Wide QRS complex tachycardias: Causes, epidemiology, and clinical manifestations

AUTHOR: Peter J Zimetbaum, MD
SECTION EDITORS: Ary L Goldberger, MD, James Hoekstra, MD
DEPUTY EDITOR: Todd F Dardas, MD, MS

All topics are updated as new evidence becomes available and our peer review process is complete.

Literature review current through: **Jan 2024.**
This topic last updated: **Nov 11, 2022.**

INTRODUCTION

Tachycardias are broadly categorized (algorithm 1) based upon the width of the QRS complex on the electrocardiogram (ECG).

- A narrow QRS complex (<120 milliseconds) reflects rapid activation of the ventricles via the normal His-Purkinje system (figure 1), which in turn suggests that the arrhythmia originates above or within the atrioventricular (AV) node (ie, a supraventricular tachycardia [SVT]).

- A widened QRS complex (≥120 milliseconds) occurs when ventricular activation is abnormally slow for one of the following reasons (see 'Differential diagnosis of WCT' below):

  - The arrhythmia originates outside of the normal conduction system and below the AV node (ie, ventricular tachycardia [VT])
  - Abnormalities within the His-Purkinje system (ie, SVT with aberrancy)
  - Pre-excitation with an SVT conducting antegrade over an accessory pathway, resulting in direct activation of the ventricular myocardium

A wide QRS complex tachycardia (WCT) represents a unique clinical challenge for three reasons:

- Diagnosing the arrhythmia is difficult – Although most WCTs are due to VT, the differential diagnosis includes a variety of SVTs. Diagnostic algorithms to differentiate these two etiologies are complex and imperfect. (See 'Differential diagnosis of WCT' below and "Wide QRS complex tachycardias: Approach to the diagnosis", section on 'Diagnosis'.)

- Urgent therapy is often required – Patients may be unstable at the onset of the arrhythmia or deteriorate rapidly at any time, particularly if the WCT is VT or SVT at an extremely rapid rate (eg, >200 beats per minute).

- Misdiagnosis of SVT when the true diagnosis is VT can lead to inappropriate therapy, which can precipitate hemodynamic collapse and cardiac arrest.

The causes, epidemiology, and clinical manifestations of patients with a WCT will be discussed here. The initial evaluation, diagnosis, and management of wide QRS complex tachycardias, as well as discussion of narrow QRS complex tachycardias, are presented separately. (See "Wide QRS complex tachycardias: Approach to the diagnosis" and "Wide QRS complex tachycardias: Approach to management" and "Overview of the acute management of tachyarrhythmias" and "Secondary prevention of sudden cardiac death in heart failure and cardiomyopathy" and "Narrow QRS complex tachycardias: Clinical manifestations, diagnosis, and evaluation".)

EPIDEMIOLOGY

Ventricular tachycardia (VT) is the most common cause of WCT, particularly in patients with a history of cardiac disease. In a series of unselected patients, VT accounted for up to 80 percent of cases of WCT [1,2]. Among patients with structural heart disease (eg, those with a prior myocardial infarction), the likelihood of a WCT being VT exceeds 90 percent [2].

Supraventricular tachycardia (SVT) results in WCT much less frequently than VT. Among patients with WCT due to SVT, aberrant conduction is the most common reason for a widened QRS (21 percent of cases in one series) [1]. However, an aberrantly conducted SVT is still much less common than VT as the cause of WCT. Antidromic AV reentrant tachycardia (AVRT) is a relatively uncommon cause of WCT (6 percent of cases in one series) [1].

CLINICAL MANIFESTATIONS

Symptoms — Patients with WCT are rarely asymptomatic, although the type and intensity of symptoms will vary depending upon the rate of the WCT, the presence or absence of significant comorbid conditions, and whether the WCT is ventricular tachycardia (VT) or supraventricular

tachycardia (SVT). Patients with WCT typically present with one or more of the following symptoms:

- Palpitations
- Chest pain
- Shortness of breath
- Syncope or presyncope
- Sudden cardiac arrest

Physical examination findings — Few physical examination findings in patients with a WCT are unique to WCT. Common findings may include:

- Tachycardia – By definition, patients will have a pulse exceeding 100 beats per minute related to the tachycardia.

- Hypotension – Patients may be hypotensive, particularly those with underlying cardiac disease who are unable to tolerate tachycardia, which may result in alterations in consciousness.

- Hypoxia and lung crackles – Patients in whom pulmonary congestion and heart failure result from the WCT may have hypoxia and crackles on lung examination. Often these patients will have underlying heart disease.

- Evidence of AV dissociation – AV dissociation, which is present in up to 75 percent of patients with VT, is not always easy to detect [3-5]. During AV dissociation, the normal coordination of atrial and ventricular contraction is lost, which may produce characteristic physical findings. The presence of AV dissociation strongly suggests VT, although its absence is less helpful.

  Although AV dissociation is typically diagnosed on the ECG, characteristic physical examination findings include (see "Wide QRS complex tachycardias: Approach to the diagnosis", section on 'AV dissociation'):

  - Marked fluctuations in the blood pressure because of the variability in the degree of left atrial contribution to left ventricular filling, stroke volume, and cardiac output.

  - Variability in the occurrence and intensity of heart sounds (especially S1; "cacophony of heart sounds"), which are heard more frequently when the rate of the tachycardia is slower.

- Cannon "A" waves – Cannon A waves are intermittent and irregular jugular venous pulsations of greater amplitude than normal waves. They reflect simultaneous atrial and ventricular activation, resulting in contraction of the right atrium against a closed tricuspid valve. Prominent A waves can also be seen during some SVTs, but they are usually regular, not irregular. Such prominent waves result from simultaneous atrial and ventricular contraction occurring with every beat. Classically, this is seen in AV nodal reentrant tachycardia (AVNRT) and has been called the "frog" sign. (See "Examination of the jugular venous pulse".)

DIFFERENTIAL DIAGNOSIS OF WCT

WCTs most often result from ventricular tachycardia (VT). Other less common causes include supraventricular tachycardia (SVT) with aberrant conduction, SVT with pre-excitation, SVT with ventricular pacing, and some types of artifact mimicking WCT (table 1).

Ventricular tachycardia — VT usually originates within the ventricular myocardium, outside of the normal conduction system, resulting in direct myocardial activation. Compared with a normally conducted supraventricular beat (which activates the ventricular myocardium via the normal AV node-His-Purkinje system), ventricular activation during VT is slower and proceeds in a different sequence. Thus, the QRS complex is wide and abnormal (waveform 1). As there may be slight changes of the activation sequence during the VT, reflecting the abnormal pathway of impulse conduction, there may be subtle changes in QRS complex morphology or in the ST-T waves.

VT may have one of three typical patterns:

- Monomorphic – Having a uniform and a fairly stable QRS morphology during an episode
- Polymorphic – Having a continuously varying QRS complex morphology and/or axis during an episode
- Bidirectional – Every other beat has a different axis as it travels alternately down different conduction pathways

The features of each form of VT are discussed separately. (See "Sustained monomorphic ventricular tachycardia: Clinical manifestations, diagnosis, and evaluation" and "Catecholaminergic polymorphic ventricular tachycardia" and "Congenital long QT syndrome: Epidemiology and clinical manifestations".)

Supraventricular tachycardia — When an SVT conducts to the ventricles via the normal AV node and His-Purkinje system, the activation wavefront spreads quickly through the ventricles,

and the QRS is usually narrow. In addition, the pathway of conduction to the ventricles is fixed and the same for each impulse, accounting for the uniformity of the QRS complexes and ST-T waves. However, SVT can also produce a widened QRS by a number of mechanisms, including aberrant conduction, pre-excitation, and the activation of ventricular pacing.

Aberrant conduction — The conduction of a supraventricular impulse can be delayed or blocked in the bundle branches or in the distal Purkinje system, resulting in a wide, abnormal QRS. This phenomenon is referred to as aberrancy. (See "Basic approach to delayed intraventricular conduction".)

Aberrant conduction may either be present at baseline or under certain conditions, such as faster heart rates.

- In patients with a left bundle branch block (LBBB), right bundle branch block, or a nonspecific intraventricular conduction delay on their baseline ECG, any SVT will have a widened QRS. Thus, if time allows, review of a baseline ECG can be helpful in differentiating VT from SVT with aberrancy. The presence of a conduction abnormality on the baseline ECG does not prove that the tachycardia is SVT with aberrancy, but the more similar the QRS during the WCT is to the QRS during sinus rhythm, the more likely it is that the WCT is an SVT with aberrancy.

  In patients with aberrancy at baseline who manifest a WCT in which the QRS complex is narrower than the baseline QRS, the WCT is likely VT originating near the ventricular septum, with early engagement of the specialized conducting system. This scenario is extremely unusual.

- In patients with a narrow QRS complex at baseline which widens at faster heart rates, conduction is normal during sinus rhythm but aberrant during the tachycardia. The most common reason for this is rate-related aberration (functional bundle branch block), in which rapidly generated impulses reach the conducting fibers before they have fully recovered from the previous impulse. Such a delay in recovery may also be the result of underlying disease of the His-Purkinje system, hyperkalemia, or the actions of antiarrhythmic drugs, particularly the class IC agents (eg, flecainide, propafenone).

  The class I antiarrhythmic drugs (table 2) can cause significant slowing of conduction during SVT and also VT. These drugs, especially class IC agents, slow conduction and have a property of "use-dependency" (a progressive decrease in impulse conduction velocity and wider QRS complex duration at faster heart rates). As a result, these drugs can cause rate-related aberration and a wide QRS complex during any SVT. However, they can also

cause VT with a very wide, bizarre QRS, which may be incessant [6,7]. (See "Cardiac excitability, mechanisms of arrhythmia, and action of antiarrhythmic drugs".)

Pre-excitation syndrome — In the pre-excitation syndromes, AV conduction can occur over the normal conduction system and also via an accessory AV pathway (figure 2A-C). These two pathways create the anatomic substrate for a reentrant circuit (macroreentrant circuit), facilitating the development of a circus movement or reentrant tachycardia known as AV reentrant tachycardia (AVRT). (See "ECG tutorial: Preexcitation syndromes" and "Atrioventricular reentrant tachycardia (AVRT) associated with an accessory pathway", section on 'Narrow complex AVRT' and "Atrioventricular reentrant tachycardia (AVRT) associated with an accessory pathway", section on 'Wide complex AVRT'.)

AVRT, which occurs both in patients with manifest pre-excitation (Wolff-Parkinson-White [WPW] syndrome) or concealed accessory pathways, can present with a narrow or a wide QRS complex:

- If antegrade conduction occurs over an accessory pathway and retrograde conduction occurs over the AV node or a second accessory pathway, the QRS complex will be wide with an unusual morphology. This is known as an **antidromic** AVRT (figure 3 and waveform 2). Antidromic AVRT is difficult to differentiate from VT because ventricular activation starts outside the normal intraventricular conduction system in both types of tachycardia (ie, there is direct myocardial activation). (See "Wide QRS complex tachycardias: Approach to the diagnosis", section on 'VT versus AVRT'.)

- If antegrade conduction to the ventricles occurs over the AV node and retrograde conduction is over the accessory pathway, the QRS complex will be narrow (unless there is aberrant conduction at baseline with a wide QRS complex). This narrow complex AVRT is known as an **orthodromic** AVRT (figure 4 and waveform 3). Orthodromic AVRT can also occur with rate-related aberrancy, creating a WCT.

In addition, patients with a manifest accessory pathway (ie, WPW syndrome) may develop a different SVT (eg, atrial tachycardia, atrial fibrillation [AF], or atrial flutter). In such cases, the atrial impulses may use the accessory pathway to conduct to the ventricles, and the QRS could be either narrow or wide, depending upon whether ventricular activation occurs over the normal conduction system, the accessory pathway, or both (waveform 4). (See "Wolff-Parkinson-White syndrome: Anatomy, epidemiology, clinical manifestations, and diagnosis", section on 'Arrhythmias associated with WPW'.)

Pacemakers — When the ventricles are activated by a pacing device, the QRS complex is generally wide:

- Most transvenous ventricular pacemakers pace the right ventricle, causing a wide QRS complex of the LBBB type. Typically, the surface ECG shows a broad R wave in lead I, indicating conduction from right to left. (See "Overview of pacemakers in heart failure".)

- Pacemakers used in cardiac resynchronization therapy (CRT) usually pace both ventricles. Although CRT generates a QRS complex that is narrower than the patient's baseline (a chronically widened QRS is one of the components of the indication for CRT), it is still usually longer than 120 milliseconds. The surface ECG usually shows a Q wave or QS complex in lead I, indicating activation from left to right, and there is usually a RBBB pattern in lead V_1. (See "Cardiac resynchronization therapy in heart failure: Indications and choice of system".)

Recognizing that a QRS complex is due to ventricular pacing can be challenging, particularly during a tachycardia. In addition to characteristic QRS morphology, a pacing "spike" or stimulus artifact can often be identified. The stimulus artifact is a narrow electrical signal too rapid to represent myocardial depolarization.

Among patients with a pacemaker or an implantable cardioverter-defibrillator, further possibilities need to be considered in addition to the usual differential diagnosis of a WCT. These include:

- In the presence of sinus tachycardia or some SVTs (eg, an atrial tachycardia, AF, or atrial flutter), the device may "track" the atrial impulse and pace the ventricle at the rapid rate, resulting in a WCT. (See "Modes of cardiac pacing: Nomenclature and selection", section on 'Mode switching'.)

- A WCT can result if ventricular paced beats are conducted retrograde (backward) through the AV node to the atrium, resulting in an atrial signal, which the pacemaker senses and tracks with another ventricular stimulus. This ventricular paced beat is also conducted retrograde, and the cycle repeats indefinitely, a process termed pacemaker-mediated tachycardia (PMT) or endless loop tachycardia. PMT usually occurs at the upper rate limit. A different mechanism of pacemaker-associated tachycardia, non-reentrant repetitive ventriculoatrial synchrony, also creates a wide complex rhythm, but usually at the lower rate limit or sensor-mediated rate rather than at the upper rate limit.

These and other arrhythmias associated with pacemakers are discussed in detail separately. (See "Unexpected rhythms with normally functioning dual-chamber pacing systems", section on 'Pacemaker-mediated tachycardia'.)

Artifact mimicking ventricular tachycardia — ECG artifact, particularly when observed on a single-lead rhythm strip, may be misdiagnosed as VT (waveform 5) [8]. The presence of narrow-complex beats that can be seen to "march" through the supposed WCT at a fixed rate strongly supports the diagnosis of artifact.

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: Arrhythmias in adults" and "Society guideline links: Ventricular arrhythmias" and "Society guideline links: Supraventricular arrhythmias".)

SUMMARY AND RECOMMENDATIONS

- **Background** – A wide QRS complex tachycardia (WCT) represents a unique clinical challenge for two reasons: Diagnosis of the arrhythmia is frequently difficult, and urgent therapy is often required. (See 'Introduction' above.)
- **Epidemiology** – Ventricular tachycardia (VT) is the most common cause of WCT, particularly in patients with a history of cardiac disease, while supraventricular tachycardia (SVT) results in WCT (due to aberrant conduction, pre-excitation, or ventricular pacing) much less frequently. WCT is identified as VT in up to 80 percent of unselected patients and more than 90 percent of patients with known structural heart disease. (See 'Epidemiology' above and 'Differential diagnosis of WCT' above.)
- **Clinical manifestations** – Patients with WCT are rarely asymptomatic, although the type and intensity of symptoms will vary depending upon the rate of the WCT, the presence or absence of significant comorbid conditions, and whether the WCT is VT or SVT. Patients with WCT typically present with one or more of the following: palpitations, chest pain, shortness of breath, syncope/presyncope, or sudden cardiac arrest. (See 'Clinical manifestations' above.)
- **Differential diagnosis** – The differential diagnosis of WCTs includes (see 'Differential diagnosis of WCT' above):
  - VT, including monomorphic VT, polymorphic VT, and bidirectional VT
  - SVT with aberrant conduction
  - SVT with conduction over an accessory pathway

- Paced ventricular rhythms
- ECG artifact

ACKNOWLEDGMENT

The UpToDate editorial staff acknowledges Leonard Ganz, MD, FHRS, FACC, who contributed to an earlier version of this topic review.

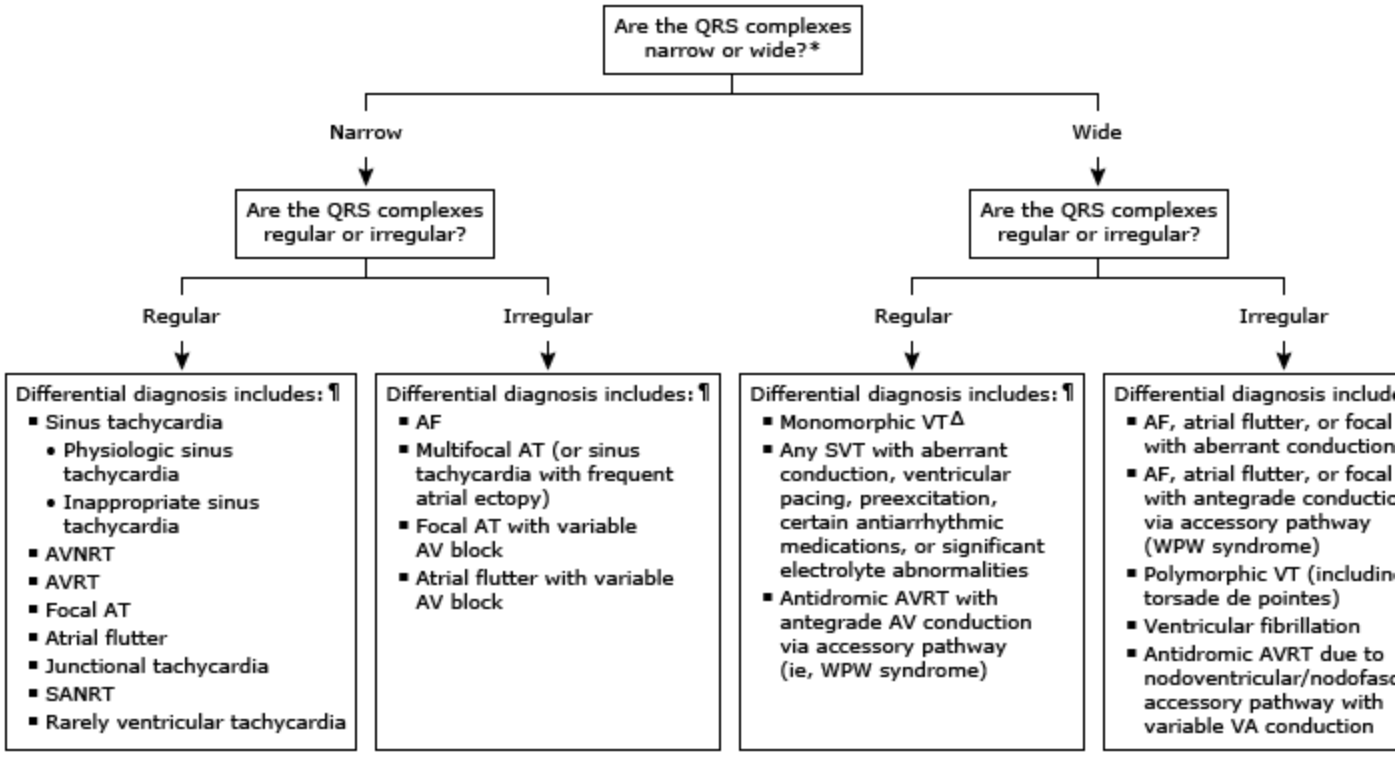
REFERENCES

1. Miller JM, Das MK. Differential diagnosis of narrow and wide complex tachycardias. In: Cardiac Electrophysiology From Cell to Bedside, 7th, Zipes DP, Jalife J, Stevenson WG (Eds), W.B. Saunders, Philadelphia 2018.
2. Vereckei A. Current algorithms for the diagnosis of wide QRS complex tachycardias. Curr Cardiol Rev 2014; 10:262.
3. Gupta AK, Thakur RK. Wide QRS complex tachycardias. Med Clin North Am 2001; 85:245.
4. Tchou P, Young P, Mahmud R, et al. Useful clinical criteria for the diagnosis of ventricular tachycardia. Am J Med 1988; 84:53.
5. Wellens HJ, Bär FW, Lie KI. The value of the electrocardiogram in the differential diagnosis of a tachycardia with a widened QRS complex. Am J Med 1978; 64:27.
6. Ranger S, Talajic M, Lemery R, et al. Kinetics of use-dependent ventricular conduction slowing by antiarrhythmic drugs in humans. Circulation 1991; 83:1987.
7. Ranger S, Talajic M, Lemery R, et al. Amplification of flecainide-induced ventricular conduction slowing by exercise. A potentially significant clinical consequence of use-dependent sodium channel blockade. Circulation 1989; 79:1000.
8. Knight BP, Pelosi F, Michaud GF, et al. Physician interpretation of electrocardiographic artifact that mimics ventricular tachycardia. Am J Med 2001; 110:335.

Topic 116601 Version 18.0

GRAPHICS

Algorithm for the initial ECG review and differential diagnosis of tachycardia



ECG: electrocardiogram; AVNRT: atrioventricular nodal reentrant tachycardia; AVRT: atrioventricular reciprocating (bypass-tract mediated) tachycardia; AT: atrial tachycardia; SANRT: sinoatrial nodal reentrant tachycardia; AF: atrial fibrillation; AV: atrioventricular; VT: ventricular tachycardia; SVT: supraventricular tachycardia; WPW: Wolff-Parkinson-White.

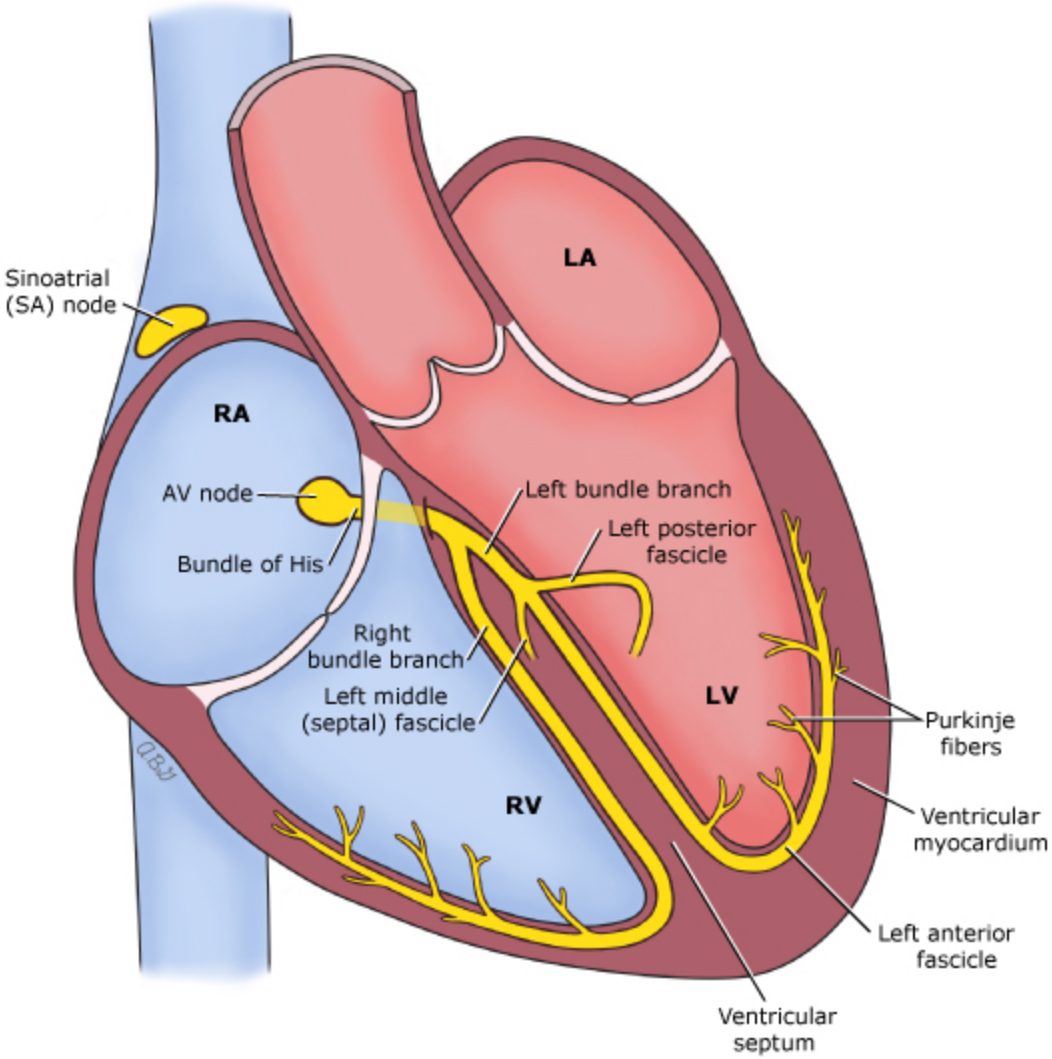
* A narrow QRS complex is <120 milliseconds in duration, whereas a wide QRS complex is ≥120 milliseconds in duration.

¶ Refer to UpToDate topic reviews for additional details on specific ECG findings and management of individual arrhythmias.

Δ Monomorphic VT accounts for 80% of wide QRS complex tachycardias; refer to UpToDate topic on diagnosis of wide QRS complex tachycardias for additional information on discriminating VT from SVT.

Graphic 117571 Version 3.0

Normal conduction system



Schematic representation of the normal intraventricular conduction system (His-Purkinje system). The Bundle of His divides into the left bundle branch and right bundle branch. The left bundle branch divides into anterior, posterior, and, in some cases, median fascicles.

AV: atrioventricular; RA: right atrium; LA: left atrium; RV: right ventricle; LV: left ventricle.

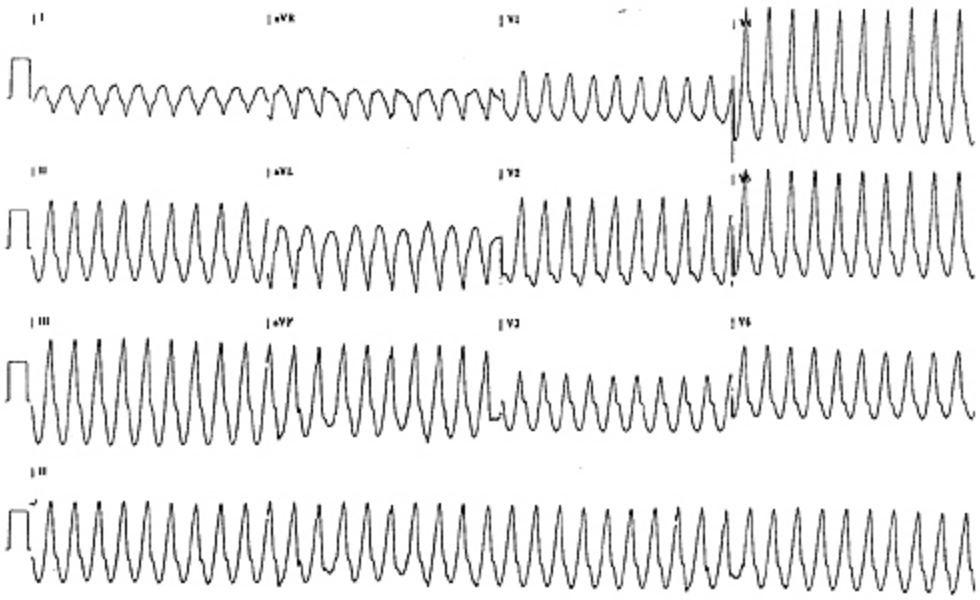
Graphic 63340 Version 6.0

Causes of a wide QRS complex tachycardia

Ventricular tachycardia (VT)
Any type of supraventricular tachycardia (SVT) with a preexistent bundle branch block or a rate-related (functional) bundle branch block
Sinus tachycardia
Atrial tachycardia
Atrial flutter
Atrioventricular nodal reentrant tachycardia
Atrioventricular reentrant tachycardia (orthodromic)
Any SVT which occurs in a patient receiving an antiarrhythmic drug, primarily class IA or IC, or in a patient with severe hyperkalemia
Any SVT with antegrade conduction via an accessory pathway (Wolff-Parkinson-White syndrome)
Sinus tachycardia
Atrial tachycardia
Atrial flutter
Atrioventricular reentrant tachycardia (antidromic)
Electronic pacemaker in certain specific settings

Graphic 51512 Version 2.0

12-lead electrocardiogram (ECG) recorded in a patient with repetitive monomorphic ventricular tachycardia (RMVT) arising from the left ventricular outflow tract (LVOT)



This electrocardiogram (ECG) illustrates repetitive monomorphic ventricular tachycardia (RMVT) with a right bundle, inferior axis morphology signifying its left ventricular site of origin. This VT was localized to the area of the aorto-mitral continuity in the left ventricular outflow tract (LVOT).

Graphic 81690 Version 3.0

Revised (2018) Vaughan Williams classification of antiarrhythmic drugs abridged table

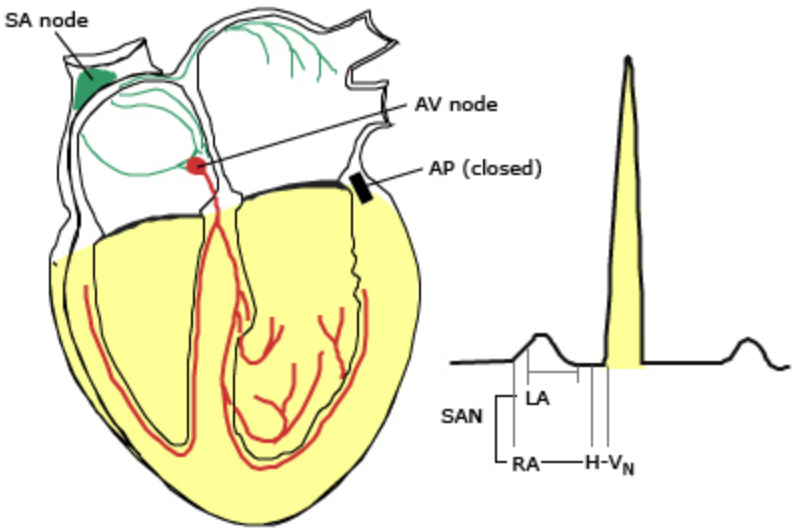
Class 0 (HCN channel blockers)
Ivabradine
Class I (voltage-gated Na+ channel blockers)
Class Ia (intermediate dissociation):
▪ Quinidine, ajmaline, disopyramide, procainamide
Class Ib (rapid dissociation):
▪ Lidocaine, mexilitine
Class Ic (slow dissociation):
▪ Propafenone, flecainide
Class Id (late current):
▪ Ranolazine
Class II (autonomic inhibitors and activators)
Class IIa (beta blockers):
▪ Nonselective: carvedilol, propranolol, nadolol
▪ Selective: atenolol, bisoprolol, betaxolol, celiprolol, esmolol, metoprolol
Class IIb (nonselective beta agonists):
▪ Isoproterenol
Class IIc (muscarinic M2 receptor inhibitors):
▪ Atropine, anisodamine, hyoscine, scopolamine
Class IId (muscarinic M2 receptor activators):
▪ Carbachol, pilocarpine, methacholine, digoxin
Class IIe (adenosine A1 receptor activators):
▪ Adenosine
Class III (K+ channel blockers and openers)
Class IIIa (voltage dependent K+ channel blockers):
▪ Ambasilide, amiodarone, dronedarone, dofetilide, ibutilide, sotalol, vernakalant

Class IIIb (metabolically dependent K+ channel openers):
▪ Nicorandil, pinacidil
Class IV (Ca++ handling modulators)
Class IVa (surface membrane Ca++ channel blockers):
▪ Bepridil, diltiazem, verapamil
Class IVb (intracellular Ca++ channel blockers):
▪ Flecainide, propafenone
Class V (mechanosensitive channel blockers):
No approved medications
Class VI (gap junction channel blockers)
No approved medications
Class VII (upstream target modulators)
Angiotensin converting enzyme inhibitors
Angiotensin receptor blockers
Omega-3 fatty acids
Statins

HCN: hyperpolarization-activated cyclic nucleotide-gated; Na: sodium; K: potassium; Ca: calcium.

Graphic 120433 Version 3.0

AV conduction with a concealed accessory pathway

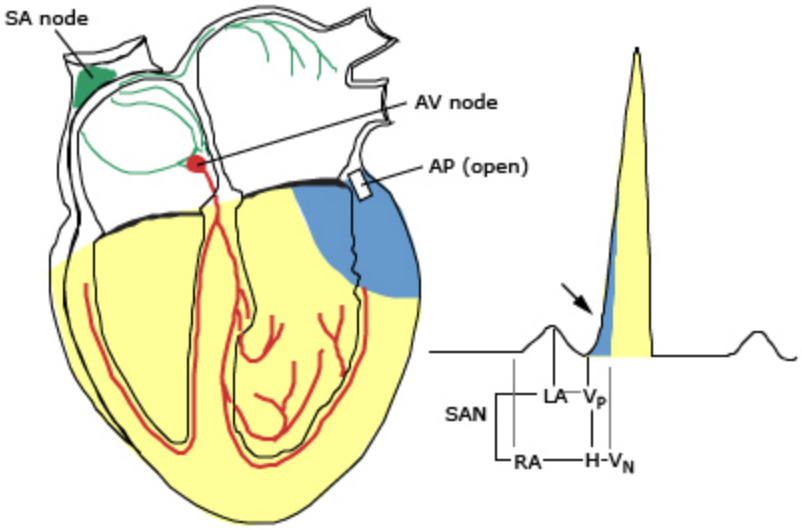


Schematic representation of AV conduction. The normal pacemaker is in the sinoatrial (SA) node at the junction of the superior vena cava and the right atrium. The SA node activates the right and left atria (shown in green). In the absence of an accessory pathway (AP) or, as in this case, if the AP is concealed, ventricular activation results from the impulse traversing the AV node, the specialized infranodal conducting system (His bundle, bundle branches, and fascicular branches, shown in red), thereby activating the ventricular myocardium (shown in yellow). The ECG shows a normal PR interval and a narrow QRS complex. The inset on the right shows the timing of SA node (SAN), right (RA) and left atrial (LA), His bundle (H), and the beginning of normal ventricular activation (V_N). All of ventricular activation (shown in yellow) is due to normal AV nodal and infranodal conduction.

AV: atrioventricular; ECG: electrocardiogram.

Graphic 73740 Version 5.0

AV conduction through an overt accessory pathway

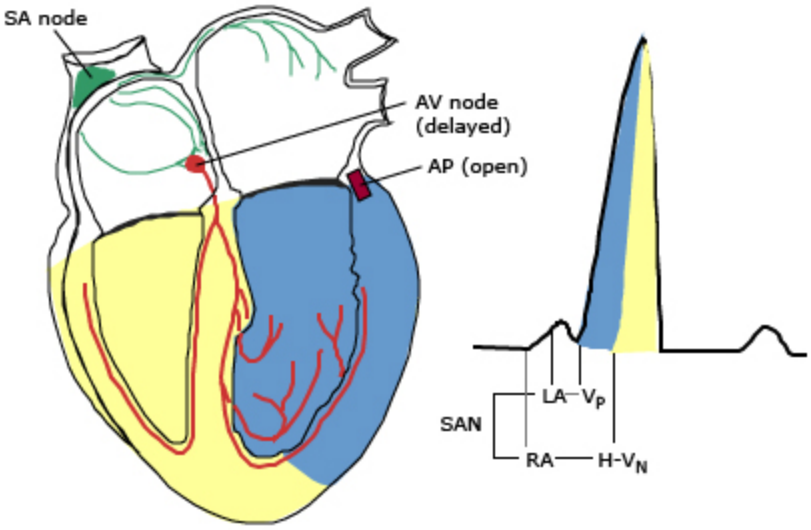


Compared with normal conduction in the preceding diagram, the accessory pathway (AP) is now overt. As a result, ventricular activation results from both early activation (pre-excitation) of the free wall of the left ventricle (shown in blue) and from normal activation (shown in yellow). The degree of unopposed pre-excitation depends upon the time required to conduct through the right and left atria, the AP, and the ventricular myocardium as compared with conduction through the normal pathways. The inset on the right shows the ECG timing of these events. The net effect is a QRS complex that is a fusion of ventricular pre-excitation (blue) and normal excitation (yellow). Early activation throughout the AP (V_P) occurs at about the same time as His bundle depolarization (H). This leads to a shorter PR interval, a small delta wave (arrow), and some prolongation of the QRS duration.

AV: atrioventricular.

Graphic 53773 Version 6.0

Conduction through an accessory pathway with AV nodal delay

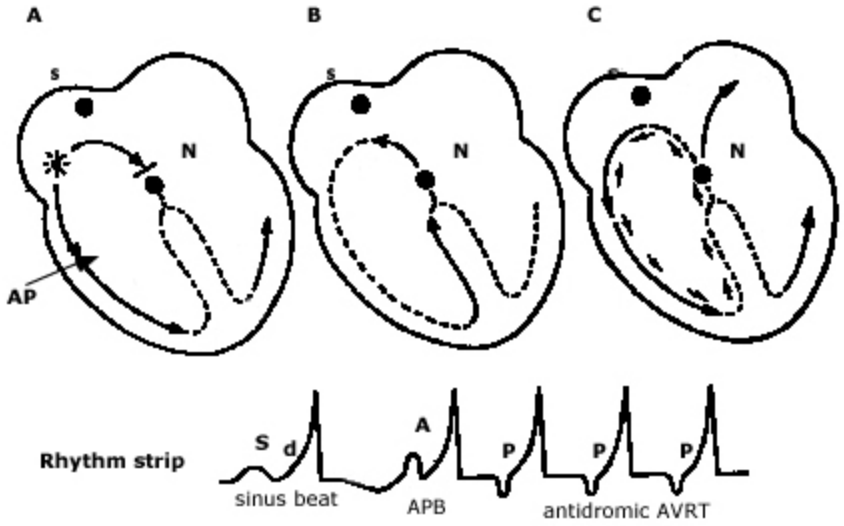


Compared with conduction through an AP with normal AV node conduction, delayed conduction through the AV node allows more of the ventricular myocardium to be activated by pre-excitation (shown in blue). The inset on the right shows the ECG timing of these events. The atrial to His interval is increased due to the AV nodal delay (RA to H); His activation is so delayed that it follows activation caused by the AP (V_P). The PR interval is short due to the pre-excitation, the delta wave (arrow) is more pronounced due to the greater and unopposed early forces (blue), and the QRS duration is prolonged due to the later than normal ventricular activation caused by the AV nodal delay (yellow).

AV: atrioventricular; ECG: electrocardiogram.

Graphic 62454 Version 6.0

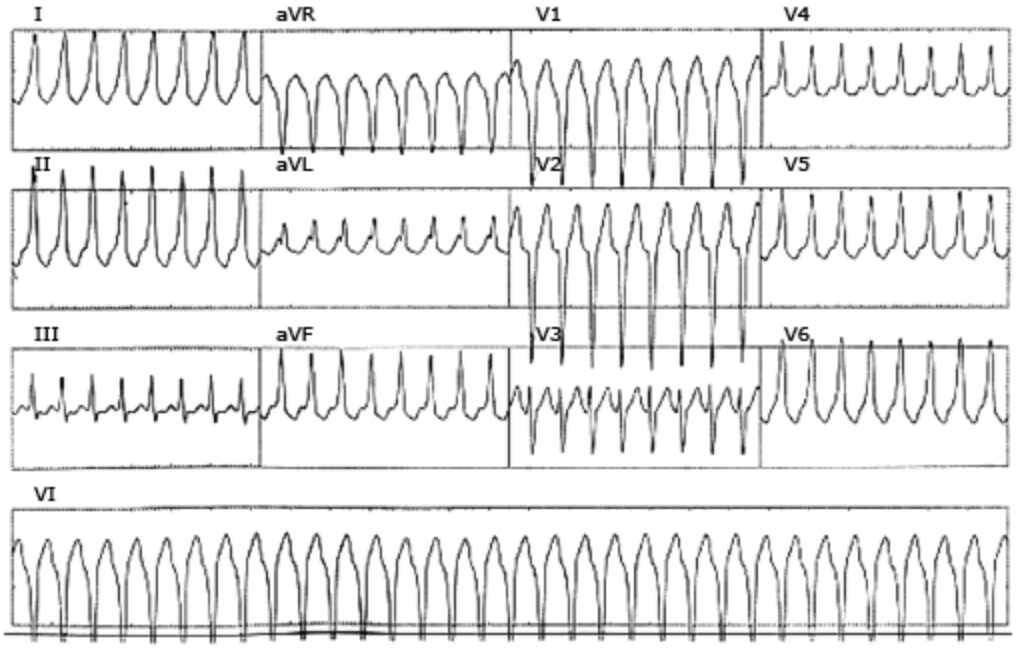
Antidromic atrioventricular reentrant tachycardia (AVRT) in the setting of an accessory AV pathway



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 the activation sequence with an atrial premature beat (APB,*). The impulse reaches the atrioventricular node (N) before it has repolarized and hence is blocked in this structure. However, the accessory pathway (AP), which has a short refractory period, is able to conduct the impulse antegradely, resulting in an APB with a widened QRS morphology similar to the sinus beat. As seen in panel B, following myocardial activation, the impulse is conducted retrogradely along the His-Purkinje system and AV node, resulting in retrograde atrial activation, seen on the rhythm strip as an inverted P wave. If this activation sequence repeats itself (panel C), a wide QRS complex antidromic atrioventricular reentrant (or reciprocating) tachycardia (AVRT) is established.

Graphic 50433 Version 4.0

12-lead electrocardiogram (ECG) showing antidromic atrioventricular reentrant tachycardia (AVRT) in a patient with an accessory AV pathway

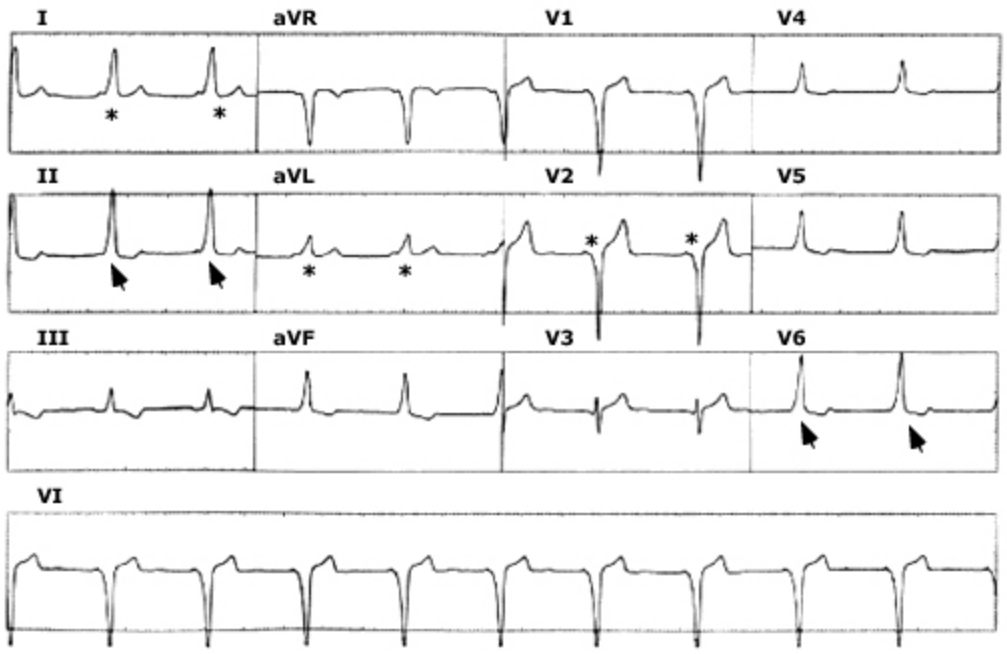


The 12-lead ECG of a patient with Wolff-Parkinson-White shows a regular tachycardia. The QRS complexes are widened and are identical to the QRS complexes seen in sinus rhythm; the antegrade conduction to the ventricle is via the accessory pathway and retrograde conduction is via the normal His-atrioventricular node pathway. This is, therefore, an antidromic atrioventricular reentrant tachycardia (AVRT).

Courtesy of Martin Burke, DO.

Graphic 54484 Version 20.0

ECG in Wolff-Parkinson-White

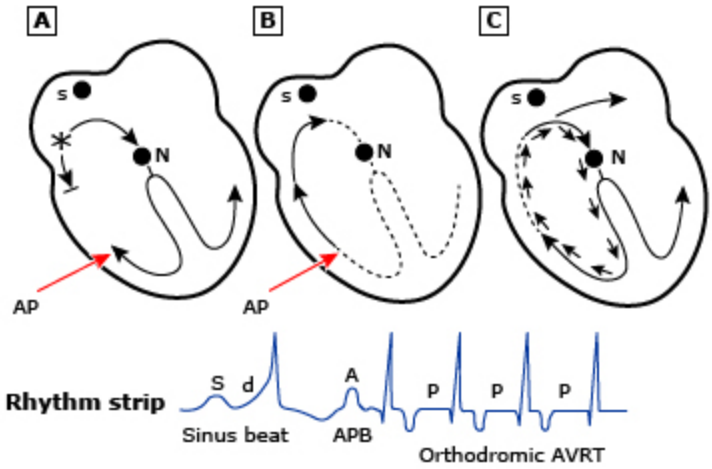


The 12-lead ECG shows the typical features of Wolff-Parkinson-White; the PR interval is short (*) and the QRS duration prolonged as a result of a delta wave (arrow), indicating ventricular preexcitation.

Courtesy of Martin Burke, DO.

Graphic 67181 Version 3.0

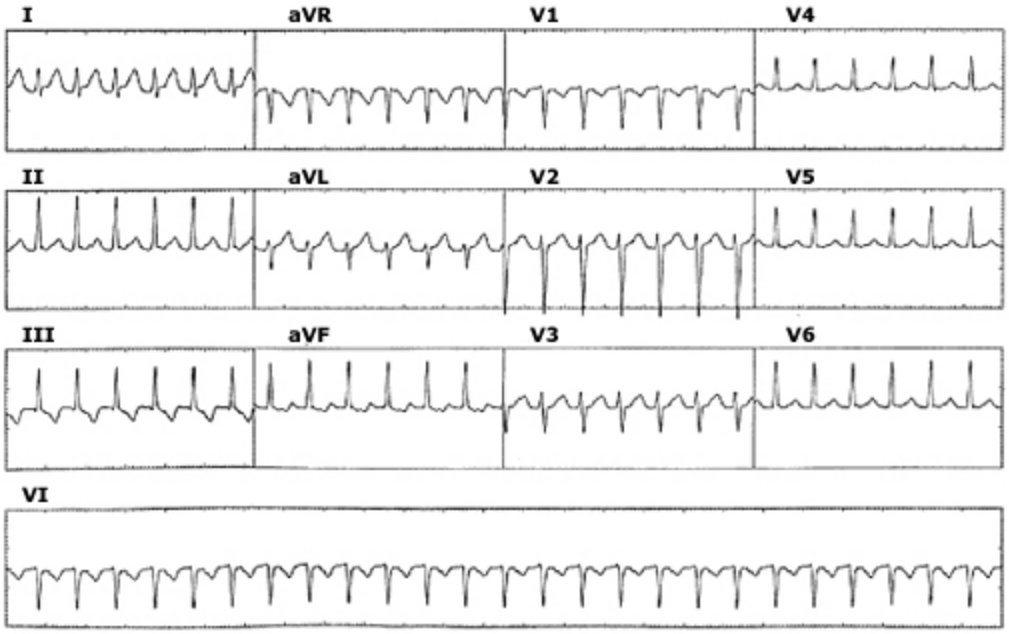
Orthodromic atrioventricular reentrant tachycardia (AVRT) in the setting of an accessory AV pathway



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

12-lead electrocardiogram (ECG) showing orthodromic atrioventricular reentrant tachycardia (AVRT) in a patient with an accessory AV pathway

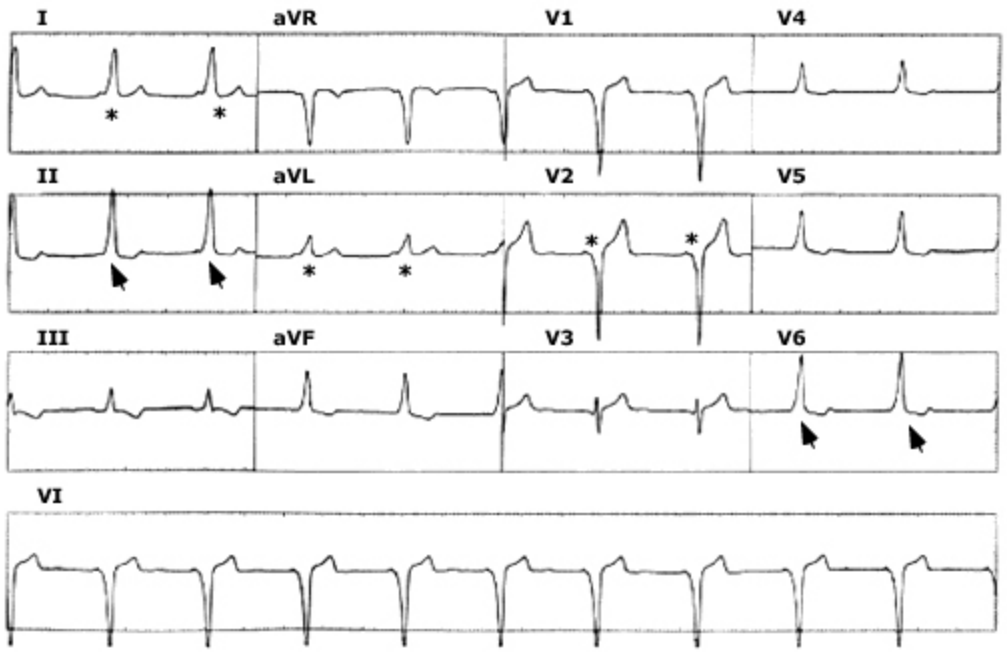


The 12-lead ECG from a patient with Wolff-Parkinson-White shows a regular tachycardia. However, in contrast to the QRS pattern during sinus rhythm, the QRS complexes are narrow, without evidence of a delta wave or pre-excitation; this is due to the fact that antegrade ventricular activation occurs via the normal atrioventricular node-His Purkinje pathway, while retrograde atrial activation is via the accessory pathway. Therefore, this is called an orthodromic atrioventricular reentrant tachycardia (OAVRT).

Courtesy of Martin Burke, DO.

Graphic 68804 Version 5.0

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

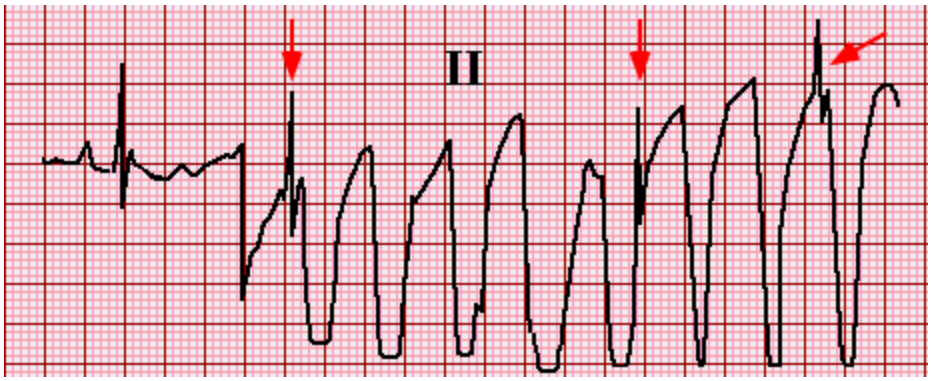
ECG pre-excited atrial fibrillation

12-lead ECG showing atrial fibrillation in a patient with antegrade conduction through both the AV node and an accessory pathway. The occasional narrow QRS complexes reflect conduction through the AV node, while the wide QRS complexes, with instantaneous rates close to 300 beats per minute, are due to antegrade conduction via an accessory pathway (probably postero-septal based on the QRS morphology).

AV: atrioventricular; ECG: electrocardiogram.

Graphic 117553 Version 2.0

Tremor artifact



This dramatic example of tremor artifact demonstrates complexes that simulate a run of ventricular tachycardia. However, QRS complexes (arrows) can clearly be seen marching through the rhythm strip.

Graphic 62575 Version 3.0