This chapter and the next focus on disturbances of cardiac rhythm with a rapid rate, namely: supraventricular and ventricular tachyarrhythmias (Fig. 14-1).

**Key Pathophysiologic Concept**

For any rapid, abnormal heart rhythm to occur, two major factors have to be present:
- A *trigger* that initiates the arrhythmia
- A *substrate* that allows the arrhythmia mechanism to continue

Tachyarrhythmias, both supraventricular and ventricular, in turn, usually start with premature beats that initiate arrhythmias by either *focal* or *reentrant* mechanisms (Fig. 14-2).

Focal tachycardias involve repetitive firing of an ectopic (nonsinus) pacemaker. In contrast, reentry involves uneven (nonuniform) spread of a depolarization wave through one pathway in the heart, with blockage along a second pathway. If this block in the second pathway is “unidirectional,” the wave may be able to reenter this second pathway from the reverse direction and then travel down pathway 1, creating an abnormal “revolving door” circuit.

Sometimes, an arrhythmia (e.g., atrioventricular nodal reentrant tachycardia) may be initiated by one mechanism (e.g., a premature beat from a focal site) and then sustained by another mechanism (e.g., reentry).

The sinus or sinoatrial (SA) node (see Chapter 13) is the physiologic (natural or intrinsic) pacemaker of the heart. The SA node normally initiates each heartbeat, producing (normal) sinus rhythm. However, pacemaker stimuli can arise from other parts of the heart, including the atrial muscle or pulmonary vein area, the atrioventricular (AV) junction, or the ventricles.

Ectopic beats are often premature; that is, they come before the next sinus beat is due. Examples include *atrial premature beats* (APBs), *AV junctional premature beats* (JPBs), and *ventricular premature beats* (VPBs). Ectopic beats can also come after a pause (delay) in the normal rhythm, as in the case of AV junctional or ventricular *escape* beats (see Chapter 13). Ectopic beats originating in the AV junction (node) or atria are referred to as *supraventricular* (i.e., literally coming from above the ventricles).

This chapter and Chapter 15 describe the major supraventricular arrhythmias, and Chapter 16 deals with ventricular tachycardias.

**ATRIAL AND OTHER SUPRAVENTRICULAR PREMATURE BEATS**

APBs* result from ectopic stimuli and are beats arising from loci in either the left or right atrium, or interatrial septum, but not the SA node itself.

*The terms atrial premature contractions, atrial premature beats, atrial premature depolarizations, and atrial extrasystoles are used synonymously. Most cardiologists prefer the designations premature beat, depolarization, and complex because not every premature stimulus is associated with an actual mechanical contraction of the atria or ventricles.*
The atria, therefore, are depolarized from an ectopic site. After an atrial or junctional depolarization, the stimulus may spread normally through the His-Purkinje system into the ventricles. For this reason, ventricular depolarization (QRS complex) is generally not affected by APBs or JPBs. The major features of APBs are listed in Box 14-1 and are depicted in Figures 14-3 to 14-6.

APBs may occur frequently or sporadically. Two APBs occurring consecutively are referred to as an atrial couplet. Sometimes, as shown in Figure 14-4, each sinus beat is followed by an APB. This pattern is referred to as atrial bigeminy.

**Clinical Significance**

APBs, conducted and blocked, are very common. They may occur in people with normal hearts or with virtually any type of organic heart disease. Thus, the presence of APBs does not imply that an individual has cardiac disease. In normal people these premature beats may be seen with emotional stress, excessive intake of caffeinated drinks, or the administration of sympathomimetic agents (epinephrine, isoproterenol). APBs may also occur with hyperthyroidism. APBs may produce palpitations; in this situation, patients may complain of feeling a “skipped beat” or an irregular pulse. APBs may also be seen with various types of structural heart disease. Frequent APBs are sometimes the forerunner of atrial fibrillation or flutter (see Chapter 15) or other supraventricular tachyarrhythmias discussed later.
The atrial depolarization (P wave) is premature, occurring before the next sinus P wave is due.

The QRS complex of the atrial premature beat (APB) is usually preceded by a visible P wave that usually has a slightly different shape or different PR interval from the P wave seen with sinus beats. The PR interval of the APB may be either longer or shorter than the PR interval of the normal beats. In some cases the P wave may be subtly hidden in the T wave of the preceding beat.

After the APB a slight pause generally occurs before the normal sinus beat resumes. This delay is due to “resetting” of the sinoatrial (SA) node pacemaker by the premature atrial stimulus. This slight delay contrasts with the longer, “fully compensatory” pause often (but not always) seen after ventricular premature beats (VPBs) (see Fig. 16-9).

The QRS complex of the APB is usually identical or very similar to the QRS complex of the preceding beats. Remember that with APBs the atrial pacemaker is in an ectopic location but the ventricles are usually depolarized in a normal way. This sequence contrasts with the generation of VPBs, in which the QRS complex is abnormally wide because of abnormal depolarization originating in the ventricles (see Chapter 16).

Occasionally, APBs result in aberrant ventricular conduction, so that the QRS complex is wider than normal. Figures 14-5 and 14-6 show examples of such APBs causing delayed (aberrant) depolarization of the right and left ventricles, respectively.

Sometimes when an APB is very premature, the stimulus reaches the atrioventricular (AV) junction just after it has been stimulated by the preceding beat. Because the AV junction, like other cardiac tissues, requires time to recover its capacity to conduct impulses, this premature atrial stimulus may reach the junction when it is still refractory. In this situation the APB may not be conducted to the ventricles and no QRS complex appears. The result is a blocked APB. The ECG shows a premature P wave not followed by a QRS complex (see Fig. 14-3B). After the blocked P wave, a brief pause occurs before the next normal beat resumes. The blocked APB, therefore, produces a slight irregularity of the heartbeat. If you do not search carefully for these blocked APBs, you may overlook them.

Major Features of Atrial Premature Beats

- The atrial depolarization (P’ wave) is premature, occurring before the next sinus P wave is due.
- The QRS complex of the atrial premature beat (APB) is usually preceded by a visible P wave that usually has a slightly different shape or different PR interval from the P wave seen with sinus beats. The PR interval of the APB may be either longer or shorter than the PR interval of the normal beats. In some cases the P wave may be subtly hidden in the T wave of the preceding beat.
- After the APB a slight pause generally occurs before the normal sinus beat resumes. This delay is due to “resetting” of the sinoatrial (SA) node pacemaker by the premature atrial stimulus. This slight delay contrasts with the longer, “fully compensatory” pause often (but not always) seen after ventricular premature beats (VPBs) (see Fig. 16-9).
- The QRS complex of the APB is usually identical or very similar to the QRS complex of the preceding beats. Remember that with APBs the atrial pacemaker is in an ectopic location but the ventricles are usually depolarized in a normal way. This sequence contrasts with the generation of VPBs, in which the QRS complex is abnormally wide because of abnormal depolarization originating in the ventricles (see Chapter 16).
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- Sometimes when an APB is very premature, the stimulus reaches the atrioventricular (AV) junction just after it has been stimulated by the preceding beat. Because the AV junction, like other cardiac tissues, requires time to recover its capacity to conduct impulses, this premature atrial stimulus may reach the junction when it is still refractory. In this situation the APB may not be conducted to the ventricles and no QRS complex appears. The result is a blocked APB. The ECG shows a premature P wave not followed by a QRS complex (see Fig. 14-3B). After the blocked P wave, a brief pause occurs before the next normal beat resumes. The blocked APB, therefore, produces a slight irregularity of the heartbeat. If you do not search carefully for these blocked APBs, you may overlook them.

**Paroxysmal Supraventricular Tachycardias**

Premature supraventricular beats (Box 14-2) may occur singly or repetitively. A sudden run of three or more such consecutive nonsinus beats constitutes an episode of paroxysmal supraventricular tachycardia (PSVT). Episodes of PSVT may be brief and nonsustained (i.e., lasting from a few beats up to 30 sec). Sustained episodes (greater than 30 sec) may last minutes, hours, or longer.

The topic of PSVT is actually quite complicated. Furthermore, the term PSVT is, itself, somewhat misleading, because some of these tachyarrhythmias may be long-lasting or even incessant, not just paroxysmal or intermittent. Therefore, the following brief discussion is intended to provide clinicians with an introduction and overview. More information is provided in the online supplement and in bibliographic references.

The major types of PSVT are shown in Figure 14-7.

**Atrial Tachycardia**

Classical atrial tachycardia (AT) is defined as three or more consecutive APBs coming from a single atrial focus and having identical nonsinus P wave morphology (Fig. 14-8). The arrhythmia focus can be located in either the left or right atrium, or proximal pulmonary veins, and fires off “automatically” in a rapid way. A variant, discussed later, is multifocal AT, in which the P waves vary because they come from different “firing” sites.

**Conduction**

Conduction occurs over the AV node and His-Purkinje system and usually produces a narrow QRS tachycardia. However, if the atrial rate is high, or the AV node is not normal, different degrees of delay and block can occur in any part of the conduction system (similar to that with isolated APBs), including PR prolongation, QRS aberration, and dropped (nonconducted) P waves that are not followed by QRS complexes (i.e., they produce functional second-degree AV block) with a regular or irregular heart rate. Also, if there is a preexisting bundle branch block or rate-related interventricular conduction delay (IVCD), the QRS during AT will remain wide and can be confused with ventricular tachycardia.
PART II Cardiac Rhythm Disturbances

Figure 14-3. A, Sinus rhythm with atrial ectopy. Note the atrial premature beat (APB) after the fourth sinus beat (arrow). B, Note also the blocked atrial premature beat, again after the fourth sinus beat (arrow). The premature P wave falls on the T wave of the preceding beat and is not followed by a QRS complex because the atrioventricular (AV) node is still in a refractory state.

Figure 14-4. Sinus rhythm with atrial bigeminy. Each sinus beat is coupled to an atrial premature (early) beat followed by a slight postectopic pause. This sequence is one of the causes of group beating pattern and must be distinguished from second-degree atrioventricular (AV) heart block in which the sinus P waves come “on time” and one is not conducted (see Chapter 17).

Figure 14-5. ECG shows sinus rhythm with three atrial premature beats. The first two (marked •) are conducted with right bundle branch block aberrancy (rSR' in lead V1). The third atrial premature beat (○) is conducted with normal ventricular activation. Notice how the first two premature P waves come so early in the cardiac cycle that they fall on the T waves of the preceding sinus beats, making these T waves slightly taller.
Termination
Termination occurs when the ectopic atrial focus stops firing (either spontaneously or after administration of an antiarrhythmic drug). The last P wave of the tachycardia usually conducts to the ventricles producing QRS complex at the end of the run. Therefore, AT almost always terminates with a QRS complex (Figs. 14-9 and 14-10). This is an important feature for differential diagnosis of other types of PSVTs (see later discussion).

Multifocal Atrial Tachycardia
As noted, a special variant is related to multiple sites of atrial stimulation and is called multifocal atrial tachycardia (MAT) (Fig 14-11). This tachyarrhythmia is characterized by multiple ectopic foci stimulating the atria. The diagnosis of MAT requires the presence of three or more consecutive (nonsinus) P waves with different shapes at a rate of 100 or more per minute. MAT contrasts with classic (unifocal) AT, which involves only a single atrial focus and produces one repetitive, nonsinus P wave. The PR intervals of P waves during MAT also vary. MAT is usually seen in patients with chronic lung disease. Because the ventricular rate is irregular and rapid, this arrhythmia is most likely to be mistaken for AF.

Clinical Considerations
Sustained AT has been observed in patients with normal hearts, but more often occurs in those with virtually any type of heart disease. The atrial rate can be variable (usually between 100 and 250 beats/min). AT, particularly at a rapid rate, may cause dizziness, lightheadedness, shortness of breath, or rarely syncope. Palpitations may be present. In susceptible individuals, AT can induce angina or chronic heart failure (CHF). Short episodes may require no special therapy, but longer runs causing symptoms are usually treated with antiarrhythmic drugs or radiofrequency (RF) catheter ablation (see later discussion). Sometimes AT can deteriorate into atrial fibrillation.

AV Nodal Reentrant (Reentry) Tachycardia
AV nodal reentrant (reentry) tachycardia (AVNRT) is a supraventricular arrhythmia, usually paroxysmal, resulting from the reentry in the AV node area. Normally, the AV node behaves as a single conductor connecting the atria and His-Purkinje-ventricular electrical network. However, in some people it can behave as two functional conduction channels with different electrical properties (so-called dual pathways). One AV nodal pathway has fast and the other has slow conduction speeds.
Figure 14-7. The three major types of paroxysmal supraventricular tachycardias (PSVTs). A, The reference is normal sinus rhythm. B, With (unifocal) atrial tachycardia (AT), a focus (X) outside the sinoatrial (SA) node fires off automatically at a rapid rate. C, With atrioventricular (AV) nodal reentrant tachycardia (AVNRT) the cardiac stimulus originates as a wave of excitation that spins around the AV nodal (junctional) area. As a result, retrograde P waves may be buried in the QRS or appear just after the QRS complex (arrow) because of nearly simultaneous activation of the atria and ventricles. D, A similar type of reentrant (circus-movement) mechanism may occur with a manifest or concealed bypass tract (BT) of the type found in Wolff-Parkinson-White syndrome (see Chapter 12 and text). This mechanism is referred to as atrioventricular reentrant tachycardia (AVRT). Note the negative P wave (arrow) in lead II, somewhat after the QRS complex.

Figure 14-8. Atrial tachycardia (AT). P wave (arrow) indicates a sinus P wave; P’ (with arrow) indicates an atrial premature beat (APB) initiating and maintaining the AT. The P’ waves of the AT are best seen in lead V1, and are more subtly detectable in lead III. They are difficult to see in leads I, II, and V2 where they merge with the T waves, slightly distorting their appearance. The sinus P waves show left atrial abnormality (broad, biphasic P waves in lead V1).
The mechanism of AVNRT initiation is presented in Figure 14-12. During sinus rhythm the atrial signal engages both “fast” and “slow” pathways. It reaches the His bundle through the fast pathway first and from there conducts to the ventricles. At the same time it turns around and goes “up” the slow pathway, colliding with the more slowly conducting down-going signal. The surface ECG registers sinus rhythm with a normal PR interval; there is no evidence of the “slow” pathway existence (beats 1 and 2 in Fig. 14-12).

If an early APB arrives at the AV node, it blocks in both pathways (P’ wave after the second QRS) producing a blocked APB. If the APB arrives a little later, however, it blocks only in the “fast” pathway while conducting over the “slow” pathway (P’ after the fourth QRS) producing marked PR interval prolongation. The signal can then turn around (reenter) at the lower pathway junction and conduct up the fast pathway, which by this time has recovered its excitability (third beat from the right, indicated by upward arrow). Then the signal reenters the slow pathway at the top of the AV node and conducts down again starting a repetitive loop of reentry (indicated by the black arrows in the diagram of the last three beats). At every turn the signal activates the atria from the top and ventricles from the bottom of the circle.

Because the arrhythmia circuit operates within the AV node (between the atria and ventricles), the activation spreads nearly simultaneously up
the atria and down the ventricles with every reen- 
trant rotation of the signal. As a result, the P 
waves can be completely hidden in the QRS com- 
plex (Fig. 14-13) or appear just after it. Because of 
retrograde (bottom-to-top) activation of the 
atria, the retrograde P waves are negative in leads 
II, III, and aVF, sometimes producing subtle but 
distinctive “pseudo-S” waves and positive in leads 
V₁ and aVR where they are referred to as “pseudo-
R” waves that are absent during sinus rhythm 
(Fig. 14-14).

Besides this “typical” (slow-fast) AVNRT there 
is an “atypical” form when the short circuit in the 
AV node moves in the opposite direction (down 
the fast and up the slow pathway, sometimes 
called “fast-slow” AVNRT). This sequence pro-
duces negative P waves in lead II, and positive P 
waves in aVR that come just in front of the QRS 
complexes.

Termination
Unlike focal AT, which terminates when the ec-
topic focus stops firing, AVNRT is a self-perpetuat-
ing rhythm that will continue indefinitely unless a 
block develops in some part of the circuit. The 
block can occur in either fast (up-going) or slow 
(down-going) AV nodal pathway. The slow AV 
nodal pathway is more susceptible to vagal
Paroxysmal Supraventricular Tachycardias

influences (see Fig. 14-15) or AV nodal blocking drugs (e.g., adenosine, \* beta blockers, calcium channel blockers, or digoxin). The block in the slow pathway usually occurs just before the signal activates the ventricles from the bottom of the circuit; therefore, the last arrhythmia deflection on the electrocardiogram is a retrograde P wave (Fig. 14-15).

Clinical Considerations
AVNRT produces a rapid and almost metronomically regular supraventricular rhythm with rates usually between 150 and 220 or so beats/min. Typically, AVNRT occurs in normal hearts and starts at a young age, particularly in young women. Until the arrhythmia is correctly identified, many of these patients are misdiagnosed as having “anxiety or panic attacks.”

One of the most commonly reported symptoms at the onset of AVNRT is the sensation of a “flip-flop” in the chest (resulting from the initiating APB), followed by rapid, regular palpitations. The arrhythmia may start with the person suddenly changing position or may be associated with psychological or physiologic stress, pregnancy, or sympathomimetic drugs. Patients may complain of pulsations in the neck due to simultaneous atrial and ventricular contraction, producing “cannon A waves” on inspection of the jugular venous waves during the neck examination. AVNRT can

\* Adenosine depresses the electrical activity in both the SA and AV nodes. Its very rapid action appears to be mediated by increasing an outward potassium current, hyperpolarizing the cells of both nodes. As a result, SA node automaticity is decreased, as are automaticity and conduction in the AV node area. Adenosine also is a vasodilator (accounting for its transient blood pressure lowering and facial flushing effects).

Figure 14-14. Pseudo-S waves (leads II, III) and pseudo-R′ waves (lead V1) caused by retrograde P waves during atrioventricular nodal reentrant tachycardia (AVNRT). Note that these waveforms disappear during sinus rhythm. See text.

Termination of AVNRT

Figure 14-15. Atrioventricular nodal reentrant tachycardia (AVNRT) terminated with carotid sinus massage (CSM), a maneuver that increases vagal tone. The block of the reentrant conduction occurs in the “slow” atrioventricular (AV) nodal pathway on its way down, before activation of the ventricles. Therefore, the QRS complex at the end of the arrhythmia is absent and the last ECG deflection is the retrograde P wave (negative in lead II, indicated by arrow). The P wave resembles an “S” wave and is sometimes called a “pseudo-S” wave with AVNRT. Note also the subtle progressive delay in the slow AV nodal pathway conduction seen prior to the arrhythmia termination, associated with longer RR intervals. Sinus rhythm then resumes abruptly.
also produce dizziness, lightheadedness, and rarely, syncope.

**Atrioventricular Reentrant (Bypass Tract–Mediated) Tachycardia**

Atrioventricular reentrant (bypass tract–mediated) tachycardia (AVRT), the third most common cause of PSVT, involves an accessory atrioventricular bypass tract (see Chapter 12), which provides the substrate for reentry. Clinicians refer to two types of bypass tracts: *manifest* and *concealed*. Manifest bypass tracts can conduct the electrical signal in both directions: from the atria to the ventricles and in reverse. During sinus rhythm, this produces the classic triad (“signature”) of WPW: delta wave, short PR interval, and wide QRS (see Chapter 12).

Importantly, bypass tract conduction can produce wide or narrow complex reentrant reciprocating tachycardias as part of the WPW syndrome depending on the direction of the reentrant loop. If the signal goes down the AV node and up the bypass tract, the ECG will have a narrow QRS (referred to by cardiologists as *orthodromic AVRT*). In contrast, if the signal goes down the bypass tract and up the AV node, a much less common finding, you will see a wide QRS; this reentrant variant is technically referred to as *antidromic AVRT*.

Importantly, clinicians should be aware that the majority of bypass tracts do *not* conduct the impulse from the atria to the ventricles and are therefore completely invisible (*concealed*) during sinus rhythm. Thus, you do not see the classic WPW signature. However, some concealed bypass tracts can conduct the impulse in the reverse direction (ventricles to atria), providing, in concert with the AV node and the infranodal conduction system, the second pathway necessary for reentry and the basis for a narrow complex tachycardia: namely, orthodromic AVRT.

**Initiation and Conduction**

During sinus rhythm, retrograde conduction through the bypass tract usually does not occur because the signal gets to the ventricular end of the bypass tract through the normal conduction system while the atrium around it is still refractory from the preceding sinus beat (Fig. 14-16A).

An early VPB that occurs close to the ventricular entrance of the concealed bypass tract can block the His-Purkinje system while conducting back to the atria through the bypass tract (Fig. 14-16B). In fact, a narrow complex tachycardia initiated by a VPB is highly suggestive of AVRT (see Figs. 14-16 and 14-17). Since the atria and ventricles activate one after another “in sequence” with AVRT as opposed to “in-parallel” as during AVNRT, the interval between the QRS and the P waves is longer in the former and P waves are often

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**Figure 14-16.** Atrioventricular reentrant tachycardia (AVRT) initiation by a ventricular premature beat (VPB). A, During sinus (SA) rhythm there is no retrograde conduction across the bypass tract (BT) or atrioventricular (AV) node because of refractoriness from the previous SA beat. B, A VPB (star) firing close to the bypass tract conducts to the atrium via the bypass tract, while simultaneously blocking in the His-Purkinje system. This sequence initiates a narrow complex tachycardia in which the impulse travels down (antegrade conduction) the AV node–His–bundle branch system and reenters the atrium by going up (retrograde conduction) the bypass tract. AVRT may also be initiated by atrial premature beats (APBs). See also Figure 14-17.
Figure 14-17. Atrioventricular reentrant tachycardia (AVRT) initiated by a ventricular premature beat (VPB). During sinus rhythm (first three beats) a short PR interval, wide QRS complex, and a delta wave are present, consistent with the classic Wolff-Parkinson-White (WPW) preexcitation pattern. A VPB (beat 4) initiates a narrow complex tachycardia. Retrograde P waves are visible as negative deflections in the middle of the T wave (black arrows). This finding corresponds to the mechanism depicted in Figure 12-5.

Figure 14-18. QRS alternans during atrioventricular reentrant tachycardia (AVRT). Note the periodically alternating (an “ABAB” pattern) QRS amplitudes, which are best seen in leads V2 and V4. Retrograde P waves are seen in the middle of T wave in leads I, II, and III (arrows). Alternans during paroxysmal supraventricular tachycardia (PSVT) should not be confused with alternans during sinus tachycardia (see Chapter 11), where it is usually an indicator of pericardial effusion and often cardiac tamponade (compare with Fig. 11-3). Also, alternans may occur with other types of PSVT (and, sometimes, ventricular tachycardia), so it is not specific for AVRT.
visible superimposed on the middle of the T wave or in the ST segment.

AVRT (as well as other PSVTs) also can produce QRS alternans—a periodic change in the QRS shape, occurring with every other beat (Fig. 14-18). This interesting pattern may be due to subtle conduction variations that occur at rapid rates.

Both the atrium and the ventricle are necessary to maintain the arrhythmia circuit. Therefore AVRT always has a 1:1 AV relationship as opposed to AT or (rarely) AVNRT. If you see more P waves than QRS complexes, AVRT can be excluded.

Termination
Because AVRT uses the AV node to conduct the signal from the atria to the ventricles (so-called orthodromic AVRT), it is vulnerable to vagal influences and AV nodal blocking agents similar to AVNRT. The reentrant circuit often terminates in the AV node on the way down before reaching the ventricles, producing a retrograde P wave at the end of arrhythmia run.

Clinical Considerations
The first episode of AVRT usually presents in childhood or young adulthood in contrast to AVNRT, which is predominantly seen in young to middle-aged female subjects. AVRT occurs more frequently in men. The accessory bypass tracts can be located on the left or right side of the heart (see Chapter 12). The symptoms, including palpitations and lightheadedness, as well as shortness of breath, are similar to AVNRT, discussed earlier.

DIFFERENTIAL DIAGNOSIS AND TREATMENT OF PSVT
The differential diagnosis of PSVT can be difficult, even for seasoned cardiologists. P waves may not be clearly visible even if present because they are hidden in T waves or ST segments, especially in a single monitor lead. Sometimes it is impossible to tell the exact mechanism of the arrhythmia (especially when initiation and termination of it are not recorded) unless an invasive electrophysiologic study is performed. A summary of major diagnostic findings is presented in Table 14-1. More detailed discussion on the differential diagnosis of PSVT is available in selected bibliographic references.

The most clinically useful diagnostic as well as therapeutic measures in terminating PSVT are aimed at achieving block in AV node conduction. These measures include vagal maneuvers, particularly the Valsalva maneuver and carotid sinus massage (CSM), and also pharmacologic interventions, especially adenosine injection.

Note: While performing CSM and adenosine injections, continuous ECG monitoring is critical to document response to the intervention. Resuscitation equipment, including an external defibrillator, should be available in case of unexpected reactions.

Note: Adenosine effect is blocked by methylxanthines (e.g., theophylline; caffeine) and potentiated by dipyridamole. The most significant electrophysiologic side effect of adenosine is induction of atrial fibrillation (which may or may not terminate spontaneously). Adenosine injection often creates an extremely uncomfortable feeling noted by patients (most frequent symptoms and signs are related to facial flushing, bronchospasm with dyspnea, and even transient asystole after arrhythmia termination). To minimize their anxiety, patients should be warned that they may feel uncomfortable.

The response of PSVT to CSM (or other vagal maneuvers) or adenosine injection is summarized in Table 14-1.

Management of an Acute PSVT Episode
The management of the first acute PSVT episode should start with vagal maneuvers (e.g., the Valsalva maneuver, CSM—see Fig. 14-15) followed by adenosine injection. Many patients find ways to terminate the arrhythmias on their own (e.g., by coughing, deep breathing, using the Valsalva maneuver, squatting, facial immersion in cold water, or with CSM).

The success of vagal maneuvers for terminating PSVT is a highly specific, but only moderately sensitive, clue to a reentrant mechanism, namely either AVNRT or AVRT, not AT.

If adenosine is not available or is contraindicated, intravenous beta blockers or calcium channel blockers can be used, but may produce hypotension. These longer acting drugs can be beneficial in patients with extremely high sympathetic tone when the arrhythmia terminates with adenosine but immediately restarts. Digoxin can also be used. Rarely, synchronized external cardioversion is required to terminate PSVT.
### TABLE 14-1 Differential Diagnosis of Supraventricular Tachycardia (SVT)*

<table>
<thead>
<tr>
<th>Type</th>
<th>Sinus Tachycardia</th>
<th>Atrial Tachycardia (AT)</th>
<th>AVNRT</th>
<th>AVRT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset/termination</td>
<td>Gradual</td>
<td>Abrupt</td>
<td>Abrupt</td>
<td>Abrupt</td>
</tr>
<tr>
<td>Heart rate</td>
<td>Varies slightly with respiration and activity</td>
<td>Nearly constant rate between 100 and 225</td>
<td>Nearly constant</td>
<td>Nearly constant</td>
</tr>
<tr>
<td>Typical initiation</td>
<td>Gradual, with acceleration of sinus P waves</td>
<td>APB identical to subsequent P' waves of AT</td>
<td>Initiated by an APB with a long PR interval</td>
<td>Initiated by a VPB or an APB</td>
</tr>
<tr>
<td>P waves during the SVT</td>
<td>Sinus</td>
<td>Identical to each other (except with MAT) but different from sinus</td>
<td>Often pseudo-R’ in V1; pseudo-S in II, III, and aVF, or P waves invisible</td>
<td>Negative in II, III, and aVF, usually shortly after QRS complex</td>
</tr>
<tr>
<td>Vagal maneuvers or adenosine injection</td>
<td>Heart rate slows down, with possible transient AV block; P waves continue</td>
<td>Rarely terminates; more commonly transient AV block may develop</td>
<td>Either no effect or PSVT terminates suddenly (usually ending with a P wave)</td>
<td>Either no effect or PSVT terminates suddenly (usually ending with a P wave)</td>
</tr>
<tr>
<td>SVT termination: last waveform seen</td>
<td>Gradual slowing of P-QRS rate; no abrupt termination</td>
<td>QRS complex</td>
<td>P’ or QRS complex</td>
<td>P’ or QRS complex</td>
</tr>
</tbody>
</table>

*Note: This group does not include atrial fibrillation or flutter; AT, AVNRT, and AVRT constitute the PSVT group.

APB, atrial premature beat; AV, atrioventricular; AVNRT, atrioventricular nodal reentrant tachycardia; AVRT, atrioventricular reentrant ("reciprocating") tachycardia; MAT, multifocal atrial tachycardia; P', premature atrial P wave; PSVT, paroxysmal supraventricular tachycardia; VPB, ventricular premature beat.

**Long-Term Management of PSVT**

Long-term PSVT management depends on the episode frequency and degree of symptoms. If episodes are rare and mild no specific treatment is necessary after termination. In more severe cases prophylactic treatment with AV nodal blocking agents or antiarrhythmic drugs may be warranted. An early referral to an electrophysiology specialist is justified as the efficacy of ablation procedures is high (in AVNRT and AVRT it approaches 95-98%), with very low risks when done by experienced operators.

PSVT does not increase thromboembolic risk. Thus, anticoagulation is not indicated.