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The Pediatric ECG

Ghazala Q. Sharieff, MD[a,b,*], Sri O. Rao, MD[c]

[a]*Children's Hospital and Health Center/University of California–San Diego, 3020 Children's Way, San Diego, CA 92123*
[b]*Pediatric Emergency Medicine, Palomar-Pomerado Hospitals/California Emergency Physicians, 555 East Valley Parkway, Escondido, CA 92025, USA*
[c]*Division of Pediatric Cardiology, Children's Hospital and Health Center, 3020 Children's Way, San Diego, CA 92123, USA*

There are many nuances to the pediatric ECG that relate to age-specific changes. These findings relate directly to changes in the myocardium and circulatory system as the individual matures from infancy to adulthood. For example, fetal circulation relies primarily on the right side of the heart and at birth the right ventricle is larger and thicker than the left ventricle. During infancy, increased physiologic stress and work of the left ventricle leads to its enlargement, such that by 6 months it is twice the thickness of the right. By adolescence, the left ventricle is at least 2.5 times as thick as the right [1]. These changes over time lead to the variability of normal ECGs in children, which can sometimes delay interpretation in the emergency department (ED).

A recent review of pediatric ED use reveals that the most common reasons for obtaining ECGs in children are chest pain, suspected dysrhythmia, seizure, syncope, drug exposure, electrical burns, electrolyte abnormalities, and abnormal physical examination findings. Of 71 pediatric ECGs reviewed over a 15-month period, 14 (20%) had clinical significance, such as prolonged QT syndrome, ventricular hypertrophy, or premature ventricular beats [2]. Although a complete review of ECG interpretation is beyond the scope of this article, the authors suggest the use of a systematic approach to ECG interpretation with special attention to rate, rhythm, axis, intervals, ventricular and atrial hypertrophy, and the presence of any ischemia or repolarization abnormalities. This article aids the reader in discerning what is truly essential on the pediatric ECG and also discusses findings in patients with congenital heart disease, hypertrophic cardiomyopathy, and myocarditis.

* Corresponding author.
E-mail address: ghazalaqs@hotmail.com (G.Q. Sharieff).

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The normal pediatric ECG

There are many systematic techniques for interpreting ECGs and no one method is particularly better than another. A caveat to the electronic interpretation that many ECG machines conduct is that they are manufactured and calibrated with adult values in the software package; hence, the machine interpretation is frequently inaccurate with children. On the other hand, they are reasonably accurate in calculating intervals that are averaged over the entire recording period. The settings of the ECG, however, must be full standard, defined as 10 mm/mV with a standard paper speed of 25 mm/sec. These settings can be changed on the machine to elucidate certain features, but a standard ECG is the only one that should be referenced to normal values. Frequently, additional right ventricular and posterior left ventricular precordial leads (V3R, V4R, and V7) are included with pediatric ECGs to provide additional information on patients who have complex congenital abnormalities. In most pediatric patients, these leads can be ignored.

Table 1 lists the normal pediatric ECG values seen in the newborn, infant, child, and adolescent [3–5]. This table lists normal ranges for heart rate, QRS axis, PR and QRS complex intervals, and R- and S-wave amplitudes, all of which significantly change with age. Rapid changes occur over the first year of life as a result of the dramatic changes in circulation and cardiac physiology. After infancy, subsequent changes are more gradual until late adolescence and adulthood.

Heart rate

In children, cardiac output is determined primarily by heart rate as opposed to stroke volume. With age, the heart rate decreases as the ventricles mature and stroke volume plays a larger role in cardiac output. Age and activity-appropriate heart rates thus must be recognized. Average

Table 1
Pediatric ECG: normal values by age

Age	HR (bpm)	QRS axis (degrees)	PR interval (sec)	QRS interval (sec)	R in V1 (mm)	S in V1 (mm)	R in V6 (mm)	S in V6 (mm)
1st wk	90–160	60–180	0.08–0.15	0.03–0.08	5–26	0–23	0–12	0–10
1–3 wk	100–180	45–160	0.08–0.15	0.03–0.08	3–21	0–16	2–16	0–10
1–2 mo	120–180	30–135	0.08–0.15	0.03–0.08	3–18	0–15	5–21	0–10
3–5 mo	105–185	0–135	0.08–0.15	0.03–0.08	3–20	0–15	6–22	0–10
6–11 mo	110–170	0–135	0.07–0.16	0.03–0.08	2–20	0.5–20	6–23	0–7
1–2 yr	90–165	0–110	0.08–0.16	0.03–0.08	2–18	0.5–21	6–23	0–7
3–4 yr	70–140	0–110	0.09–0.17	0.04–0.08	1–18	0.5–21	4–24	0–5
5–7 yr	65–140	0–110	0.09–0.17	0.04–0.08	0.5–14	0.5–24	4–26	0–4
8–11 yr	60–130	−15–110	0.09–0.17	0.04–0.09	0–14	0.5–25	4–25	0–4
12–15 yr	65–130	−15–110	0.09–0.18	0.04–0.09	0–14	0.5–21	4–25	0–4
>16 yr	50–120	−15–110	0.12–0.20	0.05–0.10	0–14	0.5–23	4–21	0–4

Courtesy of Ra'id Abdullah, MD, University of Chicago, Illinois.

resting heart rate varies with age; newborns can range from 90–160 beats per minute (bpm) and adolescents from 50–120 bpm. The average heart rate peaks about the second month of life and thereafter gradually decreases until adolescence (Fig. 1). Heart rates grossly outside the normal range for age should be scrutinized closely for dysrhythmias.

QRS axis

In utero, blood is shunted away from the lungs by the patent ductus arteriosus, and the right ventricle provides most of the systemic blood flow. As a result, the right ventricle is the dominant chamber in the newborn infant. In the neonate and young infant (up to 2 months), the ECG shows right ventricular dominance and right QRS axis deviation (Fig. 1). Most of the QRS complex is reflective of right ventricular mass. Across the precordium, the QRS complex demonstrates a large amplitude R wave (increased R-/S-wave ratio) in leads V1 and V2, and small amplitude R wave (decreased R-/S-wave ratio) in leads V5 and V6. As the cardiac and circulatory physiology matures, the left ventricle becomes increasingly dominant. Over time, the QRS axis shifts from rightward to a more normal position, and the R-wave amplitude decreases in leads V1 and V2 and increases in leads V5 and V6 (Fig. 2; and see Fig. 1).

PR interval

Similarly, the PR interval also varies with age, gradually increasing with cardiac maturity and increased muscle mass. In neonates, it ranges from 0.08–0.15 sec and in adolescents from 0.120–0.20 sec [3]. The normal shorter PR interval in children must be taken into account when considering the diagnosis of conduction and atrioventricular (AV) block.

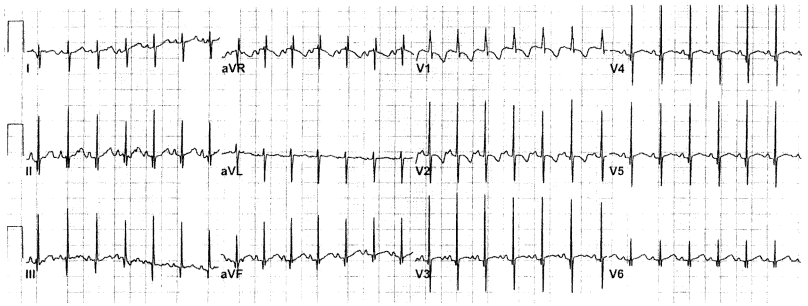

Fig. 1. Normal ECG of 4-week-old infant. The ECG demonstrates right axis deviation and large R-wave amplitude and inverted T waves in the right precordial leads (V1 and V2) indicating right ventricular dominance normally seen in early infancy. Also note the fast heart rate, which is also normal for this age group.

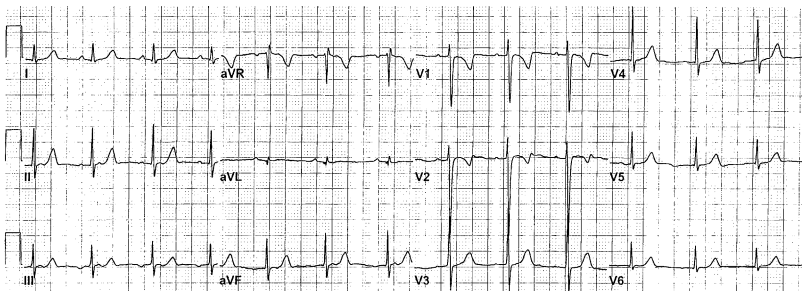

Fig. 2. Persistent juvenile pattern. This ECG in an 11-year-old boy reveals inverted T waves in leads V1 and V2 consistent with juvenile T-wave pattern. Such a finding can persist normally into adolescence.

QRS complex duration

The QRS complex duration varies with age. In children, the QRS complex duration is shorter, possibly because of decreased muscle mass, and gradually increases with age. In neonates it measures 0.030–0.08 sec and in adolescents 0.05–0.10 sec. A QRS complex duration exceeding 0.08 sec in young children (younger than 8 years of age) or exceeding 0.10 sec in older children may be pathologic. As a result, slight prolongation of what may appear as a normal QRS complex can indicate a conduction abnormality or bundle branch block in children.

QT interval

Because the QT interval varies greatly with heart rate, it is usually corrected (QTc), most commonly using Bazett's formula: $QTc = QT/\sqrt{RR}$ interval. During the first half of infancy, the normal QTc is longer than in older children and adults. In the first 6 months of life, the QTc is considered normal at less than 0.49 sec. After infancy, this cutoff is generally 0.44 sec.

T waves

In pediatric patients, T-wave changes on the ECG tend to be nonspecific and are often a source of controversy. What is agreed on is that flat or inverted T waves are normal in the newborn. In fact, the T waves in leads V1 through V3 usually are inverted after the first week of life through the age of 8 years as the so-called "juvenile" T-wave pattern (see Fig. 1). In addition, this pattern can persist into early adolescence (Fig. 2). Upright T waves in V1 after 3 days of age can be a sign of right ventricular hypertrophy (RVH).

Chamber size

An assessment of chamber size is important when analyzing the pediatric ECG for underlying clues to congenital heart abnormalities. P waves greater

than 2 mV (2 small boxes) in infants and greater than 3 mV (3 small boxes) in adolescents may indicate right atrial enlargement (RAE). Because the right atrium depolarizes before the left atrium, P-wave duration greater than 0.08 sec (2 small boxes) in infants and 0.12 sec (3 small boxes) in adolescents indicates left atrial enlargement (LAE).

RVH is best seen in leads V1 and V2 with an rSR′, QR (no S), or a pure R (no Q or S) wave. RVH also may be suggested by the presence of a large S wave in lead V6, upright T waves in leads V1–V3 after the first week of life, or persistence of the right ventricular dominance pattern of the neonate. Similarly, left ventricular hypertrophy (LVH) is suggested with the presence of tall R waves in lead V6, large S wave in lead V1, left ventricular "strain" pattern in leads V5 and V6, and a mature precordial R-wave progression in the newborn period. Biventricular hypertrophy is seen when ECG criteria for enlargement of both ventricles is seen (Table 2).

The abnormal pediatric ECG

Tachydysrhythmias

The tachydysrhythmias can be classified broadly into those that originate from loci above the AV node (supraventricular), those that originate from the AV node (AV node re-entrant tachycardias), and those that are

Table 2
ECG criteria for ventricular and atrial hypertrophy

Right ventricular hypertrophy
R wave greater than the 98th percentile in lead V1 (see Table 1)[a]
S wave greater than the 98th percentile in lead I or V6 (see Table 1)
RSR′ pattern in lead V1, with the R′ height being greater than 15 mm in infants younger than 1 year of age or greater than 10 mm in children older than 1 year of age
Q wave in lead V1
Left ventricular hypertrophy
R-wave amplitude greater than 98th percentile in lead V5 or V6 (see Table 1)
R wave less than 5th percentile in lead V1 or V2 (see Table 1)
S-wave amplitude greater than 98th percentile in lead V1 (see Table 1)
Q wave greater than 4 mm in lead V5 or V6
Inverted T wave in lead V6
Right atrial enlargement
Peaked P wave in leads II and V1 that is higher than 3 mm in infants younger than 6 months of age and greater than 2.5 mm in infants older than 6 months of age
Left atrial enlargement
P-wave duration greater than 0.08 seconds in a child younger than 12 months of age or greater than 10 ms in children 1 year and older
P wave minimal or plateau contour
Terminal or deeply inverted P wave in lead V1 or V3R

The presence of any of these is suspicious for hypertrophy. It is not necessary for all of the criteria to be met.

[a] qR wave pattern in V1 may be seen in 10% of normal newborns.

ventricular in origin. Although AV node re-entrant tachycardias are more common in adults, the vast majority of tachycardias in children are supraventricular in origin. It is important to record continuous ECG or rhythm strips with the child in tachycardia, while medication is being pushed, and when converted to sinus rhythm. On recognition of a tachycardia, stepwise questioning can help clarify the ECG tracing. Is it regular or irregular? Is the QRS complex narrow or wide? Does every P wave result in a single QRS complex?

Sinus tachycardia can be differentiated from other tachycardias by a narrow QRS complex and a P wave that precedes every QRS complex. Sinus tachycardia is a normal rhythm with activity and exercise and can be a normal physiologic response to stresses, such as fever, dehydration, volume loss, anxiety, or pain. Sinus tachycardia that occurs at rest may be a sign of sinus node dysfunction. It is important to keep in mind, however, that the normal range for heart rate is higher in children (see Table 1).

Supraventricular tachycardia (SVT) is the most common symptomatic dysrhythmia in infants and children, with a frequency of 1 in 250–1000 patients [6]. The peak incidence of SVT is during the first 2 months of life. Infants with SVT typically present with nonspecific complaints, such as fussiness, poor feeding, pallor, or lethargy. Older children may complain of chest pain, pounding in their chest, dizziness, shortness of breath, or may demonstrate an altered level of consciousness. The diagnosis often begins in triage with the nurse reporting that ''The heart rate is too fast to count.''

In newborns and infants with SVT, the heart rate is greater than 220 bpm and can be as fast as 280 bpm, whereas in older children, SVT is defined as a heart rate of more than 180 bpm [7]. On the ECG, supraventricular tachycardia is evidenced by a narrow QRS complex tachycardia without discernible P waves or beat-to-beat variability (Fig. 3). The initial ECG may be normal, however, and a 24-hour rhythm recording (eg, Holter monitor) or an event monitor may be necessary to document the dysrhythmia in cases of intermittent episodes. In children younger than 12 years of age, the most common cause of supraventricular tachycardia is an accessory atrioventricular pathway, whereas in adolescents, AV node re-entry tachycardia becomes more evident [5].

SVT can be associated with Wolff Parkinson White (WPW) syndrome. SVT in WPW syndrome generally is initiated by a premature atrial depolarization that travels to the ventricles by way of the normal atrioventricular pathway, travels retrograde through the accessory pathway, and re-enters the AV node to start a re-entrant type of tachycardia [7,8]. Antegrade conduction through the AV node followed by retrograde conduction through the accessory pathway produces a narrow complex tachycardia (orthodromic tachycardia) and is the most common form of SVT found in WPW syndrome [7,8]. Less commonly re-entry occurs with antegrade conduction through the accessory pathway and retrograde conduction through the AV node (antidromic tachycardia) to produce a wide complex tachycardia [9]. Typical

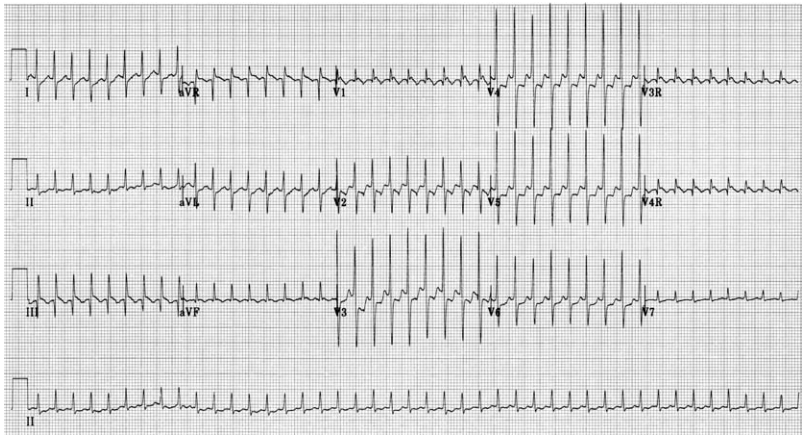

Fig. 3. Supraventricular tachycardia. This ECG demonstrates a supraventricular tachycardia in a neonate. Note the remarkably fast heart rate (260 bpm) and narrow QRS complexes without discernible P waves. It often can be difficult to interpret the ECG in this setting, because the normal QRS complex width is shorter in infants and young children.

ECG findings of WPW are a short PR interval, wide QRS complex, and a positive slurring in the upstroke of the QRS complex, known as a delta wave (Fig. 4). The ECG in most WPW SVT does not show the delta wave, because tachycardia is not conducted down through the accessory pathway. Episodes of SVT in children who have WPW usually occur early in the first year of life [9]. Episodes of SVT often resolve during infancy but may recur later in life, usually from 6–8 years of age [9].

Atrial ectopic tachycardia may be differentiated from SVT by the presence of different P-wave morphologies. Each P wave is conducted to the

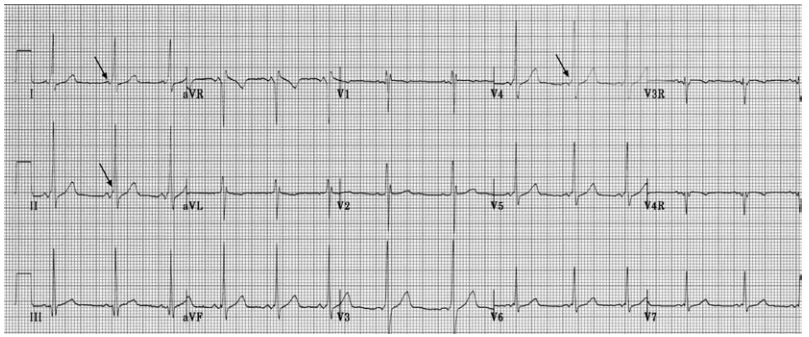

Fig. 4. Wolff Parkinson White syndrome. This ECG is of a 9-year-old boy who presented to the ED with palpitations and minor neck trauma and was found to be in a supraventricular tachycardia. This ECG was done shortly after adenosine was administered and the rhythm converted to sinus. Note the abnormally short PR interval for age and the presence of a delta wave (*arrows*) at the beginning of the QRS complex. The delta wave is not uniformly apparent in all leads.

ventricle, and because the ectopic atrial focus is faster than the SA node, the ectopic determines the ventricular rate (Fig. 5).

Although supraventricular tachycardias are more common than those of ventricular origin, it is important to remember that the normal QRS complex is shorter in duration in children than adults. As a result, a QRS complex width of 0.09 sec may seem normal on the ECG but actually represents an abnormal wide QRS complex tachycardia in an infant. The differential diagnosis of wide complex tachycardia includes sinus/supraventricular tachycardia with bundle branch block or aberrancy, antidromic AV re-entry tachycardia, ventricular tachycardia (VT), or coarse ventricular fibrillation [10]. ECG findings that support the presence of VT include AV dissociation with the ventricular rate exceeding the atrial rate, significantly prolonged QRS complex intervals, and the presence of fusion or capture beats. If there is a right bundle branch block, the presence of VT is supported by a qR complex in V1 and a deep S wave in V6. If there is a left bundle branch block present, then the presence of VT is supported by a notched S wave and an R-wave duration of >0.03 sec in V1 and V2 and a Q wave in V6 [10].

Conduction abnormalities

All degrees of AV block may occur in pediatric patients. It is important to remember that the normal PR interval in infants is shorter and lengthens as cardiac tissue matures with age. A normal appearing PR interval of 0.20 sec may thus in fact represent a pathologic first-degree AV block in an infant or young child.

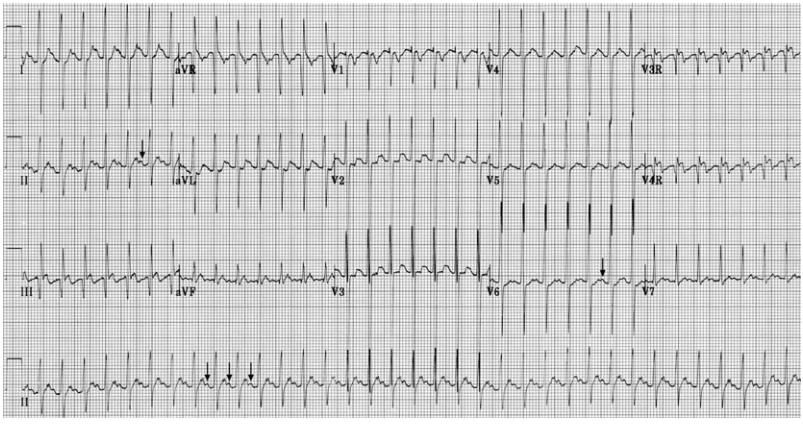

Fig. 5. Atrial ectopic tachycardia. This ECG is of an 18-month-old infant who presented with a several week history of poor feeding and vomiting. The ECG shows atrial ectopic tachycardia. Notice the different P-wave morphologies (*arrows*). Each P wave is conducted to the ventricle, and because the ectopic atrial focus is faster than the sinoatrial node, it determines the ventricular rate.

Complete heart block is a common cause of significant bradycardia in pediatric patients and may be acquired or congenital (Fig. 6). Causes of congenital heart block include structural lesions like L-transposition of the great arteries, or maternal connective tissue disorders. Acquired heart block may result from disorders such as Lyme disease, systemic lupus erythematosus, muscular dystrophies, Kawasaki disease, or rheumatic fever [11].

Bundle branch blocks (BBB) may be present when there is QRS complex prolongation abnormal for a given age. Right BBB occurs with abnormal rightward and anterior terminal forces, frequently manifesting on ECG as an rSR′ pattern in leads V1 and V2. Right BBB is more common than left BBB and can be seen after surgical repair of congenital heart defects, particularly ventricular septal defect repairs. Similarly, left BBB is seen with abnormal leftward and posterior forces, best appreciated in leads V5 and V6. Left BBB is rare in children, however, and the possibility of WPW should be considered, because this syndrome can mimic a left BBB pattern.

Congenital heart

With an incidence of 8/1000 live births, many of the structural congenital heart diseases present in the neonatal period [12]. The signs and symptoms of congenital heart disease may be nonspecific, however. Infants may present with tachypnea, sudden onset of cyanosis or pallor that may worsen with crying, sweating with feeds, lethargy, or failure to thrive [13].

Congenital heart disease lesions that present in the first 2–3 weeks of life are typically the ductal-dependent cardiac lesions. During this period the ductus arteriosus had been sustaining blood flow for these infants. When the ductus closes anatomically at 2–3 weeks of life, these infants suddenly

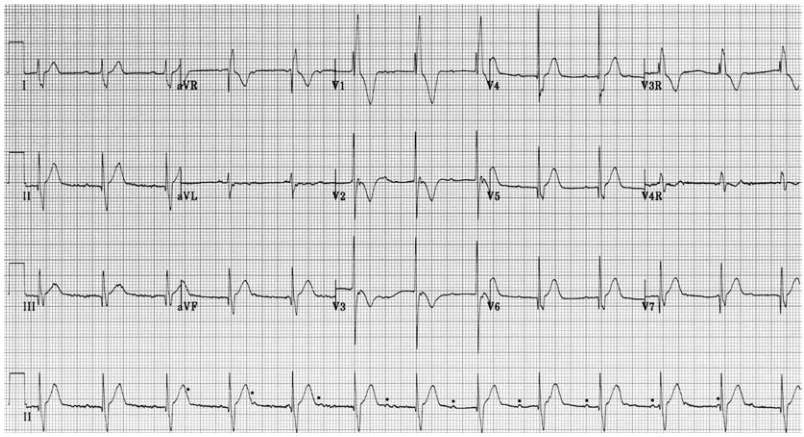

Fig. 6. Complete heart block. The QRS complexes are independent of the P waves (*dots*). This ECG is from a 6-month-old infant who had undergone recent repair of a membranous VSD that temporarily stunned the AV node.

become ill. Depending on the underlying structural abnormality, these neonates present with sudden cyanosis or signs of cardiovascular collapse. These newborns have depended on the ductus arteriosus to supply blood to the lungs—as with tetralogy of Fallot (ToF) or tricuspid atresia (TA)—or to the systemic circulation—as in the case of coarctation of the aorta (CoA) or hypoplastic left heart syndrome (HLHS). The main causes of cyanotic congenital heart disease are ToF, TA, transposition of the great arteries (TGA), truncus arteriosus, total anomalous pulmonary venous return (TAPVR), and pulmonary atresia or stenosis. Time of onset and the common associated ECG findings are listed in Table 3 [14–16].

The other class of congenital cardiac lesions that present in the first month of life are the left-to-right intracardiac shunts, such as ventricular septal or atrioventricular canal defects. As the normal pulmonary vascular resistance falls over the first month of life, any pre-existing left-to-right shunt sees a gradual increase in flow across the shunt, resulting in congestive heart failure. The differential diagnosis of congenital heart diseases that cause congestive heart failure include not only the left-to-right intracardiac

Table 3
Congenital heart abnormalities: time of onset and typical ECG findings

CHD	Onset	RVH	LVH	RAE	LAE	RAD	LAD	RBBB
PDA	2^{nd}–3^{rd} wk		+ (older child)					
ASD	Variable	+		+		+		+
VSD	2^{nd}–12^{th} wk	+	+				+	+
CoA	1^{st} wk	+ (newborn)	+ (older)			+ (newborn)		
ToF	1^{st}–12^{th} wk	+				+		+ (after repair)
TGA	1^{st} wk	+				+	+	
Truncus arteriosis	Variable, infancy	+	+					
Tricuspid atresia	1^{st}–4^{th} wk		+	+			+	
PA	Variable		+					
HLHS	1^{st} wk	+						
AS	Variable		+					
PS	1^{st}–4^{th} wk	+		+		+		
AVC	2^{nd}–3^{rd} wk	+	+	+	+		+	+
HCM	Variable, adulthood		+		+			

AS, aortic stenosis; ASD, atrial septal defect; AVC, atrioventricular canal defects; CoA, coarctation of aorta; HCM, hypertrophic cardiomyopathy; HLHS, hypoplastic left heart syndrome; LAD, left axis deviation; LAE, left atrial enlargement; LVH, left ventricular hypertrophy; PA, pulmonary atresia; PDA, patent ductus arteriosus; PS, pulmonic stenosis; RAD, right axis deviation; RAE, right atrial enlargement; RBBB, right bundle branch block; RVH, right ventricular hypertrophy; TGA, transposition of great vessels; ToF, tetralogy of Fallot; VSD, ventricular septal defect.

shunts, but also HLHS, CoA, TA, endocardial cushion defect, patent ductus arteriosus (PDA), aortic stenosis, interrupted aortic arch, aortic atresia, and mitral valve atresia [17,18].

An ECG should be obtained in all infants suspected of having congenital heart disease. Although the ECG does not make the diagnosis, it can show evidence of conduction abnormalities or chamber enlargement as a result of the congenital defect. In addition, the ECG provides a means of assessing the degree of cardiac flow obstruction, chamber hypertrophy, and the development of dysrhythmias as a result of the congenital heart disease.

Several ECG findings can be associated with specific congenital heart diseases (Table 3). The ECG can seem normal or age-appropriate for some congenital heart diseases. These include cases of PDA, mild-moderate pulmonary stenosis, TGA, ASD, VSD, and CoA, though the presence of abnormalities on the ECG is generally the rule.

RVH is the most common abnormality seen with congenital heart disease and can be seen with pulmonary stenosis, ToF, TGA, and VSD with pulmonary stenosis or pulmonary hypertension, CoA (newborn), pulmonary valve atresia, HLHS, and ASD. RVH may be difficult to distinguish during the early neonatal period because of the normal right ventricular predominance on the ECG at this age. The abnormality becomes clear, however, with later infancy and early childhood.

LVH is seen in lesions with small right ventricles, such as tricuspid atresia, pulmonary atresia with intact ventricular septum, and lesions with left ventricular outflow track obstruction (AS, CoA, hypertrophic cardiomyopathy [HCM]). LVH also can be seen in older children with PDA and larger VSD or AV canal defects (Fig. 7).

In conjunction with ventricular changes, atrial abnormalities can be detected on the ECG with congenital heart disease. RAE occurs with large

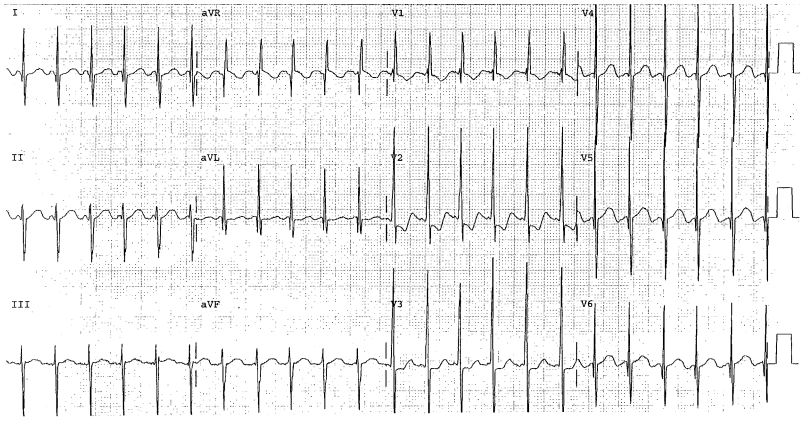

Fig. 7. Atrioventricular canal defect. This ECG is of an 8-week-old boy who had an AVC. The ECG reveals biventricular hypertrophy and left axis deviation not typical for this age group.

left-to-right shunts, causing RA volume overload, and can be seen with ASD, atrioventricular canal defects, tricuspid atresia, Ebstein anomaly, and severe pulmonary stenosis. LAE can be seen with mitral stenosis or insufficiency, left heart obstruction, and complete AV canal defects.

Abnormal QRS axis deviations are seen commonly with congenital heart defects. Right axis deviation can occur with ASD, ToF, CoA, TGA, and pulmonary stenosis. Left axis deviation can be seen with large VSD, tricuspid atresia, TGA, and complete AV canal defects (Fig. 7). Right BBB can be seen with ASD, complete AV defects, small VSD, and after repair of ToF. It is important to keep in mind, however, that incomplete right BBB can be a normal part of the involution of right ventricular forces during infancy and early childhood (Fig. 8).

Hypertrophic cardiomyopathy

Although most cases of HCM are diagnosed at 30–40 years of age, 2% of cases occur in children younger than 5 years of age and 7% occur in children younger than 10 years of age [19]. Clinical presentation varies, with patients experiencing chest pain, palpitations, shortness of breath, syncopal or near syncopal episodes, or sudden death. The hallmark anatomic finding in patients who have HCM is an asymmetric, hypertrophied, nondilated left ventricle with greater involvement of the septum than the ventricular free wall.

ECG findings include LAE and LVH, ST-segment abnormalities, T-wave inversions, Q waves, and diminished or absent R waves in the lateral leads. Premature atrial and ventricular contractions, supraventricular tachycardia, multifocal ventricular dysrhythmias, or new onset atrial fibrillation also may be present.

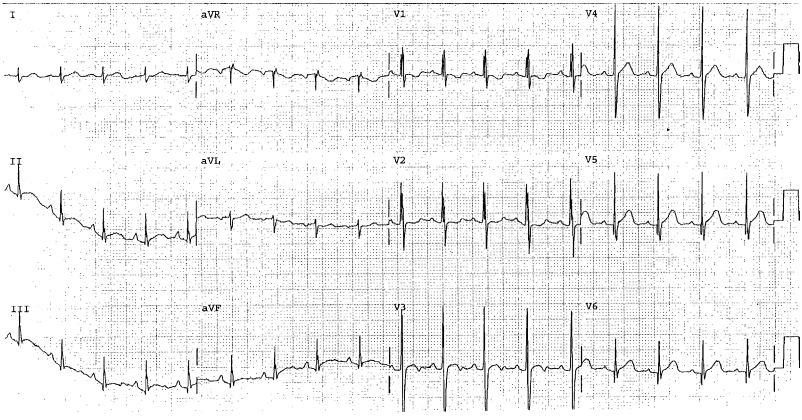

Fig. 8. Incomplete right bundle branch block. This ECG is from a 4-year-old boy. Lead V1 demonstrates an rSR′ pattern, but a relatively narrow QRS complex. The amplitude of the R′ wave approaches criteria for RVH, but this ECG also may be a normal variant.

Myocarditis

An inflammatory condition of the myocardium, this disease has numerous causes; the most common etiology in North America is viral (Coxsackie A and B, ECHO viruses, and influenza viruses) [20,21]. The clinical presentation varies depending on multiple factors, including etiology and patient age. Neonates and infants may present with symptoms such as lethargy, poor feeding, irritability, pallor, fever, and failure to thrive. Symptoms suggestive of heart failure like diaphoresis with feeding, rapid breathing, tachycardia, or respiratory distress also may be present. Older children may complain of weakness and fatigue, particularly on exertion. Signs of poor cardiac function, including signs of congestive heart failure, may be present on examination.

Multiple ECG findings may be present. Sinus tachycardia is the most common dysrhythmia. A tachycardia faster than expected for the degree of fever (10 bpm for each degree of temperature elevation) may indicate myocarditis. Many other dysrhythmias may be associated with myocarditis, including junctional tachycardias, ventricular ectopy, ventricular tachycardias, and even second- and third-degree AV blocks. Morphologically there may be T-wave flattening or inversion and low QRS complex voltage, less than 5 mm in all limb leads.

Summary

Knowledge of the basics of pediatric ECG interpretation is helpful in differentiating normal from abnormal findings. These basics include familiarity with the age-related normal findings in heart rate, intervals, axis and waveform morphologies; an understanding of cardiac physiologic changes associated with age and maturation, particularly the adaptation from right to left ventricular predominance; and a rudimentary understanding of common pediatric dysrhythmias and findings associated with congenital heart diseases.

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