Demystifying the Pediatric Electrocardiogram: Tools for the Practicing Pediatrician

Ellis Rochelson, MD,* Taylor S. Howard, MD,† Jeffrey J. Kim, MD†

*Division of Pediatric Cardiology, Children's Hospital at Montefiore, Bronx, NY
†Department of Pediatrics, Lillie Frank Abercrombie Section of Pediatric Cardiology, Texas Children's Hospital, Baylor College of Medicine, Houston, TX

EDUCATION GAP

The pediatric electrocardiogram is an important tool in the screening and diagnosis of congenital heart disease, cardiomyopathy, and arrhythmias. Interpretation of the pediatric electrocardiogram can be challenging due to changes in normal findings as a patient ages.

OBJECTIVE *After completing this article, readers should be able to:*

- Interpret a pediatric electrocardiogram and identify key abnormalities that warrant further evaluation.

INTRODUCTION

The first recording of the electrical activity of the heart—an electrocardiogram (ECG)—was performed and published by Dr Augustus Waller in 1887. (1) Dr Waller's work was further advanced by Dr Willem Einthoven, who created a 3-lead ECG machine and was awarded the Nobel Prize in Physiology and Medicine in 1924 for his work. Initially used solely for detection of arrhythmias, the ECG was soon recognized as an important tool for detecting other abnormalities, (2) for which it remains an essential tool in modern clinical medicine. During the subsequent 90 years or more, ECG technology improved significantly, with advances in tracing quality, portability, digitization, and interpretation using artificial intelligence.

In pediatrics, in addition to its important function in the diagnosis of arrhythmias (its primary purpose), the ECG plays a key role as a screening tool for various cardiac pathologies. Careful interpretation of the pediatric ECG can aid in the detection of congenital heart disease (CHD), cardiomyopathy, pericarditis, myocarditis, conduction disturbances, and inherited arrhythmia syndromes. Accurate interpretation of a pediatric ECG requires knowledge of the "red flags" for these conditions, as well as an understanding of the normal evolution of the ECG throughout a child's life.

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ABBREVIATIONS

AV	atrioventricular
CHD	congenital heart disease
EAT	ectopic atrial tachycardia
ECG	electrocardiogram
LQTS	long QT syndrome
LVH	left ventricular hypertrophy
PAC	premature atrial contraction
PVC	premature ventricular contraction
RBBB	right bundle branch block
RVH	right ventricular hypertrophy
SVT	supraventricular tachycardia
VT	ventricular tachycardia
WPW	Wolff-Parkinson-White

HOW AN ECG SIGNAL IS MADE

A modern ECG is performed using 10 electrodes connected to the surface of the skin. There are 4 limb electrodes grouped into pairs to measure electrical gradients. The 6 precordial leads—V_1 through V_6—are placed on the chest and wrap around to the left midaxillary region. In pediatrics, there are often 3 additional precordial electrodes used (V_3R, V_4R, and V_7) to allow for increased sensitivity for cardiac malposition and rotation related to CHD.

It is helpful to think of each lead on an ECG as a viewing angle of the heart's electrical activity (Fig 1). When the net balance of current toward that viewing angle is positive (net balance of current heads toward the lead), this is represented as an upward deflection in that lead. (3)

The Basics: What Means What

The Y-axis of an ECG measures voltage generated by myocardial depolarization. The standardization marker on the leftmost aspect of an ECG indicates how many vertical millimeters represent 1 mV (most commonly 10 mm = 1 mV). The X-axis of an ECG measures time. Standard ECG "paper speed" is 25 mm per second, and 1 horizontal mm represents 40 milliseconds.

The components of an ECG are named in alphabetical fashion: PQRST (Fig 2). The P wave represents atrial depolarization. The PR interval, measured from the beginning of the P wave to the beginning of the QRS complex, reflects the passage of the cardiac impulse through the atria and atrioventricular (AV) node. The QRS complex represents ventricular depolarization, and the T wave represents ventricular repolarization. The QT interval, measured from the beginning of the QRS complex to the end of the T wave, reflects the time required for ventricular depolarization and repolarization to occur.

Approach to ECG Reading

When reading a pediatric ECG, a systematic approach is advisable. We suggest the following approach: rate, rhythm, axis, intervals, forces, ST segments, and T waves.

There are several publications that have attempted to establish normal reference values for pediatric ECGs. (4)(5)(6) Most of these papers, however, have included small sample sizes and homogeneous populations, often including few nonwhite participants. Work to create a more informed understanding of norms across ethnicities is underway, (7) although limitations continue to exist. In the near future, automated interpretation may assign *z* scores to ECG data to provide more accurate screening of abnormal values. (8)

Rate. Because 1 large box (5 mm) is equivalent to a rate of 300 bpm (at standard paper speed), the heart rate can be quickly estimated as follows: 300 divided by the number of large boxes between QRS complexes (Fig 3A). Of note, in clinical practice, the average heart rate provided by automatic ECG interpretation is fairly accurate, although it should not be trusted in cases of irregular rhythms or intermittent arrhythmias.

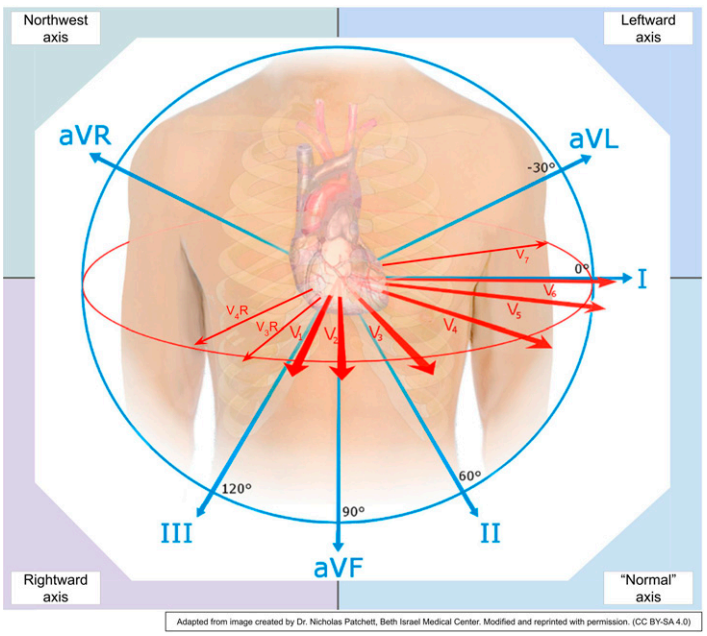

Figure 1. Electrocardiogram vectors, including additional pediatric leads (V_3R, V_4R, and V_7).

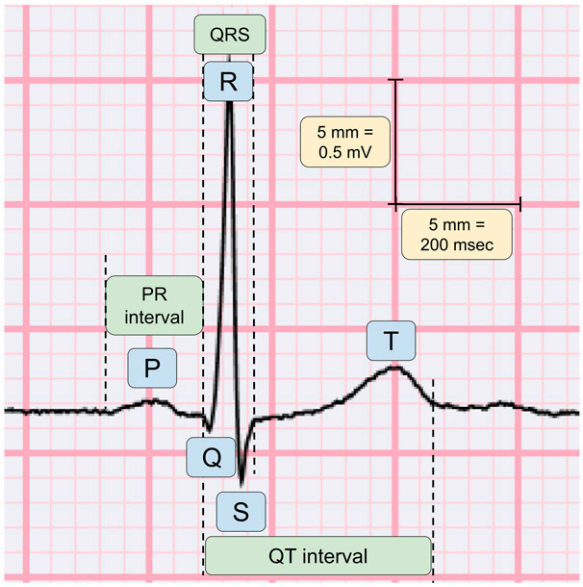

Figure 2. Electrocardiogram components.

The normal expected heart rate varies by age. In general, a normal resting heart rate is 100 to 200 bpm in infants, 75 to 150 bpm in children 2 to 8 years of age, and 50 to 120 bpm in older children. These "normal values" must be interpreted regarding the patient's clinical context. For example, in an irritable infant, sinus rates of up to 230 bpm may be seen.

Sinus bradycardia, if asymptomatic, is typically a benign phenomenon in all pediatric age groups, with the notable exception of the immediate newborn period in which neonatal resuscitation may be indicated for severe bradycardia. (9) In older neonates, infants, and children, asymptomatic sinus bradycardia carries a much more benign prognosis. Sinus bradycardia is particularly common in athletes due to high vagal tone. (10)

If the heart rate is faster than what is expected for age, a tachyarrhythmia should be considered. Causes of common pediatric tachyarrhythmias, such as supraventricular tachycardia (SVT), are discussed in a later section.

Rhythm. To evaluate the rhythm of an ECG means to determine what drives the electrical impulse. There are cardiac pacemaker cells with independent automaticity throughout the heart, including the sinoatrial (sinus) node, atrial myocytes, the AV node/His bundle, Purkinje fibers, and ventricular myocytes (Fig 4). A maxim of cardiac electrophysiology is that "the fastest cells win"—in the absence of conduction deficits, whatever pacemaker cells are firing the fastest will drive the rhythm.

Normally, the impulse is driven by the sinus node. Located near the junction of the right atrium and the superior vena cava, the sinus node initiates conduction that spreads across the atria. This direction of electrical propagation leads to a P-wave axis between 0° and 90°. (The concept of "axis" is further described in the subsequent section.) The ECG can, therefore, typically be classified as sinus rhythm if there is a P wave preceding every QRS complex and this P wave is upright in leads I and aVF. If the P-wave axis is abnormal, this may suggest an atrially driven rhythm from a site other than the sinus node, such as left atrial rhythm (inverted P wave in lead I), low atrial rhythm (inverted P-wave in aVF), or a wandering atrial pacemaker (multiple P-wave morphologies). An abnormal P-wave axis may also be seen in various forms of CHD.

Importantly, the most common cause of an abnormal P-wave axis is limb lead reversal—when the right arm lead is mistakenly placed on the left arm, and vice versa. In this scenario, the P wave will be inverted in lead I (along with right QRS axis deviation and T-wave inversion). To diagnose

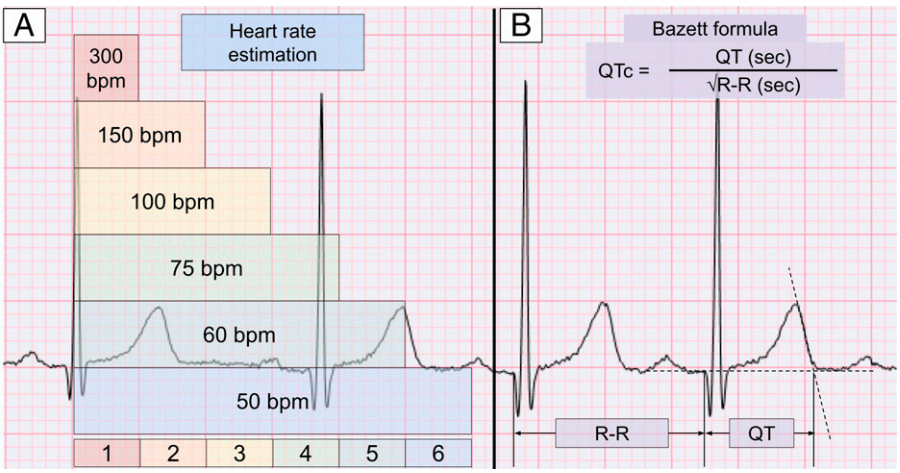

Figure 3. A. Heart rate estimation. B. Corrected QT (QTc) interval calculation (Bazett formula).

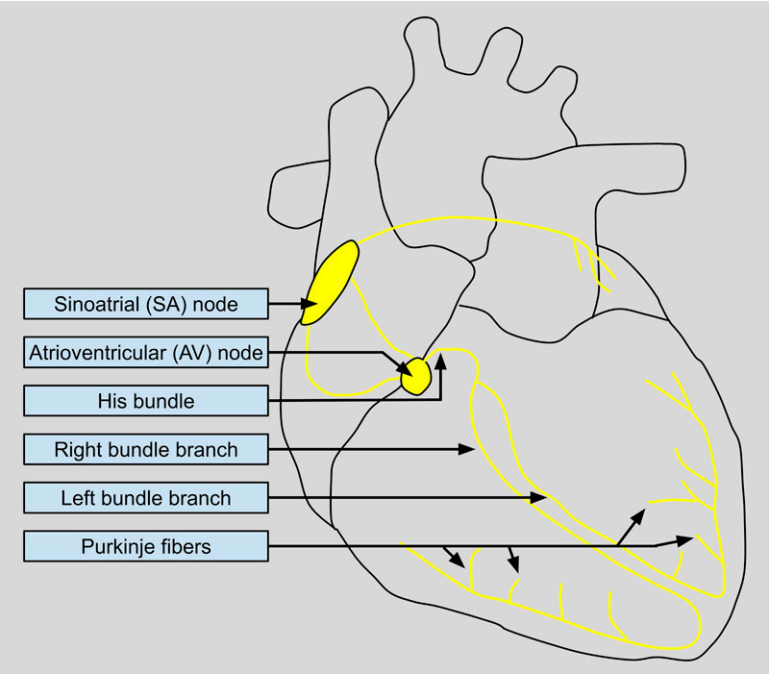

Figure 4. Simplified diagram of the cardiac conduction system.

limb lead reversal, compare vectors in leads I and V_6; if their polarities are opposite, lead reversal is likely and the ECG should be repeated.

If the rhythm is not driven by the sinus node or atrial myocytes, evidenced by QRS complexes without preceding P waves, and the rate is normal, the patient is likely in either junctional rhythm or ventricular rhythm. Junctional rhythm is characterized by a QRS morphology similar to that seen in sinus rhythm. A ventricular rhythm will, by definition, have a different QRS morphology than baseline, as it propagates from cell to cell without using the normal His-Purkinje system. This typically causes a wider QRS morphology due to uncoordinated depolarization.

Axis. Axis refers to the direction of electrical propagation, typically in the frontal plane (also known as the coronal plane.) The QRS axis refers to the vector direction of forces created by ventricular depolarization (Fig 1). The automatic calculation of the "R" axis on modern ECG software is typically accurate and can generally be relied on. To estimate the quadrant of the QRS axis, the reader should evaluate leads I and aVF. If the QRS forces are predominantly positive in lead I, the vector points toward the left. If the forces are mostly positive in lead aVF, the vector points downward. Using this paradigm, the quadrant of the QRS axis can be quickly approximated. This measurement has particular importance in pediatrics because its normal value shifts throughout childhood, and it can be used to screen for CHD. In newborns and infants, a normal QRS axis is rightward (approximately 90°–180°) due to the predominance of the right ventricle in utero. Over the first few months of life, the balance of forces shifts toward a "normal" axis that is seen in adolescence and adulthood, approximately 0° to 90°. In infancy, superior left axis deviation (0°–270°) may be associated with an AV septal defect (normal oxygen saturation) or tricuspid atresia (typically decreased oxygen saturation). Extreme axis deviation (180°–270°), also known as northwest axis, may be an abnormal but nonspecific finding that is associated with variable forms of CHD. Of note, many significant forms of CHD, including hypoplastic left heart syndrome and other severe abnormalities, may present with a normal QRS axis for age on ECG. In the older child and adolescent, right axis deviation may suggest pulmonary hypertension and/or right ventricular hypertrophy.

Intervals. The PR interval represents atrial depolarization and AV node conduction. A prolonged PR interval may be seen in first-degree AV block (see the Bradyarrhythmias subsection). A shortened PR interval, when present with a slurred upstroke of the QRS complex (delta wave), indicates a Wolff-Parkinson-White (WPW) pattern (see the Arrhythmia Syndromes subsection).

The width of the QRS interval reflects the time required for ventricular myocytes to depolarize. As discussed previously herein, if the early portion of a QRS complex is widened, in combination with a short PR interval, this suggests ventricular preexcitation. If the later portion of the QRS

complex is widened, this suggests an intraventricular conduction delay or a bundle branch block (if >120 milliseconds). Right bundle branch block (RBBB) is characterized by a widened R wave (or R′ wave) in lead V_1 (Fig 5A), and left bundle branch block will demonstrate these findings in lead V_6. Incomplete RBBB (an RBBB pattern without exceeding the normal values for QRS width) is commonly seen in childhood and can be a benign finding. (11) True RBBB may be seen after repaired CHD (eg, ventricular septal defect or tetralogy of Fallot). Left bundle branch block is significantly rarer in pediatrics. Any bundle branch block should be investigated if no obvious causation is known.

The QT interval represents ventricular repolarization. To measure the QT interval, a tangent is drawn from the steepest part of the T wave down to the isoelectric baseline (Fig 3B). The time from the beginning of the QRS complex to this point is the QT interval. (12) The QT should then be corrected for heart rate to generate a QTc interval, commonly using the Bazett equation: $QTc = QT/\sqrt{RR}$, using the preceding R-R interval. Although normal QTc intervals vary slightly by age and sex, for screening purposes we suggest a QTc range of 450 to 460 milliseconds as "borderline prolonged" and greater than 460 milliseconds as "prolonged." The QTc interval may be prolonged by electrolyte abnormalities (eg, hypokalemia, hypocalcemia), medication use, or hypothermia. Of note, QTc interval measurement is not reliable in the setting of bundle branch block. For more on long QT syndrome (LQTS), see the Arrhythmia Syndromes subsection.

Forces. The ECG is an insensitive and nonspecific tool for evaluation of atrial enlargement or ventricular hypertrophy. However, it may be useful to be aware of the ECG criteria that have been associated with these diagnoses.

For right atrial enlargement, a P-wave amplitude greater than 0.025 mV (height >2.5 mm) has been used. (13) For left atrial enlargement, a P-wave duration greater than 120 milliseconds (3 small boxes) and a terminal negative P-wave deflection greater than 40 milliseconds in lead V_1 have been used. (14) In children, massive biatrial enlargement has classically been suggestive of restrictive cardiomyopathy. (15)

For evaluation of right (RVH) and left (LVH) ventricular hypertrophy, several adult criteria have been derived in which R- and S-wave voltages are summed from varying

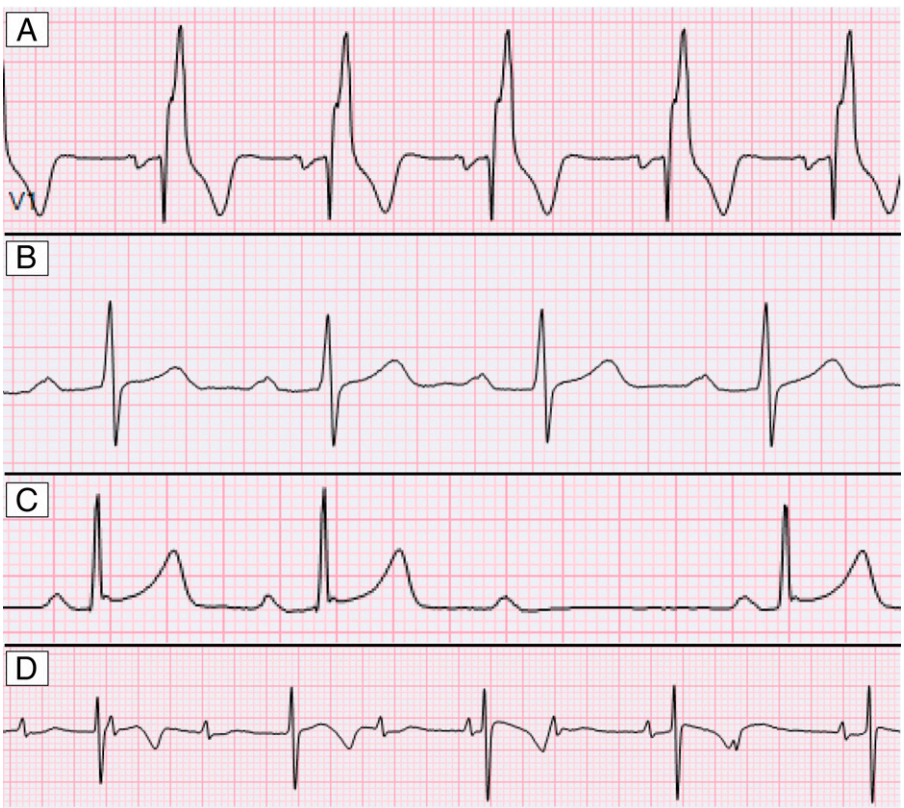

Figure 5. A. Right bundle branch block in a teenager with tetralogy of Fallot status post repair. B. First-degree atrioventricular (AV) block in a healthy adolescent athlete. C. Second-degree AV block Mobitz type 1 (Wenckebach) in a healthy adolescent athlete. D. Third-degree AV block in an infant with congenital complete AV block.

leads (see the Sokolow-Lyon criteria and the Cornell voltage criteria for LVH [16]). Some pediatric providers prefer a simpler approach, assigning the diagnosis of RVH or LVH if the R wave in V_1 or V_6, respectively, is greater than the 98th percentile for age. In general, after approximately 8 years of age, the 98th percentiles may be considered 12 mm for V_1 (RVH) and 30 mm for V_6 (LVH), although these vary in the published literature. Providers are referred to institutional norms for voltage cutoff values across age groups for preferred criteria. Another clue that may suggest RVH is the presence of an upright T wave in V_1 between 1 week and 6 years of age. (17) Supportive evidence for LVH includes T-wave inversions in the left lateral leads (V_6, V_7), sometimes described as a left ventricular strain pattern. (18) Biventricular hypertrophy should be considered if the R- and S-wave voltages in any lead are greater than 60 mm or if other combined criteria are met. The specificity of ECGs in determining hypertrophy is notoriously poor. Even dramatically tall voltages can only suggest possible hypertrophy and can never definitively make a diagnosis (a power reserved for noninvasive imaging techniques). Clinically, while acknowledging its limitations, screening for hypertrophy by ECG in children may assist in the diagnosis of hypertrophic cardiomyopathy.

ST Segments. The ST segment is one of the most important aspects of an ECG in adult patients because the pretest probability of myocardial ischemia is far greater than in children, (19) although ischemia may be seen in certain young patients. (20) The topic of ischemic ST changes is not within the scope of this article given its rarity in this patient population.

The most common benign cause of ST elevation in children is early repolarization. This finding is characterized by elevation of the QRS-ST junction (the J point), often with a notched appearance, and an upsloping (concave) ST segment (Fig 6A). It is most often seen in the inferior and lateral leads and is particularly common in athletic adolescent patients. Pediatric data suggest a benign prognosis. (21)

The most common pathologic cause of ST elevations in pediatrics is myocarditis/pericarditis. In myocarditis, a variety of ECG changes can be seen, including sinus tachycardia, AV block, prolonged QT interval, and low-voltage QRS complexes. (22) ST segments may be diffusely elevated, although ECG patterns mimicking myocardial infarction may be seen. (23) Pericarditis should be considered in a patient with chest pain, diffuse ST elevations, and PR-segment depression (Fig 6B).

In infancy, perhaps the most important cause of myocardial ischemia is ALCAPA: anomalous left coronary artery arising from the pulmonary artery. This "must not miss" diagnosis is associated with ST-segment elevation in the lateral precordial leads and deep Q waves in leads I and aVL. (24) ALCAPA must be considered in any infant with excessive irritability and these ECG findings.

T Waves. As with the ST segment, the T wave bears more importance in adults than it does in pediatrics because it can be useful for detecting ischemic changes. With that said, the detection of abnormal T-wave inversion can be an important clue for the diagnosis of young patients with cardiomyopathies or other disorders.

In the most leftward precordial leads—V_5 and V_6—the T wave should be upright at all ages. Between approximately 1 week of age and adolescence, the T waves should be inverted in V_1 and V_3R. (13) If they are upright, this may be a sign of RVH. The precordial leads should then gradually flip upright as the patient approaches adolescence. In some teenagers, the T wave in V_1 remains inverted and can be considered a normal variant.

Isolated T-wave inversions in leads III, aVR, and V_1 do not require further evaluation. Abnormal T-wave inversions in other leads are a nonspecific finding but may be associated with various types of cardiomyopathy.

Peaked T waves, a qualitative, subjective finding, may be seen in the setting of hyperkalemia; if potassium levels are severely elevated, ECG findings may progress to include a shortened QTc interval, a prolonged PR interval, and loss of visible P waves. (26) Severe hyperkalemia may be found in children with renal disease.

It is important to remember that abnormal depolarization leads to abnormal repolarization. Therefore, in most cases it is not advisable to interpret repolarization (QT interval, ST segments, or T waves) in ECGs with ventricular preexcitation, bundle branch block, or ventricular rhythm/pacing.

Arrhythmia Syndromes

WPW Syndrome. A WPW pattern on ECG is characterized by a short PR interval, a slurred upstroke of the early QRS complex (delta wave), and a widened QRS complex (Fig 7A). These findings are due to some of the antegrade conduction to the ventricles traveling down an accessory pathway crossing the tricuspid or mitral valve annulus. This depolarizes ventricular myocardial cells earlier than those activated by the usual His-Purkinje system and is, therefore, described as ventricular preexcitation. A WPW pattern may be intermittent, and its presence may vary with the patient's sinus rate. (27) Patients with WPW syndrome are at risk for reentrant SVT (see the Tachyarrhythmias subsection). Far more rarely, there may be risk of atrial fibrillation that is

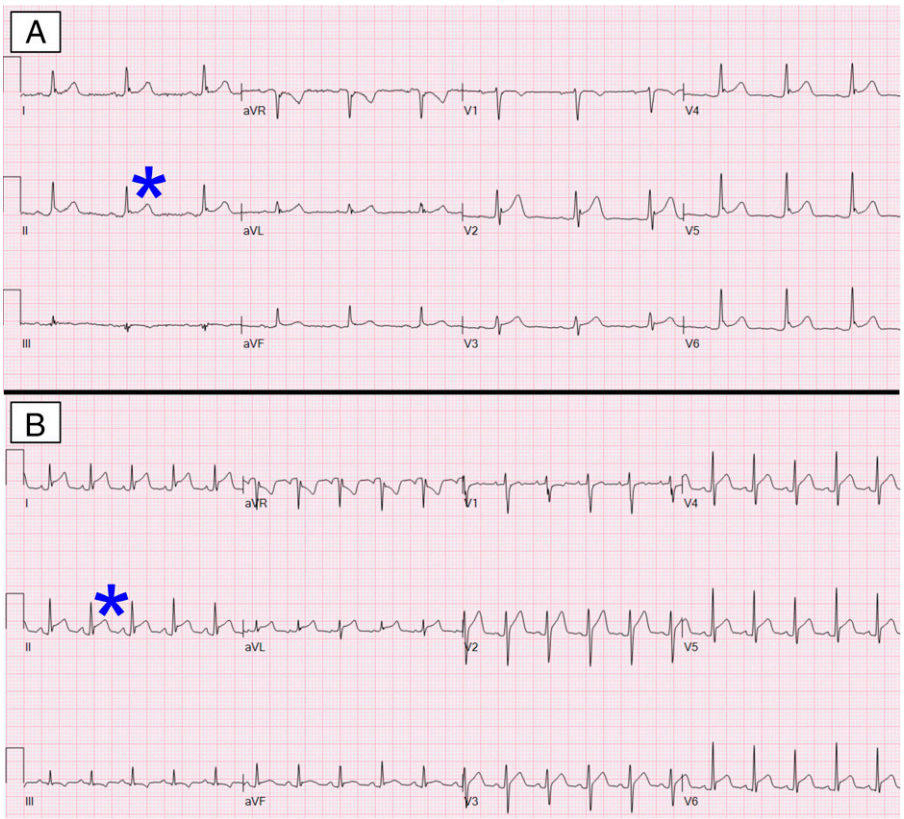

Figure 6. Early repolarization and pericarditis. A. Early repolarization in an asymptomatic teenager characterized by notched J-point elevation (*) and an upsloping (concave) ST segment, particularly in the inferolateral leads. B. Pericarditis in a teenager with large pericardial effusion characterized by diffuse ST-segment elevation (*) and PR-segment depression, often with sinus tachycardia.

rapidly conducted via the accessory pathway, leading to ventricular fibrillation and sudden death. (28)

Congenital LQTS. LQTS is an inherited arrhythmia syndrome characterized by prolonged repolarization of the ventricular myocardium. Patients with LQTS are at risk for sudden death due to torsades de pointes, a polymorphic ventricular tachycardia (VT). Certain T-wave morphologies may be associated with common types of LQTS: type 1 may have a broad T wave (Fig 8A), type 2 a notched T wave, and type 3 a delayed T wave preceded by a prolonged isoelectric period. The hallmark of LQTS is a prolonged QTc interval (often ≥500 milliseconds) without secondary causes. It can be diagnosed in the presence of a pathogenic mutation in a known *LQTS* gene, or also based on clinical scoring (family history, syncope, torsades) using the Schwartz criteria. (29)

Brugada Syndrome. Brugada syndrome is an inherited cardiac channelopathy associated with increased risk of ventricular fibrillation and sudden death. Arrhythmias may be more likely to occur with vagal events, (30) hyperthermia, (31) or during sleep. (32) The resting ECG pattern of Brugada syndrome can be diagnostic and is defined as ST elevation with "type 1 morphology" (Fig 8B) greater than or equal to 2 mm in 1 or more right precordial leads (V_1, V_2). (33) This classic type 1 morphology describes a coved ST elevation followed by T-wave depression, often confused for an RBBB. Other morphologies (types 2 and 3) of the Brugada pattern have been described, although these alone cannot be used to diagnose Brugada syndrome. The ECG findings of Brugada syndrome can be intermittent and may be unmasked by a variety of factors, including fever, sodium channel blockers, electrolyte abnormalities, and alcohol. (34)

Bradyarrhythmias

On a normal ECG, every P wave should be followed by a QRS complex. If there are normal P waves without subsequent QRS complexes, this indicates a type of AV block.

AV block can be categorized as first-, second-, or third-degree. First-degree AV block (Fig 5B) is present when the PR interval is prolonged for age, although all P waves are conducted. In a healthy, asymptomatic teenager, a prolonged PR interval can be a normal finding that does not typically require further evaluation. (10) At younger ages, when first-degree AV block is less commonly seen, pathologic causes

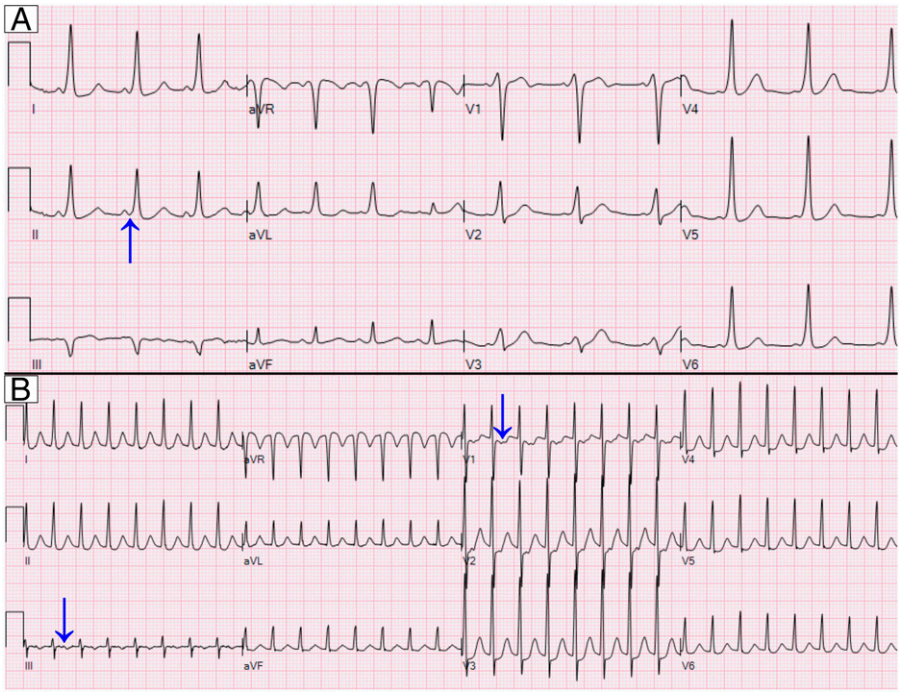

Figure 7. Wolff-Parkinson-White (WPW) syndrome and supraventricular tachycardia (SVT). A. Baseline electrocardiogram of an 11-year-old with WPW syndrome. A short PR interval with a delta wave (↑ symbol) is seen. B. Likely retrograde P waves (↓ symbols) in SVT can be seen in leads III and V_1.

may be considered, (35) including myocarditis, Lyme disease, (36) and coronavirus disease 2019 multisystem inflammatory syndrome. (37) Neuromuscular diseases such as Kearns-Sayre syndrome and Emery-Dreifuss muscular dystrophy may also present with first-degree AV block and worsen with time. (38)

Second-degree AV block is diagnosed when some P waves fail to conduct to the ventricle. In pediatrics, this is most commonly seen as second-degree AV block Mobitz type 1 (Figure 5C), known commonly as Wenckebach, characterized by progressive PR-interval prolongation followed by a nonconducted P wave. Wenckebach is commonly seen in children, particularly during sleep, and in athletic adolescents due to the effect of vagal tone on the AV node, and is often a normal finding.

More rarely seen in children is second-degree AV block Mobitz type 2, in which intermittent nonconducted P waves are seen without preceding PR-interval prolongation. This typically indicates infranodal conduction disease, affecting the His bundle or the bundle branches, and is almost always pathologic.

An important mimic of AV block in infants occurs in the setting of LQTS. Due to the prolonged QT interval, every second P wave lands during ventricular refractoriness and is nonconducted. This leads to a pattern of "pseudo 2:1 AV block." Such patients are at risk for sudden death in infancy; (39) it is, therefore, important to measure the QTc interval carefully in any infant with marked bradycardia or suspicion for 2:1 AV block.

Third-degree AV block (Fig 5D), also known as complete AV block, describes complete electrical disconnection between the atria and ventricles. It is characterized on ECG by dissociation of the P waves and QRS complexes, with a P-to-QRS ratio greater than 1. Complete AV block in a structurally normal heart may be congenital, as can be seen in pregnancies of mothers with autoimmune disease and anti-Ro or anti-La antibodies. (40) Other clinical scenarios include postoperative CHD, heterotaxy syndromes (internal organs abnormally arranged in the chest and abdomen), myocarditis, and neuromuscular diseases. The hemodynamic significance of complete AV block varies greatly, depending on the junctional or ventricular escape rate, patient's age, and pathologic mechanism. (41)

Ectopy

Premature Atrial Contractions. A premature atrial contraction (PAC) is a single early beat arising from an ectopic focus in the atria. This typically results in a narrow or native QRS complex that is preceded by a unique (different than sinus) P wave. After the PAC there is usually a pause, which results from resetting of the sinus node. A PAC may also be "blocked"—identified by an early P wave with no QRS

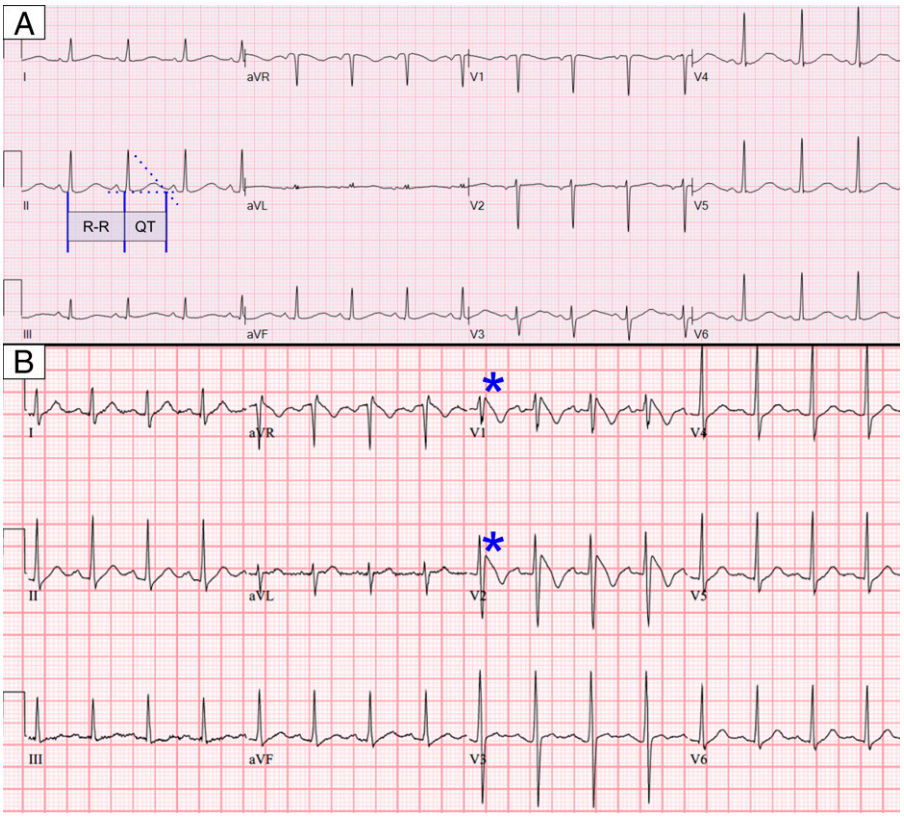

Figure 8. A. Long QT syndrome type 1, corrected QT interval of 540 milliseconds in an 8-year-old with a mutation in the *KCNQ1* gene. B. Brugada syndrome; there is coved ST-segment elevation (*) in the right precordial leads (V_1, V_2).

complex—due to AV node refractoriness, or may conduct with aberrancy (wide QRS complex). PACs are relatively common throughout childhood, particularly in the neonatal period. Most cases are idiopathic, although associations are seen with stimulant exposure, hyperthyroidism, electrolyte imbalance, structural heart disease, cardiomyopathy, cardiac tumors, respiratory ailments, or indwelling catheters. PACs are generally benign, and if no pathologic association is found, such patients can be monitored clinically.

Premature Ventricular Contractions. A premature ventricular contraction (PVC) is a single early beat arising from the ventricles, typically resulting in a wide QRS complex. PVCs are further subdivided into uniform or multiform depending on the presence of singular or multiple QRS morphologies, respectively. PVCs are also common in pediatrics, often idiopathic, and generally well-tolerated. As with PACs, however, it is imperative to perform a thorough history and physical examination to ensure no associations such as excessive caffeination, structural heart disease, cardiac dysfunction, myocarditis, or electrolyte derangement. Ambulatory ECG monitoring is valuable to determine PVC frequency because a high ectopy burden can lead to progressive cardiac dysfunction. In the absence of a secondary cause, treatment is limited to those with high ectopy burdens, dysfunction, or distressing symptoms.

Tachyarrhythmias

Supraventricular Tachycardia. Broadly speaking, SVT describes any tachycardia arising from above the ventricles. In common use, it refers more specifically to tachycardias with a reentrant mechanism. These reentrant tachycardias require 2 parallel pathways of conduction, forming an arrhythmia circuit, most commonly classified as AV nodal reentrant tachycardia or atrioventricular reentrant tachycardia (AVRT). The circuit of AV nodal reentrant tachycardia occurs entirely within the AV node, whereas AVRT is mediated by an accessory pathway connecting the atria and ventricles. A patient with AVRT may have ventricular preexcitation at baseline (WPW syndrome) or the accessory pathway may be hidden due to retrograde-only conduction ("concealed" pathway).

SVT should be considered in a child with persistent tachycardia for age, particularly in the absence of factors such as irritability or fever that may cause sinus tachycardia. In reentrant SVT, the rate is likely to remain stable with minimal variation, as its rate is largely determined by the conduction properties of the SVT circuit. In most cases the

QRS complex will be narrow. P waves are often difficult to discern, although retrograde P waves may be seen buried in the T wave; these may be best seen in leads III and V_1 (Figure 7B). (42) Administration of adenosine via rapid intravenous push, which halts conduction at the AV node, will typically terminate reentrant SVT.

In pediatrics, an important subtype of reentrant SVT is persistent junctional reciprocating tachycardia. It occurs in the presence of a slowly conducting retrograde-only accessory pathway and is characterized by a long R-P interval with deep inverted inferior P waves. This arrhythmia carries particularly high risk of tachycardia-induced cardiomyopathy if incessant and undiagnosed. (43)

Ectopic atrial tachycardia (EAT) should also be considered in the setting of an abnormal P-wave axis and narrow QRS complex tachycardia. EAT is an automatic arrhythmia from a nonsinus focus in the atrium characterized by frequent bursts of tachycardia. There may be risk of tachycardia-induced cardiomyopathy if EAT is sustained or frequent and untreated. (44)

Although rare in children, atrial flutter and atrial fibrillation may be seen. When it occurs in pediatrics, atrial flutter is most common in the first few days of neonatal life. Flutter is characterized by a fixed atrial rate (often ≥300 bpm) and there is often variable AV conduction (ventricular rate may be half or one-third the atrial rate, for example). After synchronized direct current cardioversion, recurrence is uncommon. (45) Unlike flutter, atrial fibrillation leads to ECG findings of a disorganized atrial rate and an irregularly irregular ventricular response. Atrial fibrillation may rarely be seen in healthy adolescents and is occasionally associated with a predisposition for other forms of reentrant SVT. (46)

If the ECG demonstrates wide complex tachycardia, the diagnosis is assumed to be VT until proved otherwise. There are other important causes of wide complex tachycardia to consider: sinus tachycardia with preexisting bundle branch block, SVT with aberrant conduction, antidromic reentrant tachycardia (a rare subtype of SVT), and preexcited atrial fibrillation (in a patient with WPW syndrome) with rapid ventricular response. ECG features suggestive of VT include dissociated P waves (with a QRS-to-P ratio >1), fusion beats with an intermediate QRS morphology (mix of sinus and ventricular conduction), and an abnormal QRS axis. Comparison with a previous ECG may assist in determining whether the wide QRS complex is a change from baseline.

The most common form of ventricular ectopy and VT in a healthy adolescent arises from the right or left ventricular outflow tracts. Outflow tract VT can be diagnosed on ECG by its inferior QRS axis (upright QRS in leads II, III, and aVF). Although typically benign and self-resolving, outflow tract VT may be associated with palpitations or development of cardiac dysfunction, warranting treatment with antiarrhythmic medications or transcatheter ablation.

Summary

- Based on research evidence and consensus, the pediatric electrocardiogram (ECG) is an important tool for the screening and diagnosis of congenital heart disease, cardiomyopathies, arrhythmias, and arrhythmia syndromes. (31)(47)(48)(49)(50)(51)
- Based on research evidence and consensus, ECG interpretation changes with patient age; awareness of these changes is key to identification of abnormalities. (4)(5)(6)(7)(8)
- Based on some research evidence, the QRS axis is most important in infancy as a screening tool in the setting of suspected congenital heart disease. (52)(53)(54)
- Based on some research evidence, T-wave inversion in the inferior and/or lateral leads may warrant evaluation for cardiomyopathy. (25)(46)
- Based on research evidence and consensus, if bradycardia is present, AV block or blocked atrial ectopy should be considered. Sinus bradycardia is typically benign. (10)
- Based on research evidence and consensus, if narrow QRS complex tachycardia is present and no preceding P waves are seen, supraventricular tachycardia must be ruled out. (55)
- Based on research evidence and consensus, wide QRS complex tachycardia should be considered ventricular tachycardia until proved otherwise. (42)
- Based on research evidence and consensus, in a young patient with syncope or palpitations, arrhythmia syndromes that may be seen on resting ECG should be considered. Common examples include Wolff-Parkinson-White syndrome, long QT syndrome, and Brugada syndrome. (31)(45)

References and teaching slides for this article can be found at https://doi.org/10.1542/pir.2021-005346.

PIR QUIZ

1. A patient with a history of repaired complex congenital heart disease is followed in your clinic. A routine electrocardiogram (ECG) demonstrates a P wave preceding every QRS, with the P wave always inverted in lead I. This finding is consistent with which of the following?

   A. Junctional rhythm.
   B. Left atrial rhythm.
   C. Low atrial rhythm.
   D. Sinus rhythm.
   E. Wandering pacemaker.

2. You are caring for a 3-day-old infant in the NICU with hypoxemia undergoing cardiac evaluation. An echocardiogram reveals tricuspid atresia. Which of the following best describes the expected QRS axis to be seen on ECG in this patient?

   A. A QRS axis that is similar to infants without congenital heart disease.
   B. "Normal" axis (0–90°).
   C. Northwest axis deviation (180°–270°).
   D. Rightward QRS axis (90°–180°).
   E. Superior left axis deviation (0–270°).

3. A 12-year-old girl is brought to the clinic after a syncopal episode. An ECG is obtained. Your initial impression is that she may have right bundle branch block, but on further review of the ECG you notice that there are ST-segment elevations, specifically in leads V_1 and V_2, followed by T-wave depression. Based on these ECG findings, you are concerned about which of the following diagnoses?

   A. Brugada syndrome.
   b. Congenital long QT syndrome.
   c. Hyperkalemia.
   d. Myocarditis.
   e. Wolff-Parkinson-White syndrome.

4. Which of the following ECG findings is most consistent with second-degree atrioventricular block Mobitz type 2?

   a. Dissociation between P waves and QRS complexes.
   b. Intermittent nonconducted P waves, without preceding PR-interval prolongation.
   c. PR interval prolonged for age, but all P waves are followed by a QRS.
   d. Progressive PR-interval prolongation, followed by a P wave with no conduction.
   e. P-to-QRS ratio less than 1.

5. An 8-year-old girl is brought to the emergency department with lethargy and lightheadedness. She is tachycardic at 190 bpm. An ECG shows a narrow QRS complex. Retrograde P waves can be seen, and the P-to-QRS ratio is 1. These ECG findings rule out which of the following rhythms?

   a. Atrial fibrillation.
   b. Atrioventricular reentrant tachycardia.
   c. Atrioventricular nodal reentrant tachycardia.
   d. Ectopic atrial tachycardia.
   e. Persistent junctional reciprocating tachycardia.

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