Narrow QRS complex tachycardias: Clinical manifestations, diagnosis, and evaluation

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INTRODUCTION

Tachyarrhythmias, defined as abnormal heart rhythms with a ventricular rate of 100 or more beats per minute (bpm), can result from a variety of pathologies and are frequently symptomatic. Signs and symptoms related to the tachyarrhythmia most commonly include palpitations or chest discomfort, but may also include shock, hypotension, heart failure (HF), shortness of breath, and/or decreased level of consciousness. Symptoms can sometimes be more subtle and may include fatigue, lightheadedness, or exercise intolerance. Some patients are truly asymptomatic; this may be more common in nonparoxysmal (incessant) tachycardias.

This topic will provide a broad overview of the different causes of narrow QRS complex tachycardia and an approach to their evaluation and diagnosis. An overview of the acute management of tachyarrhythmias, along with detailed discussions of specific narrow complex tachycardias (eg, atrioventricular [AV] nodal reentrant tachycardia [AVNRT], AV reentrant (or reciprocating) tachycardia [AVRT], and atrial tachycardia [AT]) and a broad discussion of wide complex tachycardias, are presented separately. (See "Overview of the acute management of tachyarrhythmias" and "Atrioventricular nodal reentrant tachycardia" and "Atrioventricular reentrant tachycardia (AVRT) associated with an accessory pathway" and "Focal atrial tachycardia" and "Wide QRS complex tachycardias: Approach to the diagnosis" and "Wide QRS complex tachycardias: Approach to management".)

DEFINITIONS

Tachycardias, with a ventricular heart rate exceeding 100 bpm, are broadly categorized based upon the width of the QRS complex on the electrocardiogram (ECG) [1].

- A narrow QRS complex (<120 ms) reflects rapid activation of the ventricles via the normal His-Purkinje system, which in turn suggests that the arrhythmia originates above or within the His bundle (ie, a supraventricular tachycardia). The mechanism of tachycardia may be in the sinus node, the atria, the AV node, the His bundle, or some combination of these sites.

- A widened QRS (≥120 ms) occurs when ventricular activation is abnormally slow. The most common reason that a QRS is widened is because the arrhythmia originates below the His bundle in the bundle branches, Purkinje fibers, or ventricular myocardium (eg, ventricular tachycardia). Alternatively, a supraventricular arrhythmia can produce a widened QRS if there are either preexisting or rate-related abnormalities within the His-Purkinje system (eg, supraventricular tachycardia with aberrancy), or if conduction occurs over an accessory pathway. Thus, wide QRS complex tachycardias may be either supraventricular or ventricular in origin. (See "Wide QRS complex tachycardias: Approach to the diagnosis".)

TYPES OF NARROW QRS COMPLEX TACHYCARDIA

Differential diagnosis of narrow QRS complex tachycardias — Narrow QRS complex tachycardias can be divided into those that require only atrial tissue for their initiation and maintenance, and those that require the AV junction (table 1) [2].

The narrow QRS complex tachycardias include:

- Sinus tachycardia (ST) (see "Sinus tachycardia: Evaluation and management")
- AV nodal reentrant tachycardia (AVNRT) (see "Atrioventricular nodal reentrant tachycardia")
- AV reentrant (or reciprocating) tachycardia (AVRT) (see "Atrioventricular reentrant tachycardia (AVRT) associated with an accessory pathway")
- Atrial tachycardia (AT, also known as ectopic atrial tachycardia [EAT]) (see "Focal atrial tachycardia")
- Inappropriate ST (see "Sinus tachycardia: Evaluation and management", section on 'Inappropriate sinus tachycardia')
- Sinoatrial nodal reentrant tachycardia (SANRT) (see "Sinoatrial nodal reentrant tachycardia (SANRT)")

- Intraatrial reentrant tachycardia (IART) (see "Intraatrial reentrant tachycardia")
- Junctional ectopic tachycardia
- Nonparoxysmal junctional tachycardia
- Atrial fibrillation (AF) (see "Atrial fibrillation: Overview and management of new-onset atrial fibrillation")
- Atrial flutter (see "Overview of atrial flutter")
- Multifocal AT (MAT) (see "Multifocal atrial tachycardia")

Paroxysmal and incessant SVT — The term "paroxysmal supraventricular tachycardias (PSVT)" defines a subset of narrow QRS complex tachycardias that are intermittent, start and stop abruptly, and have a regular ventricular response. This term is usually reserved for tachycardias that are sustained (>30 seconds). The term "incessant SVT" is used to describe an SVT that is unlikely to spontaneously terminate. AF, atrial flutter, and MAT have an irregular ventricular response and distinct clinical characteristics; these rhythms are not considered PSVTs and are described elsewhere. (See "Atrial fibrillation: Overview and management of new-onset atrial fibrillation" and "Overview of atrial flutter" and "Multifocal atrial tachycardia".)

- **Features of paroxysmal SVTs** – The prevalence of PSVT is similar in males and females (although it is slightly higher in males) and increases progressively with age. Among a prospective cohort of more than half a million United Kingdom residents, the prevalence of PSVT was approximately 29 per 10,000 persons under age 55 and increased to between 53 (females) and 74 (males) per 10,000 persons ≥65 years of age [3].

  PSVTs are often due to reentry, although the mechanisms of reentry vary (figure 1) [1,4]. The major causes of symptomatic PSVTs that are sustained and require therapy are AVNRT, which accounts for approximately 60 percent of cases; AVRT, which accounts for approximately 30 percent of cases; and, in about 10 percent of cases, an AT or SANRT [5,6]. Junctional ectopic tachycardia and nonparoxysmal junctional tachycardia are rare in adults but can represent a larger portion of PSVTs in children. Brief runs of PSVT captured on Holter monitoring or other recording devices are usually due to AT.

  - AVNRT is characterized by two pathways within the AV node or perinodal atrial tissue. (See "Atrioventricular nodal reentrant tachycardia".)

  - AVRT is characterized by an extranodal accessory pathway connecting the atrium and ventricle. The ECG shows delta waves during sinus rhythm if there is antegrade conduction via the accessory pathway, leading to a diagnosis of Wolff-Parkinson-White syndrome (WPW). The delta waves are lost during an episode of orthodromic AVRT, the most common tachyarrhythmia in patients with WPW. Most patients with AVRT,

however, have a concealed accessory pathway and do not have ventricular preexcitation during sinus rhythm. (See "Atrioventricular reentrant tachycardia (AVRT) associated with an accessory pathway" and "Wolff-Parkinson-White syndrome: Anatomy, epidemiology, clinical manifestations, and diagnosis".)

- AT is the least common form of PSVT in patients with sustained PSVT. Most ATs in patients without prior surgery or ablation are due to a focal mechanism and can arise from anywhere in the atria, such as the crista terminalis, left atrial appendage, or near the tricuspid or mitral annulus, or can arise from a large vein such as the coronary sinus or a pulmonary vein. ATs in patients with atrial scar can be due to macroreentry. The reentrant circuit does not involve the AV node or an accessory pathway. AT can be paroxysmal or, at times, incessant. AT with 1:1 (or fixed 2:1, 3:1, etc) conduction into the ventricles is a regular tachycardia, but if there is variable conduction, the tachycardia can be irregular. (See "Sinoatrial nodal reentrant tachycardia (SANRT)" and "Intraatrial reentrant tachycardia".)

- Permanent junctional reciprocating tachycardia (PJRT) is a syndrome of nearly incessant, relatively slow PSVT in a patient who can, at times, present with a tachycardia-induced cardiomyopathy and HF. The SVT mechanism in most patients with PJRT is AVRT due to a slowly conducting concealed accessory pathway, and the term PJRT is often used interchangeably with incessant, slow AVRT. However, a patient with PJRT can also have nearly incessant atypical AVNRT or AT. (See "Atrioventricular reentrant tachycardia (AVRT) associated with an accessory pathway", section on 'Permanent junctional reciprocating tachycardia' and "Focal atrial tachycardia".)

PATHOGENESIS

Reentry is the most common cause of narrow QRS complex tachycardia. Increased automaticity and triggered activity occur less frequently [7].

Mechanisms of reentry — Reviewed briefly, reentry requires two distinct electrical conduction pathways or tissues with different electrophysiologic properties that are linked proximally and distally, forming an anatomic or functional circuit (figure 2) [1,4]. The reentrant circuit may become repetitively activated, producing a sustained reentrant tachycardia. The type of arrhythmia that ensues with narrow QRS tachycardias is determined by the characteristics and location of the reentrant circuit (figure 1).

The mechanisms of reentry are discussed in detail elsewhere. (See "Reentry and the development of cardiac arrhythmias".)

Automaticity and triggered activity — Other mechanisms of narrow QRS complex tachycardia include:

- Enhanced normal automaticity (as in sinus tachycardia)
- Abnormal automaticity (resulting in an ectopic atrial or junctional tachycardia)
- "Triggered activity," which may underlie some arrhythmias due to digitalis intoxication (such as atrial tachycardia with or without AV block or arrhythmias in the setting of acute ischemia or infarction)

These mechanisms are considered in detail in topics dealing with the specific arrhythmias. (See "Focal atrial tachycardia", section on 'Mechanisms and etiology' and "Cardiac arrhythmias due to digoxin toxicity".)

CLINICAL MANIFESTATIONS

The response to a rapid heart rate can be quite variable depending on how fast the heart is beating, resultant blood pressure and tissue perfusion, underlying comorbidities, and the sensitivity of the individual patient to the symptoms. Patients with a narrow QRS complex tachycardia can present with a variety of symptoms, including:

- Palpitations
- Syncope or presyncope
- Lightheadedness or dizziness
- Diaphoresis
- Chest pain
- Shortness of breath

Most commonly, patients with a narrow QRS complex tachycardia present with palpitations, the sensation of a rapid or irregular heart beat felt in the anterior chest or neck. Usually the symptoms are abrupt in onset, although this may vary depending on the specific arrhythmia. Palpitations may be associated with diaphoresis, lightheadedness, or dizziness.

Patients with a narrow QRS complex tachycardia may also report shortness of breath or chest discomfort. While shortness of breath or chest discomfort can occur in any patient, those with underlying cardiac comorbidities (eg, coronary heart disease, cardiomyopathy or valvular heart

disease with or without HF) are more likely to present in this fashion, particularly at higher heart rates (>150 bpm).

Syncope is a rare presentation for persons presenting with a narrow QRS complex tachycardia, since in most instances the heart rate is not so rapid as to impair ventricular function and cardiac output. However, a narrow QRS complex tachycardia with a very rapid ventricular rate (>250 bpm, as might be seen in persons with AF or atrial flutter and an accessory pathway) may result in diminished cardiac output and syncope. (See 'Atrial rate' below.)

The physical examination in a patient with narrow QRS complex tachycardia reveals a rapid pulse which may be regular or irregular depending on the underlying cardiac rhythm. Cardiac auscultation also reveals a rapid heartbeat. While other physical examination findings may be present in situations where the tachycardia has led to or exacerbated another condition (eg, hypotension following syncope, lung congestion in a patient with HF), there are no other specific physical examination findings which are universally seen in patients with a narrow complex tachycardia.

DIAGNOSIS

The diagnosis of a narrow QRS complex tachycardia is usually suspected in a patient with palpitations when a pulse greater than 100 bpm is present on physical examination. The differentiation between narrow and wide QRS complex tachycardia requires only a surface ECG, which shows a heart rate greater than 100 bpm along with narrow QRS complexes that are less than 120 ms in duration. A variety of arrhythmias result in the ECG appearance of a narrow QRS complex tachycardia. (See 'Types of narrow QRS complex tachycardia' above.)

Once a narrow QRS complex tachycardia has been identified, further scrutiny of the ECG is required to identify the specific arrhythmia in a particular patient, as diagnostic evaluation and therapy will differ depending on the underlying arrhythmia. (See 'Evaluation' below.)

Invasive electrophysiology testing is not required to broadly make the diagnosis of a narrow QRS complex tachycardia, but on rare occasions it is needed to diagnose (and potentially treat with catheter ablation) the specific arrhythmia. (See 'Electrophysiologic testing' below.)

EVALUATION

Evaluation of a patient with a narrow QRS complex tachycardia involves two primary components:

- Assessment of the patient for symptoms and signs of hemodynamic stability (or instability)
- Assessment of the patient's ECG for clues to the type of tachycardia present

Often these steps are performed nearly simultaneously in an acute care setting, as the provider can be assessing the patient at the same time that the ECG is being obtained.

Assessing the patient for hemodynamic stability — The most important clinical determination to make when a narrow QRS tachycardia is noted is whether the patient is experiencing signs and symptoms related to the rapid heart rate. These can include hypotension, shortness of breath, chest pain suggestive of coronary ischemia, shock, and/or decreased level of consciousness (algorithm 1 and algorithm 2).

Determining whether a patient's symptoms are related to the tachycardia depends upon several factors, including age and the presence of underlying cardiac disease. As an example, PSVT with a heart rate of 200 bpm may be tolerated by an otherwise healthy young adult with no or few symptoms (eg, palpitations). On the other hand, AF at a rate of 120 bpm may precipitate angina in an older adult patient with significant coronary heart disease.

- Hemodynamically unstable and not sinus rhythm – If a patient has clinically significant hemodynamic instability potentially due to a narrow QRS complex tachycardia (algorithm 1), an attempt should be made as quickly as possible to determine whether the rhythm is sinus tachycardia (ST). If the rhythm is not sinus tachycardia, or if there is any doubt that the rhythm is sinus tachycardia, **urgent conversion** to sinus rhythm is recommended. (See 'Similar to sinus rhythm' below and "Overview of the acute management of tachyarrhythmias" and "Basic principles and technique of external electrical cardioversion and defibrillation".)

- Hemodynamically unstable and sinus rhythm – If it is certain that the patient's rhythm is ST and clinically significant cardiac symptoms are present (algorithm 2), management should be focused on the underlying cardiac disorder and on treating any contributing cause of the rapid heart rate (such as coronary ischemia, respiratory or cardiac failure, cardiac tamponade, hypovolemia, anemia, fever, pain, or anxiety). In patients with ST and certain forms of heart disease, such as coronary disease or aortic stenosis, treatment may need to be directed at the heart rate itself. In such cases, cautious use of an intravenous beta blocker is appropriate (eg, metoprolol 5 mg intravenously every two minutes until the heart rate is controlled, to a total of 15 mg, followed by an oral regimen). (See "Sinus tachycardia: Evaluation and management" and "Acute myocardial infarction: Role of beta blocker therapy" and "Medical management of symptomatic aortic stenosis".)

- Hemodynamically stable – If the patient is not experiencing hemodynamic instability, a nonemergent approach to the diagnosis of the patient's rhythm can be undertaken (algorithm 2) [1]. A close examination of the 12-lead ECG should permit the correct identification of the arrhythmia in 80 percent of cases [8]. In some patients, increasing the ECG paper speed (from the standard 25 mm/sec to 50 mm/sec) can improve the likelihood of a correct diagnosis, or simple vagal blocking maneuvers may slow the ventricular rate to better elucidate the underlying rhythm [9]. (See 'ECG identification of atrial activity' below and 'ECG characterization of atrial activity' below.)

Assessing the ECG for regularity of the rhythm — Following the determination of hemodynamic stability, the next step in the assessment of a narrow QRS complex tachycardia is to determine if the rhythm appears to be regular or irregular (algorithm 2). While the majority of narrow QRS complex tachycardias are associated with a regular ventricular rate (with AF being the most notable exception), underlying conduction system disease and certain drug toxicities (ie, digoxin) can lead to irregular appearing rhythms due to intermittent block of conduction between the atria and the ventricles.

Examples of regular and irregular rhythms include:

- Regular – Sinus tachycardia, AV nodal reentrant tachycardia, AV reciprocating tachycardia, atrial flutter (usually), atrial tachycardia (AT).

- Irregular – AF, multifocal AT.

If the rhythm is irregular, the ECG should be scrutinized for discrete atrial activity and for any evidence of a pattern to the irregularity. If the rhythm is irregularly irregular (that is, no pattern can be detected), the arrhythmia will almost always be either:

- AF, where no discernible discrete P waves (waveform 1 and waveform 2) can be identified. (See "The electrocardiogram in atrial fibrillation".)

- Multifocal AT, in which discrete P waves of several morphologies (waveform 3) are present. (See "Multifocal atrial tachycardia".)

If the rhythm is irregular but has some periods of regularity, other arrhythmias (Mobitz I second-degree AV block) and/or drug toxicity (ie, digoxin toxicity) should be considered. (See "Second-degree atrioventricular block: Mobitz type I (Wenckebach block)" and "Cardiac arrhythmias due to digoxin toxicity".)

ECG identification of atrial activity — Characterization of atrial activity is essential to the diagnosis of narrow complex tachycardias (algorithm 2). However, the first step in this

process, the identification of P waves, can be difficult.

Methods to aid in identifying P waves — In ideal circumstances, P waves can be easily seen on the surface ECG. However, due to the rapid rate of the tachycardia, P waves are often superimposed on other parts of the surface ECG. In cases where P waves cannot be clearly identified, the Valsalva maneuver, carotid sinus massage, or the administration of intravenous adenosine may help to clarify the diagnosis [10]. These maneuvers may also terminate the arrhythmia in some cases (especially if the rhythm is AV nodal reentrant tachycardia AVNRT or AV reentrant tachycardia [AVRT]). (See 'Possible outcomes following vagal maneuvers or adenosine administration' below.)

Valsalva maneuver — The Valsalva maneuver induces a temporary slowing of SA nodal activity and AV nodal conduction by stimulating baroreceptors in the aorta, which triggers a reflex increase in vagus nerve activity and sympathetic withdrawal. Patients must be cooperative enough to comply with instructions and must be continually monitored on a cardiac monitor in order for this maneuver to be successful. The process of performing and interpreting the hemodynamic responses to the Valsalva maneuver is discussed in detail elsewhere. (See "Vagal maneuvers", section on 'Valsalva maneuver'.)

The Valsalva maneuver is generally safe and well-tolerated, though it does require some effort on the patient's part to perform. While the Valsalva maneuver may not be as effective as adenosine in slowing the AV nodal conduction rate, it can often be accomplished while preparing for an adenosine challenge, especially in patients not eligible for carotid sinus massage. Additionally, the Valsalva maneuver may terminate some narrow complex tachycardias, although data regarding the effectiveness of the maneuver are inconclusive. In a 2013 Cochrane Review of three randomized trials (316 patients) that compared the Valsalva maneuver with other vagal maneuvers, there was insufficient evidence to support or refute the effectiveness of the Valsalva maneuver [11]. However, given the general safety of the maneuver, its lack of expense, and its occasional effectiveness, we ask patients to perform the Valsalva maneuver prior to using other vagal maneuvers or medications.

Carotid sinus massage — Carotid sinus massage induces a temporary slowing of SA nodal activity and AV nodal conduction. External pressure on the carotid bulb stimulates baroreceptors in the carotid sinus, which triggers a reflex increase in vagus nerve activity and sympathetic withdrawal. The process of performing carotid sinus massage is discussed in detail elsewhere. (See "Vagal maneuvers", section on 'Carotid sinus massage'.)

Carotid sinus massage is generally safe and well tolerated, but potential complications include profound hypotension and bradycardia (including transient loss of consciousness), transient

ischemic attack or stroke, and arrhythmias. Due to these potential complications, carotid sinus massage should be performed with simultaneous ECG and blood pressure monitoring. (See "Vagal maneuvers", section on 'Complications'.)

Intravenous adenosine — Adenosine interacts with A1 receptors on the surface of cardiac cells, activating potassium channels and causing an increase in potassium conductance (IK). Adenosine also indirectly reduces calcium influx (ICa) into cells by antagonizing catecholamine-stimulated adenylate cyclase. The resulting effects include a slowing of the sinus rate and an increase in the AV nodal conduction delay, similar to the effects seen with the Valsalva maneuver and carotid sinus massage [1].

Adenosine is used for the intravenous management of paroxysmal narrow QRS complex tachycardias in which the AV node is involved. However, like the Valsalva maneuver and carotid sinus massage, adenosine can have both diagnostic and therapeutic effects, terminating some AV node dependent arrhythmias and producing transient AV nodal block that can clarify diagnoses such as atrial flutter or AT. Adenosine is cleared from the circulation extremely rapidly, with a half-life of less than five seconds, which reduces the likelihood of serious untoward effects [4].

Administration and side effects — For intravenous adenosine administration (algorithm 3), the patient should be supine and should have ECG and blood pressure monitoring. It is valuable to perform a 12-lead rhythm strip during administration of adenosine, as there are often clues at the time of termination as to the mechanism of the PSVT. The drug is administered by rapid intravenous injection over one to two seconds at a peripheral site, followed by a normal saline flush. The rapid administration of both the drug and the saline flush is most easily accomplished through a three-way stopcock. Adenosine is typically given in increasing doses until the rhythm terminates or AV block occurs. The initial dose is 6 mg. If the initial dose is not effective, a subsequent dose of 12 mg can be given. If there is no effect from the second dose, we give another 12 mg dose or an 18 mg dose. If a central intravenous access site is used, the initial dose should not exceed 3 mg [4,10].

The effects of adenosine are blocked by methylxanthines such as theophylline and caffeine and potentiated by dipyridamole. Heart transplant recipients exhibit a supersensitive response to adenosine [12].

The most common side effects of adenosine are facial flushing (18 percent), shortness of breath, palpitations, chest pain, and lightheadedness. Patients should be warned regarding these symptoms before adenosine administration. Transient asystole is a rare complication.

In patients with an accessory pathway capable of antegrade (atrium to ventricle, antidromic reentrant tachycardia) conduction, AF can degenerate into ventricular fibrillation with adenosine administration. As a result, caution should be used when giving adenosine if an accessory pathway with antegrade conduction is a possible mechanism, and emergency resuscitation equipment should be available. (See "Wolff-Parkinson-White syndrome: Anatomy, epidemiology, clinical manifestations, and diagnosis", section on 'Atrial fibrillation'.)

Possible outcomes following vagal maneuvers or adenosine administration — Following the Valsalva maneuver, carotid sinus massage, or the intravenous administration of adenosine to patients with a narrow QRS complex tachycardia, one of four possible results may be seen:

- The slowing of SA nodal activity can cause a temporary decrease in the atrial rate (in patients with sinus tachycardia).
- The slowing of AV nodal conduction can lead to AV nodal block, which may "unmask" atrial electrical activity (ie, reveal P waves or flutter waves) by decreasing the number of QRS complexes that obscure the electrical baseline (waveform 4).
- In some cases, no response is obtained. Usually the lack of any response would suggest inadequate performance of the vagal maneuver or inadequate dosing of the adenosine (either insufficient dose or administration which was too slow such that the adenosine was metabolized prior to arrival in the heart).
- The transient slowing of AV nodal conduction can terminate some narrow QRS complex arrhythmias by interrupting a reentry circuit that requires AV nodal conduction (especially AVNRT and AVRT). A continuous ECG tracing should be recorded during these maneuvers, because the response may aid in the diagnosis [7]:
  - Termination of the tachycardia with a P wave after the last QRS complex is most common in AVRT or AVNRT and is rarely seen with AT.
  - Termination of the tachycardia with a QRS complex can be seen with AVRT, AVNRT, or AT.
  - If the tachycardia continues despite successful induction of at least some degree of AV nodal blockade, the rhythm is almost certainly AT or atrial flutter; AVRT is excluded, and AVNRT is very unlikely.

If the use of carotid sinus massage and/or adenosine does not terminate the tachycardia or permit a diagnosis for the tachycardia that was terminated, further evaluation begins

with characterization of the atrial activity (P waves) on the ECG. (See 'ECG characterization of atrial activity' below.)

ECG characterization of atrial activity — Identification and characterization of atrial activity (ie, the P wave) is central to the diagnosis of narrow QRS complex tachycardias (algorithm 2). The evaluation of atrial activity includes assessment of four features:

- The atrial rate
- The P wave morphology (ie, identical to normal sinus rhythm, retrograde, or abnormal) (see 'P wave morphology' below)
- The position of the P wave in relation to the preceding and following QRS complexes (ie, the RP relationship) [8] (see 'RP relationship' below)
- The relationship between atrial and ventricular rates (1:1 or otherwise)

It is uncommon that any one of these features can identify the mechanism of an arrhythmia. In combination, however, these features (particularly the P wave morphology and RP relationship) often provide a probable diagnosis.

Atrial rate — There is substantial overlap between the atrial rates of most narrow QRS complex tachycardias. Thus, this feature in isolation is rarely diagnostic. The exception is with very fast atrial rates (eg >250 bpm), which are generally associated with one of two diagnoses: atrial flutter or AT.

Atrial flutter has several unique features that often make it easily distinguishable from other narrow QRS complex tachycardias, including the following:

- The atrial rate is typically 250 to 350 bpm. Classically, the atrial rate is close to 300 bpm with 2:1 AV conduction, resulting in a ventricular rate of 150 bpm.

- The P waves typically exhibit a classic "sawtooth" pattern without an isoelectric baseline; these complexes are referred to as flutter waves or "F" waves (waveform 5). If this pattern is not evident initially, vagal stimulation and intravenous adenosine can reduce the ventricular rate and make the "F" waves more evident (waveform 4).

Atypical reentrant circuits (often seen post-operatively or post-ablation) and the influence of antiarrhythmic drugs can alter these classic findings, resulting in atypical flutter waves (waveform 6). The electrocardiographic and electrophysiologic features of atrial flutter are discussed in detail separately. (See "Electrocardiographic and electrophysiologic features of atrial flutter".)

P wave morphology — The P wave morphology provides insight into the site of origin of atrial activity, and therefore the mechanism of the tachycardia. The P wave should be evaluated in as many leads as possible, ideally all 12 leads of a surface ECG, although in some situations a single-lead ECG strip may be all that is available for review.

The P wave morphology can be classified into one of three general categories:

- Similar to sinus rhythm
- Retrograde
- Abnormal

Similar to sinus rhythm — The most reliable way to determine if the P wave in a narrow complex tachycardia is consistent with an origin from the sinus node is to compare the P wave during the tachycardia to that from the same patient during sinus rhythm. Although sinus P wave morphology can vary slightly with changes in heart rates, the P wave during the tachycardia should be nearly identical to that seen on the baseline ECG in all 12 leads.

If a baseline ECG is not available, characteristics of the P wave can still suggest origin from the region of the sinus node. A sinus P wave is usually upright in leads I, aVL, and the inferior leads II, III, and aVF, and negative in lead aVR. These features indicate that atrial activity is proceeding in a right-to-left and superior-to-inferior direction. (See "ECG tutorial: Basic principles of ECG analysis", section on 'P wave'.)

At atrial rates greater than approximately 140 bpm or in the presence of first degree AV block, the P wave tends to merge into the preceding T or U wave, making P wave identification difficult (waveform 7). In such cases, slowing of the ventricular response using the Valsalva maneuver, carotid sinus massage, or adenosine may allow for better analysis of the P waves.

If P wave morphology is consistent with origination from the sinus node, the differential diagnosis of the tachycardia includes:

- ST(see "Sinus tachycardia: Evaluation and management", section on 'Definition and ECG features')
- Inappropriate ST (IST) (see "Sinus tachycardia: Evaluation and management", section on 'Inappropriate sinus tachycardia')
- Sinoatrial nodal reentrant tachycardia (SANRT) (waveform 8) (see "Sinoatrial nodal reentrant tachycardia (SANRT)")
- AT, usually originating near the sinus node (see "Focal atrial tachycardia", section on 'Electrocardiographic features')

P wave morphology alone is not sufficient to distinguish these arrhythmias from one another. In persons with a P wave morphology that is consistent with origination from the sinus node, additional ECG features that assist in the diagnosis include:

- Atrial rate – In ST and IST, the rate typically ranges from 100 to 180 bpm. Faster atrial rates suggest an AT.
- Onset/offset pattern – ST and IST have smooth, gradual changes in rate. Both SANRT and AT have an abrupt onset and offset, although the rate in AT can vary significantly with autonomic tone.
- Relationship between atrial and ventricular activity – In ST and IST, there is usually a 1:1 relationship between atrial and ventricular activity.
- The response to vagal maneuvers or adenosine – With ST or IST, there is a gradual slowing of the atrial and ventricular rates, followed by a gradual resumption of the previous rate. In contrast, SNRT can terminate abruptly with these maneuvers.

Abnormal P waves — Any P wave that does not have the characteristics of a sinus P wave is considered an abnormal P wave. Abnormal P waves that are due to retrograde conduction from the ventricle to the atrium are a specific subset of abnormal P waves. (See 'Retrograde P waves' below.)

Abnormal P waves that are not retrograde are most consistent with AT, although some patients with AVRT have abnormal P waves.

Retrograde P waves — P waves that are due to retrograde conduction from the ventricle to the atrium are a specific subset of abnormal P waves. These P waves have a characteristic morphology and suggest certain diagnoses, specifically:

- AVNRT
- AVRT
- Junctional ectopic tachycardia
- Nonparoxysmal junctional tachycardia

The most common retrograde P wave morphology is associated with atrial activation that originates from the AV node and proceeds in an inferior-to-superior direction. Thus, the defining trait of a retrograde P wave is that it is negative in the inferior leads II, III, and aVF. Due to the central location of the AV node, atrial activation proceeds simultaneously leftward and rightward, so the P wave morphology in leads I, aVL, and aVR varies among patients, but it is

often relatively narrow. In addition, because the AV node is posteriorly located, the activation is usually posterior-to-anterior, producing a narrow but positive P wave in lead V1.

Retrograde P waves due to conduction via an accessory pathway can have a variety of morphologies, depending upon the site of the pathway. However, they are still usually negative in the inferior leads. (See "Wolff-Parkinson-White syndrome: Anatomy, epidemiology, clinical manifestations, and diagnosis".)

Because retrograde P waves are often associated with a short RP interval, they can blend with the terminal portion of the QRS. If a baseline ECG during sinus rhythm is available for comparison, a pseudo S wave in the inferior leads or a pseudo R' in V1 that is not present during sinus rhythm is often a retrograde P wave (figure 3) [7,8,13]. (See 'RP relationship' below.)

RP relationship — In patients with a retrograde P wave, the temporal relationship between the P wave and the R wave divides narrow complex tachycardias into two categories: short RP and long RP tachycardias. The RP interval is defined as the interval from the onset of the QRS to the onset of the P wave. Short RP tachycardias tend to be paroxysmal, while long RP tachycardias can be paroxysmal or incessant.

Short RP tachycardias — If the RP interval is less than one-half of the RR interval, the tachycardia is considered a short RP tachycardia. The differential diagnosis of a short RP tachycardia is generated by considering the P wave morphology.

- Abnormal P wave – The combination of abnormal P waves and a short RP interval is most often seen in the setting of an AT with AV nodal conduction delay. (See 'Abnormal P waves' above.)

- Retrograde P wave – The combination of retrograde P waves and a short RP interval is typical of the "common" form of AVNRT and of AVRT utilizing an accessory pathway. (See 'Retrograde P waves' above.)

In the "common" form of AVNRT (which accounts for 90 percent of AVNRT) [14], reentry occurs in the AV node and perinodal tissues. Antegrade conduction occurs down the slow pathway and retrograde conduction up the fast pathway (figure 4 and figure 5). This slow-fast pattern gives rise to retrograde P waves that may be inapparent if obscured by the QRS complex (figure 3). (See "Atrioventricular nodal reentrant tachycardia".)

AVRT utilizing an accessory pathway can be either orthodromic or antidromic. Orthodromic AVRT is more common, and in this form of the arrhythmia, antegrade conduction occurs

through the AV node, producing a narrow QRS complex, and retrograde conduction to the atrium occurs over an AV bypass tract (figure 6). (See "Atrioventricular reentrant tachycardia (AVRT) associated with an accessory pathway".)

In contrast, during antidromic AVRT, antegrade conduction occurs through the AV bypass tract and retrograde conduction occurs through the AV node or a second accessory pathway. This pattern of activation results in a wide QRS complex (thus, antidromic AVRT is not a narrow QRS complex tachycardia). (See "Wide QRS complex tachycardias: Approach to the diagnosis".)

Long RP tachycardias — If the RP interval is more than one-half of the RR interval, the tachycardia is considered a long RP tachycardia. As with short RP tachycardias, the differential diagnosis is generated by combining the PR relationship with the P wave morphology.

- Retrograde P waves – The combination of retrograde P waves and a long RP interval is usually caused by either "atypical" AVNRT or by AVRT with a slowly conducting accessory pathway (also known as permanent junctional reciprocating tachycardia [PJRT]); this combination can also be seen in AT with a focus that is close to the AV node [7]. P waves are inverted in the inferior leads as the atria are depolarized from bottom to top. (See 'Retrograde P waves' above.)

- Abnormal P wave morphology – The combination of abnormal P wave morphology and a long RP interval usually suggests some form of AT. However, this pattern can also occur in the atypical or uncommon form of AVNRT and in AVRT with a slowly conducting accessory pathway [7]. (See 'Abnormal P waves' above.)

The atypical form of AVNRT, which accounts for 10 percent of AVNRT [14], is characterized by antegrade conduction down a fast pathway and retrograde conduction through a slow pathway. As a result, the P wave occurs very late in the cardiac cycle (positioning it near to the next QRS complex) (figure 7). (See "Atrioventricular nodal reentrant tachycardia".)

In AVRT with a slowly conducting accessory pathway, antegrade conduction probably occurs through the AV node, and retrograde conduction through a slowly conducting accessory pathway [1,13]. Because of slow conduction through the retrograde limb of the circuit, the retrograde P wave occurs late in the cardiac cycle. (See "Atrioventricular reentrant tachycardia (AVRT) associated with an accessory pathway".)

Undetectable P waves — If atrial activity is not evident despite the use of carotid sinus massage and/or adenosine to "unmask" atrial activity, the most common rhythm is AVNRT [7]. Other possible rhythms are AF, AVRT, and junctional tachycardia.

Although AF is typically an irregularly irregular rhythm, when the ventricular rate is very rapid, it may appear to be more regular. Vagal maneuvers produce a transient decrease in the ventricular rate with AF, which may make the irregularity more evident.

Junctional tachycardia is an arrhythmia arising from a discrete focus within the AV node or His bundle. In children, junctional tachycardia, also known as junctional ectopic tachycardia (JET), is usually associated with significant underlying heart disease. In adults, this rhythm, generally called nonparoxysmal junctional tachycardia (NPJT), is seen with acute myocardial infarction, digitalis toxicity, and myocarditis. (See "Supraventricular arrhythmias after myocardial infarction".)

Atrial activity during junctional tachycardia is variable. Retrograde atrial activation may occur, with a P wave that either follows each QRS complex or is concealed in the QRS complex. If retrograde conduction does not occur, independent atrial activity may be seen, with complete AV dissociation that must be distinguished from AV dissociation due to complete heart block (in complete heart block, the atrial rate exceeds the ventricular rate).

Electrophysiologic testing — In some cases, narrow QRS complex tachycardias cannot be discriminated from each other using noninvasive testing. An electrophysiologic study (EPS) can provide a definitive diagnosis but is only used when a definitive diagnosis will influence therapy. [13]. For some arrhythmias, EPS with ablation can also be curative. For these reasons, invasive EP testing to define the mechanism of SVT is rarely carried out as a standalone diagnostic procedure, but rather as a "prelude" to ablation.

Diagnostic EP may be indicated to determine the mechanism of the arrhythmia in the following patients with a narrow QRS complex tachycardia:

- Those with severe symptoms such as syncope or presyncope in association with the arrhythmia, particularly if the resting ECG manifests the pattern of preexcitation with a delta wave (waveform 9) or a short PR interval without a delta wave.
- Those with underlying organic heart disease who develop angina or acute HF during their arrhythmia, even if the rate is relatively slow.

EPS and catheter ablation are discussed in greater detail elsewhere. (See "Invasive diagnostic cardiac electrophysiology studies" and "Overview of catheter ablation of cardiac arrhythmias", section on 'Introduction'.)

Other cardiac testing — In addition to close scrutiny of the available ECG data, patients with narrow QRS complex tachycardia should generally have a baseline echocardiogram to assess

for any evidence of significant underlying structural heart disease [15]. In some patients, additional cardiac testing may be required to identify or characterize an arrhythmia; its use depends upon the particular arrhythmia and symptoms but may include one or more of the following tests:

- Ambulatory ECG monitoring (for patients without a definitive ECG-based diagnosis of their arrhythmia).
- Exercise stress testing (for patients with exercise-associated arrhythmias).
- Stress testing (exercise or pharmacologic) for underlying myocardial ischemia (for those with symptoms of angina and risk factors for coronary artery disease).

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Atrial fibrillation" and "Society guideline links: Arrhythmias in adults" and "Society guideline links: Supraventricular arrhythmias".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient education: Supraventricular tachycardia (SVT) (The Basics)")

SUMMARY AND RECOMMENDATIONS

- **Definitions** – Tachycardias, defined as abnormal heart rhythms with a ventricular rate of 100 or more beats per minute (bpm), can present with either a narrow or wide QRS complex. A narrow QRS complex (<120 ms) reflects rapid activation of the ventricles via the normal His-Purkinje system, which in turn suggests that the arrhythmia originates above or within the His bundle (ie, a supraventricular tachycardia). The site of origin may be in the sinus node, the atria, the atrioventricular (AV) node, the His bundle, or some combination of these sites. (See 'Introduction' above and 'Definitions' above.)

- **Clinical manifestations** – Most commonly, patients with a narrow QRS complex tachycardia present with palpitations, usually abrupt in onset. Other presenting symptoms may include syncope or presyncope, lightheadedness, dizziness, diaphoresis, chest pain, or shortness of breath. (See 'Clinical manifestations' above.)

- **Diagnosis** – The diagnosis of a narrow QRS complex tachycardia requires only a surface ECG that shows a heart rate greater than 100 bpm along with narrow QRS complexes that are less than 120 ms in duration. (See 'Diagnosis' above.)

- **Types of narrow QRS complex tachycardia** – The differential diagnosis of narrow QRS complex tachycardias is broad (table 1), including atrial fibrillation (AF), atrial flutter, sinus tachycardia (ST), and a variety of paroxysmal supraventricular tachycardias. (See 'Types of narrow QRS complex tachycardia' above.)

- **Evaluation** – Evaluation of a patient with a narrow QRS complex tachycardia involves two primary components: assessment of the patient for symptoms and signs of hemodynamic stability (or instability), and assessment of the patient's ECG for clues to the type of tachycardia present. (See 'Evaluation' above.)

  - **Unstable patients** – If a patient has clinically significant hemodynamic instability and the rhythm is not ST, or if there is any doubt that the rhythm is sinus tachycardia, **urgent conversion** to sinus rhythm is recommended (algorithm 1).

    If it is certain that the patient's rhythm is ST, and clinically significant cardiac symptoms are present, management should be focused on the underlying cardiac disorder and on treating any contributing cause of the rapid heart rate (such as myocardial ischemia due to coronary heart disease, respiratory or cardiac failure, hypovolemia, anemia, fever, pain, or anxiety).

  - **Stable patients** – If the patient is not experiencing hemodynamic instability, a nonemergency approach to the diagnosis of the patient's rhythm can be undertaken (algorithm 2).

- **Approach to identifying the type of SVT** – Following the determination of hemodynamic stability, the next steps in the assessment of a narrow QRS complex tachycardia include:

  - Determine if the rhythm appears to be regular or irregular. (See 'Assessing the ECG for regularity of the rhythm' above.)

  - Determine the atrial activity by identifying and characterizing the atrial rate, P-wave morphology, RP relationship, and AV relationship. (See 'Atrial rate' above and 'P wave morphology' above and 'RP relationship' above.)

- **Role of electrophysiologic testing** – In some cases, narrow QRS complex tachycardias cannot be discriminated from each other using noninvasive testing. An electrophysiologic study (EPS) can provide a definitive diagnosis but is only used when a definitive diagnosis will influence therapy. (See 'Electrophysiologic testing' above.)

- **Other cardiac testing** – In some patients, additional cardiac testing may be required to identify or characterize an arrhythmia; its use depends upon the particular arrhythmia and symptoms. (See 'Other cardiac testing' above.)

REFERENCES

1. Ganz LI, Friedman PL. Supraventricular tachycardia. N Engl J Med 1995; 332:162.
2. Katritsis DG, Josephson ME. Differential diagnosis of regular, narrow-QRS tachycardias. Heart Rhythm 2015; 12:1667.
3. Khurshid S, Choi SH, Weng LC, et al. Frequency of Cardiac Rhythm Abnormalities in a Half Million Adults. Circ Arrhythm Electrophysiol 2018; 11:e006273.
4. Ferguson JD, DiMarco JP. Contemporary management of paroxysmal supraventricular tachycardia. Circulation 2003; 107:1096.
5. Trohman RG. Supraventricular tachycardia: implications for the intensivist. Crit Care Med 2000; 28:N129.
6. Knight BP, Ebinger M, Oral H, et al. Diagnostic value of tachycardia features and pacing maneuvers during paroxysmal supraventricular tachycardia. J Am Coll Cardiol 2000; 36:574.
7. Blomström-Lundqvist C, Scheinman MM, Aliot EM, et al. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias--executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice

Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Supraventricular Arrhythmias). Circulation 2003; 108:1871.

8. Kalbfleisch SJ, el-Atassi R, Calkins H, et al. Differentiation of paroxysmal narrow QRS complex tachycardias using the 12-lead electrocardiogram. J Am Coll Cardiol 1993; 21:85.
9. Accardi AJ, Miller R, Holmes JF. Enhanced diagnosis of narrow complex tachycardias with increased electrocardiograph speed. J Emerg Med 2002; 22:123.
10. Link MS. Clinical practice. Evaluation and initial treatment of supraventricular tachycardia. N Engl J Med 2012; 367:1438.
11. Smith GD, Dyson K, Taylor D, et al. Effectiveness of the Valsalva Manoeuvre for reversion of supraventricular tachycardia. Cochrane Database Syst Rev 2013; :CD009502.
12. Ellenbogen KA, Thames MD, DiMarco JP, et al. Electrophysiological effects of adenosine in the transplanted human heart. Evidence of supersensitivity. Circulation 1990; 81:821.
13. Chauhan VS, Krahn AD, Klein GJ, et al. Supraventricular tachycardia. Med Clin North Am 2001; 85:193.
14. Akhtar M, Jazayeri MR, Sra J, et al. Atrioventricular nodal reentry. Clinical, electrophysiological, and therapeutic considerations. Circulation 1993; 88:282.
15. Brugada J, Katritsis DG, Arbelo E, et al. 2019 ESC Guidelines for the management of patients with supraventricular tachycardiaThe Task Force for the management of patients with supraventricular tachycardia of the European Society of Cardiology (ESC). Eur Heart J 2020; 41:655.

Topic 943 Version 36.0

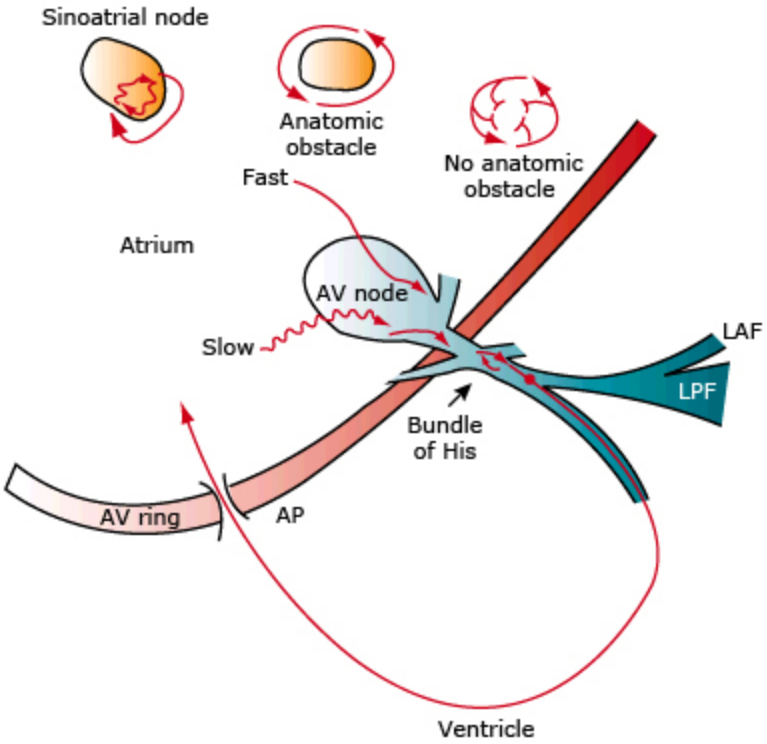
GRAPHICS

Classification of narrow QRS complex tachycardias by structures required for initiation and maintenance

Atrial tissue only	AV junction
Sinus tachycardia	AV nodal reentrant tachycardia (AVNRT)
Inappropriate sinus tachycardia	Atrioventricular reentrant tachycardia (AVRT)
Sinoatrial nodal reentrant tachycardia (SANRT)	Junctional tachycardia
Intraatrial reentrant tachycardia (IART)	Junctional ectopic tachycardia in children
Atrial tachycardia	Nonparoxysmal junctional tachycardia in adults
Multifocal atrial tachycardia	
Atrial fibrillation	
Atrial flutter	

Graphic 58019 Version 2.0

Sites of reentry in supraventricular tachyarrhythmias

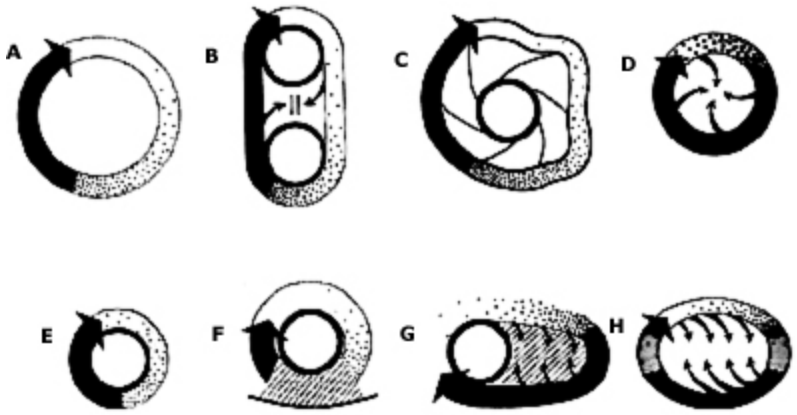


Reentry may occur around a fixed anatomic obstacle or may be functional, developing in the absence of an anatomic obstacle and resulting from the intrinsic heterogeneity of electrophysiologic properties of the myocardial tissue. Reentrant circuits leading to a supraventricular tachyarrhythmia may develop in various parts of the heart: within and around the sinoatrial node (sinus node reentry); within the atrial myocardium (atrial tachycardia, atrial flutter, or atrial fibrillation); within the atrioventricular (AV) node due to the presence of a slow and fast pathway (atrioventricular nodal reentrant tachycardia); or involving the AV node and an accessory pathway (AP) (atrioventricular reentrant tachycardia).

LAF: left anterior fascicle; LPF: left posterior fascicle.

Graphic 82249 Version 4.0

Mechanisms of reentry in cardiac arrhythmias



Schematic representation of possible reentrant circuits. The thick black arrow represents the circulating impulse; thin black lines represent advancing wavefronts in completely refractory tissue; speckled areas are partially refractory tissue; white areas are fully excitable tissue.

A is the original model of circus movement around a fixed obstacle. There is a fully excitable gap, and the length and location of the circuit are fixed.

B represents circus movement around 2 fixed anatomic obstacles. A fully excitable gap is present.

C represents rapidly conducting bundles forming closed loops that serve as preferential circuits through which the impulse may travel.

D is the leading circle type of reentry which does not require an anatomic obstacle. Instead, the impulse propagates around a functionally refractory core and among neighboring fibers that have different electrophysiologic properties. Since the refractoriness of the core is variable, the circuit size changes but will be the smallest possible circuit that can continue to propagate an impulse. Functional circuits tend to be small, rapid, and unstable.

E represents reentry around a fixed anatomic obstacle, but a fully excitable gap is absent.

F demonstrates an area of slowed conduction (hatched lines) between anatomic boundaries, while in

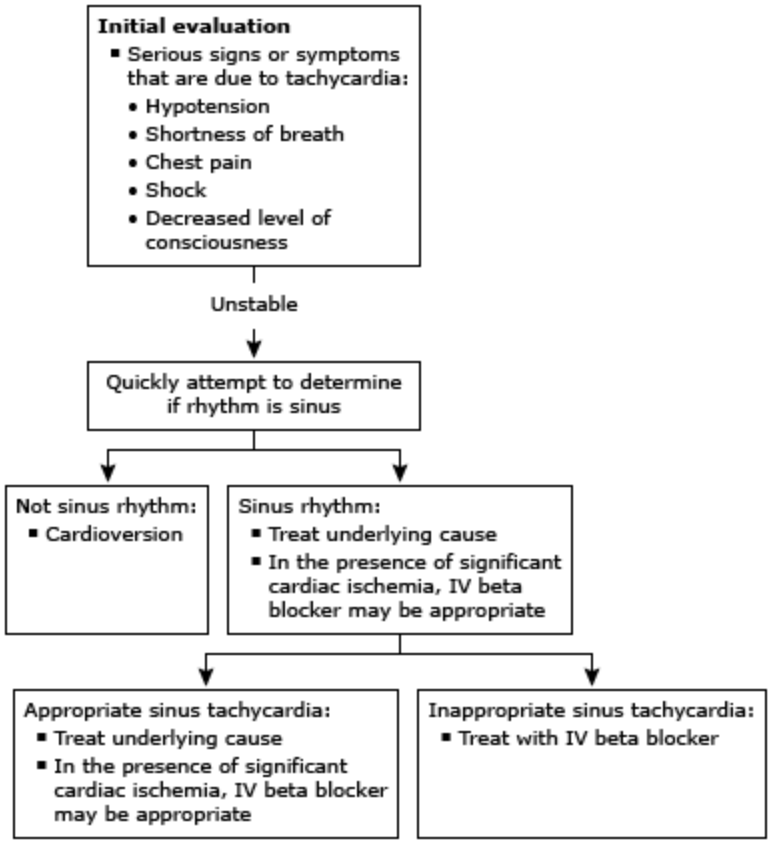
G all areas of slowed conduction neighbor an anatomic obstacle.

H represents anisotropic reentry. There are differences in the conduction of a single impulse in various fibers as a result of differences in their orientation.

Courtesy of Philip J Podrid, MD, FACC.

Graphic 52134 Version 5.0

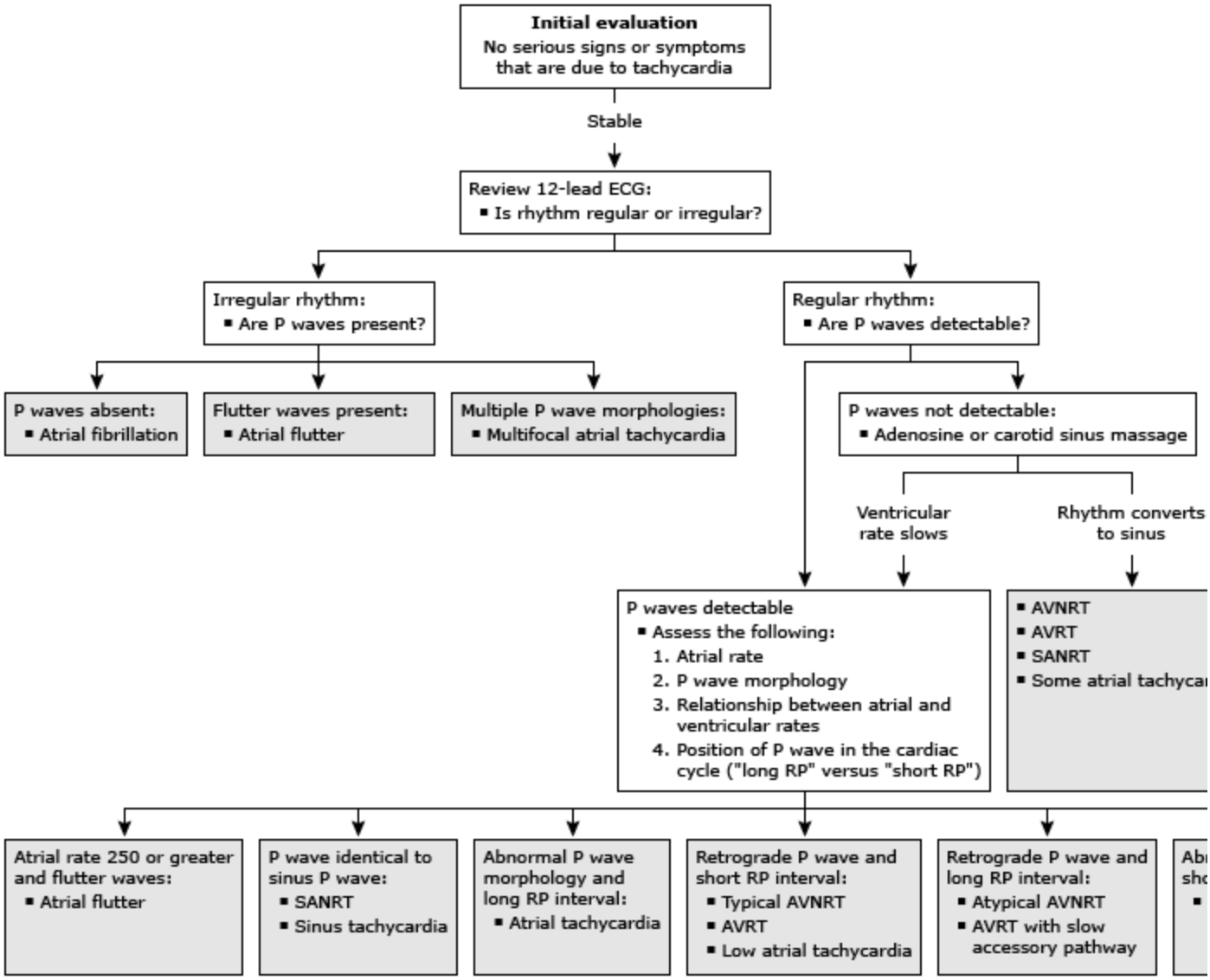
Algorithm for the evaluation of narrow QRS complex tachycardias in unstable patients



IV: intravenous.

Graphic 109811 Version 2.0

Algorithm for the evaluation of narrow QRS complex tachycardias in stable patients



ECG: electrocardiogram; AVNRT: atrioventricular nodal reentrant tachycardia; AVRT: atrioventricular reentrant tachycardia (due to an accessory pathway); SANRT: sinoatrial nodal reentrant tachycardia.

Graphic 76920 Version 4.0

Single-lead electrocardiogram (ECG) showing atrial fibrillation

Lead V1 showing coarse AF with moderate ventricular response. The two characteristic findings in AF are present: the very rapid atrial fibrillatory waves (f waves), which are variable in appearance; and the irregularly irregular ventricular response as the R-R interval between beats is unpredictable. Coarse AF may appear similar to atrial flutter. However, the variable height and duration of the f waves differentiate them from atrial flutter (F) waves, which are identical in appearance and occur at a constant rate of about 250 to 350 beats/min.

AF: atrial fibrillation.

Courtesy of Ary Goldberger, MD.

Graphic 73958 Version 6.0

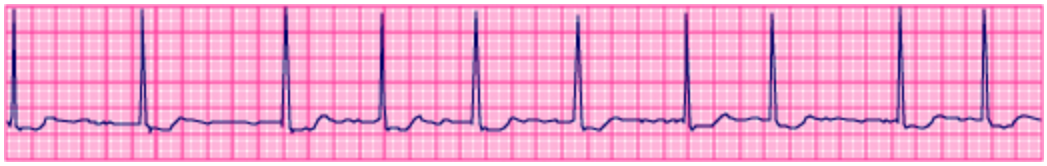
Normal rhythm strip

Normal rhythm strip in lead II. The PR interval is 0.15 sec and the QRS duration is 0.08 sec. Both the P and T waves are upright.

Courtesy of Morton F Arnsdorf, MD.

Graphic 59022 Version 3.0

Single-lead electrocardiogram (ECG) showing atrial fibrillation with minimally apparent atrial activity



F waves are not apparent in this lead, as the only finding suggestive of AF is the irregularly irrregular ventricular response.

AF: atrial fibrillation.

Courtesy of Morton Arnsdorf, MD.

Graphic 53988 Version 4.0

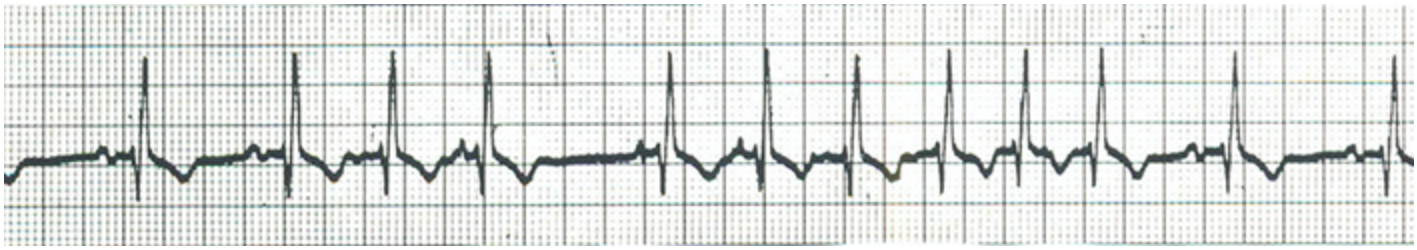
Normal rhythm strip

Normal rhythm strip in lead II. The PR interval is 0.15 sec and the QRS duration is 0.08 sec. Both the P and T waves are upright.

Courtesy of Morton F Arnsdorf, MD.

Graphic 59022 Version 3.0

Electrocardiogram single-lead multifocal atrial tachycardia



Clinical features:

- Elderly patients
- Decompensation of pulmonary disease
- Postoperative
- Arrhythmia usually does not cause severe hemodynamic compromise
- High mortality

ECG features:

- P waves have ≥3 forms
- Atrial rate is usually 100 to 200 bpm
- Atrial rate is irregular
- PR interval varies
- Isoelectric baseline between P waves
- May progress to atrial fibrillation

ECG: electrocardiogram; bpm: beats per minute.

Courtesy of Alfred Buxton, MD.

Graphic 127222 Version 1.0

Approach to adenosine dosing in stable adults with suspected supraventricular tachyarrhythmia

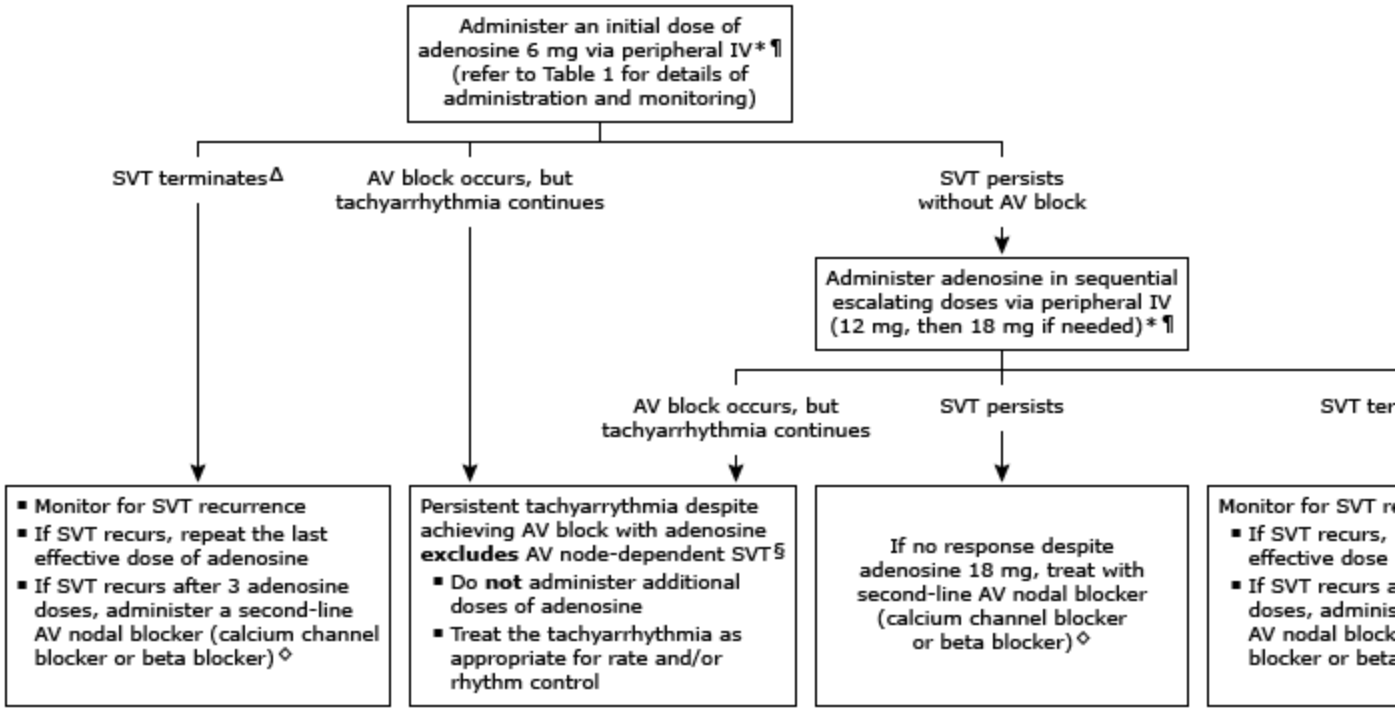


Table 1

Best practices for adenosine administration:

- The patient should be supine during adenosine administration and subsequent monitoring.
- Monitor blood pressure and record a 12-lead ECG during and for 1 to 2 minutes after administration to assess the response to adenosine. If a 12-lead ECG is impractical, record telemetry.
- Administer adenosine in the most proximal available peripheral IV site (eg, antecubutal vein); administration via a hand IV site is generally avoided. If central venous administration of adenosine is performed, lower doses are used than for peripheral administration.¶
- Administer adenosine via a 3-way stopcock, with 1 port leading to the patient, 1 port for the adenosine dose, and 1 port for normal saline flush (10 to 20 mL). Both the adenosine and normal saline syringes should be attached before adenosine is given.
- The IV adenosine dose is pushed over 1 to 2 seconds, immediately followed by turning the stopcock for normal saline flush over 1 to 2 seconds.

This figure summarizes the initial dosing, administration, and need for repeat dosing of adenosine in a hemodynamically stable adult with suspected AV node-dependent SVT. Adenosine can be used as a diagnostic aid to identify the underlying rhythm or as a therapy to terminate SVTs that rely on AV node conduction. AV node-dependent SVTs include AV nodal reentrant tachycardia (AVNRT) and AV reentrant (or reciprocating) tachycardia (AVRT). The management of suspected AV node-dependent SVT in hemodynamically stable adults commonly starts with a trial of 1 or more vagal maneuvers prior to proceeding to adenosine therapy. If the patient has hemodynamically unstable SVT, cardioversion is indicated. Refer to UpToDate content for details on management of SVTs.

AV: atrioventricular; SVT: supraventricular tachycardia; IV: intravenous; ACC: American College of Cardiology; AHA: American Heart Association; HRS: Heart Rhythm Society; ECG: electrocardiogram.

* Lower adenosine doses are indicated in certain clinical settings. If central venous administration is performed, the initial adenosine dose is 3 mg; if needed (SVT persists and there is no AV block), the dose may be increased to 6 mg and then 9 mg for subsequent doses. For heart transplant recipients, the initial adenosine dose is 1 mg; if needed (SVT persists and there is no AV block), the dose may be increased to 2 mg and then 3 mg for subsequent doses.

¶ An 18 mg dose is more likely to be required to produce AV block in patients with a body weight >70 kg, particularly in those weighing >110 kg.[1] When a third dose is indicated, an alternative approach is to administer 12 mg (instead of 18 mg) as described in the 2015 ACC/AHA/HRS Guideline for the Management of Adult Patients with Supraventricular Tachycardia.[2]

Δ SVT termination in response to adenosine includes SVT termination closely followed by SVT recurrence.

◇ Refer to UpToDate content on treatment of SVT for dosing of second-line AV nodal blockers. If the SVT persists despite second-line AV nodal blocker therapy, electrical cardioversion is performed, as discussed in UpToDate content on treatment of SVT.

§ SVTs that are **not** AV node dependent include atrial flutter, atrial fibrillation, and atrial tachycardia. When AV nodal block is induced by adenosine, the ECG may reveal atrial activity diagnostic of these rhythms.

References:

1. *Prabhu S, Mackin V, McLellan AJA, et al. Determining the optimal dose of adenosine for unmasking dormant pulmonary vein conduction following atrial fibrillation ablation: Electrophysiological and hemodynamic assessment. DORMANT-AF study. J Cardiovasc Electrophysiol 2017; 28:13.*
2. *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. Circulation 2016; 133:e506.*

Graphic 141417 Version 3.0

Atrial flutter at a rate of 250 beats/minute with 2:1 AV conduction in the presence of left bundle branch block

Although every other flutter wave can be seen at the end of the T wave in the first part of the tracing (arrows), a sinus mechanism cannot be excluded. The flutter waves become clearly apparent after carotid sinus massage is applied to slow conduction through the AV node, thereby increasing the degree of AV block.

AV: atrioventricular

Courtesy of Morton Arnsdorf, MD.

Graphic 76876 Version 3.0

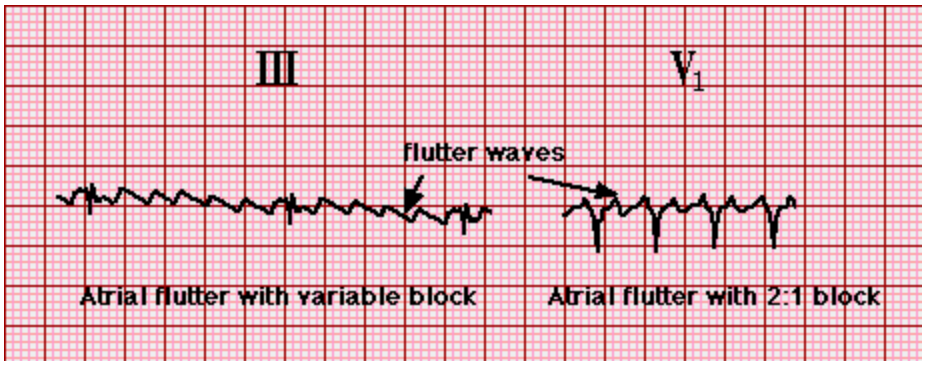
Normal rhythm strip

Normal rhythm strip in lead II. The PR interval is 0.15 sec and the QRS duration is 0.08 sec. Both the P and T waves are upright.

Courtesy of Morton F Arnsdorf, MD.

Graphic 59022 Version 3.0

Single-lead electrocardiogram showing atrial flutter



The atrial rate in this tracing of atrial waves is 300 beats per minute, and the flutter waves, which are "saw toothed," are uniform in morphology, amplitude, and cycle length. Impulse conduction to the ventricles is dependent upon the electrophysiologic characteristics of the atrioventricular node. Most commonly, every other atrial impulse is conducted and there is 2:1 block. However, when conduction through the atrioventricular node is impaired as a result of underlying disease, drugs, or increased vagal tone, there may be higher degrees of block, which may be variable, resulting in a regularly irregular ventricular rate.

Graphic 82303 Version 6.0

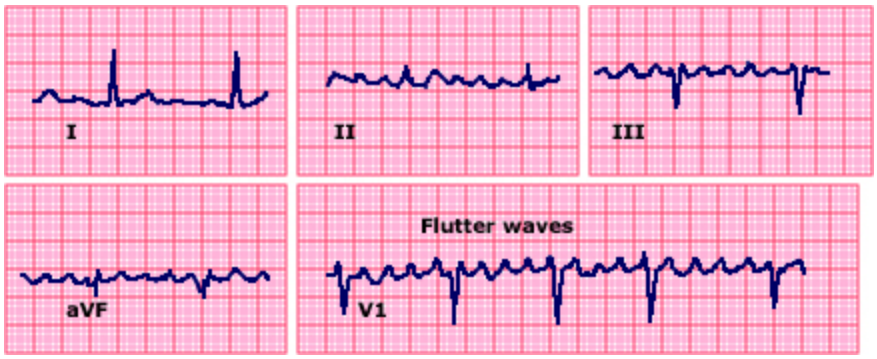
Normal rhythm strip

Normal rhythm strip in lead II. The PR interval is 0.15 sec and the QRS duration is 0.08 sec. Both the P and T waves are upright.

Courtesy of Morton F Arnsdorf, MD.

Graphic 59022 Version 3.0

Electrocardiogram in atypical atrial flutter



There are three findings that distinguish this arrhythmia from typical atrial flutter: the flutter rate is faster at 375 beats/minute; there is no isoelectric interval between the flutter waves; and there is more apparent positivity in leads II, III, and avF. The high degree of AV block is due to digoxin and some underlying AV nodal disease.

Courtesy of Morton Arnsdorf, MD.

Graphic 68846 Version 3.0

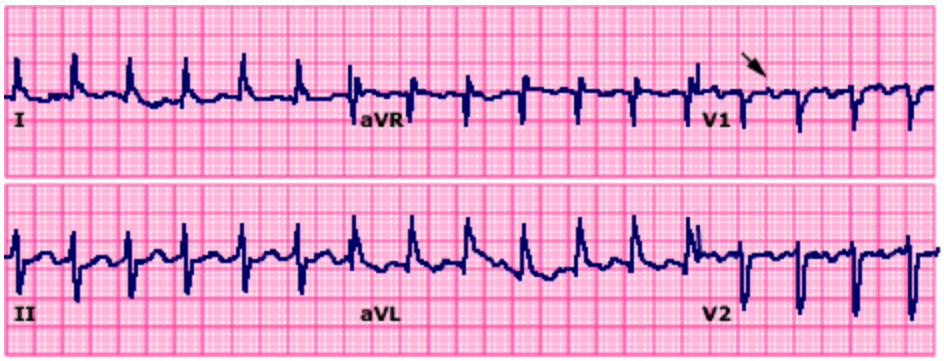
Normal rhythm strip

Normal rhythm strip in lead II. The PR interval is 0.15 sec and the QRS duration is 0.08 sec. Both the P and T waves are upright.

Courtesy of Morton F Arnsdorf, MD.

Graphic 59022 Version 3.0

Electrocardiogram (ECG) showing sinus tachycardia at a rate of 150 beats/min



Note the difficulty in separating the P waves from the T waves in the standard leads. The P waves are most evident in lead V1 (arrow) where the terminal negativity suggests left atrial enlargement.

Courtesy of Morton Arnsdorf, MD.

Graphic 78938 Version 3.0

Single-lead electrocardiogram (ECG) showing sinoatrial (SA) nodal reentrant tachycardia

Electrocardiogram showing SA nodal reentrant tachycardia. The first three beats are normal sinus beats at a rate of about 107 beats/min; the fourth beat is an atrial premature beat that is followed by a return to sinus rhythm. The eighth beat (arrow) represents the sudden onset of SA nodal reentrant tachycardia at a rate of about 145 beats/min. Since the P waves are similar to the sinus beats, the diagnosis is suggested only by the abrupt onset of the tachycardia.

Courtesy of Morton F Arnsdorf, MD.

Graphic 76364 Version 5.0

Normal rhythm strip

Normal rhythm strip in lead II. The PR interval is 0.15 sec and the QRS duration is 0.08 sec. Both the P and T waves are upright.

Courtesy of Morton F Arnsdorf, MD.

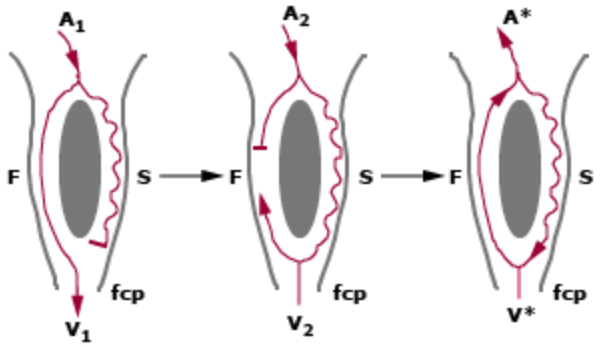
Graphic 59022 Version 3.0

Typical atrioventricular nodal reentrant tachycardia

The first two complexes are normal sinus beats with a normal P wave followed by a QRS complex. The third complex, an atrial premature beat (APB), has a prolonged PR interval; it initiates a common or typical atrioventricular nodal reentrant tachycardia (AVNRT) in which antegrade conduction to the ventricle is via the slow pathway and retrograde atrial activation is by the fast pathway. Although no distinct P wave is seen, the QRS complex has a small terminal deflection, known as a pseudo r', which is the P wave superimposed upon the terminal portion of the QRS complex.

Graphic 54290 Version 3.0

Slow-fast form of atrioventricular nodal reentrant tachycardia (AVNRT)



Representation of dual pathway physiology involving the atrioventricular (AV) node and perinodal atrial tissue in the common form of AVNRT.

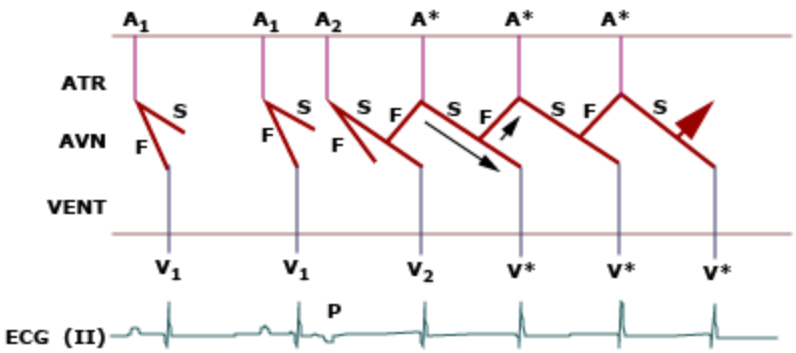
Left panel: A normal sinus beat (A_1) is conducted through the fast pathway (F) to the final common pathway (fcp) in the AV node and into the Bundle of His. The conduction through the slow pathway (S) runs into the refractory period of the impulse through the fast pathway and is extinguished.

Middle panel: A critically timed atrial premature beat (A_2) finds the fast pathway refractory in the antegrade direction but is able to conduct antegrade through the slow pathway, which has a shorter refractory period. If excitability in the fast pathway has recovered by the time the impulse reaches the fcp, there may be retrograde activation of the fast pathway.

Right panel: The retrograde impulse throws off an echo to the atrium (A*), and, if the slow pathway has recovered its excitability, the impulse reenters the slow pathway and produces ventricular depolarization (V*). If the mechanism persists, a repetitive circuit is established that creates a sustained reentrant tachycardia. The sequence of antegrade (S) and retrograde (F) conduction is called the slow-fast form of AVNRT.

Graphic 79760 Version 6.0

Generation of ECG in common form of atrioventricular nodal reentrant tachycardia (AVNRT)

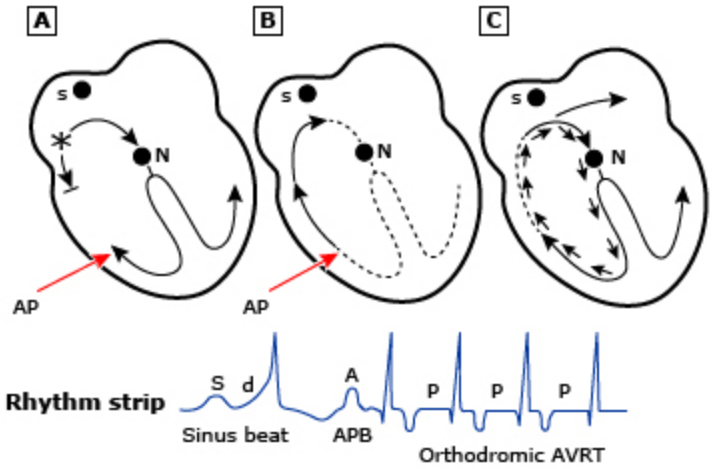


Ladder diagram of the common (slow-fast) form of AVNRT showing transmission of the impulse in atrial, atrioventricular (AV) nodal, and ventricular tissue and the resultant ECG. The first two cycles show the normal sinus beat (A_1) conducting through the fast pathway (F) to the ventricle (V_1) with the impulse blocked in the slow pathway. The premature atrial beat (A_2 on ladder diagram, P on ECG) is timed critically; it finds the fast pathway refractory but is able to conduct through slow pathway, resulting in a long PR interval, and a ventricular depolarization (V_2). It is also able to reenter and conduct retrogradely up the now recovered fast pathway, resulting in an inverted P wave (A*) that is buried in the QRS complex. This atrial echo beat (A*) then reenters the now recovered slow pathway, which conducts anterogradely and results in ventricular depolarization (V*). The cycle repeats, and AV nodal reentry is established.

ECG: electrocardiogram.

Graphic 50122 Version 6.0

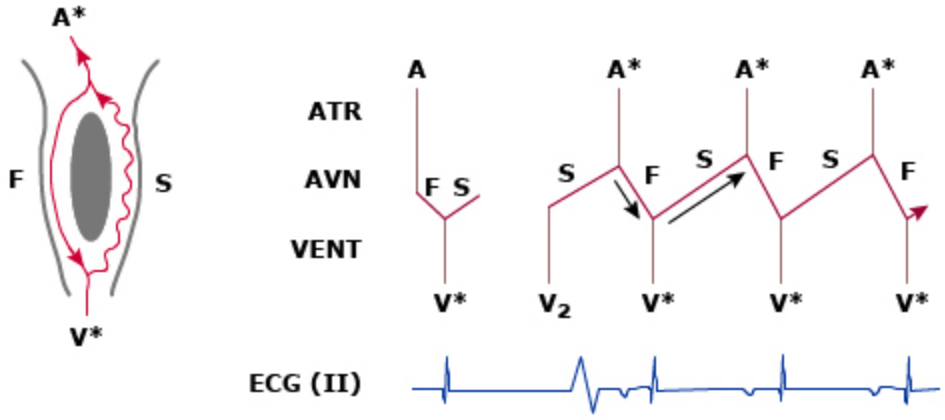
Orthodromic atrioventricular reentrant tachycardia (AVRT) in the setting of an accessory AV pathway



The rhythm strip shows a sinus (S) beat that has a short PR interval and a wide QRS complex as a result of a delta wave (d). Panel A shows an atrial premature beat (APB,*) that is blocked in the accessory pathway (AP), which has a long refractory period but is conducted antegradely through the atrioventricular node (N) and the His-Purkinje system, resulting in a normal PR interval and a narrow and normal QRS complex, as seen on the rhythm strip. After normal myocardial activation, the impulse is conducted retrogradely along the AP, activating the atrium in a retrograde fashion (panel B), which results in a negative P wave. If this activation sequence repeats itself (panel C), an orthodromic atrioventricular reentrant (or reciprocating) tachycardia (AVRT) is established.

Graphic 71302 Version 7.0

Uncommon fast-slow variant of atrioventricular nodal reentrant tachycardia (AVNRT)

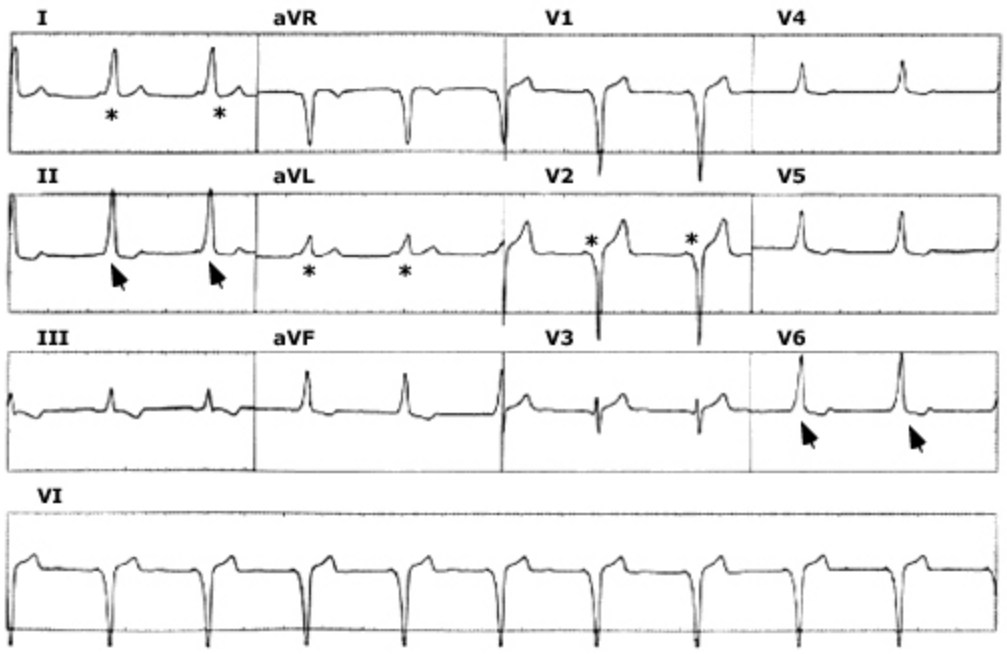


Diagrammatic representation in the circuit (left panel) and the ladder diagram (right panel) of the uncommon form of AVNRT (fast-slow variant). Antegrade conduction is through the fast (F) pathway and retrograde conduction is through the slow (S) pathway. Because of slow retrograde activation of the atrium, the P wave occurs after the QRS complex with a long RP interval and relatively short PR interval before the next QRS complex.

ECG: electrocardiogram.

Graphic 56806 Version 4.0

ECG in Wolff-Parkinson-White

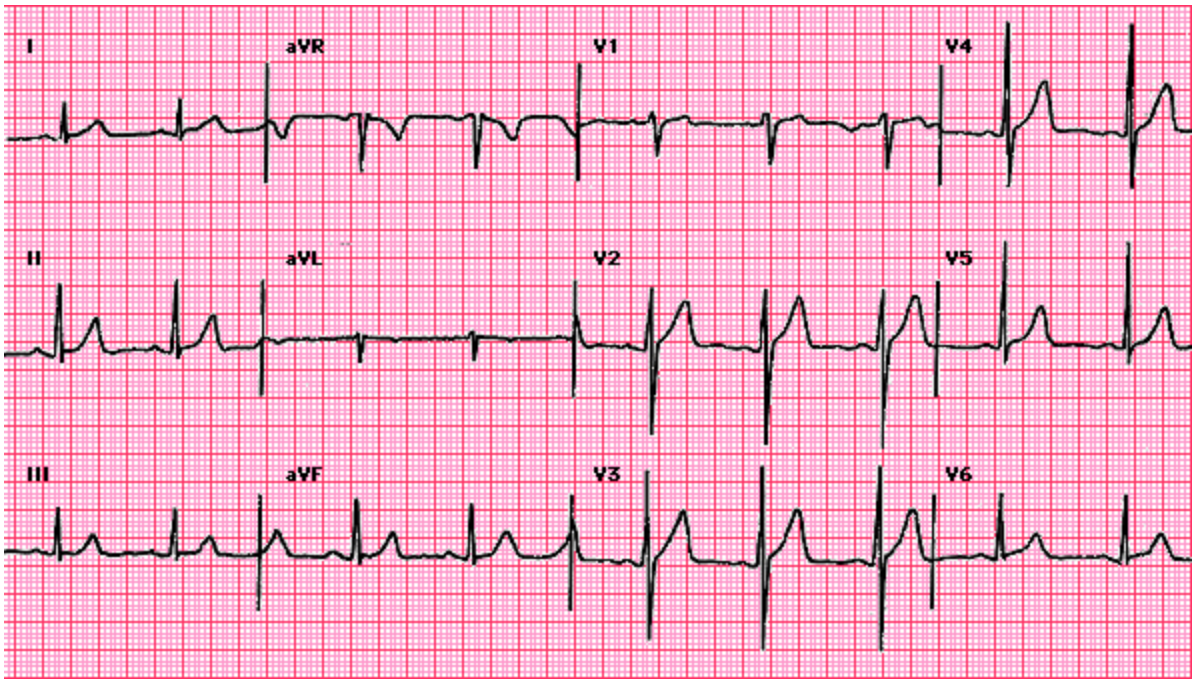


The 12-lead ECG shows the typical features of Wolff-Parkinson-White; the PR interval is short (*) and the QRS duration prolonged as a result of a delta wave (arrow), indicating ventricular preexcitation.

Courtesy of Martin Burke, DO.

Graphic 67181 Version 3.0

Normal ECG



Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0