Refractory Supraventricular Tachycardias

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SUMMARY
This essay is designed to review some of the basic and more recent contributions to our understanding of supraventricular tachyarrhythmias, their mechanisms, pertinent points in diagnosis, and treatment. Supraventricular tachycardia is discussed under the concept of reentrant mechanisms versus accelerated ectopic pacemakers. Atrial fibrillation and flutter, multifocal atrial tachycardia, supraventricular tachyarrhythmias associated with brady-tachy syndrome, and supraventricular tachycardias in ventricular preexcitation are likewise discussed. An outline of the different forms of therapy for refractory supraventricular tachyarrhythmias is provided.

Additional Indexing Words:
- Paroxysmal supraventricular tachycardia
- Atrial flutter and fibrillation
- Multifocal atrial tachycardia
- Bradycardia-tachycardia syndrome
- Nonparoxysmal supraventricular tachycardia
- Ventricular preexcitation

Supraventricular tachycardias are often regarded as benign arrhythmias compared to their ventricular counterparts. In the presence of a well-compensated heart they are usually self-limited or produce little of hemodynamic consequence. However, in the setting of myocardial disease or critical valvular obstruction such arrhythmias can be life threatening. Even in normal hearts they can be deleterious if they become prolonged and refractory to standard modes of treatment. More recently, experimental studies as well as clinical observations have increased our knowledge of the mechanisms of initiation, perpetuation, and termination of many of the supraventricular tachycardias. Likewise, the different factors that make them recalcitrant to therapy are becoming understandable. This essay is designed to review the current knowledge of the electrophysiology involved in the initiation and perpetuation of supraventricular tachycardias and to outline the different forms of therapy for refractory supraventricular tachycardias.

Paroxysmal (or Reentrant) Supraventricular Tachycardia (SVT)

At the present state of our understanding, paroxysmal supraventricular tachycardia is a more inclusive term to include what were previously termed paroxysmal atrial tachycardia, paroxysmal nodal tachycardia, and paroxysmal atrioventricular junctional tachycardia. The more pertinent differentiation depends on the mechanisms involved in the perpetuation of the tachycardia; that is, whether the rhythm persists because of a reentrant mechanism or because of an ectopic pacemaker.

In the vast majority of cases paroxysmal SVT is probably a reentry type of tachycardia. With the use of His bundle recordings and intraatrial electrograms Goldreyer and Bigger demonstrated that the site of reentry was within the A-V node in nine patients with SVT. Experimental and clinical studies have strongly supported the presence of a reentrant pathway in this arrhythmia. They have further commented that microelectrode studies offer strong evidence that some part of the atrium is involved in the reentrant circuit. Paroxysmal SVT has been initiated by a properly timed atrial premature beat (either spontaneously or artificially induced), junctional ectopic beat with an atrial echo, or a ventricular ectopic beat with an atrial echo and ventricular reciprocal beat. Rarely, it may occur in a setting of a sinus impulse with prolonged A-V conduction (figs. 1, 2).

On the surface electrocardiogram paroxysmal SVT is characterized by a rate ranging from 150 to 250 beats/min. When fully established it maintains a fixed rate. It is usually seen in young healthy individuals and patients with no evidence of heart disease or in those with preexcitation syndromes which will be discussed later. Like its mode of onset its termination is abrupt. Episodes are usually

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Supported in part by National Heart and Lung Institute grant no. 05736 and U. S. Public Health Service contract 43-67-1440 from the National Institutes of Health of the U. S. Public Health Service.
Paroxysmal supraventricular tachycardia initiated by a sinus impulse following an interpolated right ventricular paced beat and terminated by a paced ventricular stimulus in a continuous strip with electrograms from the midright atrium (MRA) and low right atrium (LRA). In the upper part of the tracing the first beat is a sinus impulse followed by an interpolated right ventricular paced beat (S) at a coupling interval of 320 msec with concealed retrograde conduction in the A-V node. The next P wave occurs on top of the T wave of the paced beat followed by a ventricular response with a P-R interval of 320 msec. This oncoming sinus impulse finds a functional refractoriness of the A-V node due to the concealed A-V conduction from the paced beat so that it is conducted antegradely with a prolonged P-R and is reflected back to the atrium with transient lasting for a few seconds to a few minutes without significant hemodynamic consequences, although the associated palpitations may be disturbing to the patient. However, it can be quite refractory and may be especially dangerous in patients with underlying heart disease or on rare occasions even in patients with normal hearts.

**Treatment and Prevention**

While the same drugs may be used for both the treatment and prevention of SVT it is important to separate conceptually these two missions since frequently the route of administration and the dosage of the drugs are different, depending on whether treatment or prevention is desired.

Whether considering treatment or prevention of paroxysmal SVT it is useful to have an order of priority of therapy starting with the least dangerous and uncomfortable form of therapy and gradually escalating to more complicated regimens. Simple physiologic measures such as a Valsalva maneuver or carotid massage should be tried initially (fig. 3). Whatever drug one chooses to use in terminating paroxysmal SVT sedation should be the first consideration. While each physician has his own list of priorities in terms of drug usage we have evolved the following pattern. If physiologic maneuvers and sedation have been unsuccessful in terminating the arrhythmia, the first drug we have chosen to use is 10 mg i.v. edrophonium bromide (Tensilon) after testing for hypersensitivity. The vagal effects of the drug may be potentiated if carotid massage is added 60 sec after administration of the drug.

If edrophonium has been unsuccessful, interruption of the arrhythmia may be attempted by pressor amines which are designed to initiate the carotid sinus reflex and thus increase vagal tone and prolong conduction in the A-V node. An alternate approach is to give a rapid-acting intravenous digitalis preparation which also acts to prolong a P-P interval of 660 msec initiating the reentrant tachycardia with a P-P of 500 msec. The first P-P interval is almost equal to the P-P interval of the sinus rhythm at the end of the tracing which means that the ventricular paced beat is interpolated. In the lower part of the tracing the last QRS of the tachycardia is followed by a conducted ventricular paced beat with a coupling interval of 360 msec. It produces retrograde concealed conduction to the A-V node which makes a portion ahead of the oncoming circus wave refractory thereby terminating the circus pathway. The last two stimuli are not conducted because they fall in the absolute refractory period of the right ventricle (paper speed = 25 mm/sec).
Paroxysmal supraventricular tachycardia initiated from a sinus impulse in a setting of 2:1 A-V block with transient A-V dissociation. QRS 1-6 are A-V junctional beats and are dissociated from the sinus rhythm. QRS 7-10 are conducted sinus beats. QRS 10 has a prolonged P-R interval brought about presumably by concealed conduction of the preceding blocked P. This allows the sinus impulse to be reflected back to the atrium (P'), initiating the reentrant tachycardia. Another explanation is that QRS 1-10 are all junctional beats with functional A-V dissociation. QRS 10 produces functional dissociation of α- and β-pathways so that the preceding P which penetrated the upper portion of the A-V node is reflected back to the atrium to initiate the tachycardia with 1:1 ventricular capture.

Conduction in the A-V node. Intravenous propranolol, the beta-blocking agent, can also be used because of its action to prolong A-V conduction.

After surveying these three possible routes of therapy we usually elect to digitalize the refractory patient rapidly because of the high success rate associated with this maneuver and the fact that it provides a useful platform on which to apply other drugs. We have been hesitant to employ pressor amines such as phenylephrine (Neo-Synephrine), methoxamine (Vasoxyl), or metaraminol (Aramine) as first-line drugs because of the danger of inducing cerebrovascular accidents in elderly or hypertensive patients. While propranolol has been
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extremely successful in converting paroxysmal SVT\(^9\) we have preferred to use it after digitalization because there is frequently an element of heart failure after prolonged tachycardia, particularly in the older patient. The addition of a negative inotropic agent such as propranolol may, and has provoked acute heart failure. Intravenous procaine amide as well as intramuscular quinidine has also been used successfully in the treatment of the arrhythmia.

In the face of an arrhythmia which has been refractory to the simple pharmacologic measures outlined above, electrical cardioversion stands as the most reasonable procedure. This approach is extremely successful. A note of caution is always introduced when cardioversion is to be used after the administration of digitalis. Well-known catastrophic arrhythmias associated with electrical cardioversion and digitalis excess can be avoided if the cardioversion attempt starts at very low doses of electrical energy and is gradually increased with careful monitoring of the electrocardiogram.\(^{10}\)

More recently the use of electrical pacing has added a new dimension to the treatment of refractory paroxysmal SVT. Transient pervenous pacing of the right atrium at varying rates or right ventricular pacing at rates slower than the tachycardia, as well as single or coupled properly timed stimuli in the right atrium, has been uniformly successful in terminating episodes of paroxysmal SVT.\(^{11}\) Such cardiac pacing or mechanically induced premature atrial contractions have also been successful in some cases of atrial flutter.\(^{2, 12}\) This approach is most valuable in cases where large doses of digitalis have been administered and where it is preferable to avoid high-energy DC shock. More recently, the induction of chronic atrial fibrillation by paired pacing of the right atrium has been reported by Cohen et al.\(^{13}\) in two patients whose ventricular responses were subsequently controlled by digitalis. Permanent pervenous pacing of the right atrium, coronary sinus, or right ventricle has been successful in the termination and prevention of recurrences of paroxysmal supraventricular tachycardia.\(^{11, 14}\) Coumel has described the successful long-term use of simultaneous atrial and ventricular pacing in certain cases.\(^{8}\) In more resistant cases surgical interruption of the A-V node with subsequent right ventricular pacing has been used.\(^{15}\)

Patients who are seriously bothered by recurrent episodes of supraventricular tachycardia are encouraged to stop indulging in cigarettes, coffee, and other high-caffeine content beverages. If this is unsuccessful we have placed patients on mild sedation, usually with low doses of phenobarbital, and digitalized them. Digitalis is chosen as the first drug because, if successful, it requires only one pill a day. If unsuccessful, other antiarrhythmic agents are added. Propranolol or quinidine alone, and in combination with digitalis, has also been successful in preventing the recurrences. Procaine amide may be substituted for quinidine, particularly if gastrointestinal side effects of quinidine are intolerable. Dilantin has been successful in preventing the arrhythmia although in the older population it has a relatively high incidence of complications such as dysequilibrium and megaloblastic anemia. Other less practical measures such as antithyroid drugs, radioactive iodine-induced hypothyroidism, interruption of cardiac sympathetics, and electrical carotid sinus nerve stimulation have been reported.\(^{16-18}\)

Nonparoxysmal (or Accelerated Ectopic) Supraventricular Tachycardia

This arrhythmia is produced by an accelerated ectopic pacemaker rather than the presence of a reentrant pathway. In the surface electrocardiogram the location of the origin of this arrhythmia in the supraventricular region, whether it be in the atria or in the A-V junctional region, is quite difficult if not impossible to determine except when it is associated with A-V dissociation.\(^{19}\)

The rate of this rhythm is usually moderate, ranging from 70 to 130 and rarely exceeding 150 beats/min. It commonly results from digitalis intoxication. It is also seen following cardiac surgery, myocardial infarction, myocarditis, and occasionally in young healthy persons without demonstrable heart disease. Following intracardiac surgery, myocardial infarction, or myocarditis, it is often transient and benign, requiring no specific treatment. When the ventricular rate is rapid or congestive failure appears, digitalis and other antiarrhythmic drugs, such as propranolol, quinidine, or procaine amide, may be employed. In rare emergency situations, DC cardioversion may be attempted, although the chances of success may be slim.

Atrial Fibrillation and Flutter

Atrial fibrillation or flutter can occur in acute self-limited forms or as chronic arrhythmias. The acute self-limited form is frequently seen in normal
otherwise healthy patients, or it may occur as a complication of an intercurrent illness such as hyperthyroidism, pericarditis, pneumonia, or pulmonary embolus as well as during the postoperative period following a thoracotomy. It has also been associated with alcoholic and heroin intoxication. Refractory or chronic atrial fibrillation commonly occurs in patients with permanent cardiac damage such as that seen in rheumatic, arteriosclerotic, and hypertensive heart disease.

The ventricular response in atrial fibrillation is determined by the degree of A-V junctional conduction. It is always associated with partial A-V nodal block. The irregularity of the ventricular response is due not only to the conduction velocity and refractory period of the A-V junction but also to the different amounts of concealed conduction in the node.20

The atrial contribution to cardiac output ranges from 20 to 25%. With the development of atrial fibrillation the atrial contribution is abolished. This is, however, of little significance in a well-compensated ventricle except during exercise.21 Clinical deterioration after the onset of atrial fibrillation is brought about not only by the loss of the atrial contribution to stroke volume but also by the increase in ventricular rate which decreases the time available for ventricular filling.

Treatment and Prevention

In young healthy patients acute paroxysmal atrial fibrillation or flutter may be so infrequent and so brief that treatment or prophylaxis is not necessary or desired by the patient. Frequently mild sedation or spontaneous sleep will provoke conversion to normal sinus rhythm in patients with this form of nondebilitating sporadic rhythm.

The initial drug of choice in the treatment of atrial fibrillation and flutter is digitalis. It controls the ventricular rate by increasing A-V block. At times it converts atrial flutter to sinus rhythm but more often to atrial fibrillation. Quinidine added to the regimen frequently converts the rhythm to a sinus mechanism by its direct effect upon the atrial musculature. Propranolol alone or in combination with digitalis and quinidine has been successful in converting some cases of atrial flutter.22, 23 Used alone it seldom leads to conversion to sinus rhythm. It may be especially useful in atrial flutter and fibrillation secondary to thyrotoxicosis not only by slowing the heart rate but also by reducing the peripheral manifestations of thyrotoxicosis.

When atrial flutter or fibrillation occurs as a paroxysmal disorder, for example following thoracic surgery or pulmonary embolism, digitalis is often sufficient to control the ventricular response, and conversion to sinus rhythm occurs spontaneously shortly thereafter. If digitalis alone does not lead to conversion to normal sinus rhythm quinidine may be added in a dose of 300 mg orally every 6 hours. Propranolol may also be used with caution starting with a dose of 10 mg every 8 hours with increases in dose to 20 mg every 6 hours if necessary.

In refractory atrial fibrillation and flutter or under emergency circumstances elective DC cardioversion under light anesthesia is the treatment of choice. If performed properly it is more successful and attended with less morbidity than quinidine conversion.24, 25 Although some workers26 have shown that nonsynchronized DC countershock is safe, we have routinely used synchronized DC countershock described by Low.10 Particular attention is given to the arterial pO2, pH, and serum potassium levels prior to cardioversion. Digitalis is discontinued 36–48 hours prior to cardioversion. Patients are usually pretreated with quinidine for 2 or 3 days before elective cardioversion in an effort to reach a serum quinidine level of approximately 4 mg/liter. Such pretreatment rapidly identifies and usually eliminates patients who won’t tolerate maintenance quinidine therapy. It also increases the chance for successful cardioversion.25

Bjerkelund and Orning emphasized the importance of anticoagulation in preventing embolism related to DC conversion.27 Freeman and Wexler28 reported a significant reduction of thromboembolic phenomena in patients with atrial fibrillation onbishydroxycoumarin (Dicumarol) and quoted similar reports in the past. However, embolization has not been a major complication except in high-risk groups such as those with long-standing mitral valve disease, chronic heart failure, and previous embolization. In low-risk groups prior anticoagulation may not be worth the delay, expense, and possibility of hemorrhagic complications.

It has been suggested that the recurrence rate of atrial fibrillation or the maintenance of normal sinus rhythm is more a function of the patient’s residual cardiac disease than a prophylactic drug program.24 However, it has been the general consensus that prophylactic drug programs do prevent recurrences of atrial fibrillation and flutter. Paroxysmal atrial flutter or fibrillation may occur so rarely and be of such brief duration that a long-term prophylactic program to prevent the rhythm is
not necessary. Patients are seldom maintained on quinidine alone, especially following conversion from atrial flutter. Digitalis is maintained to prevent a rapid ventricular response should the arrhythmia recur. More recently Stern has suggested that a combination of quinidine and propranolol may be more effective than quinidine alone in maintenance of normal sinus rhythm in patients who have had recurrent atrial fibrillation.23

**Multifocal Atrial Tachycardia**
(Chaotic Atrial Tachycardia)

Multifocal atrial tachycardia may be defined as a rhythm with an atrial rate greater than 100/min, changing P-wave morphology of at least three types in any particular rhythm strip, varying P-R intervals, and irregular P-P and R-R intervals. It is commonly confused with atrial fibrillation but closer examination of the ECG tracing reveals multiform P waves before the QRS complexes. At times the P waves may be blocked or produce an interference A-V dissociation when associated with a premature ventricular contraction or an A-V junctional premature beat (fig. 4). It may also be associated with second-degree A-V block in a setting of digitalis excess (fig. 5). Occasionally it may be confused with ventricular tachycardia when it is associated with aberrant ventricular conduction.29 The differentiation of this arrhythmia from atrial fibrillation is quite important because digitalis, which would normally be used to control ventricular rate, is often ineffective in controlling the ventricular response in multifocal atrial tachycardia. We have found it very useful to differentiate multifocal atrial tachycardia from atrial fibrillation by using an intraatrial electrode catheter.

Ischemia or injury to the sinus node has been postulated to play a major role in the genesis of this arrhythmia as in the “sick sinus syndrome.” Unlike it, however, no single dominant atrial pacemaker is present.

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

**Figure 4**

Multifocal atrial tachycardia. (A) Parts of a continuous tracing of lead II showing irregular irregularity of the P-P and R-R intervals, multiform P waves, and variable P-R intervals. (B) Unipolar intraatrial electrogram from the same patient showing P waves before each QRS except during periods of interference dissociation indicated by the arrows.
Multifocal atrial tachycardia which developed a second-degree A-V block on digitalis administration. (A) Before digitalis: note the multiform P waves, irregularly irregular P-P, R-R, and P-R intervals characteristic of MAT. (B) On digitalis: note the second-degree A-V block and occasional PVC.

Figure 5

present at any one time, and multiple hyperexcitable ectopic atrial foci or simultaneous atrial parasystolic foci may compete for control of the atria.

Multifocal atrial tachycardia is often a transient arrhythmia seen in association with acute illness but more frequently it occurs in chronically debilitated elderly patients. Because of the age of the patient population in which it occurs there is also a very high incidence of coronary artery disease. Chronic obstructive lung disease is seen in 31–84% of patients with this arrhythmia. Diabetes mellitus has been reported in more than 50%.

More recently Omori reported a case of multifocal atrial tachycardia with Wenckebach phenomenon in a 37-year-old Japanese woman followed for 13 years without demonstrable heart disease.

It is often initiated by frequent premature atrial contractions which progress to multifocal atrial tachycardia. That it heralds atrial fibrillation and usually occurs immediately following cardioversion of atrial fibrillation has been stressed by Lipson and Naimi. Current concepts of the mechanism of initiation of atrial fibrillation make the frequently encountered deterioration of multifocal atrial tachycardia to atrial fibrillation a quite logical course.

The mortality rate in reported series of multifocal atrial tachycardia is quite high although deaths have been attributed to the underlying disease rather than the arrhythmia. We have consistently observed, however, worsening of the patient’s condition once this arrhythmia sets in. Congestive failure and respiratory distress become more pronounced. Lipson and Naimi reported a case of fainting spells during episodes of multifocal atrial tachycardia.

While its course is often self-limited or paroxysmal it can be very recalcitrant indeed. Carotid sinus massage and the Valsalva maneuver, as well as vagotonic and cholinergic drugs, have been uniformly ineffective. Likewise, quinidine, diphenylhydantoin (Dilantin), and lidocaine have failed to convert the arrhythmia. Propranolol has occasionally been successful. Digitalis has been ineffective in controlling the ventricular response in most patients although occasionally it has terminated the arrhythmia.

Digitalis toxicity has been implicated in the genesis of multifocal atrial tachycardia primarily because the arrhythmia has been observed in patients who are on maintenance digitalis due to underlying heart disease and other arrhythmias. Clearly, however, it occurs in many patients not receiving digitalis. The occurrence of digitalis toxicity in multifocal atrial tachycardia is also manifested by second-degree A-V block which can be demonstrated quite easily by intracardiac electrograms. A similar observation was reported by Chung.

We have attempted cardioversion on two elderly patients but were unsuccessful even after...
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400-w-sec shocks. In refractory cases we have seen patients die without reverting to normal sinus rhythm. The most important consideration in management of these cases is the treatment of the underlying disease. Spontaneous reversion appears to parallel improvement in the patient's clinical condition. Bronchodilators such as aminophyllin and isoproterenol frequently used in chronic obstructive lung disease may produce more rapid atrial rates and hence should be avoided. 31

Bradycardia-Tachycardia Syndrome

In 1954 Short 35 described four patients with what has since become known as the bradycardia-tachycardia or brady-tachy syndrome. More recently it has been made synonymous with the “sick sinus syndrome”70 and “sinoatrial syncope—type II”. 36 These patients often present with very slow sinus rates, sinus arrest, or sinoatrial block with consequent symptomatic bradycardia. However, their course is punctuated by a gamut of paroxysmal supraventricular tachycardias, especially paroxysmal atrial flutter and fibrillation. Ventricular tachyarrhythmias are seldom seen. Suppressive drug therapy in these patients often presents difficulties since the negative chronotropic actions of the antiarrhythmic drugs frequently exaggerate the bradycardia and at times make patients more symptomatic.

It is postulated that in these patients the primary defect lies in the sinoatrial node where there are either degenerative or inflammatory changes. 37, 38 This may result in inadequate impulse formation or failure in conduction from the sinus node to atrial tissue. However, it appears that the sinus node is not the only part of the conduction system thus involved. It is not unusual to see a sinus rate of 40 beats/min or less or even transient sinus arrest with failure of lower pacemakers to “escape.” Yet, during episodes of supraventricular tachyarrhythmias a rapid ventricular response is elicited indicating that the A-V junctional tissue is capable of rapid conduction. A-V block has been recognized in patients with this syndrome. 39, 40 In 30 patients studied by Cheng and Ertem, 18 developed A-V conduction disturbances at a relatively slow atrial pacing rate suggesting a conduction defect in the A-V junction. 41 In the study of 75 patients with sinus bradycardia Narula found significant associated conduction disturbances in 50 patients. Interestingly, the 11 patients with documented brady-tachy syndrome had normal A-H and H-V intervals. Five of the 11 patients developed second-degree A-V block during atrial pacing up to 150 beats/min. 42

It has been postulated that the posttachycardia asystole in these patients results from overdrive suppression of intrinsic cardiac pacemakers during the episodes of tachyarrhythmias as has been observed during induced overdrive stimulation. 43 However, Easley and Goldstein have shown that in their patients junctional or sinus beats occur immediately after the tachycardia terminates and only then do prolonged periods of atrial asystole follow. 36

The treatment of choice in the chronic bradycardia-tachycardia syndrome is the installation of a demand ventricular pacemaker to control the bradycardia and the simultaneous use of antiarrhythmic drugs (primarily digitalis and quinidine) to control the tachyarrhythmias. 38 Fixed-rate ventricular pacing alone or in combination with antiarrhythmic drugs has also been used successfully. 40, 44 Kramer and Moss 39 have successfully employed atrial pacing from the coronary sinus in four patients with this syndrome. Although this maneuver may offer hemodynamic advantages derived from the maintenance of the normal relationship of atrial and ventricular contraction one has to be reasonably certain that A-V block is not present before attempting this approach. Atrial pacing with His bundle recordings will help determine this. Needless to say, should A-V block subsequently develop, the catheter electrode must be repositioned in the ventricle in order to insure an adequate rate of ventricular contraction in case sinoatrial bradycardia with failure of a lower pacemaker to escape should become the dominant rhythm. In more resistant cases when ventricular pacing plus antiarrhythmic drugs fail to control the tachyarrhythmia surgical interruption of the A-V node in combination with maintenance of ventricular rate with a pacemaker may be attempted. 15

Ventricular Preexcitation

One cannot complete a discussion of refractory supraventricular tachycardias without including the tachyarrhythmias associated with ventricular preexcitation. There are two main syndromes that may be labeled as such: the Wolff-Parkinson-White (W-P-W) syndrome and its variants, 45 and the Lown-Ganong-Levine (L-G-L) syndrome. 44

Since the first clear clinical description of this syndrome Wolff, Parkinson, and White have emphasized the frequent occurrence of supraventricular tachycardia in patients so affected. It is

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reported that 30–70% of patients with W-P-W eventually develop paroxysmal tachycardias. These patients frequently present the most difficult clinical challenge in the control of supraventricular tachycardias.

The use of artificial pacemakers, His bundle recordings, and direct epicardial excitation mapping in the study of atioventricular conduction have greatly enhanced our understanding of the genesis of the characteristic ECG patterns and mechanisms of tachyarrhythmias in patients with ventricular preexcitation. The more popular interpretation of the different ECG patterns seen in this syndrome is based on the concept of conduction through anomalous A-V connections. The anomalous pathways include: the Kent bundle, a muscular band between the right atrium and right ventricle in the anterolateral region of the heart; the James tract, conduction pathways from the sinoatrial node to the A-V junction with some parts bypassing the A-V node to enter the His bundle; the Mahaim fibers arising from the main His bundle or lower, and coursing directly to the myocardium; and finally the A-V connection of Ohnell, a fibromuscular band which is a counterpart of the Kent bundle and connects the left atrium and left ventricle in the posterolateral region of the heart. A typical W-P-W-QRS complex is a fusion beat formed by combination of a normally conducted impulse and an impulse coming from an anomalous pathway which shares in the depolarization of the ventricle. The degree of QRS alteration depends upon the amount contributed by the impulse passing through the anomalous pathway.

Rosenbaum et al. were the first to classify W-P-W as type A and B, and later different variants were reported. Attempts have been made to correlate the “classic” W-P-W type A and B with the anatomic location of the anomalous pathway. It has been postulated that the type A W-P-W pattern results from preexcitation through a posteriorly located atioventricular connection (Ohnell bundle), whereas the type B W-P-W pattern results from preexcitation through an anterolateral bundle of Kent. However, anatomic and electrocardiographic correlations have not been uniformly successful. Because of this difficulty in correlating location with ECG pattern, classification of the W-P-W syndrome using the 12-lead ECG alone has become increasingly more suspect.

Supraventricular tachycardia in ventricular preexcitation syndromes is commonly believed to be brought about by macroreentry mechanisms which involve one or more of the anomalous pathways and the normal A-V conduction pathway in the circus movement. The presence of either longitudinal dissociation in the A-V junction, accelerated A-V conduction, or activation of James bundle has been postulated to explain the Lown-Ganong-Levine syndrome. Supraventricular tachycardia in W-P-W has been shown to be initiated by a single properly timed pacemaker impulse or mechanically induced atrial premature beat, a ventricular premature beat with atrial echo, or a premature ventricular beat with an atrial echo and ventricular reciprocal beat.

At a critical time when the premature beat propagation is blocked in the anomalous pathway but still able to go through the normal A-V junction (or vice versa), a reentrant tachycardia is initiated. In the same manner that a properly timed stimulus in the atrium or right ventricle initiates SVT, properly timed stimuli have also been shown to revert SVT in the preexcitation syndromes to normal sinus rhythm.

The mechanisms of sudden death in these patients is seldom determinable. Cases of congestive heart failure occurring in infants, patients without underlying heart condition, and patients with associated heart disease have been reported. It appears, however, that a number of deaths may be due to fatal arrhythmias, e.g., ventricular fibrillation. Dreifus et al. recently reported a case of W-P-W where documented ventricular fibrillation occurred and reviewed six cases in the literature whose possible mechanism of sudden death was ventricular fibrillation.

**Treatment and Prevention**

Most attacks of SVT in W-P-W usually stop spontaneously or may be halted by simple measures such as a Valsalva maneuver or carotid sinus pressure which will prolong conduction through the normal A-V junctional pathway, thus interrupting the reentrant mechanism. However, in more refractory episodes even drug therapy is sometimes ineffective. Quinidine given orally or procaine amide given intravenously alone or in combination have been successful in some cases. More recently propranolol has been favored both in the control of SVT and its prevention. Quinidine and propranolol in combination have likewise been successful.
Digitalis has been used during paroxysms of SVT either alone or in combination with quinidine with successful results. However, great caution should be exercised in its use in these patients. Digitalis may act in two ways to facilitate preexcitation: it delays normal A-V conduction in the A-V junction and it may facilitate more rapid conduction through the anomalous pathway. Cardioversion is highly successful in terminating SVT in W-P-W but drug therapy is needed for its prevention. Therapy at times may be effective but it is very seldom indicated.

In a few cases in which drugs have been ineffective in the prevention of recurrent supraventricular tachycardia, pacemakers with the electrodes in the atrium or right ventricle have been used successfully to terminate the recurrent attacks. The pacemaker is a fixed-rate standby unit which is activated by applying a magnet over the generator or a demand unit which is converted to a fixed rate by the magnet. The rationale is to send a series of stimuli hoping that one stimulus will occur at the critical period in the cycle and thus interrupt the circus movement as occurs when properly timed atrial or ventricular pacemaker stimuli are applied.

Surgical approaches in the treatment of SVT in W-P-W have been twofold. First is the creation of A-V block (by surgical incision, electrocoagulation, or ligation of the A-V bundle) and secondly identification and surgical interruption of the anomalous pathway. Both procedures have been successful in the prevention of recurrent tachycardias. However, with the surgical creation of A-V block the patient requires a permanent ventricular artificial pacemaker.

The first attempt at surgical interruption of the anomalous pathway in the treatment of SVT was reported by Burchell and colleagues. A few others have been reported with variable results. The most successful of the published series was reported by Sealy, Boineau, and Wallace.

It would now appear that surgical interruption of the anomalous pathway in certain forms of W-P-W has established its place in the treatment of recalcitrant supraventricular tachycardias which have been debilitating and life threatening. Furthermore, a greater likelihood of success can be expected if electrophysiologic studies including His bundle electrograms, atrial pacing, and epicardial mapping localize the anomalous pathway in the anterolateral portion of the atrioventricular region of the heart as seen in “classic” type B W-P-W syndrome.

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_Circulation._ 1973;47:642-653
doi: 10.1161/01.CIR.47.3.642
_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:
http://circ.ahajournals.org/content/47/3/642

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