Retrograde Invasion of the Bundle Branches Producing Aberration of the QRS Complex During Supraventricular Tachycardia Studied by Programmed Electrical Stimulation


SUMMARY

Four patients with paroxysmal supraventricular tachycardia have been studied using programmed electrical stimulation of the heart and intracardiac recordings. One patient had an atrioventricular (A-V) junctional tachycardia, possibly due to a rapidly discharging protected ectopic focus, and three patients had anomalous atrioventricular connections. The ECG recordings in all four patients showed a bundle branch block pattern during tachycardia, and normal intraventricular conduction could be produced by suitably timed right ventricular premature beats. The cycle length of the tachycardia was shorter with normal intraventricular conduction than in the presence of a bundle branch block pattern in two patients. Evidence is presented indicating that the bundle branch block during tachycardia was likely to be due to concealed retrograde conduction of the appropriate bundle branch, and in the presence of an anomalous bypass, the cycle length of the tachycardia depended on the anatomical relationship of the anomalous bypass to the blocked bundle branch.

Additional Indexing Words:
Wolff-Parkinson-White Syndrome
Accessory pathways
Atroventricular conduction
Ectopic beats
Fusion beats
Junctional rhythm

A trial premature beats causing aberration of the QRS complex are common findings in clinical electrocardiography. Continued aberration during either supraventricular tachycardia or sinus rhythm is also well recognized and it has been suggested that this may be due to retrograde invasion of one or other bundle branch thereby producing a persistent bundle branch block in the appropriate bundle.1,6 In this paper four patients with documented paroxysmal supraventricular tachycardia are presented. The ECG of each patient showed a bundle branch block pattern during tachycardia and the mechanism of this was studied using intracardiac recording techniques and programmed electrical stimulation of the heart.

Patients and Methods

The clinical details of the four patients are presented in table 1. Informed consent was given by each patient and an electrophysiological study was carried out in the postabsorptive, nonsedated state. Three to four bipolar electrode catheters were introduced percutaneously through one or both femoral veins and passed to the right side of the heart. One catheter was positioned across the tricuspid valve and a His bundle electrogram obtained using the technique described by Scherlag et al.6 One catheter was positioned high in the right atrium for recording an atrial electrogram. In two patients an electrode was passed to the left atrium via a patent foramen ovale and the high left atrial electrogram recorded. The fourth catheter was used for pacing the atrium or ventricle or for producing atrial or ventricular premature beats. The recordings were made on an eight channel Elema Mingograf 81 Recorder, and the His bundle electrogram was recorded at a filter setting of 45-500 cycles/second. Leads I, II, V1 and V6 of the surface electrocardiogram were recorded simultaneously with the intracardiac recordings. All recordings were taken at a paper speed of 100 mm/sec.

A Devices* 4270 stimulator was used for atrial and ven-

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Table 1

<table>
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WPW = Wolff-Parkinson-White

Clinical Data

tricular pacing. The heart was driven at a constant frequency thus preventing any changes in excitability or refractoriness due to irregularities of rhythm. Single or double atrial or ventricular premature beats were delivered following every eighth beat of the basic driven rhythm. A variable delay circuit triggered by either the R wave of the surface electrocardiogram or the right atrial electrogram was used so that these premature beats could be delivered in the cardiac cycle at a preset delay following the preceding driven beat. In each patient the mode of initiation and termination of the tachycardia was studied and the effect of atrial and ventricular premature beats on the basic rhythm during tachycardia was studied.

The criteria we use for the diagnosis of a reciprocal mechanism rather than a rapidly discharging protected ectopic focus in patients with tachycardia are that 1) the tachycardia can be both initiated and terminated by critically timed single or double atrial or ventricular premature beats and that 2) induced premature beats during tachycardia that do not terminate the tachycardia are followed by pauses that are shorter than compensatory. In addition, in patients with atrioventricular (A-V) junctional tachycardias that are due to a reciprocal mechanism, the onset of the tachycardia should occur when a critical increase in A-V nodal conduction time has been achieved by the prematurity beat, while in patients with anomalous bypass tracts, the tachycardia is initiated when the refractory period of the bypass is reached following a critically timed atrial premature beat and normal antegrade conduction ensues by way of the A-V node-His system.

Results

Patient 1

This patient was in tachycardia at the onset of the study. The QRS complex was normal in width and configuration indicating the supraventricular origin of the tachycardia. A His bundle electrogram was not obtained on this patient. Induced ventricular premature beats during tachycardia were followed by fully compensatory pauses. The tachycardia could not be terminated by either single or double atrial or ventricular premature beats and the tachycardia was finally terminated following the administration of Verapamil, 10 mg, intravenously. Following termination, the tachycardia could not be re-initiated by single or double premature beats and a spontaneous onset of tachycardia was never observed. This failure to respond to induced premature beats led us to hypothesize that a protected ectopic focus was the most likely underlying mechanism of the tachycardia. However a protected re-entrant focus would respond in a similar manner. During tachycardia there was retrograde conduction to the atria as indicated by the fact that low atrial depolarization, as seen on the low right atrial electrogram (LRAE), occurred before high right atrial depolarization as seen on the high right atrial electrogram (HRAE) (fig. 1). The site of the abnormal focus was therefore likely to be in the atrioventricular (A-V) junctional tissues.

The effect of an induced right ventricular premature beat on the intraventricular conduction system is seen in figure 1. The first three beats show normal QRS complexes with a width of 80 msec. The fourth beat is a right ventricular premature beat and is followed by two further ventricular premature beats of similar configuration. These latter beats are probably due to repetitive firing from the pacemaker stimulus site in the ventricle. Following the third ventricular premature beat the tachycardia continues but the QRS is now wider at 110 msec and shows a right bundle branch block pattern. Figure 2 is a recording from patient one and the first three beats show a regular tachycardia with a QRS contour showing right bundle branch block pattern. The fourth beat is a fusion beat between an induced right ventricular premature beat and a beat of supraventricular origin.

Figure 1

Tracing obtained from patient 1 during tachycardia showing the development of right bundle branch block by three ventricular premature beats. Abbreviations: HRAE = high right atrial electrogram; LRAE = low right atrial electrogram; I, III, V1, V4 = surface electrocardiographic leads; AV = atrioventricular junctional region; His = bundle of His; RBB = right bundle branch; LBB = left bundle branch; VPB = ventricular premature beat; St = stimulus artifact; A = retrograde atrial depolarization. All numbers shown are in msec.
from the ectopic focus causing the tachycardia. Following the fusion beat the next QRS complex has a normal configuration and a width of 75 msec. The cycle length of the tachycardia both before and after the series of beats showing right bundle branch block was 275 msec, the same as the cycle length in the tachycardia showing right bundle branch block.

Interpretation

The initiation of right bundle branch block following the three ventricular premature beats in this patient we find difficult to explain. It is not due to a rate-related refactoriness in the right bundle because the cycle lengths of the beats showing normal conduction and those showing aberrant conduction are identical. It may be that phase four depolarization following the third ventricular premature beat is responsible; however, this would have to be occurring at a reduced level of transmembrane potential as the cycle length between the third ventricular premature beat and the first beat of the supraventricular tachycardia showing a right bundle branch block configuration is short. However, following the third ventricular premature beat (fig. 1), it will be seen that the activation front from the supraventricular focus reaches the intraventricular bundle branch system while the right bundle branch is still refractory, thus producing right bundle branch block configuration on the surface electrocardiogram. This right bundle branch block pattern is then perpetuated because we believe the supraventricular impulse passes down the left bundle, the right bundle being refractory, and penetrates the right bundle branch retrogradely. In this way the right bundle branch block is maintained as the right bundle will always be refractory to the activating impulse from the ectopic supraventricular focus following retrograde invasion of that bundle by the preceding beat. If a ventricular premature beat or fusion beat occurs late in diastole as in figure 2, then induced pre-excitation in the right bundle occurs simultaneously with left bundle activation via conduction from the His bundle and a normal QRS results. Retrograde invasion of the right bundle is prevented by premature activation of the right bundle and the next beat of the tachycardia will show normal intraventricular conduction.

Patient 2

This patient had the typical appearances of the Wolff-Parkinson-White (WPW) syndrome type B. Figure 3 is a recording of two sinus beats from patient two. The surface electrocardiogram shows the presence of a delta wave in leads I and V6 and a deep S wave in lead V1, evidence indicating a type B WPW, in keeping with an anomalous bypass on the right side of the heart. The tachycardia could be initiated in this

![Figure 2](http://circ.ahajournals.org/)

Figure 2

Tracing obtained from patient 1 during tachycardia showing conversion of right bundle branch block pattern to normal intraventricular conduction by a fusion beat. Abbreviations: FB = fusion beat.

![Figure 3](http://circ.ahajournals.org/)

Figure 3

Tracing obtained from patient 2 during sinus rhythm. Abbreviations: A = right atrial depolarization; A-H = time in msec from the low atrial depolarization on the His bundle electrogram to the His deflection; H-V = time in msec from the His deflection to the onset of ventricular activation; HLAEE = high left atrial electrogram; HBE = His bundle electrogram; H = His deflection on the His bundle electrogram; K = Kent pathway on ladder diagram; V = ventricular depolarization as seen on the His bundle electrogram.
patient by critically timed single left atrial premature beats at which time the accessory pathway became refractory and antegrade conduction occurred solely by way of the A-V node-His system. The tachycardia could be terminated by critically timed left atrial premature beats, and induced left atrial and right ventricular premature beats during tachycardia were followed by pauses less than compensatory. The QRS complex in tachycardia, following normalization by a ventricular premature beat, was narrow, of normal configuration, and was preceded by a His deflection (fig. 4).

The patient therefore had a reciprocal tachycardia in which antegrade conduction occurred by way of the A-V node-His system and retrograde conduction by way of the anomalous bypass which was thought to lie on the right side of the heart. However, the recording obtained from this patient during tachycardia (fig. 4) shows that retrograde activation of the high left atrium on the high left atrial electrogram (HLAE) occurs 70 msec before activation of the high right atrium as seen on the high right atrial electrogram (HRAE) and only 5 msec after low right atrial activation as seen on the His bundle electrogram. This observation suggests that despite the typical appearance of type B WPW on the surface electrogram during sinus rhythm, the anomalous atrioventricular pathway activated during tachycardia lies on the left side of the heart as seen in type A WPW.9 Impulses passing retrogradely to the atria through the atrioventricular junction lead to either simultaneous right and left atrial depolarization or to depolarization in quick succession of the right atria and then the left, with an interatrial conduction time not exceeding 0.03 sec.10 These findings are in accord with those from our own laboratory for retrograde atrial activation during right ventricular pacing and A-V junctional tachycardias. This patient therefore may have two anomalous A-V connections, one on the right side of the heart which is only apparent during sinus rhythm, and one on the left side of the heart which conducts retrogradely during reciprocal tachycardia. Type A and type B WPW occurring in the same patient has previously been described.11 The left atrial premature beat initiating the tachycardia always showed a right bundle branch block pattern, presumably because it occurred sufficiently early in the cardiac cycle following a preceding long cycle to find the right bundle branch still refractory. The right bundle branch block was then perpetuated during tachycardia. Figure 5 is a recording obtained from the patient during tachycardia and the first two beats show a right bundle

**Figure 4**

Tracing obtained from patient 2 during tachycardia. Abbreviations: K = Kent pathway on ladder diagram; H = His pathway on ladder diagram; V-A’ (LRA) = retrograde conduction time from onset of ventricular activation to low right atrial depolarization; V-A’ (HLA) = retrograde conduction time from onset of ventricular activation to high left atrial depolarization; V-A’ (HRA) = retrograde conduction time from onset of ventricular activation to high right atrial depolarization. All values in milliseconds.

**Figure 5**

Tracing obtained from patient 2 during tachycardia showing retrograde invasion of the right bundle branch and retrograde conduction in a left-sided pathway leading to right bundle branch block, which is converted to normal intracardiac conduction following a fusion beat. Abbreviations: Type A = Type A Wolff-Parkinson-White situated anomalous pathway; FB = fusion beat; St = stimulus artifact; A’H = retrograde conduction along anomalous pathway to His bundle; V = ventricular depolarization as seen on His bundle electrogram; H = His deflection; K = Kent pathway.

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branch block pattern. The third beat is a fusion beat between an induced right ventricular premature beat and activation of the ventricles via the A-V node-His system. Following this fusion beat the tachycardia continues but the QRS complexes are now of normal width and configuration. The cycle length of the tachycardia when a right bundle branch block pattern is present is the same as the cycle length when normal intraventricular conduction occurs, 290 msec. This suggests that the ventricular aberration is unlikely to be due to a rate-related refractoriness.

**Interpretation**

Our interpretation of the tracing is shown on the diagrams below the appropriate complexes. During tachycardia the impulse passes down the A-V node-His system and activates the ventricles and the left ventricular end of the anomalous bypass via the left bundle. The impulse passes retrogradely into the right bundle branch from the left bundle branch thus producing right bundle branch block. Each successive antegrade impulse during tachycardia finds the right bundle refractory following retrograde invasion of this branch during the preceding beat, and in this way the right bundle block is maintained. The induced right ventricular premature beat pre-excites the right ventricle and thereby normalizes the third QRS complex (fig. 5) on the surface electrocardiogram. The fourth beat of the tachycardia shows normal intraventricular conduction because the induced right ventricular premature beat prevents retrograde invasion of the right bundle branch by the reciprocating impulse which is passing antegrade through the left bundle. Because the reciprocal mechanism is occurring on the left side of the heart, the presence of block in the right bundle branch would not be expected to alter the length of the reciprocal circuit and therefore the cycle length of the tachycardia; and indeed, it remains constant at 290 msec during both right bundle block and normal intraventricular conduction.

**Patient 3**

This patient showed a type B WPW on the surface electrocardiogram, as indicated by the presence of delta waves and a deep S wave in lead V1 during sinus rhythm. Tachycardias could be initiated by critically timed single right atrial and ventricular premature beats. Following this critical right atrial premature beat, the anomalous bypass became refractory, normal antegrade conduction by way of the A-V node-His pathway occurred, and the tachycardia was initiated. The tachycardia could be terminated by critically timed single right atrial and ventricular premature beats and induced premature beats during tachycardia were followed by less than compensatory pauses. The atrial premature beat which initiated the tachycardia was always accompanied by a left bundle branch block configuration, presumably because it occurred sufficiently early in the cardiac cycle, following a preceding long cycle, to find the left bundle branch still refractory. The left bundle branch block was then perpetuated during tachycardia. A recording obtained from patient three during tachycardia is shown in figure 6. The first three beats show a left bundle branch block pattern and each beat is preceded by a His bundle potential indicating the supraventricular nature of the tachycardia. This patient therefore appears to have a reciprocal tachycardia in which antegrade conduction occurs by way of the A-V node-His system and retrograde conduction by way of an anomalous A-V connection, most likely located on the right side of the heart. The fourth beat in figure 6 shows an induced right ventricular premature beat, and following this, the tachycardia continues but the QRS complexes are now normal in width and configuration indicating normal intraventricular conduction.

**Interpretation**

During the first three beats the reciprocating impulse passes antegrade through the A-V node-His.

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![Figure 6](https://example.com/figure6.png)

Tracing obtained from patient 3 during tachycardia showing retrograde invasion of the left bundle branch and retrograde conduction in a right-sided anomalous bypass. The left bundle branch block occurring in tachycardia is converted to normal intraventricular conduction by a right ventricular premature beat (VPB). Abbreviations: Type B = Type B Wolff-Parkinson-White anomaly; A' = retrograde activation of anomalous pathway; H = His bundle deflection; V = ventricular activation; St = stimulus artifact; LBB = left bundle branch; RBB = right bundle branch.
system and activates the ventricles and the right ventricular end of the anomalous A-V connection via the right bundle branch. The impulse then invades the left bundle branch retrogradely, producing left bundle branch block. The left bundle branch block persists because the reciprocating impulse passing antegradely down the His bundle repeatedly finds the left bundle branch refractory from retrograde invasion of this bundle branch during the preceding beat. The induced ventricular premature beat leads to depolarization of both bundle branches earlier in the cardiac cycle, thus preventing retrograde invasion of the left bundle branch by the impulse passing antegradely in the right bundle branch during tachycardia. Both bundle branches now have longer to recover than in preceding cycles and therefore the next impulse arriving antegrade down the His bundle finds both bundle branches fully recovered and normal intraventricular conduction takes place as is seen in figure 6. Because the reciprocal circuit lies on the right side and the bundle branch block occurs in the left bundle, no change in the length of the reciprocal circuit, and hence no change in the cycle length of the tachycardia both during left bundle branch block and normal intraventricular conduction, would be expected. However, it will be seen that the cycles during which left bundle branch block occurs are 295 msec whereas the cycles showing normal intraventricular conduction are 5 msec shorter. An explanation for this is given in the discussion.

Patient 4

This patient had a normal resting electrocardiogram with no evidence of either the WPW syndrome or other forms of pre-excitation. The tachycardia could be initiated by suitably timed single right atrial and right ventricular premature beats associated with a critical prolongation of A-V nodal conduction time. Induced premature beats during tachycardia were followed by pauses less than compensatory, and the tachycardia could be terminated by critically timed single right atrial and ventricular premature beats. With these findings a reciprocal mechanism was thought to be the basis for the tachycardia. The atrial premature beat which initiated the tachycardia always resulted in a left bundle branch block configuration, presumably because it occurred sufficiently early in the cardiac cycle, following a preceding long cycle, to find the left bundle branch still refractory. The left bundle branch block was then perpetuated during tachycardia. A recording obtained from patient four during tachycardia is shown in figure 7. The first three beats show a left bundle branch block pattern. Each QRS complex is preceded by a His potential with a normal H-V time of 40 msec, indicating the supraventricular origin of these complexes. The fourth beat is an induced right ventricular premature beat and following this the supraventricular tachycardia continues with normal intraventricular conduction as indicated by the narrow QRS complex of normal configuration. Following the stimulus artifact of the induced ventricular premature beat, a His potential appears which is too early to have been caused by retrograde conduction. The interval between this His potential and the one preceding it is the same as the cycle length of the tachycardia, i.e., 305 msec, and so the His potential following the stimulus artifact is likely to be due to antegrade His activation occurring during the antegrade passage of the reciprocating impulse of the tachycardia. The passage of this impulse further down the specialized conducting tissue is blocked by the ventricular premature beat. This ventricular premature beat is conducted retrogradely to the atria as shown by retrograde atrial activation on the HRAE and HLAE occurring at 290 msec following the preceding atrial depolarization which is 15 msec earlier than expected if the retrograde atrial

![Figure 7](http://circ.ahajournals.org/)

**Figure 7**

Tracing obtained from patient 4 during tachycardia showing left bundle branch block due to concealed retrograde conduction of the left bundle branch followed by retrograde conduction via a left-sided anomalous atrioventricular connection. The left bundle branch block is converted to normal intraventricular conduction by a right ventricular premature beat. Abbreviations: AV = atrioventricular node; A'-H = antegrade conduction time in msec from the low atrial depolarization on the His bundle electrogram to the His deflection of the His bundle electrogram; H-A' = retrograde conduction time from the His deflection to the low atrial depolarization on the His bundle electrogram. His bundle pacing inset shows the retrograde atrial depolarization on the high right atrial electrogram and high left atrial electrogram during His bundle pacing.
activation had been due to the continuing reciprocal mechanism. However, the retrograde passage of this impulse from the ventricular premature beat could not have occurred via the His bundle as it cannot have recovered sufficiently following antegrade depolarization. Retrograde conduction must therefore have occurred via an anomalous bypass. Concealed bypasses of this type may be more common than previously recognized.12

Interpretation

The finding of an anomalous bypass operating retrogradely during a reciprocal supraventricular tachycardia explains why the cycle length of the tachycardia is shorter during normal intraventricular conduction than with left bundle branch block (see diagrams in figure 7 below the appropriate complexes). If it is postulated that this bypass lies on the left side of the heart, the ventricular end closely adjacent to the left bundle branch, then with retrograde invasion of the left bundle branch the reciprocal circuit producing left bundle branch block would consist of antegrade A-V node-His conduction, antegrade conduction in the right bundle, retrograde invasion of the left bundle, and then retrograde conduction in the anomalous bypass. The left bundle branch block pattern persists in tachycardia because the antegrade passage of the reciprocating impulse repeatedly finds the left bundle branch refractory from retrograde invasion during the preceding beat. The induced right ventricular premature beat prevents activation of the right bundle branch by the activation front passing antegradely in the His bundle and also prevents retrograde invasion of the left bundle branch. Following the induced ventricular premature beat retrograde conduction to the atria occurs through the anomalous bypass, the reciprocal mechanism continues, and activation of the ventricles occurs by way of the A-V node-His system. The proximal left bundle branch has now had longer to recover and is nonrefractory. The impulse can therefore pass antegrade through both the right and left bundle branches producing a normal intraventricular conduction pattern. The impulse passes from the left bundle branch system into the anomalous bypass and back to the atria. As can be seen from the diagrams the reciprocal circuit is shorter when normal intraventricular conduction occurs than when left bundle branch block occurs. It therefore follows that the cycle length of the tachycardia will be shorter when normal intraventricular conduction occurs than when left bundle branch block is present as the diagram confirms. The anomalous pathway in this patient must be located on the left side of the heart for the reason just given. However, because retrograde activation of both the high right and left atrium occurs simultaneously, unlike the classical retrograde activation sequence in type A WPW when high left atrial activation occurs before high right atrial activation, the pathway must also be relatively close to the specialized conduction system. The fact that retrograde activation of both the high right and left atria also occurs simultaneously during His bundle pacing in this patient (see inset on figure 7) and is similar to the retrograde activation sequence occurring during tachycardia is further evidence that the retrograde pathway lies close to the specialized conduction system, but on its left side.

Discussion

It might be expected that during a supraventricular tachycardia the refractory period of one of the bundle branches is such that for a given rate that bundle remains refractory and conduction to the ventricles proceeds down the other bundle branch. However, this cannot explain the bundle branch block pattern in the four patients described in this paper. In patients one and two the cycle length of the tachycardia was identical for the complexes showing a bundle branch block pattern and for those complexes showing normal intraventricular conduction. In patients three and four the cycle length of the tachycardia for the complexes showing a bundle branch block pattern was longer than the cycle length of the tachycardia during which normal intraventricular conduction occurred. An increased heart rate cannot therefore be the cause of the aberrancy of the QRS complex seen in these four patients.

Moe et al.5 in 1965 showed experimentally in the dog heart that during regular driving of the right atrium a critically timed atrial premature beat reached the right bundle branch while it was still refractory and that activation of the right bundle distal to the level of block occurred via antegrade conduction in the left bundle branch and retrograde invasion of the right bundle branch distal to the level of block in the right bundle. This right bundle branch block pattern could then be maintained by continuing to drive the right atrium at a critical frequency. Under these circumstances the right bundle branch will remain refractory because of regular retrograde invasion of the right bundle during the preceding driven beat. Evidence for retrograde invasion of a bundle branch in man has been obtained by Cohen et al.13 who showed that following an induced atrial premature beat a right bundle branch block configuration occurred. This aberrant QRS complex was followed by retrograde activation of the His bundle suggesting that, following antegrade conduction in the left bundle branch, the blocked right bundle

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branch had been invaded retrogradely, thereby producing a His bundle echo.

Two separate aspects of bundle branch block occurring during tachycardia should be considered. First, the initiation of the bundle branch block must be explained. In patients two, three, and four described in this paper, the bundle branch block and tachycardia were initiated by an atrial premature beat which had a short coupling interval and followed a preceding long basic paced cycle length. The bundle branch block was presumably due to arrival of the atrial premature impulse during the refractory period of one of the bundle branches, which is longer than that of the A-V nodal refractory period. The second aspect concerns the perpetuation of the bundle branch block during tachycardia. The perpetuation of the aberrant intraventricular conduction could not be explained on the basis of an increased ventricular rate during supraventricular tachycardia. Also, in all cases, the bundle branch block could be converted to normal intraventricular conduction by an appropriately timed ventricular premature beat or a fusion beat. These findings strongly suggested to us that the perpetuation of the aberrant intraventricular conduction during tachycardia was due to concealed retrograde conduction of one of the main bundle branches.

Further evidence for retrograde invasion of the bundle branches leading to aberration of the ventricular ECG pattern is supplied by examining the cycle length of the tachycardia with and without bundle branch block in the patients with anomalous atrioventricular connections. Patient two had a left-sided bypass, and right bundle branch block appeared during tachycardia. Examination of the diagrams in figure 5 demonstrates that the length of the reciprocal circuit will be the same irrespective of whether right bundle branch block or normal intraventricular conduction is present. If the length of the reciprocal circuit is constant, then the cycle length of the tachycardia will be the same whether or not bundle branch block is recorded. Indeed, the cycle length is 240 msec for both the beats showing right bundle branch block and those showing normal intraventricular conduction.

Patient three had a right-sided bypass and left bundle branch block and examination of the diagrams in figure 6 shows that the length of the reciprocal circuit, and therefore the cycle length of the tachycardia, will be the same in the presence of left bundle branch block or in normal intraventricular conduction. However, in this patient the cycle length of the tachycardia in the presence of left bundle branch block was 295 msec whereas it was 290 msec when normal intraventricular conduction occurred. This difference could be explained on the basis of observer error where such small intervals are involved. However, a more attractive explanation can be put forward. Durrer et al. found that endocardial activation of the right ventricle started 5-10 msec after the onset of the left ventricular cavity potential. This would explain why during normal antegrade A-V nodal-His conduction the onset of ventricular activation in the presence of left bundle branch block could occur 5-10 msec later than when normal intraventricular conduction was present. This delay would apply equally during a supraventricular tachycardia with antegrade His conduction, and a lengthening of the cycle length by 5 msec in the presence of left bundle branch block could be explained by this small delay in ventricular activation when the ventricles are activated solely by the right bundle branch.

This explanation also fits the shortening of the cycle length in tachycardia in patient four (fig. 7). In patient four the left bundle branch block cannot be explained on the basis of an increased ventricular rate and is most logically explained on the basis of the presence of a left-sided anomalous bypass and retrograde invasion of the left bundle branch. In the presence of left bundle branch block the intraventricular conduction time (H-V) might be expected to be longer than during normal intraventricular conduction and the tachycardia cycle length during left bundle branch block would therefore be longer by an appropriate amount. However in patient four the H-V time was 40 msec during both left bundle branch block and normal intraventricular conduction. A bypass is thought to be present in this patient, and if it were located on the left side of the specialized conduction system, then the longer cycle lengths in the presence of left bundle branch block can be explained on the basis of retrograde invasion of the left bundle with retrograde conduction in the bypass producing a longer reciprocal circuit than when normal intraventricular conduction occurs (see diagrams in fig. 7).

No anomalous A-V connection was present in patient one, and as illustrated in the diagrams in figures 1 and 2, the cycle length of the tachycardia was the same both during the right bundle branch block and normal intraventricular conduction.

If, in patients with a bundle branch block pattern during supraventricular tachycardia, the cycle length of the tachycardia shortens when the ECG pattern reverts to normal intraventricular conduction, then the presence of an anomalous pathway on the same side as the blocked bundle branch should be suspected.

References


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