Paroxysmal supraventricular tachycardia (PSVT) has been a well-recognized clinical syndrome since the early days of electrocardiography. The clinical syndrome was defined in European literature in the 19th century. In 1867, Cotton reported on an “unusually rapid action of the heart,” and this was followed by further observations by French and German scientists. Variously referred to as Bouveret’s syndrome and later as paroxysmal atrial tachycardia, it was described classically as “a fully unprovoked tachycardia attack, lasting a few seconds or several days, in patients who as a rule have otherwise healthy hearts.” It has been electrocardiographically defined in the 10th Bethesda Conference on Optimal Electrocadioigraphy as “a tachycardia usually characterized by an atrial rate of 140 to 240 beats per minute (bpm) and by an abrupt onset and termination. It may or may not be associated with intact A-V conduction. Specific electrophysiological studies may elicit specific mechanisms such as retrograde and anterograde pathways and sites of reentry.”

In the past 25 years, this has been an intensively studied arrhythmia, with extensive definition of its genesis, presentation, subtypes, and electrophysiology. Pharmacologic therapy and, later, nonpharmacologic therapy have been investigated and refined. This is now a classic story in the evolution of clinical cardiac electrophysiology and forms a fundamental cornerstone in the modern treatment of cardiac arrhythmias.

Epidemiology

The epidemiology of PSVT has not been widely investigated in modern times. Early electrocardiographic reports were useful in arrhythmia detection for patients presenting with sustained palpitations and persistent episodes of supraventricular tachycardias (SVT) but had little role for this purpose in the large body of patients with brief SVT events terminating before presentation to the physician. These efforts were supplemented by the advent of ambulatory electrocardiography, which documented a large number of cardiac arrhythmias in asymptomatic patients. Conversely, it confirmed that many symptoms experienced by patients with or without heart disease that are suggestive of tachyarrhythmias occur when no arrhythmias or simply premature beats are documented on monitoring. Epidemiologic data are extensively colored by the selection criteria and the electrocardiographic documentation mode used in the study. Brodsky and coworkers reported that ambulatory electrocardiographic recordings in 50 male medical students detected atrial premature beats in 56%, but only one had more than 100 beats in 24 hours. The limited period of observation precludes objective assessment of development of SVT events in these subjects. Thus, Hinkle et al. in a study of 301 men with a median age of 56 years, detected various supraventricular arrhythmias in 76% of these individuals. However, coronary disease was present in 20% of these patients.
Clark et al. studied an apparently normal population ranging from 16 to 65 years old and noted a low incidence of supraventricular arrhythmias.\textsuperscript{8,9} In contrast, recent longitudinal studies with telemetric monitoring and even implanted cardiac pacemakers document a very high incidence of asymptomatic and symptomatic atrial arrhythmias, particularly atrial fibrillation in patients with bradyarrhythmias.\textsuperscript{10} Thus, it would be appropriate to infer that the true incidence of PSVT in the general population remains unknown due to its evanescent nature and limited methods of detection. It would also be appropriate to surmise that the incidence may be higher than generally believed over a long observation period. Atrial arrhythmias also increase with age.\textsuperscript{11} In the Cardiovascular Health Study, short runs of PSVT occurred in 50% of men and 48% of women, doubling in prevalence in octogenarians. Twenty-eight percent of nursing home residents demonstrate PSVT.\textsuperscript{11}

More data are available for the preexcitation syndromes, particularly the Wolff-Parkinson-White (WPW) syndrome. Electrocardiographic studies of healthy individuals suggest that the incidence of this condition is 3 in 1000 of the general population.\textsuperscript{12} Early studies suggested that the morbidity and mortality in WPW syndrome with tachyarrhythmias were greater in adults with ventricular fibrillation occurring in patients with atrial fibrillation and antegrade preexcitation.\textsuperscript{13} However, Klein et al. have reported on the natural history of asymptomatic WPW syndrome; they noted a very low incidence of major morbidity and serious symptomatic arrhythmias including atrial fibrillation with rapid antegrade conduction and mortality.\textsuperscript{14}

Supraventricular arrhythmias are seen at all ages and are particularly common in infants, children, and young adults. Age-related behavior of these rhythms has also been the subject of epidemiologic study. In a study of infants younger than 1 year of age, Mantakas et al. noted that associated congenital heart disease was present in 35%, and fully 90% developed SVTs with narrow (35%) or wide QRS (45%) complexes.\textsuperscript{15} Most patients (90%) improved with growth or remained stable, but patients with congenital heart disease could have refractory arrhythmias. The quality and duration of life in children with WPW syndrome without clinical dysrhythmias have been reported to be normal.

**Anatomy and Pathology**

**NORMAL ANATOMY OF THE ATRIOVENTRICULAR JUNCTION**

In order to understand the pathologic base for atrioventricular (AV) nodal tachycardia and AV reentry tachycardia, the normal anatomy of the AV node and its approaches, including the atria and the AV bundle, is briefly reviewed in the following regions of interest: (1) approaches to the AV node including the atrial septum; (2) AV node; and (3) AV nodo-bundle junction.\textsuperscript{16-27}

**APPROACHES TO THE ATRIOVENTRICULAR NODE**

Approaches to the AV node include the atrial myocardium located in the anterior, superior, midseptal, and inferior regions as they converge to the AV node. The approaches beneath the coronary sinus area may be called the posterior or inferior approaches. In addition, the approaches also originate from the tricuspid valve. The left-sided approaches include those from the left atrial myocardium and the mitral valve. The superior approaches include the pectinate muscles as they merge from the superior, lateral, and posterior walls of the right atrium toward the AV nodal area, atrial septum, and Todaro’s tendon. Approaches to the AV node are formed by different types of myocardial fibers coming from different directions as they merge toward the AV nodal area (Fig. 14-1). Histologically, in general the cells are relatively loosely arranged with lighter staining smaller cells. The size and shape of the myocardial fibers vary considerably from one approach to the other in the vicinity of the node. In general, there is increase in the elastic collagen connective tissue intermingling with the cells, fat, and a large amount of nerve fibers. At the electron microscopic level, AC

![FIGURE 14-1 Schematic representation of the atrioventricular (AV) junction depicting the approaches to the AV node. AVN, AV node; CFB, central fibrous body; LA, left atrial myocardium—left atrial approaches; LV, left ventricular side of the septum; MV, approaches from the mitral valve; RA, right atrial myocardium—superior approaches; AS, atrial septum; S, superior approaches; I, inferior approaches; RV, right ventricular side of the septum—right ventricular approaches; TV, approaches from the tricuspid valve; VS, summit of the ventricular septum. Arrows point to the approaches to the AV node from the tricuspid valve area, right atrial aspect, right ventricular aspect, atrial septal aspect, left atrial aspect, and mitral valvular aspect. (With permission from Bharati S, Lev M: The anatomy of the normal conduction system: Disease-related changes and their relationship to arrhythmogenesis. In Podrid PJ, Kowey PR [eds]: Cardiac Arrhythmias, Mechanism, Diagnosis and Management. Baltimore, Williams & Wilkins, 1995, p 1.)](image)
level, the mitochondria and myofibrils of the atrial myocardial cells are not as well organized as the ventricular cells, and some of them do not have a transverse tubular system.\textsuperscript{16}

**SIGNIFICANCE OF VARIATIONS IN THE SIZE OF APPROACHES TO THE ATRIOVENTRICULAR NODE**

The atrial myocardial fibers including the collagen, elastic tissue, and nerve elements in the approaches to the AV nodal area can vary in size, shape, and direction, suggesting that there may be functional differences in the speed of conduction.\textsuperscript{16-20} Anatomically, one approach to the AV node may be more dominant than the other. For example, dominant posterior approaches with less prominent superior approaches in the human suggest the possibility of dominant slow pathway conduction. Likewise, dominant superior approaches with practically absent inferior approaches suggest the possibility of dominance of fast pathway conduction or other alterations in AV nodal conduction. Myocardial fibers in the atria may get entrapped within the central fibrous body and join the AV node. In other instances, approaches to the AV node and the AV node may be entrapped within the tricuspid or mitral valve annulus or the base of the aortic valve. During an altered physiologic state, these anomalies may form an anatomic substrate for a reentry circuit resulting in various types of supraventricular or junctional arrhythmias.\textsuperscript{16-18}

**THE ATRIOVENTRICULAR NODE**

The AV node is normally in continuity with its atrial approaches.\textsuperscript{16-23,26} It is a sizable structure and is usually located near the annulus of the septal leaflet of the tricuspid valve. In the adult, it measures approximately 5 to 7 mm in length and 2 to 5 mm in width. The node extends closely beneath the endocardium of the right atrium, adjacent to the septal leaflet of the tricuspid valve, and lies very close to the right ventricular aspect of the ventricular septum and the central fibrous body. The size and shape of the node are not uniform in nature and vary considerably from heart to heart. At the light microscopic level, the AV node consists of a meshwork of cells that are approximately the size of atrial cells but are smaller than ventricular cells. Histologically, the AV node may be divided into three layers: (1) superficial or subendocardial, (2) intermediate or midzone, and (3) deep or innermost layer.\textsuperscript{16-18}

**SUPERFICIAL OR SUBENDOCARDIAL LAYER OF THE ATRIOVENTRICULAR NODE**

The approaches that come from various directions merge gradually with the superficial or subendocardial part of the AV node. These fibers are loosely arranged with smaller nodelike cells oriented along the atrial cells, some intermingling with the atrial cells, fat, elastic tissue, collagen, and nerve fibers. A distinct increase in fat occurs with normal aging of the heart.\textsuperscript{16-18}

**INTERMEDIATE OR MIDLAYER OF THE AV NODE**

AV nodal cells are more or less compact; however, the orientation and arrangement of the cells vary considerably. The collagen and elastic tissue content is less than that seen in the superficial layer with fewer nerve fibers. In the older age group, fat may be present in the intermediate layer of the node.\textsuperscript{16-18}

**DEEP OR INTERMOST LAYER OF THE NODE**

AV nodal cells are tightly arranged and may be considered compact. However, the arrangement and orientation of these cells also vary considerably. There is some amount of collagen and elastic tissue, though somewhat less than the intermediate and superficial layers. Fat may be seen intermingling with the nodal cells in the older age group. At the light microscopic level, the nodal fibers vary from the periphery toward the central fibrous body.\textsuperscript{16-18} At the electron microscopic level, there are fewer myofibrils and mitochondria, which are randomly arranged. The cytoplasmic reticulum is poorly developed, and there is no transverse tubular system. It is not known whether the AV node contains more glycogen than the surrounding atrial and ventricular mycardium. The gap junctions are scarce, but desmosomes are frequent. Fascia adherens are more than in the sinoatrial nodal cells but not as frequent as in atrial and ventricular myocardial cells.\textsuperscript{16-18}

**BLOOD SUPPLY AND NERVE SUPPLY TO THE ATRIOVENTRICULAR NODE**

In approximately 90\% of hearts, the AV node is supplied by ramus septi fibrosi, a branch from the right coronary artery reinforced by branches from the anterior descending coronary artery.\textsuperscript{16-23} Copious nerve cells surround the AV node, especially in the atrial septum adjacent to the AV node and nerve fibers within the node. The exact distribution and destination of the nerves in the human in the AV nodal area are still unknown. However, the rich autonomic innervation of the sinoatrial node and AV nodal areas in the canine heart indicates that the sinoatrial node is particularly responsive to parasympathetic adrenergic regulation, whereas the AV nodal conduction is preferentially sensitive to sympathetic adrenergic regulation.

**VARIATION IN SIZE, SHAPE, AND LOCATION OF THE ATRIOVENTRICULAR NODE**

The node lies beneath the septal leaflet of the tricuspid valve in close proximity to the right ventricular aspect of the ventricular septum and the central fibrous body. The AV node may be draped over the central fibrous body within the atrial septum, or some of the fibers may be in part within the tricuspid valve annulus and may be in part within the central fibrous body. In some cases the fibers may be situated toward the left atrial aspect.
or in part within the mitral valve annulus, or they may be situated close to the base of the aortic valve.\textsuperscript{16-23} AV nodal–like cells may also be seen near the tricuspid, mitral, and aortic valve annuli. Some of these nodelike cells may enter the central fibrous body and eventually join the regular posterior AV node. In addition, an accessory AV node may be seen in the parietal wall of the right atrium near the annulus of the tricuspid valve, anterosuperiorly.\textsuperscript{16} Note that not all the AV nodal cells eventually form the AV bundle; some remain, such as leftover nodal cells, and lie adjacent to the central fibrous body or near the valves.

**MAHAIM FIBERS**

Conduction fibers from the AV node, AV bundle, and left bundle branch may join the ventricular myocardium. They have been referred to as Mahaim fibers or paraspecific fibers of Mahaim. They may form the substrate for a unique variety of ventricular preexcitation. The myocardial fibers resemble the cells of the tissue of origin and gradually take over the characteristics of the ventricular myocardial cells. Mahaim fibers may be present from the AV node to the right, left, or midpoint of the ventricular septum.\textsuperscript{16-23}

**ACCESSORY ATRIOVENTRICULAR BYPASS PATHWAYS—FIBERS OF KENT**

Accessory AV bypass pathways bypassing the AV node are seen in normal infants up to 6 months of age.\textsuperscript{21} The myocardial cells on the atrial side resemble atrial cells, and those on the ventricular aspect resemble ventricular cells.\textsuperscript{16-22} In adults, it has been well documented that such pathways cause preexcitation with varying types of supraventricular arrhythmias.

**AV NODO-BUNDLE JUNCTION**

The AV node penetrates the central fibrous body to become the penetrating part of the AV bundle. The penetrating AV bundle undergoes or assumes different shapes and contours. The orientation of fibers differs significantly. The nodo-bundle junction is very small, measuring approximately 1 to 1.5 mm in greatest dimension or less in the majority of hearts. The nodo-bundle junction becomes a part of the AV node and may be considered as the most distal part of the AV node or the beginning part of the penetrating AV bundle.\textsuperscript{16-18} The AV nodal cells that are close to the tricuspid valve and the posterior approaches are first molded to form the penetrating AV bundle. On the other hand, the nodal fibers from the anterior/superior aspect are the last to lose their continuity with the superior approaches as they enter the central fibrous body to form the AV bundle. Thus, the formation of the AV bundle within the central fibrous body occurs differentially. The posterior part of the AV node penetrates the central fibrous body earlier than the anterior or superior part. The superior part of the AV node includes the nodal fibers closer to the left atrial side, atrial septal, and right atrial aspects.\textsuperscript{16-18}

**FUNCTIONAL SIGNIFICANCE OF THE NODO-BUNDLE JUNCTION**

The variation in sizes of the superior and inferior components of the node, as well as the variations in which they form the AV bundle, may provide a substrate for arrhythmias. Since histologically the nodo-bundle junction has the characteristics of both the AV node and the AV bundle, its functional properties may be intermediate, having characteristics of both the node and the penetrating AV bundle. Likewise, the atrial myocardial approaches from the mitral valve, tricuspid valve, the right ventricular myocardium, and the atrial septum tend to get entrapped within the central fibrous body and may later join the AV node or the nodo-bundle junction, or both. The normal variations in morphology of the AV junction have the potential for supporting AV nodal arrhythmias.\textsuperscript{16-18} The variation in the morphology of the AV junctional area also probably predisposes to varying types of physiologic phenomena, including dual AV nodal pathways and other junctional arrhythmias.\textsuperscript{15,16} For example, an anterior AV node in the parietal wall of the atrium or near the tricuspid valve annulus or double AV node may alter its conduction velocity. Dual AV nodal pathways may be normal or abnormal. They may be transient or become permanent.\textsuperscript{16-18}

**ACCESSORY ATRIOVENTRICULAR NODE AND ITS RELATIONSHIP TO PREEXCITATION AND ATRIOVENTRICULAR JUNCTIONAL TACHYCARDIAS**

Few pathologic studies document an accessory AV node being responsible for preexcitation or other arrhythmias originating from the AV junction. Figure 14-2 is an example of a 5-month-old child with a history of intractable junctional tachycardia. He died suddenly, and postmortem examination demonstrated a displaced coronary sinus relative to the septal leaflet of the tricuspid valve with a double AV node and double AV bundle (see Figs. 14-2A and B). In other instances, the AV node has been located in part within the central fibrous body with a left-sided AV bundle, or the right AV node has been completely interrupted by sutures, and a left-sided AV node was connected to the atrial septum. It was recently documented that an accessory AV node located anterior to the AV junction directly communicated with the right atrium and right ventricle in a curved fashion that produced ventricular preexcitation and formed the retrograde limb of typical AV reentrant tachycardia (AVRT). In another patient with typical preexcitation and recurrent AVRT who died suddenly, there were no conventional anomalous pathways on the right side. Instead, an anterior accessory AV node in the right atrium continued as the infundibular right ventricular myocardium.

These types of abnormalities of the AV junction are seen in children as well as adults. Pathologic study in a 13-month-old infant with hypertrophic cardiomyopathy and paroxysmal SVTs, consistent with AV reciprocating tachycardia using a concealed posterior accessory
pathway, revealed that the central fibrous body was abnormally formed with numerous Mahaim fibers (nodoventricular) on both sides of the septum with fibrosis of the left bundle branch (Fig. 14-3).

Concepts and Classification

The fundamental concepts underlying recurrent PSVT have been elucidated by decades of experimental and clinical electrophysiological investigation. Early experimental studies of Moe and associates and the seminal clinical studies of Durrer, Wellens, Castellanos, Rosen, and Denes, among others, helped define the mechanisms and substrates involved in these arrhythmias.28-31 Extensive pathologic studies as mentioned earlier have further enhanced our understanding of the anatomic basis of these arrhythmias. Fundamental to our understanding is the concept that these arrhythmias may be due to either enhanced automaticity or reentry. While the latter may predominate in certain populations and clinical practice, there is considerable overlap in clinical and electrocardiographic features. Automatic arrhythmias may arise from the sinoatrial region and from working atrial myocardium or the AV junction. Reentrant rhythms may arise in these structures as well and may also involve accessory AV connections or other variants in the preexcitation syndrome. Table 14-1 provides a tabulation of PSVT categories by electrophysiological mechanisms and substrate. A summary of the basic concepts involved in the genesis of these arrhythmias is provided in the following discussion.

Basic Electrophysiology

The anatomic and electrophysiological substrate and physiology of SVTs have been given fresh investigative impetus by the evolution of catheter ablation procedures. Based on anatomic dissections and pathologic study of involved hearts, the accessory bypass tract has been recognized as working myocardial bundles that may cross the AV annulus at any location or bridge elements of the specialized conduction system with atrial or ventricular working myocardium. The reentrant circuit in AVRTs has four components—the atrium, normal AV
node, ventricle, and accessory pathway. In some instances, the tachycardia is located in the atrium or AV node and simply uses the accessory pathway for conduction to the ventricles, the so-called bystander pathway. In contrast, AV nodal pathways are believed to be located within or in the immediate environs of the AV node. This latter structure has been stratified into transitional regions with the atrium (AN region), compact node (N region), and the His Bundle (NH region). The N region is synonymous with the anatomic descriptions of the compact AV node. The reentrant circuit in AV nodal reentry is confined to the AV node and adjoining atrial myocardium. A concise discussion of these two substrates and their physiology is used as the basic structure to define specific variants of these arrhythmias.

ATRIOVENTRICULAR NODAL REENTRANT TACHYCARDIAS

Pioneering anatomic studies of Tawara and Kent and physiologic demonstration of dual AV nodal physiology in dogs by Moe have laid the foundation for our current understanding of this condition. Tawara and others examined the compact AV node, but more recent studies have focused on the connections of the node. Moe and coworkers first suggested the presence of functionally and spatially distinct pathways with fast and slow conduction properties in the canine AV node. Subsequently, cellular electrophysiological properties of the node and its environs were studied (see Chapter 13). The AN and N regions lie in the triangle of Koch framed by Todaro’s tendon, the tricuspid annulus, and the ostium of the coronary sinus. This anatomic location is critical to understanding the technique of catheter ablation of this arrhythmia. The N region is located at the apex of the triangle where the NH region in the nonbranching part of the AV bundle penetrates the central fibrous body to become the His bundle. Considerable uncertainty existed in our understanding of the AN region and the atrial connections of the AV node. Truex and Smythe demonstrated atrial extensions of the compact AV node, with a prominent posterior tail that extended to the ostium of the coronary sinus. Anderson and coworkers defined superficial, deep, and posterior zones in these connections. The superficial zone extended into the anterior and superior part of the node, the posterior zone into the inferior and posterior aspect of the compact node, and the deep fibers connected the left atrial septum to the node. This latter connection was noted by Anderson to join the distal part of the node, suggesting the possibility of a lesser degree of AV nodal conduction modulation.

### TABLE 14-1 Classification of Regular Supraventricular Tachycardias with Narrow QRS Complexes

<table>
<thead>
<tr>
<th>Sinus Node Disorders</th>
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<tbody>
<tr>
<td>Paroxysmal sinus tachycardia</td>
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<tr>
<td>Nonparoxysmal sinoatrial tachycardia</td>
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<tr>
<td>Atrioventricular (AV) Nodal Reentrant Tachycardias</td>
<td></td>
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<tr>
<td>Slow-fast (type 1)</td>
<td></td>
</tr>
<tr>
<td>Fast-slow (type 2)</td>
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<tr>
<td>Slow-slow (type 3)</td>
<td></td>
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<tr>
<td>Reentrant and Ectopic Atrial Tachycardias</td>
<td></td>
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<tr>
<td>Intra-atrial reentrant tachycardia</td>
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<tr>
<td>Automatic atrial tachycardia (unifocal or multifocal)</td>
<td></td>
</tr>
<tr>
<td>Preexcitation Syndrome: Wolff-Parkinson-White Syndrome</td>
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<tr>
<td>Orthodromic atrioventricular reentry</td>
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<tr>
<td>Permanent junctional reciprocating tachycardia</td>
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</tr>
<tr>
<td>Antidromic atrioventricular reentry</td>
<td></td>
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<tr>
<td>Atrial tachycardia, atrial flutter, or atrial fibrillation with or without accessory pathway conduction</td>
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<tr>
<td>Other Preexcitation Syndromes: Mahaim Conduction</td>
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<tr>
<td>Nodoventricular and nodofascicular reentry</td>
<td></td>
</tr>
<tr>
<td>Atrial tachycardia, AV nodal reentry, or atrial fibrillation with nodoventricular or nodofascicular bystander conduction</td>
<td></td>
</tr>
<tr>
<td>Other Preexcitation Syndromes: Lown-Ganong-Levine syndrome</td>
<td></td>
</tr>
<tr>
<td>Atrial tachycardia, atrial flutter, or atrial fibrillation with enhanced AV nodal conduction</td>
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<tr>
<td>Automatic AV Junctional Tachycardias</td>
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by slow currents and more rapid AV conduction. Definition of these three inputs further refined our clinical techniques of AV nodal modification or ablation. Racker also described discrete superior, medial, and lateral atrionodal bundles. The medial and lateral bundles converged into a single proximal AV bundle leading into the compact AV node. These two bundles are believed to form in part or in entirety the anterior “fast” and posterior “slow” AV nodal physiologic pathways.

Moe and colleagues demonstrated the electrophysiological substrate for AV nodal reentrant tachycardias (AVNRTs) by developing the concept of dual AV nodal pathways as its basis. They noted a marked prolongation of AV nodal conduction when a premature atrial extrastimulus was delivered at a critical coupling interval. This was often associated with an echo beat, postulated to be due to two AV nodal pathways—a β-pathway with a faster conduction and long refractoriness and an α-pathway with shorter refractoriness and slower conduction times. At critical coupling intervals, the premature beat shifts conduction from the β-to the α-pathway and reengages the β-pathway retrogradely to result in an atrial echo beat. More recent, elegant, isolated canine and pig hearts, McGuire, de Bakker, and Janse have shown nodal type action potentials in cells around both mitral and tricuspid valve rings. These cells are separated from atrial cells by a zone of cells with intermediate action potentials. Adenosine reduced amplitude and upstroke velocity of action potentials in nodal-type cells, but their morphologic characteristics were indistinguishable from atrial myocytes. However, they could be distinguished by the absence of connexin 43, which was present in atrium and ventricle. The posterior AV nodal approaches were dissociated by pacing from the atrium and AV node and echo beats used the slow posterior pathway retrogradely and preceded atrial activation. These posterior approaches were not used in fast pathway conduction.

**HUMAN CORRELATES OF EXPERIMENTAL OBSERVATIONS**

Clinical correlation of these experimental concepts of AV nodal physiology was demonstrated by the early work of Bigger and Goldreyer, as well as Denes, Rosen, and coworkers. The critical role of AV nodal conduction delay in the onset of SVT was recognized. Denes et al. demonstrated longitudinal dissociation of the AV nodal conduction in patients with recurrent SVT. The critical link between anatomic and physiologic concepts in humans was provided by a landmark study by Sung et al. They demonstrated the posterior input and output of the “slow” AV nodal pathway and the anterior location of the “fast” AV nodal conduction pathway. Submerged in controversy for some time, this observation formed the basis for the development of fast and slow pathway ablation for the cure of AV nodal reentry. Several years later, this study was directly validated in intraoperative studies in humans. McGuire and coworkers mapped Koch’s triangle to define the earliest atrial activation during AVNRT in patients during cardiac surgery. A zone of slow conduction was found in the triangle in 64% of patients. Atrial activation patterns confirmed that the fast pathway was connecting at the apex of the triangle near the AV node–His bundle junction, while the slow pathway did so at the orifice of the coronary sinus near the posterior aspect of the AV node. Two types of AVNRT were distinguished: (1) the common variety or type 1, called the “slow-fast” form, which used the slow pathway for anterograde propagation and the fast pathway for retrograde conduction and (2) the uncommon type or type 2, called “fast-slow” AVNRT. Contiguous ablation lesions can affect both pathways, confirming their proximity. The presence of multiple “slow” pathways has also been identified, resulting in a third form of reentry, called “slow-slow” or type 3 AVNRT. These different types and pathways support the concept that these arrhythmias are supported by AV nodal tissue, transitional atrio-nodal inputs, and other perinodal tissues, which have varying electrophysiological properties and functionally simulate distinct electrical pathways.

**ATRIOVENTRICULAR REENTRANT TACHYCARDIAS**

The classification of preexcitation syndromes has evolved since the original eponyms were given to Kent, Mahaim, and James fibers. The long proposed and accepted classification by the European Study Group is shown in Table 14-2. However, limitations of this classification are being recognized, and correlation with the previous eponyms remains occasionally tangential. The major reason for this observation is the recognition that decremental conduction is a property not solely confined to the AV node but also seen with accessory AV connections. Speculation around the embryologic basis of these connections swirls around the original suggestion by Gallagher that these may be displaced AV nodal and specialized conduction system tissues. Decremental conduction has been observed in posteroseptal AV connections in the permanent form of junctional reciprocating tachycardia, as well as in atriofascicular bypass tracts, which commonly link the parietal atrial myocardium in the right atrium to the right bundle branch at its distal portion and are detected as Mahaim physiology. The tachycardia propagates antegrade over the atriofascicular pathway and retrogradely over the normal AV conduction system and involves the atrium and ventricle as critical elements in the circuit.

**TABLE 14-2 Classification of Preexcitation Syndromes**

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<tbody>
<tr>
<td>1.</td>
<td>Atrioventricular (AV) bypass tracts providing direct connections between the atrium and ventricle</td>
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<tr>
<td>2.</td>
<td>Nodoventricular connections between the AV node and ventricular myocardium</td>
</tr>
<tr>
<td>3.</td>
<td>Fasciculoventricular connections between the fascicles of the specialized conduction system and the ventricular myocardium</td>
</tr>
<tr>
<td>4.</td>
<td>AV nodal bypass tracts with direct connections between the atrium and His bundle</td>
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</table>
This is quite distinct from the nodoventricular connections originally described by Mahaim. In this latter instance, the tachycardia has a similar propagation sequence but can exist totally without atrial involvement and has been referred to as a “subjunctional tachycardia.”

The anatomic basis for AVRT is the accessory AV pathway, a small band of working myocardium bridging the AV annulus. The location of these pathways is most frequent in the left ventricular free wall, followed by posteroseptal and paraseptal locations and, least commonly, in the right atrial free wall and left anterior AV annulus. Multiple AV connections are seen in approximately 15% of patients. Typical dimensions for these locations and such connections have been elucidated by pathologic studies. In cadaver hearts the posterior septal space was noted to extend from the coronary sinus orifice for 2.3 ± 0.4 cm, and the length of the left ventricular free wall was 5 ± 1 cm. Posteroseptal accessory pathways were located in the proximal 1.5 cm of the coronary sinus in the posterior septum and those between 1.5 and 3 cm could be either in the left free wall or the posterior septal space. Posteroseptal accessory pathways beyond 3 cm were invariably in the free wall.

**BASIC ELECTROPHYSIOLOGICAL CONCEPTS IN PREEXCITATION SYNDROMES**

AV accessory pathways, as well as AV nodal bypass tracts, generally exhibit “all or none” conduction behavior during electrophysiological evaluation. Rapid non-decremental conduction up to the point of refractoriness is the norm and is exhibited during antegrade and retrograde conduction, especially when competing AV nodal conduction is absent. When decremental conduction in response to progressive premature stimulation is observed, it is usually due to switching of conduction to the AV nodal-His axis, though enhanced AV nodal conduction may occur in the same patient. When the accessory pathway conducts antegrade, the most common manifestation is WPW syndrome with a short P–R interval and δ wave (due to ventricular preexcitation by the pathway) and prolongation of QRS duration (due to abnormal intraventricular conduction patterns). In some instances, when the antegrade refractoriness of the accessory pathway is particularly long or if the pathway fails to permit such conduction, it may remain concealed and only manifest when retrograde propagation occurs over echo beats or AVRT. Patients with concealed accessory pathways have normal surface ECGs. Rarely, decremental conduction is observed in the anomalous pathway. While this is occasionally due to nodoventricular connections, more often this is due to slowly propagating accessory pathways or other causes of Mahaim physiology. The electrophysiological basis for decremental conduction in accessory pathways has been speculative. Suggestions include impedance mismatch at the interface between the pathway and atrial or ventricular myocardium or extreme tortuosity of these fibers in some instances, which has been seen pathologically. Investigations suggest that block in such pathways usually occurs at the ventricular connection and may be related to anisotropy, fiber narrowing, or altered intercellular junctions. Age and autonomic state–related changes in electrophysiological properties may lead to intermittent manifestations of preexcitation syndromes and the arrhythmias supported by them in a patient’s lifetime. It is common for incessant tachycardias and manifest preexcitation of infancy to wane during childhood and adolescence or for there to be an initial appearance of the condition in young adults. Aging generally can impair pathway function, and it is uncommon, though not rare, for the condition to manifest itself for the first time in an elderly patient.

**SUPRAVENTRICULAR TACHYCARDIAS DUE TO ENHANCED AUTOMATICITY**

Accelerated junctional rhythms manifest as supraventricular tachyarrhythmias have been recorded with particular frequency in the era of extensive digitalis use without blood concentration determinations. They have been noted in postoperative patients, after myocardial infarction and during electrolyte abnormalities. These arrhythmias are believed to be due to triggered automaticity resulting from delayed afterdepolarizations in most instances. Pacing can enhance the afterdepolarizations and they can be increased in amplitude by increase in extracellular calcium concentrations, which can be seen in digitalis toxicity. Ischemia in experimental models can also result in accelerated, triggered automaticity in the coronary sinus region. Hypokalemia can predispose to afterdepolarizations. Acceleration is often seen in triggered rhythms at initiation and can clinically manifest as nonparoxysmal junctional tachycardias on the ECG. The triggered arrhythmias can often be induced by overdrive pacing and do not necessarily resume after pacing ceases, unlike other automatic rhythms. Triggered rhythms have also been induced in human diseased atrial tissues resected at surgery and in coronary sinus cells during experimental studies.

**Clinical Presentation**

PSVT presents most commonly as sudden onset of palpitations and may be associated with chest discomfort, dyspnea, near syncope, and syncope. It is of variable duration and may last from seconds to days. Termination is usually sudden as well, though in some forms gradual disappearance may be noted. Chest discomfort in children and adults without overt heart disease may be related to the perception of rapid heart action; after an episode, it may persist in a milder form for a period of time. In older patients and in the presence of heart disease, this may be related to myocardial ischemia. Dyspnea may be a prominent symptom, often in patients with preexisting left ventricular dysfunction, when pulmonary congestion may worsen due to poor forward cardiac output. Symptoms suggestive of near syncope and syncope are seen with extremely rapid
PSVT and result from a compromise of cardiac output. In many forms of PSVT, atrial and ventricular activation are not timed sequentially to achieve appropriate ventricular filling. Rapid rates further compromise this, and forward cardiac output can be seriously compromised, especially with heart rates greater than 200 bpm. In some patients, PSVT can be minimally symptomatic or even asymptomatic. In children and infants, incessant PSVT can lead to a tachycardia-induced cardiomyopathy with symptoms of left ventricular failure, failure to thrive, and syncope. Very rapid or hemodynamically unstable episodes of this arrhythmia have been known to precipitate myocardial infarction, ventricular tachyarrhythmias, and cardiac arrest.

**Electrocardiography**

The distinguishing electrocardiographic features of PSVT reflect the underlying mechanism of the arrhythmia. The major criteria that have been used to separate these mechanisms depend on the features of onset, the position of the P wave in the R–R interval during SVT, the presence or absence of QRS alternans, cycle length variation, the P-wave morphology, effects of bundle branch block (BBB), the presence of a pseudo R' deflection in V1, and whether there is ventricular preexcitation. Some of these criteria have proven to be particularly useful, but there is considerable overlap among the ECG manifestations of the different mechanisms of SVT.

1. **Onset.**

   AVNRT is usually triggered by a premature atrial beat that differs in morphology from sinus rhythm. As shown in Figure 14-4, initiation of the tachycardia is characterized by sudden prolongation of the P–R interval because conduction from the premature beat blocks in the fast pathway and conducts down the slow pathway. Reentry occurs if the fast pathway has recovered and is capable of conducting retrograde. In contrast, while a triggered or reentrant atrial tachycardia may be initiated by an APD, these arrhythmias are not heralded by marked P-R prolongation with the onset of tachycardia. Automatic atrial tachycardias are characterized by gradual acceleration and a P wave morphology that differs from sinus rhythm. AVRT mediated by accessory pathways may be triggered by either premature atrial or ventricular beats. Ventricular ectopy can induce AVRT or atrial tachycardias, but it is a far less common mode of induction for these arrhythmias.

2. **Position of the P wave.**

   During typical AVNRT the atria are generally activated simultaneously with the ventricles, so the P wave is buried within the QRS complex, though in some cases it may extend into the early portion of the ST segment (Fig. 14-5A). In the atypical form of AVNRT, during which antegrade conduction is mediated by the fast pathway and retrograde conduction by the slow pathway, the P wave may fall into the second half of the R–R interval because of slow retrograde conduction to the atria. During orthodromic AVRT mediated by an accessory pathway, retrograde atrial activation generally begins about 70 milliseconds (ms) after the onset of the surface QRS and extends well into the ST segment in the first half of the R–R interval (see Fig. 14-5B). In patients with atrial tachycardias the P wave is usually detected in the second half of the R–R interval. The exception is patients with atrial tachycardias in whom AV conduction is delayed because of the effects of antiarrhythmic drugs or intrinsic conduction system disease. Kalbfleisch evaluated ECGs in patients who had undergone electrophysiology studies and found that the P wave was in the first half of the R–R interval in 91% of patients with AVNRT, 87% of patients with AVRT, and 11% of patients with atrial tachycardias. Green observed that ECGs recorded from some patients with SVT exhibited QRS alternans and, when present, it was usually associated with AVRT mediated by an accessory pathway. Subsequent studies by Kay and Kalbfleisch demonstrated that QRS alternans depends on the abrupt onset of SVT and is more common in rapid tachycardias. In their studies, the incidence of QRS alternans was 27% to 38% in orthodromic AVRT and 13% to 23% in patients with AVNRT. It was much less common in patients with atrial tachycardias. The differences between the results of these studies may reflect criteria used to identify QRS alternans and the number of leads used for the recordings. For example, the alteration in QRS amplitude may only be apparent in selected leads. Recordings obtained from telemetric monitoring or using

![Figure 14-4](image-url) Sinus rhythm is interrupted by APDs that have a different P-wave morphology. The second APD blocks in the fast pathway and conducts down the slow pathway to induce atrio-ventricular node reentry. P-waves are not evident during the tachycardia because they are buried in the QRS complex.
FIGURE 14-5  A, Narrow complex supraventricular tachycardia (SVT) at a rate of 240 beats per minute (bpm) with no retrograde or antegrade P wave being visible in the RR cycle. The tachycardia was subsequently confirmed to be type 1 atrioventricular nodal reentrant tachycardia on electrophysiological study. B, Narrow complex SVT at a rate of 206 bpm with the retrograde P wave being clearly visible in the mid-RR cycle, especially in lead V1. The tachycardia was subsequently confirmed to be AVRT with a retrogradely conducting posteroseptal accessory pathway on electrophysiological study.
only a limited number of surface leads may not demonstrate QRS alternans quite as well as a 12-lead ECG.\textsuperscript{48}

4. **Rate and Cycle Length Alternations.**

Several studies have evaluated the rate of SVT without demonstrating significant differences that would be useful to discriminate the underlying mechanism.\textsuperscript{44-46} Cycle length variation is relatively uncommon in the reentrant tachycardias. One would expect reentrant tachycardias such as AVRT and AVNRT to have relatively constant cycle lengths as they usually do, but sometimes variable conduction in one limb of the circuit may lead to variations in cycle length even during reentry. Figure 14-6 shows unusual and striking variation in the R–R intervals recorded during orthodromic AVRT mediated by a left lateral accessory pathway in a patient who also had dual AV node physiology. The variation in R–R interval is attributable to differences in conduction through the AV node depending on whether antegrade conduction occurred over the fast or slow pathway.

5. **P wave Morphology.**

When a P wave can be identified during SVT, it is often difficult to determine the morphology and axis because it may be obscured by ventricular repolarization. Kalbfleisch reported that when the P wave was visible, its axis could be determined in the vertical, horizontal, and anterior-posterior planes in only 32%, 11%, and 9% of patients.\textsuperscript{43} One might expect the P wave morphology to be more accurately analyzed during atrial tachycardias, because the P wave falls in the second half of the R–R interval and is less likely to be obscured by the T wave. Tang developed an algorithm to differentiate left atrial from right atrial focal tachycardias based on surface ECGs recorded from patients who underwent ablation procedures.\textsuperscript{49} They found that negative P waves in the inferior leads differentiated inferior from superior foci, and a negative P wave in aVR was characteristic of tachycardias arising along the crista terminalis. All these investigations are limited in that the number of patients studied is relatively small, and prospective analysis of P wave morphology is often hindered by inability to accurately separate the terminal part of the T wave and P wave during the tachycardia.

6. **Effect of BBB.**

The development of BBB during SVT may provide a clue to the diagnosis of AVRT. Coumel and colleagues first observed that the development of BBB ipsilateral to an accessory pathway may result in cycle length prolongation (decrease in rate) because the circuit is prolonged.\textsuperscript{52,53} Figure 14-7 was recorded during transition from SVT with left

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**FIGURE 14-6** Surface leads I, aVF, and V1 demonstrate marked cycle length variability in a patient with a concealed left lateral accessory pathway and dual AV node physiology. The irregular R–R intervals were recorded during orthodromic atrioventricular (AV) reentry and were attributable to intermittent conduction down the slow AV node pathway.
BBB to a narrow QRS from a patient with a left lateral accessory pathway. Concomitantly, the cycle length of the tachycardia decreased by 40 ms (300 to 260 ms), resulting in an increase in the rate from 200 to 230 bpm. The rate of tachycardias dependent on right-sided accessory pathways may decrease with the development of right BBB, but tachycardias dependent on accessory pathways located in the septum do not change rate appreciably with BBB because the circuit is not significantly prolonged. Even when BBB develops on the same side as the accessory pathway, the rate does not invariably change because the increase in ventriculo-atrial conduction time may be associated with shortening of antegrade conduction time in the AV node, so that the net effects on the tachycardia cycle length are negated.

7. **Pseudo R’ in V1.**

The development of a pseudo R’ in V1 is observed more frequently in AVNRT than in either AVRT or atrial tachycardias. Although this is not a particularly sensitive criterion (58%), it is relatively specific for AVNRT (91%). It is attributable to distortion of the terminal portion of the QRS by a retrograde P wave.

8. **Preexcited Tachycardias.**

Tachycardias associated with ventricular preexcitation may be attributable to antidromic AVRT, atrial fibrillation or atrial tachycardias that conduct over an accessory pathway, or tachycardias mediated by a Mahaim fiber. As shown in Figure 14-8, maximal preexcitation is present during antidromic AVRT because ventricular activation occurs exclusively through the accessory pathway. ECGs recorded during antidromic SVT show a regular, wide, monomorphic QRS complex that resembles ventricular tachycardia. Retrograde P waves may be detectable during the first half of the R–R interval, but they are extremely difficult to appreciate because they are obscured by the marked repolarization abnormality associated with preexcited complexes. When evident, P waves have a 1:1 relationship with the QRS because block of conduction in either the accessory pathway or AV node would terminate the tachycardia. Sometimes sudden changes in cycle length are observed during antidromic AVRT that reflects which bundle is used during retrograde conduction. During atrial fibrillation with ventricular preexcitation, the ventricular rate may be rapid, the R–R intervals are irregular, and the QRS morphology varies depending on the degree to which the ventricle is activated by the accessory pathway or the AV node. The irregular rate and QRS morphology differentiate this arrhythmia from antidromic AVRT or monomorphic ventricular tachycardia. Patients with preexcited R–R intervals shorter than 220 to 250 ms have accessory pathways with short refractory properties and are at increased risk that atrial fibrillation could induce ventricular fibrillation because of rapid conduction over the accessory pathway.

Tachycardias that are mediated by Mahaim fibers, shown in Figure 14-9, exhibit a QRS morphology that resembles left BBB. The QRS axis is typically between 0 and −75 degrees; QRS duration is 0.15 seconds or less; an R wave is present in limb lead I; an rS is seen in precordial lead V1; and there is transition in the precordial leads from a predominantly negative to positive QRS complex in leads V4 to V6.

9. **Patterns of Ventricular Preexcitation.**

The ECG pattern of ventricular preexcitation provides useful information about the location of the accessory pathway and whether more than one pathway may be present. Several authors have studied the ECG manifestations of preexcitation and have developed criteria for localization of the
The accuracy of these methods depends on the degree of ventricular preexcitation at the time of the recording and whether there is underlying heart disease that modifies the usual patterns of preexcitation. These criteria have evolved over time as additional experience has been acquired. They are based on the concept that δ wave polarity, QRS axis, and R wave transition in the precordial leads reflect the position of the accessory pathway. When preexcitation is pronounced, left-sided pathways have a prominent R wave in V1 with a positive δ wave. If the pathway is located on the posterior aspect of the mitral annulus, the polarity of the δ wave is generally negative in the inferior leads, and a QS complex is present. Pathways that are located more anterior-laterally on the mitral annulus have negative δ waves and QS morphology in aVL, but...
the δ waves are positive in the inferior leads. Posterior septal pathways have negative δ waves in the inferior leads, but the R/S ratio is less than 1 in V1. There is an abrupt transition in to R/S ratio greater than 1 in V2. Arruda found that subepicardial accessory pathways characteristically have clear negative δ waves in lead II in the first 20 ms after the onset of the δ wave. Accessory pathways located on the right side exhibit an R/S less than 1 in V1 and have delayed δ wave progression. The polarity of the δ waves in the inferior limb leads is negative if the pathway is posterior. Positive δ waves in the inferior leads suggest a more anterior position. Pathways located in the middle to anterior septum have positive δ waves in the inferior leads and may have negative δ waves in V1. They are distinguished by the R/S ratio greater than 1 in lead III with anteroseptal pathways and R/S equal to 1 with midseptal pathways.

The assessment of accessory pathway locations is difficult when preexcitation is not pronounced. In one of the larger series, Chiang evaluated a stepwise algorithm that was based on the R/S ratio in V2, the δ wave polarity in lead III (initial 40 ms), the δ wave polarity in V1 (initial 60 ms), and the δ wave polarity in aVF (initial 40 ms). The algorithm correctly predicted the location of the accessory pathway in 93% of the patients. Arruda developed an algorithm base on 135 consecutive patients and prospectively applied it to 121 consecutive patients with an overall sensitivity of 90% and specificity of 99%. Their criteria were based on the initial 20 ms of the δ wave in leads I, II, aVF, and the R/S ratios in III and V1. The algorithm they employed is shown in Table 14-3. The presence of multiple accessory pathways may be recognized by variable patterns of preexcitation with characteristics of more than one pathway or hybrid patterns that do not fit the usual algorithms. Fananapazir found that in patients with multiple pathways, more than one pattern of preexcitation was apparent in only 32% of recordings made during sinus rhythm, but the presence of more than one pathway was recognized in 55% of recordings obtained during atrial fibrillation.

Diagnostic Approach to the Patient with Supraventricular Tachycardia

Investigation of SVT is based on understanding the underlying mechanism of the tachycardia and clinical context in which it occurs. A systematic approach should start with the history and physical examination, which provides two types of information: (1) the presence and type of symptoms and (2) the clinical context, particularly the existence of associated heart disease. Electrocardiographic documentation of the arrhythmia is an essential prerequisite to tachycardia management.

CLINICAL EVALUATION

Careful history taking may provide valuable information. SVT may be symptomatic or asymptomatic. Palpitations due to tachycardia are the most common symptom.
Episodes of palpitations with abrupt onset and termination, followed by polyuria, suggest PSVT. This syndrome, known in French literature as “syndrome de Bouveret,” was described in 1888, before the advent of electrocardiography. It is characteristic of paroxysmal junctional tachycardia but may be found in other types of supraventricular or ventricular tachycardias, such as verapamil-sensitive ventricular tachycardia. Occasionally, SVT may be the cause of syncope. A history of palpitations preceding syncope is often reported in this presentation. When syncope or presyncope occurs immediately after termination of fast palpitations, the tachycardia-bradycardia syndrome should be suspected. Other symptoms may be associated with SVT and are indicative of poor tolerance (e.g., syncope, dizzy spells, chest discomfort, dyspnea, or even pulmonary edema). SVT may be mildly symptomatic or asymptomatic and discovered incidentally on recordings performed for another reason. Sometimes, the arrhythmia is specifically suspected due to its complications (e.g., asymptomatic paroxysmal atrial fibrillation in a patient with a cerebrovascular accident suspected to be of embolic origin). Physical examination is focused on any associated heart disease but, in general, paroxysmal SVT occurs in patients without organic heart disease. In contrast, heart disease is present in 70% of patients with atrial fibrillation.

**DIFFERENTIAL DIAGNOSIS OF SUPRAVENTRICULAR TACHYCARDIA FROM THE ECG (Table 14-4)**

Electrocardiographic documentation of the tachycardia is essential for the proper diagnosis and management of SVT. Recording of the tachycardia episode is easily obtained when tachycardia occurs frequently or is of prolonged duration. SVT by definition arises above the bifurcation of the His bundle, either in the atria or the AV junction and, therefore, is generally associated with narrow QRS complexes. SVT may sometimes present with wide QRS complexes either because the patient had a preexisting BBB or because aberrant conduction is present. Differentiating SVT from ventricular tachycardia may, at times, be difficult, particularly when preexcitation is present.

a. **Tachycardia with narrow QRS complexes** (Table 14-5). Most regular SVTs use the AV node either passively as in atrial tachycardias and atrial flutter or as a critical component of the circuit as in PSVT. The diagnostic approach to tachycardias with narrow QRS complexes should be undertaken in a stepwise fashion.66-68

The first step is to assess the regularity of the R-R interval.
1. If the R–R interval is irregular, atrial fibrillation or atrial flutter should be considered as the most likely diagnosis. However, when atrial fibrillation is associated with rapid ventricular response, it may seem regular. Permanent junctional reciprocating tachycardia (PJRT) is an SVT in which the R–R interval is often irregular. This tachycardia described by Coumel et al. was found to be related to a concealed accessory connection capable of decremental conduction. The tachycardia uses the AV node in the antegrade conduction and a slow conducting accessory connection in the retrograde direction (Fig. 14-10). This tachycardia may be incessant, accounting for the descriptive term “permanent” in its title. It becomes sustained in clinical situations with increased catecholamine output such as exercise or emotion. It warrants therapy as it may have a deleterious effect on cardiac function in addition to tachycardias related symptom relief. The differential diagnoses include atypical AVNRT using the fast pathway in the antegrade direction and the slow pathway in the retrograde direction, and atrial tachycardia arising from the inferior atrium near the coronary sinus ostium.

2. When the R–R intervals have been assessed to be regular, the next step is also to look for the P waves (“cherchez le P”) as advocated by Marriott. The presence, morphology, and position as to the QRS complexes are important in the diagnosis of the site of origin and for the suspected mechanism of narrow QRS complex tachycardias. When the QRS complexes are preceded by P waves, which are different in configuration from the sinus P waves and conducted with a P–R interval equal or longer than the P–R interval of the sinus P waves, the most likely diagnosis is atrial tachycardia arising from an ectopic focus. The other mechanism of this type of SVT is intra-atrial or sinoatrial (SA) reentrant tachycardia, a diagnosis that requires an electrophysiological study for substantiation. If the P waves have the same configuration as the sinus P waves, the differential diagnosis includes appropriate or “inappropriate” sinus nodal tachycardia. Inappropriate sinus node tachycardia is a rare arrhythmia that has been recognized recently. This atrial tachycardia is characterized by an inappropriate and exaggerated acceleration of heart rate during physiologic stresses. While its mechanism remains speculative, there are a number of possible hypotheses as to its basis. These include an ectopic atrial focus located in the SA node area, a normal SA node with increased response to the sympathetic tone or failure to respond to vagal stimulation, and an intrinsic anomaly of the SA node. When the P waves are submerged within the QRS complex in SVT and are therefore not identifiable, the most likely diagnosis is type 1 AVNRT (i.e., tachycardia involving the AV node and

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TABLE 14-4 Classification of Supraventricular Tachycardias

<table>
<thead>
<tr>
<th>Atrial Origin</th>
<th>AV Junction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrial fibrillation</td>
<td>Atrioventricular nodal reentrant tachycardia (AVNRT)</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>Common type: slow pathway anterograde–fast pathway retrograde</td>
</tr>
<tr>
<td>Atrial tachycardia</td>
<td>Uncommon type: fast pathway anterograde–slow pathway retrograde</td>
</tr>
<tr>
<td>Enhanced automaticity: atrial focus</td>
<td>“Slow-slow”: slow pathway anterograde–slow pathway retrograde</td>
</tr>
<tr>
<td>Reentry: atrial or sinoatrial</td>
<td>Atrioventricular reentrant tachycardia (AVRT)</td>
</tr>
<tr>
<td>PJRT</td>
<td>Prereciliation may be overt or concealed on ECG in sinus rhythm (see text)</td>
</tr>
<tr>
<td>AV junction anterograde–accessory pathway retrograde</td>
<td></td>
</tr>
</tbody>
</table>

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TABLE 14-5 Diagnostic Algorithm for Narrow QRS Complex Tachycardia

<table>
<thead>
<tr>
<th>R–R interval irregular</th>
<th>R–R interval regular</th>
</tr>
</thead>
<tbody>
<tr>
<td>Look for the P wave</td>
<td>Look for the P wave</td>
</tr>
<tr>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>F waves 250–350</td>
<td>Yes Ectopic</td>
</tr>
<tr>
<td>Atrial fibrillation (AF)</td>
<td>AVNRT</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>Atrial flutter</td>
</tr>
<tr>
<td>Atrial tachycardia</td>
<td>Rapid AF</td>
</tr>
<tr>
<td>No</td>
<td>Yes PR&gt;RP</td>
</tr>
<tr>
<td>Yes</td>
<td>Atrial tachycardia</td>
</tr>
<tr>
<td>AVRT (WPW)</td>
<td>PJRT</td>
</tr>
<tr>
<td>Yes PR&lt;RP</td>
<td>Atypical AVNRT</td>
</tr>
</tbody>
</table>
using in the typical form a slow pathway in the anterograde direction and a fast pathway in the retrograde direction, resulting in a P wave within or immediately after the QRS complex.\textsuperscript{71,72} Wellens has described an electrocardiographic sign, which is suggestive of AVNRT. It consists of an incomplete BBB pattern (RSR') in lead V1 during the tachycardia that is not present in the 12-lead ECG in sinus rhythm.\textsuperscript{72} However, atrial flutter with 2:1 conduction should be suspected as a differential diagnosis if the ventricular rate of the SVT with narrow QRS complexes is around 150 bpm. If the P waves during SVT have been identified and follow the QRS complexes at a significant interval resulting in an R–P interval equal to or greater than the P–R interval, the most likely diagnosis is orthodromic AVRT (i.e., involving the AV node in the antegrade direction and an accessory AV pathway in the retrograde direction). Other helpful electrocardiographic clues that have been reported and shed light on the mechanism of SVT include a negative P wave in leads I and V1, due to left-to-right atrial activation in AVRT using a left-sided AV connection in its retrograde limb.\textsuperscript{72} When two recordings of the tachycardia are available, one with narrow QRS complexes and the other with left BBB with a longer cycle length, a finding first described in Paris, the diagnosis of AVRT involving a left-sided accessory connection can be made.\textsuperscript{52,73} The presence of QRS alternation is also in favor of AVRT.

The differential diagnosis between PSVT and atrial fibrillation with rapid ventricular response or atrial flutter with 1:1 conduction may at times be difficult. Vagal maneuvers, particularly carotid sinus massage and adenosine injection, may be of great help in clinical diagnosis (Fig. 14-11). In AVRT or AVNRT, the arrhythmia may terminate abruptly or remain unaffected. In contrast, atrial fibrillation or flutter is rarely terminated by these techniques but can be slowed in its ventricular response.

FIGURE 14-10 Twelve-lead ECG of a tachycardia related to a slow conducting accessory pathway used in the retrograde direction. Note in the left panel that the R–R intervals are slightly irregular as the pathway is capable of decremental conduction.

FIGURE 14-11 Termination of narrow QRS complex tachycardia with carotid sinus massage (CSM).
thus exposing the underlying atrial flutter or fibrillation waves. PSVT and atrial flutter/AF may also coexist in the same patient. Adenosine administration may also be used as a diagnostic test to assess dual AV nodal pathway conduction, efficacy of slow pathway ablation, and detection of concealed accessory pathways. 74

b. **Tachycardia with regular wide QRS complex** (Table 14-6). Regular SVT may present with wide (>0.12 sec) QRS complex, and differentiating SVT from VT may be difficult. Another etiology of wide QRS complex tachycardia is the preexcitation syndrome, which may be overt (WPW syndrome) or concealed (with the pathway only conducting in the retrograde direction), and this is discussed later. It bears re-emphasizing that the vast majority of regular SVTs occur in patients without organic heart disease in contrast to patients with VT. Three clues may be used in the differentiation diagnosis of wide QRS tachycardias.

1. The age of the patient; in the child and young adult, preexcitation is more common than ventricular tachycardia. In this instance the ECG in sinus rhythm, if available, shows preexcitation, and the ECG during the tachycardias shows an identical morphology.

2. In making this distinction in SVT diagnosis, it is necessary to locate the P wave during tachycardias. The presence of AV dissociation is generally diagnostic of ventricular tachycardia. However, it is only present in about 40% of ventricular tachycardias. Differentiating VT from SVT may require an electrophysiological study with endocardial recordings. 66,75-79

3. Wellens et al. have described a number of criteria that may be of help differentiating SVT from VT. 79 A few that we have found particularly useful include left axis deviation (beyond −30 degrees), QRS complexes wider than 0.14 sec, and the QRS morphology in V1 and V6 (i.e., a monophasic R wave or a QR pattern in V1, or an rS or a QS pattern in V6), all of which favor a diagnosis of VT. Another clue that might be helpful is the presence of BBB in sinus rhythm. An identical morphology during the tachycardia is suggestive of SVT as well. When the QRS complex is narrow in sinus rhythm and wide during SVT, aberrant conduction is also a consideration. We have found it practical to consider aberrancy only when the typical pattern of right or left BBB is present during tachycardia. This still does not exclude the possible diagnosis of VT. For these reasons, it is a wise rule "to consider any tachycardia with wide QRS complexes as being VT unless proven otherwise" (Agustin Castellanos Jr.). Detailed electrophysiological studies are indicated in all patients with wide QRS complex tachycardias.

c. **Tachycardia with preexcited QRS complexes** (Fig. 14-12). The differential diagnosis of wide QRS complex tachycardia may be extremely difficult if preexcitation is present. Fortunately, tachycardias with preexcited QRS complexes represent less than 5% of all wide QRS complex tachycardias. A number of mechanisms may account for a tachycardia with preexcited QRS complexes. The most common is atrial fibrillation with conduction over the Kent bundle. The R–R interval is frequently irregular, and the QRS width may change from one complex to
the other. Atrial flutter with 1:1 conduction over an accessory connection is another possibility; however, this diagnosis should be suspected when the ventricular rate ranges from 250 to 300 bpm. In a young person, a tachycardia with a left BBB and left axis deviation should suggest the possibility of a Mahaim fiber. The other mechanisms may require endocavitary recordings and can only be ascertained by a detailed electrophysiological study.

d. Noninvasive investigations.

As previously mentioned, electrocardiographic documentation is an essential step in the diagnostic approach to SVT. This can be achieved by several methods.

1. Ambulatory ECG Monitoring.

Ambulatory ECG recordings are only warranted when symptoms are frequent enough to allow documentation within the 24- or 48-hour recordings. Otherwise, the information provided by such monitoring is limited to the possible trigger (extrasystoles). Occasionally, monitoring of the tachycardia shows that it is similar to sinus tachycardia, making ambulatory ECG recordings extremely useful in the diagnosis of “inappropriate” sinus node tachycardias in our experience. Chest pain and dyspnea precipitated by the tachyarrhythmia may be the dominant or stand-alone symptoms, and monitoring may be useful in correlating these symptoms to the tachycardia.

2. Event Recorders.

If the episodes of tachycardia are infrequent or of short duration, event recorders are often useful. In recent years, the advances in technology have prompted the development of recorders with transtelephonic transmission and will allow transtelephonic surveillance of selected patients. Daily monitoring and symptomatic transmissions permit the clinician to assess arrhythmia-related symptoms.

3. Telemetric Monitoring.

When the tachycardia is severely symptomatic and occurs frequently enough to be recorded during a hospital stay, telemetric monitoring in the hospital offers another diagnostic option. However, this surveillance requires hospitalization and is associated with a high cost, which often makes this technique impractical for any prolonged period.


Exercise testing is particularly valuable when the tachycardia is precipitated by exercise or is otherwise believed to be catecholamine dependent. For example, atrial tachycardia and atrial fibrillation are not uncommonly induced by exercise. Although junctional tachycardias may occasionally be precipitated by exercise, this test is seldom indicated in patients with paroxysmal SVT. Exercise testing is a valuable tool in patients suspected to have “inappropriate sinus node tachycardia.”

5. Implantable Loop Recorders and Other Devices.

Implantable loop recorders are subcutaneous ECG recording devices without intracardiac electrodes. They have been developed recently for symptom-ECG correlation. Loop recorders, such as the “Reveal Plus” system, have been used recently in syncope and myocardial infarction populations. While such devices can document tachycardias, large-scale study of the yield of such systems in PSVT is unavailable. Furthermore, these devices cannot reliably differentiate VT from SVT. In patients with previously implanted pacemakers or defibrillators, documentation of arrhythmia and its site of origin is generally easy to achieve.

6. Electrophysiological Study (Fig. 14-13).

Electrophysiological studies are used for diagnostic and therapeutic purposes in patients with PSVT. SVT initiation during these
Clinical Electrophysiology

ATRIOVENTRICULAR NODAL REENTRANT TACHYCARDIA

Concept of Dual Atrioventricular Nodal Pathway

During electrophysiological evaluation, typical AVNRT manifests antegrade conduction via a “slow” pathway and retrograde conduction via a “fast” pathway. The electrophysiological characteristic is the presence of dual AV nodal pathway physiology demonstrated by a discontinuous AV node functional curve. In contrast, atypical AVNRT has antegrade conduction through a “fast” or “slow” pathway and retrograde conduction through a “slow” pathway. Discontinuous AV nodal conduction is defined as a sudden increment of 50 ms or greater in A–H or H–A interval (“jump”) with a decrement in prematurity of the extrastimulus by 10 to 20 ms. In some patients a jump (>50 ms) of any consecutive A–H intervals during incremental atrial pacing would be found, which might be a manifestation of dual AV nodal pathways.

Two important concepts of dual AV nodal pathways have been further clarified by provocative pharmacologic testing and catheter ablation in patients with AV nodal reentrant tachycardia. One major question has been whether dual AV nodal pathways are fully intranodal and due to longitudinal dissociation of AV nodal tissue or extranodal involving the separate atrial inputs into the AV node. From clinical studies of slow pathway potentials, LH (low followed by high) frequency potentials are observed during asynchronous activation of muscle bundles above and below the coronary sinus orifice; HL (high followed by low) frequency potentials are caused by asynchronous activation of atrial cells and a band of nodal-type cells that may represent the substrate of the slow pathway. Thus, the slow and fast pathways are likely to be atrionodal approaches or connections rather than discrete intranodal pathways. The results of catheter ablation, indicate that the fast and slow pathways have their origins outside the limits of the compact AV node, and the tissues targeted during successful ablation are composed of ordinary working atrial myocardium surrounding the AV node itself. Furthermore, AV or VA conduction block during AVNRT favors the concept that atrial and ventricular tissue is not involved in maintenance of this tachycardia (Fig. 14-14). The electrophysiological characteristics of the retrograde pathway during the tachycardia differ from those of the anterogradely conducting pathway; these characteristics can be demonstrated by the differential response to atrial or ventricular pacing, and response to antiarrhythmic drugs. For example, procainamide prolongs conduction time in the retrogradely conducting, but not in the anterogradely conducting, pathway. Also, pacing at short cycle lengths prolongs antegrade, but not retrograde, AV nodal conduction time in some patients.

Unusual Physiology of Dual Atrioventricular Nodal Pathways

Some patients with AVNRT have multiple antegrade and retrograde AV nodal pathways with multiple discontinuities in the AV node function curve or dual AV nodal pathways with a continuous curve during programmed electrical stimulation. Furthermore, variant forms (slow-slow, slow-intermediate, fast-intermediate) of AVNRT have been noted (Figs. 14-15 and 14-16). Whether multiple antegrade and retrograde AV nodal pathways originate from anatomically different pathways or represent anisotropic conduction–induced functional pathways is still controversial. Several investigators have demonstrated the marked heterogeneity of the transitional cells surrounding the compact AV nodal pathway. The nonuniform properties of the AV node can produce anisotropic conduction and suggest that the antegrade and retrograde fast pathways are anatomically distant from the multiple “antegrade slow” and “retrograde slow” or intermediate pathways, respectively. Clinical studies have demonstrated that successful ablation or modification of “retrograde slow” and intermediate pathways occurs at different sites from “antegrade fast or slow” pathway, and the possibility of anatomically different antegrade or retrograde multiple pathways should be considered. Furthermore, in the patients who have successful ablation of multiple “antegrade slow” pathways or “retrograde slow” and
intermediate pathways at a single site, anisotropic conduction over the low septal area of the right atrium is a possible explanation for the presence of multiple antegrade and retrograde AV nodal pathways.\textsuperscript{86-88,91}

Patients with AVNRT can have continuous AV node conduction curves. These patients do not exhibit an AH jump using two extrastimuli and two drive cycle lengths during atrial pacing from the high right atrium and coronary sinus ostium. The possible mechanisms of the continuous AV node function curves in AVNRT include (1) the functional refractory period of the atrium limits the prematurity with which atrial premature depolarization will encounter the refractoriness in the AV node, which in turn produces inability to dissociate the fast and slow AV node pathways, and (2) fast and slow AV nodal pathways have similar refractory periods and conduction time.\textsuperscript{87}

[FIGURE 14-14 A, Right ventricular stimulus induces atrioventricular nodal reentrant tachycardia (AVNRT). Although the last pacing beat (with vertical line) does not conduct to the atrium, AVNRT still happens. B, AVNRT with occasional retrograde conduction to the atrium (oblique arrows).]

ATRIOVENTRICULAR REENTRY TACHYCARDIA

Anatomy and Electrophysiology of Accessory Pathways

The oblique orientation of most accessory pathways has been demonstrated by detailed endocardial and epicardial mapping techniques.\textsuperscript{92-94} The atrial and ventricular insertion sites of accessory pathways can be up to 2 cm disparate in location; furthermore, some accessory pathways have antegrade and retrograde conduction fibers at different locations, and this finding has been proven by different ablation sites for antegrade and retrograde conduction.\textsuperscript{95} Thus, the anatomic and functional dissociation of the accessory pathway into atrial and ventricular insertions and antegrade and retrograde components is possible. Approximately 90% of
FIGURE 14-15  A–D, Atrioventricular (AV) nodal conduction curve with multiple jumps. Five patterns of tachycardia (slow-fast form) induction demonstrated by AV node conduction curves ($A_2H_2$ versus $A_1A_2$). $A_2H_2$, atrio–His bundle conduction interval in response to atrial extrastimulus; $A_1A_2$, coupling interval of atrial extrastimulus. Open circles indicate fast pathway conduction; open circles with central cross indicate slow pathway conduction without initiation or maintenance of sustained tachycardia; closed circles indicate slow pathway conduction with initiation and maintenance of sustained tachycardia; closed circles with central cross indicate slow pathway conduction with initiation but without maintenance of sustained tachycardia.  A, Only the first slow pathway is used for induction and maintenance of sustained tachycardia (pattern 1).  B, Only the second slow pathway is used for induction and maintenance of sustained tachycardia (pattern 2).  C, The first slow pathway is used during sustained tachycardia; either the first or the second slow pathway is used for initiation of tachycardia (pattern 3).  D, The first slow pathway is used during sustained tachycardia; any of the three slow pathways is used for initiation of tachycardia (pattern 4).  E, The third slow pathway is used during sustained tachycardia; either the second or third slow pathway is used for initiation of tachycardia (pattern 5). (From Tai CT, et al: J Am Coll Cardiol 1996;28:725-31.)  F, AV nodal conduction curve without AH jump. Curve relating $A_2H_2$ interval to prematurity of atrial extrastimuli ($A_1A_2$). Before ablation, a smooth curve without evidence of dual AV nodal pathway is present (○). After critical AH delay, tachycardia also is induced (★). Ablation in the slow pathway zone eliminates the “tail” of the curve (△). (From Tai CT, et al: Circulation 1997;95:2541-7.)
FIGURE 14-16 Recordings show four types of atrioventricular nodal reentrant tachycardia (AVNRT) and echo. A and B, the baseline state. C and D, intravenous infusion of isoproterenol. A, induction of slow-fast form of AVNRT by atrial extrastimulus, with the earliest atrial activation at the ostium of the coronary sinus (OCS). D, a slow-slow form AVNR echo before termination of slow-intermediate form of AVNRT. $A_2$ and $H_2$, atrial and His bundle response to the atrial extrastimulus ($S_2$), respectively; HRA, HBE$_1$, HBE$_2$, PCS, and MCS, electrograms recorded from the high right atrium, the distal His-bundle area, proximal His-bundle area, proximal coronary sinus, and middle coronary sinus, respectively; $S_1$, basic paced beats; $S_2$, extrastimulus. (From Tai CT, et al: Am J Cardiol 1996;77:52-8.)
AV accessory pathways have fast conduction properties, and the other accessory pathways (including Mahaim fibers) show decremental conduction properties during atrial or ventricular stimuli with shorter coupling intervals. These pathways with decremental conduction may be sensitive to several antiarrhythmic drugs, including verapamil and adenosine. Accessory pathways in the right free wall and posteroseptal areas have a higher incidence of decremental conduction properties. When decremental conduction is present, the possibility of Mahaim fiber, such as atriofascicular or nodoventricular pathway, must be considered. Several studies have demonstrated that most of the ventricular insertion sites of these particular bypass tracts are close to the right bundle branch, and the typical Mahaim fiber potential can be recorded along the tricuspid annulus in patients with the atriofascicular pathways (Fig. 14-17).

Electrophysiological Findings in Atrioventricular Reentry Tachycardia

The manifest accessory pathway can be diagnosed from the 12-lead surface ECG with typical δ wave and is confirmed by a reduced, absent, or negative H–V interval. During electrophysiological study, recordings should be obtained from the tricuspid and mitral annulus directly or indirectly from the coronary sinus as well as the normal AV nodal His conduction system. Atrial and ventricular pacing and extrastimulation, isoproterenol provocation, and induction of atrial fibrillation to assess antegrade conduction over an accessory pathway are essential elements of electrophysiological study. Ventricular pacing and extrastimulation can define retrograde conduction properties such as refractoriness and conduction time and location of the pathway. Switching of conduction between the accessory pathway and AV nodal–His axis can be demonstrated upon reaching effective refractoriness of one or the other conduction pathway. Atrial pacing or extrastimulation can accentuate antegrade preexcitation up to the refractoriness of the pathway. Tachycardia induction requires unidirectional block in one of the AV conduction pathways (AV node/His or accessory pathway) coupled with critical conduction delay in the circuit.

For the diagnosis of accessory pathway–mediated AV reentry tachycardia, a premature ventricular depolarization can be delivered during the tachycardia at a time when the His bundle is refractory, and the impulse still conducts to the atrium; this indicates that retrograde propagation conducts to the atrium over a pathway other than the normal AV conduction system. The definition of AV reentry tachycardia involves reentry over one or more AV accessory pathways and the AV node, and the classic classification of AV reentry tachycardia includes orthodromic and antidromic tachycardias. For the initiation of orthodromic tachycardia, a critical degree of AV or VA delay, which can be in the AV node or His-Purkinje system, is usually necessary.

**FIGURE 14-17** A mapping catheter along the posterolateral aspect of tricuspid annulus records the Mahaim fiber potential (arrow in A). Application of radiofrequency energy on this point eliminates Mahaim fiber conduction with disappearance of ventricular preexcitation (B).
However, dual AV nodal pathway physiology, with or without AV nodal reentrant tachycardia, can be noted in some patients (Fig. 14-18). Ventricular pacing from different sites can provide valuable information about retrograde conduction through the AV node or via a septal pathway (Fig. 14-19). The incidence of antidromic AV reentry tachycardia is much lower than orthodromic AV reentry tachycardia. Rapid conduction in retrograde AV nodal–His axis is necessary for initiation and maintenance of antidromic tachycardia. The atrial premature beat usually can advance the next preexcited ventricular complex through the antegrade accessory pathway, or it can terminate the AV reentry tachycardia through collision with the previous retrograde wavefront (Fig. 14-20). The incidence of multiple accessory pathways is about 5% to 20% and antidromic tachycardia is common in this situation.

The most difficult situation for differential diagnosis of AV reentry tachycardia is the so-called Mahaim tachycardia, including atriofascicular or nodofascicular (or nodoventricular) reentry tachycardia and AV nodal reentry tachycardia with innocent bystander bypass tract. These arrhythmias often appear as wide complex tachycardias with a left BBB and left axis deviation morphology. However, presence of VA dissociation favors nodofascicular tachycardia.

**Principles of Management**

The principles of management in many forms of paroxysmal SVT have undergone a sea of change with a better understanding of the natural history and progression of these arrhythmias, as well as the development of more effective ablative and drug therapy for many of these rhythms. Immediate and long-term prophylactic therapy differs significantly in considerations in selection and clinical application. Immediate therapy is directed at resolution of an individual arrhythmic event. Prophylaxis is largely focused on curative approaches, though suppressive and tachycardia termination methods exist and are occasionally applied in specific clinical scenarios. The spontaneous occurrence of a single SVT event may or may not mandate immediate therapy based on symptomatology and certainly does not mandate prophylactic therapy unless recurrence is anticipated, patterns of recurrence and duration become clearer, or the event is potentially malignant or life threatening. Immediate therapy is indicated if a patient develops angina, heart failure, or syncopal symptomatology during SVT. Sustained palpitations can impair functionality and result in serious patient discomfort and require immediate termination. In SVT observed during infancy and childhood, resolution can be observed with growth and development in selected individuals. Diagnosis of these rhythms even in preterm infants has resulted in better characterization of the clinical outcome and therapy selection. While observation with a conservative management approach was more prevalent in this and older populations, the advent of effective curative therapy in the form of catheter ablation has lowered the threshold for intervention in both adult and pediatric populations. As mentioned earlier, a change in pattern of SVT over time is not uncommon and should be considered in prophylaxis.
The success and safety of ablation of AVNRT, AVRT, and other focal atrial tachycardias has offered promise of cure. The prevalence of these arrhythmias in young populations with the prospect of lifelong recurrent SVT has rapidly motivated physicians to offer catheter ablation as definitive first-line treatment after resolution of the acute episode. Drug therapy is generally reserved for acute management of a symptomatic episode, short-term prophylaxis as a bridge to cure, or rarely in patients in whom ablation is contraindicated or not feasible. While device therapy has had a niche role and can be effective in many of these rhythms, it is now largely relegated to an adjunctive role when implantation of a device is contemplated for other clinical indications in a patient with recurrent SVT. Device therapy is most often initiated for other indications, with activation of antitachycardia pacing for coexisting SVT termination. The use of device therapy in patients with failed ablation and drug therapy is an increasingly rare event.

Evidence-Based Therapy

Spontaneous termination of SVT is common and can occur quite early. Waxman et al. concluded that the spontaneous termination of SVT occurred within the AV node. Further pharmacologic testing indicated that the initial hypotension during SVT onset provokes a sympathetic response to raise blood pressure, which in turn enhances vagal tone that can terminate the arrhythmia. Immediate therapy in this SVT usually consists of vagal or other physical maneuvers to terminate the event or the parenteral administration of a short-acting effective AV nodal blocking agent. Commonly used vagal maneuvers include unilateral carotid sinus massage, diving reflex activation with placement of the face in cold water, or a Valsalva maneuver. These are often quite effective and can be performed by the patient or a health professional. In a comparative clinical study, the efficacy of the initial use of the Valsalva maneuver was 19% and of carotid sinus massage was 11%. However, the sequential use of both techniques improved overall success to 28%.

Due to its extremely short half-life, safety, and efficacy, intravenous adenosine has become the drug of choice in the United States. A single peripheral bolus of 6 or 12 mg followed by saline bolus for rapid transit with minimal dilution is employed. In clinical trials, the efficacy of a single 6 mg intravenous bolus was 63%, rising to 91% at 12 mg. Central administration is not more effective than peripheral bolus injection, but lower doses of 3 mg and 6 mg are more effective. Adenosine may result in complete but very transient AV block or sinus arrest in individual patients; it resolves spontaneously within a few seconds and reinitiation of SVT occurs in less than 10% of patients. In a field study, vagal maneuvers were followed by adenosine administration. There was a 90% conversion of PSVT, consistent with dose-ranging studies, and there was no difference in
FIGURE 14-20  A, A 12-lead ECG during sinus rhythm. B, Surface 12-lead ECG of anterograde tachycardia. C, Rapid atrial pacing (S) induces anterograde atrioventricular (AV) reentry tachycardia with antegrade conduction through right free wall accessory pathway and retrograde conduction through slow AV nodal pathway (earliest retrograde atrial activation in the CS ostium). D, One atrial premature beat preexcites the ventricle through the accessory pathway; however, this atrial premature beat (S) also collides with retrograde atrial activation, and AV reentry tachycardia is terminated.
the asystolic pause seen in PSVT or AF patients. Adenosine use may also help unmask the underlying mechanism of SVT. It is commonly used as a test to block the AV node and elicit accessory pathway conduction during electrophysiological study and may show δ waves transiently in SVT due to AV reentry. In other instances, it may show underlying dual AV nodal physiology in patients with AVNRT.

Adenosine is expensive and other alternative agents that are highly effective include intravenous calcium blockers such as diltiazem or verapamil, type 1 agents or β-blockers. Verapamil is typically administered as a bolus in 5 mg aliquots, and a total of 10 to 15 mg is almost invariably effective. In refractory patients, use of a drug infusion may be helpful. In a direct comparison, the efficacy of intravenous verapamil was 73% in one study and did not differ significantly from adenosine, but hypotension was more common with verapamil. Rarely, fast pathway block with the use of intravenous type 1 agents such as procainamide may be contemplated. Intravenous flecainide has been used in a dose of 2 mg/kg body weight and terminates AVNRT and AVRT with more than 90% efficacy. Electrical reinitiation of PSVT is uncommon and is associated with a markedly slower tachycardia. Comparison of intravenous diltiazem at a dose of 0.2 mg/kg and esmolol at 0.5 mg/kg showed superior efficacy of diltiazem. New intravenous agents include doxefiltide, which has been tested in the WPW syndrome in patients with atrial fibrillation and AVRT. The overall efficacy in one study was 71%. Intravenous propafenone was also highly effective in the same patient population in another study.

It is now extremely rare to use electrical antitachycardia therapies for acute conversion of SVT. However, it is important for the treating physician to know that atrial antitachycardia pacing and cardioversion can be used effectively for this purpose. Bursts, programmed extrastimuli, or ramp pacing in the atrium can effectively terminate AVNRT, and this may also be achieved with rapid ventricular pacing in many patients. Thus, in the presence of pacing lead systems on a temporary or permanent basis, any of these modes may effectively terminate an episode of AVNRT without major adverse sequelae in most patients. Induction of atrial fibrillation may occasionally occur with atrial pacing methods, and ventricular tachyarrhythmias may be rarely initiated in a predisposed patient with rapid ventricular pacing. Direct current cardioversion is highly effective but is rarely needed even in emergent circumstances. However, in patients with syncope and very rapid PSVT, this should be considered in emergent circumstances.

Oral single-dose drug therapy to terminate a single arrhythmic event has been gaining currency, particularly in Europe. This is widely promoted in cardioversion of atrial fibrillation. However, this principle can be applied to a single PSVT event that is not highly symptomatic and can be tolerated by the patient. β-Blockers, calcium blockers, and even type 1 drugs can be considered, but in contrast to atrial fibrillation, formal efficacy and safety studies are lacking.

**Prophylaxis of Recurrent Paroxysmal Supraventricular Tachycardias**

Prophylaxis of recurrent AVNRT has been attempted with drug therapy, ablation, and device therapy. Catheter ablation is the first-line therapy for prophylaxis of PSVT in virtually all populations and age groups, is curative in nature, and uses a variety of techniques. It may be avoided in patients who have major contraindications to the procedure such as uncontrolled infections, bleeding diatheses, unstable cardiovascular hemodynamics, and implanted prosthetic heart valves, as well as in very elderly and debilitated patients. However, successful procedures have been performed in our laboratory in patients in their 10th decade of life. In AV nodal tachycardia, the most widely used technique is ablation of the slow AV nodal pathway, while in AVRT, ablation of the accessory bypass tract is performed. The technical details of each technique are described in Section V of this textbook, Pharmacologic and Interventional Therapies. Catheter ablation can be performed using map-guided or anatomically based approaches in AV nodal reentry but is invariably performed using map-guided methods for AVRTs, as well as in other preexcitation syndrome substrates. In brief, slow pathway ablation is performed at or above the posterior aspect of the Triangle of Koch just above the coronary sinus ostium. Fast pathway ablation is performed in the superior part of the triangle, just above the AV node–His bundle axis. Specific ablation methods are used for free wall accessory pathways in the left or right atrium, anteroseptal, midseptal, posteroseptal, and paraseptal accessory pathways. Finally, ablation of the nodoventricular and fasciculo-ventricular and atrio-His fibers has also been successfully performed with map-guided methods. The success of these techniques in suppression of recurrent SVT exceeds 90%, with complication rates of 1% or less. Mortality is rare with inadvertent complete AV block and myocardial perforation being the most important major complications, estimated at 1% to 2%. Successful cure of AVRT can vary with ablation site, with left free wall and posteroseptal or paraseptal pathways showing higher efficacy rates than right free wall or right anteroseptal pathways. Alternative ablation techniques for AV nodal tachycardia include ablation of the fast AV nodal pathway. Fast pathway ablation can be equally effective in cure of AV nodal reentry but is associated with a higher complication rate with respect to AV nodal block, approaching 5% in some series. Similarly, ablation of intermediate septal pathways in the WPW syndrome can have a similar risk of complete AV block. It is rarely necessary and generally inadvisable to perform complete AV nodal ablation in either arrhythmia. In a comparative clinical trial, catheter ablation provided superior control of symptoms and better quality of life than drug therapy in patients with recurrent PSVT.

Prospective clinical studies have documented a high degree of efficacy with catheter ablation in this condition. Tables 14-7 and 14-8 show the NASPE voluntary registry of 3423 procedures performed at 68 centers in the United States. It reports efficacy rates for AV
junctional ablation, AVNRT ablation, and accessory pathway ablation by tract location at teaching and non-teaching hospitals in the United States. Note that efficacy rates exceed 90% for all arrhythmias, and there is virtually no significant difference in outcome by location. In addition, comparison of data for large-volume (>100 procedures/yr) and lower-volume centers did not show differences, indicating that the learning curve for the procedure was over. There was a slightly lower success rate with right free wall or septal pathways compared with free wall pathways. Significant complications in this series of patients undergoing AV junction ablation included a very low mortality and a small incidence of sudden death due to polymorphous VT after ablation and cardiac pacing at relatively rapid rates is recommended for several weeks after AV junction ablation. For patients undergoing slow pathway ablation, the risk of second-degree AV block was 0.16% and for complete heart block was 0.74%. Complications included a low incidence of cardiac tamponade, AV block, and rare instances of coronary occlusion and pulmonary embolism. The overall complication rate for this procedure is estimated at less than 1%.

Drug therapy has been largely relegated to temporary, intermittent, or second-line choice. Digoxin therapy, long established for this purpose, has now been supplanted by more effective β-blocker, calcium blocker, and types 1C and III drug therapy in the last 2 decades. In prospective clinical trials, oral flecainide therapy was associated with an actuarial 79% to 82% freedom from symptomatic PSVT events compared with only 15% on placebo at 60 days (P < .001). The median time to the first symptomatic PSVT event was greater in the flecainide group, and the interval between attacks was increased by flecainide. Similarly, propafenone reduced recurrent PSVT in prospective studies by 80%, Oral verapamil was shown to be effective in the prophylaxis of SVT in comparison with placebo, with reduced need for pharmacologic cardioversion and event rates. In a comparison with flecainide therapy, it was equally effective and well tolerated. In one study, both drugs showed a marked reduction in the frequency of attacks of PSVT, with a small advantage for flecainide. Thirty percent of patients on flecainide had resolution of symptomatic attacks versus 13% of the patients on verapamil (P = .026). Eleven percent of patients discontinued flecainide, and 19% discontinued verapamil for inefficacy at 1 year. Both drugs were well tolerated; 19% of the flecainide group discontinued therapy due to adverse effects, compared with 24% discontinuing verapamil for this reason. Randomized controlled trials of propafenone with placebo show a sixfold increase in time to first PSVT recurrence at a dose of 600 mg/day. While the higher dose of 900 mg was even more effective if tolerated, there was a significant increase in adverse effects. Comparative studies have shown propafenone and flecainide to have similar efficacy and safety. Newer agents include dofetilide, azimilide, and sotalol. While data on dofetilide are limited, at a dose of 500 µg/day, dofetilide is equally effective as propafenone at a relatively low dose of 450 mg/day. The probability of freedom from recurrent PSVT was 50% and 54%, respectively, with a 6% probability on placebo. Thus, these efficacy rates remain well below levels seen with catheter ablation and relegate this approach to a second line of therapy. In addition, the improvement of quality of life with catheter ablation is superior to medical therapy. The improvement in quality of life was seen in patients with moderate or severe symptoms due to PSVT. The safety profile of type 1C agents in the elderly is also of concern due to the risk of proarrhythmia.

## TABLE 14-7 Percentage of Successful Ablations and Complications for Teaching Versus Nonteaching Hospitals

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Teaching Hospital, % Success</th>
<th>Nonteaching Hospital, % Success</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrioventricular junction</td>
<td>171/176, 97.2</td>
<td>458/470, 97.4</td>
</tr>
<tr>
<td>Atrioventricular nodal reentrant tachycardia</td>
<td>456/476, 95.8</td>
<td>705/732, 93.2</td>
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<tr>
<td>AP (total)</td>
<td>255/275, 92.7</td>
<td>372/399, 93.2</td>
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<tr>
<td>Left free wall</td>
<td>160/172, 93.6</td>
<td>232/247, 93.9</td>
</tr>
<tr>
<td>Right free wall</td>
<td>26/27, 96.3</td>
<td>54/56, 96.4</td>
</tr>
<tr>
<td>Septal</td>
<td>75/83, 90.4</td>
<td>90/103, 87.4</td>
</tr>
</tbody>
</table>

## TABLE 14-8 Percentage of Successful Ablations and Complications for Medical Centers Handling More or Fewer Than 100 Cases in 1998

<table>
<thead>
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<th>Procedure</th>
<th>With &gt;100 Cases, % Success</th>
<th>With &lt;100 Cases, % Success</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrioventricular junction</td>
<td>354/366, 96.7</td>
<td>275/280, 98.2</td>
</tr>
<tr>
<td>Atrioventricular nodal reentrant tachycardia</td>
<td>603/627, 96.2</td>
<td>558/581, 95.8</td>
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<tr>
<td>AP (total)</td>
<td>315/339, 92.9</td>
<td>312/335, 93.1</td>
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<tr>
<td>Left free wall</td>
<td>201/216, 93.1</td>
<td>191/202, 94.6</td>
</tr>
<tr>
<td>Right free wall</td>
<td>37/39, 94.9</td>
<td>43/44, 97.7</td>
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<tr>
<td>Septal</td>
<td>144/160, 90.0</td>
<td>85/98, 86.7</td>
</tr>
</tbody>
</table>

Surgical Ablation of Atrioventricular Nodal Reentrant Tachycardia

Surgical ablation of PSVT has been well developed since the 1980s. Established and progressively refined techniques have been used since that period. The surgical basis for SVT ablation involves resection or modification of the substrate for the arrhythmia applying and actually pioneering many of the principles that have come in vogue in catheter ablation procedures. Experimental studies have refined surgical techniques since the original description of SVT control by ligature of the His bundle. Since that time, complete AV block has been avoided, and modification of selective pathways with preservation of normal AV conduction has been pursued. In animal studies, McGuire and coworkers have disconnected the anterior part of the
AV node and showed mild impairment of AV nodal conduction, and ventriculo-atrial conduction was destroyed in 50% of the animals. This helped define the contribution of each of these connections to AV conduction patterns in this animal model. It also suggested that the fast pathway is the preferred route of conduction through the AV node, but other inputs can assume these responsibilities with limited loss of efficacy. This became the basis for one surgical approach to cure AVNRT by fast pathway interruption. More recently, slow pathway interruption by surgical or catheter ablation methods has become the procedure of choice, with lesser opportunity of impaired AV conduction and a high degree of efficacy. However, in failures, ablation alternatives such as elimination of the fast pathway or left-sided connections should be considered.

Accessory pathway ablation using an endocardial or epicardial approach using linear incision at the mapped pathway location was widely used in the 1980s until it was supplanted by catheter ablation. Near-perfect efficacy has been described in the largest series with mortality rates of less than 1% in patients without cardiac disease or other surgical procedures. The details of the mapping and surgical technique are beyond the purview of this chapter and are described elsewhere in this text or in referenced literature. Suffice it to state that the epicardial approach to accessory pathway eliminated the need for cardiopulmonary bypass, and cryoablation of the AV groove resulted in a safe and highly effective procedure. Other energy sources such as laser have also been applied clinically. The usefulness of this technique lies in the ability to perform curative ablation procedures in PSVT patients undergoing cardiac surgery for other indications and in patients with failed catheter ablation attempts, particularly if multiple accessory pathways are present. The role of this approach in sino-atrial node reentry or nonparoxysmal sinus tachycardia or other atrial tachycardias is less well established. While surgical success in select patients has been reported, this is not as effective an approach in these substrates.

Device Therapy

The use of antitachycardia devices for PSVT termination and prevention has become of historical significance except in a few specific situations with newer devices. It is mentioned here for completion, as well as for limited use in future devices. Early studies established the efficacy of antitachycardia pacemakers in recurrent PSVT. Atrial burst and ramp pacing have been shown to be highly effective in termination of PSVT and common atrial flutter with implanted pacemaker devices. Prevention of PSVT in different substrates has also been shown. Permanent dual-chamber pacing with a short A-V interval can prevent reentrant PSVT due to collision of the atrial and ventricular wavefronts in the critical elements of the PSVT circuit. This pacing mode has been employed effectively for this purpose. Long-term management of PSVT is feasible with automatic and patient activated antitachycardia pacing. Current devices have these pacing modes available in their repertoire. The use of these modes may be considered in patients with implanted pacemakers or pacemaker defibrillators for other arrhythmia indications, who have coexisting PSVT of modest frequency. Incessant or highly frequent PSVT should be considered for ablative therapy.

REFERENCES