Analysis of Re-entry Mechanisms in Three Patients with Concealed Wolff-Parkinson-White Syndrome

By Helmut Neuss, M.D., Martin Schlepper, M.D., and Jochen Thormann, M.D.

SUMMARY
Three patients with recurring attacks of supraventricular tachycardia and no electrocardiographic evidence of the Wolff-Parkinson-White syndrome (WPW syndrome) were studied using intracardiac recordings and atrial stimulation. The findings are interpreted as evidence of a concealed WPW syndrome. In all patients there was antegrade block of the anomalous atrioventricular (A-V) pathway while retrograde conduction was unimpaired and allowed the initiation of the observed reciprocating tachycardias. The diagnosis was based on the assumption that the ventricular myocardium was an essential link in the re-entry circuit. The three most important findings to support this assumption are: 1) retrograde conduction time, measured by the Q-A' interval (Q in ECG to atrial echo), and the rate of tachycardia were dependent on the mode of intraventricular conduction; 2) the first Q-A' interval of the tachycardia was independent of the A-H interval (initiation of atrial impulse to first activation of the His bundle) of the initiating premature atrial depolarization (PAD); 3) there was retrograde conduction following a ventricular premature beat during tachycardia at a time when the A-V node and/or the bundle of His would be refractory.

Additional Indexing Words:
His bundle recordings Unidirectional block Reciprocating tachycardia

The manifestation of re-entry mechanisms that do not follow the accessory pathway has been shown in patients with the Wolff-Parkinson-White (WPW) syndrome. Atrioventricular nodal (A-V) re-entry should therefore be considered a possible mechanism of supraventricular tachycardias (SVT) in patients with a WPW syndrome. Differentiation between A-V nodal re-entry and re-entry via an accessory pathway (AP) is based on the identification of the structures involved. If, for example, the ventricular myocardium is found to be an essential link in the re-entry circuit, then the postulation of re-entry confined only to the A-V junction can be excluded and reciprocation via an accessory pathway should be assumed.

In three patients without electrocardiographic evidence of a WPW syndrome, participation of the ventricular myocardium in the re-entry circuit was found to be present during paroxysmal SVT. This observation provided sufficient evidence to establish the diagnosis of ventriculo-atrial conduction via an AP as the underlying mechanism in these SVTs.

Methods
The patients consented to the electrophysiological studies, were in a nonsedated state and were not on antrirhythmic medication at the time of examination.

A multipolar electrode catheter was placed via the right femoral vein at the right side of the ventricular septum adjacent to the bundle of His, according to the method described by Scherlag et al. From this catheter, filtered, low right atrial potentials and His bundle depolarizations were recorded by selecting two suitable electrodes. A second quadrupolar catheter was positioned via the femoral vein in the right atrium. Two electrodes were used to record a high right atrial electrogram (AE), while the remaining two electrodes served for atrial stimulation. Simultaneously, several leads of surface ECG were registered. Surface ECG and intracardiac electrograms were recorded on magnetic tape and displayed with a paper speed of 100 mm/sec using an ink writing system. The atrial stimuli consisted of rectangular impulses of 2.0 msec duration and twice diastolic threshold. Stami were applied either in the form of premature atrial depolarizations (PAD) after each eighth spontaneous or paced beat, or continuously with an increasing rate.

Results
Case 1
A 17-year-old boy related a history of frequent episodes of tachycardia. The ECG at sinus rhythm (70 beats/min) showed a P-Q interval of 120 msec without evidence of WPW syndrome.

Sinus rhythm (100 beats/min) was present at the beginning of the electrophysiological study. P-Q interval measured 125 msec, with a P-A interval (P wave to atrial deflection in the His bundle electrogram [HBE]) of 30 msec, an A-H interval (atrial deflection in the HBE to His bundle potential) of 45 msec, and
an H-Q interval (His bundle potential to the onset of QRS complexes on the ECG) of 50 msec (see sinus beats in figs. 1, 2). Increasing the atrial rate to 200 beats/min, the A-H interval lengthened to only 80 msec. The H-Q interval and the QRS configuration remained unchanged.

At sinus rhythm a PAD with a coupling interval (P wave to stimulus artifact = P-S interval) of 360 msec was conducted with an unchanged A-H interval, while the H-Q interval increased to 80 msec and right bundle branch block (RBBB) occurred (fig. 1). This QRS complex was followed by an atrial echo (A'), which then initiated SVT. In all atrial potentials interpreted as atrial echoes (A'), deflections in AE occurred 20 msec prior to atrial potentials in the HBE. While the Q-A' interval (measured from the onset of QRS on the ECG to the initial rapid deflection of atrial potentials in AE) was 140 msec in the two QRS complexes with RBBB pattern, normalization of intraventricular conduction was accompanied by an abrupt shortening of the Q-A' interval to 80 msec. Concomitantly, the cycle length of tachycardia shortened from 300 to 250 msec.

When the coupling interval (P-S) was decreased to 280 msec (fig. 2), a PAD was conducted with nodal delay (A-H, 85 msec) as well as with delay in the His-Purkinje system (HPS) with an H-Q interval of 120 msec. The resulting QRS was of left bundle branch block (LBBB) configuration and was followed by an atrial echo. The Q-A' interval was then 80 msec, identical to that found during SVT with normal intraven-

**Figure 1**
ECG (leads I, II, and III), high right atrial electrogram (AE) and His bundle electrogram (HBE) in case 1. Paper speed 100 mm/sec. Time intervals in msec. In this and all subsequent figures: \( A \) = atrial potentials; \( A' \) = atrial echo; \( H \) = His bundle potentials; \( V \) = ventricular potentials; \( S \) = stimulus artifact. SVT is induced by a PAD with a coupling interval of 360 msec.

**Figure 2**
ECG (leads I, II and III), high right atrial electrogram (AE) and His bundle electrogram (HBE) in case 1. Panel a) At sinus rhythm a PAD with a coupling interval of 280 msec results in a QRS complex with LBBB patterns. Panel b) A PAD with the same coupling interval was blocked distal to the bundle of His and no atrial echo was elicited.
tricular conduction. A SVT was not initiated since block distal to the bundle of His prevented excitation from reaching the ventricles.

A PAD with the same coupling interval (280 msec) was blocked distal the bundle of His (fig. 2). This block was most probably due to the longer preceding R-R interval (670 msec compared to 590 msec in fig. 2a). While A-V nodal conduction was delayed to the same extent as in figure 2a, conduction block below the bundle of His prevented the appearance of an atrial echo.

At a driving cycle length of 400 msec (150 beats/min), PADS with coupling intervals shorter than 270 msec repeatedly elicited SVT. These PADS were conducted without apparent ventricular aberrancy and unchanged H-Q intervals. The minimal prolongation of A-H interval needed to start a SVT was found to be 90 msec. If the A-H interval of the induced PAD was longer than 90 msec, the Q-A' interval remained constant.

Supraventricular tachycardia in this patient could be terminated by rapid atrial stimulation. There was atrial capture at a paced rate of 280 beats/min and a 2° A-V block with Wenckebach periods. If pacing was stopped and the last stimulated atrial depolarization was blocked proximal to the bundle of His, the tachycardia was terminated.

Interpretation

The short P-Q interval, due to a short A-H interval, with a narrow QRS complex in a patient with SVT suggests the existence of the syndrome of Lown, Canon, and Levine7 and re-entry confined to the A-V junction should be considered as a possible mechanism in SVT.8 The evidence obtained from catheterization studies reveals, however, that the tachycardia could not possibly result from longitudinal dissociation of the A-V junction. The dependency of retrograde conduction time (Q-A' interval) and the rate of tachycardia on intraventricular conduction proves that the ventricular myocardium is an essential link in the re-entry circuit.

When RBBB pattern occurred during SVT, Q-A' was 140 msec and the cycle length of tachycardia 300 msec. Normalization of intraventricular conduction during SVT shortened the Q-A' interval (80 msec) and the cycle length of tachycardia (250 msec). The postulation of an AP between the right ventricle and the right atrium may explain this relationship. During normal intraventricular conduction and during delayed left ventricular activation in LBBB (fig. 2a), the right ventricular myocardium, and thereby the insertion of the AP, are activated without delay. Retrograde conduction time (Q-A') is therefore short (80 msec) and identical in both instances. During RBBB the excitation wave reaches the right ventricle and consequently the postulated right-sided AP with delay. Under these conditions retrograde conduction time will be prolonged (Q-A', 140 msec). Since the re-entry time is composed of the sum of conduction times in each involved part, it is evident that the rate of tachycardia is slower when there is delay due to RBBB. This is in accordance with the assumption of a re-entry mechanism in which the right ventricle takes part.

The unusual sequence of atrial potentials in atrial echoes cannot possibly be explained by a migration of the position of AE electrodes since the sequence of atrial potentials during sinus beats or atrial echoes was reproducible during the entire study. While the sequence is compatible with sinoatrial re-entry, this alternative hypothesis does not explain our findings. Our explanation is that, given a lateral right ventricular insertion of AP, spread of retrograde atrial excitation via the AP may reach the AE electrodes before the electrodes positioned in the vicinity of the A-V node (HBE).

Case 2

The 36-year-old male patient was admitted because of frequent paroxysms of rapid heart action. Clinical examination revealed no pathologic findings. The ECG on admission showed a normal P-Q interval (160 msec) and no signs of WPW syndrome.

During sinus rhythm (90 beats/min) the P-Q interval was 140 msec at the beginning of the electrophysiological study and its components included P-A, 45 msec; A-H, 55 msec; H-Q, 40 msec. One-to-one A-V conduction continued up to an atrial driving rate of 200 beats/min, but A-H increased to only 90 msec. At sinus rhythm, PADS with a coupling interval shorter than 300 msec elicited SVT with rates between 185 and 240 beats/min. At the beginning of these runs, QRS complexes showed a LBBB pattern (fig. 3). Normalization of intraventricular conduction was accompanied by sudden decrease in cycle length (R-R intervals shortened from 320 to 270 msec) and a shortening of the Q-A' intervals (Q-A' in LBBB, 190 msec; in incomplete LBBB, 150 msec; and in normalized QRS complexes, 130 msec).

At a basic heart rate of 100 beats/min, PADS with coupling intervals of 290 msec and shorter elicited atrial echo phenomena and runs of SVT. Paroxysms triggered at this heart rate always showed a RBBB pattern with a cycle length of 240–260 msec (fig. 3b). The Q-A' intervals were identical in beats having a RBBB configuration to those of undisturbed intraventricular conduction (130 msec).

In a few runs of SVT with normal QRS complexes, sudden prolongations of A'-H were observed (fig. 4).
A'-H intervals corresponding to QRS complexes 1–5, 7, 8, 10 and 11 in figure 4 ranged from 110–130 msec. A'-H intervals preceding the sixth and ninth QRS complexes were prolonged to 200 and 210 msec respectively. This led to a prolongation of Rv-Rs and Rv-Ra intervals to 360 msec and to phasic aberrant intraventricular conduction of QRS complex nos. 7 and 10 showing incomplete left bundle branch pattern. The corresponding Q-A' intervals were again prolonged in a progression analogous to the Q-A' intervals during intraventricular conduction disturbances presented in figure 3.

Interpretation

As in case 1, the relation of both the Q-A' interval and the rate of SVT to intraventricular conduction disturbances can serve as evidence that the ventricular myocardium is part of the re-entry circuit. It is thus reasonable to assume the existence of an AP between left ventricle and left atrium since retrograde conduction time (Q-A' interval) was longer during SVT with complete and incomplete LBBB.

Case 3

A 25-year-old female patient gave a history of recurrent episodes of tachycardia. With a normal P-Q interval (150–160 msec), the ventricular complex did not show evidence of WPW morphology.

During sinus rhythm (90 beats/min), the P-Q interval of 160 msec was subdivided into a P-A, 40 msec; A-H, 70 msec; H-Q, 50 msec. At coupling intervals (P-S) less than 360 msec, PADs evoked SVT with rates ranging between 185–210 beats/min, as long as they were conducted with a P-Q interval of at least 180 msec. The Q-A' interval during these runs always measured 130 msec, whether intraventricular conduction was normal or disturbed by functional RBBB (fig. 5). Also normalization of QRS configuration did not cause abrupt changes in the rate of tachycardia. The beat-to-beat alterations seen in figure 5 were due to variations of the A'-H and H-Q intervals. A refractory-dependent LBBB could never be evoked by PADs.

After administration of 50 mg ajmaline, SVT could be elicited during sinus rhythm (100 beats/min) if PADs were conducted with a P-Q interval of 240 msec. The cycle length of SVT was constant (345–350 msec) due to a prolongation of the Q-A' interval to 180 msec, while the A'-H interval remained unchanged. During these runs right ventricular extrasystoles occurred spontaneously. At a coupling interval of 270 msec, the ventricular depolarization occurred 20 msec prior to the expected antegrade H potential (fig. 6). This H spike can be identified at the beginning of the ventricular potential. Following this premature ventricular depolarization, 220 msec later, an A' potential occurred, reducing the A'-A' interval to 320 msec. This premature beat caused the prolongation of the following A'-H interval. Similar findings could also be observed prior to the administration of ajmaline with spontaneously occurring right ventricular premature beats.

Interpretation

In this case normal conduction and conduction disturbances in the right bundle branch did not alter the Q-A' interval, and consequently did not change the rate of tachycardia. While by this finding, an AP between the right atrium and the right ventricle can be ruled out, A-V junctional re-entry cannot be excluded. The most important argument for V-A conduction via an AP is the shortening of the A'-A' interval by right ventricular extrasystoles. They did not occur prematurely enough to reach the His bundle (i.e., the final common pathway if SVT were due to longitudinal dissociation of the A-V node) prior to the arrival of orthograde excitation. A few ventricular extrasystoles were even followed by an antegrade H potential at the very beginning of the V potentials (fig.
CONCEALED WPW SYNDROME

Figure 4
ECG (leads I and III), atrial electrogram (AE) and His bundle (HB) recording in case 2. SVT with alternating A'-H intervals and phasic aberrant intraventricular conduction. Intermittent LBBB alters the Q-A' interval.

6), so that a reverse conduction via His bundle and A-V node is excluded. A similar observation has recently been reported by Zipes et al.* Because a reduction of A'-A' interval results, V-A conduction can only occur via an AP directly between the left ventricle and the left atrium. A left-sided AP would explain the additional increase in retrograde conduction time of Q-A' from 180 to 220 msec following right ventricular extrasystoles. The existence of a right-sided AP can be ruled out since the Q-A' interval would be expected to remain unchanged, or at least not to differ to that extent.

Figure 5
ECG (leads V, and I), high right atrial electrogram (AE), and His bundle electrogram (HB) in case 3. Supraventricular tachycardia is induced by a PAD with coupling interval of 280 msec. Conduction disturbance in the right bundle branch has no effect on the Q-A' interval.
Discussion

The electrophysiological data obtained in all our patients favor the postulation of V-A conduction during SVT via an AP bypassing the A-V junction.

Reciprocal activation resulting in the occurrence of echo phenomena is only possible when the re-entry time is prolonged sufficiently to allow the recovery of the previously excited re-entry path. In SVT due to longitudinal dissociation of the A-V node, the return level is localized above the bifurcation of the specialized conduction system, i.e., proximal to the bundle of His. The occurrence of echo phenomena is dependent, therefore, on a critical A-H prolongation. On the other hand, when the ventricular myocardium is involved in the re-entry circuit, conduction delay may occur in the HPS exclusively, as was observed in the first patient (fig. 1).

Atrial echoes via an AP could be elicited only if the ventricular depolarization occurred after a PAD. While block distal to the bundle of His should not affect re-entry localized in the A-V junction, it will prevent atrial echoes via an AP. This differentiating criterion was recently pointed out by Lau et al. and is an argument for V-A conduction via an accessory pathway in case 1 (fig. 2b).

The continuation of the first Q-A' interval in beats with identical patterns of intraventricular conduction, but various prolongations of the A-H interval of the PAD may serve as a further argument for V-A conduction by means of an AP. By careful analysis of V-A conduction in patients with WPW syndrome, Wellens and Durrer have recently shown that refractory-dependent delay in retrograde conduction time via an AP is very slight. Therefore, the coupling interval of the atrial echo (Q-A') should be constant. In the case of longitudinal dissociation of the A-V junction, the timing of QRS and A' may vary in SVT as the timing is a function of antegrade and retrograde intranodal conduction times.

Further evidence of accessory V-A conduction is provided by the behavior of the Q-A' interval during SVT with and without functional bundle branch block. This behavior also allows conclusions regarding the insertion of the AP. In the first patient the postulation of an AP inserting into the right ventricle is reached because of delay shown during RBBB, while in the second patient an AP inserting into the left ventricle is hypothesized because of delay during LBBB. Therefore, under the given conditions, the Q-A' intervals were longer, and accordingly the rate of SVT low. This agrees with the interpretation given by Slama and Coumel in four patients with concealed WPW syndrome.

In the third patient there was spontaneously occurring RBBB during SVT also. The Q-A' interval and the rate of tachycardia were not altered however. According to this finding, V-A conduction via a rightsided AP can be excluded. The fact that following ventricular premature beats V-A conduction occurred at a time when the His bundle was refractory provides convincing proof that re-entry did not involve the A-V junction (fig. 6).

While V-A conduction via the postulated AP was unimpaired up to high rates (1:1 V-A conduction during SVT), no evidence for accessory A-V conduction could be ascertained in our three cases, either during sinus rhythm or during high rate atrial pacing or with PADs. Generally, unidirectional block of the AP should be considered, particularly if intra-atrial conduction disturbances as the cause for concealed WPW syndrome can be ruled out, for instance, by studying the intra- and interatrial activation sequences. Left atrial stimulation may help to differentiate between concealed WPW syndrome caused by interatrial conduction disturbances and instances of true unidirectional block of the AP.

Considering the electrophysiological findings obtained by Fuente et al., unidirectional block of an AP is probably not rare. It is obvious that the therapeutic approach of the tachycardia in cases with concealed WPW syndrome might be different from cases with A-V junctional tachycardias. It therefore is advisable to identify the re-entry path in patients with recurrent tachycardias whose ECGs did not show WPW morphology. In patients with therapy-resistant tachycardias due to this mechanism, invasive diagnostic methods such as epicardial mapping should be considered, since a surgical intervention might be the only successful remedy.

Circulation, Volume 51, January 1975
CONCEALED WPW SYNDROME

References

Analysis of re-entry mechanisms in the three patients with concealed Wolff-Parkinson-White syndrome.
H Neuss, M Schlepper and J Thormann

Circulation. 1975;51:75-81
doi: 10.1161/01.CIR.51.1.75
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1975 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/51/1/75

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/