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Paroxysmal Supraventricular Tachycardias

OUTLINE

A "supraventricular" origin of a tachycardia implies the obligatory involvement of one or more cardiac structures above the bifurcation of the His bundle (HB), including the atrial myocardium, atrioventricular node (AVN), proximal HB, coronary sinus (CS), pulmonary veins, venae cavae, or abnormal atrioventricular (AV) connections other than the HB (i.e., bypass tracts [BTs]).[1]

Narrow QRS complex supraventricular tachycardia (SVT) is a tachyarrhythmia with a rate greater than 100 beats/min and a QRS duration of less than 120 milliseconds. Narrow complex SVTs include sinus tachycardia, inappropriate sinus tachycardia, sinoatrial nodal reentrant tachycardia, focal atrial tachycardia (AT), multifocal AT, atrial fibrillation (AF), atrial flutter (AFL), junctional tachycardia, atrioventricular nodal reentrant tachycardia (AVNRT), and atrioventricular reentrant tachycardia (AVRT). These tachycardias can be divided into those that require only atrial tissue for their initiation and maintenance (sinus tachycardia, AT, AF, and AFL), and those that require the AV junction (junctional tachycardia, AVNRT, and AVRT).

Paroxysmal SVT is the term generally applied to intermittent SVT other than AF, AFL, and multifocal AT, and describes a clinical syndrome characterized by the presence of a regular and rapid tachycardia of abrupt onset and termination (Fig. 20.1). The major causes are AVNRT (approximately 50% to 60% of cases), AVRT (approximately 30% of cases), and focal AT (approximately 10% of cases).

EPIDEMIOLOGY AND NATURAL HISTORY

Paroxysmal SVT with sudden onset and termination is relatively common. In the United States, the estimated prevalence in the general population is 2.29 per 1000, with an incidence of 36 per 100,000 person-years. Paroxysmal SVT in the absence of structural heart disease can present at any age but most commonly first presents between ages 12 and 30 years. The risk of developing paroxysmal SVT is twofold greater in women than men.[1]

In a large cohort of patients with symptomatic paroxysmal SVT referred for ablation, AVNRT was the most common mechanism (56%), followed by AVRT (27%) and AT (17%). However, the mechanism of paroxysmal SVT was significantly influenced by both age and gender. The majority of patients with AVRT are men (55%), whereas the majority of patients with AVNRT and AT are women (70% and 62%, respectively). As patients grew older, there was a significant and progressive decline in the number of patients presenting with AVRT, which was the predominant mechanism in the first decade, and a striking increase in AVNRT and AT (Fig. 20.2). These trends were similar in both genders, although AVNRT replaced AVRT as the predominant mechanism much earlier in women.[2]

AVNRT is the predominant mechanism overall in patients undergoing ablation, and after the age of 20 years it accounts for the largest number of ablations in each age group. AVNRT is unusual in children under 5 years of age, and typically initially manifests in early life, often in the teens. There is a striking 2:1 predominance of women in the AVNRT group, which remains without clear physiological or anatomical explanation. Female sex and older age (teens vs. early childhood years) favor the diagnosis of AVNRT over AVRT.[3]

AVRT presents earlier in life than AVNRT (most commonly in the first two decades of life), with an average of more than 10 years separating the time of clinical presentation of AVRT versus AVNRT. The early predominance of AVRT is consistent with the congenital nature of the substrate. However, a minority of patients have relatively late onset of symptoms associated with AVRT and thus continue to account for a small proportion of ablations in older patients. Men account for a higher proportion of AVRT at all ages.[4]

Focal ATs comprise a progressively greater proportion of paroxysmal SVT with increasing age, accounting for 23% of paroxysmal SVTs in

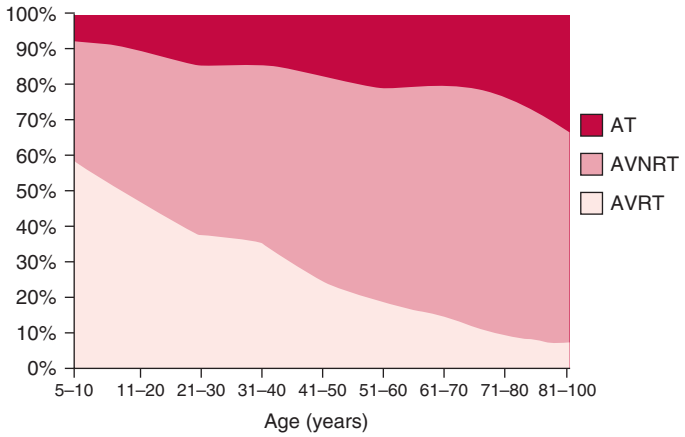


Fig. 20.1 Proportion of Paroxysmal Supraventricular Tachycardia Mechanisms by Age. *AT,* Atrial tachycardia; *AVNRT,* atrioventricular nodal reentrant tachycardia; *AVRT,* atrioventricular reentrant tachycardia. (From Porter MJ, Morton JB, Denman R, et al. Influence of age and gender on the mechanism of supraventricular tachycardia. *Heart Rhythm.* 2004;1:393.)

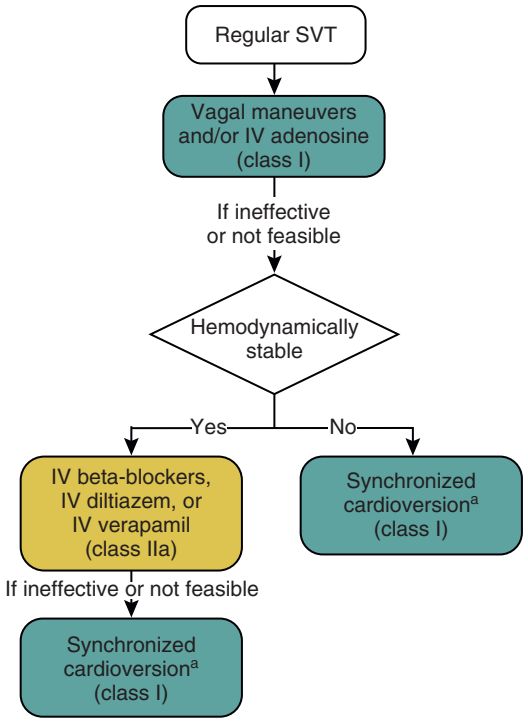


Fig. 20.2 Acute Treatment of Regular Supraventricular Tachycardia *(SVT)* of Unknown Mechanism. Drugs listed alphabetically. [a]For rhythms that break or recur spontaneously, synchronized cardioversion is not appropriate. *IV,* Intravenous. (From Page RL, Joglar JA, Caldwell MA, et al. 2015 ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *J Am Coll Cardiol.* 2016;67:e27–e115.)

patients older than 70 years. Although there is a greater absolute number of women with AT, the proportion of AT in both genders is similar. Age-related changes in the atrial electrophysiology (EP) substrate (including cellular coupling and autonomic influences) likely contribute to the increased incidence of AT in older individuals.[2] In adults, focal ATs can occur in the absence of structural heart disease; nonetheless, the incidence of associated structural heart disease is higher in AT than other types of paroxysmal SVT.[5] The long-term prognosis in patients with focal AT is generally benign, except for those with incessant ATs, which can precipitate tachycardia-induced cardiomyopathy.[1]

Unlike AF and AFL, paroxysmal SVT does not appear to be a risk marker for stroke. In fact, unexplained stroke is rare in patients with SVT. Nonetheless, a strong link exists between SVT and atrial arrhythmias, particularly paroxysmal AF, even in patients with no ventricular preexcitation. The prevalence of a history of AF in the SVT patient population is approximately 2.6% compared to 0.4% to 2% in the general population. In addition, AVRT and, less frequently, AVNRT can serve as triggers for AF.[6]

CLINICAL PRESENTATION

The clinical syndrome of paroxysmal SVT is characterized as a regular rapid tachycardia of abrupt onset and termination. Episodes can last from seconds to several hours. Patients commonly describe palpitations and heart racing, frequently associated with complaints of dyspnea, weakness, chest pain, dizziness, or even frank syncope. The impact of SVT on quality of life varies according to the frequency and duration of episodes, and the severity of symptoms.[1] Patients often learn to use certain maneuvers such as carotid sinus massage or the Valsalva maneuver to terminate the arrhythmia, although many require pharmacological treatment to achieve this.

Neck pounding can occur during tachycardia, which is related to pulsatile reversed flow when the right atrium (RA) contracts against a closed tricuspid valve. The physical examination correlate of this phenomenon is continuous pulsing cannon A waves in the jugular venous waveform (described as the "frog" sign). This clinical feature has been reported to distinguish typical AVNRT from orthodromic AVRT. Although the atrial contraction during orthodromic AVRT does occur against closed AV valves, the longer ventriculoatrial (VA) interval during the tachycardia results in separate ventricular and then atrial contractions and, hence, relatively lower RA and venous pressures. Therefore symptoms of "shirt flapping" or "neck pounding" are experienced less commonly in patients with AVRT than those with AVNRT (17% vs. 50%). In addition, polyuria, which related to higher RA pressures and elevated levels of atrial natriuretic protein, is more common in patients with AVNRT compared with patients who have AVRT.[1]

Dizziness can occur initially because of hypotension, but it then disappears when the sympathetic response to the SVT stabilizes the blood pressure (typically within 30 to 60 seconds). Reductions of blood pressure and cardiac output (and the associated reflex sympathetic activity) are likely to be most prominent in SVTs with simultaneous atrial and ventricular activation (e.g., typical AVNRT) than those with short VA intervals (e.g., slow-slow AVNRT or orthodromic AVRT), and least prominent in long RP tachycardias (e.g., atypical AVNRT, permanent junctional reciprocating tachycardia [PJRT], and AT).[1]

True syncope is rare but can occur, especially in elderly patients. Syncope at the initiation of SVT is commonly vagally mediated, especially in young patients, and is benign in nature. However, malignant ventricular arrhythmias as a cause of syncope should be considered in patients with manifest preexcitation. In these patients, AVRT can deteriorate into AF, which can be associated with very fast ventricular rates, with possible degeneration into ventricular fibrillation (VF).

In patients with underlying structural heart disease, symptoms can be more severe. Decompensation of underlying heart failure or ischemic heart disease can be precipitated by episodes of SVT. Truly paroxysmal SVT rarely leads to a tachycardia-induced cardiomyopathy. However, PJRT and focal AT can manifest as a frequently recurring or incessant tachycardia that can precipitate cardiomyopathy and heart failure. Elimination of the tachycardia results in normalization of left ventricular (LV) function within a few months in the vast majority of patients.[7]

INITIAL EVALUATION

History, physical examination, and an electrocardiogram (ECG) constitute an appropriate initial evaluation of patients presenting with symptoms suggestive of paroxysmal SVT. However, clinical symptoms are not usually helpful in distinguishing different forms of paroxysmal SVT. A 12-lead ECG during tachycardia can be helpful for defining the mechanism of paroxysmal SVT. Ambulatory 24- or 48-hour Holter recording can be used for documentation of the arrhythmia in patients with frequent (i.e., several episodes per week) but self-terminating tachycardias. A cardiac event monitor is often more useful than a 24-hour recording in patients with less frequent arrhythmias. Implantable loop recorders can be helpful in selected cases with rare episodes associated with severe symptoms of hemodynamic instability (e.g., syncope).

An echocardiographic examination should be considered in patients with documented sustained SVT to exclude the possibility of structural heart disease. Exercise testing is rarely useful for diagnosis unless the arrhythmia is clearly triggered by exertion. Further diagnostic studies are indicated only if there are signs or symptoms that suggest structural heart disease. Of note, troponin I and T levels (especially high-sensitivity Troponin T) are elevated in a significant proportion (more than 50%) of patients presenting with SVT, but this elevation does not constitute a clear biomarker of clinically significant coronary artery disease.[8] Similarly, striking ST segment depression during SVT, that resolves quickly on cessation of tachycardia, is not uncommon but does not signify obstructive coronary artery disease.

Invasive EP testing is not indicated unless a decision to proceed with catheter ablation is undertaken. EP testing with subsequent catheter ablation may also be used for diagnosis and therapy in cases with a clear history of paroxysmal regular palpitations. It may also be considered in patients with preexcitation or disabling symptoms without ECG documentation of an arrhythmia.

PRINCIPLES OF MANAGEMENT

Acute Management

Most episodes of paroxysmal SVT require intact 1:1 AVN conduction for continuation and are therefore classified as AVN-dependent. AVN conduction and refractoriness can be modified by vagal maneuvers and by many pharmacological agents and thus are the weak links targeted by most acute therapies. Termination of a sustained episode of SVT is usually accomplished by producing a transient block in the AVN (Fig. 20.3).

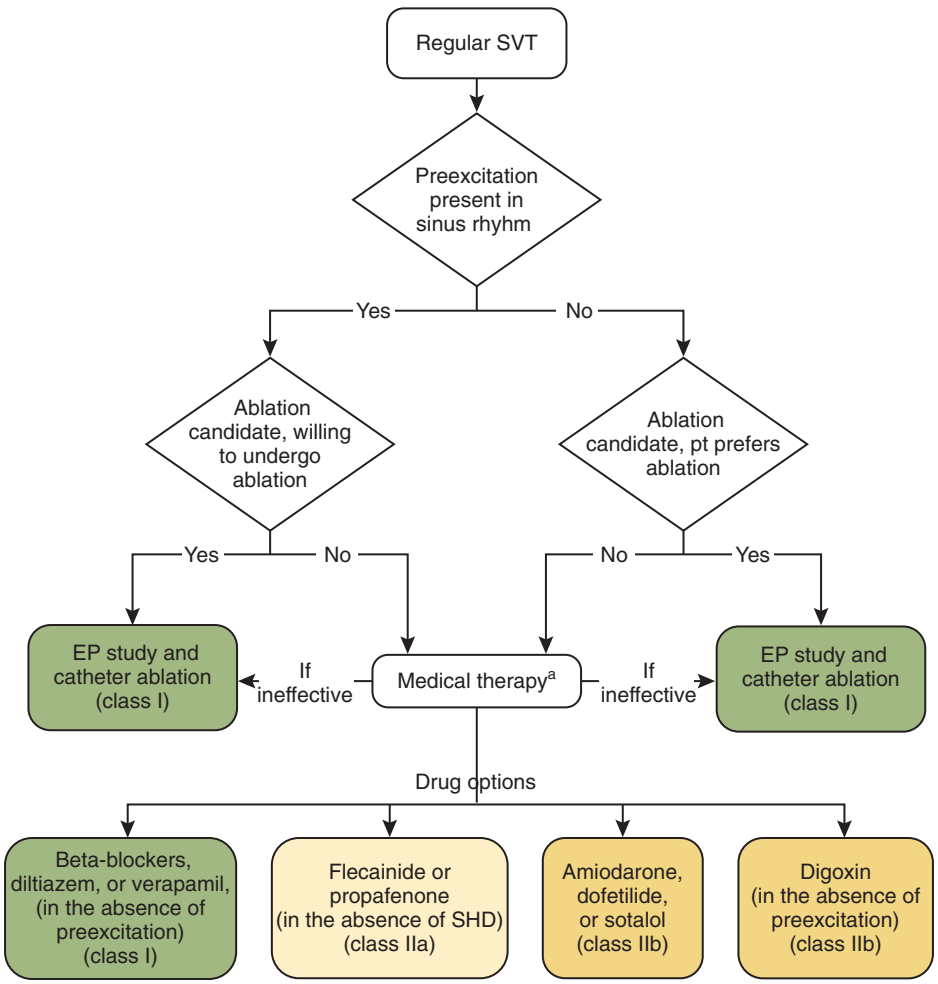


Fig. 20.3 Ongoing Management of Supraventricular Tachycardia *(SVT)* of Unknown Mechanism. Drugs listed alphabetically. [a]Clinical follow-up without treatment is also an option. *EP,* Electrophysiology; *pt,* patient; *SHD,* structural heart disease (including ischemic heart disease); *SVT,* supraventricular tachycardia. (From Page RL, Joglar JA, Caldwell MA, et al. 2015 ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *J Am Coll Cardiol.* 2016;67:e27–e115.)

For acute conversion of SVT, vagal maneuvers (including Valsalva and carotid sinus massage) are the first-line intervention, although their success rate remains limited (less than 30%). Valsalva is the most effective technique in adults, but carotid sinus massage can also be effective. The so-called modified Valsalva (patient supine, with legs slightly elevated to increase venous return and decrease reflex sympathetic tone) has also been useful. Facial immersion in water is the most reliable method in infants. Vagal maneuvers are less effective once a sympathetic response to paroxysmal SVT has become established, so patients should be advised to try them soon after onset of symptoms.[1]

When vagal maneuvers are unsuccessful, termination can be achieved with antiarrhythmic drugs whose primary effects increase AVN refractoriness or decrease AVN conduction. In most patients, the drug of choice is intravenous (IV) adenosine. In hemodynamically stable patients, IV diltiazem, verapamil, or beta-blockers are appropriate therapy for SVT that is refractory to adenosine or recurs after initial termination, and are successful in 64% to 98% of cases. Electrical cardioversion is recommended for acute treatment in patients with hemodynamically unstable SVT when vagal maneuvers or adenosine are ineffective or not feasible.[1]

The advantages of adenosine include its rapid onset of action (usually within 10 to 25 seconds via a peripheral vein), short half-life (less than 10 seconds), and high degree of efficacy. The effective dose of adenosine is usually 6 to 12 mg, given as a rapid bolus over 1 to 2 seconds at a peripheral site, followed by a vigorous flush of normal saline. If a central IV access site is used, the initial dose should not exceed 3 mg and may be as little as 1 mg. Doses up to 12 mg terminate over 90% of paroxysmal SVT episodes (predominantly AVRT and AVNRT). Sequential dosing can be given at 60-second intervals because of adenosine's rapid metabolism. In AVNRT, termination is usually caused by block in the slow pathway. In AVRT, termination occurs secondary to block in the AVN. Termination can also occur indirectly, that is, because of adenosine-induced premature atrial complexes (PACs) or premature ventricular complexes (PVCs). Adenosine also can terminate a significant proportion of focal ATs; therefore termination of an SVT in response to adenosine is not helpful in differentiating AT from other SVTs. Even when AT persists, adenosine can be useful diagnostically by producing transient AV block and unmasking the independent atrial activity during AT. Of note, adenosine is cleared rapidly; hence, reinitiation of paroxysmal SVT after initial termination can occur. Either repeated administration of the same dose of adenosine or substitution of a calcium channel blocker or beta-blocker typically is effective.

Importantly, adenosine shortens the atrial refractory period, and atrial ectopy can induce AF. This can be dangerous if the patient has a BT capable of rapid anterograde conduction, which can result in a rapid ventricular response that can degenerate into VF. In patients with Wolff-Parkinson-White (WPW) syndrome and AF, adenosine can result in a rapid ventricular response that can degenerate into VF. However, this problem has not been observed frequently, and the use of adenosine for diagnosis and termination of regular SVTs, including AVRT, is appropriate as long as close patient observation and preparedness to treat potential complications, such as with immediate electrical cardioversion/defibrillation, are maintained.

The AVN action potential is calcium channel–dependent, and the non–dihydropyridine calcium channel blockers verapamil and diltiazem are effective for terminating AVN-dependent paroxysmal SVT. The recommended dosage of verapamil is 5 mg IV over 2 minutes, followed in 5 to 10 minutes by a second 5- to 7.5-mg dose. The recommended dose of diltiazem is 20 mg IV followed, if necessary, by a second dose of 25 to 35 mg. Paroxysmal SVT termination should occur within 5 minutes of the end of the infusion, and more than 90% of patients with AVN-dependent paroxysmal SVT respond. As with adenosine, transient arrhythmias, including PACs, PVCs, AF, and bradycardia, can be seen after paroxysmal SVT termination with calcium channel blockers. Hypotension can also develop, particularly if the paroxysmal SVT does not terminate. Adenosine and verapamil have been reported to have a similar high efficacy in terminating paroxysmal SVT, with a rate of success ranging from 59% to 100% for adenosine and from 73% to 98.8% for verapamil, according to the dose and mode of administration. However, data also suggest that the efficacy of adenosine and verapamil is influenced by the arrhythmia rate. Adenosine appears to be more effective at terminating SVT with faster rates. In contrast, the efficacy of verapamil in restoring sinus rhythm was inversely related to the rate of paroxysmal SVT.

IV beta-blockers including propranolol (1 to 3 mg), metoprolol (5 mg), and esmolol (500 μg/kg over 1 minute and a 50-μg/kg per minute infusion) are also useful for acute termination. Digoxin (0.5 to 1.0 mg) is considered the least effective of the four categories of drugs available, but is a useful alternative when there is a contraindication to the other agents.

AVN-dependent paroxysmal SVT can present with a wide QRS complex in patients with fixed or functional aberration, or if a BT is used for anterograde conduction. Most wide complex tachycardias, however, are caused by mechanisms that can worsen after IV administration of beta-blockers and calcium channel blockers. Unless there is strong evidence that a wide QRS tachycardia is AVN-dependent, verapamil, diltiazem, and beta-blockers should not be used.

Limited data are available on the acute pharmacological therapy of ATs. Vagal maneuvers only rarely terminate AT, and the response to adenosine is variable. IV beta-blockers, diltiazem, or verapamil have modest efficacy (30% to 50%) in terminating the focal AT or slowing the ventricular rate. Class I or III antiarrhythmic drugs given orally or parenterally may be considered for refractory ATs. Adenosine does not slow or terminate microreentrant AT. In contrast, triggered activity ATs typically terminate abruptly in response to adenosine and do not spontaneously reinitiate. Automatic ATs are either slowed transiently by adenosine before gradual resumption of the AT rate or suppressed transiently before spontaneous reinitiation.[9] Microreentrant ATs can terminate in response to carotid sinus massage and vagal maneuvers. Triggered activity ATs can also terminate in response to carotid sinus massage, vagal maneuvers, verapamil, beta-blockers, and sodium channel blockers. During automatic AT, carotid sinus massage can cause AV block and can slow the atrial rate; however, these interventions generally do not terminate the AT. Only beta-blockers have been useful in termination of paroxysmal (but not incessant) automatic AT. Termination of automatic AT is usually preceded by a cool-down phenomenon of the AT rate.[1,9]

In general, most stable patients with SVT respond to pharmacological therapy, with conversion success rates of 80% to 98%. Electrical cardioversion is also recommended for patients with hemodynamically stable SVT when pharmacological therapy is ineffective or contraindicated. However, electrical cardioversion is inappropriate if the SVT is terminating and reinitiating spontaneously.[1]

In patients with manifest preexcitation during normal sinus rhythm (NSR) presenting with AVRT (orthodromic or antidromic), vagal maneuvers are the first-line intervention for tachycardia termination. For persistent SVT, adenosine is recommended. Importantly, adenosine should be used with caution because it can induce AF with a rapid ventricular rate in the presence of an anterogradely conducting BT. This is unusual and should not be viewed as a contraindication to adenosine use, but one should be prepared for emergency cardioversion before administering adenosine to SVT patients. For refractory AVRT, IV diltiazem, verapamil, or beta-blockers may be considered to block conduction in the AVN, which represents the retrograde or anterograde limb in the AVRT circuit. AVN blocking drugs, however, are ineffective

in patients with an antidromic AVRT that utilizes two separate BTs for anterograde and retrograde conduction. Drug treatment directed at the BT (ibutilide, procainamide, flecainide) may be considered. When drug therapy fails or hemodynamic instability is present, electrical cardioversion should be considered.[1]

Chronic Management

Most paroxysmal SVTs are generally benign and do not influence survival; therefore the primary indication for treatment is to alleviate symptoms and improve quality of life. The threshold for initiation of therapy and the decision to treat SVT with oral pharmacological therapy or catheter ablation depends on the frequency and duration of the arrhythmia, severity of symptoms, presence of concomitant structural heart disease, and patient preference (Fig. 20.4). The threshold for treatment is also influenced by whether the patient is a competitive athlete, a woman considering pregnancy, or someone with a high-risk occupation (e.g., pilots, bus drivers).[1]

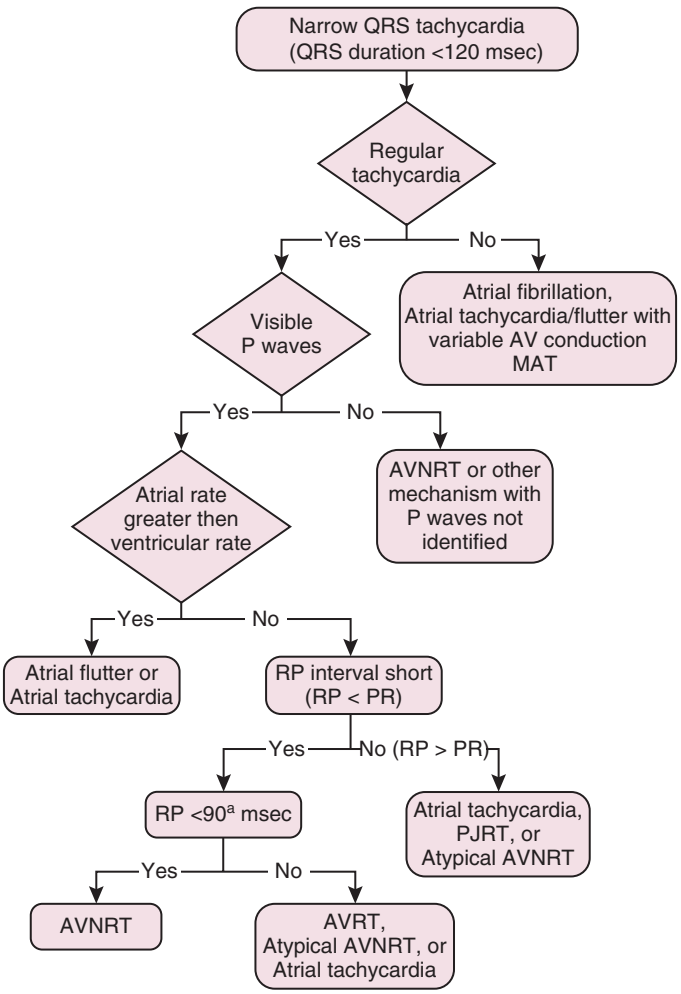


Fig. 20.4 Differential Diagnosis of Narrow QRS Tachycardia. Patients with junctional tachycardia may mimic the pattern of slow-fast atrioventricular nodal reentrant tachycardia *(AVNRT)* and may show atrioventricular *(AV)* dissociation and/or marked irregularity in the junctional rate. [a]RP refers to the interval from the onset of surface QRS to the onset of visible P wave (note that the 90-millisecond interval is defined from the surface electrocardiogram, as opposed to the 70- millisecond ventriculoatrial interval that is used for intracardiac diagnosis). *AVRT,* Atrioventricular reentrant tachycardia; *MAT,* multifocal atrial tachycardia; *PJRT,* permanent junctional reciprocating tachycardia. (From Page RL, Joglar JA, Caldwell MA, et al. 2015 ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *J Am Coll Cardiol.* 2016;67:e27–e115.)

Catheter Ablation

Given the high success rates and the low complication rate, catheter ablation is the treatment of choice in patients who desire to avoid or are unresponsive or intolerant to drug therapy.[1] Catheter ablation is also recommended for incessant SVT, even in asymptomatic patients, especially when tachycardia-induced cardiomyopathy has developed.

Pharmacological Therapy

For patients requiring therapy who are reluctant to undergo catheter ablation, drug therapy remains a viable alternative. Calcium channel blockers and beta-blockers may be considered in patients without ventricular preexcitation during NSR. Verapamil, propranolol, and digoxin likely have equivalent efficacy, and can improve symptoms in 60% to 80% of patients. However, verapamil, diltiazem, and beta-blockers are generally preferred to digoxin. The effective dose of digoxin is usually higher than that commonly used in clinical practice today. Given the risk of toxicity, digoxin should be reserved for patients who cannot take beta-blockers, diltiazem, or verapamil or a class IC agent (flecainide or propafenone) and must be used with caution in the presence of renal dysfunction.[1]

In patients who do not respond, classes IC and III drugs may be considered. Flecainide and propafenone affect the AVN and BTs, reduce SVT frequency in 86% to 93% of patients, and may be considered in patients without ischemic or structural heart disease. Sotalol, dofetilide, and amiodarone are second-line agents. The probability of remaining free of SVT after 6 months of treatment is about 50% for dofetilide and 54% for propafenone, compared to 6% for placebo. However, the potential benefit should be balanced by the potential risks of proarrhythmia and toxicity. Because sympathetic stimulation can antagonize the effects of many antiarrhythmic agents, concomitant therapy with a beta-blocker can improve efficacy.[1]

Pharmacological management of ATs has not been well evaluated in controlled clinical trials. Depending on the mechanism responsible for the arrhythmia, beta-blockers or calcium channel blockers may be considered as first-line drug therapy. Class IC agents in combination with an AVN blocking agent, or class III agents (sotalol and amiodarone) are second-line therapy (**see Chapter 11**).

Patients with SVT should be educated on how to perform vagal maneuvers. Those with well-tolerated episodes of paroxysmal SVT that always terminate spontaneously or with vagal maneuvers do not require chronic prophylactic therapy. Selected patients may be treated only for acute episodes. Outpatients may use a single oral dose of verapamil, diltiazem, or propranolol to acutely terminate an episode of SVT. This so-called pill in the pocket is a reasonable treatment option for patients who have SVT episodes that are sustained but infrequent enough that daily preventive therapy is not desired. This approach necessitates the use of a drug that has a short onset of action (i.e., immediate-release preparations); crushing the drug tablet can potentially facilitate absorption. Candidate patients should be free of significant LV dysfunction, sinus bradycardia, and preexcitation.[1]

For patients with ventricular preexcitation, antiarrhythmic drugs to block BT conduction are considered. Class IC agents (in patients without structural heart disease or ischemic heart disease), and class III drugs, such as sotalol and dofetilide, may be considered. Oral amiodarone is a last resort therapy, given the associated risks of long-term therapy. In general, antiarrhythmic drug therapy can offer symptomatic improvement in up to 90% of patients, although complete disappearance of symptoms is observed in only 30%. Chronic oral beta-blockers,

verapamil, and diltiazem may be used for the treatment of patients with WPW syndrome, particularly if their BT has been demonstrated to be incapable of rapid anterograde conduction. However, these agents must be used with caution and after a discussion with the patient concerning the potential risk of rapid conduction over the BT if AF develops. Digoxin, on the other hand, should be avoided because it can shorten the refractory period of the BT and, hence, is potentially harmful in patients with manifest BTs.[1]

ELECTROCARDIOGRAPHIC FEATURES

Regularity of the Tachycardia

Most SVTs are associated with a regular ventricular rate. If the rhythm is irregular, the ECG should be scrutinized for discrete atrial activity and for any evidence of a pattern in the irregularity (e.g., grouped beating typical of Wenckebach periodicity). If the rhythm is irregularly irregular (i.e., no pattern can be detected), the mechanism of the arrhythmia is multifocal AT, AFL with variable AV conduction, or AF (Fig. 20.5). Multifocal AT is an irregularly irregular atrial rhythm characterized by more than three different P wave morphologies, with the P waves separated by isoelectric intervals and associated with varying P-P, R-R, and PR intervals (**see Fig. 11.2**). On the other hand, AF is characterized by rapid and irregular atrial fibrillatory activity and, in the presence of normal AVN conduction, by an irregularly irregular ventricular response. P waves cannot be detected in AF, although coarse fibrillatory waves and prominent U waves can sometimes give the appearance of P waves. At times, the fibrillatory activity is so fine as to be undetectable. If the patient's rhythm is regular or has a clearly discernible pattern, the ECG should next be assessed for P waves (atrial activity).[1]

Atrial Activity

The P waves may be easily discernible; however, frequently, comparison with a normal baseline ECG is needed and can reveal a slight alteration in the QRS, ST segment, or T waves, suggesting the presence of the P wave. If the P waves cannot be clearly identified, carotid sinus massage or the administration of IV adenosine may help clarify the diagnosis. These maneuvers may also terminate the SVT.[1]

Atrial Rate

An atrial rate greater than 250 beats/min is usually caused by AFL. However, overlap exists, and AT and AVRT can occasionally be faster than 250 beats/min, especially in youngsters. Although AVRT tends to be faster than AVNRT and AT, significant overlap exists and this criterion does not usually help in distinguishing among different SVTs.[10]

P Wave Morphology

P waves indicate the result of atrial activation and may be broadly classified as concentric or eccentric. A P wave morphology identical to a sinus P wave suggests sinus tachycardia, inappropriate sinus tachycardia, sinoatrial nodal reentrant tachycardia, or AT arising close to the region of the sinus node. A nonsinus P wave morphology can be observed during AVNRT (P wave is concentric due to midline retrograde activation; **see Fig. 17.8**), AVRT (P wave can be eccentric or concentric due

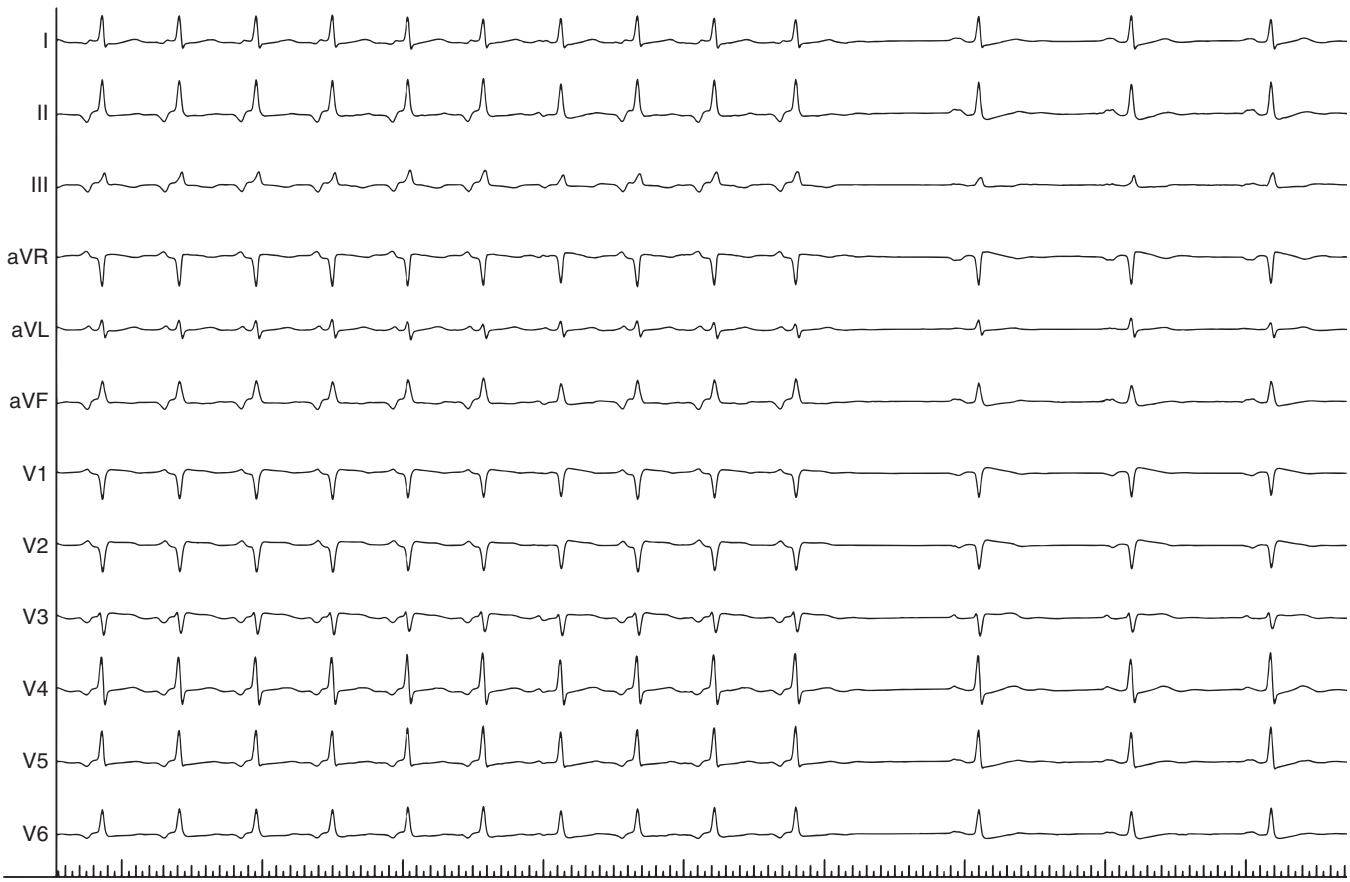


Fig. 20.5 Supraventricular Tachycardia. Surface electrocardiogram of a narrow complex supraventricular tachycardia with a long RP interval. The tachycardia terminates spontaneously and sinus rhythm ensues *(at left)*.

to retrograde conduction over the BT; **see Fig. 18.12**), AT (P wave can be eccentric or concentric), and AFL (lack of distinct isoelectric baselines between atrial deflections is suggestive of AFL, but can also be seen occasionally in AT; **see Fig. 12.6**). The P waves may not be discernible on the ECG, which suggests typical AVNRT or, less commonly, AVRT (especially in the presence of bundle branch block [BBB] contralateral to the BT).[1] It is best to characterize P waves during SVT morphologically at the outset (concentric, eccentric) rather than attaching a mode of activation (i.e., retrogradely activated); for example, upright P waves in the inferior leads, though usually due to AT, may nonetheless be retrogradely conducted over an anterior BT, and although inverted P waves in inferior leads are often retrogradely conducted, focal AT from the coronary sinus ostium (CS os) can have an identical appearance. Thus not all retrograde P waves are inverted in the inferior leads, and not all inverted P waves in inferior leads are retrogradely conducted.

Characterization of P/QRS Relationship

RP-PR Interval Ratio

In general, SVTs are classified according to the RP interval as "short RP" (RP < PR) or "long RP" (RP > PR) tachycardias (see Fig. 20.5). The RP-to-PR ratio is not diagnostic of any tachycardia type but does help in creating a differential diagnosis of the possible tachycardia types. During short RP SVTs, the ECG will show P waves inscribed within the ST-T wave with an RP interval that is less than half the tachycardia R-R interval. Such SVTs include typical AVNRT (most common), orthodromic AVRT, AT with prolonged AV conduction, and slow-slow AVNRT. A very short RP interval (less than 90 milliseconds) is consistent with typical AVNRT and excludes AVRT. In typical AVNRT, the P wave is not usually visible because of the simultaneous atrial and ventricular activation. The P wave may distort the initial portion of the QRS (mimicking a q wave in inferior leads) or lie just within the QRS (inapparent) or distort the terminal portion of the QRS (mimicking an s wave in inferior leads or r′ in lead V_1; **see Fig. 17.8**).

Long RP SVTs (see Fig. 20.1) include AT (most common), atypical (fast-slow) AVNRT, and AVRT using a slowly conducting AV BT (e.g., PJRT). If the PR interval during the SVT is shorter than that during NSR, AT and AVRT are very unlikely, and atypical AVNRT, which is associated with an apparent shortening of the PR interval, is the likely diagnosis. ATs originating close to the AV junction are also a possibility.

The PR intervals during AT are appropriate for the AT rate and are usually longer than those during NSR. The faster the AT rate, the longer the PR interval. Thus the PR interval can be shorter than, longer than, or equal to the RP interval. The PR interval may also be equal to the RR, and the P wave may fall inside the preceding QRS, thus mimicking typical AVNRT.

Slow-slow AVNRT can be associated with RP intervals and P wave morphology similar to that during orthodromic AVRT using a posteroseptal AV BT. However, although both SVTs have the earliest atrial activation in the posteroseptal region, conduction time from that site to the HB region is significantly longer in AVNRT than in orthodromic AVRT. The results are a significantly longer RP interval in lead V_1 and a significantly larger difference in the RP interval between lead V_1 and inferior leads during AVNRT. Therefore ΔRP interval (V_1–III) of more than 20 milliseconds suggests slow-slow AVNRT (sensitivity, 71%; specificity, 87%).[11]

When the P wave is not visible, typical AVNRT is the most likely diagnosis, but AT with a long PR interval (P wave is obscured within the QRS) and junctional tachycardia are also possible.

Atrial-Ventricular Relationship

SVTs with an A/V ratio of 1 (i.e., equal number of atrial and ventricular events) include AVNRT, AVRT, and AT. On the other hand, an A/V ratio during the SVT of greater than 1 indicates the presence of AV block and that the ventricles are not required for the SVT circuit, thereby excluding AVRT and suggesting either AT (most common; **see Fig. 11.7**) or AVNRT (rare; **see Fig. 17.9**). AV dissociation (even complete AV block) can be observed during AT (most common) or AVNRT (rare).[1] VA block during SVT is rare, but can occur in automatic junctional tachycardia with retrograde VA block and orthodromic AVRT using a nodofascicular or nodoventricular BT for retrograde conduction. Intra-Hisian reentry is another potential mechanism, but it is a theoretical entity whose clinical occurrence has not convincingly been demonstrated.[12–14]

QRS Morphology

The QRS morphology during SVT is usually the same as in NSR. However, SVT can present as a wide complex tachycardia (QRS duration greater than 120 milliseconds) due to rate-related aberrant conduction, preexisting intraventricular conduction disturbance (IVCD), or ventricular preexcitation over an anterogradely conducting BT.[1]

Functional aberration during SVT is much more common in orthodromic AVRT than AVNRT or AT (90% of SVTs with sustained left bundle branch block [LBBB] are orthodromic AVRTs). Thus the mere presence of LBBB aberrancy during SVT is suggestive of orthodromic AVRT, but can still occur in other types of SVTs.[15]

Preexcited SVTs include antidromic AVRT, whereby the BT is an essential part of the tachycardia circuit, and AVNRT and AT with ventricular preexcitation over a bystander BT.

QRS alternans during fast SVTs is most commonly seen in orthodromic AVRT but can also be seen with other types of SVTs as well. However, when QRS alternans occurs during a relatively slow SVT, the diagnosis is almost always orthodromic AVRT.

Effects of Interventions

Carotid sinus massage or adenosine can result in one of four possible effects: (1) temporary decrease in the atrial rate in patients with sinus tachycardia or automatic AT; (2) slowing of AVN conduction and AVN block, which can unmask atrial electrical activity—that is, reveal P waves or flutter waves in patients with AT or AFL by decreasing the number of QRS complexes that obscure the electrical baseline; (3) terminating the SVT; or (4) no effect is observed.

Carotid sinus massage or adenosine can terminate the SVT, especially if the rhythm is AVNRT or AVRT by transient slowing and block of AVN conduction and interrupting the reentry circuit; termination of focal AT can occur but is much less common. A continuous ECG tracing should be recorded during these maneuvers because the response can aid in the diagnosis and changes can be subtle (rate slowing or transient AV block). Termination of the tachycardia with a P wave after the last QRS complex is most common in AVRT and typical AVNRT and is rarely seen with AT (**see Fig. 18.36**), whereas termination of the tachycardia with a QRS complex is more common with AT, atypical AVNRT, and PJRT (**see eFig. 18.5**). If the tachycardia continues despite development of AV block, the rhythm is almost certainly AT or AFL; AVRT is excluded and AVNRT is very unlikely.[1]

ELECTROPHYSIOLOGICAL TESTING

Discussion in this section will focus on differential diagnosis of narrow QRS complex paroxysmal SVTs, including focal AT, orthodromic AVRT, and AVNRT. The goals of EP testing in these patients include the following: (1) evaluation of baseline cardiac electrophysiology; (2) induction of SVT; (3) evaluation of the mode of initiation of the SVT; (4) definition of atrial activation sequence during the SVT; (5) definition of the A/V relationship at the onset and during the SVT; (6) evaluation of the effect of BBB on the tachycardia cycle length (TCL) and VA

interval; (7) evaluation of the SVT circuit and requirement for the atria, HB, or ventricles in the initiation and maintenance of the SVT; (8) evaluation of the SVT response to programmed atrial and ventricular stimulation; and (9) evaluation of the effects of drugs and physiological maneuvers on the SVT.

Baseline Observations During Sinus Rhythm

The presence of preexcitation during NSR suggests AVRT as the likely diagnosis; however, it does not exclude other causes of SVT during which the BT is an innocent bystander. Furthermore, the absence of preexcitation during NSR does not exclude the presence of an AV BT or the diagnosis of AVRT.

Programmed Electrical Stimulation During Sinus Rhythm

The programmed stimulation protocol typically includes: (1) ventricular burst pacing from the right ventricular (RV) apex (down to the pacing cycle length [PCL] at which VA block develops); (2) single and double ventricular extrastimulus (VES; down to the ventricular effective refractory period [ERP]) at multiple PCLs (600 to 400 milliseconds) from the RV apex; (3) atrial burst pacing from the high RA and CS (down to the PCL at which 2:1 atrial capture occurs); (4) single and double atrial extrastimulations (AESs) (down to the atrial ERP) at multiple PCLs (600 to 400 milliseconds) from the high RA and CS; and (5) administration of isoproterenol infusion (0.5 to 4 μg/min) or epinephrine (0.05 to 0.2 μg/kg per minute) infusion as needed to facilitate tachycardia induction or sustenance.

Programmed Atrial Stimulation During Sinus Rhythm

Dual AVN physiology. Although the demonstration of dual AVN physiology during programmed atrial stimulation favors AVNRT as the mechanism of SVT (positive predictive value greater than 80%), it is not an uncommon finding in patients with other types of SVTs. Furthermore, failure to demonstrate dual AVN physiology does not exclude the possibility of AVNRT, and might be related to similar fast and slow AVN pathway ERPs. Then, dissociation of refractoriness of the fast and slow AVN pathways can be necessary (**see Chapter 17**).

Ventricular preexcitation. Atrial stimulation can help unmask preexcitation if it is not manifest during NSR because of fast AVN conduction, slow BT conduction, or distant (left lateral) BT location. AES and atrial pacing from any atrial site result in slowing of AVN conduction and, consequently, unmask or increase the degree of preexcitation over the AV BT (**see Fig. 18.23**). Moreover, atrial stimulation close to the BT insertion site results in maximal preexcitation and the shortest P-delta interval because of the ability to advance the activation of the AV BT down to its ERP from pacing at this site caused by the lack of intervening atrial tissue, whose conduction time and refractoriness can otherwise limit the ability of the AES to stimulate the BT prematurely (**see Fig. 18.24**).

The failure of atrial stimulation to increase the amount of preexcitation can be caused by: (1) markedly enhanced AVN conduction; (2) the presence of another AV BT; (3) pacing-induced block in the AV BT because of a long ERP of the BT (longer than that of the AVN); (4) total preexcitation already present at the basal state caused by prolonged or absent AVN-His-Purkinje system (HPS) conduction; (5) decremental conduction in the BT; or (6) the presence of a fasciculoventricular BT rather than an AV BT.

Extra atrial beats. AES and atrial pacing can trigger extra atrial beats or echo beats. Those beats can be caused by different mechanisms.

Intraatrial reentrant beats. These beats usually occur at short coupling intervals, and can originate anywhere in the atrium. Therefore the atrial activation sequence depends on the site of origin of the beat. The more premature the AES, the more likely it will induce nonspecific intraatrial reentrant beats and short runs of irregular AT or AF.

Catheter-induced atrial beats. These beats usually have the earliest activation site recorded at that particular catheter tip and have the same atrial activation sequence as the atrial impulse produced by pacing from that catheter. Portions of the catheter proximal to the tip usually do not elicit mechanically induced ectopic impulses.

AVN echo beats. AVN echoes occur in the presence of anterograde dual AVN physiology (**see Fig. 4.23**). Such beats require anterograde block of the atrial stimulus in the fast AVN pathway, anterograde conduction down the slow pathway, and then retrograde conduction up the fast pathway. AVN echo beats have several features: they appear reproducibly after a critical A_2-H_2 interval; the atrial activation sequence is consistent with retrograde conduction over the fast pathway, with the earliest atrial activation site in the HB; and the VA interval is very short, but it can be longer if the atrial stimulus causes anterograde concealment (and not just block) in the fast pathway.

AV echo beats. AV echo beats occur secondary to anterograde conduction of the atrial stimulus over the AVN-HPS and retrograde conduction over a BT (concealed or bidirectional BT). If preexcitation is manifest during atrial stimulation, the last atrial impulse inducing the echo beat will demonstrate loss of preexcitation because of anterograde block in the BT, and the atrial activation sequence and P wave morphology of the echo beat will depend on the location of the BT mediating VA conduction (**see Fig. 3.9**). These beats have a relatively short VA interval (QRS onset to earliest atrial activation) but always longer than 70 milliseconds. Moreover, the VA interval of the AV echo beat remains constant, regardless of the varying coupling interval of the AES triggering the echo beat ("VA linking"). Of note, AV echo beats also can occur secondary to anterograde conduction of the atrial stimulus over a manifest BT and retrograde conduction over the AVN, in which setting the last paced beat is associated with anterograde block in the AVN and fully preexcited QRS complex.

Programmed Ventricular Stimulation During Sinus Rhythm

Retrograde VA conduction. The absence of VA conduction (at ventricular PCLs greater than 600 milliseconds and despite isoproterenol administration) or the presence of decremental VA conduction makes the presence of a retrogradely conducting BT unlikely.

The normal AVN response to rate-incremental ventricular pacing or progressively premature single VESs is a gradual delay of VA conduction (manifest as gradual prolongation of the VA and His bundle–atrial [HA] intervals) as the PCL or VES coupling interval decreases. Nondecremental VA conduction suggests BT conduction, although fast pathway conduction in patients with AVNRT often shows minimal decrement. Nonetheless, some BTs with retrograde decremental conduction properties can also exhibit prolongation of conduction time and VA interval with ventricular pacing or VES. In addition, at short ventricular PCLs or VES coupling intervals, intramyocardial conduction delay can occur, resulting in prolongation in the VA interval; however, the local VA interval at the BT location remains unchanged. Furthermore, short ventricular PCLs or VES coupling intervals can encroach on the BT refractoriness, causing some decremental conduction, with a consequent increase in the surface VA interval and the local VA interval.

During the delivery of progressively premature single VESs, an abrupt increase in the VA conduction interval is often observed. This may be due to a variety of reasons including: (1) retrograde block in the AVN fast pathway and subsequent VA conduction over the slow pathway; (2) retrograde block in the right bundle branch (RB) and subsequent retrograde conduction over the left bundle branch (LB);

or (3) retrograde block in the BT and subsequent VA conduction only over the AVN.

Retrograde atrial activation sequence. VA conduction over the AVN produces a classic concentric atrial activation sequence starting in the anteroseptal or posteroseptal region of the RA because of retrograde conduction over either the fast or the slow AVN pathways, respectively. The duration of atrial activation is short because both atria are roughly simultaneously activated. Thus, if a normal P wave lasts 80 milliseconds (about 40 milliseconds for each atrium), a concentrically activated P wave (and total atrial activation time) approximates 40 milliseconds.

Eccentric retrograde atrial activation (i.e., lateral left atrium or RA earlier than AV junction and opposite chamber) can also occur. In the presence of a retrogradely conducting AV BT (whether manifest or concealed), atrial activation can result from conduction over the BT, over the AVN, or a fusion of both (**see Fig. 18.25**). Conduction over the BT alone is the most common pattern at short PCLs or short VES coupling intervals. In this setting, the VA conduction time is fairly constant over a wide range of PCLs and VES coupling intervals (given the absence of intraventricular conduction abnormalities or additional BTs). On the other hand, retrograde conduction over both the BT and HPS-AVN is especially common when RV pacing is performed in the presence of a left-sided BT at long PCLs or long VES coupling intervals. This occurs because it is easier to engage the RB and conduct retrogradely through the AVN than it is to reach a distant left-sided BT. In this setting, the atrial activation pattern depends on the refractoriness and conduction times over both pathways and usually exhibits a variable degree of fusion. In addition, VA conduction can proceed over the HPS-AVN alone, resulting in a normal pattern of VA conduction, or can be absent because of block in both the HPS-AVN and BT, which is especially common with short PCLs and very early VES.

An eccentric atrial activation sequence in response to ventricular stimulation suggests the presence of an AV BT mediating VA conduction (**see Fig. 18.25**). However, a concentric retrograde atrial activation sequence does not exclude the presence of a septal or paraseptal BT, or a free-wall BT located far from the pacing site, allowing for preferential VA conduction over the AVN. In addition, AVN slow pathway conduction can be associated with an eccentric atrial activation sequence in the CS. Accurate analysis of the atrial activation sequence frequently requires the use of multielectrode catheters around the tricuspid annulus and deep in the CS.[16]

Retrograde dual AVN physiology. Demonstration of retrograde dual AVN physiology during programmed ventricular stimulation suggests AVNRT (occurring most commonly during atypical AVNRT), but it can also be observed with other SVTs. Importantly, failure to demonstrate retrograde dual AVN physiology in patients with AVNRT can be the result of similar fast and slow AVN pathway ERPs, in which setting dissociation of refractoriness of the fast and slow AVN pathways is required (**see Chapter 17**).

VA block at a ventricular PCL greater than 600 milliseconds or decremental VA conduction during ventricular pacing makes the presence of a retrogradely conducting BT unlikely, except for decrementally conducting BTs and the rare catecholamine-dependent BTs. In addition, development of VA block during ventricular pacing in response to adenosine suggests the absence of a BT.

VES during His bundle refractoriness. A VES delivered when the HB is refractory (i.e., when the His potential is already manifest or within 35 to 55 milliseconds before the time of the expected His potential) that results in atrial activation is diagnostic of the presence of a retrogradely conducting BT. Because the HPS-AVN is already refractory and cannot mediate VA conduction, retrograde atrial activation from ventricular stimulation will necessarily be mediated by a BT.

Furthermore, an earlier VES that does conduct to the HB and results in an atrial activation that either precedes HB activation (**see Fig. 18.27**) or is associated with an apparent HA interval shorter than that during drive complexes indicates "atrial preexcitation" via an AV BT.

Importantly, if a VES delivered when the HB is refractory does not result in atrial activation, this does not necessarily exclude the presence of a retrogradely conducting AV BT, because such a VES can be associated with retrograde block in the BT itself (**see Fig. 18.25**). In addition, the lack of such a response does not exclude the presence of unidirectional (anterograde-only) AV BTs.

Differential-site RV pacing. Differential-site RV pacing can help exclude the presence of a retrogradely conducting septal AV BT. The response to differential-site RV pacing can be evaluated by comparing two variables between RV basal and RV apical pacing: the VA interval (i.e., the stimulus-to-atrial [S-A] interval) and atrial activation sequence (**see Fig. 18.28**).

VA interval. In the absence of a septal BT, the RV apex, although anatomically more distant from the atrium than the RV base, is nonetheless electrically closer because of its proximity to the entrance to the HPS (i.e., RB terminus). Therefore the VA interval is shorter during pacing from the RV apex than that during RV basal pacing. In contrast, in the presence of a septal BT, the RV base is closer than the RV apex to the BT ventricular insertion site. As a result, the VA interval is shorter during RV basal pacing compared to apical pacing. Therefore, when the VA interval during RV apical pacing is longer than that during RV basal pacing, a retrogradely conducting AV BT is diagnosed (**see Fig. 18.28**), and when the VA interval during RV apical pacing is shorter than that during RV basal pacing, a retrogradely conducting *septal* BT is excluded. Importantly, while this maneuver suggests AVN conduction, it may not exclude the presence of a free wall or a slowly conducting BT. It is important to ensure that the basal pacing site captures neither the HB or the RB, nor the atrium, which would yield spurious results.

Atrial activation sequence. In the absence of a retrogradely conducting BT, the atrial activation sequence remains identical regardless of the RV pacing site because retrograde VA conduction propagates only over the AVN in both settings. In contrast, in the presence of a septal BT, atrial activation results from VA conduction over the BT during pacing at the RV base (because of its immediate proximity to the BT), and over the AVN, the BT, or a fusion of both during pacing at the RV apex. Therefore a change in retrograde atrial activation sequence in response to differential RV pacing (RV base vs. RV apex) indicates the presence of an AV BT, but a constant atrial activation sequence is not helpful in excluding the presence of a BT, because AVN-HPS conduction delay can allow retrograde VA conduction to occur over the BT during RV pacing from both the apex and the base.

Limitations. This maneuver does not exclude the presence of a distant right or left free-wall BT because the site of pacing is far from the BT; as a consequence, pacing from the RV apex or RV base may result in preferential VA conduction exclusively over the AVN and a constant atrial activation sequence. This can be avoided by moving the basal pacing site closer to the putative BT location along the tricuspid or mitral annulus.[17]

In addition, this maneuver does not exclude the presence of a slowly conducting BT. The VA interval criterion identifies the actual route of VA conduction and therefore the fastest path of this conduction; hence, a slowly conducting BT would be missed in the presence of fast VA conduction over the HPS-AVN.

The occurrence of right bundle branch block (RBBB) (but not LBBB) also can alter the significance of the VA interval criterion, especially

when VA conduction propagates over the HPS-AVN. In the presence of retrograde RBBB, VA conduction occurs over the LB-HB; therefore the VA interval depends on the distance between the pacing site and the LB rather than the RB, and access of the paced wavefront to the LB can be faster for RV basilar or septal pacing compared with pacing from the RV apex (Fig. 20.6).

Of note, the exact entrance to the HPS (i.e., the terminus of the RB) is difficult to identify; the entrance site can be located in the mid-septum and not at the RV apex. In such situations, both the RV base and apex can be equidistant from the entrance to the HPS so that pacing at either location will produce a constant VA interval during retrograde conduction over the AVN. Therefore patient-to-patient variability with regard to the distance of the pacing catheter to the RB terminus can potentially introduce conflicting results. This problem is largely resolved by first pacing from the HB region (while avoiding HB capture) and then moving the pacing catheter in a stepwise fashion along the septum toward the RV apex. Pacing from sequential sites in this path brings the catheter closer to the RB terminus (entrance of the HPS), as reflected by shortening of the VA interval for AVN conduction, but not for BT conduction. As the pacing catheter is moved farther apically, the pacing sites become less useful diagnostically because the relative distance from the RB terminus to the insertion of the BT becomes less clear.[17]

Retrograde RBBB during VES. Retrograde RBBB occurs frequently during VES testing, and it can be diagnosed by observing the retrograde His potential during the drive train and its abrupt delay following the VES. Often, however, it is difficult to visualize the retrograde His potential during the pacing train; nevertheless, the sudden appearance of an easily distinguished retrograde His potential, separate from the ventricular electrogram following the VES, can be sufficient to recognize retrograde RBBB.

Prolongation of the VH interval is observed on development of retrograde RBBB because conduction must traverse the interventricular septum (which requires approximately 60 to 70 milliseconds in normal hearts), conduct retrogradely via the LB, and ascend to reach the HB. Although an increase in the VH interval necessarily occurs with retrograde

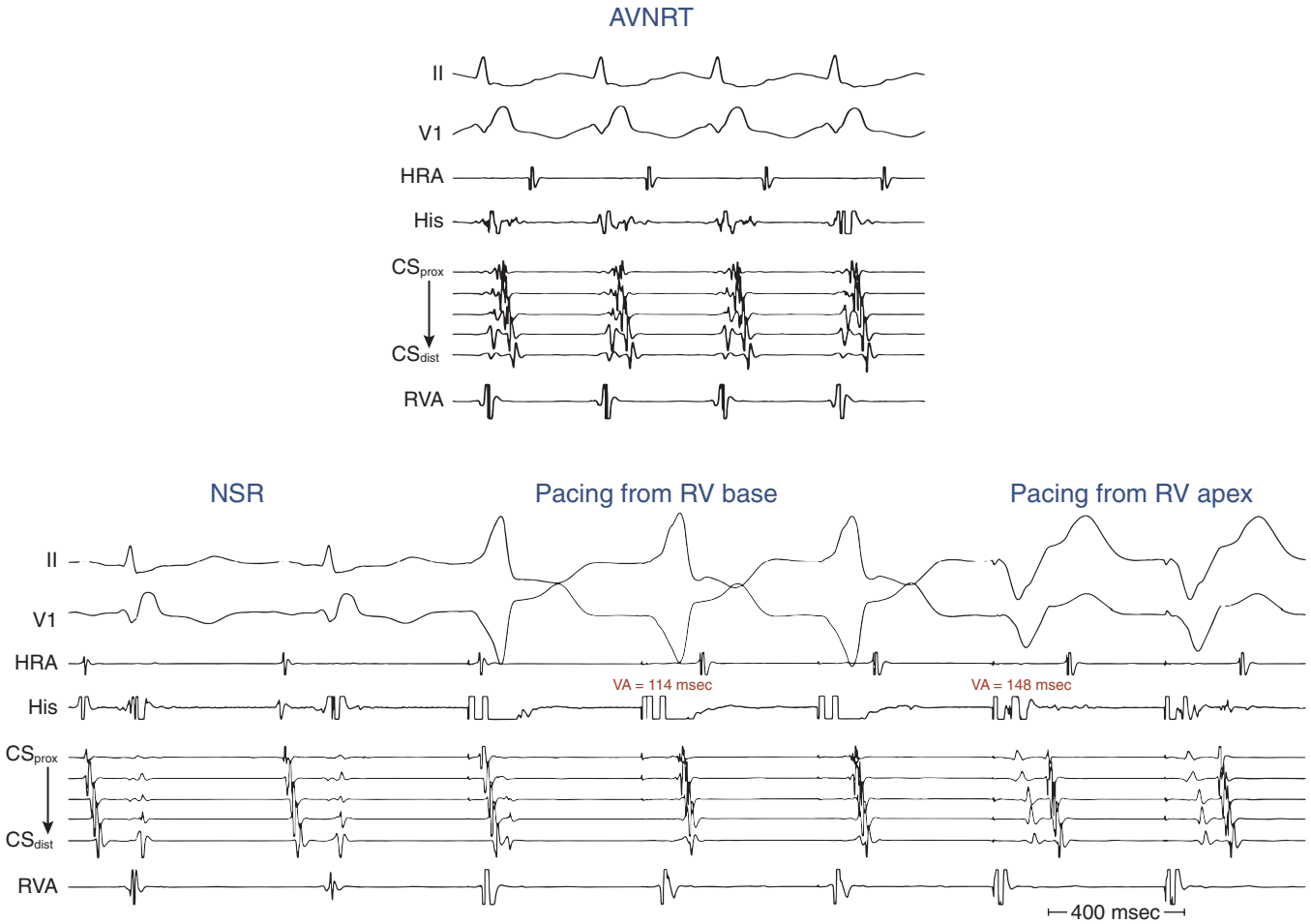


Fig. 20.6 Differential-Site Right Ventricular *(RV)* Pacing During Sinus Rhythm in the Presence of Right Bundle Branch Block (RBBB). Comparison between RV basal versus apical pacing during normal sinus rhythm *(NSR, bottom panel)* in a patient with typical atrioventricular nodal reentrant tachycardia *(AVNRT)* and RBBB *(top panel)* but no bypass tracts (BTs). RBBB is also observed during NSR (beginning of *bottom panel*). In the absence of a retrogradely conducting BT, pacing from the RV apex is expected to result in a shorter ventriculoatrial *(VA)* interval than pacing from the RV base. However, in this case, the presence of RBBB produces misleading results because retrograde VA conduction occurs over the left bundle branch–His bundle, and the VA interval depends on the distance between the pacing site to the left bundle branch, as opposed to the right bundle branch. *CS_{dist}*, Distal coronary sinus; *CS_{prox}*, proximal coronary sinus; *HRA,* high right atrium; *RVA,* right ventricular apex.

RBBB, whether a similar increase occurs in the VA interval depends on the nature of VA conduction (over the AVN vs. BT).

Measurement of the effect of the development of retrograde RBBB during VES on the retrograde VH and VA intervals can help in distinguishing between retrograde AVN and BT conduction. In the absence of a BT, the AVN can be activated in a retrograde fashion only after retrograde activation of the HB; as a consequence, VA activation will necessarily be delayed with retrograde RBBB, and the increase in the VA interval will be similar to the increase in the VH interval. On the other hand, when retrograde conduction is via a BT, there will be no expected increase in the VA interval when retrograde RBBB is induced. Thus the increase in the VA interval is minimal and always less than the increase in the VH interval.[18]

Extra ventricular beats. Ventricular stimulation can trigger extra ventricular beats or echo beats. These beats can be caused by different mechanisms.

Bundle branch reentrant beats. During RV stimulation at close coupling intervals, progressive retrograde conduction delay and block occur in the RB, so that retrograde HB activation occurs via the LB. At this point, the His potential usually follows the local ventricular electrogram. Further decrease in the VES coupling interval produces an increase in retrograde HPS conduction delay. When a critical degree of HPS delay (S_2-H_2 interval) is attained, the impulse can return down the initially blocked RB and result in a QRS of similar morphology to the paced QRS at the RV apex—specifically, it will look like a typical LBBB pattern with left axis deviation because ventricular activation originates from conduction over the RB. The HV interval of the bundle branch reentry beat is usually longer than or equal to the HV interval during NSR. Retrograde atrial activation over the AVN, if present, follows the His potential (**see Fig. 4.26**); if over a left-sided BT, atrial activation follows the QRS.

AVN echo beats. AVN echoes are caused by reentry in the AVN in patients with retrograde dual AVN physiology (**see Fig. 17.13**). The last paced beat conducts retrogradely up the slow AVN pathway and then anterogradely down the fast pathway to produce the echo beat. AVN echoes appear reproducibly after a critical H_2-A_2 interval (or V_2-A_2 interval, when the His potential cannot be seen), and manifest as extra beats with a normal anterograde QRS morphology and atrial activity preceding the His potential before the echo beat. This phenomenon can occur at long or short coupling intervals and depends only on the degree of retrograde AVN conduction delay. In most cases, this delay is achieved before the appearance of a retrograde His potential beyond the local ventricular electrogram (i.e., before retrograde block in the RB).

AV echo beats. These beats occur secondary to retrograde block in the HPS-AVN and retrograde VA conduction over an AV BT, followed by anterograde conduction over the AVN. Alternatively, echo beats can result from retrograde block in the BT and retrograde VA conduction over the AVN-HPS, followed by anterograde conduction over the AV-BT. In the first setting, the echo beat displays a narrow QRS; in the latter setting, the echo beat is fully preexcited (**see Fig. 18.25**).

Intraventricular reentrant beats. This response occurs most commonly in the setting of a cardiac pathological condition, especially coronary artery disease, and usually occurs at short coupling intervals. It can have any QRS morphology, but more often RBBB than LBBB in patients with prior myocardial infarction. These responses are usually nonsustained (1 to 30 complexes) and typically polymorphic. In patients without prior clinical ventricular arrhythmias, such responses are of no clinical significance.

Catheter-induced ventricular beats. Such beats usually have the earliest ventricular activation site recorded at that particular catheter tip and have the same QRS morphology as the QRS produced by pacing from that catheter.

Para-Hisian Pacing During Sinus Rhythm

Concept of para-Hisian pacing. The para-Hisian pacing site is unique because it is anatomically close but electrically distant from the HB. Para-Hisian pacing at high output simultaneously captures the HB or proximal RB, as well as the adjacent ventricular myocardium. At lower output, direct HB-RB capture is lost and retrograde activation of the HB is delayed because the HB and RB are insulated from the adjacent myocardium and the peripheral inputs to the Purkinje system are located far from the para-Hisian pacing site. By maintaining local ventricular capture while intermittently losing HB-RB capture, retrograde VA conduction can be classified as dependent on the timing of local ventricular activation (BT), HB activation (AVN), or both (fusion).

Para-Hisian pacing can result in capture of the ventricle (indicated by a wide paced QRS), the atrium (indicated by atrial activation in the HB region immediately following the pacing artifact), the HB (indicated by narrow paced QRS), or any combination of these (**see Fig. 18.29**). Careful attention must be given to minimize the atrial signal seen on the recording from the pacing electrode pair to ensure that local atrial capture does not occur during pacing.

Technique of para-Hisian pacing. Ideally, two quadripolar catheters (one for pacing and another for recording) or a single octapolar catheter (for both pacing and recording) is placed at the distal HB-RB region. Alternatively, a single quadripolar HB catheter (which is typically used during a diagnostic EP study) is used, taking into account that such an approach would limit the ability to record the retrograde His potential and HA interval.[19]

Overdrive ventricular pacing is performed (from the pair of electrodes on the HB catheter that records activation of the distal HB-RB) at a long PCL (greater than 500 milliseconds) and high output. During pacing, direct HB-RB capture is indicated by narrowing of the paced QRS width. The pacing output and pulse width are then decreased until the paced QRS widens, which is associated with a delay in the timing of the retrograde HB potential, indicating loss of HB-RB capture. The pacing output is increased and decreased to gain and lose HB-RB capture, respectively, while local ventricular capture is maintained. Occasionally, the HB can be captured uniquely (without myocardial capture), resulting in a QRS identical to the patient's normally conducted QRS.[19]

Response to para-Hisian pacing. When the ventricle and HB are captured simultaneously, the wavefront activates the ventricles over the HPS and results in a relatively narrow QRS. The wavefront can also travel retrogradely over the AVN to activate the atrium with an SA interval (i.e., the interval from the pacing stimulus to the atrial electrogram) that represents conduction time over the proximal part of the HB and AVN (i.e., SA interval = HA interval) because the onset of ventricular activation and HB activation occur simultaneously (i.e., stimulus–His bundle [SH] interval = 0).[19]

When the ventricle is captured but not the atrium or HB, the wavefront activates the ventricles by muscle-to-muscle conduction, resulting in a wide QRS with LBBB morphology caused by pacing in the RV. Once the wavefront reaches the RV apex, it conducts retrogradely up the RB and then over the HB and AVN to activate the atrium. In this setting, the SA interval represents the conduction time from the RV base to the HB (SH interval) plus the conduction time over the HB and AVN (HA interval). Thus, normally (in the absence of a retrogradely conducing BT), para-Hisian pacing results in a shorter SA interval when the HB (or HB plus RV) is captured than the SA interval when only the ventricle is captured.[19]

In the presence of a septal AV BT, the SA interval usually remains fixed regardless of whether the HB is being captured, because in both situations the paced impulse travels retrogradely over the BT, with constant conduction time to the atrium as long as local ventricular myocardium is being captured. Atrial activation in this setting can be secondary to activation over the BT, especially when only the ventricle is captured, or a result of fusion of conduction over both the BT and AVN, especially when both the ventricle and the HB are captured. Nevertheless, because VA conduction time over the BT is faster than that over the AVN, the timing of the earliest atrial activation (i.e., the SA and local VA intervals) remains constant, regardless of whether HB-RB capture occurs and regardless of whether atrial activation occurs exclusively over the AV BT or as a fusion of conduction over both the AV BT and AVN.[19]

Seven patterns of response to para-Hisian pacing can be observed (Box 20.1; **see Figs. 18.29 and 18.30**). In patients with retrogradely conducing BTs, in whom retrograde conduction occurs over both the AVN and BT during para-Hisian pacing, the amount of atrial myocardium activated by each of the two pathways (atrial fusion) is dependent on four variables: (1) the magnitude of delay in retrograde activation of the HB (i.e., SH interval); (2) retrograde conduction time over the AVN (HA interval); (3) intraventricular conduction time from the para-Hisian pacing site to the ventricular end of the BT (SV_{BT}); and (4) retrograde conduction time over the BT (VA_{BT}). The first two variables (SH plus HA) form the SA interval resulting from retrograde VA conduction over the AVN, and the latter two variables (SV_{BT} plus VA_{BT}) form the SA interval resulting from retrograde VA conduction over the BT. The amount of the atria activated by the AVN is greater during HB-RB capture, secondary to a minimal SH interval (i.e., SA interval = HA interval). Loss of HB-RB capture results in prolongation of the SH interval and, therefore, an increase in the amount of atria activated by the BT, resulting in a change in the retrograde atrial activation sequence. Consequently, a change in the retrograde atrial activation sequence with loss of HB-RB capture always indicates the presence of

BOX 20.1 Response Patterns to Para-Hisian Pacing

Pattern 1 (AVN/AVN Pattern)

- Retrograde conduction occurs exclusively over the AVN regardless of whether the HB-RB is captured.
- Loss of HB-RB capture results in an increase in the SA interval in all electrograms equal to the increase in the SH interval, with no change in the atrial activation sequence. The HA interval remains essentially the same.
- This response indicates that retrograde conduction is dependent on HB activation and not on local ventricular activation.
- This pattern is observed in all patients with AVNRT and is not observed in any patient with a septal or right free-wall BT. However, this pattern can be observed in some patients with a left free-wall BT or PJRT, in which case retrograde AVN conduction masks the presence of retrograde BT conduction.

Pattern 2 (BT-BT Pattern)

- Retrograde conduction occurs exclusively over a single BT.
- The SA interval is identical during HB-RB capture and noncapture, indicating that retrograde conduction is dependent on local ventricular activation and not on HB activation.
- This pattern does not exclude the presence of retrograde conduction over the AVN with longer conduction time or a second BT with longer conduction time or located far from the pacing site.

Pattern 3 (BT-BT_L Pattern)

- Retrograde conduction occurs exclusively over a BT.
- Loss of HB-RB capture is associated with a delay in the timing of ventricular activation close to the BT. This results in an increase in the SA interval in all electrograms, with no change in the atrial activation sequence. The local VA interval, recorded close to the BT, remains approximately the same. The increase in the SA interval is less than the increase in the SH interval. Therefore the HA interval is shortened with loss of HB-RB capture, indicating that retrograde conduction cannot be occurring over the AVN. Two mechanisms have been identified accounting for the delay in timing of ventricular activation close to the BT.
- Activation of the HPS results in earlier ventricular activation near some BTs located far from the para-Hisian pacing site, such as left lateral or anterolateral BTs.
- Decreasing the pacing output to lose HB-RB capture occasionally results in a small delay in ventricular activation close to the pacing site.
- Pattern 3 is referred to as the BT-BT_L pattern, where BT_L refers to a lengthening of the SA interval with loss of HB-RB capture.

Pattern 4 (AVN-BT Pattern)

- Loss of HB-RB capture is associated with atrial activation exclusively over the BT.
- Loss of HB-RB capture results in an increase in SA and local VA intervals in all electrograms, with the least increase occurring in the electrogram closest to the BT.
- The HA interval shortens, indicating that the atrium near the AVN is activated by the BT before retrograde conduction over the AVN is complete.

Pattern 5 (AVN-Fusion Pattern)

- Loss of HB-RB capture results in activation of part of the atria by the AVN and part by the BT.
- Loss of HB-RB capture is associated with an increase in SA and local VA intervals in all electrograms.
- The HA interval remains constant, indicating that part of the atria was still activated by the AVN.

Pattern 6 (Fusion-BT Pattern)

- Loss of HB-RB capture results in atrial activation exclusively over the BT.
- Loss of HB-RB capture is associated with no change in the SA or local VA intervals recorded near the BT.
- In the HB electrogram, the SA interval increases, but not as much as the SH interval, leading to a decrease in the HA interval. This indicates that the atrial myocardium in that region is no longer activated by the AVN.

Pattern 7 (Fusion-Fusion Pattern)

- The atria continue to be activated by both the AVN and the BT during loss of HB-RB capture, with more of the atria activated by the BT than during HB-RB capture.
- Like pattern 6, loss of HB-RB capture is associated with minimal change in the SA or local VA intervals recorded close to the BT; however, the HA interval remains essentially the same, indicating that part of the atria is still activated by the AVN.

AVN, Atrioventricular node; *AVNRT,* atrioventricular nodal reentrant tachycardia; *BT,* bypass tract; *HA,* His bundle–atrial; *HB-RB,* His bundle–right bundle branch; *HPS,* His-Purkinje system; *PJRT,* permanent junctional reciprocating tachycardia; *SA,* stimulus-atrial; *SH,* stimulus–His bundle; *VA,* ventriculoatrial.

retrograde conduction over both the BT and AVN. There are four such patterns (patterns 4 through 7). In patterns 4 and 5, HB-RB capture is associated with activation of the atria exclusively by retrograde conduction over the AVN. In patterns 6 and 7, HB-RB capture results in atrial activation over both the AVN and the BT.[19]

Interpretation of results of para-Hisian pacing. The response to para-Hisian pacing can be determined by comparing the following four variables between HB-RB capture and noncapture while maintaining local ventricular capture and no atrial capture: (1) atrial activation sequence, (2) SA interval, (3) local VA interval, and (4) HA interval (**see Figs. 18.29 and 18.30**).

The SA interval is defined as the interval between the pacing stimulus and atrial electrogram. It should be recorded at multiple sites, including those close to the site of earliest atrial activation during SVT.

The local VA interval is defined as the local ventricular-to-atrial electrogram interval in the electrode position with the earliest retrograde atrial activation time. For the local VA to be relied on, it actually has to be measured at the site of earliest atrial activation (this requires positioning a catheter at the site of earliest atrial activation recorded during SVT). The high RA catheter, for example, may not be satisfactory for evaluation of the local VA interval in the presence of a septal BT.

The HA interval is recorded in the HB electrogram; however, this measurement can be obtained only if two catheters are placed in the HB position (one catheter for pacing and a second catheter for recording) or if an octapolar catheter is used for pacing and sensing around the HB. The use of a single quadripolar HB catheter, which is typically used during a diagnostic EP study, negates the ability to record the retrograde His potential and HA interval during pacing. However, the combination of the SA and local VA intervals is sufficient to identify the presence of a retrograde BT.

If the SA (and local VA) interval at any site remains fixed, regardless of whether HB-RB capture occurs, while the HA interval shortens, retrograde conduction is occurring only over an AV BT. In this setting, the HA interval shortens on loss of HB-RB capture because the HB and atrium are activated in parallel and HB activation is delayed because of prolongation of the SH interval, while atrial activation timing remains unchanged because it results from retrograde conduction over the AV BT and is independent of the timing of HB activation. On the other hand, if the SA (and local VA) interval increases in all electrograms (including the electrode recording the earliest atrial activation) coincident with loss of HB-RB capture, while the HA interval remains essentially the same, retrograde conduction is occurring only over the AVN.

An identical retrograde atrial activation sequence during HB-RB capture and noncapture indicates that retrograde conduction is occurring over the same system (either the BT or AVN) and does not help prove or exclude the presence of a BT (especially a septal BT; **see Fig. 18.30**). A change in the retrograde atrial activation sequence with loss of HB-RB capture, however, indicates the presence of retrograde conduction over both a BT and the AVN. Morphological change in the atrial electrogram recorded at the AV junction without overlapping the ventricular electrogram also seems to have diagnostic significance, indicating the presence of both BT and AVN conduction.

Limitations of para-Hisian pacing. The location of the BT and the retrograde conduction time over the BT must be taken into account when interpreting the results of para-Hisian pacing. For superoparaseptal BTs, the SV_{BT} interval is short. For BTs located progressively farther from the para-Hisian pacing site, the SV_{BT} increases progressively. This is not a significant factor for midseptal, posteroseptal, or most right free-wall BTs. However, for left free-wall BTs, which are located far from the pacing site, the SV_{BT} interval can be sufficiently long to have the entire atria activated by the AVN, even during loss of HB-RB capture. In this setting, para-Hisian pacing can produce an AVN retrograde conduction pattern, regardless of whether the HB-RB is captured (pattern 1: AVN-AVN), and fail to identify the presence of retrograde BT conduction (because of the long SV_{BT}). However, a left lateral BT should not be a diagnostic challenge because of the obvious eccentric retrograde atrial activation sequence during orthodromic AVRT, and para-Hisian pacing is performed mainly to investigate the presence of a septal BT. In addition, for BTs located far from the para-Hisian pacing site, it is important to record atrial activation close to the suspected site of the BT. Otherwise, the change in the atrial activation sequence may not be identified, incorrectly suggesting that retrograde conduction is occurring over just the AVN. This is most likely to occur in patients with short retrograde AVN conduction (short HA interval) and a BT located far from the pacing site.

Para-Hisian pacing may fail to identify retrograde conduction over a slowly conducting BT (e.g., PJRT) because of the long VA_{BT} interval. Performing para-Hisian entrainment or resetting during SVT can help in these situations (see later). In addition, although para-Hisian pacing during NSR can help prove the presence of an AV BT, it does not show whether that BT is operative during the SVT.

In patients with very proximal retrograde RBBB, RB capture may fail to produce early retrograde activation of the HB, limiting the use of para-Hisian pacing in these patients. This observation suggests that HB-RB capture actually represents capture of the proximal RB and not HB capture. This is supported by the observation that, during HB-RB capture, the HB potential is often recorded 10 to 20 milliseconds after the pacing stimulus. Importantly, para-Hisian pacing has been performed successfully in many patients with more distal RBBB (**see Fig. 18.29**).

Assurance of lack of atrial capture by the pacing stimulus is important for correct interpretation of the results of para-Hisian pacing. Atrial capture is indicated by a very short SA interval in the electrodes just proximal to the pacing electrodes. It is sometimes helpful to withdraw the para-Hisian pacing catheter until atrial capture alone is seen; if the SA interval in the presence of ventricular capture is not longer than this, atrial capture was also present and the test should be repeated at a more distal pacing site.

Dual-Chamber Sequential Extrastimulation During Sinus Rhythm

Dual-chamber sequential extrastimulation is a useful maneuver for identifying retrogradely conducting AV BTs, even slowly conducting BTs and BTs with decremental properties. This maneuver relies on concealed AVN conduction during a critically timed AES to cause transient retrograde AVN blockade at the time a VES is delivered, thereby allowing the BT to become manifest with the VES (analogous to delivering a VES during HB refractoriness).[20]

The dual-chamber sequential extrastimulation maneuver consists of an eight-beat drive train of simultaneous atrial and RV pacing at 600 milliseconds, followed by an AES (A_2) delivered at a coupling interval equal to the AVN ERP, followed by a VES (V_2) delivered at a coupling interval equal to the drive train cycle length (CL) (600 milliseconds). Repeat drives are then performed with decrements of 10 milliseconds for V_2 until VA block is observed.[20]

The critically timed A_2 prolongs the AVN refractory period via concealed anterograde conduction, causing V_2 to block in the AVN when it would have conducted had A_2 not been delivered. If a BT is present, V_2 conducts back to the atrium while the AVN remains refractory, resulting in a retrograde atrial activation pattern consistent with exclusive BT conduction. Although there can also be some degree of concealed anterograde conduction into the BT during A_2 stimulation, the more pronounced decremental properties of AVN tissue should

prolong AVN refractoriness to a greater degree than that of the BT, allowing exclusive retrograde conduction over the BT to remain intact during V_2 stimulation.[20]

This maneuver has several potential limitations. First, atrial ERP may exceed anterograde AVN ERP. In addition, local atrial ERP at the site of BT insertion can render the atrium refractory to the wavefront traveling retrogradely over the BT. Therefore atrial pacing during this maneuver ideally should be performed at a site in close proximity to the atrial insertion of the BT if possible. Furthermore, the AES may cause anterograde concealed conduction in the BT, potentially resulting in BT conduction block during delivery of V_2. The success of this pacing maneuver relies on the differential effects of concealed conduction into the AVN and BT, with greater extension of refractoriness in the former than the latter.[20]

Induction of Tachycardia

Initiation by Programmed Atrial Stimulation

Inducibility. All types of paroxysmal SVTs can be inducible with atrial stimulation (except automatic AT). Initiation can require catecholamines (isoproterenol) with any type of SVT, and this observation does not help for differential diagnosis. However, automatic AT can start spontaneously (without atrial stimulation) during isoproterenol or epinephrine infusion.

SVT initiation that is reproducibly dependent on a critical atrial–His bundle (AH) interval is classic for typical AVNRT (**see Fig. 17.11**). This is because anterograde block in the fast pathway in conjunction with sufficient delay in anterograde conduction over the slow pathway ("critical AH interval") are necessary to allow for recovery of the fast pathway to conduct retrogradely and initiate AVN reentry. Atypical AVNRT is usually initiated with modest prolongation of the AH interval along the fast pathway with anterograde block in the slow pathway, followed by retrograde slow conduction over the slow pathway. Therefore a critical AH interval delay is not obvious (**see Fig. 17.12**).

Orthodromic AVRT usually requires some AV delay for initiation; however, the delay can occur anywhere along the AVN-HPS axis. In patients with baseline manifest preexcitation, initiation of orthodromic AVRT is usually associated with anterograde block in the AV BT and loss of preexcitation following the initiating atrial stimulus, which would then allow that BT to conduct retrogradely during the SVT.

No delay in the AH or PR interval is required for initiation of AT, although it can occur. AV block can also occur at initiation. Automatic ATs cannot be reproducibly initiated by AES or atrial pacing.

Warm-up. Progressive shortening of the TCL for several beats (warm-up) before its ultimate rate is achieved is characteristic of automatic AT, but may occur in other SVTs as well.

VA interval. If the VA interval of the first tachycardia beat is reproducibly identical to that during the rest of the SVT, AT is very unlikely, and such "VA linking" is suggestive of typical AVNRT and orthodromic AVRT.

Initiation by Programmed Ventricular Stimulation

Inducibility. Ventricular stimulation commonly induces AVRT and AVNRT. On the other hand, it is uncommon to initiate AT with VES or ventricular pacing because decremental retrograde conduction over the AVN prevents adequate prematurity of atrial activation.

Postpacing intervals. If SVT is induced during VES, the interval between the VES (delivered from the RV apex) that initiates SVT and the first cycle of tachycardia can provide information about the circuit that are essentially equivalent to those observed with ventricular entrainment (see later). The interval from the VES to atrial activation (surface VA or "S-A" interval) is compared to the surface VA interval during SVT, and the post-VES return cycle (i.e., the interval from the VES to the subsequent RV apical depolarization) is compared to the TCL. An SA interval that exceeds the surface VA interval during SVT by less than 85 milliseconds is consistent with orthodromic AVRT. Similarly, when the post-VES return cycle exceeds the TCL by less than 115 milliseconds, orthodromic AVRT is indicated. The small differences between those intervals are related to the proximity of the RV apical pacing site to the AVRT reentry circuit. Larger differences in these intervals are observed in the setting of AVNRT or orthodromic AVRT utilizing a left lateral BT or a septal BT with decremental conduction. The unique advantage of this technique is that they do not have a requirement for sustained tachycardia that can be successfully entrained.[21]

Furthermore, when induction of the SVT is achieved by ventricular pacing at a CL similar to the subsequent TCL or by a VES that activates the HB at a coupling interval (i.e., H_1-H_2 interval) similar to the H-H interval during the SVT (i.e., similar to the TCL), the HA interval following the initiating ventricular stimulus is then compared with that during the SVT. During AVNRT, the HA interval of the ventricular stimulus initiating the SVT is longer than the HA interval during the SVT, because both the HB and atrium are activated in sequence during ventricular stimulation but in parallel during AVNRT (**see Fig. 17.14**). This is even exaggerated by the fact that the AVN usually exhibits greater decremental conduction with repetitive engagement of impulses than to a single impulse at a similar coupling interval. Therefore the more prolonged the HA interval with the initiating ventricular stimulus, the more likely the SVT is AVNRT. On the other hand, if the SVT uses an AV BT for retrograde conduction, the HA interval during the initiating ventricular stimulus (at a coupling interval comparable to the TCL) is shorter than that during orthodromic AVRT because the HB and atrium are activated in parallel during ventricular pacing (when atrial activation is mediated by retrograde BT conduction), but in sequence during SVT.

Tachycardia Features

Atrial Activation Sequence

During typical AVNRT, the initial site of atrial activation is usually recorded in the HB catheter at the apex of the triangle of Koch. In contrast, the initial site of atrial activation during atypical AVNRT is usually recorded at the base of the triangle of Koch or CS os (**see Fig. 17.2**). On the other hand, in orthodromic AVRT, the initial site of atrial activation depends on the location of the AV BT, but is always near the AV groove, without multiple breakthrough points. It is comparable to that during ventricular pacing when VA conduction occurs exclusively over the AV BT. The atrial activation sequence during AT depends on the origin of the AT, and can simulate that of other types of SVTs.

In summary, eccentric atrial activation during SVT excludes typical and atypical AVNRT, except for the left variant of AVNRT, during which the earliest atrial activation occurs in the proximal or mid-CS. Furthermore, an eccentric atrial activation sequence that originates away from the AV rings is diagnostic of AT and excludes both AVNRT and AVRT.

Atrial-Ventricular Relationship

PR-RP intervals. During AT, the PR interval is appropriate for the AT rate and is usually longer than that during NSR. The faster the AT rate, the longer the PR interval. Thus the PR interval can be shorter, longer, or equal to the RP interval. The PR interval can also be equal to the R-R and the P wave can then fall within the preceding QRS, mimicking typical AVNRT.

During typical AVNRT, the RP interval is very short (−40 to 75 milliseconds); in contrast, during atypical fast-slow AVNRT, the RP interval is longer than the PR interval. On the other hand, during orthodromic AVRT, the RP interval is short but longer than that in typical AVNRT because the wavefront has to activate the ventricle before it

reaches the AV BT and subsequently conduct retrogradely to the atrium. Thus the ventricle and atrium are activated in sequence, in contrast to AVNRT, during which the ventricle and atrium are activated in parallel, resulting in abbreviation of the VA interval. Consequently, a very short VA interval (less than 70 milliseconds) or short V–high RA interval (less than 95 milliseconds) largely excludes orthodromic AVRT, and favors typical AVNRT. However, VA intervals shorter than 70 milliseconds have occasionally been observed in adult patients with orthodromic AVRT utilizing left lateral or left posteroseptal BTs.[22,23]

Because the retrograde limb of the reentry circuit in PJRT is the slow BT (which conducts more slowly than the AVN), the RP interval is longer than the PR interval, similar to fast-slow AVNRT (**see Figs. 18.14 and 18.39**).

AV block. The presence of AV block during SVT excludes AVRT, is uncommon during AVNRT, and strongly favors AT (Fig. 20.7). AV block occurs commonly during AT, with either Wenckebach periodicity or fixed-ratio block. AV block may also occur during AVNRT because of block below the reentry circuit (usually below the HB and infrequently in the lower common pathway), which can occur especially at the onset of the SVT, during acceleration of the SVT, and following a PVC or a VES (**see Fig. 17.9**).

VA block. VA block during SVT is a rare phenomenon, and may occasionally be observed in infraatrial SVTs, including junctional ectopic tachycardia and orthodromic AVRT using a nodofascicular or nodoventricular BT for retrograde conduction. VA block during AVNRT has been reported (**see eFig. 17.2**). Intra-Hisian reentry is another potential mechanism, but it is a theoretical entity whose clinical occurrence has not been convincingly demonstrated.[12–14]

Variation of the P/QRS relationship. Spontaneous changes in the PR and RP intervals with fixed atrial-atrial (A-A) interval favor AT and exclude orthodromic AVRT (Fig. 20.8). On the other hand, spontaneous changes in TCL accompanied by a constant VA interval ("VA linking") suggest orthodromic AVRT (**see Fig. 18.36**). During orthodromic AVRT, the RP interval remains fixed, regardless of oscillations in TCL from whatever cause or changes in the PR (AH) interval. Thus the RP/PR ratio may vary, and the TCL is most closely associated with the PR interval (i.e., anterograde slow conduction). In contrast to the classic fast AV BTs, the RP interval during PJRT is not fixed because the BT serving as the retrograde limb of the reentrant circuit has decremental properties.

Variation of the P/QRS relationship (with changes in the AH interval, HA interval, and AH/HA ratio), with or without block, can occur during AVNRT, especially in atypical or slow-slow AVNRT. This phenomenon usually occurs during initiation or termination of the tachycardia or in cases of nonsustained tachycardias, likely because of decremental conduction in the lower common pathway.

The ECG manifestation of P/QRS variations, with or without AV block during tachycardia, should not be misdiagnosed as AT; they can be atypical or, rarely, typical forms of AVNRT. Moreover, the variations could be of such magnitude that a long RP tachycardia can masquerade for brief periods of time as short RP tachycardia.

Oscillation in Tachycardia Cycle Length

Analysis of TCL variability can provide useful diagnostic information that is available even when episodes of SVT are nonsustained. TCL variability of at least 15 milliseconds in magnitude was found to occur

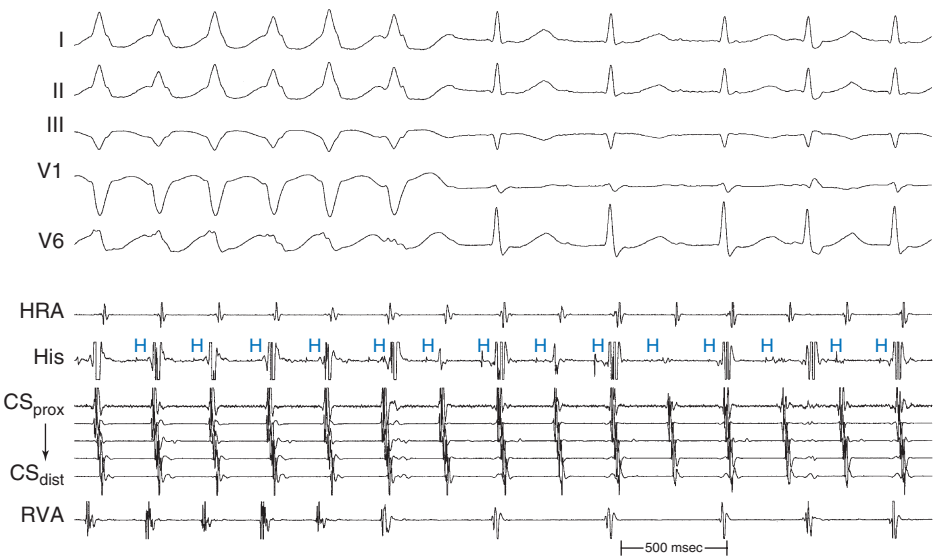


Fig. 20.7 Supraventricular Tachycardia (SVT) With Concentric Atrial Activation Sequence and Intermittent Atrioventricular (AV) Block. *At left,* A wide complex tachycardia with left bundle branch block (LBBB) pattern and 1:1 AV ratio is shown. Simultaneous atrial and ventricular activation is observed, excluding atrioventricular reentrant tachycardia (AVRT) as the mechanism of the tachycardia, and favoring typical atrioventricular nodal reentrant tachycardia (AVNRT) or atrial tachycardia (AT) with a long PR interval. *At right,* 2:1 AV block is observed without disruption of the tachycardia. Normalization of QRS morphology is observed during the period of 2:1 AV block, suggesting that wide QRS morphology during 1:1 AV conduction was a result of functional LBBB. The development of AV block with continuation of the tachycardia confirms that the ventricle is not part of the tachycardia circuit and, thus, excludes AVRT. The presence of His potentials, even during the blocked beats, suggests that the block is infra-Hisian. The observation of AV block favors AT, but does not exclude AVNRT. The ventriculoatrial (VA) interval remains constant following both the narrow and wide QRS complexes, which suggests AVNRT. Other pacing maneuvers confirmed that this SVT was in fact typical AVNRT. *CS_{dist},* Distal coronary sinus; *CS_{prox},* proximal coronary sinus; *HRA,* high right atrium; *RVA,* right ventricular apex.

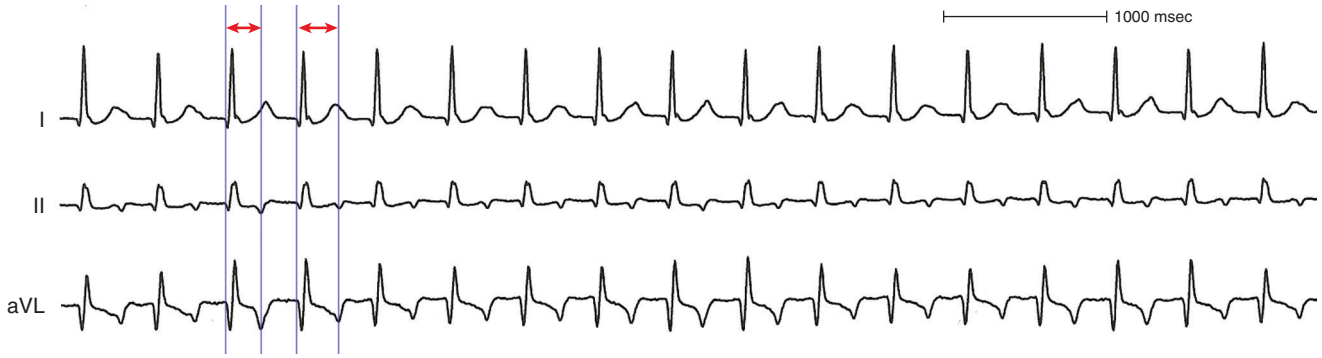


Fig. 20.8 Supraventricular Tachycardia (SVT) With Variable RP Intervals. Three surface electrocardiogram leads are shown. The P waves are inscribed within the T waves, and are more discernible with negative polarity in lead aVL. Note that the RP interval varies during the SVT *(arrows)* with constant atrial-atrial intervals, consistent with atrial tachycardia as the mechanism of the SVT and excludes orthodromic atrioventricular reentrant tachycardia.

in up to 73% of paroxysmal SVTs and was equally prevalent in AT, AVNRT, and orthodromic AVRT.

Changes in atrial CL preceding similar changes in subsequent ventricular CL strongly favor AT or atypical AVNRT (**see Fig. 17.15**). In contrast, when the change in atrial CL is predicted by the change in preceding ventricular CL, typical AVNRT (**see eFig. 17.2**) or orthodromic AVRT is the most likely mechanism (**see Fig. 18.36**).

The AVN participates either actively or passively in all types of narrow complex SVTs and the AV interval can vary depending on the preceding atrial CL and autonomic tone. A change in anterograde or retrograde AVN conduction can result in TCL variability in AVNRT or orthodromic AVRT. However, during AT, changes in anterograde AVN conduction produce only changes in ventricular CL, whereas the atrial CL remains unaffected. Therefore spontaneous changes in the ventricular CL in conjunction with fixed A-A interval (typically manifesting as variations in the PR and RP intervals) favor AT.[24]

Changes in atrial CL during AT result from changes in the CL of the atrial focus itself, and this causes similar variability in the conducted ventricular CL. Therefore, when there is CL variability in both the atrium and ventricle, changes in atrial CL during AT would be expected to precede and predict the changes in ventricular CL. However, ventricular CL variability can be caused by changes in AV conduction instead of changes in the CL of an AT, in which case ventricular CL variability may not be predicted by a prior change in atrial CL during AT. Nevertheless, because there is no VA conduction during AT, ventricular CL variability alone would not be expected to result in atrial CL variability during AT.[24]

In contrast to AT, typical AVNRT and orthodromic AVRT generally have CL variability because of changes in anterograde AVN conduction. Because retrograde conduction through a fast AVN pathway or a BT is generally much less variable than anterograde conduction through the AVN, the changes in ventricular CL that result from variability in anterograde AVN conduction would be expected to precede the subsequent changes in atrial CL. This explains why the change in atrial CL does not predict the change in subsequent ventricular CL in typical AVNRT and orthodromic AVRT. On the other hand, in atypical AVNRT, anterograde conduction occurs over the more stable fast AVN pathway and retrograde conduction is more subject to variability. This explains the finding that changes in atrial CL predict the changes in subsequent ventricular CL in atypical AVNRT (as is the case in AT).[24]

In addition, orthodromic AVRT in the presence of dual AVN physiology can be associated with anterograde conduction alternating over the slow and fast AVN pathways, resulting in a regular irregularity of the TCL (alternating long and short cycles). Importantly, the RP interval during the SVT remains constant, regardless of the PR interval.

Effects of Bundle Branch Block

The development of BBB during SVT that does not influence the TCL (A-A or H-H interval) or the VA interval is consistent with AT, AVNRT (**see Fig. 17.9**), and orthodromic AVRT using a BT in the ventricle contralateral to the BBB, because the ventricle affected by conduction delay is not part of the SVT circuit (**see Fig. 18.35**), but excludes orthodromic AVRT using a BT ipsilateral to the BBB.

BBB ipsilateral to the AV BT mediating orthodromic AVRT results in prolongation of the "surface VA interval" (QRS onset to atrial electrogram) compared to that during narrow QRS tachycardia because of the extra time required for the impulse to travel from the AVN down the HB and contralateral bundle branch, and transseptally to the ipsilateral ventricle to reach the AV BT and then activate the atrium (**see Fig. 18.37**). However, the "local VA interval" (measured at the site of BT insertion) remains constant. In addition, the TCL usually increases in concordance with the increase in the surface VA interval as a result of ipsilateral BBB because of the now-larger tachycardia circuit; however, because the time the wavefront spends outside the AVN is now longer, AVN conduction may improve, resulting in shortening in the AH interval (and PR interval), which can potentially counterbalance, at least in part, the effects of prolongation of the VA interval on the total TCL. Thus the surface VA interval and not the TCL should be used to assess the effects of BBB on the SVT (**see Fig. 18.12**).

Prolongation of the surface VA interval during SVT in response to BBB by more than 35 milliseconds suggests that an ipsilateral free-wall AV BT is present and is participating in the SVT (i.e., diagnostic of orthodromic AVRT). On the other hand, prolongation of the surface VA by 25 to 35 milliseconds suggests a septal AV BT (posteroseptal AV BT in association with LBBB, and superoparaseptal AV BT in association with RBBB) (Fig. 20.9). In contrast, BBB contralateral to the AV BT does not influence the VA interval or TCL because the contralateral ventricle is not part of the reentrant circuit (**see Figs. 18.35 and 18.37**).

Because the occurrence of BBB during SVT is much more common in orthodromic AVRT than AVNRT or AT (90% of SVTs with sustained LBBB are orthodromic AVRTs), the mere presence of LBBB aberrancy during SVT is suggestive of orthodromic AVRT, but can still occur in other types of SVTs.

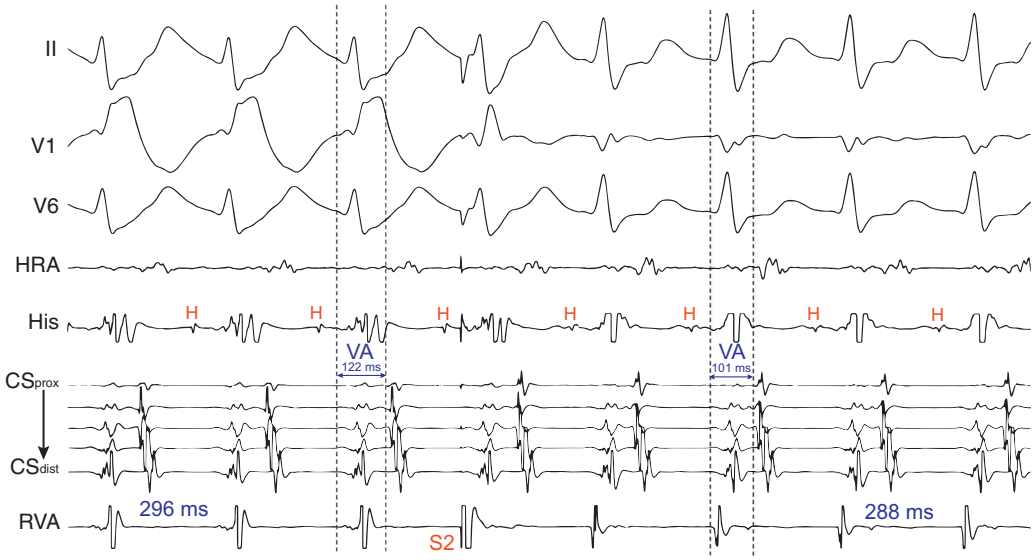


Fig. 20.9 Effect of Right Bundle Branch Block (RBBB) on Supraventricular Tachycardia (SVT). RBBB is initially present during an SVT with short RP interval and concentric atrial activation. Introduction of a ventricular extrastimulus *(S2)* from the right ventricular apex *(RVA)* during the tachycardia is followed by resolution of RBBB ("peeling back" refractoriness). Note that the loss of RBBB is associated with shortening of the ventriculoatrial *(VA)* interval (by 21 milliseconds) and a milder degree of shortening of the tachycardia cycle length (by 8 milliseconds), indicating the presence and participation of a septal BT in the reentrant circuit, which establishes the diagnosis of orthodromic atrioventricular reentrant tachycardia. CS_{dist}, Distal coronary sinus; CS_{prox}, proximal coronary sinus; *HRA,* high right atrium.

Tachycardia Termination and Response to Physiological and Pharmacological Maneuvers

Spontaneous termination. Spontaneous termination of orthodromic AVRT usually occurs because of anterograde gradual slowing and then block in the AVN, sometimes causing initial oscillation in the TCL, with alternate complexes demonstrating a Wenckebach periodicity before block. However, termination with retrograde block in the AV BT can occur without any perturbations of the TCL during very rapid orthodromic AVRT or following a sudden shortening of the TCL (e.g., after resolution of ipsilateral BBB or shift of anterograde conduction from the slow to the fast AVN pathway).

Spontaneous termination of AVNRT occurs because of block in the fast or slow pathway. However, the better the retrograde fast pathway conduction, the less likely that it is the site of block.

Spontaneous termination of AT is usually accompanied by progressive prolongation of the A-A interval, with or without changes in AV conduction. During AT with 1 : 1 AV conduction, the last beat of AT is conducted to the ventricle. Spontaneous termination of SVT with a P wave not followed by a QRS practically excludes AT, except coincidentally or in the case of a nonconducted PAC terminating the AT (neither of which will be reproducible).

Termination with adenosine. Adenosine terminates the vast majority of orthodromic AVRTs (by blocking AVN conduction) and AVNRTs (by blocking slow pathway conduction). Also, a significant proportion of focal ATs can be terminated by adenosine, typically (80%) prior to the onset of AV block (i.e., termination occurs with a tachycardia P wave that is followed by a conducted QRS). Although adenosine may not terminate ATs, it can help confirm the diagnosis of AT when it causes transient AV block without terminating the tachycardia. In addition, adenosine can help identify automatic ATs, which are generally transiently slowed but not terminated, with gradual resumption of the AT rate.

Adenosine generally terminates atypical AVNRT by gradual slowing and then block in the retrograde slow pathway. Adenosine usually terminates PJRT by block in the AVN (two-thirds) or in the BT (one-third) (**see eFig. 18.5**). Although the mode of tachycardia termination by adenosine has been suggested to distinguish between PJRT and atypical AVNRT, one report has shown that termination of atypical AVNRT with adenosine can also result from block in the fast AVN pathway and, therefore, its value in distinguishing between atypical AVNRT and PJRT is questionable.

In summary, the mere termination of SVT in response to adenosine is usually not helpful in differentiating SVTs. However, the pattern of SVT termination can be helpful in two situations: First, reproducible termination of the SVT with a QRS not followed by a P wave excludes orthodromic AVRT using a rapidly conducting AV BT as the retrograde limb (adenosine blocks the AVN and not the BT), is unusual in typical AVNRT (adenosine blocks the slow pathway with little or no effect on fast pathway conduction), and is consistent with AT, PJRT, or atypical AVNRT. Second, reproducible termination of the SVT with a P wave not followed by a QRS excludes AT, because it occurs in AT only if adenosine terminates the AT at the same moment it causes AV block, which is an unlikely coincidence (Fig. 20.10).[25]

Termination with vagal maneuvers. Carotid sinus massage and vagal maneuvers can slow or terminate sinus node reentrant tachycardia and 25% of microreentrant ATs (especially those with long TCLs and those arising in the RA), and, to a lesser degree, triggered-activity ATs. However, these interventions may slow but do not generally terminate automatic AT.

Orthodromic AVRT usually terminates with gradual slowing and then block in the AVN. Typical AVNRT usually terminates with gradual anterograde slowing and then block in the slow pathway; block in the fast pathway is uncommon. In addition, carotid sinus massage and vagal maneuvers terminate atypical AVNRT by gradual slowing and

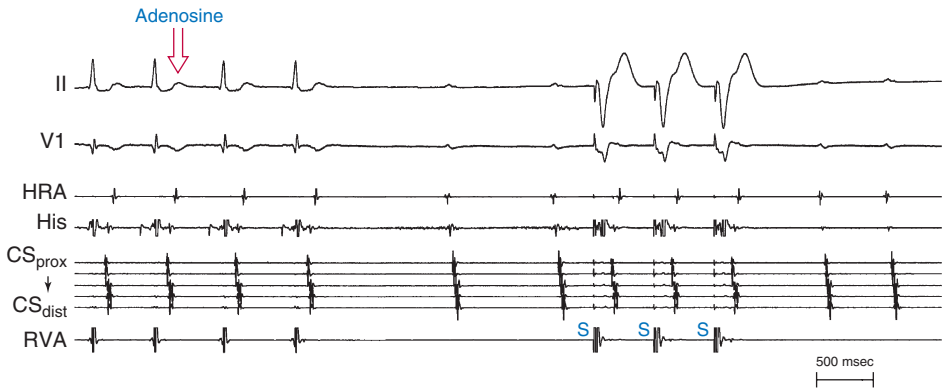


Fig. 20.10 Termination of Supraventricular Tachycardia (SVT) With Adenosine. Administration of adenosine during an SVT with concentric atrial activation sequence and short RP interval results in termination of the SVT with an atrial complex not followed by a QRS, which is inconsistent with atrial tachycardia. Following termination of the SVT, complete atrioventricular (AV) block during sinus rhythm is observed; however, ventricular pacing is associated with intact ventriculoatrial (VA) conduction and a retrograde atrial activation sequence identical to that during the SVT, which suggests that VA conduction is not mediated by the atrioventricular node (AVN) but by an AV bypass tract (BT). The SVT is in fact an orthodromic atrioventricular reentrant tachycardia using a concealed septal BT, and termination with adenosine was the result of block in the AVN. Retrograde conduction over the BT was not affected by adenosine. *CS_{dist}*, Distal coronary sinus; *CS_{prox}*, proximal coronary sinus; *HRA*, high right atrium; *RVA*, right ventricular apex.

then block in the retrograde slow pathway (**see Fig. 17.15**). Carotid sinus massage usually terminates PJRT by block in either the AVN or the BT.

Diagnostic Maneuvers During Tachycardia

Atrial Extrastimulation During Tachycardia

Resetting. An AES can reset AT, AVNRT, and orthodromic AVRT, and demonstration of resetting by itself is not helpful in distinguishing among the different types of SVTs. However, several characteristics of tachycardia resetting can help in the differential diagnosis.

Manifest atrial fusion. Resetting with manifest atrial fusion (as demonstrated by intermediate P wave morphologies on the surface ECG, or partial activation change recorded by multiple endocardial electrodes) can be demonstrated only in orthodromic AVRT and macroreentrant atrial tachycardia (MRAT), but not with AVNRT or focal AT. For atrial fusion (i.e., fusion of atrial activation from both the tachycardia wavefront and the AES) to occur, the AES should be able to enter the reentrant circuit while at the same time the tachycardia wavefront should be able to exit the circuit. This requires spatial separation between the entry and exit sites to the reentrant circuit, a condition that seems to be lacking in the setting of AVNRT and focal AT.

Resetting response curve. AES can reset microreentrant AT, AVNRT, and orthodromic AVRT with a resetting response classic for reentry (increasing or mixed response). In contrast, triggered activity AT exhibits a decreasing resetting response curve, and automatic AT exhibits an increasing response curve.

Termination. Termination of SVT with an AES is not usually helpful for the differential diagnosis. An AES can reproducibly terminate reentrant AT, AVNRT, and orthodromic AVRT. Termination of triggered activity AT is less reproducible. AES generally does not terminate automatic AT.

Atrial Overdrive Pacing During Tachycardia

Entrainment. Overdrive pacing at a CL approximately 10 to 30 milliseconds shorter than the TCL is usually able to entrain reentrant AT, AVNRT, and orthodromic AVRT but not triggered activity or automatic ATs.

Manifest atrial fusion. As discussed earlier, entrainment with manifest atrial fusion can be demonstrated only in AVRT and MRAT, but not with AVNRT or focal AT (**see Fig. 18.40**). Therefore, during entrainment of AVNRT and focal AT by atrial pacing, the atrial activation sequence and P wave morphology are always similar to those of pure paced morphology. Importantly, variable degrees of surface P wave or intracardiac electrogram fusion can be observed at the beginning of overdrive pacing, and should not be mistaken for constant fusion during entrainment.[26,27]

ΔAH interval. During entrainment at a PCL close to the TCL, the AH interval during entrainment is longer than that during AVNRT. In contrast, in the setting of AT and orthodromic AVRT, the AH interval is comparable during SVT and entrainment with atrial pacing.

Acceleration. Overdrive pacing during triggered activity–related AT generally produces acceleration of the atrial CL. Following atrial overdrive pacing during AT due to triggered activity, the return atrial CL tends to shorten with shortening of the PCL.

Overdrive suppression. Automatic AT cannot be entrained by atrial pacing; however, rapid atrial pacing results in overdrive suppression of the AT rate. The AT resumes after cessation of atrial pacing but at a slower rate and gradually speeds up (warms up) to return to prepacing TCL. The atrial return CL following cessation is progressively prolonged with an increase in the duration or rate of the overdrive pacing train. This is in contrast to the return cycles and postpacing intervals (PPIs) that remain fixed following entrainment of reentrant circuits (microreentrant AT, AVNRT, and orthodromic AVNRT), regardless of the length of the pacing drive.

Termination. Termination of SVT with atrial pacing is not usually helpful for differential diagnosis. Atrial pacing can reproducibly terminate reentrant AT, AVNRT, and orthodromic AVRT, but not automatic AT. Termination of triggered activity AT is less reproducible.

VA linking. Following cessation of overdrive atrial pacing during SVT, the VA interval (the interval between the last captured QRS complex and the first tachycardia atrial complex) can help distinguish between the different mechanisms of SVT.

VA linking following different pacing rates and durations. Overdrive atrial pacing is performed from the same atrial site

(e.g., proximal CS) at different PCLs (10, 20, and 30 milliseconds shorter than the TCL), and for different durations (e.g., 10 and 20 seconds). If "VA linking" is present (i.e., the VA interval is reproducibly constant despite pacing at different CLs or for different durations and is similar to that during SVT), AT is unlikely. If no VA linking is demonstrable, AT is more likely than other types of SVTs (**see Fig. 18.40**). VA linking occurs in the setting of typical AVNRT and orthodromic AVRT because the timing of atrial activation is dependent on the preceding ventricular activation and is the result of retrograde VA conduction over the AVN fast pathway (during typical AVNRT) or the BT (during orthodromic AVRT), which is relatively fixed and constant. As a consequence, postpacing VA intervals are fixed and similar to those during tachycardia (with less than 10 milliseconds variation) after different attempts at atrial entrainment, regardless of the site, duration, or CL of the entraining atrial pacing drive. Conversely, following cessation of overdrive pacing (with 1:1 AV conduction) during focal AT, the postpacing VA interval can vary significantly from the VA interval during AT (especially when pacing at different rates or durations), because the timing of the tachycardia atrial return cycle is not related to the preceding ventricular activation.[11,22,28]

VA linking following different pacing sites. Overdrive atrial pacing is performed from the high RA and proximal CS, at the same PCL (10 to 30 milliseconds shorter than the TCL), and for the same duration. When comparing the postpacing VA intervals following overdrive pacing from the high RA versus from the proximal CS, a maximal difference in the postpacing VA intervals (ΔVA interval) of more than 14 milliseconds is diagnostic with AT, whereas a ΔVA interval of less than 14 milliseconds favors AVNRT or orthodromic AVRT. This is because, in the setting of AT, the first atrial return cycle following cessation of pacing is dependent on the distance between the AT origin and pacing site, atrial conduction properties, and mode of the resetting response of the AT, and it is not related to the preceding ventricular activation. Hence, the postpacing VA intervals vary among the pacing sites, and the ΔVA interval is relatively large (more than 14 milliseconds).[28]

Ventricular Extrastimulation During Tachycardia

Resetting. Single VESs are delivered during tachycardia to scan diastole, initially at long coupling intervals (10 to 20 milliseconds shorter than the TCL), then with progressively shorter coupling intervals (10-millisecond decrements) until loss of ventricular capture.

VES can reset any SVT, and resetting by itself is not diagnostic of a specific type of SVT. Orthodromic AVRT is the easiest SVT to be reset and even terminated by VES (**see Fig. 18.42**).[22] Significant portions of the ipsilateral ventricle or ventricular septum are requisite components of the orthodromic AVRT circuit, making it vulnerable to penetration and resetting even by a late-coupled VES. However, a VES delivered in the contralateral ventricle may not affect the circuit, and resetting is facilitated by delivery of the VES closer to the potential BT location.

For AVNRT, the ability of a VES to affect the SVT depends on its ability to activate the HB prematurely and penetrate the AVN. Even when HB activation is advanced by the VES, the ability of the paced impulse to invade the AVN will depend on the length of the lower common pathway; the longer the lower common pathway, the more the timing of HB activation must be advanced. In fast-slow or slow-slow AVNRT, which typically has a long lower common pathway, the HB activation must be advanced by more than 30 to 60 milliseconds. In contrast, in slow-fast AVNRT, the lower common pathway (if present) is shorter and the tachycardia is typically reset by the VES as soon as the HB activation is advanced.

During AT, a VES can advance the next atrial activation when given the chance to conduct retrogradely and prematurely to the atrium, which requires significantly shorter VES coupling intervals.[11]

Certain situations during resetting can help discriminate between the different SVT mechanisms, including the following:

Preexcitation index. The preexcitation index (defined as the difference between the TCL and the longest VES coupling interval at which atrial capture occurs during tachycardia) of 100 milliseconds or more characterizes AVNRT, whereas an index less than 45 milliseconds is consistent with AVRT with a septal BT.[4]

Resetting with different atrial activation sequence. The retrograde atrial activation sequence following a VES that resets the tachycardia is usually similar to that during SVT in the setting of AVNRT and orthodromic AVRT, because it should conduct over the tachycardia retrograde limb (except in the presence of a bystander retrogradely conducting AV BT). In contrast, a retrograde atrial activation sequence following the VES is usually different from that during AT, except for ATs originating close to the AV junction.

Manifest ventricular fusion. Resetting with manifest QRS fusion can be observed during orthodromic AVRT, especially during pacing at a site closer to the BT ventricular insertion site than the entrance of the reentrant circuit to ventricular tissue (i.e., the HPS). Such a phenomenon, on the other hand, cannot occur during AVNRT or focal AT because of the lack of spatial separation of the entrance and exit to the tachycardia circuit.

Failure of resetting. Late-coupled VESs may fail to reset the SVT, even orthodromic AVRT, especially when delivered from a site far from the BT location. However, failure of early single or double VESs to reset the SVT, despite advancement of ventricular electrograms in the electrode recording the earliest atrial activation during the SVT (which would be close to the potential BT ventricular insertion site) by more than 30 milliseconds, excludes orthodromic AVRT.[29]

Resetting without atrial activation. The ability of a VES to reset the SVT without atrial activation (i.e., the VES advances the subsequent His potential and QRS) excludes AT and orthodromic AVRT, because it proves that the atrium is not part of the SVT circuit.

Resetting with delay of atrial activation. The ability of a VES to delay the next atrial activation excludes AT. This can be observed more often during PJRT (**see Fig. 18.39**) and less commonly during AVNRT, and is caused by decremental conduction in the retrograde limb of the tachycardia circuit (the decrementally conducting concealed BT in the setting of PJRT).

Resetting during His bundle refractoriness. A VES delivered when the HB is refractory (i.e., when the His potential is already manifest or within 35 to 55 milliseconds before the time of the expected His potential) that advances (accelerates) the next atrial activation is diagnostic of the presence of a retrogradely conducting BT. Such a VES has to conduct and advance atrial activation via a BT because the HPS-AVN is already refractory and cannot mediate retrograde conduction of the VES to the atrium (Fig. 20.11; **see Fig. 18.42**). Although such an observation proves the presence of a retrogradely conducting AV BT, it does not prove its participation in the SVT (i.e., does not prove the diagnosis of orthodromic AVRT), because both AT and AVNRT can coexist with a bystander AV BT. However, if this VES advances atrial activation with an activation sequence identical to that during the SVT, this suggests that the SVT is orthodromic AVRT and the BT is participating in the SVT, although it does not exclude the rare case of an AT originating at a site close to the atrial insertion site of a bystander AV BT. Although such a VES can advance atrial activation during AT through fast retrograde conduction over a bystander BT, it should not be able to delay an AT beat by conduction over the AV BT. Thus a VES delivered when the HB is refractory that delays the next atrial activation indicates that the VES was conducted with some delay over the BT (due to decremental BT conduction) and that the next atrial activation was dependent on this slower conduction; hence, the BT is participating in the SVT, proving

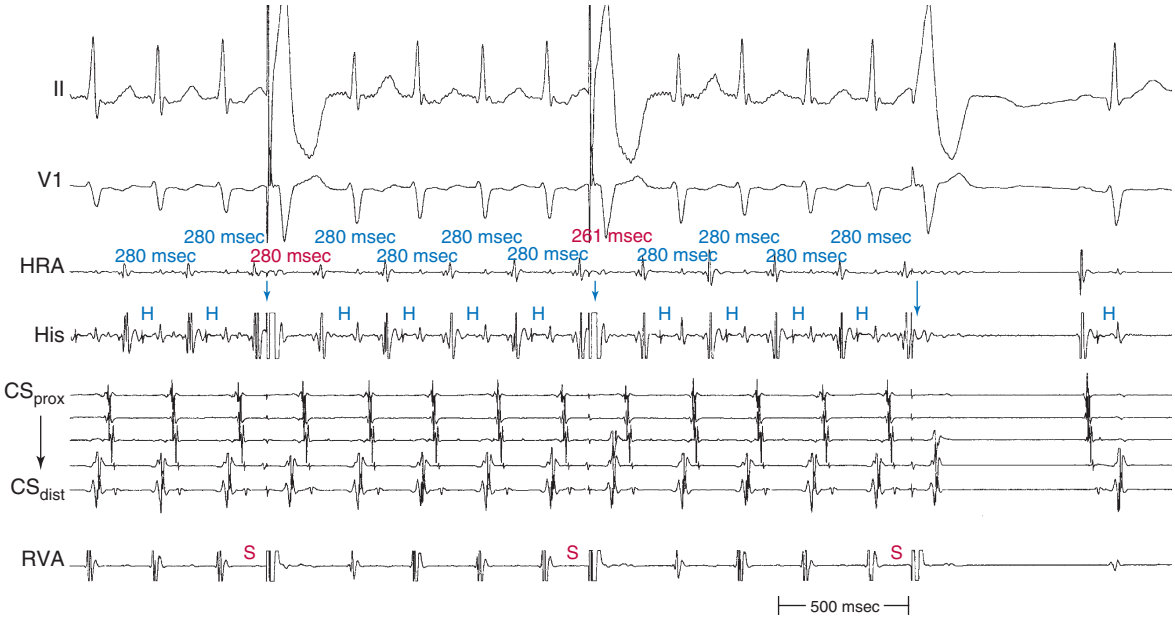


Fig. 20.11 Ventricular Extrastimulation (VES) During Supraventricular Tachycardia (SVT). VESs were delivered at progressively shorter coupling intervals during a narrow complex SVT with concentric atrial activation sequence. Timing of the anticipated anterograde His bundle (HB) activation is indicated by the blue arrows. The first VES is delivered during HB refractoriness and fails to reset the tachycardia, a phenomenon that does not help in the differential diagnosis. The second VES is delivered slightly earlier but still during HB refractoriness, and it does accelerate the following atrial activation. Atrial activation sequence during the reset atrial complex is identical to that during SVT. This observation excludes atrioventricular nodal reentrant tachycardia (AVNRT), and favors orthodromic atrioventricular reentrant tachycardia (AVRT), but does not exclude the rare example of atrial tachycardia (AT) with a bystander bypass tract with atrial insertion close to the AT focus. The third VES is delivered during HB refractoriness (within 40 milliseconds before the anticipated anterograde HB activation) and it terminates the tachycardia without conducting to the atrium. This observation, when reproducible, excludes both AVNRT and AT, and proves that the tachycardia is an orthodromic AVRT. *CS_{dist}*, Distal coronary sinus; *CS_{prox}*, proximal coronary sinus; *HRA*, high right atrium; *RVA*, right ventricular apex.

orthodromic AVRT (e.g., PJRT or nodofascicular reentrant tachycardia) as the mechanism of the SVT. Similarly, a VES delivered when the HB is refractory that terminates the SVT without atrial activation is diagnostic of AVRT.[4,30]

Termination. Termination of AVNRT with a single VES is difficult and occurs rarely when the TCL is less than 350 milliseconds; such termination favors the diagnosis of orthodromic AVRT, which can usually be readily terminated by single or double VESs. Termination of the SVT with a VES delivered when the HB is refractory excludes AVNRT and AT (see Fig. 20.11), except in the presence of an innocent bystander BT mediating VA conduction. Reproducible termination of the SVT with a VES not followed by atrial activation excludes AT (**see Fig. 18.42**) and, if this occurs with a VES delivered while the HB is refractory, it excludes both AT and AVNRT (see Fig. 20.11).

Ventricular Overdrive Pacing During Tachycardia

Ventricular pacing is performed at a CL 10 to 30 milliseconds shorter than the TCL; the PCL is then progressively reduced by 10 to 20 milliseconds in a stepwise fashion with cessation of ventricular pacing after each PCL to verify continuation versus termination of the SVT. The presence of ventricular capture during ventricular pacing should be verified. In addition, the presence of 1:1 VA conduction and acceleration of the atrial rate to the PCL should be carefully examined (Fig. 20.12). It is also important to verify the continuation of the SVT following cessation of ventricular pacing and whether SVT termination, with or without reinduction of the SVT, has occurred during ventricular pacing (Fig. 20.13).

VA dissociation. When overdrive ventricular pacing during SVT fails to accelerate the atrial CL to the PCL (i.e., the ventricles are dissociated from the tachycardia), AVRT is excluded, AT is the most likely diagnosis, but AVNRT is still possible.

Atrial activation sequence. As noted, the retrograde atrial activation sequence during ventricular pacing is usually similar to that during the SVT in the setting of AVNRT and orthodromic AVRT because it should conduct over the tachycardia retrograde limb. On the other hand, the retrograde atrial activation sequence during ventricular pacing is usually different from that during AT, except for ATs originating close to the AV junction. A pitfall of this criterion is the presence of a bystander AV BT, which can provide another retrograde route capable of mediating retrograde conduction during ventricular pacing without being part of the SVT circuit. In this setting, ventricular pacing can result in a retrograde atrial activation sequence different from that of AVNRT or orthodromic AVRT. The presence of such an AV BT, however, is generally easy to verify with ventricular stimulation during NSR.

Entrainment. Ventricular pacing is almost always able to entrain AVNRT and orthodromic AVRT and, if 1:1 VA conduction is maintained, reentrant AT. Although the mere demonstration of tachycardia entrainment does not help discrimination between the different SVT mechanisms, several parameters during ventricular entrainment can help establish the lower portion of the tachycardia circuit as macroreentry

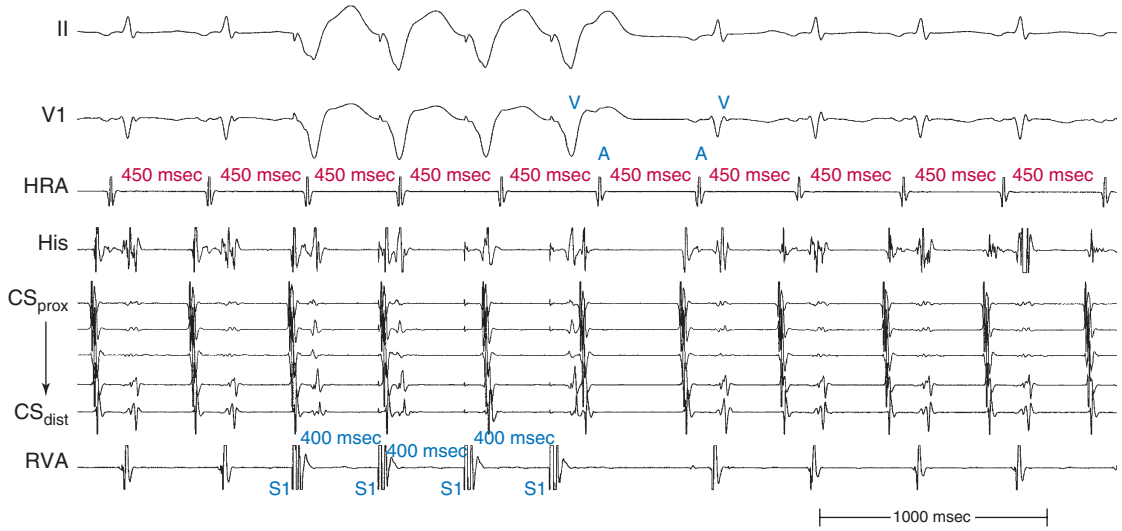


Fig. 20.12 Overdrive Ventricular Pacing During Supraventricular Tachycardia (SVT). The SVT has a long RP interval and concentric atrial activation sequence, which favors atypical atrioventricular nodal reentrant tachycardia (AVNRT), atrial tachycardia (AT) originating close to the atrioventricular junction, or orthodromic atrioventricular reentrant tachycardia (AVRT) using a slowly conducting septal bypass tract. Overdrive ventricular pacing during the tachycardia fails to entrain the tachycardia or capture the atrium (no ventriculoatrial [VA] conduction) because the tachycardia atrial cycle length (CL; 450 milliseconds) remains stable and unaltered by the faster ventricular pacing CL (400 milliseconds). Therefore analysis of the postpacing interval or VA interval during pacing versus SVT is invalid. Analysis of the response sequence following cessation of pacing (atrial-ventricular vs. atrial-atrial-ventricular [A-A-V] response) is also invalid in view of the lack of 1 : 1 VA conduction during ventricular pacing (resulting in a pseudo–A-A-V response). Nonetheless, the fact that ventricular pacing has dissociated the atrium from the ventricle excludes AVRT. However, other pacing maneuvers are required for differentiation between atypical AVNRT and AT. *CS_{dist}*, Distal coronary sinus; *CS_{prox}*, proximal coronary sinus; *HRA,* high right atrium; *RVA,* right ventricular apex.

involving the HPS/ventricle (AVRT) or not (AVNRT, AT).[30] These parameters include the presence of manifest ventricular fusion, VA interval during ventricular pacing as compared with that during SVT, the PPI, and differential-site ventricular entrainment.[26,27]

Manifest ventricular fusion. As discussed previously, manifest ventricular fusion during SVT entrainment is proof that the ventricle is part of the SVT circuit (i.e., diagnostic of AVRT), excluding AVNRT, AT, and junctional tachycardia (**see Fig. 18.43**). During ventricular entrainment of AVNRT, AT, or junctional tachycardia, the QRS morphology is that of pure ventricular pacing. A requirement for the presence of fusion is spatial separation between the sites of entrance to and exit from the reentrant circuit, which is absent in the setting of AVNRT, AT, and junctional tachycardia, each of which has a single "gate" to the ventricles (the HB). In orthodromic AVRT, the entrance (HPS) and exit (BT ventricular insertion site) of the reentrant circuit to and from ventricular tissue) are separated from each other. In this setting, the paced wavefront can activate a portion of the ventricles and enter the AVRT circuit and at the same time the tachycardia wavefront emerge from the reentry circuit at a distant exit site and activate another part of the ventricles. Also, the relative proximity of the pacing site to the entry and exit sites of the reentry circuit is a critical determinant for the occurrence of fusion during resetting and entrainment. Pacing at a site closer to the BT ventricular insertion site (e.g., LV pacing in the setting of left free-wall BTs, and RV basal pacing in the setting of right-sided or septal BTs) than the entrance of the reentrant circuit to ventricular tissue (i.e., the HPS) results in a larger degree of fusion of QRS morphology between baseline morphology during orthodromic AVRT and that of fully paced QRS (**see Fig. 18.43**).[31,32]

It is important to understand, however, that overdrive pacing of a tachycardia of any mechanism (including focal AT, junctional tachycardia, and AVNRT) can result in a certain degree of fusion, especially when the PCL is only slightly shorter than the TCL. Such fusion, however, is unstable during the same pacing drive at a constant CL because the pacing stimuli fall on a progressively earlier portion of the tachycardia cycle, producing progressively less fusion and more fully paced morphology. Such phenomena should be distinguished from entrainment, and sometimes this requires pacing for long periods to demonstrate variable degrees of fusion.

ΔVA interval. Entrainment of the SVT by RV pacing can help differentiate orthodromic AVRT from AVNRT by evaluating the VA interval during SVT (measured from the onset of surface QRS to high RA electrogram) versus the VA interval during pacing (i.e., SA interval measured from the ventricular pacing stimulus to the high RA electrogram). The ventricle and atrium are activated in sequence during orthodromic AVRT and during ventricular pacing, but in parallel during AVNRT. Therefore the VA interval during orthodromic AVRT approximates that during ventricular pacing (**see Fig. 18.43**). In contrast, the VA interval during AVNRT would be much shorter than that during ventricular pacing (**see Fig. 17.19**). In general, a difference in the VA interval (ΔVA [$VA_{pacing} - VA_{SVT}$]) greater than 85 milliseconds is consistent with AVNRT, whereas a ΔVA of less than 85 milliseconds is consistent with orthodromic AVRT (Fig. 20.14).[33,34]

This measurement resembles the "stimulus-exit" interval versus "electrogram-exit" interval criterion used in entrainment mapping of macroreentrant AT and VT. In the setting of AVNRT and AVRT, the "electrogram" is represented by the QRS onset, and the "exit" is

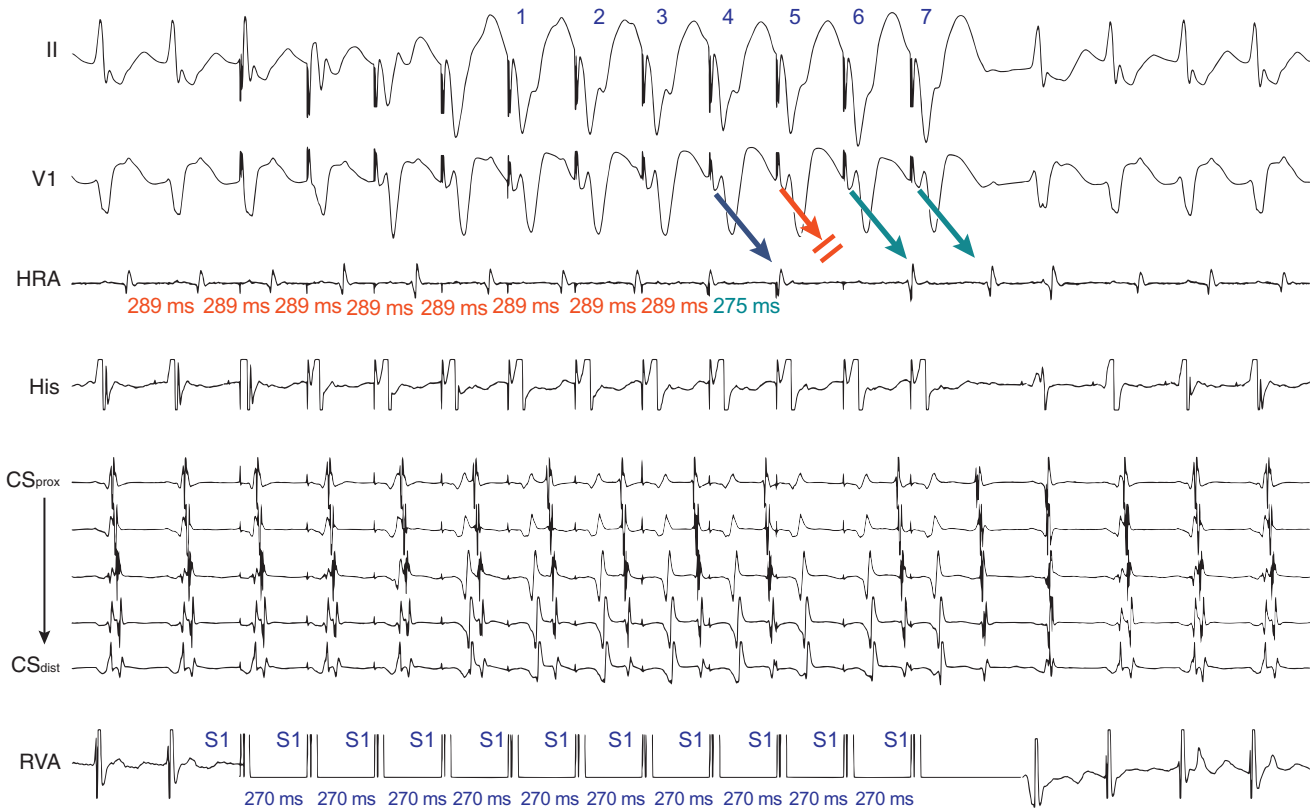


Fig. 20.13 Overdrive Ventricular Pacing During Atrioventricular Nodal Reentrant Tachycardia (AVNRT). The tachycardia has a short RP interval and concentric atrial activation sequence and simultaneous atrial and ventricular activation, consistent with typical AVNRT. Overdrive ventricular pacing *(S1)* started from the right ventricular apex *(RVA)* at a cycle length (CL) 18 milliseconds shorter than the tachycardia CL. The numbers 1 to 7 indicate the paced QRS complexes with stable (purely paced) morphology. Note that the first constant-appearing QRS complex to reset atrial activation is QRS #4 (as indicated by the *blue arrow*), which excludes atrioventricular reentrant tachycardia, and is consistent with AVNRT. Also note that the tachycardia terminates after that, and QRS #5 is not followed by atrial activation *(red arrow)*. QRS #6 and #7 conduct retrogradely to the atrium *(green arrows)* and reinitiate typical AVNRT. Since the tachycardia was terminated and then reinitiated during the same pacing drive, entrainment is not present, and analysis of the postpacing interval is not valid. CS_{dist}, Distal coronary sinus; CS_{prox}, proximal coronary sinus; *HRA,* high right atrium.

represented by the subsequent atrial electrogram, which is orthodromically captured during ventricular entrainment and represents the exit site from the reentry circuit to the atrium. Hence, the stimulus to the subsequent atrial-entrained electrogram interval (SA interval) is compared to the electrogram (QRS onset) to the subsequent atrial electrogram during tachycardia (VA interval). As a consequence, the shorter the difference between the two intervals, the closer the pacing site to the reentry circuit.[32]

Postpacing interval. The PPI reflects conduction time from the ventricular pacing site to the SVT circuit, once around the reentry circuit, and then back to the pacing site. The difference between the PPI and TCL (PPI – TCL) represents the conduction time from the pacing site to the reentry circuit and back. Thus the (PPI – TCL) difference can qualitatively estimate how far (in terms of conduction time) the reentrant circuit is from the pacing site; the greater the (PPI – TCL) difference, the longer the conduction time between the pacing site and reentry circuit, and the greater the "electrical" distance is between the pacing site and the circuit.[32]

Because the RV is closer to the AVRT circuit as compared to the AVNRT circuit, the (PPI – TCL) difference following entrainment from the RV apex is smaller in the setting of AVRT than during AVNRT. In fact, a (PPI – TCL) difference of more than 115 milliseconds was found to identify AVNRT (**see Figs. 17.19** and Fig. 20.14), whereas a (PPI – TCL) difference of less than 115 milliseconds was consistent with orthodromic AVRT (**see Fig. 18.43**). For borderline values, ventricular pacing at the RV base can help exaggerate the (PPI – TCL) difference in the setting of AVNRT, but without significant changes in the setting of orthodromic AVRT (see differential-site RV entrainment later).[11,35]

A relatively common phenomenon encountered during entrainment of orthodromic AVRT by ventricular pacing is the prolongation of the AH interval because of decremental conduction properties of the AVN or (in the presence of dual AVN physiology) a jump of anterograde conduction from the fast pathway to the slow pathway. The prolonged AH interval on the last entrained beat will contribute to prolongation of the PPI that is not reflective of the distance of the pacing site from the circuit. Thus the (PPI – TCL) differences obtained after entrainment of orthodromic AVRT employing a septal BT can actually overlap with those observed after entrainment of AVNRT. "Correction" of the PPI by subtracting the increment in AVN conduction time in the first PPI (postpacing AH interval minus prepacing AH interval) from the (PPI – TCL) difference ("corrected" [PPI – TCL]) has been found to improve the accuracy of this criterion. The difference between AV intervals

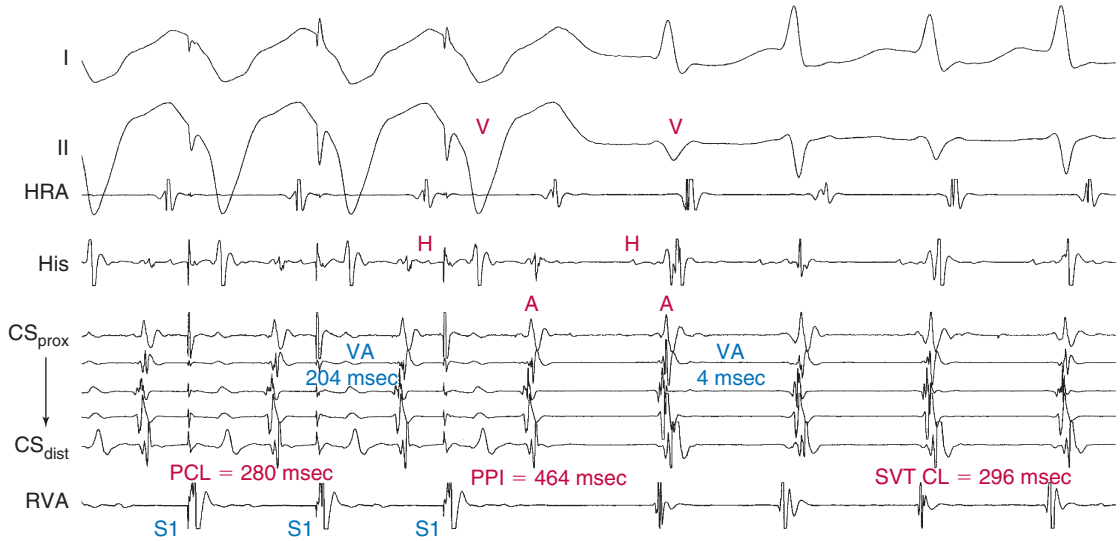


Fig. 20.14 Ventricular Entrainment of Supraventricular Tachycardia *(SVT).* Ventricular entrainment if achieved with pacing from the RVA. Several features in this tracing can help in the differential diagnosis of the SVT. First, atrial and ventricular activation occur simultaneously during the SVT, which excludes atrioventricular reentrant tachycardia (AVRT). Second, atrial activation during the SVT is eccentric, with earliest activation in the mid–coronary sinus (CS), which favors atrial tachycardia (AT) over atrioventricular nodal reentrant tachycardia (AVNRT). Third, the atrial activation sequence during ventricular pacing is identical to that during SVT, which favors AVNRT and AVRT over AT. Fourth, following cessation over ventricular pacing, the postpacing interval *(PPI)* minus SVT cycle length (CL), (PPI – SVT CL), is more than 115 milliseconds, and the Δventriculoatrial *(VA)* interval ($VA_{pacing} - VA_{SVT}$) is more than 85 milliseconds, which favors AVNRT over AVRT. Fifth, although characterization of the activation sequence following cessation of ventricular pacing (atrial-atrial-ventricular [A-A-V] vs. atrial-ventricular [A-V] response) is unclear (because of the simultaneous occurrence of atrial and ventricular activation in the first tachycardia complex, replacing ventricular activation with HB activation [i.e., characterizing the response as A-A-H or A-H instead of A-A-V or A-V, respectively]) reveals an A-H response, which favors AVRT and AVNRT over AT. In summary, AVRT can be reliably excluded by the simultaneous atrial and ventricular activation. AT is excluded by the A-H response following cessation of ventricular pacing and by the identical atrial activation sequence during the SVT and ventricular pacing. The left variant of typical AVRT (with an eccentric atrial activation sequence) is the mechanism of the SVT. CS_{dist}, Distal coronary sinus; CS_{prox}, proximal coronary sinus; *HRA,* high right atrium; *PCL,* pacing cycle length; *RVA,* right ventricular apex.

(postpacing AV interval minus prepacing AV interval) can be taken for the latter adjustment when a His deflection is not clearly visible (assuming the HV interval remains constant). In a study of patients with both typical and atypical forms of AVNRT, as well as orthodromic AVRT using septal and free-wall BTs, a corrected (PPI – TCL) difference of less than 110 milliseconds was found more accurate in identifying orthodromic AVRT from AVNRT than the uncorrected (PPI – TCL) difference.[36–38]

Of note, determinations of the corrected (PPI – TCL) and ΔVA ($VA_{pacing} - VA_{SVT}$) after resetting with single or double VESs from the RV apex, are of similar value for discrimination between AVNRT and orthodromic AVRT even when the SVT is interrupted by ventricular pacing. Corrected (PPI – TCL) of more than 110 milliseconds and ΔVA of more than 110 milliseconds after resetting are consistent with AVNRT.[39]

Importantly, there are several potential pitfalls to the ΔVA interval and PPI criteria discussed previously. The TCL and VA interval are often perturbed for a few cycles after entrainment. For this reason, care should be taken not to measure unstable intervals immediately after ventricular pacing. In addition, oscillations in the TCL and VA intervals can occur spontaneously during the SVT. The discriminant points chosen may not apply when the spontaneous variability is greater than 30 milliseconds. Also, it is possible to mistake isorhythmic VA dissociation for entrainment if the pacing train is not long enough or the PCL is too close to the TCL. Furthermore, this test is less reliable and should be used with caution in patients with left lateral BTs. In addition, these criteria may not apply to BTs with significant decremental properties, although small decremental intervals are unlikely to provide a false result.

Conventional SVT criteria during entrainment from the ventricle establish the lower portion of the tachycardia circuit as macroreentrant involving the HPS/ventricle (AVRT) or not. Importantly, slow, decremental BT conduction (e.g., during PJRT or nodofascicular reentrant tachycardia) can affect the sensitivity of the ΔVA and ΔHA criteria for orthodromic AVRT, although the specificity of these criteria remains high. Therefore any standard criteria that is positive for orthodromic AVRT ([PPI – TCL] < 115 milliseconds, corrected [PPI – TCL] < 110 milliseconds, ΔVA < 85 milliseconds, and ΔHA < 0 milliseconds) is considered diagnostic for orthodromic AVRT, despite discordance among them, which can occur up to 50% of the time. In addition, a higher cutoff value of 125 milliseconds was found to increase the sensitivity for orthodromic AVRT while maintaining a high specificity.[30]

Differential-site RV entrainment. Differential-site RV entrainment (from RV apex vs. RV base) can help distinguish AVNRT from orthodromic AVRT. Because the reentrant circuit in AVNRT is confined above the HB and does not involve the ventricle, the base of the RV is electrically more distant (although anatomically closer) from the tachycardia

circuit than the RV apex, given that the His-Purkinje network directly inserts near the RV apex. Consequently, the PPI after entrainment of AVNRT from the RV base is longer than that following entrainment from the RV apex. The difference in PPI from the RV base versus the RV apex is largely composed of the extra time required to reach the circuit from the base versus the apex (approximately 30 milliseconds). Conversely, in orthodromic AVRT, in which the ventricles are an obligatory part of the circuit, the basal pacing site relative to the RV apex is variably related to the circuit, closer than the RV apex with septal BTs and equidistant with free-wall BTs, but the paced wavefront from the RV apex or RV base tends to have, on average, approximately equal access and proximity to the reentrant circuit involved in orthodromic AVRT. Therefore the time taken to reach the circuit (and hence the PPI) tends to be similar, irrespective of the location of the BT.

Correction of the PPI (to avoid potential error introduced by decremental conduction within the AVN during ventricular pacing) increases the accuracy of this method, although the degree of decrement is not expected to be materially different from basal rather than apical pacing as long as the pacing rates are the same or similar. The "corrected PPI" is obtained by subtracting any increase in the AV interval of the return cycle beat (as compared with the AV interval during SVT). A differential corrected (PPI – TCL) difference of more than 30 milliseconds after transient entrainment was found to be consistent with AVNRT (i.e., corrected [PPI – TCL] difference following pacing from the RV base was consistently at least 30 milliseconds longer than that following pacing from the RV apex). In contrast, a corrected (PPI – TCL) difference of less than 30 milliseconds was observed in all cases of orthodromic AVRT. In addition, a differential VA interval (VA interval during entrainment from RV base vs. RV septum) of more than 20 milliseconds was consistent with AVNRT, whereas a differential VA interval of less than 20 milliseconds was consistent with orthodromic AVRT.[40,41]

The main advantage of this technique is that the differential VA interval can be calculated from the last paced beat if the tachycardia is terminated after transient entrainment.

Length of pacing drive required for entrainment. Assessing timing and type of response of SVT to RV pacing can help differentiate orthodromic AVRT from AVNRT. In the setting of orthodromic AVRT, ventricular myocardium is the only intervening tissue between the pacing wavefront and the ventricular insertion site of the BT. Therefore once ventricular capture is achieved during RV pacing, the paced wavefront propagates to the ventricular insertion site of the BT quickly and resets the tachycardia (i.e., advances or delays atrial activation time). Consequently, RV pacing results in resetting of AVRT with septal or right-sided BT immediately once the RV is fully captured by the paced wavefront. During AVNRT, on the other hand, the pacing site is distant from the SVT circuit, and the paced wavefront has to propagate though ventricular tissue, then through the HPS, followed by AVN tissue prior to resetting the tachycardia. As a consequence, resetting of AVNRT is delayed for several captured paced beats as compared with orthodromic AVRT (eFig. 20.1). This observation is consistent with the concept that overdrive pacing begins to advance a reentrant tachycardia precisely when the total pacing prematurity exceeds the (PPI – TCL) after correcting for conduction velocity changes associated with the shorter CL.[42]

After initiation of synchronized RV pacing during SVT at a CL 10 to 40 milliseconds shorter than the TCL, once constant-appearing paced QRS complexes (either pure capture or fixed fusion) are observed, the number of constant-appearing paced QRS complexes required to accelerate atrial activation timing to the PCL is determined. The first atrial beat accelerated to the PCL is identified by demonstrating a fixed ventricular stimulus–to–atrial capture interval (SA interval). One report demonstrated that resetting of the SVT occurring by the first beat with stable paced QRS morphology identifies all orthodromic AVRT and essentially excludes AVNRT with high accuracy. On the other hand, resetting of the SVT occurring only after two or more beats with stable (fully paced) QRS morphology is consistent with AVNRT (see Fig. 20.13).

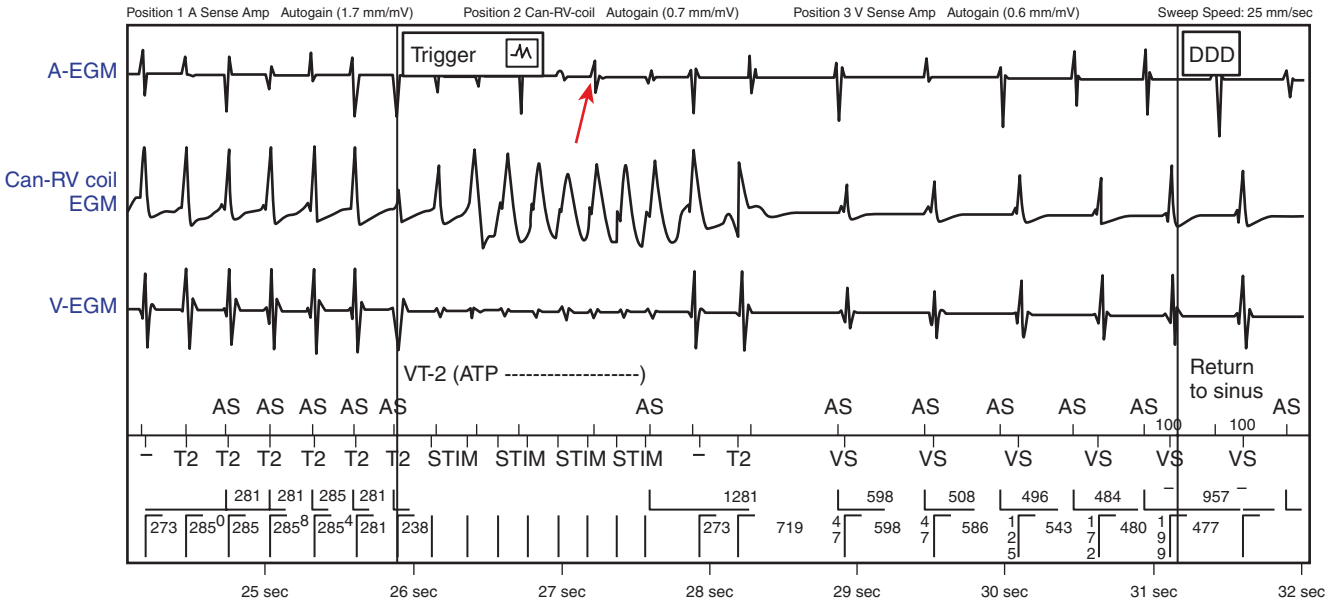
The major advantage of this method is its independence of tachycardia continuation after cessation of pacing. However, it is likely that these criteria may not be applicable at PCLs more than 40 milliseconds shorter than the TCL or with prematurity of more than 80% of the TCL (which can occur with poorly synchronized stimulation), because resetting of the tachycardia can occur earlier in AVNRT in response to a greater degree of penetration into the tachycardia circuit. In addition, these findings may not apply in cases in which AVNRT occurs in the setting of a bystander BT because SVT may be reset using the bystander BT. In addition, although this technique is effective for differentiating septal pathways from atypical AVNRT, it may be less useful in patients with BTs remote from the pacing stimulus (e.g., left free-wall BTs) (Fig. 20.15).[43,44]

Atrial resetting during the transition zone. On initiation of synchronized RV pacing trains during SVT at a rate slightly faster than the TCL, there is a transition zone during which the pacing train fuses with anterograde ventricular activation (i.e., the zone in which the paced QRS complexes show progressive fusion with the SVT complexes) until a stable QRS morphology is observed (either completely paced or constantly fused). The transitional zone begins with progressive fusion beats between the paced and tachycardia wavefronts and ends with the first beat of stable QRS morphology, the latter representing constant fusion in the setting of orthodromic AVRT and a fully paced QRS morphology in patients with AVNRT or AT. In patients with AVNRT or AT, acceleration of the timing of atrial activation cannot occur through the AVN during the transition zone. The reason is that the HB is expected to be refractory, as indicated by at least some ventricular activation still occurring by anterograde conduction over the HPS. If perturbation of atrial timing occurs during the transition zone, it indicates the presence of a retrogradely conducting BT, which can be an integral part of the SVT circuit (i.e., orthodromic AVRT) or a bystander. In one report, these criteria showed excellent diagnostic accuracy and could be applied regardless of whether entrainment was achieved or whether the SVT terminated during pacing. Perturbation of atrial timing of at least 15 milliseconds or a fixed SA interval measured from the last beat of the transition zone was seen in all the patients with orthodromic AVRT and in none of the patients with AVNRT or AT (unless a bystander retrogradely conducting BT is present).[41,45]

Atrial and ventricular electrogram sequence following cessation of ventricular pacing

Technique. During SVT, synchronized ventricular overdrive pacing is initiated at a PCL 10 to 30 milliseconds shorter than the TCL until 1:1 VA conduction occurs, at which point pacing is stopped. If pacing results in termination of the tachycardia, SVT is reinduced, and the maneuver is repeated. If ventricular pacing does not terminate the tachycardia and the presence of stable 1:1 VA conduction is verified, the electrogram sequence immediately after the last paced ventricular complex is categorized as an atrial-ventricular (A-V) or atrial-atrial-ventricular (A-A-V) pattern (**see Fig. 11.16**).

Interpretation. During AVNRT or orthodromic AVRT, when the ventricle is paced at a CL shorter than the TCL and all electrograms are accelerated to the pacing rate without terminating the tachycardia, VA conduction occurs through the retrograde limb of the circuit. Therefore, after the last paced ventricular complex, the anterograde limb of the tachycardia circuit is not refractory, and the last entrained retrograde atrial complex can conduct to the ventricle. This results in an A-V response following cessation of pacing. However, when the ventricle is paced during AT and 1:1 VA conduction is produced, retrograde conduction occurs through the AVN. In this setting, the last retrograde



eFig. 20.1 Antitachycardia Pacing *(ATP)* Terminating Supraventricular Tachycardia (SVT). Intracardiac atrial, shock lead, and ventricular electrograms (*A-EGM, Can-RV coil EGM,* and *V-EGM,* respectively) stored by the implantable cardioverter defibrillator (ICD) during an episode of tachycardia at a cycle length (CL) of 275 to 285 milliseconds and a 1 : 1 atrioventricular relationship. The tachycardia triggered ATP therapy by the ICD with a burst of eight ventricular paced beats. ATP terminates the tachycardia, and sinus rhythm is restored. Note that the first three captured complexes during ATP failed to conduct to the atrium, and atrial activity continued unperturbed, which excludes ventricular tachycardia as the mechanism of the tachycardia. Furthermore, the first reset (accelerated) atrial complex occurs after the fourth captured paced ventricular complex *(red arrow)*, which is inconsistent with atrioventricular reentrant tachycardia (AVRT) and favors atrioventricular nodal reentrant tachycardia (AVNRT) or atrial tachycardia. The simultaneous atrial and ventricular activation during the tachycardia at the baseline line also excludes AVRT and favors AVNRT.

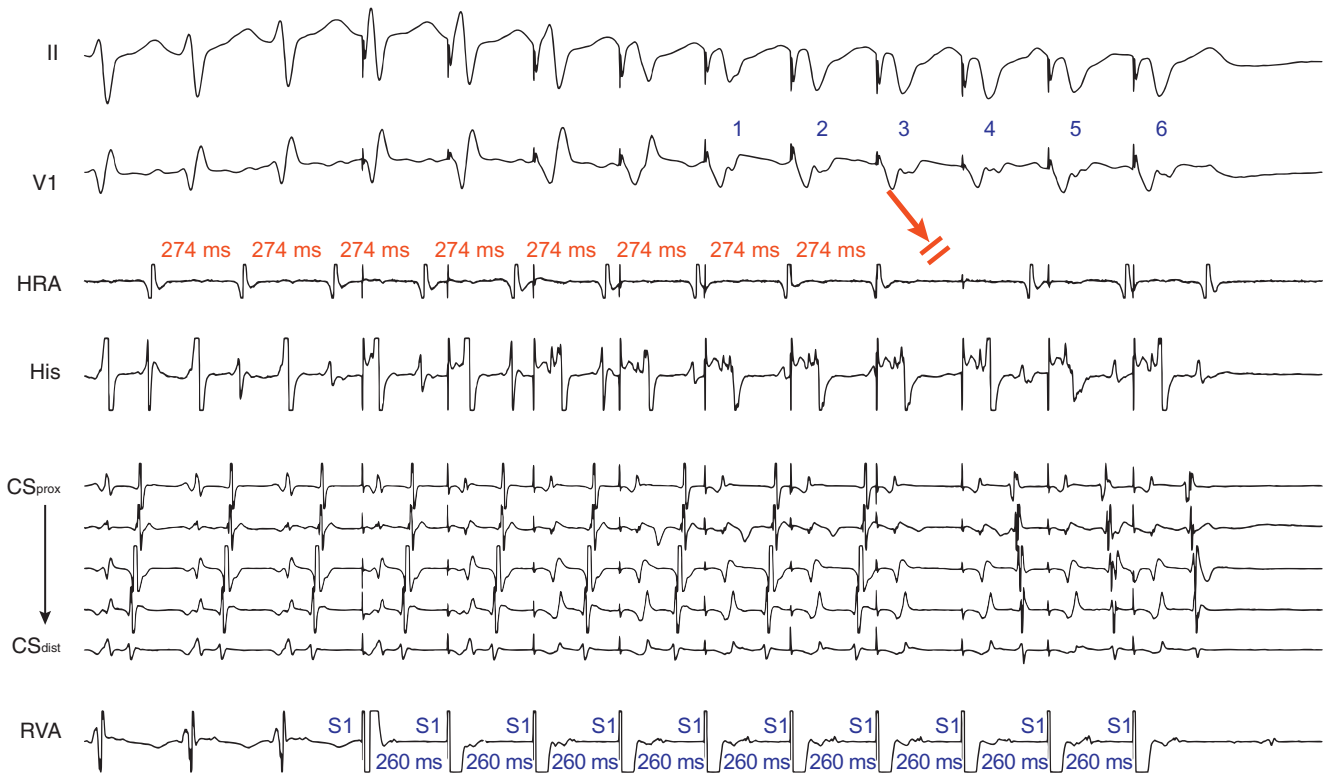


Fig. 20.15 Overdrive Ventricular Pacing During Atrioventricular Reentrant Tachycardia (AVRT). The tachycardia has a short RP interval and an eccentric atrial activation sequence consistent with orthodromic AVRT utilizing a left lateral bypass tract as the retrograde limb. Overdrive ventricular pacing *(S1)* started from the right ventricular apex *(RVA)* at a cycle length (CL) 14 milliseconds shorter than the tachycardia CL. The numbers 1 to 6 indicate the paced QRS complexes with stable morphology. Note that the first constant-appearing QRS complex to affect atrial activation is QRS #3 (as indicated by the *red arrow*). Although this finding typically indicates atrioventricular nodal reentrant tachycardia, and excludes orthodromic AVRT utilizing a septal bypass tract (BT), it can also be observed in orthodromic AVRT when the BT is remote from the ventricular pacing site, like in this case. CS_{dist}, Distal coronary sinus; CS_{prox}, proximal coronary sinus; *HRA,* high right atrium.

atrial complex resulting from ventricular pacing is unable to conduct back to the ventricle because the AVN is refractory to anterograde conduction and there is no alternate route for anterograde conduction to the ventricle available (unlike AVNRT and AVRT), and the result is an A-A-V response.

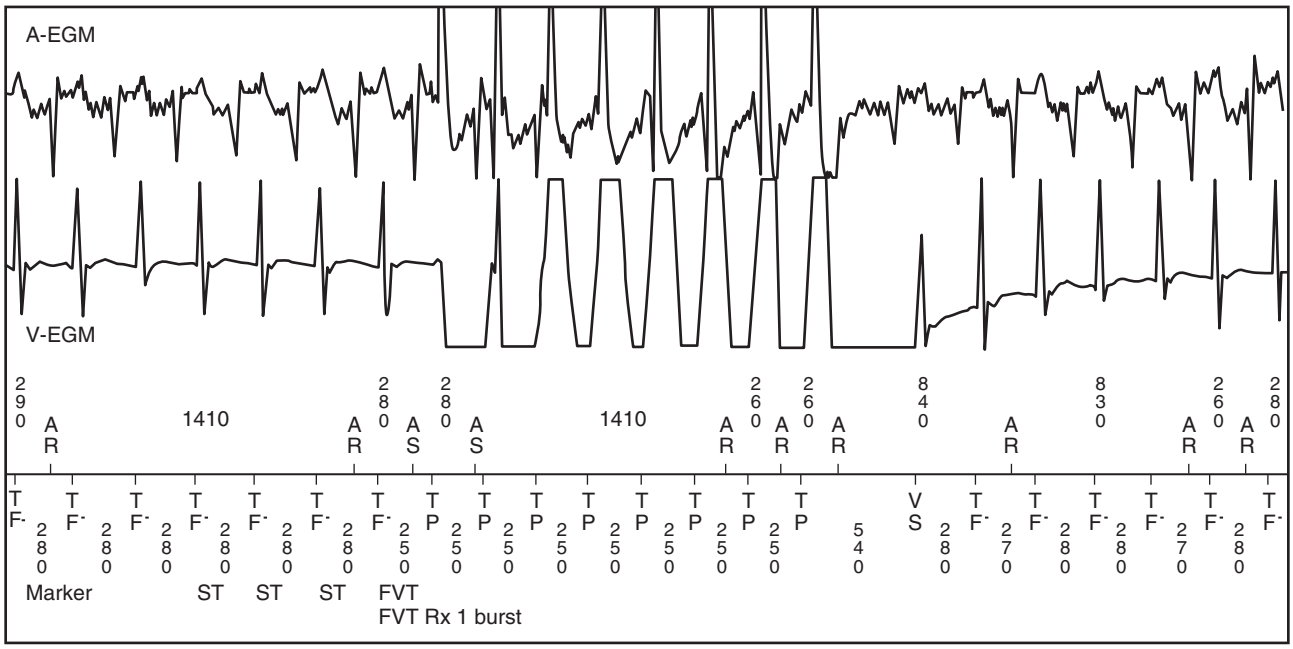
Pitfalls. This pacing maneuver is not useful when 1:1 VA conduction during ventricular pacing is absent. Thus, when determining the response after ventricular pacing during SVT, the presence of 1:1 VA conduction must be confirmed. Isorhythmic VA dissociation can mimic 1:1 VA conduction, especially when the pacing train is not long enough or the PCL is too slow (**see Fig. 11.17**).

Pseudo–A-A-V response. A pseudo–A-A-V response can occur during atypical AVNRT (eFig. 20.2). Because retrograde conduction during ventricular pacing occurs through the slow pathway, the VA interval is long and can be longer than the PCL (V-V interval), so that the last paced QRS is followed first by the atrial complex resulting from slow VA conduction of the preceding paced QRS and then by the atrial complex resulting from the last paced QRS. To avoid this potential pitfall, the A-A intervals are measured during and after ventricular overdrive pacing, and the last atrial electrogram resulting from VA conduction during ventricular pacing is carefully examined. The last retrograde atrial complex characteristically occurs at an A-A interval equal to the ventricular PCL, whereas the first tachycardia atrial complex usually occurs at a longer return CL.

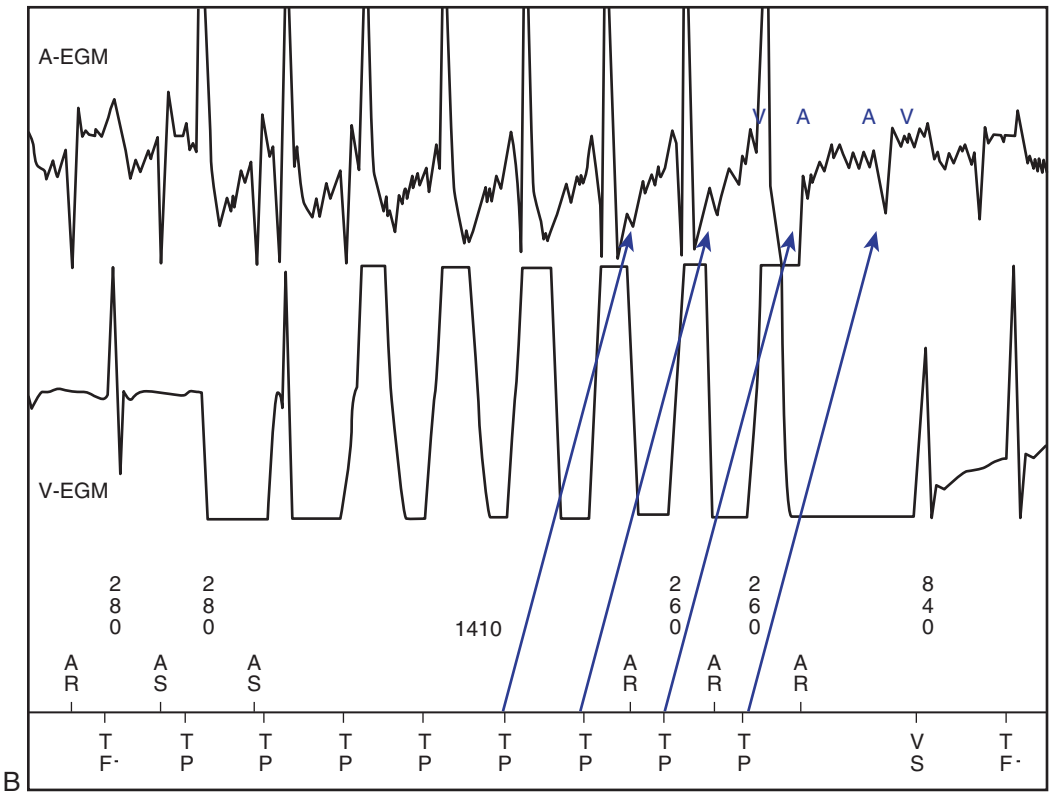
A pseudo–A-A-V response can also occur when 1:1 VA conduction is absent during overdrive ventricular pacing (see Fig. 20.12), during typical AVNRT with long HV intervals or short HA intervals (whereby atrial activation may precede ventricular activation), and in patients with a bystander BT. Replacing ventricular activation with His activation (i.e., characterizing the response as A-A-H or A-H instead of A-A-V or A-V, respectively) can be more accurate and can help eliminate the pseudo–A-A-V response in patients with AVNRT and long HV intervals, short HA intervals, or both.[46]

Pseudo–A-V response. A pseudo–A-V response can occur with an automatic AT when the maneuver is performed during isoproterenol infusion. Ventricular pacing with 1:1 VA conduction can result in overdrive suppression of the atrial focus, and isoproterenol can enhance junctional automaticity, so that an apparent A-V response occurs. Therefore, when ventricular pacing is performed during an isoproterenol infusion, it is important to determine that the response after cessation of ventricular pacing is reproducible.[28]

A pseudo–A-V response can theoretically occur when AT coexists with retrograde dual AVN pathways or a bystander BT. In such cases, the last retrograde atrial complex would have an alternative route for anterograde conduction to the ventricle, other than the one used for retrograde VA conduction during ventricular pacing, thus resulting in an A-V response. However, clinical occurrence of these theoretical scenarios has not been observed, probably because of retrograde penetration



A



B

eFig. 20.2 Antitachycardia Pacing (ATP) During Supraventricular Tachycardia (SVT). (A) Intracardiac atrial and ventricular electrograms (*A-EGM* and *V-EGM,* respectively) stored by the implantable cardioverter defibrillator (ICD) during an episode of SVT at a cycle length (CL) of 280 to 290 milliseconds and a 1:1 atrioventricular relationship. The SVT inappropriately triggered ATP therapy by the ICD with a burst of eight ventricular paced beats at a CL of 250 milliseconds. ATP fails to terminate the tachycardia, which resumes at the baseline CL. (B) Magnification of a portion of the upper panel showing atrial and ventricular intracardiac electrograms during ATP. Arrows track ventriculoatrial (VA) conduction following the last four paced ventricular complexes. VA conduction during ATP is present and can be verified by demonstrating acceleration of the atrial rate to that of the paced ventricular rate. After cessation of ATP, the tachycardia resumes with an atrial-atrial-ventricular (A-A-V) electrogram sequence, which is evident on interrogation of the atrial and ventricular channel electrograms *(lower panel)*. However, careful analysis to identify the last reset atrial complex (occurring at a CL similar to the pacing CL) indicates a pseudo–A-A-V response rather than an A-A-V electrogram sequence, which is consistent with atypical AV nodal reentrant tachycardia as the mechanism of the SVT. See text for discussion.

of both AVN pathways or of the AVN and the BT during ventricular pacing, so that both pathways are refractory to anterograde conduction on cessation of pacing.[28]

Termination. Ventricular pacing can easily terminate orthodromic AVRT, and failure to terminate the SVT with ventricular pacing argues against orthodromic AVRT. Termination of AVNRT is also common, but AT is less likely to be terminated by ventricular pacing.

Para-Hisian Pacing During Tachycardia

Para-Hisian entrainment or resetting

Technique. Entrainment of the tachycardia is performed by pacing at the para-Hisian region using the HB catheter at a PCL 10 to 30 milliseconds shorter than the TCL. Entrainment is confirmed when the atrial CL accelerates to the PCL, without a change in the atrial activation sequence, and the tachycardia continues after pacing is discontinued.[47]

Para-Hisian entrainment is performed by alternately pacing at high-energy output for HB-RB capture or lower energy output for HB-RB noncapture. Entrainment with HB-RB capture is recorded separately from that without HB-RB capture. The SA (stimulus-to-earliest atrial activation) and local VA intervals during HB-RB capture and noncapture are then examined.

One must be cautious about performing the para-Hisian entrainment maneuver by simply decreasing the pacing energy output during the same run to achieve HB-RB noncapture. That is, even though the SVT may have been entrained during HB-RB capture, on loss of HB-RB capture, the initial paced complexes typically do not entrain the SVT. This initial failure of entrainment occurs because of the sudden increase in the distance from the pacing site to the actual reentrant circuit. During HB-RB capture of AVNRT, the pacing site is near the circuit (the HB-RB); however, during HB-RB noncapture, the pacing site (the basal RV myocardium) is well outside the circuit. This limitation would not apply if HB-RB noncapture is performed *prior* to HB-RB capture. That is, if the pacing output is increased while the SVT is being entrained during HB-RB noncapture, the circuit almost certainly will be entrained on HB-RB capture (unless the SVT terminates).

If para-Hisian entrainment cannot be performed because of repetitive termination of the tachycardia during entrainment attempts, isoproterenol or epinephrine infusion may be used to help sustain the rhythm. Alternatively, single or double VESs can be given to reset the tachycardia (para-Hisian resetting). These VESs are delivered at progressively shorter coupling intervals until the first VES that reliably advances or resets the tachycardia. This is performed alternately with high- or low-energy outputs to achieve HB-RB capture and noncapture, respectively. As with para-Hisian entrainment, the retrograde atrial activation sequence and timing are compared during para-Hisian resetting to characterize the response.

Interpretation. In AVNRT (typical or atypical), the AVN-AVN pattern is observed in response to para-Hisian entrainment or resetting. Both the SA and the local VA intervals increase during HB-RB noncapture compared with HB-RB capture.

In orthodromic AVRT, the BT-BT pattern or BT-BT_L pattern is observed. In the setting of a BT-BT pattern, the SA and local VA intervals are usually not significantly different between HB-RB capture and noncapture. Conversely, in the case of a BT-BT_L pattern, the SA interval increases on HB-RB noncapture, but without significant change in the local VA interval.

A ΔSA interval of less than 40 milliseconds was found to be a reasonable guide to separating the AVN-AVN from the BT-BT response. Patients with AVNRT uniformly have a ΔSA interval of greater than 40 milliseconds, whereas those with AVRT have a ΔSA interval of less than 40 milliseconds (except for rare patients with a left lateral BT). Using the Δ local VA interval (instead of the ΔSA interval) provides a more accurate parameter for discrimination between AVNRT and AVRT.

Fusion patterns during para-Hisian entrainment or resetting have not been observed in patients with AVNRT. This is a potential advantage over para-Hisian pacing during NSR in identifying the presence of a BT. Because retrograde VA conduction can only proceed over a single route during entrainment of the SVT (assuming that a complex scenario such as multiple BTs is not present), the various forms of retrograde fusion that might be seen with para-Hisian pacing during NSR cannot occur with para-Hisian entrainment or resetting.

Para-Hisian overdrive pacing

Technique. During SVT, para-Hisian pacing is performed at a PCL 10 to 30 milliseconds shorter than the TCL. Responses to this maneuver are then classified as follows: (1) entrainment occurs when the atrial CL is accelerated to the PCL, without a change in the atrial activation sequence, and the tachycardia resumed after pacing was discontinued; (2) termination occurs when pacing results in termination of the tachycardia; and (3) AV dissociation occurs when HB capture is confirmed and no change in the atrial CL is observed.

When response 1 or 2 is observed, the number of beats required to enter the tachycardia circuit is determined. Any change in atrial timing (advance or delay of at least 10 milliseconds) or termination of the SVT is considered to represent entry into the tachycardia circuit. The surface ECG is examined to determine the first beat where a consistent nonfused paced QRS morphology is observed. This beat is taken as beat 1 for the purposes of analysis. The number of beats required to enter the tachycardia circuit is determined by counting from beat 1 each time the maneuver is performed.

Interpretation. Because the HB is a necessary component for the circuit in AVRT, overdrive capture of the HB during AVRT should result in immediate entry into the tachycardia. In contrast, HB overdrive pacing during AVNRT may not immediately enter the circuit because the HB is not an obligate component. Therefore entry into the tachycardia circuit within 1 beat indicates AVRT, whereas entry into the circuit occurring only after 3 or more beats is consistent with AVNRT.[48]

This technique does not require confirmation of both HB capture and noncapture. In addition, unlike para-Hisian entrainment whereby termination of the SVT occurs frequently and precludes analysis of ΔSA interval, termination of SVT during para-Hisian overdrive pacing serves as confirmation that the pacing stimulus has entered and perturbed the tachycardia circuit.[48]

Of note, this principle should apply to any tachycardia using a BT and the HB, including atriofascicular, concealed nodofascicular, decremental, and left lateral BTs. Moreover, it should be useful in distinguishing AVNRT with bystander pathway participation from antidromic AVRT.[48]

Diagnostic Maneuvers During Sinus Rhythm After Tachycardia Termination

When pacing the atrium or ventricle at the TCL, it is important that the autonomic tone be similar to its state during the tachycardia because alterations of autonomic tone can independently influence AV or VA conduction.

Atrial Pacing at the Tachycardia Cycle Length

ΔAH interval. The difference in the AH interval between atrial pacing (at the TCL) and SVT can allow differentiation of fast-slow AVNRT from other types of long RP tachycardias. A ΔAH (HA_{pacing} – AH_{SVT}) greater than 40 milliseconds favors AVNRT. In contrast, during AT and orthodromic AVRT utilizing a septal BT, the AH interval during SVT approximates that during atrial pacing. In contrast, a ΔAH of less than 20 milliseconds favors AT and orthodromic AVRT. This has only

been tested with RA pacing during right ATs and should be applied with caution when a left AT is suspected.[11]

The ΔAH ($AH_{pacing} - AH_{SVT}$) can also help differentiate typical (slow-fast) AVNRT from AT with a long PR interval. Atrial pacing during NSR at the TCL yields an AH interval that is similar to that during AT but shorter than the AH interval during typical AVNRT. During atrial pacing and AT, AV conduction occurs preferentially over the fast pathway and, hence, is expected to be associated with similar AH and PR intervals (under comparable autonomic tone). In contrast, AV conduction during typical AVNRT occurs over the slow pathway, resulting in a long AH interval.

Ventricular Pacing at the Tachycardia Cycle Length

ΔHA interval. To help distinguish between orthodromic AVRT and AVNRT, ventricular pacing is performed during NSR (with PCL equal to the TCL), and the HA interval during SVT is compared to that during ventricular pacing. The HA interval is measured from the *end* of the His potential (where the impulse leaves the HB to enter the AVN) to the atrial electrogram in the high RA recording and the ΔHA interval ($HA_{pacing} - HA_{SVT}$) is calculated.

In the setting of orthodromic AVRT, the ΔHA interval is typically less than −10 milliseconds. Ventricular pacing results in HA and VA intervals that are shorter than those during orthodromic AVRT, because the HB and atrium are activated sequentially during orthodromic AVRT but in parallel during ventricular pacing whereby the atrium is activated via the BT (**see Fig. 18.43**).

In contrast, in the setting of AVNRT the ΔHA interval is typically more than −10 milliseconds because the HA interval during AVNRT is shortened by parallel activation of both the HB and the atrium during the tachycardia (i.e., the HA interval is a "pseudo-interval" that represents activation times of the HB and atrium), whereas the HB and atrium are activated sequentially during ventricular pacing in the absence of a BT (i.e., the HA interval represents a true conduction time interval from the HB to the atrium). The ΔHA interval is even more pronounced in atypical AVNRT, which has a lower common pathway that is longer than that in typical AVNRT. In focal junctional tachycardia, the ΔHA interval is typically close to 0.[49]

The main limitation of the ΔHA interval criterion is the ability to record the retrograde His potential during ventricular pacing. Retrograde His potential generally appears before the local ventricular electrogram in the HB tracing, and can be verified by the introduction of a VES that causes the His potential to occur after the local ventricular electrogram. Moreover, pacing from different sites (e.g., midseptum) may allow earlier penetration into the HPS and facilitate observation of a retrograde His potential. When the retrograde His potential is not visualized, using the ΔVA interval instead of the ΔHA interval is not as accurate in discriminating orthodromic AVRT from AVNRT.[4]

Another limitation is that VA conduction during ventricular pacing may not occur over the BT but propagates preferentially over the HPS-AVN, leading to earlier atrial activation over this pathway than over the BT. If this were the case, the HA interval during ventricular pacing would be shorter than that observed if the atrium were activated via the BT. This would yield a more negative ΔHA interval.

VA block. In the setting of AT and AVNRT, and under comparable autonomic tone, 1:1 VA conduction over the AVN may or may not be maintained during ventricular pacing at a PCL similar to the TCL because of possible retrograde block in the AVN or the lower common pathway. Anterograde conduction properties of the AVN or the lower common pathway may allow 1:1 conduction from the AT or AVNRT circuit down to the ventricle, but its retrograde conduction properties may not allow 1:1 retrograde conduction from the ventricle up to the atrium during ventricular pacing at a CL similar to the TCL. On the contrary, in the setting of orthodromic AVRT, 1:1 VA conduction is expected to be maintained because of the presence of an AV BT that is capable of mediating VA conduction at rate at least as fast as that during the AVRT. Therefore, if VA block is observed during ventricular pacing at the TCL, orthodromic AVRT is excluded (with the exception of orthodromic AVRT using a slowly conducting AV BT), and AT or AVNRT with lower common pathway physiology is more likely.

Atrial activation sequence. The retrograde atrial activation sequence during ventricular pacing is usually similar to that during SVT in the setting of AVNRT, but slight differences of activation sequence can also be observed when the retrogradely conducing AVN pathway during AVNRT is different from that utilized during ventricular pacing. In the setting of orthodromic AVRT, a retrograde atrial activation sequence during ventricular pacing can be similar to or different from that during tachycardia, depending on whether retrograde VA conduction during ventricular pacing propagates over the AVN, the BT, or both. For AT, the retrograde atrial activation sequence during ventricular pacing is usually different from that during AT, except for ATs originating close to the AV junction.

PRACTICAL APPROACH TO ELECTROPHYSIOLOGICAL DIAGNOSIS OF SUPRAVENTRICULAR TACHYCARDIA

It is important to understand that there is no single diagnostic maneuver or algorithm that is adequate in distinguishing among the different types of SVTs in all cases. Each maneuver has its own applications and limitations, and almost all described diagnostic maneuvers have exceptions to their primary interpretation.[4] Although several diagnostic criteria were found to have high specificity, sensitivity is frequently limited. Therefore the investigator will often need to use a combination of SVT features and pacing maneuvers to establish an accurate diagnosis. Systematic evaluation of all possibilities and adherence to fundamental EP principles will help establish the correct diagnosis. Each step during the EP study in these patients can offer valuable information to the vigilant investigator that, if recognized, can potentially reduce procedure time and improve outcome.

It is important for the electrophysiologist to be fully conversant with the maneuvers used for the differential diagnosis of the different arrhythmias. Exercising the application of these techniques on a routine basis, even when the diagnosis of the underlying arrhythmia mechanism has been established, helps the operator to correctly apply those diagnostic maneuvers in the more challenging cases and become familiar with the pitfalls, exceptions, and the spectrum of behavior of each technique.

Tables 20.1 to 20.3 and Fig. 20.16 outline some of the proposed strategies for the EP diagnosis of narrow complex SVTs. Baseline tachycardia features, atrial and ventricular programmed stimulation during the tachycardia and then during sinus rhythm after tachycardia termination, provide a diagnosis of the mechanism of SVT in the vast majority of cases.

Step 1

Characterize the A/V relationship, RP-PR ratio, and atrial activation sequence during tachycardia (see Table 20.1). The differential diagnosis is most challenging in the setting of SVTs with a concentric atrial activation sequence and a 1:1 A/V relationship. SVTs with very short RP intervals (simultaneous atrial and ventricular activation) and a concentric atrial activation sequence include: (1) typical AVNRT; (2) AT with a focus close to the AV junction with a long PR interval; (3) and junctional tachycardia. The differential diagnosis of a short

TABLE 20.1 Diagnostic Strategy for Narrow QRS Supraventricular Tachycardia: Tachycardia Features

Atrial activation sequence	• Eccentric atrial activation sequence excludes AVNRT (except for the left variant of AVNRT). • An initial atrial activation site away from the AV groove and AV junction is diagnostic of AT and excludes both AVNRT and orthodromic AVRT.
RP-PR interval ratio	• Short RP with VA interval of <70 msec or a ventricular–to–high RA interval of <95 msec during SVT excludes orthodromic AVRT, and is consistent with AVNRT (most common), but can occur during AT with a long PR interval or junctional tachycardia (rare). • Short RP with VA interval of >70 msec suggests orthodromic AVRT (most common), slow-slow AVNRT, and AT with a long PR interval. • Long RP suggests fast-slow AVNRT, PJRT, and AT.
AV block	• Spontaneous or induced AV block with continuation of the tachycardia is consistent with AT, excludes AVRT, and is uncommon in AVNRT.
VA block	• VA block excludes orthodromic AVRT, and rarely occurs during AVNRT. • Other potential mechanisms of SVT with VA block include junctional tachycardia with retrograde VA block and nodofascicular or nodoventricular reentrant tachycardia.
Effects of BBB	• BBB does not affect the TCL or VA interval in AT, AVNRT, or orthodromic AVRT using a BT contralateral to the BBB. • BBB that prolongs the VA interval, with or without affecting the TCL, is diagnostic of orthodromic AVRT using an AV BT ipsilateral to the BBB and excludes AT and AVNRT.
Tachycardia CL variations	• Spontaneous changes in PR and RP intervals with a fixed A-A interval is consistent with AT, and excludes orthodromic AVRT. • Spontaneous changes in the TCL accompanied by a constant VA interval ("VA linking") favor orthodromic AVRT. • Changes in atrial CL preceding similar changes in subsequent ventricular CL strongly favor AT or atypical AVNRT. • Changes in ventricular CL preceded by similar changes in the atrial CL favor typical AVNRT or orthodromic AVRT.
Tachycardia termination	• Termination of SVT (spontaneous or in response to adenosine or vagal maneuvers) with a P wave not followed by a QRS practically excludes AT, except in the case of a nonconducted PAC terminating the AT. • Reproducible SVT termination in response to adenosine with a QRS not followed by a P wave excludes orthodromic AVRT using a rapidly conducting AV BT as the retrograde limb, is unusual in typical AVNRT, and is consistent with AT, PJRT, or atypical AVNRT.

A-A, Atrial-atrial; *AT,* atrial tachycardia; *AV,* atrioventricular; *AVNRT,* atrioventricular nodal reentrant tachycardia; *AVRT,* atrioventricular reentrant tachycardia; *BBB,* bundle branch block; *BT,* bypass tract; *CL,* cycle length; *PAC,* premature atrial complex; *PJRT,* permanent junctional reciprocating tachycardia; *RA,* right atrium; *SVT,* supraventricular tachycardia; *TCL,* tachycardia cycle length; *VA,* ventriculoatrial.

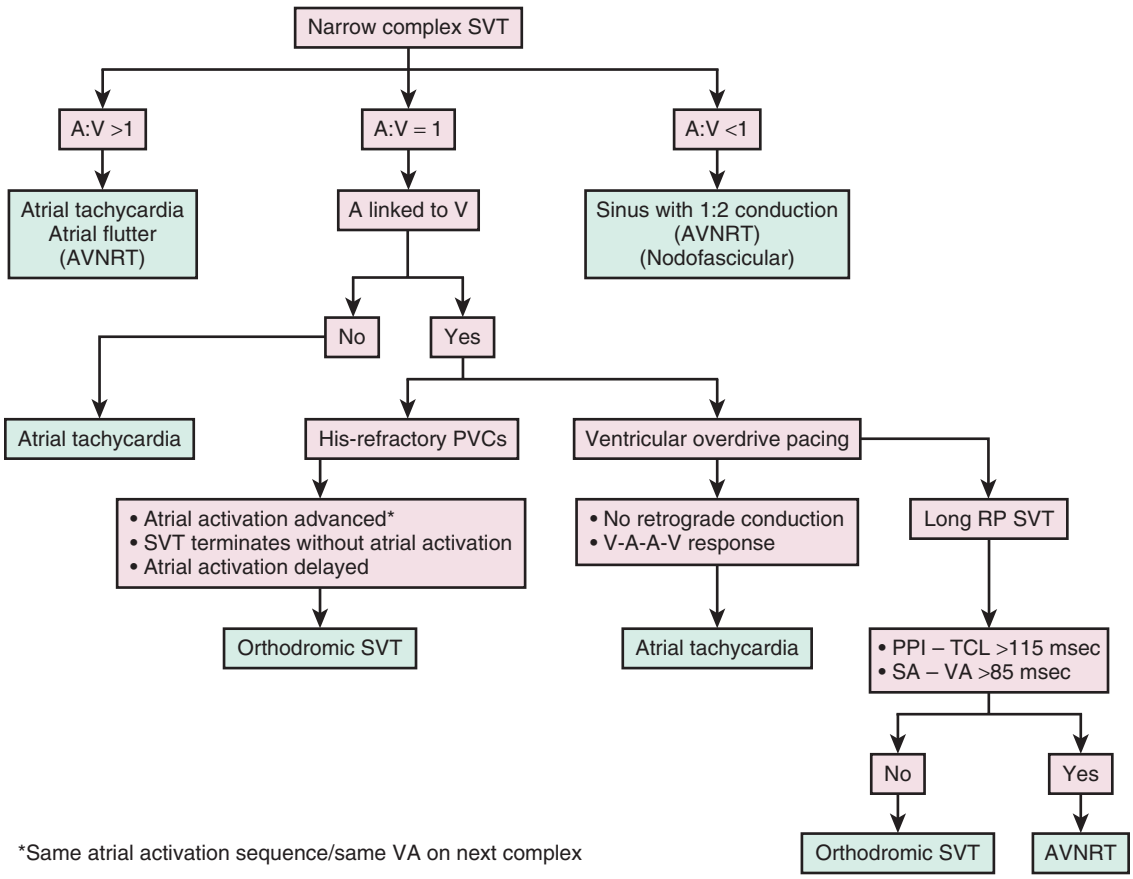


Fig. 20.16 Algorithm for Diagnosis of Narrow Complex Supraventricular Tachycardia *(SVT).* Items in parentheses are rarely seen. *AVNRT,* Atrioventricular nodal reentry; *CL,* cycle length; *PPI,* postpacing interval; *PVC,* premature ventricular complex; *SA,* stimulus-to-atrial interval; *TCL,* tachycardia cycle length; *VA,* ventriculoatrial interval.

TABLE 20.2 Diagnostic Strategy for Narrow QRS Supraventricular Tachycardia: Programmed Electrical Stimulation During Tachycardia

Ventricular Extrastimulation	
Resetting with ventricular fusion	• Resetting with manifest QRS fusion is consistent with orthodromic AVRT and excludes both AVNRT and focal AT.
Preexcitation index	• Preexcitation index (the difference between the TCL and the longest VES coupling interval at which atrial capture occurs during tachycardia) ≥100 msec characterizes AVNRT, whereas an index <45 msec is consistent with AVRT with a septal BT.
VA interval	• ΔVA interval ($VA_{pacing} - VA_{SVT}$) >110 msec (after resetting with single or double VESs from the RV apex) is consistent with AVNRT.
Postpacing interval	• Corrected (PPI – TCL) >110 msec (after resetting with single or double VESs from the RV apex) is consistent with AVNRT.
When the VES advances the next atrial activation	• Advancement of the next atrial impulse occurring with a VES delivered when the HB is not refractory usually does not help differentiate among the different types of SVTs. • If advancement occurs when the HB is refractory, AVNRT is excluded. • If advancement occurs with an atrial activation sequence similar to that during SVT, regardless of the timing of the VES, AT is less likely than AVNRT or orthodromic AVRT. • When advancement occurs when the HB is refractory, with an atrial activation sequence similar to that during SVT, AVNRT is excluded, AT is unlikely, and orthodromic AVRT is the most likely diagnosis.
When the VES delays the next atrial activation	• When the VES causes delay in the next atrial activation, regardless of the timing of the VES, AT is excluded, and when such delay occurs with a VES delivered when the HB is refractory, both AVNRT and AT are excluded.
When the VES resets the next QRS without atrial activation	• When a VES resets the SVT without atrial activation (i.e., the VES advances the subsequent His potential and QRS), both AT and orthodromic AVRT are excluded.
When the VES terminates the SVT	• When termination occurs reproducibly with a VES delivered when the HB is refractory, AVNRT is excluded, and the presence of a retrogradely conducting AV BT is diagnosed. • If the atrial activation sequence following the VES is similar to that during the SVT, orthodromic AVRT is indicated and AT is practically excluded, except for the rare case in which AT originates close to the atrial insertion site of an innocent bystander AV BT. • When termination occurs reproducibly with a VES that does not activate the atrium, regardless of the timing of the VES in relation to the HB, AT is excluded; when this phenomenon is observed with a VES delivered when the HB is refractory, both AT and AVNRT are excluded and orthodromic AVRT is diagnosed.
When the VES fails to affect the next atrial activation	• If resetting does not occur with a relatively late VES, this usually does not help in the differential diagnosis of SVT. • If resetting does not occur with an early VES, despite advancement (by >30 msec) of the local ventricular activation at ventricular sites near the site of earliest atrial activation, orthodromic AVRT and the presence of a retrogradely conducting AV BT are excluded.
Ventricular Pacing	
VA dissociation	• When overdrive ventricular pacing during SVT fails to accelerate atrial CL to the PCL (i.e., the ventricles are dissociated from the tachycardia), AVRT is excluded, AT is the most likely diagnosis, but AVNRT is still possible.
VA interval	• ΔVA interval ($VA_{pacing} - VA_{SVT}$) >85 msec is consistent with AVNRT. • ΔVA of <85 msec is consistent with orthodromic AVRT.
Postpacing interval	• (PPI – TCL) >115 msec (or corrected [PPI – TCL] >110 msec) is consistent with AVNRT. • (PPI – TCL) <115 msec (or corrected [PPI – TCL] <110 msec) is consistent with orthodromic AVRT.
Entrainment with ventricular fusion	• Manifest ventricular fusion during entrainment indicates AVRT and excludes both AVNRT and AT.
Differential-site right ventricular entrainment	• Differential corrected (PPI – TCL) of >30 msec after transient entrainment from the RV apex vs. the RV base is consistent with AVNRT. • Differential corrected (PPI – TCL) of <30 msec is consistent with orthodromic AVRT. • Differential VA interval (ventricular stimulus-to-atrial interval during entrainment from RV base vs. RV septum) of >20 msec is consistent with AVNRT. • Differential VA interval of <20 msec is consistent with orthodromic AVRT.
Atrial resetting during the transition zone	• Atrial resetting (perturbation of atrial timing by >15 msec) during the transition zone is consistent with orthodromic AVRT and excludes AVNRT or AT (unless a bystander retrogradely conducting BT is present).
Length of pacing drive required for entrainment	• Acceleration of the SVT to the PCL occurring by the first beat with stable paced QRS morphology is consistent with orthodromic AVRT and essentially excludes AVNRT. • Acceleration of the SVT to the PCL occurring only after ≥2 beats with stable paced QRS morphology is consistent with AVNRT and excludes orthodromic AVRT.
Atrial activation sequence during ventricular pacing	• Atrial activation sequence during pacing different from that during the SVT is consistent with AT and practically excludes both orthodromic AVRT and AVNRT. • Atrial activation sequence during pacing similar to that during the SVT favors orthodromic AVRT or AVNRT over AT.
Atrial and ventricular electrogram sequence following cessation of ventricular pacing	• A-A-V response is consistent with AT (as long as pseudo–A-A-V response is excluded). • A-V response is consistent with AVNRT and orthodromic AVRT.

Continued

TABLE 20.2 Diagnostic Strategy for Narrow QRS Supraventricular Tachycardia: Programmed Electrical Stimulation During Tachycardia—cont'd

Atrial Extrastimulation	
Resetting with atrial fusion	• Demonstration of resetting with manifest atrial fusion excludes AVNRT and focal AT.
Atrial Pacing	
Entrainment during atrial pacing	• Demonstration of entrainment excludes automatic and triggered activity ATs.
Entrainment with atrial fusion	• Demonstration of entrainment with manifest atrial fusion excludes AVNRT and focal AT.
Overdrive suppression	• Overdrive suppression of the SVT favors automatic AT and excludes AVNRT and orthodromic AVRT.
AH interval	• ΔAH interval (AH_{pacing} – AH_{SVT}) of >40 msec favors AVNRT over AT and orthodromic AVRT. • ΔAH interval of <20 msec favors AT and orthodromic AVRT over AVNRT.
VA linking	• On cessation of overdrive atrial pacing (with 1 : 1 AV conduction), if the VA interval following the last entrained QRS is reproducibly constant (<10 msec variation), despite pacing at different CLs or for different durations (VA linking) and similar to that during SVT, AT is unlikely. • If no VA linking is demonstrable, AT is more likely than other types of SVT.
Differential-site atrial pacing	• On cessation of overdrive atrial pacing (with 1 : 1 AV conduction) from different atrial sites (high RA and proximal CS) at the same PCL, a maximal difference in the postpacing VA intervals (the interval from last captured ventricular electrogram to the earliest atrial electrogram of the initial tachycardia beat after pacing) among the different atrial pacing sites (ΔVA interval) of >14 msec is consistent with AT. • ΔVA interval of <14 msec favors AVNRT or orthodromic AVRT over AT.
Para-Hisian Entrainment or Resetting	
SA and local VA intervals	• Prolongation of the SA and the local VA intervals on loss of HB capture, compared with that during HB capture is consistent with AVNRT. • SA and the local VA intervals that remain constant regardless of whether the HB-RB is being captured indicate orthodromic AVRT. • SA interval prolongation on HB-RB noncapture, but without significant change in the local VA interval, indicates orthodromic AVRT. • The change in the stimulus to earliest atrial activation time (ΔSA interval) with loss of HB-RB capture >40 msec is consistent with AVNRT. • ΔSA interval <40 msec indicates AVRT (except for rare patients with a left lateral BT).
The number of beats required to capture the atrium with para-Hisian pacing during SVT	• Entry into the tachycardia circuit within 1 beat indicates AVRT, whereas entry into the circuit occurring only after 3 or more beats is consistent with AVNRT.

A-A-V, Atrial-atrial-ventricular; *A-V,* atrial-ventricular; *AH,* atrial-His bundle interval; *AT,* atrial tachycardia; *AV,* atrioventricular; *AVNRT,* atrioventricular nodal reentrant tachycardia; *AVRT,* atrioventricular reentrant tachycardia; *BT,* bypass tract; *CL,* cycle length; *CS,* coronary sinus; *HB,* His bundle; *PCL,* pacing cycle length; *PPI,* postpacing interval; *RA,* right atrium; *RB,* right bundle branch; *RV,* right ventricle; *SA,* stimulus-atrial; *SVT,* supraventricular tachycardia; *TCL,* tachycardia cycle length; *VA,* ventriculoatrial; *VES,* ventricular extrastimulus.

TABLE 20.3 Diagnostic Strategy for Narrow QRS Supraventricular Tachycardia: Programmed Electrical Stimulation During Sinus Rhythm

Tachycardia Induction	
Catecholamine administration	• Spontaneous SVT initiation during isoproterenol administration is consistent with automatic AT or junctional tachycardia.
AES	• "Critical AH" required for SVT induction is consistent with typical AVNRT. • If the VA interval of the first tachycardia beat is reproducibly identical to that during the rest of the SVT ("VA linking"), AT is very unlikely, and is suggestive of typical AVNRT and orthodromic AVRT.
VES	• ΔVA (VA_{VES} – VA_{SVT}) <85 msec is consistent with orthodromic AVRT. • ΔVA interval >85 msec favors AVNRT. • (PPI_{VES} – TCL) <115 msec is consistent with orthodromic AVRT. • (PPI_{VES} – TCL) >115 msec favors AVNRT.
Atrial Pacing From High Right Atrium at Tachycardia Cycle Length	
AH interval	• ΔAH interval (AH_{pacing} – AH_{SVT}) of >40 msec favors AVNRT over AT and orthodromic AVRT. • ΔAH interval of <20 msec favors AT and orthodromic AVRT over AVNRT.
Ventricular Pacing From Right Ventricular Apex at Tachycardia Cycle Length	
HA interval	• ΔHA interval (HA_{pacing} – HA_{SVT}) of more than –10 msec favors AVNRT. • ΔHA interval of less than –10 msec favors orthodromic AVRT.

TABLE 20.3 Diagnostic Strategy for Narrow QRS Supraventricular Tachycardia: Programmed Electrical Stimulation During Sinus Rhythm—cont'd

Retrograde atrial activation sequence	• Retrograde atrial activation sequence during ventricular pacing similar to that during SVT favors orthodromic AVRT and AVNRT over AT.
VA block	• The presence of VA block or decremental VA conduction during ventricular pacing at a CL similar to the TCL and under comparable autonomic tone argues against orthodromic AVRT and favors AT and AVNRT.
Differential-Site Right Ventricular Pacing	
VA interval	• When the VA (S-A) interval during RV apical pacing is shorter than that during RV basal pacing, a retrogradely conducting septal BT is excluded. • When the VA (S-A) interval during RV apical pacing is longer than that during RV basal pacing, a retrogradely conducting AV BT is diagnosed.
Atrial activation sequence	• Retrograde atrial activation sequence that is different depending on the site of ventricular pacing indicates the presence of a BT. • Constant atrial activation sequence does not help exclude or prove the presence of an AV BT.
Para-Hisian Pacing	
Atrial activation sequence	• Identical retrograde atrial activation sequence, with and without HB capture, indicates that retrograde conduction is occurring over the same system during HB-RB capture and noncapture (either the BT or AVN) and does not help prove or exclude the presence of BT. • Retrograde atrial activation sequence that is different depending on whether HB is captured indicates the presence of a BT.
HA and VA intervals	• VA (S-A) interval (recorded at multiple sites, including close to the site of earliest atrial activation during SVT) that is constant regardless of whether the HB-RB is being captured indicates the presence of a BT. • Prolongation of the VA (S-A) interval on loss of HB capture, compared with that during HB capture, excludes the presence of a retrogradely conducting BT, except for slowly conducting and far free-wall BTs.
Dual-Chamber Sequential Extrastimulation	
VA conduction	• VA conduction of V_2 indicates the presence of a retrogradely conducting BT.

AES, Atrial extrastimulation; *AH,* atrial-His bundle interval; *AT,* atrial tachycardia; *AV,* atrioventricular; *AVN,* atrioventricular node; *AVNRT,* atrioventricular nodal reentrant tachycardia; *AVRT,* atrioventricular reentrant tachycardia; *BT,* bypass tract; *CL,* cycle length; *HA,* His bundle–atrial; *HB,* His bundle; *PPI,* postpacing interval; *RB,* right bundle branch; *RV,* right ventricle; *S-A,* stimulus-atrial; *SVT,* supraventricular tachycardia; *TCL,* tachycardia cycle length; *VA,* ventriculoatrial; *VES,* ventricular extrastimulus.

RP SVT with a concentric atrial activation sequence includes (1) AT with a focus close to the AV junction; (2) orthodromic AVRT using a septal or paraseptal BT; and (3) slow-slow AVNRT. Long RP SVTs with a concentric atrial activation sequence include: (1) atypical (fast-slow) AVNRT; (2) PJRT; and (3) nodofascicular reentrant tachycardia (orthodromic AVRT using a concealed nodofascicular/nodoventricular BT). Narrow-complex tachycardia with VA block is rare; the differential diagnosis includes (1) typical AVNRT; (2) junctional tachycardia; and (3) nodofascicular reentrant tachycardia (orthodromic AVRT using a concealed nodofascicular/nodoventricular BT).

Step 2
Overdrive Ventricular Pacing During the Supraventricular Tachycardia

Ventricular pacing during the SVT represents the single most important diagnostic maneuver and can provide several clues to the diagnosis of most SVTs. Therefore it is preferable to employ this maneuver as an initial step in the diagnostic approach. When sustained SVT is inducible, overdrive pacing from the RV apex and RV base is performed at a PCL 10 to 30 milliseconds shorter than the TCL; the PCL is then progressively reduced by 10 to 20 milliseconds in a stepwise fashion with cessation of ventricular pacing after each PCL to verify continuation versus termination of the SVT. Several diagnostic criteria can be applied including the VA interval during pacing versus tachycardia, PPI, atrial activation sequence, presence of manifest ventricular fusion, and the length of pacing drive required to enter the tachycardia circuit, and the atrial and ventricular electrogram sequence following cessation of pacing (see Table 20.2).[30]

Step 3
Ventricular Extrastimulation During Supraventricular Tachycardia

Subsequently, a VES is delivered when the HB is refractory and then at progressively shorter VES coupling intervals (approximately 10-millisecond stepwise shortening of the VES coupling interval) so as to scan all of diastole. First, ventricular capture of the VES should be verified, and then the effect of the VES on the following atrial activation (advancement, delay, termination, or no effect) should be evaluated, as well as the timing of the VES in relation to the expected His potential during the SVT. Furthermore, conduction of the VES to the atrium and sequence of atrial activation following the VES should be carefully examined (see Fig. 20.11).[30]

Step 4
Overdrive Atrial Pacing During the Supraventricular Tachycardia

Atrial pacing is performed at a PCL 10 to 20 milliseconds shorter than the TCL. The PCL is then progressively reduced by 10 to 20 milliseconds in a stepwise fashion, with discontinuation of atrial pacing after each PCL to ensure continuation versus termination of the SVT. The AH interval, and presence of entrainment, atrial fusion, and VA linking should be evaluated (see Table 20.2).[30]

Step 5

After tachycardia termination, ventricular and atrial pacing at the TCL, differential site RV pacing (RV apex vs. base), and para-Hisian pacing maneuvers are performed (see Table 20.3).

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