Pediatric Dysrhythmias

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The overall incidence of arrhythmias is 13.9 per 100,000 emergency department (ED) visits and 55.1 per 100,000 pediatric ED visits (children under 18 years of age) [1]. Among children with arrhythmias, the most common dysrhythmias are sinus tachycardia (50%), supraventricular tachycardia (13%), bradycardia (6%), and atrial fibrillation (4.6%) [1].

The presentation of dysrhythmias can serve as a diagnostic challenge to most clinicians because most children present with vague and nonspecific symptoms such as “fussiness” or “difficulty feeding.” Despite the infrequency and vague presenting symptoms, it is critical to identify and appropriately manage these disorders. When left they are unrecognized and untreated, dysrhythmias can lead to cardiopulmonary compromise and arrest.

The electrocardiogram in pediatrics

The most common reasons for obtaining EKGs in children are chest pain, suspected dysrhythmias, seizures, syncope, drug exposure, electrical burns, electrolyte abnormalities, and abnormal physical examination findings. Of all of these, the most life-threatening findings are those caused by electrolyte disturbances, drug exposure, and burns [2].

Although a complete EKG interpretation is beyond the scope of this chapter, it is advisable to use a systematic approach, with special attention to rate, rhythm, axis, ventricular and atrial hypertrophy, and the presence of any ischemia or

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repolarization abnormalities. More specifically, it is essential to interpret pediatric EKG’s based on age-specific rates and intervals (Table 1) [3–5]. The EKG can be evaluated further for rhythm, chamber size, and T-wave morphology.

### Tachydysrhythmias

Tachycardia is defined as a heart rate beyond the upper limit of normal for the patient’s age. In adults, the heart rate is greater than 100 beats per minute (BPM). Tachycardias can be classified broadly into those that originate from loci above the atrioventricular (AV) node (ie, supraventricular), from the AV node (AV node reentrant tachycardias), and from the ventricle. The majority of tachycardias are supraventricular (SVT) in origin. Those that are ventricular in origin are associated typically with hemodynamic compromise [4]. When tachycardia is recognized, step-wise questioning can help evaluate the EKG tracing. Is it regular or irregular? Is the QRS complex narrow or wide? Does every P wave result in a single QRS complex? Once these have been established, the treatment options are considered according to whether the patient has a pulse and the presenting rhythm on EKG (Fig. 1) [6].

### Sinus tachycardia

Sinus tachycardia can be differentiated from other tachycardias by a narrow QRS axis and a P wave that precedes every QRS complex. The rate is usually greater than 140 BPM in children and greater than 160 BPM in infants. Sinus tachycardia is typically benign. The pulse rate has been shown to increase linearly with temperature in children older than 2 months of age. For every 1°C (1.8°F) increase in body temperature, the pulse rate increases by an average of 9.6 BPM [7]. Sinus tachycardia can also be associated with such underlying conditions as
Fig. 1. Tachycardia algorithm. PEA, pulseless electrical activity; SVT, supraventricular tachycardia; V-Fib, ventricular fibrillation; V-Tach, ventricular tachycardia. (Data from Hazinski M, Zaritsky A, Nadkarini V, et al. PALS provider manual. Dallas (TX): American Heart Association; 2002.)
hypoxia, anemia, hypovolemia, shock, myocardial ischemia, pulmonary edema, hyperthyroidism, medications (catecholamines), hypocalcemia, and illicit drug use. Most commonly, it is a result of dehydration and hypovolemia [1,4]. Because children augment cardiac output by increasing the heart rate rather than the stroke volume, heart rate increases appear early, whereas hypotension is a late sign of dehydration. Treatment aimed at correcting the heart rate alone may be harmful to the patient because the tachycardia is a compensatory response to sustain adequate cardiac output. For this reason, the treatment of sinus tachycardia is largely targeted at treating the underlying disorder, rather than treating the tachycardia itself.

**Ventricular tachycardia**

Although it is rare in children, ventricular tachycardia is an important rhythm to recognize and treat promptly. Nonperfusing ventricular rhythms are seen in up to 19% of pediatric cardiac arrests, when sudden infant death syndrome (SIDS) cases are excluded [8]. Although the heart may be contracting and pulses are palpable in some patients who have ventricular tachycardia, those contractions are hemodynamically inefficient and can lead ultimately to syncope and death if left untreated. Furthermore, ventricular tachycardia can decompensate into ventricular fibrillation, which is a nonperfusing, terminal arrhythmia.

Ventricular tachycardia may result from electrolyte disturbances (hyperkalemia, hypokalemia, and hypocalcemia), metabolic abnormalities, congenital heart disorders, myocarditis, or drug toxicity. Other causes include cardiomyopathies, cardiac tumors, acquired heart disease, prolonged QT syndrome, and idiopathic causes.

On electrocardiograms, the QRS complexes have a wide configuration. The QRS duration is prolonged, ranging from 0.06 to 0.14 seconds. Complexes may appear monomorphic with a uniform contour and absent or retrograde P waves. Alternatively, the QRS complexes may appear polymorphic or vary randomly as is seen in torsades de pointes. EKG findings that further support the presence of ventricular tachycardia include the presence of AV dissociation with the ventricular rate exceeding the atrial rate (Fig. 2).

In a patient who has ventricular tachycardia, the urgency of treatment depends on the patient’s clinical status. Initially, the airway, breathing, and circulation (ABCs) must be maintained, and it must be determined whether the patient has a pulse and is hemodynamically stable. The American Heart Association has set forth treatment algorithms [6] to facilitate prompt treatment for this potentially fatal rhythm (see Fig. 1).

Ventricular tachycardia with a pulse in an unstable patient warrants immediate synchronized cardioversion at 0.5 to 1 J/kg. It is important to pretreat conscious patients with light sedation (eg, midazolam, 0.1 mg/kg). Pharmacologic interventions include amiodarone (5 mg/kg intravenously [IV] over 20–60 min; maximum single dose, 150 mg; maximum daily dose, 15 mg/kg/d), procainamide (15 mg IV over 30–60 min), or lidocaine (1 mg/kg IV bolus, repeat every
5–10 min, with max total of 3 mg/kg) [9]. When using procainamide, the infusion is stopped once the arrhythmia resolves if the QRS complex widens to ≥50% over the baseline or if hypotension ensues. Pulseless ventricular tachycardia should be treated as ventricular fibrillation (see below).

After cardioversion, the return to normal sinus rhythm is usually transient. The medication used to achieve sinus rhythm must be given as a continuous infusion using lidocaine (20–50 μg/kg/min), amiodarone (7–15 mg/kg/d), or procainamide (20–80 μg/kg/min [maximum dose of 2 g/24 h]) [6]. In polymorphic ventricular tachycardia, temporary atrial or ventricular pacing may be required. Overall, the treatment goal is to keep the heart rate at less than 150 BPM in infants and less than 130 BPM in older children.

**Premature ventricular contractions**

A premature ventricular contraction (PVC) is a premature, wide QRS complex that has a distinct configuration and is not preceded by a P wave. They may appear in a pattern of two consecutive PVCs (couplet), an alternating PVC with a normal QRS complex (bigeminy), or in which every third beat is a PVC (trigeminy). The occurrence of three or more consecutive PVCs is considered ventricular tachycardia. The sinoatrial (SA) node maintains a normal conduction pace, and the PVC replaces a normal QRS wave while maintaining a rhythm.
Although most children who have PVCs are otherwise healthy, PVCs can also be associated with congenital heart disease, mitral valve prolapse, prolonged QT syndrome, and cardiomyopathies (dilated and hypertrophic). Malignant origins include electrolyte imbalances, drug toxicities (eg, general anesthesia, digoxin, catecholamines, amphetamines, sympathomimetics, and phenothiazines), cardiac injury, cardiac tumors, myocarditis (Lyme and viral diseases), hypoxia, and an intraventricular catheter.

For the most part, patients who have PVCs are asymptomatic; however, when they are unrecognized and untreated, there is a risk of developing ventricular tachycardia in patients who have a serious underlying cause. When they are examined, 50% to 75% of otherwise normal children may have PVCs seen on Holter monitoring [4]. It is crucial to determine whether the heart has an underlying pathology. This can be accomplished by history and physical examination, a 12-lead electrocardiogram, and a chest radiograph. If the findings of all of these tests are normal, no further investigation is necessary.

Premature ventricular contractions are considered malignant if they are associated with underlying heart disease; there is a history of syncope or family history of sudden death; precipitated by or increased with activity; exhibit multiform morphology; they are symptomatic of runs of PVCs; or there are frequent episodes of paroxysmal ventricular tachycardia. Children presenting with premature ventricular contractions require evaluation and possibly treatment in conditions that are likely to cause cardiopulmonary compromise. This occurs whenever there are two or more PVCs in a row, they are multifocal in origin, there is an “R-on-T” phenomenon, or if there is underlying heart disease. The R-on-T phenomenon is an instance in which a PVC occurs on the T wave, which is considered a vulnerable period of stimulating abnormal rhythms. This can be seen with hypoxia or hypokalemia and may result in life-threatening arrhythmias [10]. For those patients who have an underlying cause (eg, electrolyte abnormality, hypoxia, or severe acidosis), the treatment consists of managing the underlying cause. The treatment consists largely of IV lidocaine (1 mg/kg/dose), followed by a lidocaine drip (20–50 μg/kg/min). Amiodarone, procainamide, and β blockers are reserved for conditions that are refractory to lidocaine [11].

In asymptomatic patients who present with isolated PVCs and normal cardiac structure and function, no treatment is necessary. For patients who have couplets, multiform PVS, or frequent PVCs, a referral to a pediatric cardiologist for further investigation is indicated. It is prudent to advise any patient who has PVCs to avoid stimulants such as caffeine, theophylline, and pseudoephedrine because the stimulants may precipitate more frequent PVCs.

**Ventricular fibrillation**

Ventricular fibrillation is an uncommon rhythm in the pediatric population but is certainly life threatening. The hallmark is chaotic, irregular ventricular
contractions without circulation to the body. On the electrocardiogram, the rhythm is one of bizarre QRS complexes with varying sizes and configurations and a rapid, irregular rate. Causes of ventricular fibrillation include postoperative complications from congenital heart disease repair, severe hypoxemia, hyperkalemia, medications (digitalis, quinidine, catecholamines, and anesthesia), myocarditis, and myocardial infarction.

Ventricular fibrillation is an uncommon cause of cardiac arrest in infants less than 1 year of age but increases with growing age. With the increasing use and efficacy of automated external defibrillators (AEDs) in the adult setting, controversy has arisen in the use of AEDs in the prehospital treatment of children in cardiac arrest. Currently, AEDs are approved for patients older than 8 years of age [8]. However, according to the American Heart Association International Liaison Committee on Resuscitation, AEDs may be used in 1- to 8-year-old children who have no signs of circulation, ideally with the pediatric dose. For a lone rescuer responding to a child who does not have signs of circulation, cardiopulmonary resuscitation (CPR) for 1 minute is recommended before attaching an AED or activating emergency medical services. For documented ventricular fibrillation or pulseless ventricular tachycardia, defibrillation is recommended [8].

Because ventricular fibrillation is a nonperfusing rhythm, CPR must be initiated immediately. Of note, ventricular fibrillation is treated the same as ventricular tachycardia without a pulse. Defibrillation is initiated at 2 J/kg, increased from 2 to 4 J/kg, and then followed by a third shock at 4 J/kg. If defibrillation is unsuccessful, epinephrine (0.01 mg/kg, 1:10,000 solution) should be given and repeated every 3 to 5 minutes as necessary.

If pulseless ventricular tachycardia is refractory to defibrillation, antiarrhythmic drugs are indicated, such as amiodarone (5 mg/kg, IV bolus) or lidocaine (1 mg/kg, IV bolus, and repeated to a maximum of 3 mg/kg). Although the pediatric dosing of amiodarone has not been clearly established, the recommended loading dose of 5 mg/kg IV may be given over 20–60 minutes. If rate control is not achieved, the dose may be repeated in increments of 5 mg/kg IV, to a maximum of 15 mg/kg/d IV [12]. For polymorphic ventricular tachycardia (torsades des pointes), the mainstay of treatment is magnesium (20–50 mg/kg, IV).

Supraventricular tachycardia

SVT is the most common symptomatic dysrhythmia in infants and children. In newborns and infants who have SVT, the heart rate is greater than 220 BPM. In older children, it is defined as having a heart rate of more than 180 BPM [13]. The ECG shows a narrow complex tachycardia, either without discernible P waves or with retrograde P waves with an abnormal axis (Fig. 3). The QRS duration is normal but is occasionally increased with aberrancy. It is further characterized by little or no variation in the heart rate.
There are three types of supraventricular tachycardia. The most common is the AV reentrant tachycardia phenomenon. In addition to the normal conduction from the SA node to the AV node to the bundle of His to the Purkinje fibers, there is an accessory “bypass” pathway in conjunction with the AV node. This pathway is an anatomically separate bypass tract, such as the bundle of Kent, which is seen in Wolff-Parkinson-White (WPW) syndrome. Conduction through this accessory pathway occurs more rapidly than through the normal conduction pathway, creating a cyclic pattern of reentry independent of the SA node. Typical ECG findings of WPW are a short PR interval, a wide QRS, and a positive inflection in the upstroke of the QRS complex, known as the delta wave (Fig. 4). This characteristic finding is evident only after the rhythm is converted to a sinus rhythm [14].

The second type of SVT is the AV nodal, or junctional, tachycardia, which is a cyclical reentrant pattern from dual AV node pathways that are depolarized simultaneously. The third type of SVT, ectopic atrial tachycardia, is rare and is manifested by the rapid firing of a single ectopic focus in the atrium. The hallmark of ectopic atrial tachycardia is the presence of different P-wave morphologies. Each P wave is conducted to the ventricle and, because the ectopic atrial focus is faster than the SA node, it takes over the rate determination [4].

The majority of infants with SVT present at less than 4 months of age, in a male-to-female ratio of 3:2 [15]. Among this group, almost half of the conditions have an idiopathic cause, whereas 24% are associated with conditions such as
fever and drug exposure, 23% are caused by congenital heart disease (most commonly Ebstein’s anomaly, single ventricle, L-transposition), and 10% to 20% are the result of WPW syndrome [4]. Among older children, causes are more likely to be WPW, concealed bypass tracts, or congenital heart disease. The AV reentrant type of tachycardia is more common in children less than 12 years of age, whereas the AV node type of tachycardia becomes more evident in adolescents [16]. Other causes include hyperdynamic cardiac activity as is seen in response to catecholamine release, drug use, and postoperative cardiac repair. Toxic causes of SVT include stimulants, β agonists, anticholinergics, salicylates, theophylline, tricyclics, and phenothiazines. Nontoxic causes include anxiety, anemia, sedative and ethanol withdrawal, dehydration, acidosis, exercise, fever, hypoglycemia, hypoxemia, and pain [13].

The diagnosis often begins in triage where the nurse reports a heart rate that is “too fast to count.” In newborns and infants who present with SVT, the heart rate is often between 220 and 280 BPM [13]. Most patients do not have an underlying cause to account for the tachycardia, such as fever, dehydration, fluid or blood loss, anxiety, or pain. Infants often present with nonspecific complaints such as “fussiness,” lethargy, poor feeding, pallor, sweating with feeds, or simply “not acting right.” If congestive heart failure (CHF) is present, caretakers may describe pallor, cough, and respiratory distress. Although many infants can tolerate SVT well for 24 hours, within 48 hours, 50% of them will develop heart failure and may deteriorate rapidly [13]. In contrast, CHF rarely occurs in older children, who are usually able to describe palpitations, chest pain, dizziness, or shortness of breath. Important historical factors include a relationship to exercise, meals,
stress, color changes, neurologic changes, or syncope. A medical history significant for cardiac problems, current medications, allergies, or a family history of sudden death or cardiac disease should be investigated.

The management of SVT always begins with ensuring that the patient is maintaining airway, breathing, and cardiovascular status. It is important to promptly administer oxygen and to obtain a 12-lead EKG with a rhythm strip. It is of utmost importance, to expeditiously differentiate between patients who are stable and those who are unstable. In a child presenting with unstable SVT with severe heart failure and poor perfusion, synchronized cardioversion is initiated at 0.5 J/kg and can be increased up to 1 J/kg. Adenosine may be given before cardioversion if intravenous access has already been established. In unstable patients, cardioversion should not be delayed for attempts at IV access or sedation [6].

In children who present with asymptomatic SVT or with mild heart failure, vagal maneuvers such as ice to the face in an infant or blowing through a straw in an older child may be attempted [16]. If that is unsuccessful, adenosine is administered through an IV that is preferably close to the heart. Because of its extremely short half-life, adenosine must be pushed and flushed (with 5 cc normal saline) quickly, to be effective. The initial dose of adenosine is 0.1 mg/kg (up to 6 mg) and can be increased to 0.2 mg/kg/dose (up to 12 mg) if the first dose is ineffective [9]. An effective response is a brief period of asystole on EKG, with the return of a normal sinus rhythm. Failure to terminate the dysrhythmia after the second dose of adenosine in a stable patient should prompt consultation with a pediatric cardiologist. Adenosine can be therapeutic as well as diagnostic; however, it is not effective with nonreciprocating atrial tachycardia, atrial flutter, atrial fibrillation, or ventricular tachycardia. There are minimal hemodynamic consequences associated with adenosine administration [17]. Contraindications include a deinnervated heart (eg, transplant) and second- or third-degree heart block. Additionally, adenosine can worsen bronchospasm in asthmatics and increase heart block or precipitate ventricular arrhythmias in those taking carbamazepine, verapamil, or digoxin.

Alternative medications include procainamide (15 mg/kg, IV, over 30–60 min or at 20–80 μg/kg/min), amiodarone (5 mg/kg over 20–60 min, with a maximum single dose of 150 mg and a maximum daily dose of 15 mg/kg). Of note, amiodarone should not be used in newborns during the first month of life because it contains the preservative benzyl alcohol that has been associated with a gasping syndrome, which is characterized by metabolic acidosis and the “striking onset of gasping respiration, hypotension, bradycardia, and cardiovascular collapse” [18,19]. β blockers such as propranolol or esmolol may be used but with caution because they may induce hypotension [20]. In addition, verapamil should be avoided in children less than 1 year of age because cardiovascular collapse and death can occur [13]. Further precautions should be taken in the use of digoxin because it may act as a proarrhythmic agent in SVT associated with WPW. The long-term management of SVT may include β blockers, procainamide, sotalol, amiodarone, or flecainide. When pharmacologic treatments fail, radiofrequency
Catheter ablation has an 85% to 95% success rate of preventing recurrence of SVT [21].

The evaluation of SVT includes attempts at elucidating the cause of SVT to prevent future episodes. Laboratory studies may include electrolytes (especially potassium, calcium, magnesium, and glucose), complete blood count, toxicology screen, blood gas, and thyroid function tests. Additionally, creatine kinase and troponins may be added if myocarditis is suspected. Imaging studies can include a chest radiograph (including anteroposterior and lateral views) and an echocardiogram. Once stabilized, the majority of patients who present with SVTs will need to be admitted to a hospital, to investigate the underlying cause of SVT and the potential for long-term medical management or radiofrequency ablation. One could consider safely discharging a patient who has a history of SVT, has presented with minor symptoms such as palpitations, or has had a clear precipitant.

**Atrial flutter**

Atrial flutter is an uncommon rhythm presenting in the pediatric population. Atrial rates may present in the range of 240 to 450 BPM [4], with the ventricular response depending on the AV nodal conduction. The pacemaker lies in an ectopic focus.

Causes of atrial flutter in children are attributed largely to structural heart disease, including a dilated atria, myocarditis, or acute infection. It is associated most notably with postoperative complications of congenital heart disease repairs, such as atrial septal defect (ASD) repairs, the Mustard procedure for D-transposition of the great arteries, or the Fontan procedure for single ventricle. These procedures cause atrial flutter through disruption in the conduction system, as happens when there is suturing through the atrial septum. Occasionally, patients who have undergone ventricular surgeries such as tetralogy of Fallot repairs may present with atrial arrhythmias. Atrial flutter is also seen in such conditions as Duchenne’s muscular dystrophy and central nervous system injury.

On an electrocardiogram, the hallmark pattern is “saw-toothed” flutter waves, which is best viewed in leads II, III, and V1. The atrial rate is, on average, approximately 300 atrial BPM [3]. Because the AV node cannot respond this quickly, there is an AV block, which can present as a 2:1, 3:1, or 4:1 block. The QRS complex is generally normal in configuration (Fig. 5).

Significant cardiac pathology usually accompanies atrial flutter. Because cardiac output is determined by the ventricular rate, with atrial flutter, the ventricular rate is too fast to maintain an efficient cardiac output. Atrial arrhythmias are an important cause of morbidity and mortality in those with congenital heart disease.

Initially, the clinician must recognize whether the patient is hemodynamically stable. An unstable patient may warrant electrical cardioversion, with the con-
sideration of adding heparin to prevent embolization [3,22]. In patients who are receiving digoxin, it is advisable to avoid electrical cardioversion, unless the condition is life threatening, because the combination is associated with malignant ventricular arrhythmias [23,24]. Alternatives for patients receiving digoxin are rapid atrial pacing with catheterization or lower current settings [22]. For patients who are hemodynamically stable, digoxin is administered to increase AV blockade, thereby slowing the ventricular rate. Propranolol, 1.0 to 4.0 mg/kg/d, orally, divided three to four times daily, may also be added. Recurrences are then prevented, by administering Quinidine.

**Atrial fibrillation**

Atrial fibrillation is another rare rhythm that presents in children. It is defined as disorganized, rapid atrial activity, with atrial rates ranging from 350 to 600 BPM [11]. The ventricular rate is variable and depends on a varying AV block. The rhythm of atrial fibrillation is described as being “irregularly irregular,” alternating between fast and slow rates. On electrocardiography, the hallmark features are irregular atrial waves, with beat-to-beat variability of the atrial size and shape. This is best recognized in lead V1. The QRS complexes appear normal. Children at an increased risk of developing atrial fibrillation include those who have an underlying structural heart defect (such as congenital mitral valve disease and hyperthyroidism) and those who have undergone an intra-atrial operative procedure. Atrial fibrillation is also associated with
decreased cardiac output. With a significantly increased ventricular rate, incoordination ensues between the atria and ventricles, thereby decreasing cardiac output.

When the child presents to the emergency department, the clinician must promptly recognize whether he or she is hemodynamically stable or has cardiac compromise. Hemodynamically unstable patients warrant immediate cardioversion. However, in patients who are hemodynamically stable, digoxin can be administered for ventricular rate control, allowing for a 24-hour time period to assess its efficacy. After that time period elapses and digoxin proves to be ineffective, a second medication may be added such as propranolol, esmolol, or procainamide. In patients who have undergone cardioversion, recurrence is common. During admission, cardioverted patients are often started on an agent to keep them in normal sinus rhythm (eg, amiodarone, procainamide, quinidine, or a β blocker) [25].

**Bradydysrhythmias**

Bradycardia is defined as a heart rate slower than the lower limit of normal for the patient’s age (Table 1), whereas in adults, it is defined as a heart rate less than 60 BPM. Mechanisms of bradycardia include depression of the pacemaker in the sinus node and conduction system blocks. Complete heart block is a common cause of significant bradycardia in pediatric patients and may be acquired or congenital.

Bradycardia in children may be attributable to vagal stimulation, hypoxemia, acidosis, or an acute elevation of intracranial pressure. The most common cause of bradycardia in the pediatric population is hypoxemia. It is important to correct hypoxemia before increasing the heart rate in children.

The management of bradycardia includes the identification of the cause and appropriate cardiopulmonary resuscitation, with assisted ventilation, oxygenation, and chest compressions as indicated. If symptomatic bradycardia persists despite initial resuscitative measures, pharmacologic intervention is initiated with epinephrine (0.01 mg/kg IV; 0.1 mL/kg of 1:10,000 solution) or atropine (0.02 mg/kg, IV, minimum 0.1 mg; maximum single dose is 0.5 mg in children and 1 mg in adolescents). Epinephrine is the initial drug of choice in children with symptomatic bradycardia. Chest compressions are indicated for neonates or children with heart rates less than 60 BPM with hemodynamic compromise [6].

**Sinus bradycardia**

Sinus bradycardia, includes a heart rate less than the lower limit of normal for the patient’s age (see Table 1), with P waves preceding each QRS complex on an EKG. Usually, the heart rate is less than 80 BPM in infants and less than 60 BPM
in older children [26]. Sinus bradycardia is a predominantly benign entity, seen most often in athletes and during sleep.

Sinus bradycardia can also be associated with underlying causes. One such ominous cause is an acute onset of increased intracranial pressure as part of Cushing’s triad of bradycardia, hypertension, and irregular respirations. An important cause of bradycardia is respiratory compromise. Therefore, the adequacy of the patient’s oxygenation and ventilation should be assessed rapidly. Bradycardia can also be associated with hyperkalemia, hypercalcemia, hypoxia, hypothermia, hypothyroidism, and medications (eg, digitalis and β blockers). As with sinus tachycardia, the treatment of sinus bradycardia is targeted at the treatment of the underlying cause.

An important distinction must be made between sinus bradycardia and junctional (nodal) bradycardia. On electrocardiography, junctional bradycardia has either no P waves or inverted P waves after QRS complexes. QRS complexes have a normal configuration and generally have rates between 40 and 60 BPM. Junctional bradycardia may occur in an otherwise normal heart or postoperatively, in cases of digitalis toxicity, or with increased vagal tone. If the patient is asymptomatic, no treatment is indicated. However, if the patient has signs of decreased cardiac output, atropine or pacing may be indicated [4].

**Conduction abnormalities**

**First-degree atrioventricular block**

First-degree AV block is an abnormal delay in conduction through the AV node. This type of AV block is a disturbance in the conduction between the normal sinus impulse and its eventual ventricular response. This manifests as a prolonged PR interval on electrocardiography. Meanwhile, the heart is maintained in sinus rhythm, with a normal QRS configuration. There are no dropped beats.

First-degree heart block can be an incidental finding on an otherwise normal EKG reading. Common causes include otherwise healthy children with an infectious disease. It may further be associated with myocarditis (eg, rheumatic fever and Lyme disease), cardiomyopathies, and congenital heart disease (ASD and Ebstein’s anomaly).

**Second-degree atrioventricular block: Mobitz type I (Wenckebach) and type II**

In the Mobitz type I block, otherwise known as the Wenckebach phenomenon, the PR interval lengthens progressively until a QRS complex is dropped. This usually occurs over three to six cardiac cycles, followed by a long diastolic pause, and then the cycle resumes. There are occasional and frequent P waves that conduct, and the QRS configuration is normal. The block is caused by an increased refractory period at the level of the AV node. Although this can be seen in otherwise healthy individuals, it can also be seen in patients who have
myocarditis, myocardial infarctions, cardiomyopathies, congenital heart disease, digoxin toxicity, and postoperative cardiac repairs.

The Mobitz type II second-degree heart block is known as the “all or none” phenomena. There is either normal AV conduction with a normal PR interval or a completely blocked conduction. The failure of conduction is at the level of the bundle of His, with a prolongation of the refractory period in the His-Purkinje system. Because some of the atrial impulses are not conducted to the ventricle, the ventricular rate depends on the number of conducted atrial impulses.

**Third-degree heart block**

Third-degree heart block, otherwise known as complete heart block, occurs when none of the atrial impulses is conducted to the ventricles. There is a complete loss of rhythm conduction from a working atrial pacemaker, thereby allowing the ventricular pacemaker to take over. On electrocardiography, the P waves are completely dissociated from the QRS waves. Even though they are dissociated, both the atrial and ventricular rhythms are regular, maintaining regular PP and RR intervals, respectively. The QRS duration is usually normal if the block is usually proximal to the bundle of His, whereas a wide QRS complex indicates that the block is most likely in the bundle branches (eg, surgically induced complete heart block). Oftentimes, the ventricular rhythm is slower than normal (Fig. 6).
Complete heart block may be an isolated anomaly. It may also be congenital and is associated with structural lesions, such as in L-transposition of the great arteries and maternal connective tissue disorders. Acquired heart block may result from cardiac surgery, especially when there is suturing in the atrium. This effect can be either transient, resolving within 8 days postoperatively, or permanent. Other causes include infectious causes such as myocarditis, Lyme disease, rheumatic fever, and diphtheria, and inflammatory disorders such as Kawasaki disease and systemic lupus erythematosus. Complete heart block is also associated with myocardial infarction, cardiac tumors, muscular dystrophies, hypocalcemia, and drug overdoses.

Children presenting with first-degree heart block are largely asymptomatic but have the potential to progress to further heart block, including second- and third-degree heart blocks. Those presenting with second-degree, type I (Wenckebach), rarely progress to complete heart block, whereas second-degree, type II, block frequently progresses to complete heart block [4]. Those children who present with complete heart block, most notably in infancy, may present with signs of congestive heart failure. Older children may present with syncopal attacks, otherwise known as Stokes-Adams attacks, with heart rates less than 40 to 45 BPM or even sudden death.

Patients who have complete heart block may present with symptoms related to hypoperfusion, including fatigue, dizziness, impaired exercise tolerance, syncope, confusion, and even sudden death [27]. Acquired or surgically induced heart block generally has a slower ventricular rate, with rates between 40 and 50 BPM, than is seen in congenital heart block, which is generally 50 to 80 BPM [16].

No treatment is indicated for a first-degree degree heart block. However, if suspicious features are present, patients may require evaluation for underlying disease (eg, Lyme disease or rheumatic fever). For second-degree heart blocks, treatment is directed at the underlying cause. In patients who have Mobitz type II second-degree heart block, a prophylactic pacemaker may be warranted because there is a risk of progressing to complete heart block. For those who present with a complete heart block, the mainstay of therapy is a pacemaker. While awaiting pacemaker insertion, it may be necessary to administer atropine or isoproterenol, which temporarily increases the heart rate.

**Prolonged QT syndrome**

Prolonged QT syndrome, otherwise referred to as long QT syndrome (LQTS), is a disorder of delayed ventricular repolarization, characterized by prolongation of the QT interval, as seen on electrocardiography. Prolongation of the QT interval may be either hereditary or acquired. Jervell-Lange-Nielsen syndrome is an autosomal recessive form of prolonged QT syndrome associated with congenital deafness, whereas Romano-Ward syndrome is an autosomal dominant form that is not associated with deafness. Congenital LQTS, which often presents in
childhood, has an estimated incidence of 1 per 10,000 to 1 per 15,000 and is responsible for 3000 to 4000 cases of sudden death each year in the United States. Patients with the acquired type of LQTS usually present in the fifth or sixth decades of life [28]. The most common causes are medications (Box 1) and electrolyte abnormalities such as hypokalemia, hypocalcemia, and hypomagnesemia.

Patients with LQTS commonly present between the ages of 9 and 15 years of age with recurrent episodes of near or frank syncope [29]. Of the patients who have acquired LQTS, 60% of affected individuals are symptomatic at diagnosis [30]. Syncopal episodes are often precipitated by intense emotion, vigorous physical activity, or loud noises. Syncopal episodes may be mistaken for seizures because they can result in the loss of consciousness, tonic-clonic movements, and temporary residual disorientation following the event. A spontaneous return of consciousness usually follows a syncopal episode, but the dysrhythmia has the potential to degenerate into ventricular fibrillation and sudden death. Approximately 10% of children with LQTS present with sudden death, with the youngest children being more likely to die suddenly [31]. LQTS may present in infancy as SIDS or later in life as incidents of near-drowning. Children may also present with milder symptoms such as diaphoresis, palpitations, or lightheadedness.

When a patient presents with syncope, there are a number of historical factors that should be viewed as warning signs of potential cardiac disease and sudden death. Syncope that occurs with exertion is almost always an ominous sign. The strongest risk factors for developing malignant dysrhythmias or sudden death include a history of syncope.

The hallmark dysrhythmia of LQTS, is a polymorphic ventricular tachycardia known as torsades de pointes (“twisting of the points”), a French term first used in 1966 by Dessertenne to describe a QRS axis shifting back and forth around the baseline (Fig. 7) [32]. During this dysrhythmic episode, the cardiac output is markedly impaired, often resulting in syncope or seizures. Although many of these events are self-limiting, with the spontaneous return of consciousness, the dysrhythmia has the potential to degenerate into ventricular fibrillation and

### Box 1. Drugs that prolong the QT interval

- Antiarrhythmics (class 1A and 3)
- Antiemetic (droperidol)
- Antifungals (ketoconazole)
- Antihistamines (astemizole, terfenidine)
- Antimicrobials (erythromycin, trimethoprim-sulfamethoxazole)
- Antipsychotics (haloperidol, risperidone)
- Organophosphate insecticides
- Phenothiazines (thioridazine)
- Promotility agents (cisapride)
- Tricyclic antidepressants (amitriptyline)
sudden death. With the potential for fatal consequences in undiagnosed affected individuals, the recognition of LQTS is of paramount importance.

LQTS should be considered and an EKG be obtained on any patient presenting with a suggestive history, including first-degree relatives of known LQTS carrier, a family history of syncope, seizures, sudden death, SIDS, a seizure of unknown cause, or an unexplained near-drowning. Other risk factors include congenital deafness or bradycardia in infants. Suggestive features of syncopal episodes include those that are triggered by emotion, exertion, or stress or those associated with chest pain or palpitations. The QT interval should be measured manually, with lead II generally accepted as being the most accurate. To account for the normal physiologic shortening of the QT interval that occurs with increasing heart rate, the corrected QT interval (QTc) is calculated using the Bazett formula $QTc = \frac{QT}{\sqrt{RR}}$. For the greatest accuracy, three consecutive QT intervals and three consecutive preceding RR intervals should be measured and averaged. The current practice identifies a QTc interval of $\geq 460$ ms as prolonged. A QTc value between 420 and 460 ms is considered borderline and warrants additional assessment. Although EKGs automatically calculate the QT and QTc, in patients who have a suggestive history, an EKG with a manual calculation of the QTc should be performed because the computer calculation often is inaccurate. If the diagnosis of LQTS is suspected but the screening EKG is not diagnostic, increasing sympathetic activity such as with vagal maneuvers may trigger abnormalities on electrocardiography. These abnormalities include QT interval prolongation, prominent U waves, T-wave alternans, and ventricular dysrhythmias.

Patients presenting with LQTS may require emergency intervention. Patients presenting with an episode of polymorphic ventricular tachycardia or torsades de pointes of unknown origin should receive magnesium (25–50 mg/kg, IV, maximum 2 g). Serum electrolytes and a toxicology screen should be obtained. β blockers may be useful in suppressing catecholamine surges and any further dysrhythmic activity. Patients who exhibit torsades des pointes caused by prolonged QT may worsen acutely, whereas those who have a normal QT interval improve. Patients with recurrent ventricular tachycardia may require temporary transcutaneous ventricular pacing.

Any patient who has a compatible history, a borderline prolongation of the QT interval with symptoms, or an identified prolonged QT syndrome should be referred to a cardiologist for further management. Admission is limited to those
who are symptomatic or have cardiovascular compromise. Therapy is aimed at reducing sympathetic activity of the heart, either pharmacologically or surgically. β blockers are generally recommended as the initial therapy of choice, which has been shown to effectively eradicate dysrhythmias in 60% of patients and to decrease mortality from 71% in untreated patient to 6% in those who are treated [28,36]. The most commonly used β blockers are propranolol (2–4 mg/kg/d, maximum 60 mg/d) and nadolol (0.5–1 mg/kg/d, maximum 2.5 mg/kg/d). Patients with severe asthma, in whom β blockers are contraindicated, may be candidates for pacemaker therapy.

Once a patient is diagnosed with LQTS, an EKG should be performed on all other family members. All affected individuals regardless of age should be restricted from competitive sports but not necessarily recreational sports. Patients should be educated to avoid triggering factors such as certain medications, loud noises, emotionally stressful situations, and dehydration. Because of the high risk of unexpected cardiac events, family members and close friends should be instructed in CPR and even consider purchasing a home AED.

Summary

In an acute care setting, it is necessary to quickly determine which pediatric ECG findings are normal, which are abnormal, and which must be addressed.
immediately. A systematic approach to the ECG is essential so that subtle abnormalities are less likely to be missed. A continuous review of the pediatric advanced life support algorithms is imperative in order for the emergency physician to care for children with dysrhythmias.

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References


