore, less anterograde AV nodal concealed conduction by the reentrant atrial impulse could have accounted for the shortening of S-HRA' at a faster ventricular paced rate.

In our patient with dual AV nodal pathways and an unusual form of supraventricular tachycardia (Case 2), the fast retrograde pathway was the fast AV nodal pathway with a normal retrograde atrial activation sequence, while the slow retrograde pathway could represent either a slow AV nodal pathway or a slow accessory paraseptal pathway with an abnormal retrograde atrial activation sequence (12–15). However, the slow retrograde pathway used during simultaneous retrograde fast and slow pathway conduction and during the unusual form of supraventricular tachycardia could not be the same anterograde slow AV nodal pathway that was demonstrated by atrial extrastimulus testing. If they were the same, the AV nodal reentrant echo using the slow AV nodal pathway for anterograde and the fast AV nodal pathway for retrograde conduction could not have been induced by the ventricular extrastimulus when retrograde conduction occurred through the retrograde slow pathway and returned anterogradely through the slow AV nodal pathway.

**Thus, there were three AV pathways in this patient (Case 2).** The fast AV nodal pathway was capable of both anterograde and retrograde conduction, whereas the slow AV nodal pathway was probably capable of only anterograde conduction. The slow retrograde pathway, most likely representing a paraseptal accessory pathway, was probably capable of only retrograde conduction. The presence of triple pathways in this patient provided a substrate for multiple reentries, although clinically only the unusual form of supraventricular tachycardia using the fast AV nodal pathway for anterograde and the paraseptal accessory pathway for retrograde conduction was observed. Simultaneous retrograde fast and slow pathway conduction in this patient also resulted in pseudoshortening of VA interval during ventricular pacing. Pseudoshortening of the A-H interval during atrial pacing due to simultaneous fast and slow pathway conduction in a patient with dual AV nodal pathways has been described previously (8).

**Implications.** Double atrial responses to a single ventricular impulse may result from simultaneous conduction through two retrograde pathways when pacing-induced conduction delay in the slower pathway is sufficient to allow recovery of the atria and response to the second slower retrograde impulse. This phenomenon, when it occurs during ventricular pacing, may be misinterpreted as second degree VA block. When it occurs during spontaneous ventricular beats, the second atrial response may mimic a late blocked atrial ectopic beat, and thus complicate electrocardiographic interpretation. It may also initiate the “endless loop tachycardia’’ in patients with a physiologic dual chamber pacemaker (16).

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**References**


