A-V Nodal Reentrance

An Unexpected Mechanism of Paroxysmal Tachycardia in a Patient with Preexcitation

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SUMMARY

Electrophysiologic studies were performed in a patient with type B preexcitation and recurrent paroxysmal supraventricular tachycardia (PSVT). Atrial pacing at rates of 80/min and faster produced QRS normalization suggesting failure of the anomalous pathway. At a paced rate of 120/min, Wenckebach periods proximal to the His bundle as well as episodes of PSVT were noted. These episodes all occurred when a critical P-H interval of 300 msec was achieved. PSVT was also induced by coupled atrial pacing. At coupling intervals of 800–530 msec, anomalous pathway conduction was intact. At coupling intervals of 520 msec or shorter, QRS normalization occurred. At intervals of 480–280 msec, PSVT was induced. The P-H interval of the coupled beat inducing PSVT was always between 300 and 350 msec. Intraatrial recording revealed atrial echoes following each QRS of the PSVT.

In summary, critical A-V nodal delay appeared necessary for induction of PSVT. This delay was achieved either by coupled pacing or during pacing-induced Wenckebach periods. The PSVT occurred at rates at which the anomalous pathway would seem to be inoperative. These findings strongly support A-V nodal reentry as the mechanism of PSVT in this patient without participation of the anomalous pathway. In this patient and other patients with similar findings, surgical ablation of the anomalous pathway would probably not be useful in prevention of recurrent PSVT.

Additional Indexing Words:
His bundle electrogram
Wolff-Parkinson-White syndrome
Dual pathways
Longitudinal dissociation
A-V conduction
Atrial pacing

CURRENT EVIDENCE suggests that most cases of preexcitation reflect the presence of an anomalous A-V connection in the A-V ring (Kent bundle).1-8 Paroxysmal supraventricular tachycardia (PSVT) complicating preexcitation appears to reflect circus movements utilizing both the anomalous and normal A-V pathways. Epicardial mapping may allow demonstration of the site of insertion of the anomalous pathway.2-8 Surgical destruction of this site may produce cure of preexcitation and prevention of subsequent PSVT.4-5

In this report, we describe a patient with preexcitation and recurrent PSVT. Electrophysiologic studies, utilizing His bundle recording and cardiac stimulation, strongly suggest that PSVT reflected A-V nodal reentrance, without dependence upon the anomalous pathway. The findings imply that in some patients with preexcitation, surgical destruction of the anomalous pathway will fail to influence subsequent occurrence of PSVT.

Report of Case

A 25-year-old male was admitted to Cook County Hospital following a syncopal episode. The patient had a history of frequent palpitation and attacks of “fast heart rates.” There was no other history of cardiovascular disease.

Physical examination and routine laboratory investigation was within normal limits. Electrocardiograms revealed typical type B preexcitation with a short P-R, prolonged QRS, and positive delta wave in V6. Several episodes of PSVT were recorded during hospitalization, all with a narrow QRS (fig. 1).
Electrophysiologic Studies

His bundle (H) electrograms were recorded on a multichannel oscilloscopic photographic recorder (Electronics-for-Medicine, Dr 16) using previously described catheter techniques, at paper speeds of 100 and 200 mm/sec.9,10 Atrial pacing was performed with a bipolar pacing catheter placed against the lateral wall of the right atrium. Stimuli were supplied by a battery-powered coupled pulse generator (Medtronics 5837) in both the demand and coupled modes.

The presence of delta waves were used as evidence of preexcitation.10 P-delta interval was measured from the onset of the P wave to the onset of the delta wave. P-H interval was measured from the onset of the P wave to the first peak potential of the His bundle electrogram. H-V interval was measured from the first peak potential of the His bundle electrogram to the onset of the QRS as recorded from multiple surface ECG leads. During atrial pacing, P-delta, and P-H were measured from the pacing stimulus. Latency time, from the stimulus to the onset of the atrial electrogram remained constant with pacing at varied rates.

At the onset of the study, the tripolar catheter was first positioned in the atrium for recording of atrial electrograms. Coupled atrial stimulation with the bipolar pacing catheter provoked several episodes of PSVT. These were all identical in regard to induction (fig. 2). In each episode, a coupled premature stimulus was conducted with a long P-R and QRS normalization. The narrow QRS was followed by an atrial echo and a run of PSVT at rates of 128–134/min. Each complex of the PSVT was narrow and followed by an atrial echo. These findings could be interpreted in two ways: (1) PSVT could be reentrant, dependent upon unidirectional block in the anomalous pathway and slow conduction in the normal pathway for induction.11–13 PSVT in this circumstance would depend upon antegrade conduction in the normal pathway and retrograde conduction in the anomalous pathway. (2) PSVT could be reentrant utilizing only the A-V node without dependence on anomalous pathway conduction (see below).

The tripolar catheter was then placed across the tricuspid valve for recording of His bundle electrograms. In order to achieve a stable catheter position, it was necessary to pass the catheter slightly more distally in the right ventricle than usual. In this position, the atrial electrogram recorded was small-to-absent. The H potential may have been from low in the His bundle or high in the right-bundle branch.

Control recordings during sinus rhythm revealed a P-delta interval of 110 msec and a P-H interval of 130

Figure 1
Electrocardiogram demonstrating preexcitation. This case is classified as type B, because predominant initial forces are directed leftward.

Figure 2
Induction of PSVT with a coupled atrial extrastimulus. Shown are ECG leads V1, II, and III with an intraatrial electrograms (AE). P = P waves; R = QRS; A = atrial electrograms. The first complexes are conducted beats with preexcitation. The coupled extrastimulus (arrow) is conducted with a long P-R and QRS normalization. This QRS is followed by an atrial echo and a run of PSVT at a rate of 128 beats/min. Note that each QRS of the PSVT is followed by an atrial echo. Paper speed is 100 mm/sec and time lines are at 1 sec on this and all subsequent illustrations.

Circulation, Volume XLVII, June 1973
Examination of these episodes revealed a constant pattern. During the Wenckebach period, progressive P-H prolongation was noted. At a P-H of 300 msec PSVT was induced.

Effective refractory periods (ERP) were measured with an atrial extrastimulus supplied after every eighth to tenth sinus beat. The interval from the preceding sinus P wave (P) to the extrastimulus (Pi) was measured for determination of refractory periods. The cardiac cycle was scanned at P-Pi intervals of 800–270 msec in 10–20-msec steps. At coupling intervals of 800–530 msec, anomalous pathway conduction remained intact (fig. 5A). At a coupling interval of 520 msec (anomalous pathway ERP) or shorter, the anomalous pathway failed with normalization of the QRS and P-H prolongation (fig. 5B and C). At coupling intervals of less than 480 msec, episodes of PSVT were provoked by the coupled stimulus (fig. 4D and E). These episodes of PSVT occurred when the P-H of the coupled beat was between 300 and 350 msec. At a coupling interval of 270 msec, the effective refractory period of the atrium was achieved (fig. 5F). The A-V nodal ERP could not be measured because the atrial ERP was limiting. The episodes of PSVT induced during the study were all easily converted with coupled atrial pacing.

The findings suggest A-V nodal reentrance as the mechanism of PSVT in this patient. This is based upon the following observations: (1) PSVT at a rate of 128–134/min was evoked by premature atrial stimulation. In contrast, the anomalous pathway would not support conduction above a paced rate of 80/min. This suggests that the anomalous pathway was not utilized during PSVT in this patient. (2) There was a gap between the anomalous pathway ERP (520 msec) and the zone for induction of PSVT (490–280 msec). The P-H of the coupled beat in this zone was between 300 and 350 msec. One could postulate that PSVT utilized the anomalous pathway. If this was the case, unidirectional block of the anomalous pathway occurred at a coupling interval of 520 msec. However, for PSVT to be induced, the P-H of 300–350 msec was necessary for recovery of the anomalous pathway for retrograde conduction. A more likely possibility is that the gap is an artifact and not directly related to the genesis of the PSVT. If PSVT was due to A-V nodal reentrance, then the critical P-H for induction of reentry was between 300 and 350 msec. This occurred at coupling intervals of 490–280 msec, which were shorter than the anomalous pathway ERP. (3) Strongly supporting the latter view was the occurrence PSVT at a paced rate of 120/min during Wenckebach sequences. At this rate, the anomalous pathway was inoperative. PSVT occurred at a critical P-H of 300 msec, a P-H in the range of P-H intervals evoked by the coupled extrastimuli which induced PSVT.

Discussion

Both pathologic as well as electrophysiology studies suggest that most cases of preexcitation
Continuous recordings showing induction of PSVT with pacing-induced Wenckebach periods. The first two and last two pacing impulses of a series of impulses are labeled with arrows. The pacing rate is 120 beats/min. Note the Wenckebach sequence with increase in P-H from 200 to 300 msec. In the cycle marked X, there is sudden shortening of the R-R intervals because of induction of PSVT following the critical P-H of 300 msec. The pacing spikes in the cycle marked X and all spikes in all cycles following probably do not capture the atria. Because of catheter position, atrial electrograms of the atrial echoes are not well visualized but appear to be seen following the first, second, and third complexes of the bottom tracings.

Figure 4

Continuous recordings showing induction of PSVT with pacing-induced Wenckebach periods. The first two and last two pacing impulses of a series of impulses are labeled with arrows. The pacing rate is 120 beats/min. Note the Wenckebach sequence with increase in P-H from 200 to 300 msec. In the cycle marked X, there is sudden shortening of the R-R intervals because of induction of PSVT following the critical P-H of 300 msec. The pacing spikes in the cycle marked X and all spikes in all cycles following probably do not capture the atria. Because of catheter position, atrial electrograms of the atrial echoes are not well visualized but appear to be seen following the first, second, and third complexes of the bottom tracings.

reflect the presence of an anomalous pathway located in atrioventricular ring (Kent bundle). These anomalous connections predispose to the PSVT frequently seen in patients with preexcitation, serving as one limb of a circus movement, which also utilizes the normal pathway (A-V node and His bundle). Premature atrial stimulation suggests the following mechanism of PSVT in many of these patients. A critically timed atrial premature stimulus blocks in the anomalous pathway with slow propagation in the normal pathway. This results in a premature beat with a long P-R interval and narrow QRS. During slow conduction in the normal pathway, the anomalous pathway recovers and it thus is available for retrograde conduction back to the atria. During PSVT, the normal pathway serves as the antegrade route from atria to ventricles and the anomalous pathway serves as the retrograde pathway back to the atria. The reverse type of reciprocation has also been described in which the premature antegrade response blocks in the normal pathway but does conduct in the anomalous pathway, returning then via the normal pathway. QRS complexes with this type of PSVT are aberrant.

Another type of reentrance appears to occur in many patients with PSVT, but without preexcitation. In these patients longitudinal dissociation occurs within the A-V node with development of two functional pathways, each with its own
conduction properties. Bigger and Goldreyer studying six consecutive patients with PSVT, demonstrated that critically timed atrial stimulation could provoke atrial echoes as well as PSVT. In these cases, it was postulated that the premature stimulus inducing the tachycardia was blocked in one A-V nodal pathway, which during slow conduction in the other pathway recovered and was available for retrograde conduction. Episodes of reentrant tachycardia could then occur using one A-V nodal pathway as antegrade route and the other as retrograde route. The anatomic basis for such functional dissociation has not been defined, but microelectrode studies in rabbit hearts have demonstrated a site of reentry in the A-N region of the A-V node during experimental PSVT.

Goldreyer and Damato further evaluated the mechanisms of PSVT in patients without preexcitation. They demonstrated that the occurrence of A-V nodal reentrance was dependent upon the development of a critical degree of A-V nodal slowing, as measured as a critical P-H interval with coupled premature stimulation. They further showed that when this critical P-H occurred during pacing-induced Wenckebach periods, that A-V nodal reentry occurred. In a subsequent paper, it was demonstrated that other means of inducing critical A-V nodal delays also could allow the demonstration A-V nodal reentrance. In a patient with A-V dissociation, retrograde concealed conduction allowed sinus beats to be transmitted with critical P-H intervals allowing the demonstration of reciprocation.

Superficial examination of the patient presently reported suggests reentrant tachycardia utilizing the anomalous pathway, in that premature atrial stimulation produced QRS normalization, P-R prolongation, and PSVT. One would postulate that the premature impulse blocked in the anomalous pathway and conducted slowly in the normal pathway with reentry via the anomalous pathway. However, examination of the electrophysiologic data strongly suggests A-V nodal reentry as the mechanism of the tachycardia. This is based on the following observations: (1) The anomalous pathway failed at a paced rate of 80 beats/min. This failure was total, with complete normalization of QRS at this and all faster rates. The PSVT in this patient occurred at rates of 128-134 beats/min. It is unlikely that the anomalous pathway could support conduction at this rate. (2) Coupled pacing revealed the anomalous pathway-effective refractory period to be 520 msec while the echo zone for induction of PSVT ranged from 490 to 280 msec. This is consistent with PSVT utilizing the anomalous pathway as the retrograde route from ventricles to atrium with the critical P-H of 300-350 msec being necessary for recovery of this pathway. The more likely alternative is to postulate A-V nodal reentrance with a P-H of 300 msec necessary for the
occurrence of A-V nodal reentry. (3) Pacing-induced Wenckebach periods induced PSVT. This always occurred at a critical P-H of 300 msec. The findings closely resemble those described by Goldreyer and Damato for diagnosis of A-V nodal reentrance.

A word of caution is necessary regarding our conclusions. These are partly based upon the premise that antegrade properties of the anomalous pathway reflect its retrograde properties. We have assumed that the anomalous pathway cannot conduct at rates of 80/min or faster in a retrograde direction, as would be necessary if the anomalous pathway were the retrograde route to the atria during PSVT. Since we have not studied retrograde conduction with ventricular pacing in this patient, this is an unprovable assumption. However, the additional supporting evidence for A-V nodal reentrance cited in the previous paragraph, (observations nos. 2 and 3) are still valid.

Clinical Implications

The demonstration of A-V nodal reentry as a mechanism of PSVT in a patient with preexcitation has important therapeutic implications. Most important of these relates to current surgical techinics being utilized in patients with preexcitation. Destruction of the anomalous pathway in a patient with preexcitation and A-V nodal reentrant PSVT would probably have no effect on the future occurrence of tachycardia. Although technical inadequacies of mapping and surgical techinics might explain surgical failures for prevention of recurrent tachyarrhythmias, the presence of A-V nodal reentry is another potential cause of failure.

A second point of interest concerns the relationship of A-V nodal reentrance and the presence of preexcitation. A-V nodal reentrance in the present case could be a chance phenomenon of simultaneous occurrence of two abnormalities of cardiac conduction. It is also possible that patients with preexcitation may also have related functional abnormalities of the A-V node.

The present case suggests the necessity for careful analysis of additional cases of preexcitation, with specific attention to the functional evaluation of both the normal and anomalous pathways. This can be done by utilizing techinics of cardiac stimulation with simultaneous His bundle recording. This type of study would certainly be indicated in patients with preexcitation who are being evaluated for surgical therapy.

References


Circulation, Volume XLVII, June 1973


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Circulation. 1973;47:1267-1273
doi: 10.1161/01.CIR.47.6.1267
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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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:
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