PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA
(PSVT)

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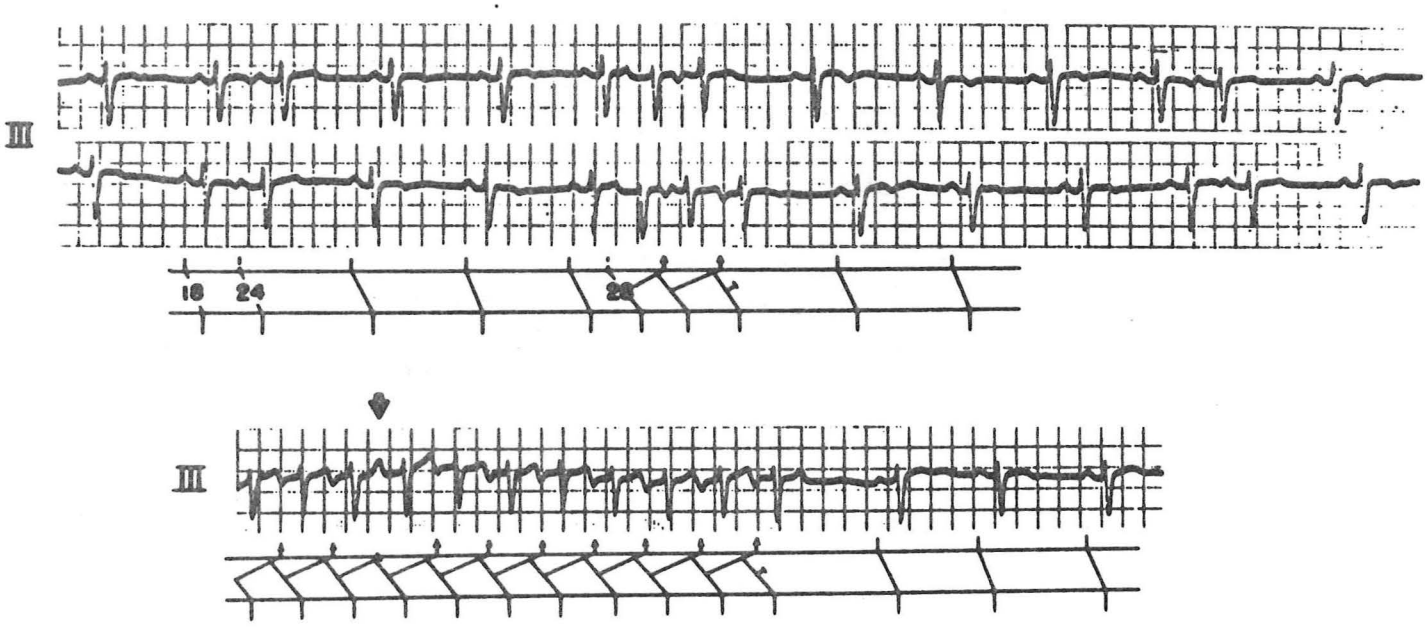

MAY 4, 1978

Atrial tachycardias were originally described in 1867 by Cotton (1) and later in 1887 by Bristowe (2) and in 1889 by Bouveret (3). These cases were presumably due to paroxysmal supraventricular tachycardia due to the nature and rates of the rhythm disturbance though not proven. Supraventricular tachycardia was found by Katz and Pick in 671 tracings (1.1%) and atrial tachycardia in 291 tracings (0.6%) when the electrocardiograms were reviewed in 50,000 consecutive patients (4). However, it should be noted that these incidence figures may be misleading as this was the incidence in patients, not in the general population.

Kissane et al found 361 patients (3.6%) with supraventricular paroxysmal tachycardia out of 9950 patients (5). The cardiac status of these patients was as follows:

Cardiac Status	Number of Patients	Percent
Normal heart	122	34%
Rheumatic heart disease	123	34%
Atherosclerotic heart disease	51	14%
Hypertensive heart disease	12	3%
Thyrotoxic heart disease	16	5%
Other	37	10%

More recent series have shown similar distributions (6,7).

Paroxysmal supraventricular tachycardia (PSVT) has been described in 4-8% of patients with an acute myocardial infarction (8,9,10). PAT with A-V heart block has frequently been associated with digitalis intoxication (10). In patients who are susceptible to PSVT, paroxysms have been precipitated by 1) deep inspiration (11,12), 2) hyperventilation (13), 3) exercise (14), 4) changes in position (15), 5) swallowing (16), and 6) emotional stress (17).

HEMODYNAMIC EFFECTS

In patients without organic heart disease, paroxysmal supraventricular tachycardia causes little change in cardiac output or disability to the patient early, but with prolonged episodes some impairment may occur (18). Wegria et al (19) and Maxwell et al (20) have shown experimentally that small decreases in cardiac output occur with a paced tachycardia but not with exercise-induced tachycardia nor with tachycardia secondary to hypothalamic stimulation (21).

Coronary blood flow appears to be maximal during sinus tachycardia between rates of 160 to 180 per minute in normal hearts. With sinus tachycardia rates between 180 and 200, coronary artery flow tends to decrease slightly (17). Corday et al (22) found that coronary blood flow decreased by 38.2% in patients with atrial tachycardia followed by an increase in flow at the cessation of the tachycardia.

Initially, a decrease in systolic, diastolic and pulse pressure occurs which may be to shock levels; this is particularly marked in the upright position (18,23). Below is shown an arterial pressure as a patient develops paroxysmal supraventricular tachycardia.

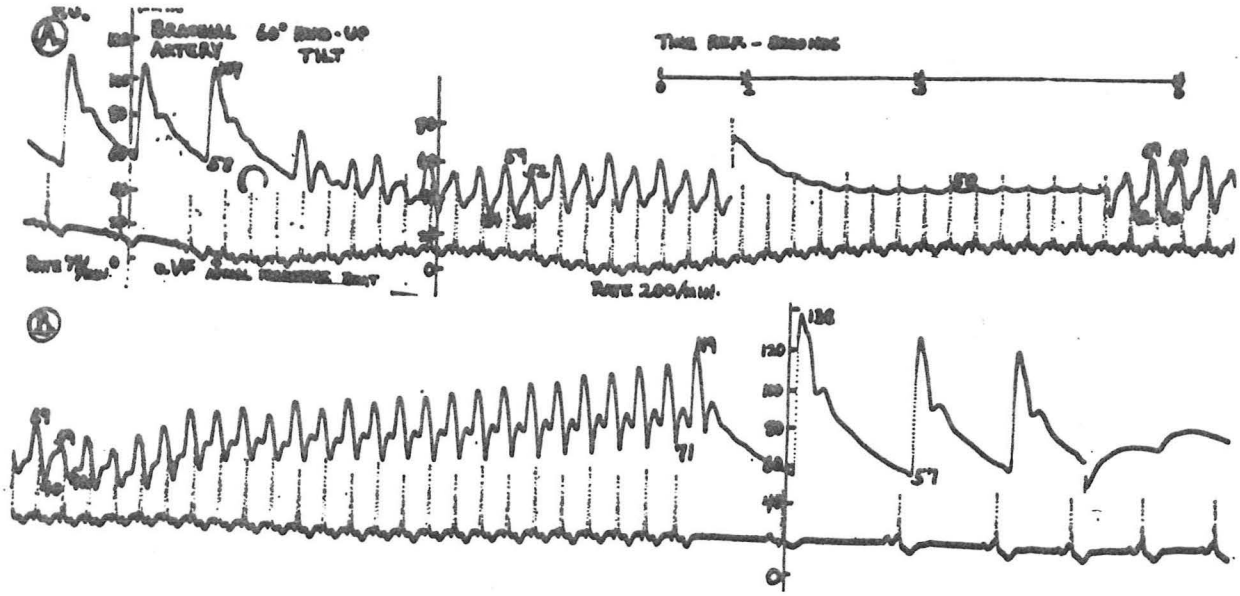

FIGURE 1. Illustrates the drop in brachial artery pressure almost immediately after the onset of paroxysmal atrial tachycardia (18).

A) Note the brachial artery pressure, which was 107/58, dropped to as low as 52/34. The hypotensive state is the result of inadequate ventricular filling due to shortening of diastole as a consequence of the tachycardia. As a result, there is a drop in cardiac output.

B) Note the return of pressure towards normal as the tachycardia persists due to increased ventricular filling due to elevation of atrial pressures with a return of cardiac output towards normal. With cessation of the tachyarrhythmia there is overshoot of arterial pressure.

Systemic arterial pulsus alternans was common early after the initiation of paroxysmal supraventricular tachycardia particularly in the upright position (18).

Right atrial pressure was elevated indicating that decreased venous return was not responsible for the fall in pressure and cardiac output (18,23). Pulmonary artery pressures did not change significantly (18). Sudden rises in left atrial pressure occurred with the onset of the tachycardia becoming maximal after 20 seconds.

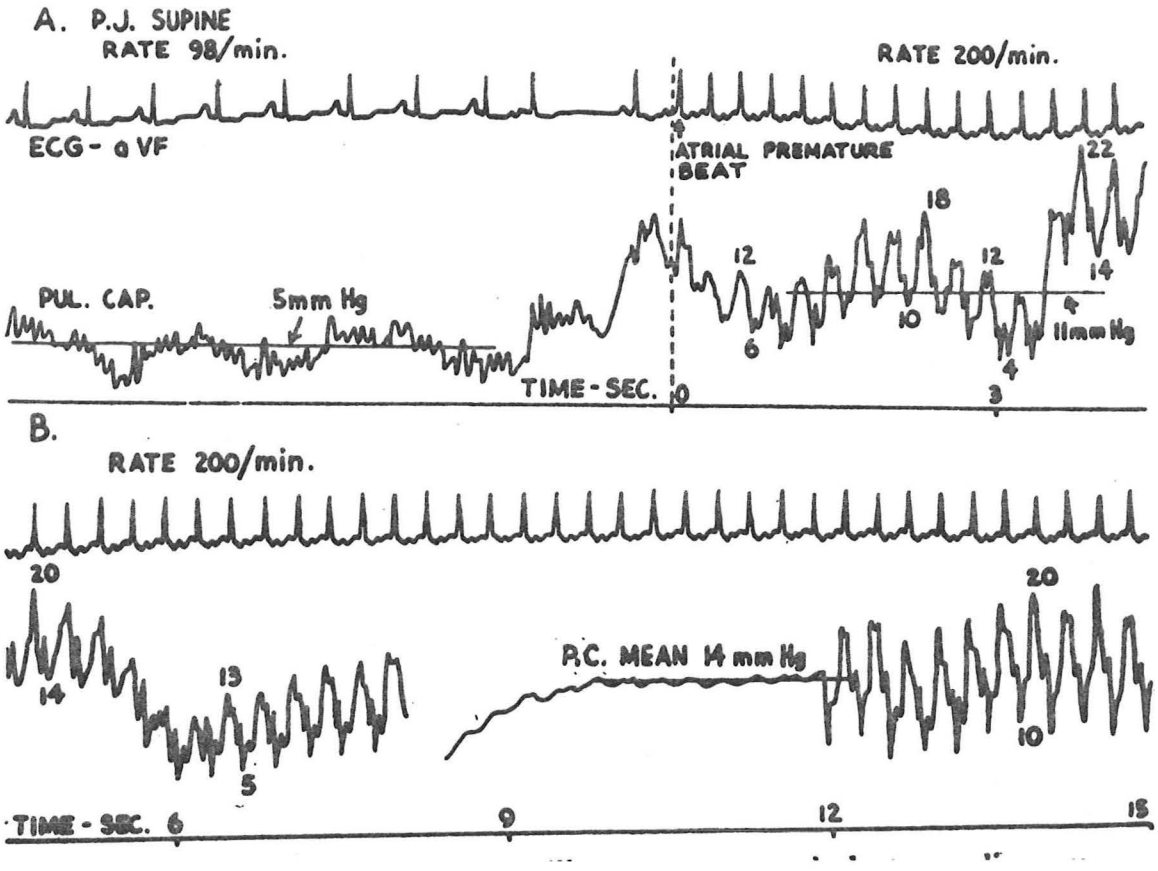

FIGURE 2. Pulmonary Capillary Wedge Pressure with PAT.
A) Note the sudden increase in pressure with the onset of tachycardia and the large a waves that also occur.

Large a waves (atrial waves) occur presumably because atrial systole starts during ventricular systole and the atrial contraction against closed atrioventricular valves results in the large a waves (24).

A marked decrease in stroke volume also occurs (18,23). As cardiac output either stays the same or diminishes slightly and heart rate more than doubles, then stroke volume decreases more than 50%.

In patients with prolonged tachycardia or with concomittant heart disease, marked falls in cardiac output may occur leading to hypotension, vascular collapse, heart failure, angina pectoris, and/or myocardial infarction (17). In post tachycardia beats there is a sudden increase in systolic and pulse pressure with systolic overshoot (18,23).

SYMPTOMS AND SIGNS

Paroxysmal supraventricular tachycardia (PSVT) usually has sudden onset and termination. In patients with normal hearts the symptoms are usually mild. Many patients note palpitations or an uneasy feeling in their chest. The longer the paroxysms the more symptoms patients notice; the majority of patients do not even recognize short bursts of PSVT. Some patients describe precordial discomfort or pain, weakness, dizziness or syncope, nausea, and/or vomiting. Those that have pain rarely describe a pain suggestive of angina pectoris with a normal heart though a small percentage describe classic angina pectoris (17).

In older patients and patients with atherosclerotic heart disease severe chest pain mimicing acute myocardial infarction and with impressive ST-T changes may occur. A few patients may develop congestive heart failure, shock and/or vascular collapse (17). When Wolff (25) reviewed 253 cases of paroxysmal tachycardia, 40 patients (16%) had classic anginal pain; the majority of these patients were elderly or had known atherosclerotic heart disease or aortic valvular disease. An additional 16% had chest pain not considered to be anginal in character. All patients had heart rates greater than 150 when chest pain developed. Interestingly, only 10 of 37 patients with recent myocardial infarction developed chest pain. Vascular collapse also occurred in 16%; these patients always had rates of 200/minute or greater. When rates were greater than 200, vascular collapse occurred in 50% of normal patients and those with old coronary artery disease, 75% of those with recent myocardial infarctions, and 100% of patients with mitral stenosis (25).

Auscultation during paroxysmal supraventricular tachycardia usually reveals constant heart sounds except for respiratory variation. Gallops may be present. Murmurs frequently diminish or disappear (17).

Polyuria with a rapid diuresis of up to 3 liters in the first 30-90 minutes after the onset of the tachycardia has been reported (26-29). The water diuresis is believed to be secondary to atrial distention from the elevated atrial pressures resulting in inhibition of antidiuretic hormone secretion (27). The mechanism of the salt diuresis is unknown (29).

Deaths have occurred in patients with concomitant heart disease, in WPW alone and in WPW after administration of digitalis (25).

CLASSIFICATION

Recent use of electrophysiologic techniques to study paroxysmal supraventricular tachycardia have greatly aided in the classification of mechanisms. The study of mechanisms has become important to understand newer modes of therapy including pharmacologic, pacemaker, and surgical therapy. Whereas paroxysmal supraventricular tachycardia was defined either as paroxysmal atrial tachycardia (PAT) or nodal tachycardia, it now includes arrhythmias originating in the sinus node, the atrium, the AV junction, the AV node, and the bundle of His.

Paroxysmal supraventricular tachycardia has been classified by Coumel and Barold (30) as follows:

CLASSIFICATION OF SUPRAVENTRICULAR TACHYCARDIA

1. *AV junctional tachycardia*
   - A. *Reciprocating tachycardia within the AV node*
     - 1) *AV nodal re-entry: Paroxysmal and permanent forms of AV junctional tachycardias*
     - 2) *Reciprocating tachycardias associated with the pre-excitation syndrome*
   - B. *Re-entry in the distal portion of the AV junction (His bundle)*
   - C. *Unifocal*
2. *Atrial Tachycardia*
   - A. *Sinus node re-entry*
   - B. *Atrial re-entry*
   - C. *Unifocal*
3. *Bypass pathway re-entry*

DISTRIBUTION OF SUPRAVENTRICULAR TACHYCARDIAS BY RE-ENTRY SITE

	Sinus node and atrium	*AV node*	*Bundle of Kent*	*Indeterminant*
Wellens		*88%*	*12%*	
Akhtar		*65%*	*20%*	*15%*
Narula	*12%*	*56%*	*28%*	*4%*

RE-ENTRY MECHANISMS

The creation of a circus movement on re-entry requires 3 conditions: 1) a closed circuit, 2) unidirectional block somewhere in the circuit, and 3) slow conduction.

Extensive experimental work in animals has shown the presence of a closed circuit inside the AV node (31-33). These have been arbitrarily named the alpha and beta pathways (Figure 3). The alpha pathway has a short refractory time and a long conduction time. The beta pathway has a long refractory period and a short conduction time.

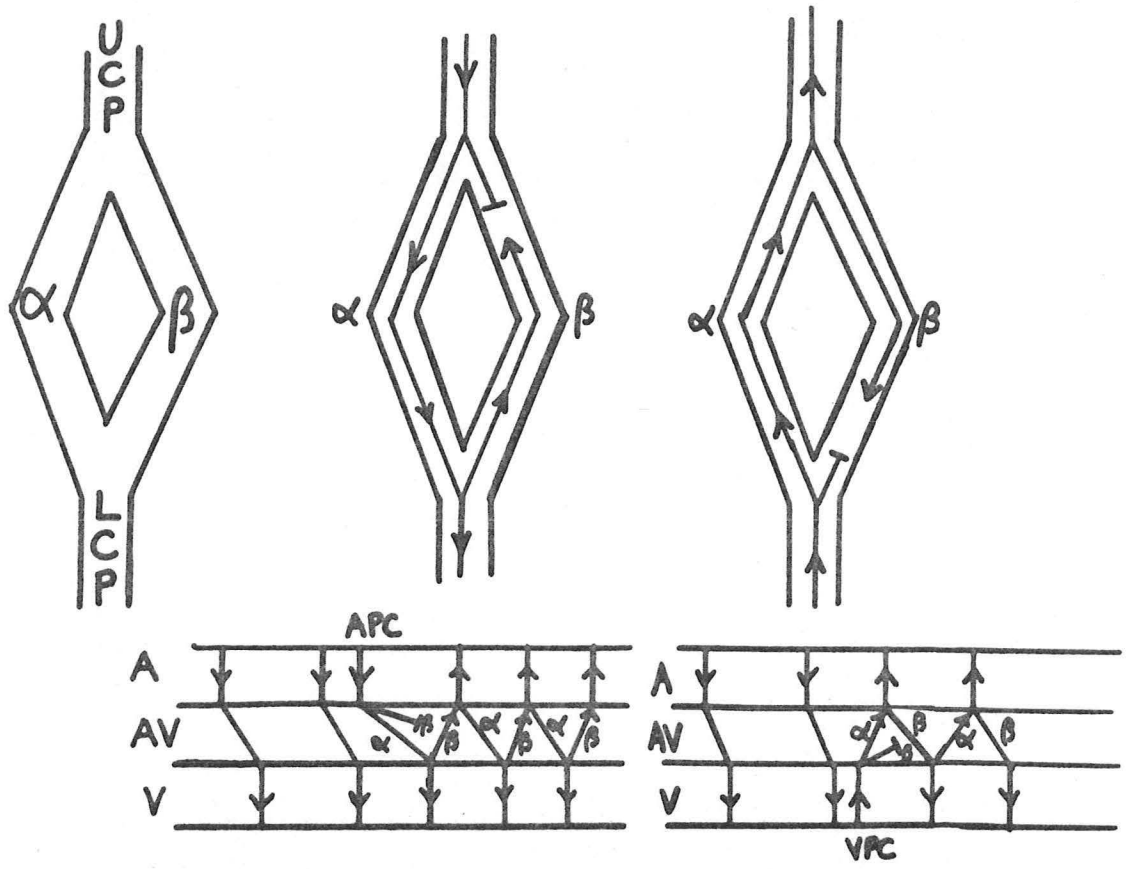

FIGURE 3. Diagrammatic representation of longitudinal dissociation of the AV node. UCP = upper common pathway. LCP = lower common pathway (30).

There is good experimental evidence that dual A-V nodal pathways exist in man (34).

Unidirectional block is usually caused by the difference in refractory periods of the two pathways. The majority of the time neither pathway is refractory and when an impulse travels from the atrium it is conducted down both pathways, however the ventricle is mostly activated by the beta pathway as it has a shorter conduction time. A premature atrial contraction may find the alpha pathway able to conduct while the beta pathway is still refractory due to a longer refractory period; hence unidirectional block may occur as diagrammed in the middle panel of Figure 3. Likewise, a premature ventricular contraction with retrograde conduction may find the alpha pathway able to conduct but the beta pathway refractory as is shown in the right panel of Figure 3.

The third and most important component is slow conduction. Usually when slow conduction is present there is unidirectional block because the slow conduction is down the alpha pathway prolonging A-V conduction hence the beta pathway is blocked. There is a critical window in which slow conduction must fall to cause re-entry. If the slow conduction is not slow enough then return up the beta pathway may arrive back at the alpha pathway while the alpha pathway is still refractory. Most commonly the slow conduction occurs in the alpha pathway and PR prolongation occurs. Occasionally the slow conduction occurs down the beta pathway and when this occurs there is a long R-P interval (35).

The WPW syndrome which will be discussed more in depth later provides proof that the re-entry pathway can be entered from either dirction (36,37). Similarly, a ventricular impulse may have retrograde conduction into the alpha pathway and have retrograde block in the beta pathway which is then entered at its junction with the upper common pathway with antegrade conduction back down the beta pathway which is demonstrated in the right-hand panel of Figure 3 (38). The A-V node has both the anatomic and electrophysiologic properties necessary for re-entry to occur (39-41). All three conditions - a closed circuit, unidirectional block, and slow conduction must be present for A-V nodal re-entry to occur. WPW presents very good evidence that all 3 properties are present when tachycardia is produced. In WPW the delta wave proves the existance of an accessary pathway and therefore a closed circuit. Disappearance of the delta wave during the tachycardia shows evidence that unidirectional block occurs in the bundle of Kent and prolongation of the PR interval in the first beat shows evidence that slow conduction occurs simultaneously with unidirectional block. With A-V nodal re-entry it is not possible to demonstrate a closed circuit and unidirectional block directly though there is good electrophysiologic data for them in humans and direct evidence in animals. Figure 4, panel C reveals the one factor with A-V nodal re-entry that can usually be found and that is slow conduction as exemplified by the long PR that occurs with the premature atrial contraction which sets up the properties needed for re-entry. Hence, there is good evidence that in A-V junctional re-entry all 3 mechanisms-closed circuit, unidirectional block, and slow conduction exist.

A-V Junctional Reciprocating Tachycardia

Re-entry in the A-V junction usually occurs when conduction is prolonged through the A-V node thereby establishing the criteria needed for re-entry. Generally when re-entry occurs the slow conduction is sufficient.

CONDITIONS WHICH TRIGGER RECIPROCAL TACHYCARDIA

1. *Premature atrial contraction with 1^{o} A-V block*
2. *Normal sinus rhythm with 1^{o} A-V block*
3. *Premature ventricular contraction with delayed retrograde conduction*
4. *Premature ventricular contraction causing 1^{o} A-V block of the sinus beat following the PVC*
5. *Wenckebach form of 2^{o} A-V block*
6. *3^{o} A-V block in the upper or lower common pathway not involving the alpha and beta pathways*

Figure 4 reveals how a premature atrial contraction with 1° A-V block can trigger re-entry.

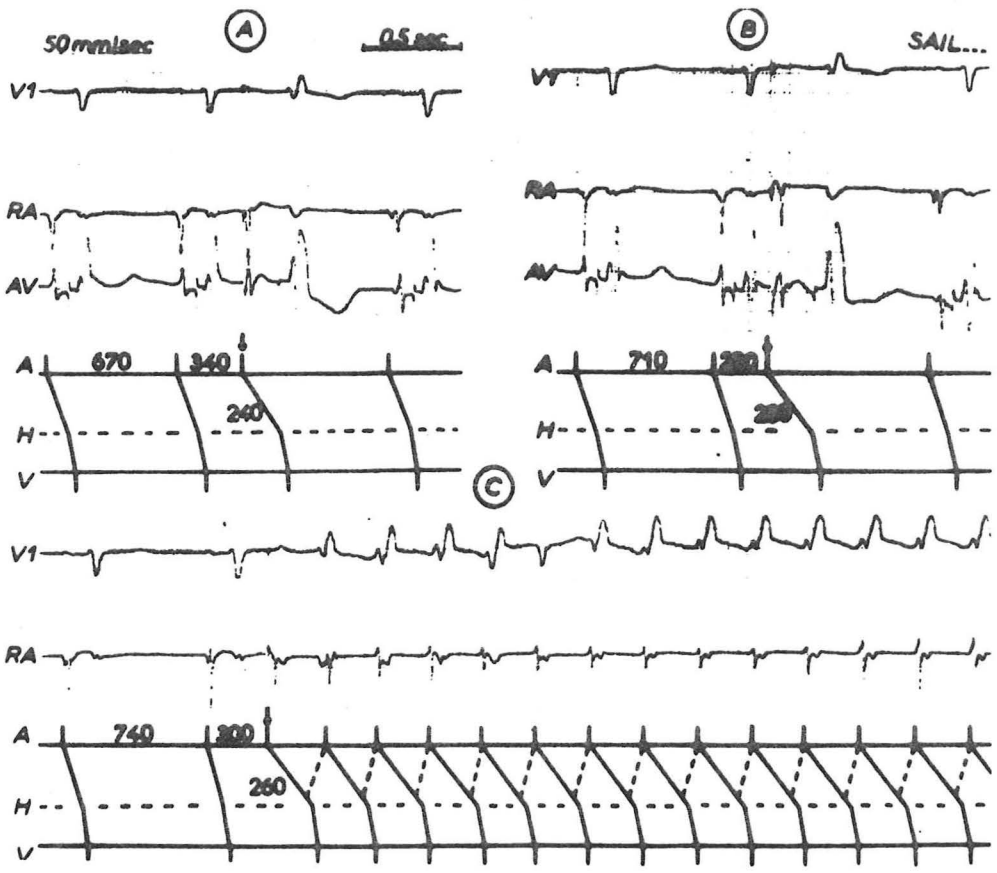

FIGURE 4. Initiation of paroxysmal A-V junctional reciprocating tachycardia by atrial stimulation. A) An atrial premature beat 340 msec after the sinus P wave, is conducted with functional RBBB and a P-R interval of 240 msec. B) Reduction of the coupling interval to 280 msec causes a longer P-R interval (280 msec) but the tachycardia does not start (Possibly due to the atrial rates not allowing the proper setting to occur for re-entry such as bidirectional block 2° to the rate). C) Tachycardia starts after a lesser prolongation of the P-R interval (260 msec) as compared to B when the basic sinus P-P interval has lengthened to 740 msec. The fifth beat of the tachycardia appears to be a ventricular fusion beat. (RA = right atrial electrogram)(30).

In the next figure an example of premature ventricular contractions initiating PSVT is shown.

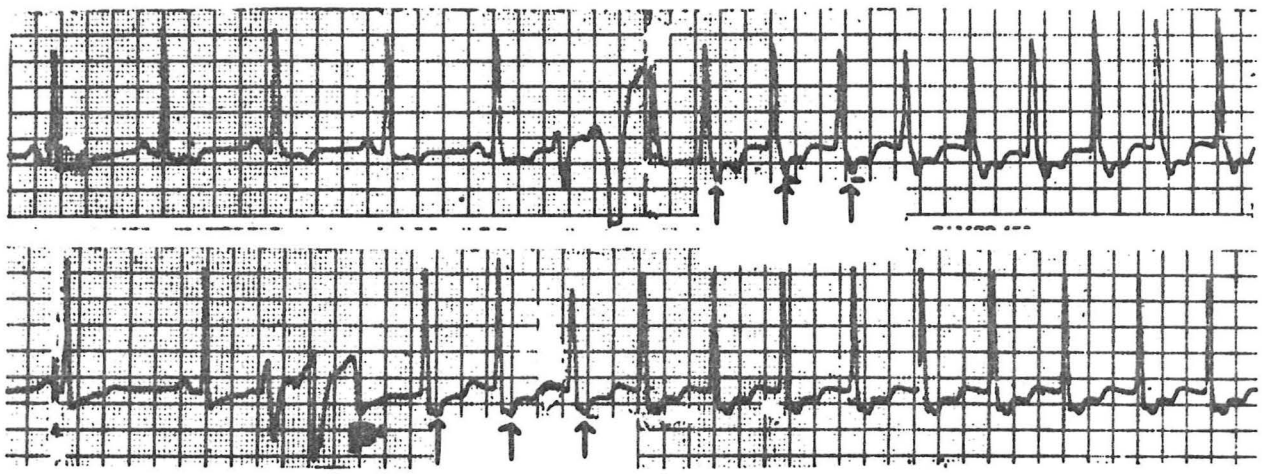

FIGURE 5. Initiation of A-V junctional reciprocating tachycardia by retrograde ventriculo-atrial (VA) conduction. The tracings were those of a patient with a long history of paroxysmal supraventricular tachycardia and were recorded during myocardial ischemia when frequent ventricular extrasystoles were present. Repeated episodes of A-V junctional tachycardia were consistently triggered by twin ventricular premature beats but never by single ones. The bottom strip shows how retrograde VA conduction coupled with delayed anterograde conduction initiates paroxysmal A-V junctional reciprocating tachycardia. Retrograde activation presumably engaged the alpha pathway and returned along the beta pathway to initiate re-entry (P^1 = retrograde P wave)(30).

The next set of figures reveal how re-entry may occur from one direction but not the other.

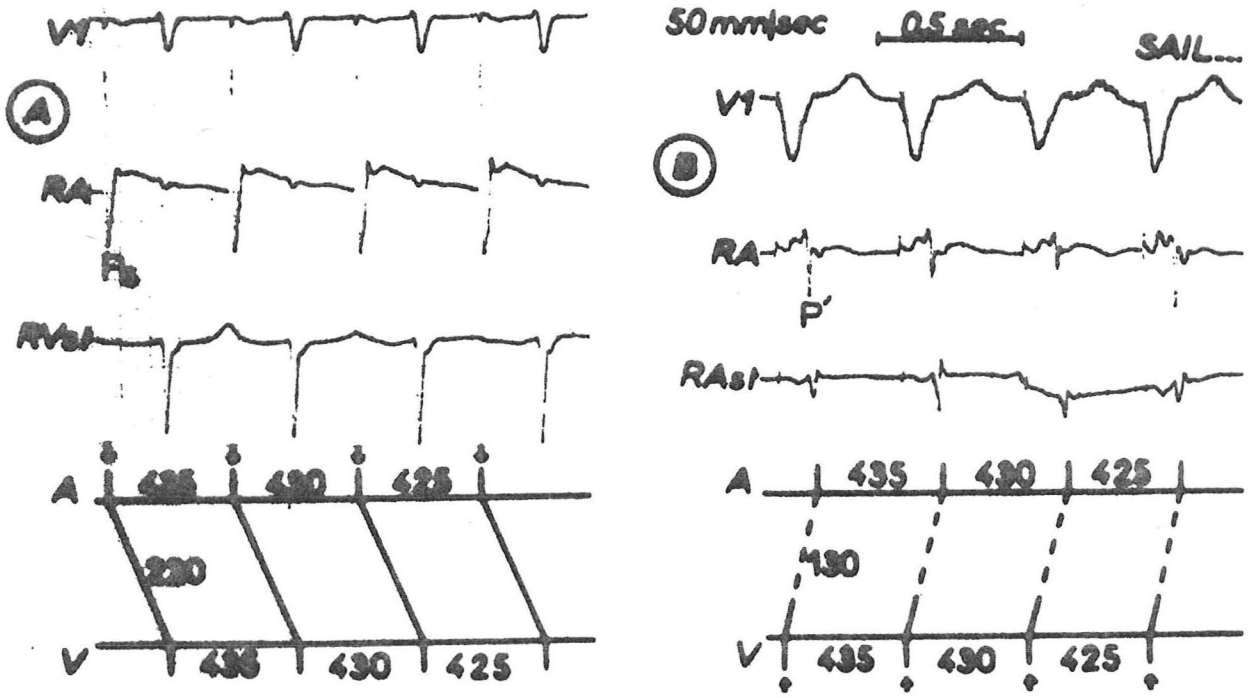

FIGURE 6A and B. Evaluation of anterograde and retrograde conduction and initiation of the tachycardia by ventricular stimulation. A) anterograde conduction time was measured as the interval between the pacemaker stimulus in the atrium and the intrinsic deflection of the bipolar electrogram in the right ventricle (RV_{st}) where ventricular stimulation was applied to study retrograde conduction. B) Retrograde conduction time was measured as the interval between the pacemaker stimulus in the right ventricle and the intrinsic deflection of the bipolar electrogram in the right atrium (RA_{st}) where atrial stimulation was previously done. According to these measurements, anterograde and retrograde conduction times differ considerably at the same rates (220 vs 130 msec) suggesting the presence of an accessory pathway responsible for rapid retrograde conduction.

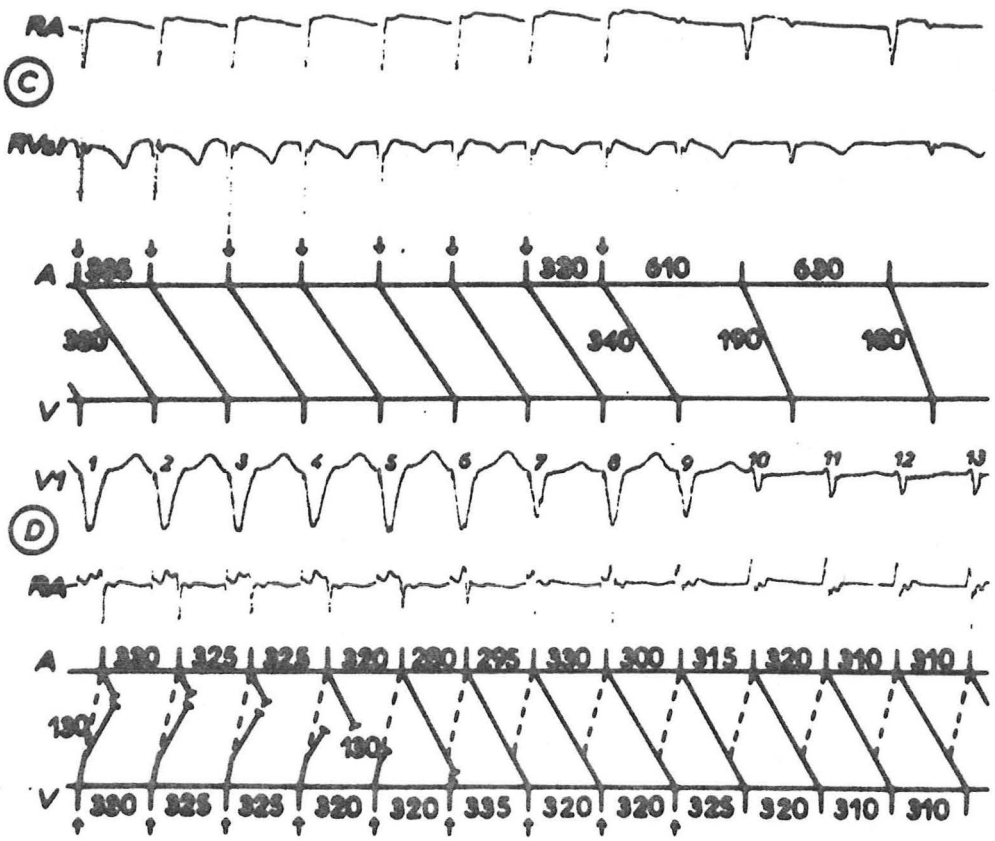

FIGURE 6 C and D. Evaluation of anterograde and retrograde conduction and initiation of the tachycardia by ventricular stimulation. C) More rapid atrial stimulation caused progressive prolongation of A-V conduction.. An atrial cycle of 320 msec causes an anterograde conduction of 340 msec. D) The frequency of ventricular stimulation was increased until a cycle length of 320 msec was reached. Retrograde conduction still remains unchanged at 130 msec, but reciprocating tachycardia is initiated. This suggests unidirectional block related to rate (in other words lesser degrees of retrograde concealed conduction allowed re-entry without apparent prolongation of conduction time)(30).

Wenckebach phenomena can also trigger an A-V junctional reciprocating tachycardia.

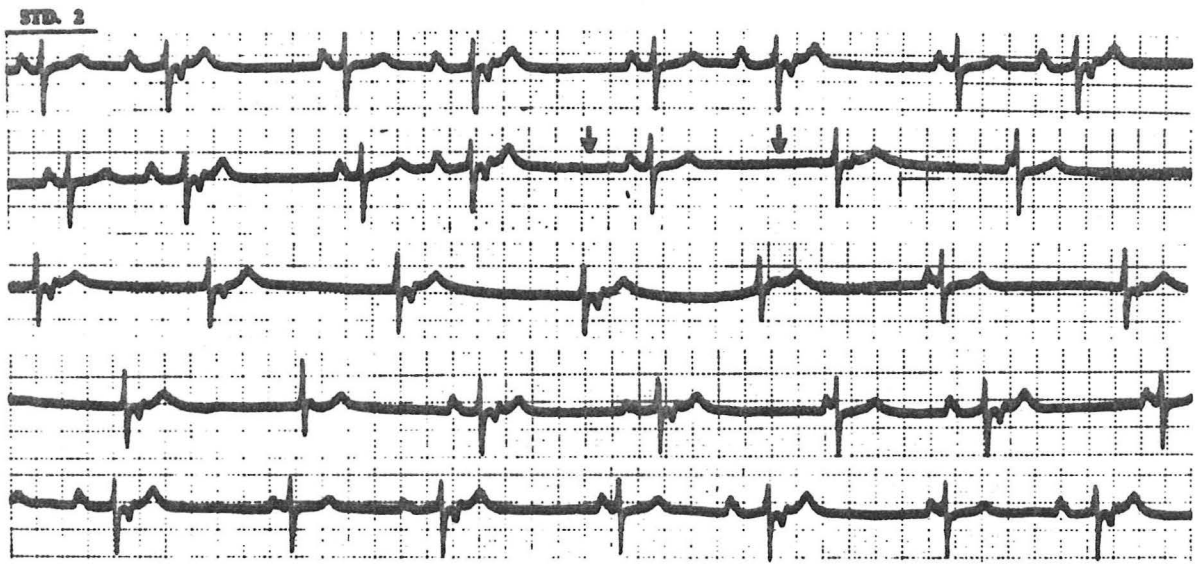

FIGURE 7A. Initiation of atrial echo by the Wenckebach mechanism. Note that the first beat of the cycle has a normal PR interval. The second beat of the cycle has a prolonged PR interval and an atrial echo (retrograde conduction) occurs. Because of the atrial echo the Wenkebach cycle is broken and does not progress to a blocked beat (42).

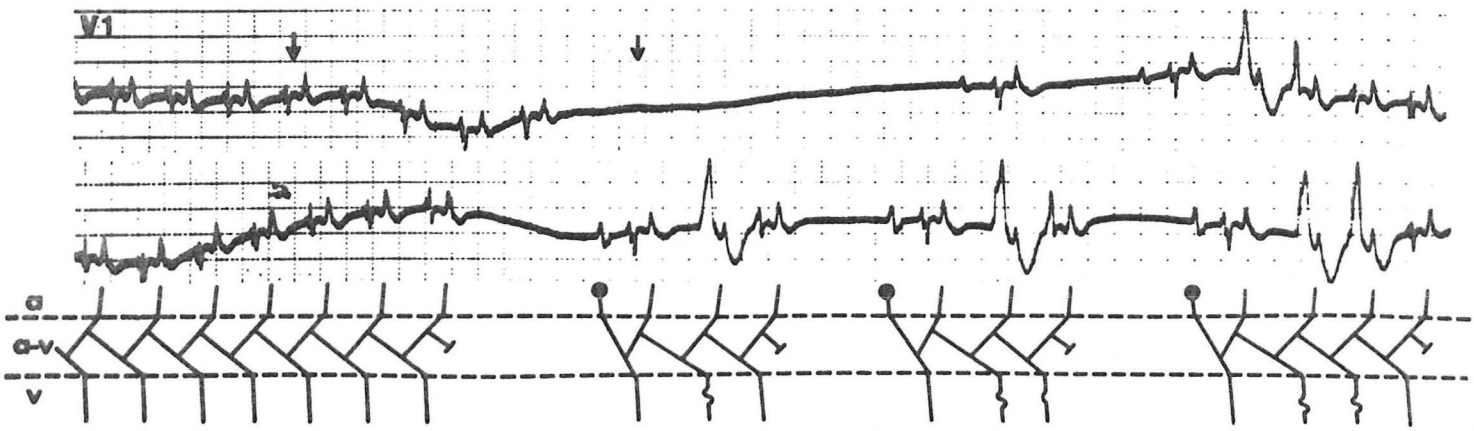

FIGURE 7B. Paroxysmal supraventricular tachycardia precipitated by Wenckebach mechanism. This is a later strip of the same patient as shown in Figure 7A and shows an A-V junctional reciprocating tachycardia initiated by the Wenckebach mechanism (42).

Figure 8 reveals a patient with complete heart block who has reciprocal beats.

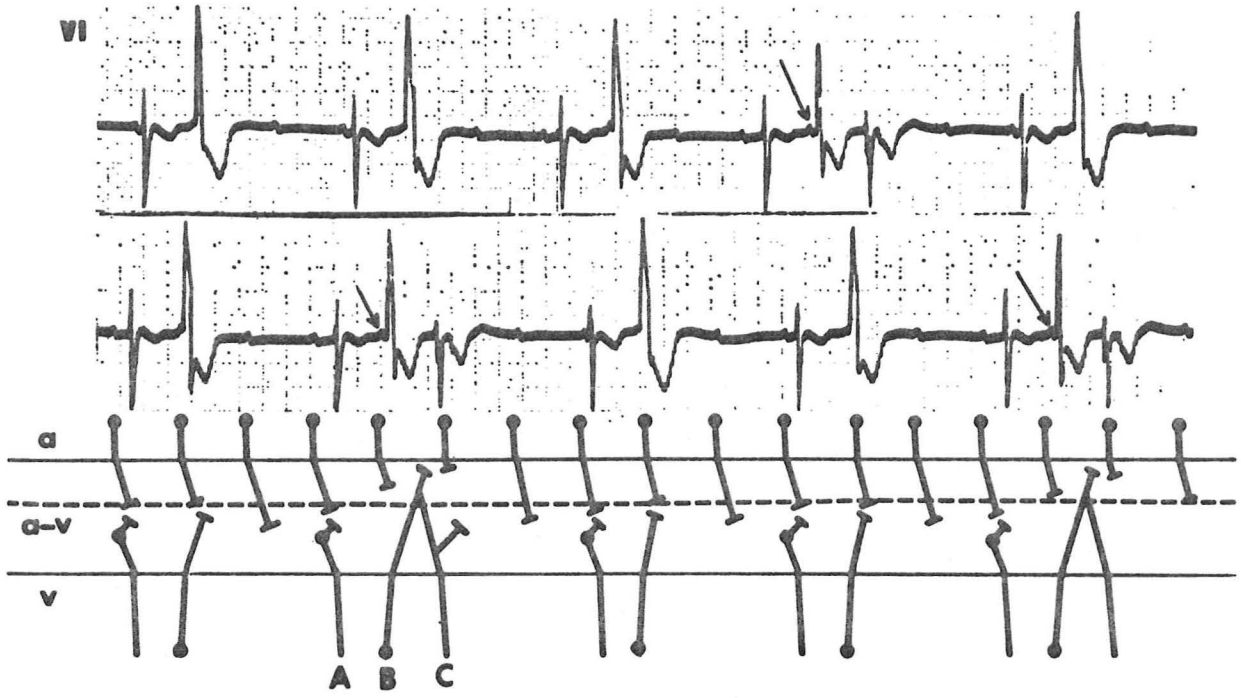

FIGURE 8. Complete A-V block with A-V junctional reciprocal beats. Complete A-V block is present presumably high in the upper common pathway. Premature ventricular contractions cause a ventricular echo (anterograde conduction) from A-V junctional reciprocation (42).

As the upper and lower common pathways are not essential to have re-entry once initiated, examples of anterograde and retrograde block can be seen. More commonly there is retrograde block with absence of P waves intermittently or completely. Anterograde block can also occur but much more rarely as shown in Figure 9.

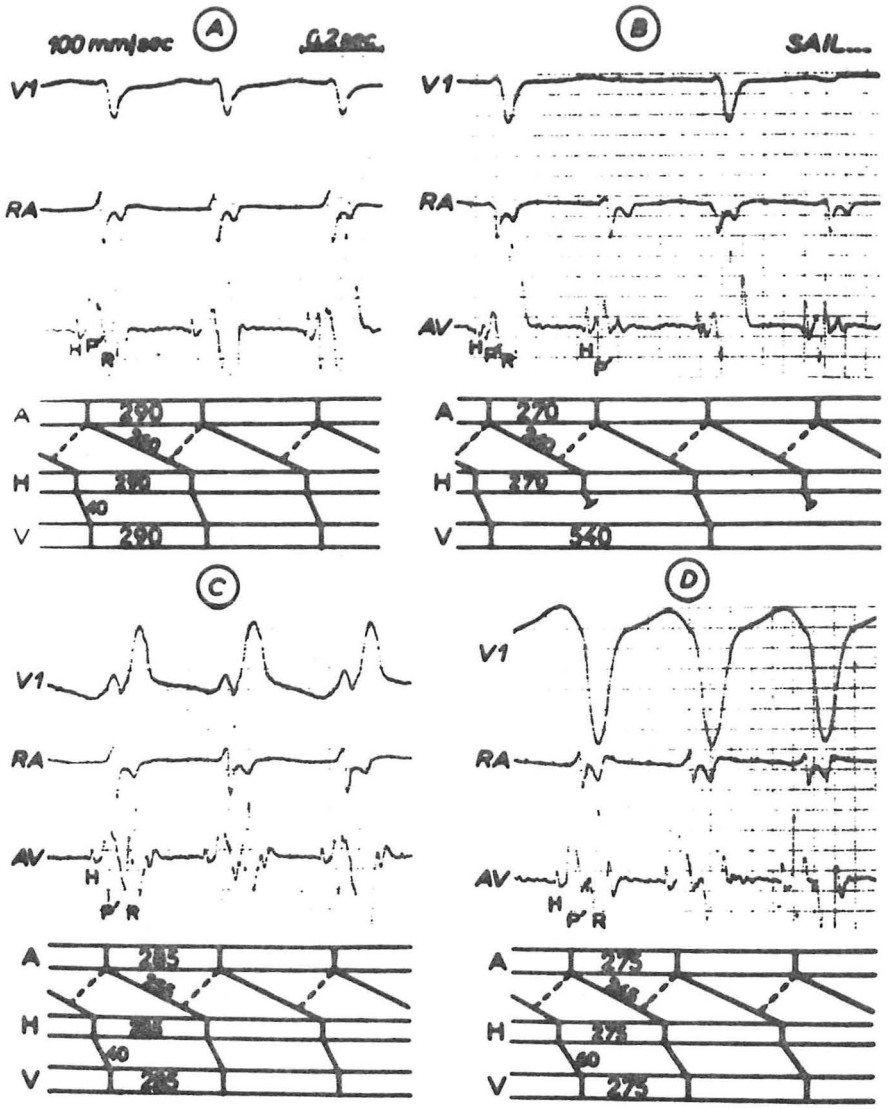

FIGURE 9. His bundle and right atrial recordings during A-V junctional reciprocal tachycardia. A) Tachycardia with 1:1 conduction and narrow QRS complexes (H-V = 40 msec). B) Tachycardia with 2:1 block below the site of recording of the His bundle potential. This proves the existence of a lower common pathway. C) Tachycardia with 1:1 conduction and functional RBBB. The H-V interval remains normal. D) Tachycardia with 1:1 conduction and functional LBBB; the H-V interval lengthens to 50 msec. Comparison of the P-H intervals and cycle lengths of the tachycardia (P-P interval) shows they are linked in a parallel fashion. This relationship excludes with great certainty the presence of an ectopic focus, atrial or junctional (30).

Stimulation of the atrium or ventricle by use of pacemakers has become a useful tool in the diagnosis of mechanism of a paroxysmal supraventricular tachycardia (43-47). Stimulation of the atrium or ventricle may fail to capture the chamber paced if too early. A little later the stimulation may capture the paced chamber and then block in the A-V node and may disrupt the re-entry. Stimulation a little later may capture both the paced chamber and the opposite chamber thus resetting the re-entry mechanism. Pacing too late will only caputre the paced chamber and block the next impulse in that chamber causing a compensatory pause. The mechanisms are shown diagrammatically in Figure 10.

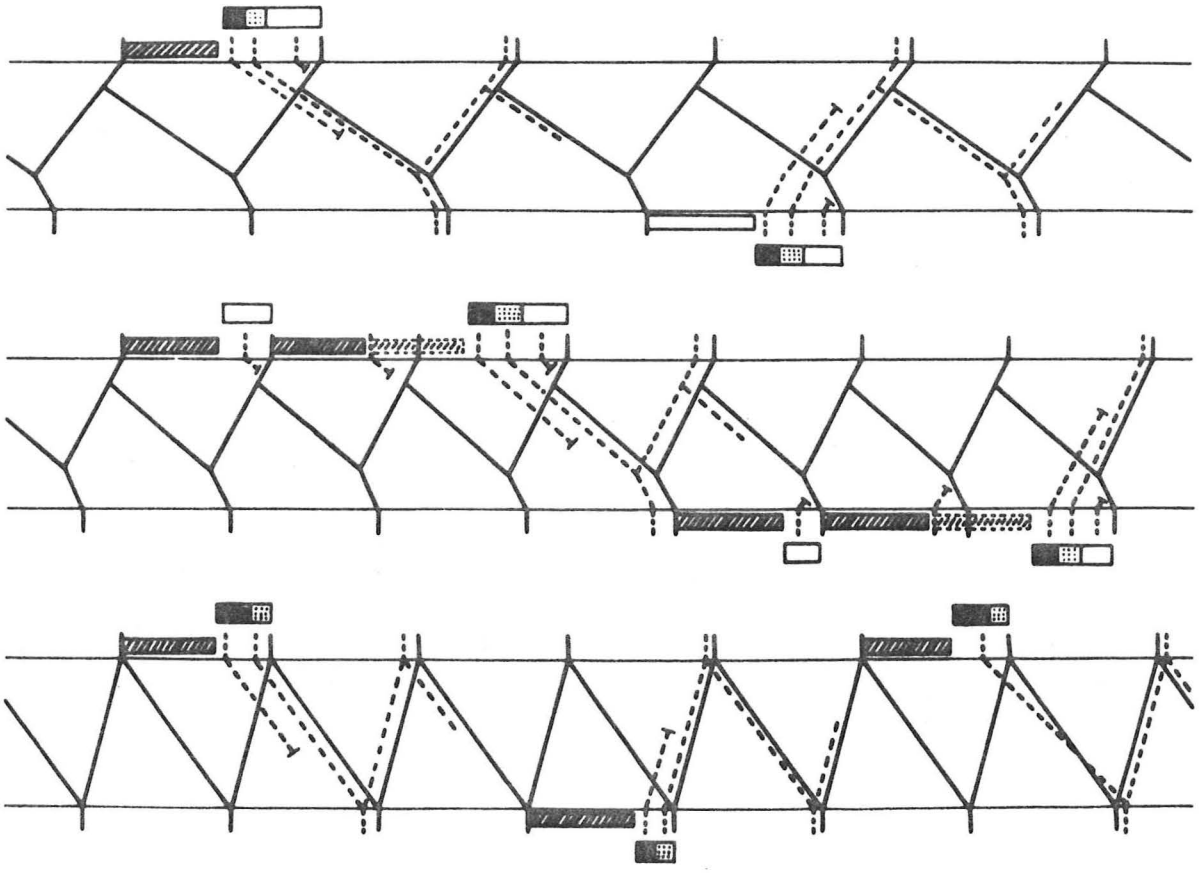

FIGURE 10. Diagrammatic representation of the possible effects of electrical stimulation during reciprocal tachycardia. Upper panel: intranodal reciprocating tachycardia with a relatively slow rate (less than 150 to 160/min). Three "zones" can be delineated as atrial or ventricular stimulation is given progressively earlier; failure to capture the opposite chamber (blank area), capture of the opposite chamber (dotted area), and blocked stimulation terminating the tachycardia (black area). Middle panel: the rate of the intranodal reciprocating tachycardia is faster than in the upper panel. Single atrial or ventricular stimulation can never be given early enough to capture

or terminate the tachycardia. Twice stimulation either from the atrium or the ventricle allows the first stimulus to change the temporal relationship of the refractory period (of the chamber being stimulated) with respect to the tachycardia. This "peeling" allows the second stimulus access to the re-entry pathway with the capture or termination of the tachycardia. Lower panel: If the circus movement involves the atrium and ventricle, as in WPW syndrome, the initial and common pathways disappear. This greatly facilitates capture even in a fast tachycardia. A zone of "capture with delay" (horizontally hatched area) is often evident (30).

Hence a single stimulus may capture or terminate an A-V junctional tachycardia only if the rate is slow enough. As the rate is usually faster than 150 beats/minute, twin stimuli are generally needed to capture or terminate a tachycardia because the stimulus must reach one limb of the pathway to influence the pathway. As WPW has one limb of the pathway in the atrium and the other in the ventricle then a single stimulus can always reach a limb of the pathway. Therefore, twin stimuli are usually required to terminate an A-V junctional reciprocal tachycardia. Figure 11 shows a single stimulus capturing and twin stimuli terminating a reciprocal tachycardia.

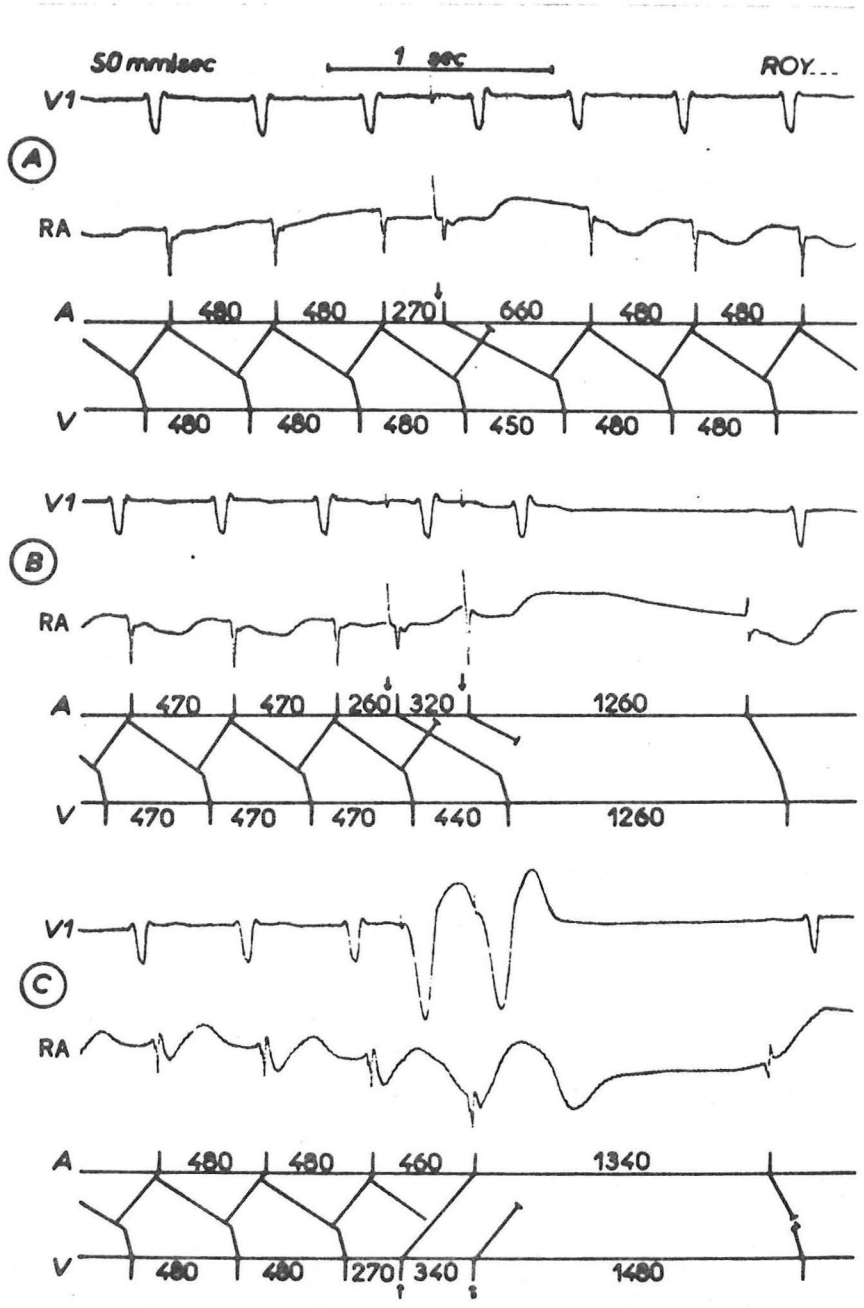

FIGURE 11. Termination of relatively slow A-V junctional reciprocating tachycardia by twin stimuli. The tachycardia could not be terminated by a single atrial or ventricular stimulus. A) A single atrial stimulus causes a capture beat. B) Termination by twin paced atrial beats. C) Termination by twin paced ventricular beats (30).

Permanent or Chronic Form of Reciprocating A-V Junctional Tachycardia

Though several workers had postulated a re-entry mechanism in the permanent form of A-V junctional tachycardia (35,48-52), it was not confirmed until 1967 when Coumel et al (53) proved the mechanism with programmed stimulation techniques. The permanent form of tachycardia is relatively rare. Patients generally are in the tachycardia the majority of the time with frequently only a few normal beats between bursts of tachycardia. The tachycardia generally has rates between 120 and 250 beats/min. Several characteristics have been described by Coumel and Barold (30):

> "In the paroxysmal form of A-V junctional reciprocating tachycardia, the morphology and timing of the retrograde P waves vary considerably but these two features are very constant and useful diagnostically in the permanent form of reciprocating tachycardia which has the following features: (a) the retrograde P waves have the classic morphology of retrograde activation by being clearly negative in leads 2,3, aVF; (b) the retrograde P waves are closer to the next QRS complex than the previous ones and the apparent P-R interval is not necessarily short, its value depends upon the heart rate which is rarely less than 120/min and occasionally up to 250/min; (c) isolated atrial echo beats representing the abortive onset of tachycardia constitute a particularly characteristic feature of this tachycardia and occur more frequently in adults than in children; (d) the initiating mechanism of the tachycardia is its most important characteristic and explains its relapsing nature."

The conduction time is already sufficiently slow for re-entry to occur in the form of tachycardia. The only missing ingredient is unidirectional block which can occur by modest changes in the P-P interval. Hence, as the sinus rhythm is always changing it frequently is greater than the threshold rate needed to cause unidirectional block. The threshold rate may also vary greatly from minute to minute with the autonomic tone. Figure 12 reveals such a rhythm.

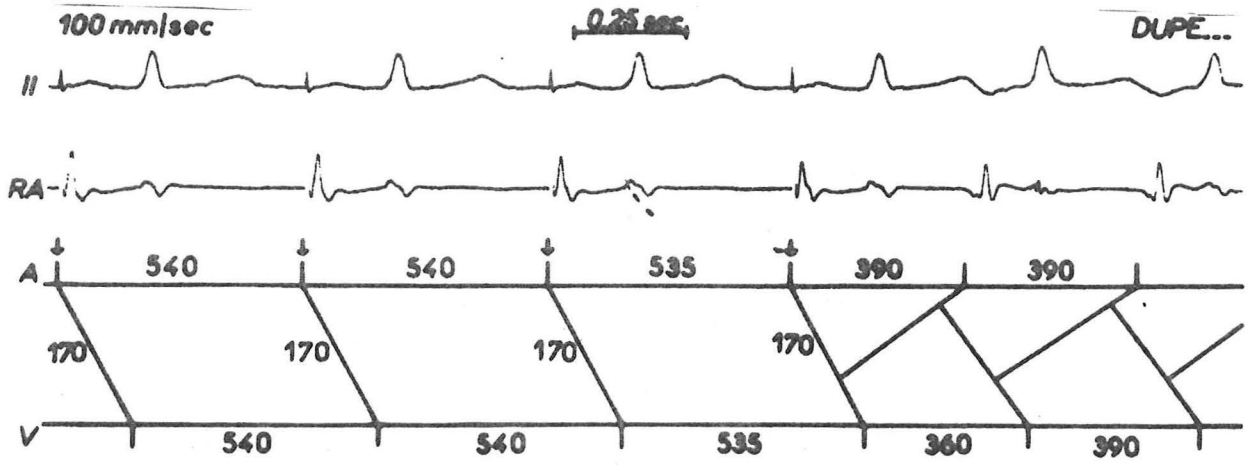

FIGURE 12. Initiation of permanent form of A-V junctional reciprocating tachycardia by atrial pacing. The rate of atrial stimulation was progressively increased to measure the critical P-P interval (535 msec) required to initiate the tachycardia. Note the tachycardia starts without any prolongation of the PR interval (30).

Figure 13 reveals several facets of this type of tachycardia.

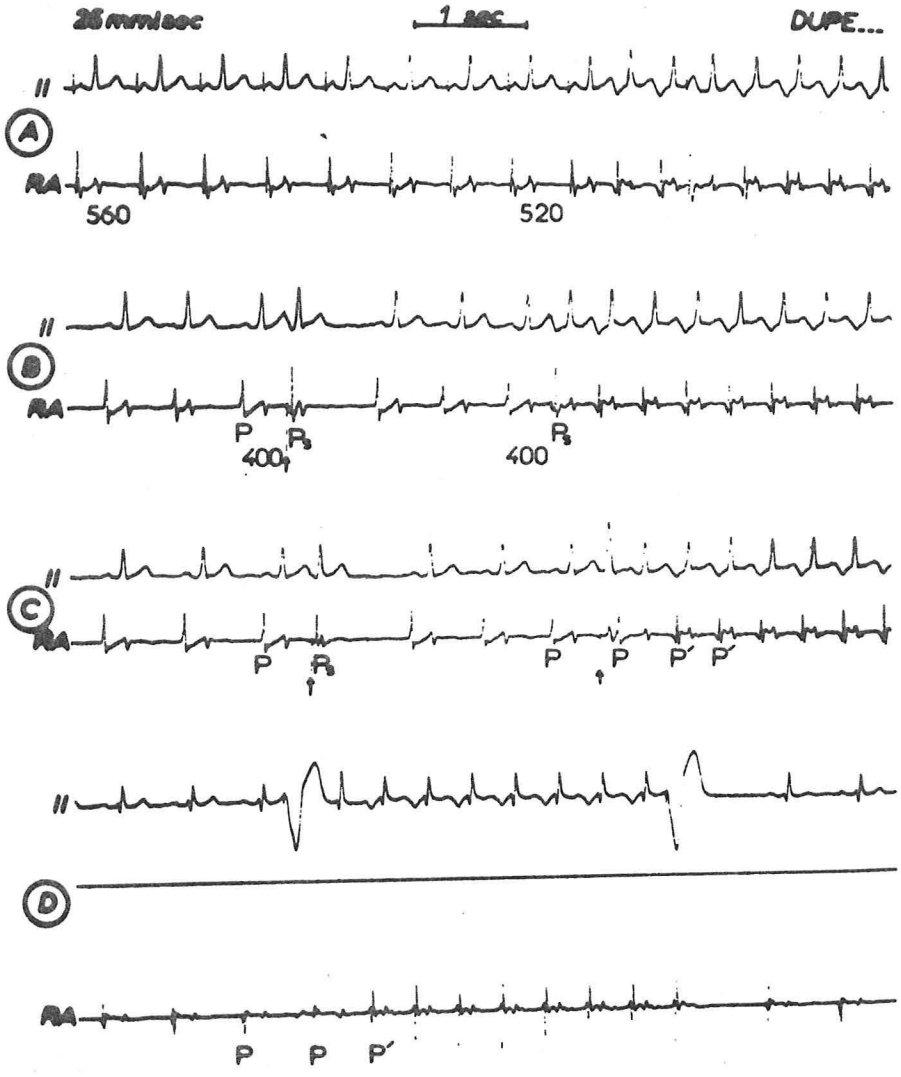

FIGURE 13. Stimulation studies in permanent A-V junctional reciprocating tachycardia (same patient as Figure 12). A) Initiation of tachycardia by progressive acceleration of atrial pacing rate. B) The first stimulus (P_S) activates the atrium and His bundle simultaneously and does not initiate tachycardia. The second stimulus (P_S) activates the atrium only and the tachycardia starts. C) The first stimulus activates the atrium and the His bundle simultaneously as in panel B. The second stimulus activates the His bundle only and again triggers the tachycardia. D) The first paced ventricular beat initiates the tachycardia and the second paced ventricular beat terminates it (30).

This arrhythmia may be further complicated by co-existance of the other type of A-V junctional reciprocating tachycardia as shown in the next figure.

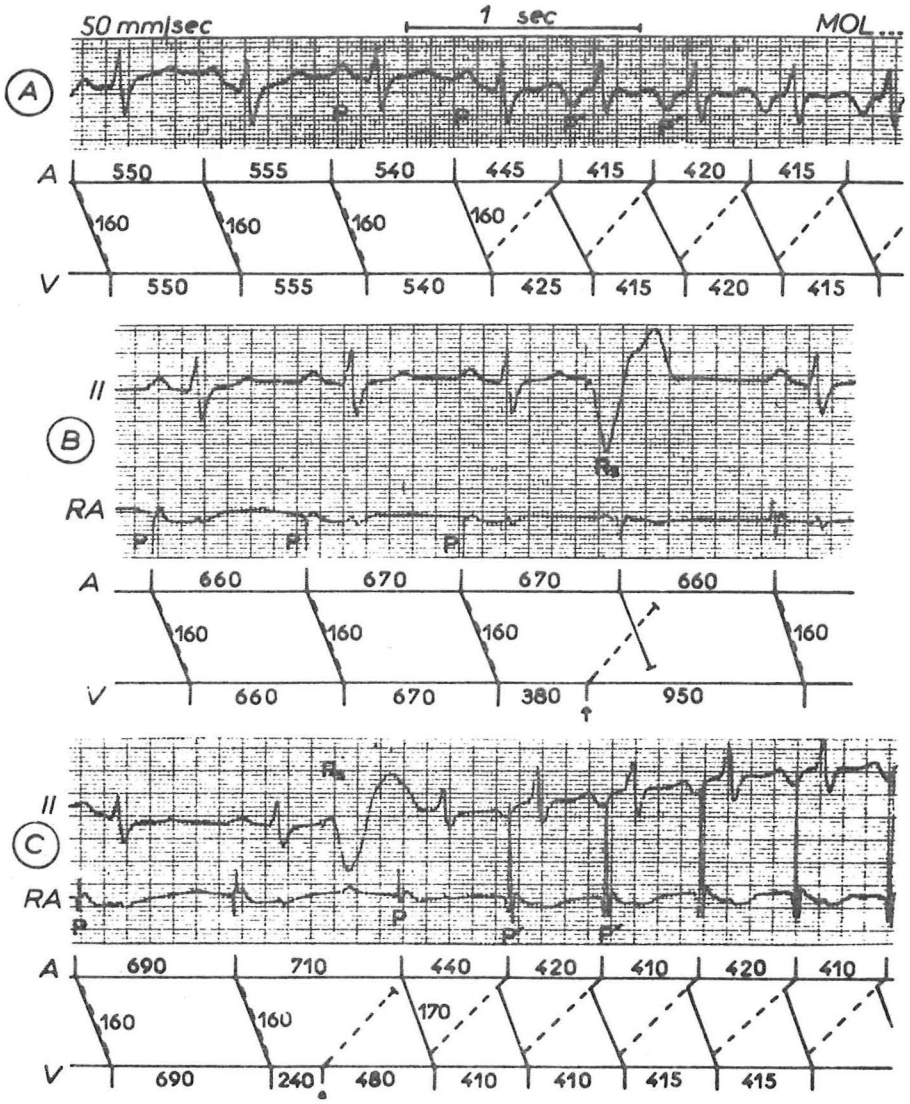

FIGURE 14. Permanent form of A-V junctional tachycardia. A) Tachycardia starts when the P-P interval shortens to a critical value while the P-R interval remains constant. B) A late paced ventricular beat does not initiate tachycardia. C) An earlier paced ventricular beat starts the tachycardia by blocking the beta pathway and prolonging the P-R interval thus allowing the tachycardia to be initiated without reaching the critical P-P interval (30).

Re-entry In The Bundle of His

Recent studies have strongly suggested that re-entry can occur in the bundle of His as well as in the A-V node (54-56). However, these have not been definitely proven and if they exist are apparently very rare.

Nonparoxysmal Junctional Tachycardia (NPJT)

Nonparoxysmal junctional tachycardia is felt to be a result of an accelerated pacemaker and not re-entry (57,58). The rate is between 70 and 130 and rarely exceeds 150/min. It usually begins gradually rather than having a sudden onset. Most of the time intermittant A-V dissociation is seen as the rate is slightly faster than the sinus or atrial rate and retrograde block is seen. It is usually associated with digitalis intoxication, actue myocardial infarction, myocarditis or rarely with no other cause.

Sinus Node Re-entry

Experimental studies in isolated tissue, intact animals, and man have suggested that re-entry can occur in the sinus node (59-64). The necessary prerequisites for sinus node re-entry have been described: 1) slow propagation within the sinus node (65-67) and 2) unidirectional block because the effective refractory period of the sinus node may exceed that of the surrounding tissue (59). In the following two diagrams sinus node re-entry is diagrammed.

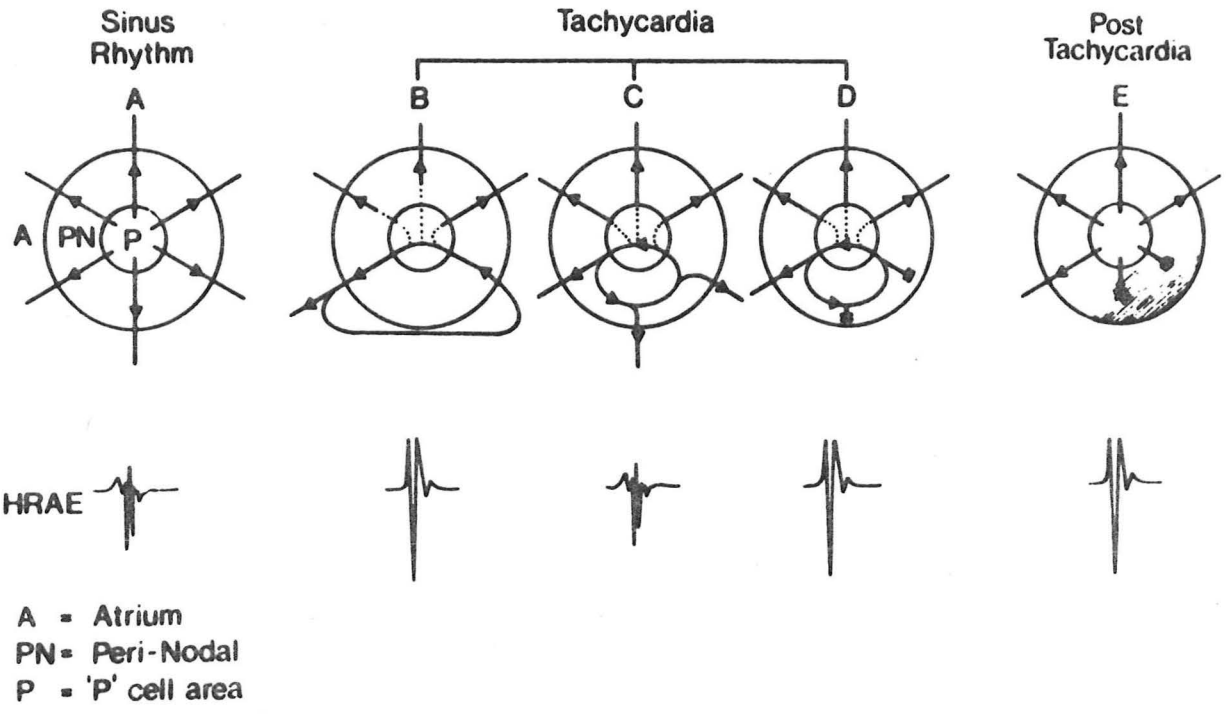

FIGURE 15. Diagrammatic representation of the different sequences of activation within and around the SA node; and the resultant configurations of the high right atrial electrograms (HRAE) during: A) sinus rhythm (radial excitation originating from the center of the SA node); B to D) sinus node re-entry tachycardias (PRST); and E) the first few escape beats of sinus rhythm immediately after the termination of PRST. The re-entry tachycardia circuit may include the atrium, the perinodal cells and the central pacemaker region of the sinus node (B) in which case the local sequence of atrial activation during PRST may differ from that in sinus rhythm (A) resulting in an altered HRAE and possibly also an altered P wave on the surface ECG, since some of the exit pathways used in sinus rhythm are now used retrogradely as re-entrant pathways. Alternatively, the circuit may be entirely within the SA node when the sequence of the atrial activation during PRST (C) may be identical to that in sinus rhythm unless rate dependent aberration is induced in some of the exit pathways (D). 'Polarization' of the sinus node exit pathways used retrogradely in PRST may occur particularly following prolonged attacks, leaving these pathways relatively refractory for a varying period which may extend over one to four cycles of the subsequent sinus rhythm. In this case the HRAE in the few cycles of sinus rhythm will retain a configuration similar to that seen in PRST, (E) (68).

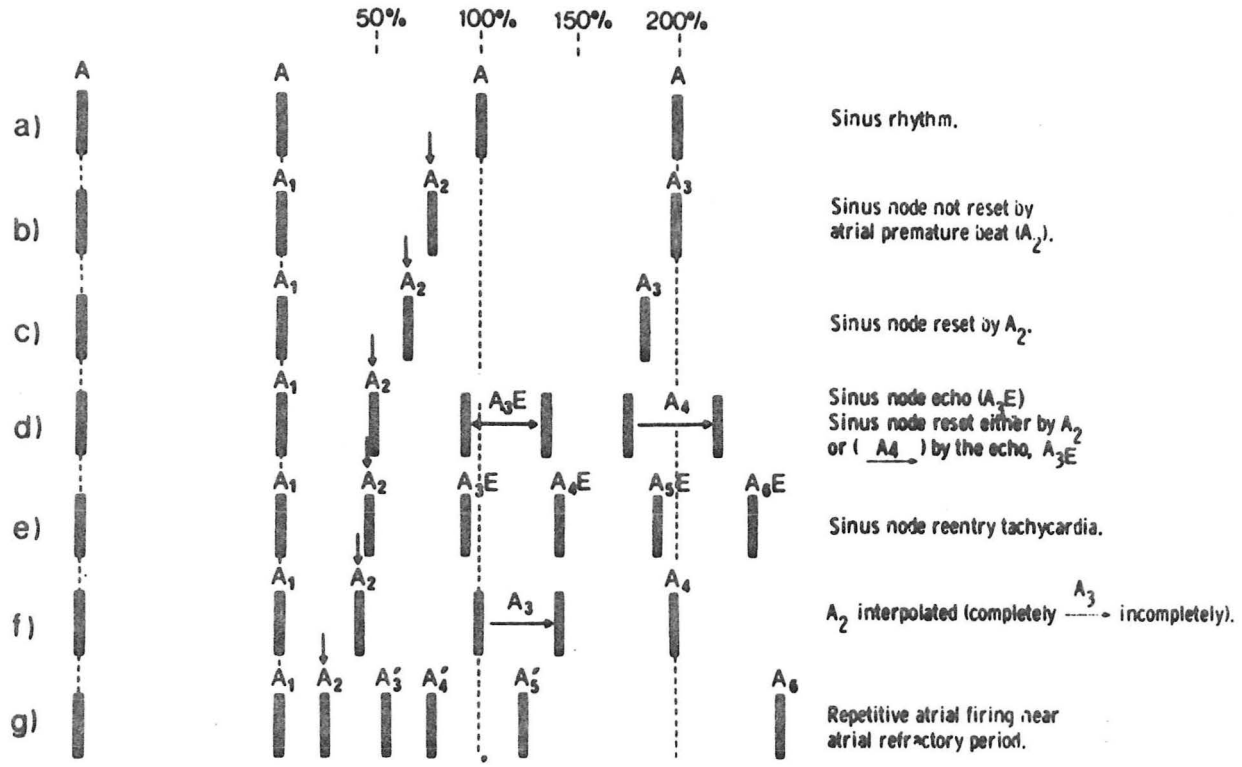

FIGURE 16. Diagram of the responses which may be obtained in the course of a single atrial extrastimulus test during sinus rhythm. Both A and A_1 represent high right atrial electrograms of the basic sinus rhythm. A_2 is the induced atrial extrastimulus while A_3 to A_6 are return beats following the intraduction of A_2. The letter "E" after a response indicates sinus node re-entry (echoes or tachycardia). Abnormal sequences of intra-atrial depolarization are indicated as in A''_3 - A''_5 (68).

An electrocardiogram from a patient with sinus node re-entry is shown in Figure 17.

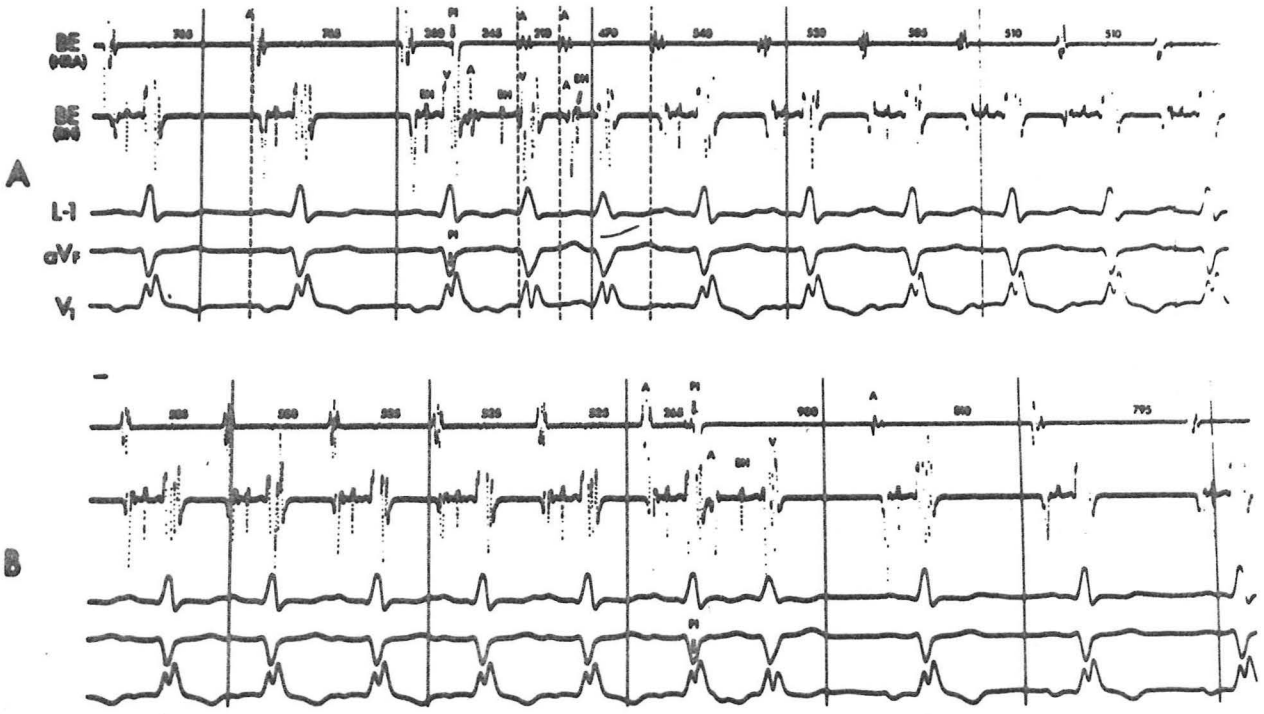

FIGURE 17. Initiation and termination of supraventricular tachycardia due to sustained sinus node re-entry by a single premature atrial beat. Panels A and B are continuous. A) During normal sinus rhythm an induced premature atrial beat at a coupling of 260 msec results in sustained sinus node re-entry and supraventricular tachycardia with a cycle length of 500 to 520 msec. The SVT and sinus node re-entry are terminated by a single atrial premature beat. Note that during SVT, the sequence of activation is from high right atrium (HRA) to low right atrium (B-BH). The broken line demarcates the onset of the A wave in the HRA both during normal sinus rhythm and re-entrant beats in the sinus node (30).

Unifocal Atrial Tachycardia

Automatic ectopic focii have been identified in the atrium (69,70). Coumel (70) has described a 15 year old boy with intractible supraventricular tachycardia. At surgery an ectopic focus was found and surgically cured.

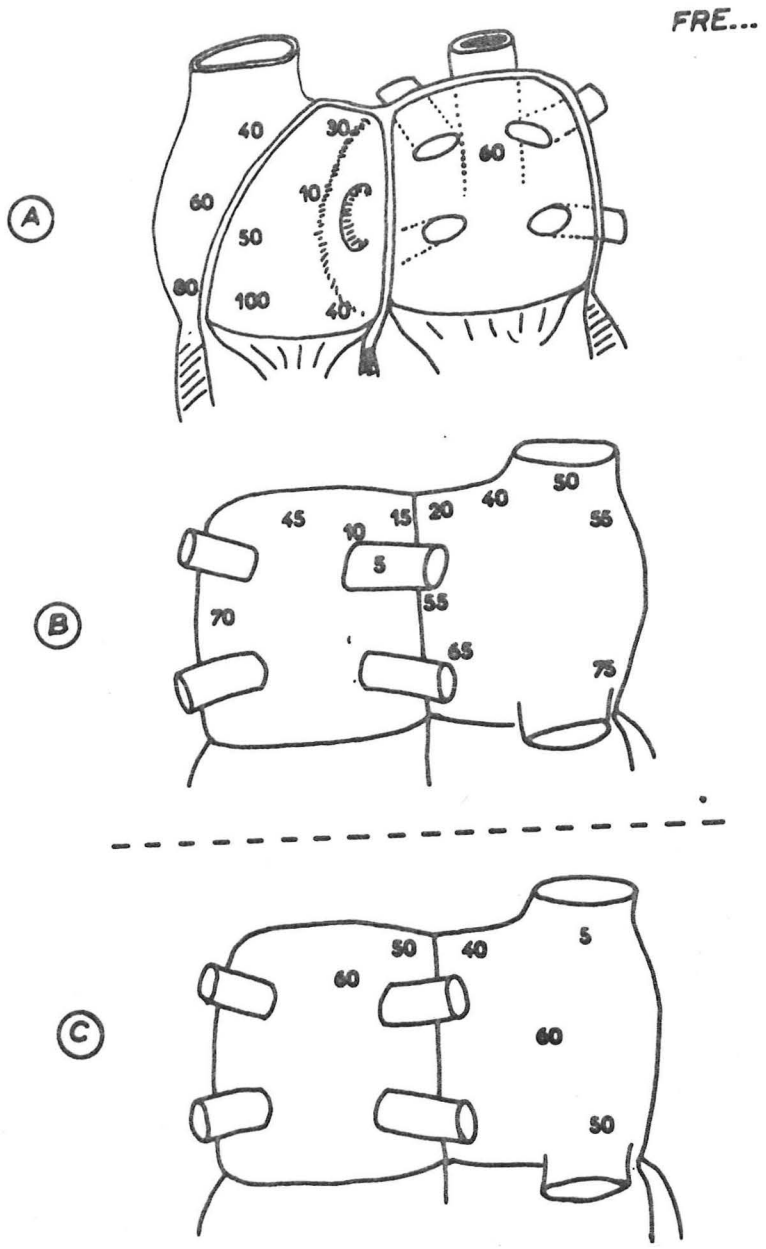

FIGURE 18. Endocardial and epicardial mapping of atrial activation during SVT. A) Endocardial mapping of the right atrium. The point of earliest atrial activation (with respect to the onset of the P wave on the surface ECG) is situated in the region of the upper third of the posterior part of the atrial septum. B) Epicardial mapping at operation shows that the earliest point of activation during SVT is at the origin of the right superior pulmonary vein. C) After abolition of the tachycardia by the pressure of the surgeon's finger, epicardial mapping shows atrial activation from the sinus node.

Accessory Pathways

Accessory pathways have been postulated as a cause of PSVT for years but only recently have they been documented (71-94). Four separate syndromes with accessory pathways have been described.

ACCESSORY PATHWAYS

1. *The syndrome of short PR interval and narrow QRS complexes (Lown-Ganong-Levine)*
2. *Wolff-Parkinson-White syndrome*
3. *Exclusive unidirectional retrograde "pre-excitation"*
4. *Latent bypass pathways*

Anatomically and electrophysiologically defined pathways are shown in Figure 19.

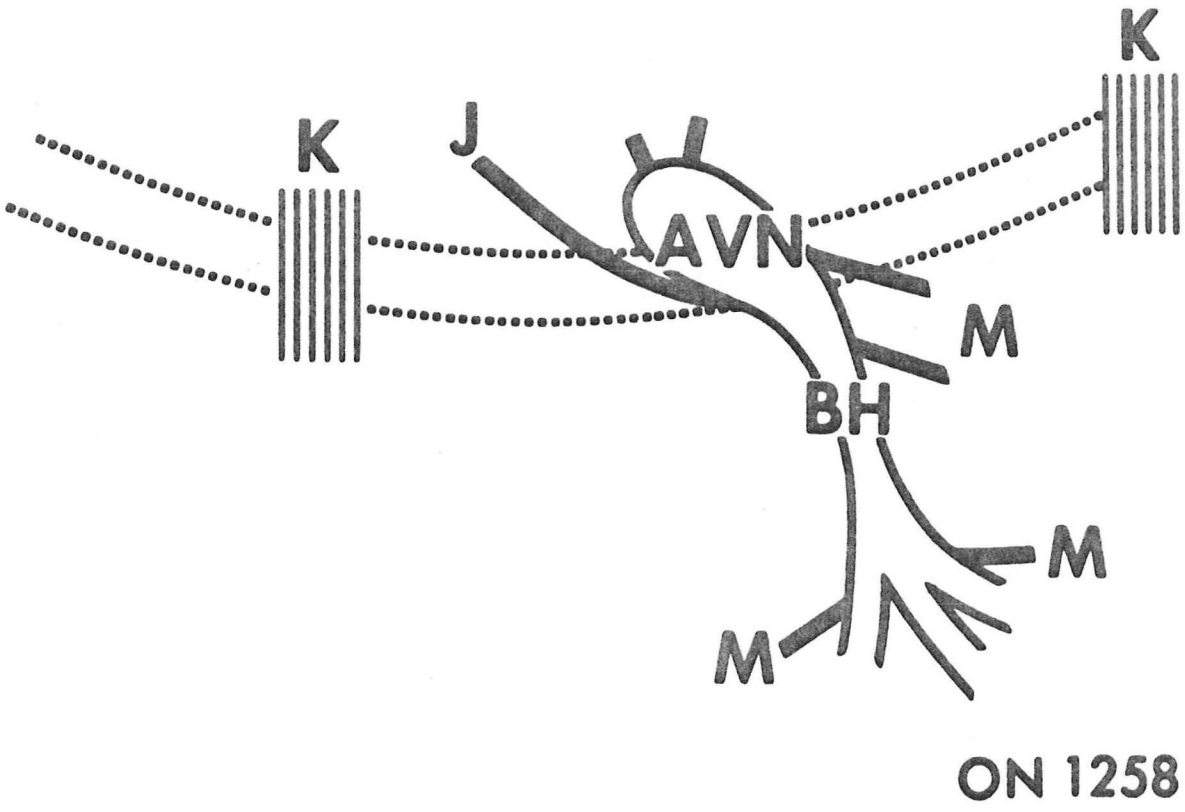

FIGURE 19. Diagrammatic representation of the various possible accessory pathways. K = Kent fibers, J = James fibers, M = Mahaim fibers, AVN = A-V node, BH = bundle of His (95).

Different pathways cause different PR intervals, QRS configurations, and bundle of His recordings. The pathways can be distinguished by atrial pacing in some cases. Sample ECGs and His electrograms are shown in the next figure.

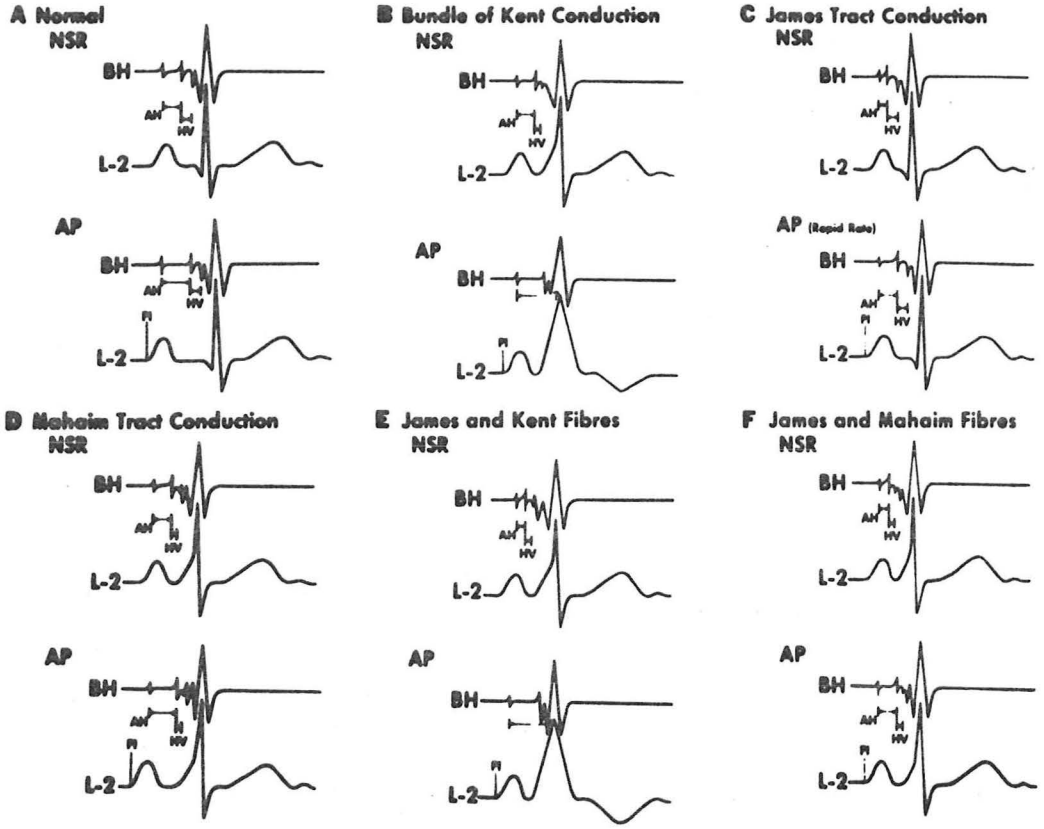

FIGURE 20. Pattern of BH recordings in various types of anomalous connections during NSR and atrial pacing (AP). A) In a normal case the A-H lengthens but H-V remains constant on AP. B) The BH either slightly precedes or occurs simultaneously with or within the QRS. The BH moves into the QRS on AP due to a lengthening of AH time (a normal response); QRS comples becomes more aberrant. C) The A-H is very short and lengthens slightly on AP. The normal QRS complex and H-V interval are unchanged with AP. D) The A-H is normal and lengthens with AP. The shape of the QRS and a very short H-V time are unchanged with AP. E) Response with AP is similar to B. F) This is similar to C except for the presence of the delta wave (95).

The findings in these syndromes are summarized in the next table.

Patterns of BH recordings during NSR and AP associated with various accessory pathways

		Normal AV pathway	*Anomalous connections*				
			Kent	*James*	*Mahaim*	*James, Kent*	*James, Mahaim*
NSR	QRS	Normal	Δ	Normal	Δ	Normal or Δ	Δ
	P–R	Normal	Short	Short	Short or normal	Short	Short
	A–H	Normal	Normal	Short	Normal	Short	Short
	H–V	Normal	Short or minus	Usually normal	Short	Normal, short, or minus	Short
AP	QRS	Constant	Usually widens	Constant	Constant+	Usually widens	Constant
	P–R	Prolongs	Constant	Constant or slightly prolongs	Prolongs	Slightly prolongs or constant	Slightly prolongs
	A–H	Prolongs	Prolongs	Constant or slightly prolongs	Prolongs	Slightly prolongs	Slightly prolongs
	H–V	Constant	Minus	Constant	Constant+	Short or minus	Constant
BH Pacing	QRS	Normal	Normal	Normal	Δ	Normal	Δ
	PI–R	=H–V	=H–V*	=H–V	=H–V	=H–V*	=H–V

Abbreviations: NSR = normal sinus rhythm, AP = atrial pacing, BH = His bundle, Δ = delta wave, minus = BH follows the onset of the QRS complex, PI–R = pacing impulse to the QRS interval, + = constant when Mahaim fibers originate from the BH but may vary if the origin is from the AV node.

*H–V in non-WPW beats.

Short PR, Normal QRS Syndrome

The short PR, normal QRS syndrome or Lown-Ganong-Levine as called by some is felt to be an anomalous James pathway, however this is not proven (96,97). It is felt that the James pathway allows bypass of the A-V node so that the PR is short but re-entry into the bundle of His causes a normal QRS. A re-entry might occur if the James pathway developed unidirectional block and the slow conduction in the A-V node would then set up the criteria for re-entry. It is possible that the re-entry site could be within the A-V node or be due to a different bypass tract than the James fiber. This is at best an uncommon cause of PSVT.

Wolff-Parkinson-White

Wolff-Parkinson-White syndrome is almost always due to a bundle of Kent (98-101). Theoretically a combination of James fibers and Mahaim fibers could cause all the findings in WPW; they have only been shown to have a role in a very rare number of cases and probably are only a rare cause of WPW with the bundle of Kent being the primary mechanism. It is now recognized that patients with WPW also have atrial fibrillation and flutter and may develop ventricular fibrillation if the anomalous pathway can conduct greater than 300/min. Patients presenting to Gallagher (102) for surgical intervention had the arrhythmias as shown in the next figure.

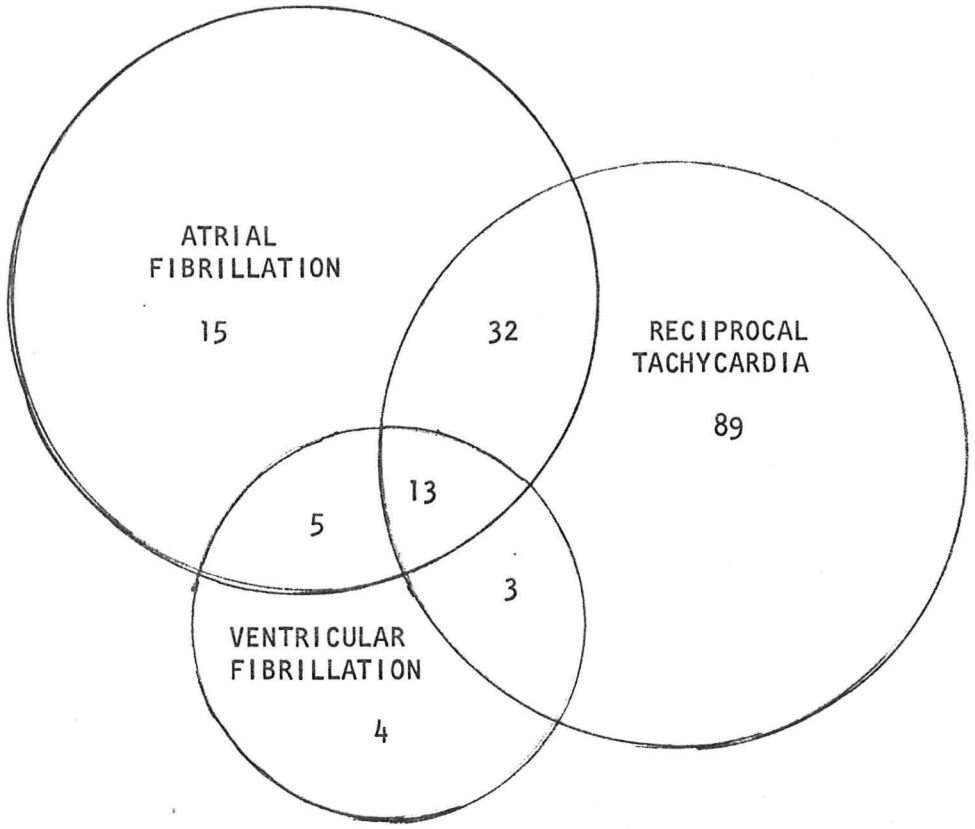

FIGURE 21. The presenting arrhythmias in patients with WPW (102).

During normal sinus rhythm there may be conduction down both the accessory pathway and through the A-V node. Because of the rapid conduction down the accessory pathway there is fusion with the normally conducted impulse. This fusion causes the development of a delta wave and shortening of the PR interval and widening of the QRS. The PZ and PS intervals are unchanged. In Figure 22 the alterations in ECG are diagnosed.

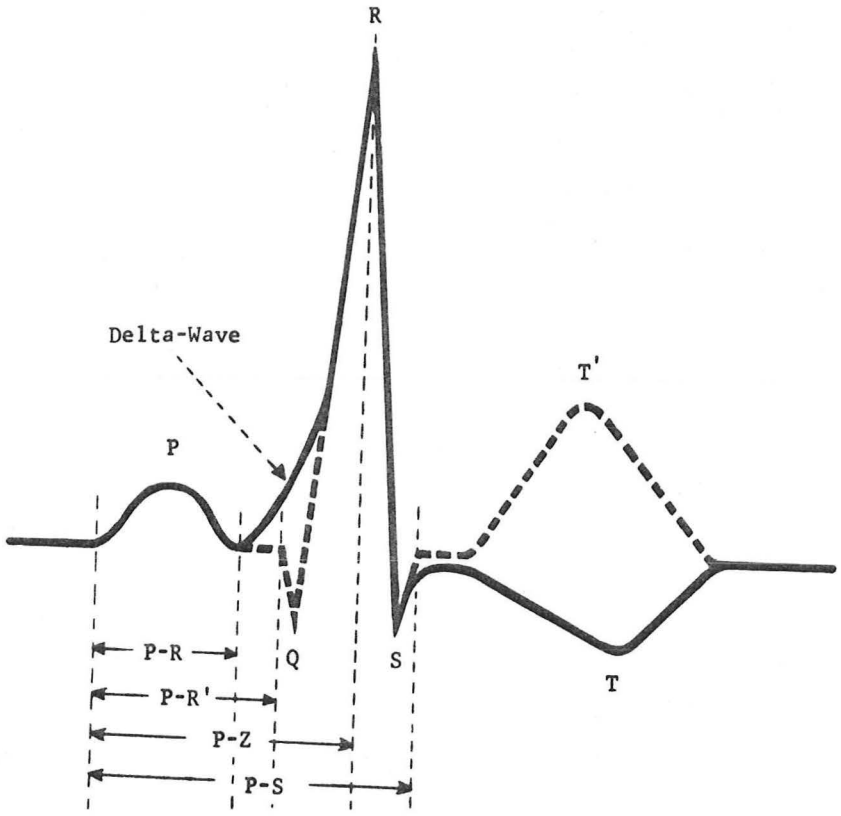

FIGURE 22. The uninterrupted line indicates an anomalous conduction in WPW syndrome whereas the dotted line indicates a normal conduction. The PR interval is shorter than the PR^{1} interval because of a delta wave. Note that the PZ and PS intervals are constant during anomalous and normal conduction. The T wave in WPW is inverted because of secondary T wave changes (103).

Classically the syndrome has been divided into type A and B depending on the direction of the dominant QRS deflection in V_1. In type A the delta wave and the remainder of the QRS are predominantly upright in V_1 with a negative delta wave in lead I. In type B the delta wave and the remainder of the QRS are predominantly negative in V_1 with a positive delta wave in lead I (95). Examples are shown in Figures 23 and 24.

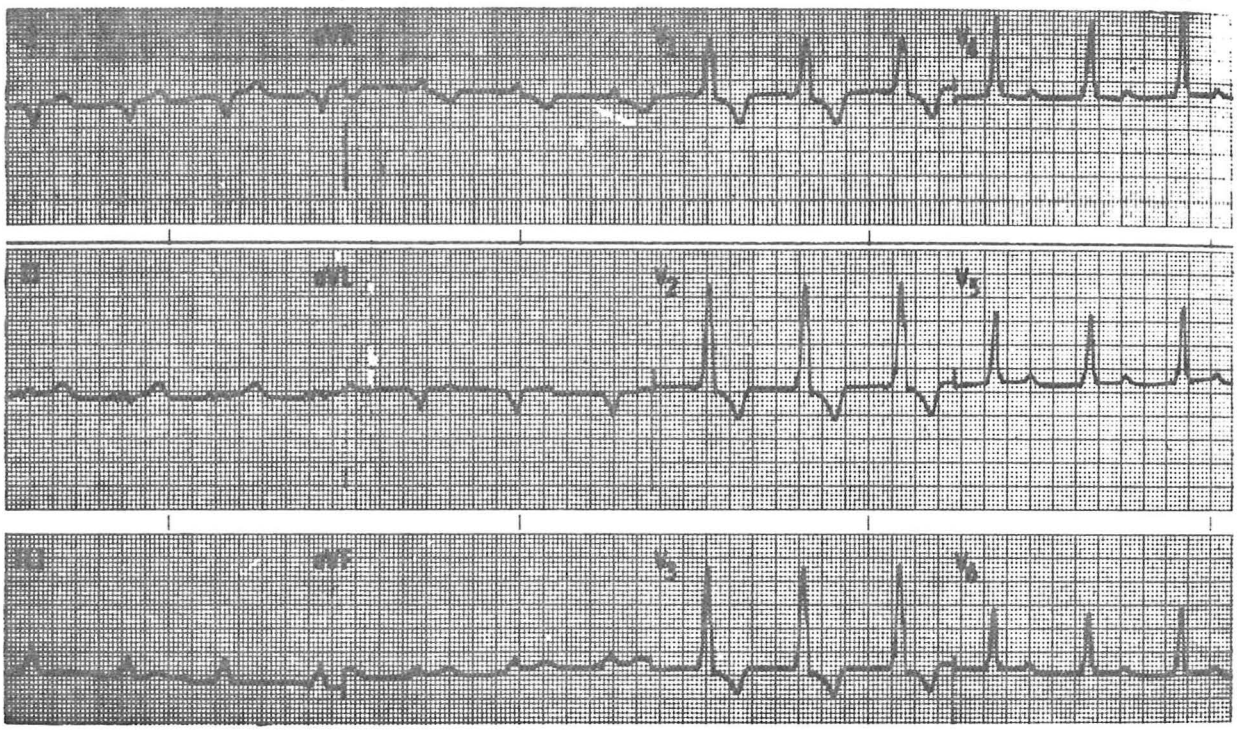

FIGURE 23. Wolff-Parkinson-White syndrome, Type A. The ECG findings resemble right bundle branch block, bilateral bundle branch block, posterior and high lateral myocardial infarction (103).

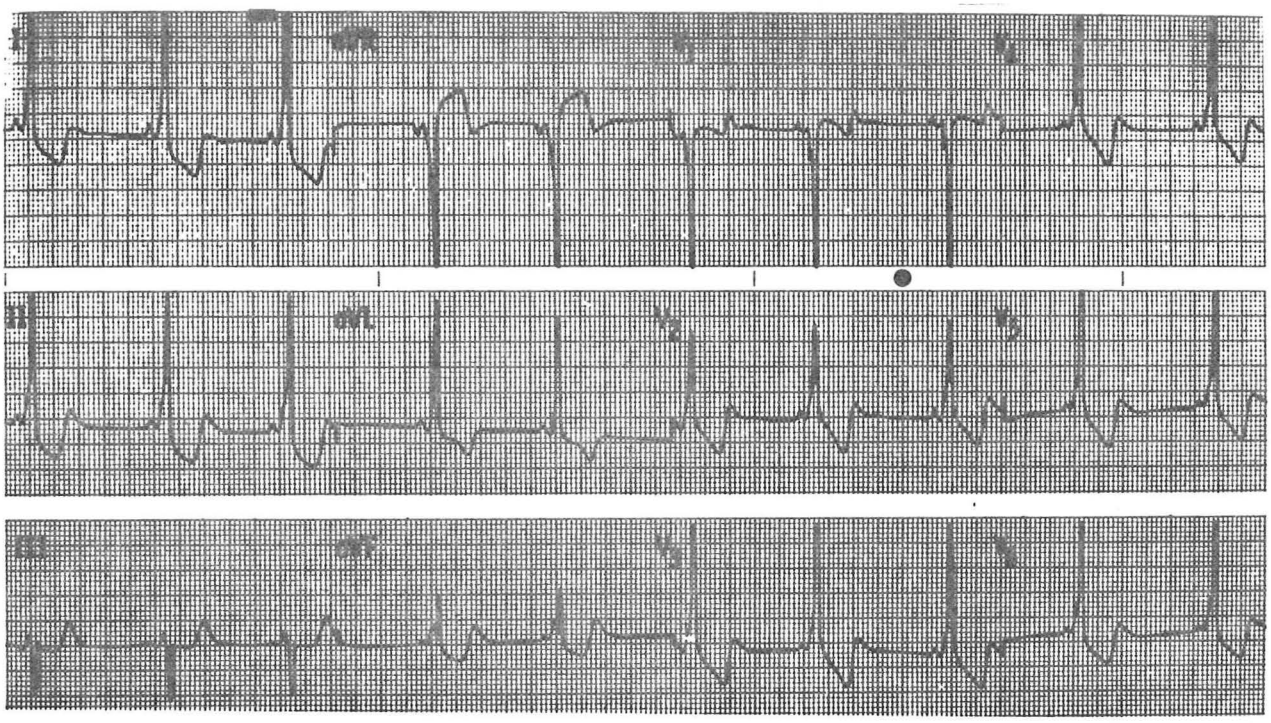

FIGURE 24. Wolff-Parkinson-White syndrome, type B. Left ventricular hypertrophy or LBBB is closely simulated (103).

Also many intermediate types have been described. It was felt that the type would predict where the accessory pathway might be located but this may be fraught with errors as bundle branch blocks may coexist.

The predominant mechanism for the reciprocal tachycardia is unidirectional block in the bundle of Kent with slow conduction through the A-V node and re-entry via the ventricle, bundle of Kent, and atrium (74,77-79,81,88,101). This causes a normal QRS usually and negative P waves in 2, 3, aVF; the QRS and P activity must be present as this is an integral part of the re-entry pathway as diagrammed in Figure 25.

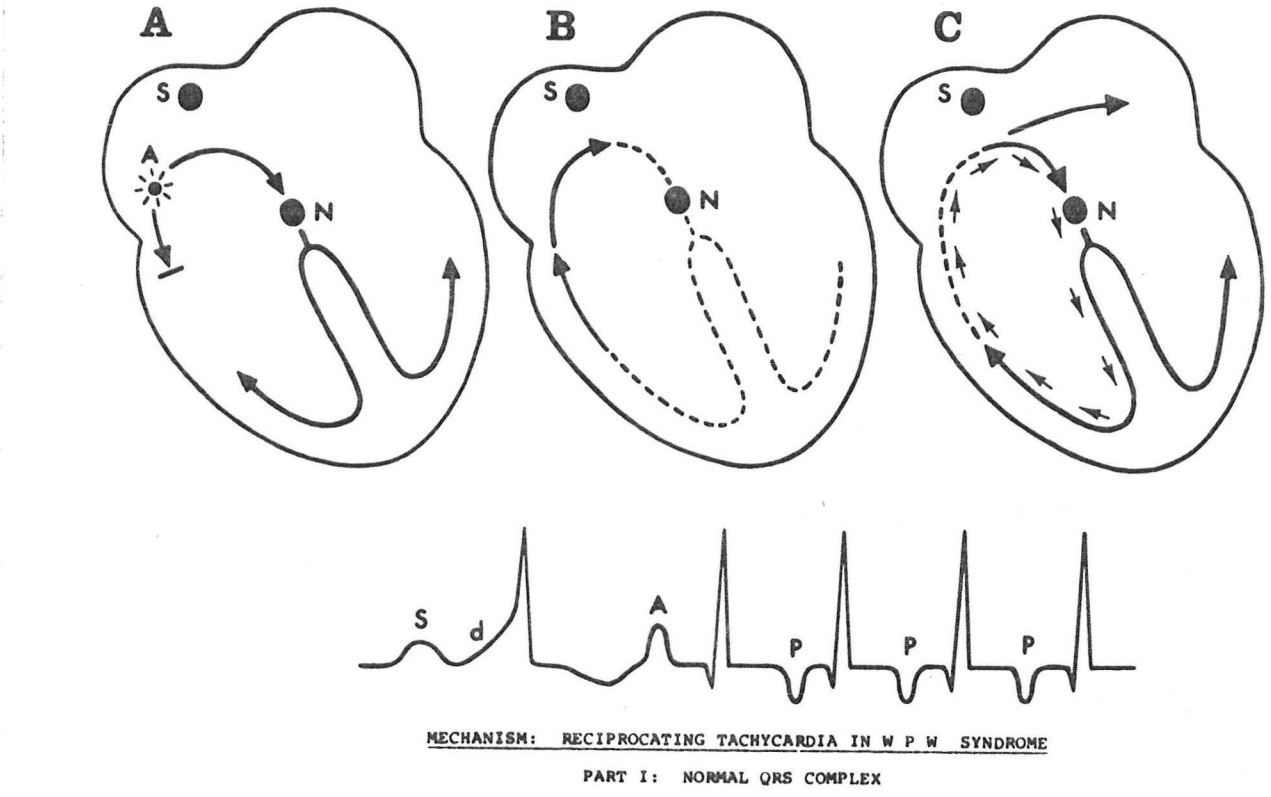

FIGURE 25. Diagram illustrating the mechanism of a reciprocating tachycardia with normal QRS complex in the WPW syndrome. In diagram A, an atrial premature impulse (marked A) is conducted to the A-V node (marked N), but the atrial premature impulse is blocked in the anomalous pathway. The atrial premature impulse is then conducted to both ventricles via the bundle branch system (diagram A). In diagram B, the atrial impulse is conducted to the atria in retrograde fashion to produce an inverted P wave. In diagram C, the impulse is conducted in a clockwise fashion producing reciprocating (re-entry) cycle, and the same cycle may repeat indefinitely. Note the QRS complex during the tachycardia is normal. (S = sinus node; d = delta wave; P = inverted P wave) (103).

While the majority of patients have normal QRS with tachycardia some have abnormal QRS's. Figure 25 shows one mechanism for an abnormal QRS.

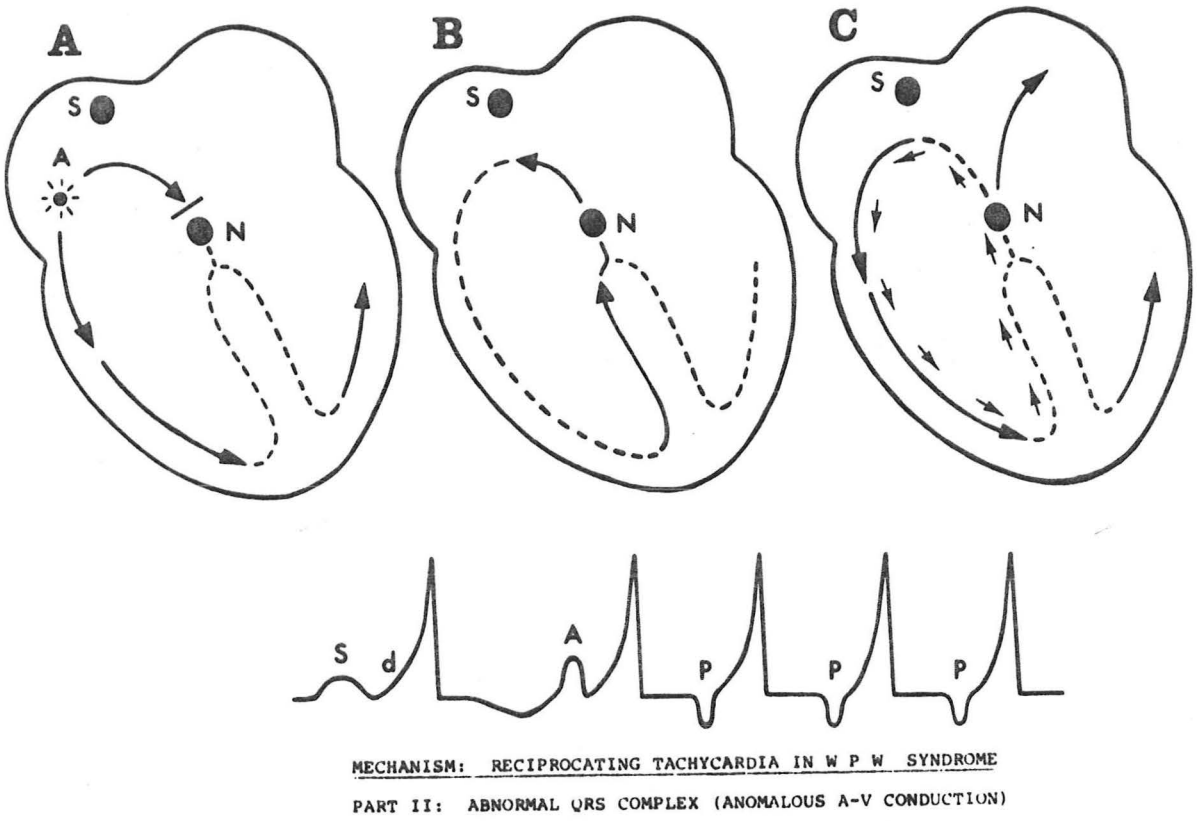

FIGURE 26. Diagram illustrating a reciprocating tachycardia with anomalous conduction in the WPW syndrome. The re-entry is counterclockwise, which is exactly the reverse to that shown in Figure 25.(103).

Bundle branch blocks may also occur causing an abnormal QRS as shown in Figure 27.

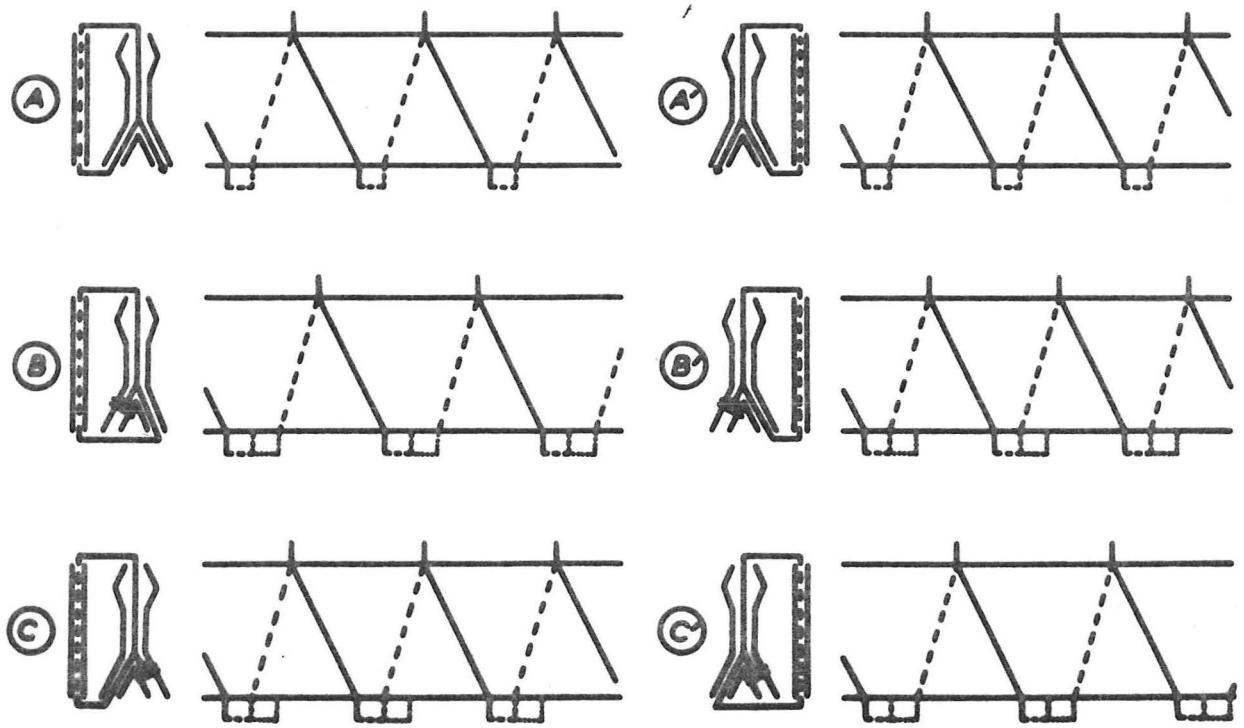

FIGURE 27. Bundle branch block dependent rate of reciprocating tachycardia in the WPW syndrome. A right-sided accessory pathway is present in A, B, and C. The rate of the reciprocating tachycardia is the same when QRS complexes are narrow or widened due to LBBB, but it is slower when RBBB is present. A left-sided accessory path is present in A', B', and C'. The rate of the reciprocating tachycardia is the same when the QRS complexes are narrow or widened by RBBB, but it is slower when LBBB is present (101).

Deaths have been reported in WPW usually secondary to digitalis administration in a patient with an accessory bundle with a very short refractory period or in an accessory pathway that can conduct >300/minute (95).

Persons with WPW may have more than one pathway and may have re-entry tachycardia due to other mechanisms as shown in Figure 28.

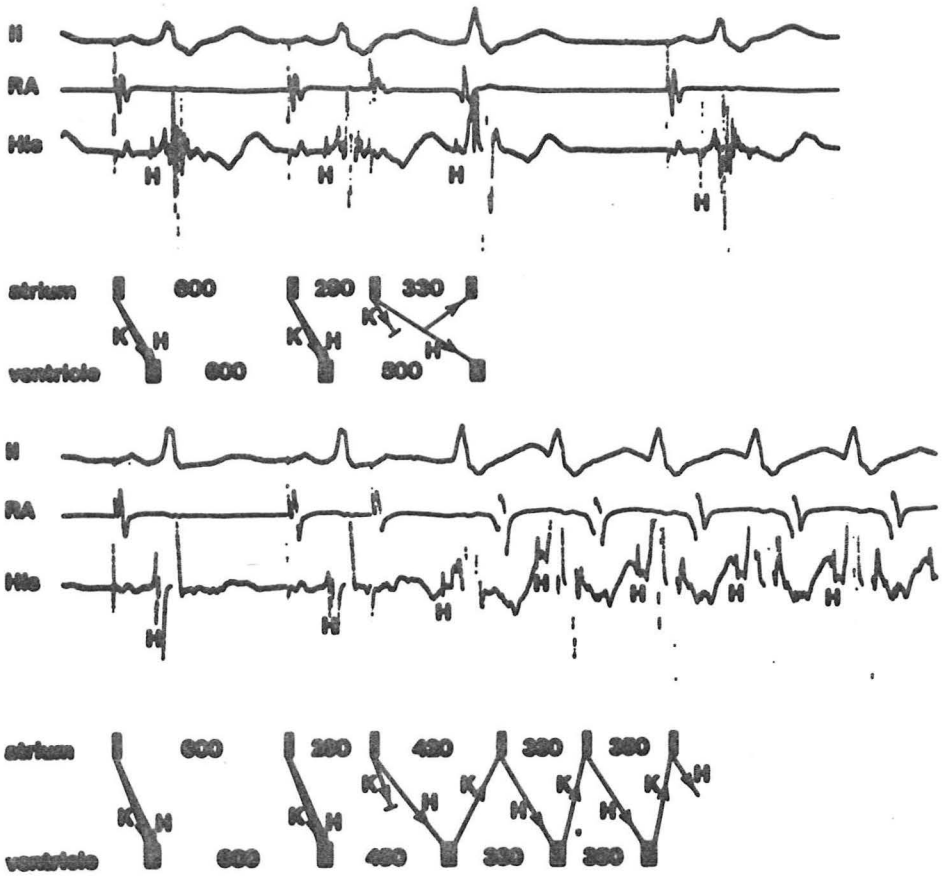

FIGURE 28. A-V junctional reciprocating tachycardia in a patient with WPW. On the upper strip is shown a patient with WPW; a premature atrial contraction causes block in the bundle of Kent and prolongs A-V conduction causing A-V nodal re-entry or echo. On the bottom strips, a premature atrial beat causes block in the bundle of Kent which sets up a WPW type of re-entry (101).

Exclusive Unidirectional Retrograde Pre-excitation

Retrograde re-entry has been shown by Narula in the absence of antegrade conduction (95). This presents a normal ECG when normal sinus rhythm is present and a narrow QRS with PSVT. Only careful electrophysiological studies can discern this mode of re-entry.

Latent Bypass Fibers

There is growing evidence that patients may have latent pathways which are only brought to light with an acute myocardial infarction or other acute event (95). In patients with WPW, additional latent pathways are found in 10-20%.

MANAGEMENT OF SUPRAVENTRICULAR TACHYCARDIA - GENERAL

The treatment of PSVT depends upon the type of mechanism involved, the symptomatology, coexistant diseases, and whether you are trying to convert the rhythm or prevent it. At times there is a precipitating cause such as thyrotoxicosis, pheochromocytoma, anxiety, or a triggering arrhythmia. Obviously the treatment of choice is to treat the precipitating cause. Triggering arrhythmias such as 2^{o} or 3^{o} heart block or PVC's should be suspected in patients developing PSVT late in life and patients should be screened carefully for these and treatment should be aimed at the triggering arrhythmia. Most patients who develop PSVT for the first time probably will not have the arrhythmia again and except for WPW probably should not be treated other than acutely.

WPW presents some special problems. It is now recognized that some patients with WPW present with ventricular fibrillation. Also some patients who are minimally symptomatic may also have ventricular fibrillation. For these reasons most if not all patients with WPW should have electrophysiological studies to see if the pathway will conduct faster than 300/minute. An unresolved question is whether patients with a WPW pattern on ECG but no symptoms should also be studied as the initial presentation may be ventricular fibrillation.

Patients who have recurrent arrhythmias and are symptomatic should also have electrophysiologic studies to see if there is a lesion that can be surgically corrected. Most PSVT, with all or part of the re-entry outside the A-V node, can be approached surgically today. In addition finding the refractory periods of the various pathways might alter drug therapy.

There are three mechanisms for breaking or inhibiting the re-entry pathway. One method is to depress the action potential in the re-entry path so that the impulse can not be conducted. A second method is to prolong the refractory period in the re-entry pathway so that bidirectional block occurs and hence no re-entry. A third mechanism is to enhance conduction in the re-entry pathway so that there is no unidirectional block (104).

The effects of commonly used antiarrhythmias are shown in the following table.

	Atrium	*AV node*	*His–Purkinje*	*Ventricle*	*Accessory pathway*
Length of Effective Refractory Period					
Digitalis	±	+	?	±	−
Procainamide	+	0	+	+	+
Quinidine	+	±	+	+	+
Ajmalin	+	0	+	+	+
Diphenylhydantoin	±	−	−	±	±
Propranolol	±	+	?	±	0
Lidocaine	±	0	?	+	+
Verapamil	±	+	?	±	±
Conduction Velocity					
Digitalis	±	−	?	±	?
Procainamide	−	±	−	−	?
Quinidine	−	±	−	−	?
Ajmalin	−	±	−	−	?
Diphenylhydantoin	±	+	0	+	?
Propranolol	−	−	0	−	?
Lidocaine	−	±	?	±	?
Verapamil	0	−	0	0	?

+=increased, −=decreased, 0=no change, ±=inconsistent, ?=not known

(105)

The effect of these drugs may vary with the type of arrhythmia and the refractoriness of the re-entry pathway. For example digitalis in WPW decreases the effective refractory period in the bundle of Kent. If the refractory period is long in the bundle of Kent and digitalis is administered, the effective refractory period will decrease in the bundle of Kent and increase in the A-V node; this may make the refractory periods almost identical and control the arrhythmia very effectively. However, in a few patients with WPW the effective refractory period in the bundle of Kent is short and digitalis will make it shorter; this will lead to atrial fibrillation, atrial flutter, and/or ventricular fibrillation. In these patients with short refractory periods the acute treatment should be lidocaine followed by procainamide and/or quinidine to prolong conduction. In patients with a very short refractory period in the bundle of Kent propranolol may slow conduction in the A-V node making re-entry more likely. Hence, patients who have short refractory periods, particularly those patients with WPW presenting with atrial fibrillation and atrial flutter, should be treated with lidocaine, procainamide, quinidine, or cardioversion and propranolol and digitalis should be avoided. Those patients who have long refractory periods should be treated with vagal maneuvers, propranolol, digitalis, or cardioversion (103).

ACUTE MANAGEMENT OF PSVT

1. *Vagal maneuvers*
   a) *Carotid massage*
   b) *Valsalva*
   c) *Diving reflex (106)*
   d) *Vasopressors (preferabley neosynephrine) not used as frequently*
   e) *Ipecac*
2. *Propranolol 1-5 mg IV over 15-20 minutes (contraindicated in WPW with short refractory period particularly with atrial flutter or fibrillation)*
3. *Digitalis (contraindicated in WPW with short refractory period particularly with atrial flutter or fibrillation)*
4. *Cardioversion*
5. *Sedation*

WPW WITH SHORT REFRACTORY PERIOD AND/OR ATRIAL FIBRILLATION OR FLUTTER

1. *Lidocaine*
2. *Procainamide*
3. *Quinidine*
4. *Cardioversion*
5. *Sedation*

PROPHYLAXIS AGAINST ATTACKS - EXCEPT FOR WPW WITH SHORT REFRACTORY PERIOD

1. *Digitalis*
2. *Quinidine*
3. *Propranolol*

and/or

1. *Propranolol*
2. *Digitalis*
3. *Quinidine*

4. *Sedation or tranquilizer*
5. *Procainamide*
6. *? surgery*
7. *? pacing*

SURGERY FOR WPW

Surgery has proven very effective in WPW with surgical cures of 92-96% with virtually no mortality in recent series. Surgery can also be considered for sinus node re-entry and ectopic atrial foci (101). Surgery should probably be performed in all patients who have WPW with ventricular fibrillation or a pathway that can conduct greater than 300 beats/minute. Surgery should also be considered in patients with refractory WPW.

FIGURE 29. (next page) Epicardial mapping of accessory pathway to reveal right atrial to right ventricular connection. The surgical incision is shown in the middle panels. The lower panels reveal the pre- and post-op ECG's.

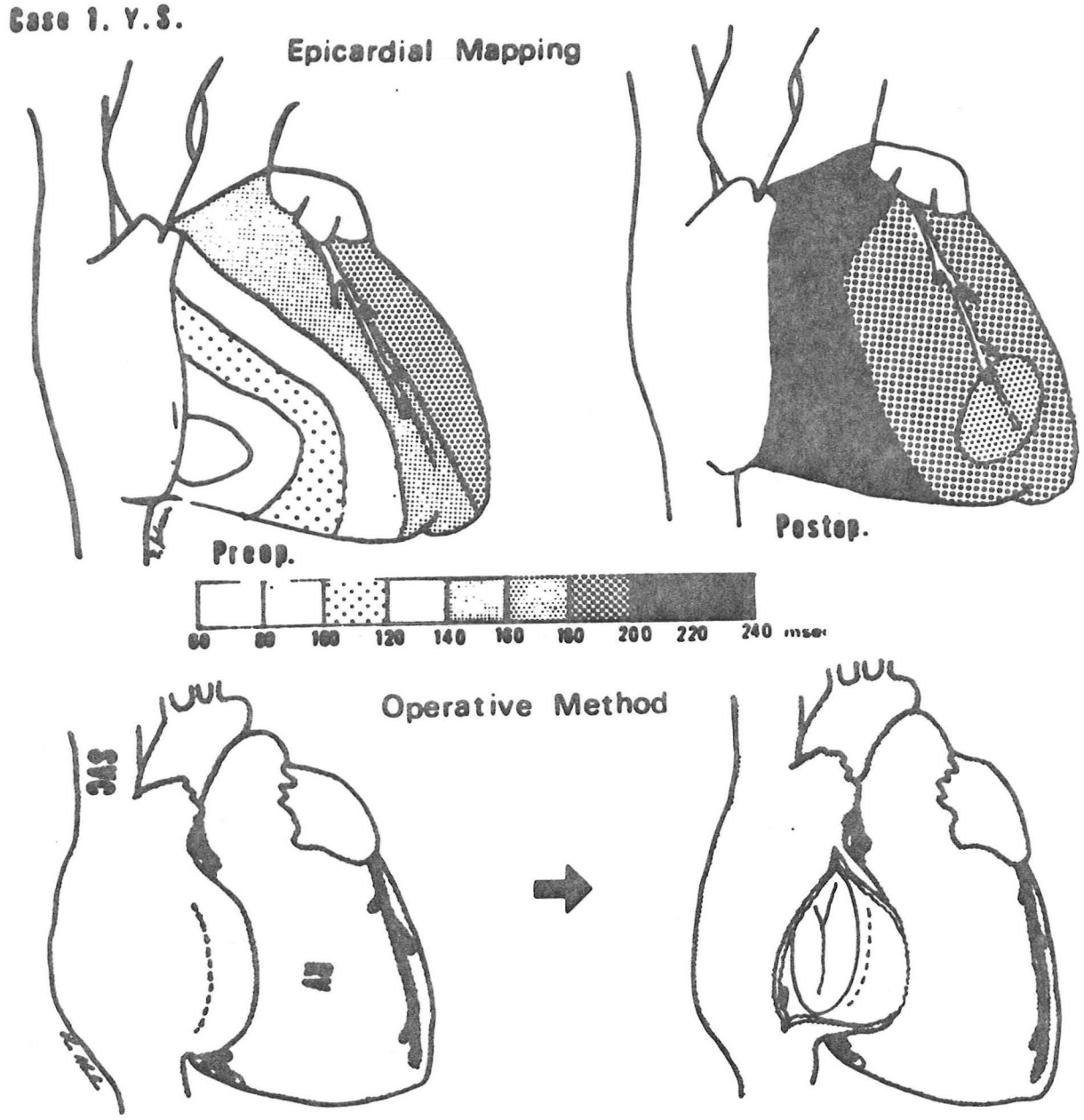
Case 1. Y.S.
Epicardial Mapping
Postop.
Preop.
60 80 100 120 140 160 180 200 220 240 msec
Operative Method
SVC
RA
I II III aVR aVL aVF V1 V2 V3 V4 V5 V6
PRE OP
POST OP
3 Y.

FIGURE 29

Pacing (107-113)

Pacing may be helpful in the management of refractory PSVT. If the triggering mechanism is sinus bradycardia, sinus arrest (sick sinus syndrome), 2^{o} of 3^{o} A-V block permanent demand pacing may be helpful in preventing the triggering mechanism; usually drugs must be given with the pacing to achieve control. Overdrive pacing may also be used to pace the heart at a faster rate than normal to suppress the tachycardias. Rate adjustable pacemakers should be used in these patients.

A demand atrial or ventricular pacemaker may be used to break a tachycardia particularly WPW in a patient that can be terminated by a single stimulus by placing a magnet over the pacemaker to connect it to fixed pacing.

Programmed atrial or ventricular pacemakers either automatic or triggered externally by magnet or radiofrequency transmitter can be used to deliver a series of short stimuli at preselected intervals to terminate a tachycardia.

Finally simultaneous pacing of the atria and ventricles can be accomplished so that fusion occurs in the bypass pathways causing bidirectional block.

SUMMARY

Recent advances in electrophysiology have greatly increased our understanding of PSVT and allowed better therapeutic techniques to be developed for refractory and high risk patients.

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