

Arrhythmias are defined as abnormalities of electrical conduction and rhythm of the heart and encompass a broad range of congenital and acquired disease states. Although relatively rare in the general pediatric population, arrhythmias can lead to significant morbidity and mortality, especially when occurring during the postoperative care of children with CHD. Figure 34-1 and Figure 34-2 provide algorithms for the diagnosis of narrow- and wide-complex tachyarrhythmias, respectively.

Sinus-Rhythm Abnormalities

Sinus rhythm occurs when electrical impulses originating in the sinoatrial node (SA node) propagate throughout the atria and coalesce at the center of the heart in the AV node before continuing via the bundle of His and Purkinje fibers to the individual right/left ventricular bundles. These electrical impulses are most commonly translated into an ECG as the P-Q-R-S-T wave forms. The waveforms provide a visual depiction of the SA nodal/atrial impulse (P wave) and subsequent ventricular depolarization (Q-R-S waves) and repolarization (T wave) activity.

Rhythm that occurs in a sinus fashion but is faster than established upper limits of normal for age is labeled *sinus tachycardia*. It often occurs in response to stress or painful stimuli, fever, anemia, hypovolemia, high catecholamine state, or medications. Treatment is directed at the underlying cause. Conversely, slow heart rates below the lower limits of heart rate for age are labeled *sinus bradycardia* and are usually benign and without hemodynamic significance. Sinus bradycardia can be observed during sleep but also occur in conditions of high vagal tone, hypothermia, hypotension, hypoxemia, acidosis, drugs, electrolyte abnormalities, or increased intracranial pressure. Treatment, if needed, is usually focused on addressing the underlying cause. Patients with certain forms of CHD, including heterotaxy syndrome, may be more prone to bradycardia that may indeed be clinically significant.

Sinus node dysfunction (also known as sick-sinus syndrome) may also present with slow heart rates frequently alternating with periods of tachycardia and is most commonly due to secondary causes such as cardiac surgery, infection (myocarditis), trauma, ischemia, or cardioactive drugs rather than a primary arrhythmia. In symptomatic cases, permanent pacemaker implantation may be needed for definitive therapy.

Absent SA node activity may contribute to the development of *junctional rhythm*, a condition characterized by QRS complexes that have an identical morphology to that of sinus rhythm but lack preceding P waves. Pacing the atrium at 10-20 bpm above the junctional rate demonstrates normal AV nodal conduction, restores AV synchrony, and leads to improvement in stroke volume and cardiac output.

Conduction Abnormalities

Abnormal AV conduction occurs when transmission of the normal SA node impulses is delayed or blocked due to an abnormality in the conduction system, specifically of the

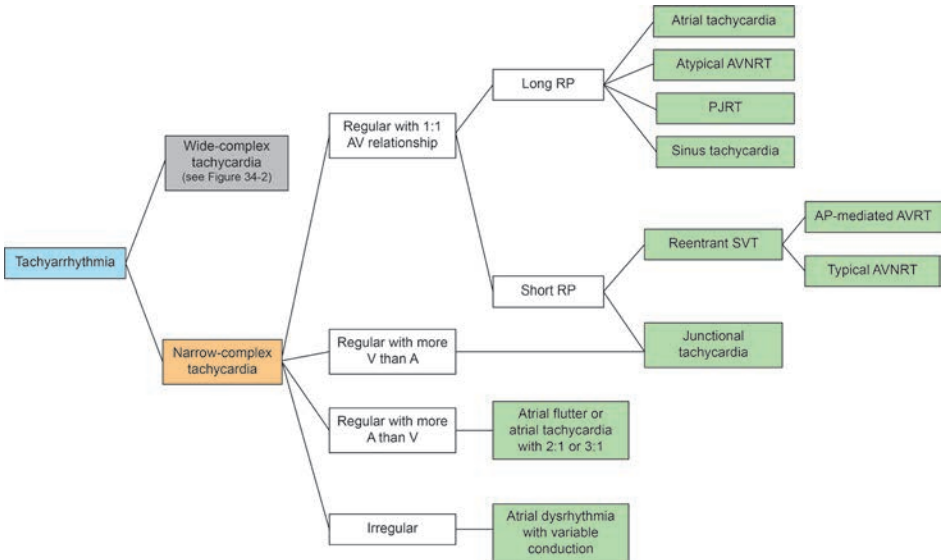


Figure 34-1. Diagnostic algorithm for narrow-complex tachyarrhythmias. PJRT: Permanent junctional reciprocating tachycardia.

AV node or His-Purkinje system. AV conduction deficits are covered in Chapter 75 and only briefly mentioned here.

First-degree AV block results in stable prolongation of the PR interval above the upper limits of normal for age and heart rate and is a result of an abnormal delay in conduction through the AV node. This is typically a benign phenomenon.

Second-degree AV block results from intermittent failure of AV conduction and is categorized into two common forms: Mobitz type I (Wenckebach phenomenon) and Mobitz type II. Type I second-degree AV block occurs at the level of the AV node yielding progressive lengthening of the PR interval until it fails to conduct the atrial impulse to the ventricle. Type II second-degree AV block occurs below the level of the bundle of His and is defined as the sudden loss of AV conduction occurring after normal sinus rhythm (no evidence of PR-interval prolongation). Although less common than type I, type-II second-degree block is a more serious form of AV conduction disorder in which progression to complete heart block with hemodynamic compromise is more likely. Higher grade forms of second-degree AV block occur when two successive P waves fail to be followed by QRS complexes.

Third-degree (complete) AV block is defined as complete interruption of atrial impulse transmission resulting in atrial and ventricular activity that is independent of each other (AV dissociation). Surface ECG morphology will show regular P waves at a rate appropriate for age but with independent QRS complexes occurring at regular and slower rates than the atrial rate (junctional escape). Congenital complete heart block occurs in 1/20,000 live births in association with structural heart disease (e.g.,

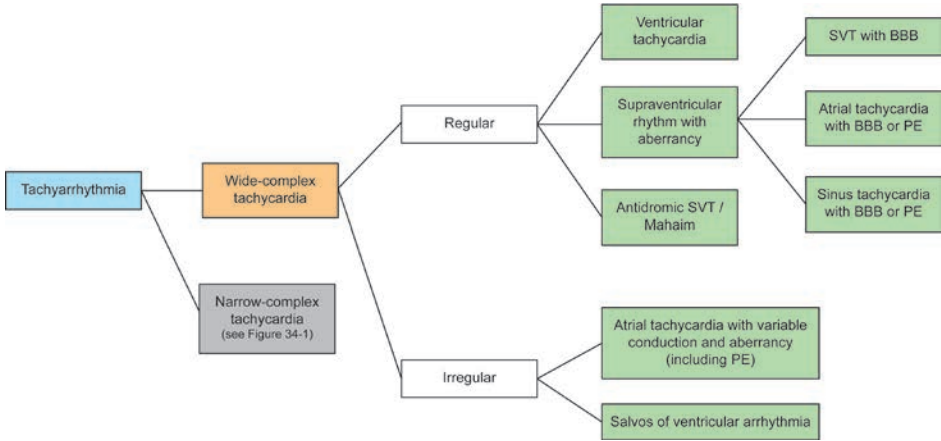


Figure 34-2. Diagnostic algorithm for wide-complex tachyarrhythmias. BBB: Bundle branch block, PE: Preexcitation.

L-transposition of the great vessels, heterotaxy syndrome with polysplenia/left atrial isomerism) or associated with maternal collagen vascular abnormalities (e.g., systemic lupus erythematosus, Sjögren syndrome). Postoperative surgical AV block may occur after some types of cardiac operations. Over 60% of patients usually recover normal conduction within the first 10 postoperative days. Permanent cardiac pacing is indicated for patients without recovery after 7 to 10 days postoperatively.

Supraventricular Arrhythmias

Premature atrial contractions (PAC) are relatively common in infants and small children and represent a benign phenomenon. Each QRS complex is preceded by a P wave that may have a normal axis or suggest an axis directed from outside the SA node.

Supraventricular tachycardia (SVT) represents the most common arrhythmia occurring in the pediatric population and is commonly divided into 2 main categories: reentrant and automatic. Both forms of SVT are characterized by a narrow or baseline QRS-complex morphology and can occur in structurally normal hearts as well as in various forms of CHD. Evaluation includes a 15-lead ECG and a continuous rhythm strip to evaluate onset, termination, and response to medications like adenosine or pacing maneuvers. Figure 34-1 displays a common diagnostic algorithm for classification of narrow-complex tachycardias, including SVT.

Common forms of narrow-complex tachycardias seen in infancy as well as adolescence include *atrioventricular reentrant tachycardia* (AVRT) and *atrioventricular nodal reentrant tachycardia* (AVNRT). AVRT is the most common type of SVT encountered in infancy/childhood and results from electrical signals crossing an accessory pathway of conduction tissue between the atria and ventricles. A cycle of conduction propagating normally down the AV node but returning to the atria via the accessory pathway creates the reentrant circuit which can be terminated with vagal maneuvers, adenosine,

or, in cases of poor perfusion and hemodynamic compromise, synchronized cardioversion (0.5-1 Joule/kg). Surface ECG will usually demonstrate P waves immediately after QRS complexes or within the ST segment or T wave (i.e., short RP tachycardia). AVNRT occurs most commonly in adolescents and young adults and is characterized by a similar reentrant circuit that occurs primarily within the AV node. Surface ECG makes discerning P waves difficult as they are often buried within the QRS complex. The management strategy for AVNRT is similar to AVRT.

Narrow complex tachycardias that result from automatic foci of electrical activity (e.g., *ectopic atrial tachycardia* [EAT], *multifocal atrial tachycardia* [MAT]) do not respond to electrical cardioversion and generally require pharmacologic therapies in addition to the avoidance of sympathetic stimulants like fever, pain/agitation, inotropic agents, and the correction of electrolyte imbalances.

Junctional ectopic tachycardia (JET) is a narrow-complex tachycardia that can occur with AV dissociation. Narrow QRS complexes occur at a rate faster than P waves due to an automatic focus of electrical activity within the AV node or junction. This arrhythmia often occurs in the perioperative setting as a result of cardiac manipulation and dissection around the RA. The distinction from accelerated junctional rhythm is based on heart rate (typically greater than 160 or 170 bpm) and hemodynamic status of the patient. When occurring in the postoperative period, JET is usually transient and self-limited, lasting from 24 to 72 hours but can result in hemodynamic instability, significant morbidity, and may contribute to mortality. Management is usually multimodal with focus on minimizing stimulation to the patient, avoidance of hyperthermia, use of antiarrhythmic medications, and temporary atrial pacing to overdrive the junctional rate. The use of ECMO should also be considered as a rescue modality for cases of JET refractory to conventional therapies.

Atrial fibrillation and *atrial flutter* are both less common in the general pediatric population yet are more often encountered in patients with CHD. Both conditions may benefit from the use of adenosine as a diagnostic maneuver to uncover underlying atrial activity (flutter waves vs. irregularly irregular atrial activity). Acute management of atrial fibrillation in the hemodynamically stable patient should focus on ventricular rate control and determination of underlying etiology. Normal sinus rhythm may also be restored with synchronized cardioversion after an appropriate evaluation for intracardiac thrombus. Synchronized cardioversion can also terminate atrial flutter, although atrial overdrive pacing can be utilized successfully as well.

Ventricular Arrhythmias

Premature ventricular contractions (PVC) result in early- and wide-QRS complexes without preceding P waves due to early activation of ventricular myocardium from an ectopic focus. In the patient with a structurally normal heart, PVCs of a single QRS morphology (uniform) without associated symptoms are generally considered benign. PVCs may have more significance if they are multifocal, occur in very high frequency, occur with symptoms of syncope, are accompanied by a family history of sudden death, or are associated with underlying heart disease.

Ventricular tachycardia (VT) is defined as 3 or more PVCs in series at a heart rate

>120 bpm in adults or more than 20% greater than the preceding sinus rate. The acute onset of VT may be due to hypoxia, acidosis, electrolyte/metabolic derangements, or in the context of depressed myocardial function, poor hemodynamics, prior surgical interventions, myocardial tumors, cardiomyopathies, myocarditis, acute injury (trauma), and primary channelopathies. Polymorphic VT in the form of “torsades de pointes” manifests as both positive and negative oscillations of the QRS complex around an isoelectric baseline. Polymorphic VT can be secondary to drug therapy, myocardial ischemia, neurologic injury, or may occur in long-QT syndrome. Cardioversion of torsades de pointes should be performed in sustained cases, although there is risk of degradation to ventricular fibrillation (VF). Magnesium sulfate should be administered as first line therapy.

Ventricular fibrillation (VF) results from asynchronous ventricular depolarizations that create chaotic and ineffective muscle contractions and loss of cardiac output. Immediate attention to the hemodynamic status of a patient is essential to the care of patients with VT and/or VF. CPR should be instituted in the unstable patient while cardiodefibrillator pads are placed in preparation for cardioversion/defibrillation (2-4 Joules/kg). Pharmacologic therapies including amiodarone and lidocaine may be indicated for the patient with stable VT or as part of the management of pulseless VT/VF as per the Pediatric Advanced Life Support algorithm (AHA guidelines).

Suggested Reading

Valdes SO, Kim JJ, Miller-Hance WC. Arrhythmias: Diagnosis and management. In: Andropoulos DB, Stayer S, Mossad EB, Miller-Hance WC (eds). *Anesthesia for Congenital Heart Disease*. 3rd ed. John Wiley and Sons; 2015.