Unusual Properties of Accessory Pathways

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SUMMARY

Electrophysiologic evaluation in five patients who exhibited manifestations of the pre-excitation syndrome and/or supraventricular tachycardia provided functional evidence that the properties of the accessory fibers may be quite variable. The unusual responses to electrical stimulation in these patients included: 1) failure to depolarize the His bundle from the site of ventricular pre-excitation; 2) supraventricular tachycardia with two types of re-entry; 3) anterograde block in the accessory pathway with intact retrograde conduction; and 4) retrograde delay or block in the accessory pathway and A-V node with intact anterograde conduction.

In addition to these anomalies in conduction over accessory pathways we demonstrated in these patients the occurrence of retrograde functional right bundle branch block and retrograde "gap" phenomenon, and ventricular reciprocation without recording intervening atrial discharge.

DURING ELECTROPHYSIOLOGIC EVALUATION of patients who exhibited manifestations of the pre-excitation syndrome and/or supraventricular tachycardias, we observed a group of patients who demonstrated unusual responses to electrical stimulation. Taken singly, these patients represent merely a series of isolated examples; taken as a group, however, they illustrate the complexities and variable properties of accessory conduction and underscore the need for thorough electrophysiologic evaluation to expose and understand these variations. It is the purpose of this report to present data derived from these studies to support the following observations: 1) failure to depolarize the His bundle from the site of ventricular pre-excitation; 2) supraventricular tachycardia with two types of re-entry; 3) anterograde block in the accessory pathway with intact retrograde conduction; and 4) retrograde delay or block in the accessory pathway and A-V node with intact anterograde conduction.

Methods

Data obtained from five patients (table 1) comprise the basis for this report. All patients were informed about, and consented to, the study. The patients were selected for electrophysiologic evaluation because they manifested electrocardiographic evidence of ventricular pre-excitation or had hard-to-control supraventricular tachycardias (SVT). Patients were not receiving cardiac medications at the time of study.

In all five patients, right and left atrial activity was evaluated by means of multiple standard surface electrocardiograms and simultaneous bipolar tracings obtained with quadripolar (USCI 5655) and bipolar (Medtronic 5821) electrode catheters placed, respectively, within the right atrial cavity and the esophagus, posterior to the left atrium. In addition, bundle of His recordings were obtained using standard methods. Tracings were recorded on a multichannel oscilloscopic photographic recorder (Electronics for Medicine DR8) at a paper speed of 100 mm/sec, using filter settings between 40 and 500 Hz and 1 second time lines. Bipolar atrial or ventricular pacing was performed through the distal two poles of the quadripolar electrode catheter, using a pulse generator which delivered pulses of 2 msec duration and 2 to 4 volts intensity, through an isolation transformer (Digipulser Model 830, Isopulser Model 850, WP Instruments, Inc.).

Refractory periods were determined by terminating a train of ten to twelve regularly occurring basic stimuli (S1) with a premature stimulus (S2) at progressively shorter S1-S2 intervals. The anterograde effective refractory period of the atrioventricular (A-V) node (ERP A-VN) for a specific basic driving rate is defined as the longest A1-A2 interval following which A2 just failed to reach the bundle of His. The
antegrade functional refractory period of the A-V node (FRPAVN) is defined as the shortest H1-H2 interval conducted from the atrium. The longest A1-A2 interval which just failed to reach the ventricles over the anomalous pathways as judged by a QRS complex which did not display pre-excitation characteristics and had normal or prolonged values for A-H and H-V intervals is defined as the refractory period of the anomalous pathway.

Results

Failure to Depolarize the His Bundle from the Site of Ventricular Pre-excitation

Case 1. A 13-year-old boy presented with a long history of palpitations. Physical examination was normal. ECG revealed type B pre-excitation. Early premature atrial stimulation repeatedly precipitated atrial flutter as the sole tachycardia during the course of study.

During basic cycle lengths (BCL) of 700 and 710 msec, the His bundle spike was lost within the inscription of the ventricular electrogram (fig. 1A, C). Premature atrial stimulation (A1-A2, 320-330 msec) produced expected A-V nodal delay with A-H prolongation and a constant A-V interval (80 msec). At these A1-A2 intervals, activation of the bundle of His occurred near or following the completion of ventricular depolarization, after A-H intervals of 215 and 225 msec. The QRS complex failed to alter significantly in the premature cycle, presumably because conduction traveled to the ventricles primarily over the accessory pathway during the basic cycles. Shortening the premature cycle by 10 msec (fig. 1B, D), resulted in conduction delay or block in the accessory pathway, further A-H prolongation (220 msec and 235 msec) and a less abnormal QRS contour. His activation now preceded ventricular discharge by a short interval (18 msec and 15 msec) and maintained a contour similar to the His deflection seen in figure 1A and 1C. The more prolonged A-H intervals following longer A1-A2 intervals in figure 1C, D may reflect a slight change in vagal tone or the slight difference in BCLs.

Antegrade conduction must have been responsible for His discharge in figure 1B and 1D. A comparison between figure 1A and 1B, and between figure 1C and 1D reveals similar His contour and A-H interval, and makes it likely that antegrade conduction was responsible for His activation in the premature cycles shown in figures 1A and 1C also. If this is so, then ventricular activation in response to the premature atrial stimulus in figure 1A and 1C failed to activate His retrogradely in time to prevent His discharge over the normal A-V pathway; i.e., retrograde conduction delay of block existed between ventricular depolarization initiated by conduction over the bypass tract and the His bundle. An alternate, equally plausible explanation is that the His bundle, depolarized during the ventricular response to A1, was still refractory when the ventricles depolarized in response to A2. The refractory period of the His bundle would therefore have to exceed 320-330 msec. With sufficient A-H delay, it might be possible for the premature impulse traveling the normal A-V nodal route (fig. 1A and 1C) to depolarize the ventricles a second time following initial ventricular activation over the bypass route, or perhaps to establish A-V nodal re-entry unrelated to the bypass tract (see case 2).
The short H-V interval and less abnormal QRS distortion seen in response to the premature atrial stimulus in figure 1B and 1D is also of interest. Three possible explanations exist: 1) Conduction reached the ventricles over an infra-Hisian tract. The patient could have two accessory A-V pathways "in parallel," comprised of a Kent bundle from atrium to ventricle, and Mahaim fibers from His to ventricle. Block in the Kent bundle would allow ventricular activation to occur over the Mahaim fibers, resulting in a shortened H-V interval and QRS distortion. 2) Conduction in the accessory pathway did not block entirely, but traveled slowly to the ventricles and arrived slightly ahead of conduction traveling in a parallel fashion over the normal A-V route, resulting in a fusion QRS complex. The varied QRS contour seen in figure 1B and 1D and changing H-V interval support the presence of fusion complexes. The small changes in H-V interval cannot be considered accurate measurements at a paper speed of 100 mm/sec, but do indicate that the H-V times varied with the different QRS contours. 3) The spike recorded was a right bundle branch (RBB) potential. We did not attempt His bundle pacing, which might have been helpful. Although we can not exclude the possibility that the spike recorded represented a RBB potential, it seems less likely because of the large A deflection and because of the normal H-V interval (35 msec) recorded on other occasions without shifting the catheter electrode, e.g. following the precipitation of atrial flutter (bottom panel, fig. 1).

Assuming the deflection to be a RBB spike still would not aid in understanding the varying degrees of QRS distortion following A2 in figure 1B and D and would make the late spike in response to A2 in figure 1A and C a RBB potential, indicating that ventricular depolarization failed to activate the RBB.

**SVT With Two Types of Re-entry**

Case 2. A 42-year-old white male for many years had SVT resistant to medical control. Physical examination was normal. Electrocardiogram revealed type A pre-excitation syndrome established during right atrial pacing. Premature atrial stimulation precipitated SVT which manifested two different types of QRS complexes, arbitrarily called type I and type II (fig. 2), which were thought to be due to re-entry over different pathways. The terms, type I and type II are used merely for convenience and not necessarily to suggest a classification of re-entrant mechanisms in the WPW syndrome.

Type I SVT. During shorter basic driving cycles, A1-A2 intervals between 220-240 msec resulted in H1-H2 intervals of 290-400 msec and precipitation of type I SVT on 12 occasions (fig. 3, table 2). Conduction to the ventricles in response to A2 must have occurred totally over the anomalous pathway because H2 followed V2 and QRS contours of the premature complex showed maximum aberration. A2-V2 intervals did not lengthen and V1-V2 intervals remained within 5 msec of their respective A1-A2 intervals. On each occa-
sion, SVT was established with the same specific pattern for the sequence of atrial activation as recorded in three atrial leads: low right atrium, followed by high right atrium and activity recorded in the esophageal lead, simultaneously. Activity recorded in the His bundle electrode lead revealed the sequence of activation to be ventricles-His-atria (figs. 2 and 3). The retrograde H-A interval measured only 50 msec (at rates of 170-200/min) and we considered the possibility that retrograde conduction, as well as anterograde conduction, occurred over a bypass tract. However, on three occasions, an A1-A2 interval of 190 msec to 200 msec was followed by an H1-H2 interval of 295 msec to 305 msec and an atrial echo but no ventricular complex (fig. 4). A series of spontaneous complexes often followed. Block of A2 must have occurred in the bypass tract as well as distal to the His bundle. This observation strongly suggested that A-V nodal re-entry provided the pathways for the atrial echo and that the accessory pathway was not required. If A-V nodal re-entry were also the mechanism maintaining type I SVT, then the H-A interval would not represent a true measure of retrograde conduction time but would be influenced by the point at which the reflected wave front returned to the atrium; therefore, a second bypass tract is not necessary to account for the short H-A interval.

Premature atrial stimulation during type I SVT affected the accessory and normal pathways differently and provided further support for this argument. Early premature right atrial stimulation conducted to the ventricles over the accessory pathway, but blocked proximal to the His bundle and terminated type I SVT (fig. 5). This observation supports the importance of A-V nodal conduction to perpetuate type I SVT. Later premature atrial stimulation conducted to the ventricles over the accessory pathway, but in addition, reached the His bundle and simply shifted following cycles to an earlier time without terminating the SVT. Of note was that, for this later premature atrial cycle, the A-H interval lengthened while the A-V interval remained constant, supporting the presence of anterograde conduction to both His bundle and ventricle, but over different pathways.

We produced A-V nodal Wenckebach at atrial cycle lengths of 240 msec (faster rates were not employed) in an attempt to initiate type I SVT. However, 1:1 ventricular activation occurred over the accessory pathway and type I SVT was not initiated, possibly because the short atrial cycle lengths prevented A-V nodal return from discharging the atria (see fig. 15, reference *). Pacing the atrium at slower cycle lengths (290 msec) initiated type I SVT but the mechanism was not clear.

![Figure 4](http://circ.ahajournals.org/)

**Figure 4**

Case 2. Atrial echo with block distal to the bundle of His following an A1-A2 interval of 200 msec. The first atrial deflection is a spontaneous sinus impulse, followed by two atrial stimuli (pacemaker spikes indicated by arrows). Last stimulus (unlabeled) was ineffective owing to atrial refractoriness. Conventions as in figure 1.
In summary, the most attractive, simple but inclusive explanation for the type I SVT appears to be an SVT maintained by A-V nodal re-entry, independent from the accessory pathway. However, anterograde conduction traveled from the atrium to the ventricles over the bypass tract, but the latter was not a part of the re-entry circuit.

Type II SVT. During slightly longer BCLs in this same patient, A1-A2 intervals between 220-230 msec failed to reach the ventricles over the bypass tract and resulted in H1-H2 intervals between 300-310 msec, and V1-V2 intervals between 350-370 msec. H2-V2 interval lengthened (table 2) and the QRS contours exhibited a left bundle branch block (LBBB). The first atrial echo following A2 and therefore, the onset of type II SVT, occurred after greater delay than was the case with type I SVT and the sequence of atrial activation also differed: atrial depolarization recorded in all three atrial leads occurred simultaneously. Activity recorded in the His bundle electrode lead revealed the sequence of activation to be atria-His-ventricles (figs. 2 and 6). The same A-V nodal re-entry responsible for type I SVT seems unlikely for the following reasons: H1-H2 intervals associated with an A2 that precipitated type II SVT in most instances were less than those which were followed by type I SVT; the sequence of atrial activation as well as that of His and ventricular discharge differed; anterograde bypass conduction was never manifested at cycle lengths considerably in excess of the bypass refractory period; and a delay of over 500 msec occurred between A2 and the next spontaneous atrial depolarization. We have explained the mechanism responsible for type II SVT as follows: short A1-A2 intervals following the slightly longer BCL resulted in anterograde block in the accessory pathway as well as in the left bundle branch. Delayed activation of the left ventricle (presumably the location for a type A accessory pathway) resulted in delayed activation of the bypass tract. Conduction returned to the atrium over the accessory pathway and then traveled anterogradely over the normal A-V node-His bundle route. Retrograde conduction over the accessory pathway explains why ventricular activation over the bypass was not seen during type II SVT. The longer re-entry circuit also explains the generally slower rates for type II SVT. The V-A interval (200 msec) during type II SVT does not reflect retrograde conduction time over the bypass tract since the time at which the bypass tract was discharged is unknown. On five occasions, A1-A2 intervals of 200 msec were followed by single atrial echoes and QRS with LBBB. However, the V1-V2 intervals were less than those which were followed by type II SVT, and this may account for failure to initiate sustained type II SVT (table 2).

Anterograde Block in Accessory Pathway With Intact Retrograde Conduction

Case 3. A 29-year-old white male had a history of intermittent SVT since age 15, unsatisfactorily controlled with digitalis, quinidine, and propranolol. Physical examination was unremarkable. ECG revealed premature ventricular extrasystoles with a right bundle branch block pattern and episodes of SVT manifesting a functional left bundle branch block (LBBB).

During a basic cycle of 580 msec, premature high right atrial stimulation lengthened A-H interval at A1-A2 intervals less than 350 msec, without definite evidence for A-V nodal bypass. We did not attempt to pace other atrial sites. A1-A2 intervals less than 400 msec resulted in functional LBBB. ERPAVN occurred at an A1-A2 interval of 250 msec and the FRPAN was 330 msec at an A1-A2 interval of 305 msec. A1-A2 inter-

![Figure 5](image-url)

**Figure 5**

Case 2. Termination of type I SVT with an early premature atrial stimulus. Following premature atrial discharge (A'), conduction blocked proximal to the His bundle and SVT terminated. A' conducted to the ventricles (V') over the accessory pathway. The resulting premature ventricular interval (265 msec) equaled the premature atrial interval recorded in BEE, and supports the presence of a left-sided bypass. Continued atrial pacing captured the atria at a rate which produced partial pre-excitation of the ventricles. Spontaneous SVT intervals, 350 msec. Pacing stimulus intervals, 390 msec. The numbers 225, 265, and 290 indicate the premature atrial cycle lengths recorded for different parts of the atria in three different leads.

![Figure 6](image-url)

**Figure 6**

vals of 280-290 msec precipitated a sustained SVT manifesting LBBB (fig. 7, left panel). In figure 8, an A-A2 interval of 290 msec lengthened the A2-H2 interval to 150 msec and precipitated an SVT with functional LBBB. The H-V interval lengthened to 45 msec and then stabilized between 50 msec and 55 msec during the SVT which had a cycle length of 330 msec. The contour of the H potential following A2 may represent delayed conduction through the bundle of His into or may simply be due to catheter movement. A single premature right atrial stimulus delivered 270-280 msec after the last spontaneous QRS complex of the SVT terminated the SVT (not shown).

After the SVT was initiated, single premature right ventricular stimuli occurring 290-340 msec after the last spontaneous QRS complex shortened the following atrial cycle by an amount equal to the prematurity of the stimulus on 10 occasions without interrupting the SVT. Single premature right ventricular stimuli between 240-280 msec terminated the SVT on nine occasions. In figure 9, pacemaker-induced premature right ventricular depolarization during the SVT (top, arrow) occurred 290 msec after a spontaneous QRS complex. His activation, immediately preceding the ventricular stimulus (interrupted line), occurred on time and without contour change. Therefore, the premature ventricular stimulus cannot have discharged the bundle of His retrogradely. However, the atrial response following the premature ventricular depolarization occurred early, after an interval of only 290 msec. The premature ventricular depolarization must have activated the atrium over a route which bypassed the refractory bundle of His, i.e., an A-V accessory pathway which conducted only retrogradely from ventricle to atrium. In the lower panel of this

continuous recording, premature right ventricular stimulation (arrow), 280 msec following a spontaneous QRS complex, terminated the SVT. However, the last His response occurred precisely on time and without contour change, just after the onset of ventricular activation which the pacemaker stimulus initiated (interrupted line). Since it is improbable that excita-

Figure 7

Case 3. Anterograde block in accessory pathway with intact retrograde conduction. Left panel, A-A2 intervals (X axis) plotted against corresponding H1-H2 intervals (Y axis) during right atrial pacing at a basic cycle length (BCL) of 580 msec. Right panel, V1-V2 intervals (X axis) plotted against corresponding A1-A2 intervals (Y axis) during right ventricular pacing at a BCL of 540 msec. Retrograde block to the atria occurred at V1-V2 intervals less than 330 msec.

Figure 8

Case 3. Initiation of SVT with LBBB following A1-A2 interval of 290 msec. H-V interval lengthened to 50-55 msec during SVT which had a cycle length of 330 msec. Conventions as in figure 1.
tion from the premature ventricular discharge reached the His bundle retrogradely in only a few msec and without altering the contour or timing of the His spike, one must conclude that a portion of the re-entrant pathway sustaining this SVT was located below the bundle of His and could be discharged by premature ventricular depolarization. We interpret these observations to indicate that anterograde A-V conduction traveled only over the normal A-V node–His bundle route during sinus rhythm as well as during SVT; the accessory pathway did not conduct in an anterograde direction.13,14 Retrograde conduction to the atria traveled over an accessory pathway which bypassed the A-V node during SVT. Late premature right ventricular stimulation found the accessory pathway excitable, shortened the succeeding atrial cycle but perpetuated the SVT. Early premature right ventricular stimulation partially invaded the incompletely recovered accessory pathway, eventually blocked and terminated the SVT by making the accessory pathway and/or ventricular muscle refractory to the impulse descending the normal A-V node–His bundle route. The presence of LBBB during the SVT raises the question whether re-entry traveled over the bundle branches.15,16 This seems unlikely because A1-A2 intervals of 380 msec-295 msec provoked LBBB with H-V delay but failed to initiate SVT (fig. 7, left panel). Also, the probable sequence of activation during bundle branch re-entry would be ventricle-His-atrium rather than atrium-His-ventricle.

The atrial response which followed premature ventricular stimulation in figure 9 (top) occurred early (290 msec) but failed to prolong the succeeding A-H interval. This observation, coupled with the facts that A2-H2 remained constant through A1-A2 values of 350 msec (fig. 7, left) and that the A-H interval was only 60 msec when the atria were paced at a BCL of 580 msec (fig. 8) raise the question whether an A-V nodal bypass, such as James fibers with “relatively” slow conduction and “relatively” long refractory period or which bypassed only a portion of the A-V node existed in parallel to the unidirectionally conducting A-V bypass. Obviously, alternative explanations such as a short A-V node, preferential intranodal conduction and so forth are possible.

We evaluated retrograde V-A conduction, at a time when the patient did not have the SVT, by prematurely stimulating the apex of the right ventricle in the same fashion that we evaluated anterograde conduction. At premature intervals (V1-V2) less than 330 msec, retrograde conduction to the atria failed, resulting in identical values for FRP and ERP (fig. 7, right panel). This is an unusual response because, unlike anterograde A-V nodal conduction, V2-A2 remained fixed and then conduction suddenly failed without the usual nodal delay. Of course, this factor alone does not indicate the presence of retrograde bypass conduction, but only that the characteristics of retrograde conduction were unusual during this method of testing.17 It is also important to emphasize that, during this method of testing, V2 failed to reach the atria at V1-V2 intervals less than 330 msec following a BCL of 540 msec. The VT cycle length was about 340 msec and therefore V-A conduction over the bypass might be expected to occur at shorter premature ventricular cycle lengths during the SVT.

At V1-V2 intervals of 240-290 msec, V2 was followed by a late deflection which had the appearance of a His potential18 and by two spontaneous QRS complexes (figs. 7 & 10). The first spontaneous QRS had a LBBB pattern (VLB), while the second QRS was preceded by atrial and His deflections and had a normal contour (VN). VN did not occur in this context without being preceded by VLB. Since V1-V2 intervals less than 260 msec were followed by a late His spike and by VLB on only one occasion, it would appear likely that VLB was in some fashion dependent on delayed retrograde His excitation and did not represent simply a repetitive ventricular response following stimulation during the ventricular relative refractory period or vulnerable period. VLB was very likely a ventricular reciprocal beat, without intervening (recordable) atrial activity, possibly due to re-entry within the A-V node19 or His bundle20 (functional LBBB would be expected at this
short interval). Since \( V_{LB} \) did not display exactly the same LBBB QRS contour the patient 'normally' exhibited, one can postulate an alternate re-entry route over the bundle branches: \( V_2 \) resulted in retrograde functional RBBB and delayed retrograde activation traveled to the bundle of His over the left bundle branch system. The impulse then returned to the ventricle over the right bundle branch to produce a QRS with a LBBB pattern (\( V_{LB} \)). The lack of retrograde atrial excitation at these short \( V_1-V_2 \) intervals eliminates the possibility of \( V_{LB} \) being due to conduction over the ventricle-atrium bypass. If the bypass were located in the left ventricle, \( V_{LB} \) would allow the bypass sufficient time to repolarize and result in delayed bypass activation. Conduction could then return to the atrium over the bypass and then back to the ventricle over the normal route to produce \( V_N \). However, no sustained SVT ever followed any of the \( V_1-V_2 \) intervals because \( V_N \) resulted in normal activation time for the left ventricle before the accessory pathway had time to recover and continue the re-entrant excitation.

Sustained SVT did result on 9 occasions when a spontaneous premature ventricular extrasystole of RBBB contour and left axis deviation (\( V_E \)) was followed by a paced premature ventricular systole (\( V_2 \)) at a \( V_E-V_2 \) interval of 270 to 310 msec (not shown). The V-A interval for \( V_E \) was 95 msec, compared to the V-A interval of 210 msec during right ventricular pacing and can be explained by early entry into the left-sided bypass because the spontaneous premature ventricular impulse arose in the left ventricle, possibly in the postero-inferior division. Since \( V_E \) resulted in early activation of the bypass tract and presumably somewhat later activation of the A-V node, retrograde conduction to the atria might occur over the bypass tract at a \( V_E-V_2 \) interval (270-310 msec) normally expected to fail in both pathways (fig. 7). If \( V_2 \) did fail retrogradely in the A-V node, then the atrial response to \( V_2 \) could return to the ventricles at a time when the normal A-V route had recovered, and thus initiate SVT. Short \( V_1-V_2 \) intervals (fig. 10) failed to initiate sustained SVT while short \( V_E-V_2 \) intervals repeatedly initiated SVT because, in the former instance, \( V_N \) followed \( V_{LB} \) too closely (310 msec) to allow time for the bypass to recover.

In summary we interpret the data obtained in this patient to indicate the presence of a left-sided accessory pathway from ventricle to atrium which only conducted retrogradely. Premature right ventricular stimulation during the spontaneous SVT conducted to the atrium or terminated the SVT at times when the His bundle was refractory. Premature right ventricular stimulation initiated the SVT only when the bypass was allowed sufficient time to recover excitability and maintain the re-entrant excitation, (e.g. following a left-sided premature ventricular extrasystole). Premature ventricular stimulation resulted in ventricular echoes without intervening atrial depolarization being recorded.

Case 4. A 32-year-old female suffered from recurrent SVT which was difficult to control medically. Physical examination was normal.

Premature atrial stimulation or rapid atrial pacing at a high right atrial site lengthened A-H interval normally and there was no evidence to support the presence of anterograde bypass conduction. Other atrial sites were not paced. \( A_1-A_2 \) intervals between 250 msec and 280 msec following a BCL of 455 msec provoked a sustained SVT on 8 occasions (fig. 11, top). Similar events occurred with a BCL of 555 msec. Competitive right atrial pacing terminated the SVT. During premature right ventricular pacing at \( V_1-V_2 \) intervals greater than 310 msec, \( V_2 \) conducted retrogradely to the atria. At \( V_1-V_2 \) intervals between 290 and 310 msec, \( V_2 \) failed to conduct to the atria but at \( V_1-V_2 \) intervals between 230 and 290 msec, \( V_2 \) was followed by a delayed H spike and conduction to the atria at an \( H_2-A_2 \) interval of 40 msec. The \( H_2-A_2 \) interval remained constant even though the \( V_1-H_2 \) interval (the H spike was obscured by the \( V_1 \) electrogram and therefore \( H_1-H_2 \) could not be measured) shortened 25 msec following a \( V_1-V_2 \) interval of 230 msec (fig. 11, bottom). Similar events occurred with a BCL of 455 msec. These observations are best explained by a retrograde "gap" owing to retrograde functional RBBB, which delayed \( H_2 \). The short \( H_2-A_2 \) in-

![Figure 11](image-url)

*Figure 11* 4. Anterograde block in accessory pathway with intact retrograde conduction exposed by the "gap" phenomenon. Last basic atrial cycle (\( A_1-A_2 \)) and premature atrial cycle (\( A_1-A_2 \)) are shown in top tracing, left panel; only the premature cycle (\( A_1-A_2 \)) is shown in the right panel. SVT followed \( A_1-A_2 \) interval of 280 msec. Last basic ventricular cycle (\( V_1-V_2 \)) and premature ventricular cycle (\( V_1-V_2 \)) are shown in bottom tracing, left panel; only the premature ventricular cycles (\( V_1-V_2 \)) are shown in the middle and right panels. Conventions as in figure 1.
interval which did not lengthen as the $V_1$-$H_2$ cycle shortened can be explained by the presence of a James bypass which conducted only retrogradely. The refractory period of the bypass was rather long since it failed to conduct at $V_1$-$V_2$ intervals of 290 to 310 msec and only resumed conduction when the $V_1$-$H_2$ interval was 360 msec (fig. 11, bottom, middle panel). Premature ventricular stimulation did not precipitate SVT. However, since the response of retrograde A-V nodal conduction following a premature ventricular stimulus in normal patients is largely unknown, the short $H_2$-$A_2$ interval must be interpreted cautiously; a normal variant can not be entirely excluded at this time.17 For example, A-H conduction time may exceed H-A conduction time in the normal individual because a different portion of the atrium is taken to represent the onset of low right atrial activity in the anterograde direction than that which is taken to represent low right atrial activity in the retrograde direction. Actual nodal conduction times may be the same in both directions.

Retrograde Conduction Delay or Block in the Accessory Pathway and A-V Node With Intact Anterograde Conduction

Case 5. A 34-year-old white male with extensive coronary artery disease had undergone coronary vein bypass surgery for severe angina pectoris. A syncopal spell one year following the surgical procedure and the demonstration of type B pre-excitation in the surface ECG prompted an electrophysiologic evaluation. Tachycardia had never been documented. A fourth heart sound was noted at the time of the physical examination.

Premature right atrial stimulation during a basic cycle length of 700 msec revealed the effective refractory period of the bypass tract to be 390 msec, while the ERP ANV was 320 msec. Right atrial pacing at progressively shorter cycle lengths produced block in the accessory pathway when the atrial cycle length was reduced to intervals less than 460 msec. Once block was established in the accessory pathway in this fashion, the atrial cycle length could be lengthened gradually to 650 msec while still maintaining block in the accessory pathway (fig. 12). This observation may be explained by assuming that retrograde (concealed) re-entry of the bypass tract following normal ventricular excitation caused the accessory pathway to be refractory to the next atrial impulse. Since atrial echoes never resulted, the concealed re-entry must enter the bypass tract but fail to reach the atrium because the upper portion of the bypass tract, depolarized anterogradely during the atrial discharge, was still refractory.25 An alternate explanation is that ventricular activation, “displaced” to the right by the long P-R interval during normal conduction, renders the ventricles still refractory when the bypass is activated in the following cycle.26 This hypothesis is not applicable because the refractory period of the ventricles in the top panel (fig. 12) would have to exceed 280 msec (interval from onset of normal QRS to anticipated time of arrival at ventricles of Kent activation in following cycle) while the ventricular refractory period in the lower panel would have to exceed 510 msec. The determined ventricular refractory period in this patient (BCL 600 msec) was < 280 msec. A more likely explanation is depicted by the events shown in figure 13. Ventricular pacing at a cycle length of 600 msec did not conduct to the atria, which discharged spontaneously and independently at cycle lengths of about 1000 msec (fig. 13, top).17 However, it could be shown that a portion of the A-V node and accessory pathway were depolarized retrogradely, by the effects of ventricular discharge on subsequent atrial conduction (fig. 13, bottom). The independently discharging atria failed to conduct to the ventricles at short R-P intervals, conducted with a normal QRS complex at longer R-P intervals, and conducted over the accessory route at the longest R-P intervals. In summary, the observations made in this patient can be explained by assuming normal anterograde conduction in the A-V node and accessory pathway. Retrograde conduction from the ventricles could enter both pathways but encountered delay or block and failed to excite the atria. This factor would prevent precipitation of a SVT supported by re-

Figure 12
Case 5. Retrograde conduction delay or block in the accessory pathway and A-V node with intact anterograde conduction; concealed re-entry in the bypass tract. Top and bottom panels represent beginning and end of a continuous recording during which right atrial cycle length was at first progressively shortened to produce block in the accessory pathway (top) and then gradually lengthened to demonstrate maintenance of block in the accessory pathway at longer cycle lengths (bottom). Middle portion of recording with intermediate pacing cycle lengths is not shown. Conventions as in figure 1.

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entry which required retrograde conduction to the atrium over one of these two pathways. SVT could not be provoked in this patient.

Discussion

In 1967, Durrer et al.\textsuperscript{10} observed that an early premature atrial systole could be made to block in the accessory pathway and conduct to the ventricles exclusively over the normal A-V node–His bundle route. They were able to initiate SVT with appropriately timed atrial and ventricular extrasystoles. Their studies have been confirmed by others and it is generally accepted that in many (but not all) patients with the pre-excitation syndrome and SVT, the refractory period of the accessory pathway exceeds that of the normal A-V node, enabling a premature atrial depolarization to propagate anterogradely in the normal pathway and return to the atrium over the accessory pathway.\textsuperscript{9, 17–20} Drugs like digitalis may terminate or prevent SVT by decreasing the difference in refractory periods between the two pathways;\textsuperscript{20} drugs which increase the disparity of the functional properties may promote SVT.

As more patients with pre-excitation syndrome are studied, it is obvious that many exceptions exist to the classic electrophysiology briefly outlined above. Occasionally, SVT may be sustained by anterograde conduction over the accessory route and return to the atrium over the normal A-V node.\textsuperscript{17, 22} Bypass tracts with exceedingly short refractory periods have been noted,\textsuperscript{31} and the presence of multiple bypass tracts have been postulated for some patients.\textsuperscript{35}

The present study extends the observations on the variety of results obtained following electrical stimulation in patients with the pre-excitation syndrome. We have demonstrated that anterograde conduction over the normal A-V node may discharge the His bundle toward the end of ventricular depolarization when the latter was initiated by conduction over the accessory pathway (cases 1 and 2). This may be caused by retrograde conduction delay or block from the site of ventricular pre-excitation or because the His bundle remained refractory during ventricular discharge.\textsuperscript{33}

Nodal re-entry, either sinus\textsuperscript{34} or A-V,\textsuperscript{7, 8, 35} may sustain SVT in some patients with the pre-excitation syndrome. Type I SVT (case 2) was probably due to A-V nodal re-entry independent of the accessory pathway; the ventricles were still activated anterogradely via the accessory pathway. Distinguishing SVT maintained by A-V nodal re-entry with anterograde conduction to the ventricles over the bypass from re-entry involving anterograde bypass and retrograde A-V nodal transmission may be quite difficult.\textsuperscript{17} In case 2, type II SVT demonstrated characteristics which differed markedly from type I SVT and were explained by a different re-entrant route, anterogradely over the A-V node and retrogradely over the accessory pathway. Tracings in this patient also point out the usefulness of recording multiple atrial leads in order to identify different patterns of atrial activation.\textsuperscript{35}

One-way conduction is a well-established electrophysiologic phenomenon and perhaps it should not be unexpected to demonstrate its existence in accessory fibers (cases 3, 4, 5). Recent reports have confirmed these observations\textsuperscript{17, 37} and pointed out that left atrial stimulation may expose a left-sided bypass not apparent during sinus rhythm.\textsuperscript{37} The presence of unidirectional conduction may have clinical importance: if an accessory pathway is capable of only V-A transmission, then it may provide the requisite alternate route to initiate and sustain SVT, or to initiate atrial flutter or fibrillation, but remain clinically unrecognized.\textsuperscript{13} Excessively rapid ventricular rates in response to atrial flutter or fibrillation would not be expected in this situation since the normal route provides the only anterograde pathway. If both the A-V node and accessory pathway demonstrate one-way conduction in the same direction, then these two pathways by themselves could not support a re-entry circuit involving atrium and ventricle (case 5).\textsuperscript{17}

In case 3 (fig. 10), premature ventricular stimulation provoked a ventricular reciprocal beat (V1,\textsubscript{B}) that was not preceded by recordable atrial activity. If the return to the ventricles occurred within the A-V node,
this example would offer evidence that the atrium — or at least atrial activity recorded via the esophageal electrode — was not required as a link in the reentrant pathway.38, 39 However, it is just as reasonable to conclude, from this example, that the reentrant circuit traveled over the bundle branches, in which case the atrium would not be needed to complete the conduction loop.15, 16, 23

Concealed re-entry into the accessory pathway is postulated to explain maintenance of block in the accessory pathway at atrial cycle lengths which were longer than those cycle lengths required to initiate the block (figs. 12, 13). A similar observation has been made during rate dependent aberrancy and a similar explanation has been offered.40 Retrograde functional RBBB has been clearly demonstrated in the canine heart during in vivo premature stimulation of the free running false tendon.21 This factor can account for a delayed His response following premature right ventricular stimulation6 (fig. 10, 11) and establish a retrograde “gap” in V-A impulse transmission similar to that which is seen in the anterograde direction.23, 24

Although various pathways have been demonstrated anatomically which may explain the electrocardiographic and electrophysiologic counterparts in patients with the pre-excitation syndrome and its variants, the functional significance of the anatomic substrata remains incompletely resolved.41 A recent study correlated electrophysiologic and anatomic observations to convincingly establish the role of a lateral A-V connection in a dog with preexcitation.42 The results of surgery in man also lend credence to the presence of lateral A-V connections in some patients with the pre-excitation syndrome.43 However, the role played by anatomic entities, such as the fibers described by James,44 in explaining electrocardiograms characterized by a short P-R interval and normal QRS,45, 46 or A-H intervals that are only borderline short and do not lengthen “normally” in response to premature stimulation or rapid pacing (cases 3 & 4) is presently unknown. Similar questions can be applied to the functional significance of Mahaim fibers (case 1). A discontinuous annulus fibrosus has recently been offered as an important morphologic component.47 But anatomic conclusions derived from electrophysiologic data as well as electrophysiologic conclusions derived from anatomic data have to be made cautiously.48 Our study presents functional data which support the conclusion that the electrophysiologic properties of accessory fibers may be quite variable. Understanding these variations may provide important therapeutic implications regarding drug and surgical management, but this study does not furnish further insight regarding the anatomic framework.

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PROPERTIES OF ACCESSORY PATHWAYS


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Unusual Properties of Accessory Pathways
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