

Erratum

www.elsevier.com/locate/ajem

# The pediatric electrocardiogram Part I: Age-related interpretation<sup>☆</sup>

## Matthew O'Connor MD<sup>a</sup>, Nancy McDaniel MD<sup>b</sup>, William J. Brady MD<sup>c,\*</sup>

<sup>a</sup>Department of Pediatrics, Children's Medical Center, University of Virginia Health System, Charlottesville, VA 22908, USA <sup>b</sup>Division of Cardiology, Department of Pediatrics, Children's Medical Center, University of Virginia Health System, Charlottesville, VA 22908, USA

<sup>c</sup>Department of Emergency Medicine, University of Virginia Health System, Charlottesville, VA 22908, USA

Received 27 July 2007; accepted 2 August 2007

**Abstract** Emergency physicians attending to pediatric patients in acute care settings use electrocardiograms (ECGs) for a variety of reasons, including syncope, chest pain, ingestion, suspected dysrhythmias, and as part of the initial evaluation of suspected congenital heart disease. Thus, it is important for emergency and acute care providers to be familiar with the normal pediatric ECG in addition to common ECG abnormalities seen in the pediatric population. The purpose of this 3-part review will be to review (1) age-related changes in the pediatric ECG, (2) common arrhythmias encountered in the pediatric population, and (3) ECG indicators of structural and congenital heart disease in the pediatric population.

© 2008 Elsevier Inc. All rights reserved.

### 1. Introduction

Emergency physicians attending to pediatric patients in acute care settings use electrocardiograms (ECGs) for a variety of reasons, including syncope, chest pain, ingestion, suspected dysrhythmias, and as part of the initial evaluation of suspected congenital heart disease. Most providers, however, lack specialized training in pediatric cardiology, and the possibility of errors in interpretation exists because of the relative rarity of cardiac pathology in children as well as the dynamic nature of the pediatric ECG. Several studies have investigated the accuracy of pediatric ECG interpretation in the emergency department

\* Corresponding author.

and found discrepancy rates in the interpretation between emergency department providers and pediatric cardiologists of 13% to 32% [1,2]. Thus, it is important for emergency and acute care providers to be familiar with the normal pediatric ECG in addition to common ECG abnormalities seen in the pediatric population. The purpose of this 3-part review will be to review (1) age-related changes in the pediatric ECG, (2) common arrhythmias encountered in the pediatric population, and (3) ECG indicators of structural and congenital heart disease in the pediatric population.

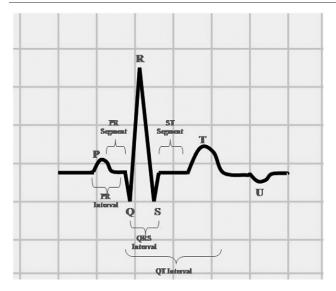
### 2. Interpretation of the ECG

The most important aspect of proper ECG interpretation is ensuring that the ECG is completely reviewed and all

<sup>&</sup>lt;sup>☆</sup> In the February issue (Am J Emerg Med 2008:26;221-8) this article published with incorrect figures. The article with corrected art appears above.

E-mail address: wb4z@virginia.edu (W.J. Brady).

<sup>0735-6757/\$ –</sup> see front matter  ${\ensuremath{\mathbb C}}$  2008 Elsevier Inc. All rights reserved. doi:10.1016/j.ajem.2008.03.030



**Fig. 1** Idealized ECG waveform demonstrating constituent waves and intervals. Standard paper speed is 25 mm/s. Time is represented along horizontal axis; each small block represents 0.04 seconds. Voltage amplitude is represented along the vertical axis; each small block represents 0.1 mV.

necessary data abstracted from it. Most modern ECG platforms provide automated interpretation based upon agerelated pediatric norms; however, the accuracy of computerassisted interpretation is not ideal [3]. The pediatric ECG format is similar to the adult, with the exception that additional right precordial leads (V<sub>3</sub>R, V<sub>4</sub>R) may be used to provide additional information about the right ventricle, disorders of which are frequently seen in children with congenital heart disease. A thorough review of the basics of ECG interpretation can be found in many textbooks. Fig. 1 demonstrates a normal ECG showing gridlines, waves, and intervals.

### 3. Age-related changes

Interpretation of the pediatric ECG is heavily dependent on patient age. The pediatric ECG changes quite dramatically during childhood, particularly during infancy. For example, heart rates considered normal in adults would be likely pathologically bradycardic in the infant. Many of the changes that occur in the ECG reflect the anatomical dominance of the right ventricle during neonatal life. At birth, the right ventricle is thick on account of high pulmonary artery pressure in utero. With the expected fall in pulmonary artery pressure during infancy, right ventricular wall stress and thickness decrease until right ventricular pressure approximates that of the adult, typically by 6 months of age. Electrocardiogram norms for children stratified for age have been compiled [4]. For further discussion of pediatric ECG norms and interpretation, the reader is referred to several excellent textbooks upon which the following discussion is based [5-8].

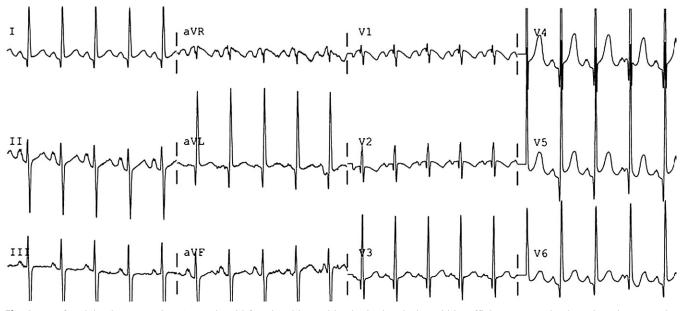
Heart rates are the most obvious manifestation of agerelated variability within the pediatric ECG. The normal mean heart rate for newborn infants 1 to 6 months of age ranges from 125 to 145 beats/min (bpm) with the normal resting heart rate of 80 bpm in adults typically not achieved until mid-adolescence. These changes can be accounted for by the gradual increase in vagal tone that accompanies aging. Young children may also be anxious during ECG acquisition, causing an artifactual increase in the heart rate. Normal values for all aspects of the pediatric ECG are easily found in many handbooks and textbooks.

P waves, which represent atrial depolarization, are typically best reviewed in leads II or V<sub>1</sub>. If the cardiac rhythm originates from the sinus node, the expected vector of depolarization will be from right to left and superior to inferior. Thus, the P wave deflection should be positive (upward) in leads I, II, and aVF. Any other manifestation of P wave orientation suggests a nonsinus site of atrial rhythm. P wave morphology is also important for assessing right and left atrial size (Figs. 2-4). Right atrial enlargement (sometimes referred to as *P pulmonale*) is defined as a tall, broad, and peaked P wave in lead II, with elevations greater than 3.0 mm in infants 0 to 6 months and those greater than 2.5 mm in others marking the upper limits of normal (Fig. 2). Left atrial enlargement (P mitrale) criteria include a widened and deeply notched P wave in lead II or a deep biphasic P wave in lead  $V_1$  (Fig. 3). Right atrial enlargement may be seen with any condition causing right atrial volume overload, as in atrial septal defect, Ebstein's anomaly of the tricuspid valve, or patients who have undergone the Fontan procedure for various forms of single-ventricle lesions. Left atrial enlargement may be seen with either mitral stenosis or mitral regurgitation.

Atrioventricular conduction is assessed via the PR interval. Prolongation of the PR interval suggests either conduction delay or block within the AV node. Simple prolongation of the PR interval indicates first-degree AV block (Fig. 5). Gradual prolongation indicates second-degree AV block, with type I second-degree AV block (Wenkebach) demonstrating progressive PR prolongation proceeding to failure of AV conduction (Fig. 6) and type II



**Fig. 2** Right atrial enlargement in a 5 year old male with cardiomyopathy. Note the peaked, large amplitude p waves in lead II.



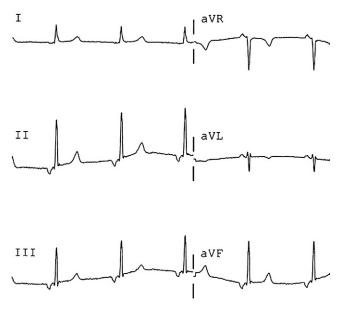
**Fig. 3** Left atrial enlargement in a 9 months old female with combined mitral and tricuspid insufficiency. Note the deep, broad p waves in lead V1 and tall, notched p waves in lead II.

second-degree AV block (Möbitz) demonstrating random failure of AV conduction without PR prolongation. Thirddegree AV block is manifested by AV dissociation, a state in which impulses from the AV node are not conducted to the ventricles (Fig. 7). Diagnosis of this rhythm requires examination of the rhythm strip to confirm AV dissociation, because examination of only a few ECG impulses may demonstrate normal-appearing PR intervals. In children, the PR interval is shorter than that of adults, presumably because of a lesser degree of muscle mass. The PR interval of infants and younger children is approximately 100 milliseconds and lengthens over childhood to the normal adult range of 150 milliseconds. Abnormalities of atrioventricular conduction are frequently seen in the postoperative state after repair of congenital heart disease. The finding of thirddegree heart block, particularly in infants, should prompt an investigation for neonatal lupus and transplacentally acquired antibodies against Ro and La.

Determination of the mean frontal plane QRS axis is an important tool for investigating potential cardiac pathology in children and adults. QRS axis may be determined in several ways, but the most commonly used method at the bedside is to investigate the direction of deflection of the QRS complex in the limb leads. The most isoelectric limb lead is identified, and the mean QRS vector will be orthogonal to this limb lead. An easy to remember rule of thumb is that if the deflections in leads I and aVF are positive, then the QRS axis will be between  $0^{\circ}$  and  $+90^{\circ}$ , which roughly corresponds to "normal axis." Because of the dynamic nature of right ventricular muscle mass and size during childhood, however, normal values for QRS axis change rather dramatically from infancy to adolescence [9], with infants demonstrating a normal rightward axis that

transitions to the expected leftward axis seen in adults (Fig. 8). One abnormality of axis in children that should be recognized is the "extreme superior axis," with a frontal plane QRS axis of  $-90^{\circ}$  to  $-180^{\circ}$  (or  $0^{\circ}$  to  $+270^{\circ}$ ), seen frequently in infants with atrioventricular canal or ostium primum atrial septal defects.

The QRS duration in infants and children is shorter than that of adults, once again, because of decreased cardiac muscle mass. QRS duration typically ranges from 50 to 80 milliseconds in childhood. In children, however, it is



**Fig. 4** Abnormal p wave axis in an 11 year old female presenting with syncope. Note the downward deflection of the p waves in the inferior limb leads.

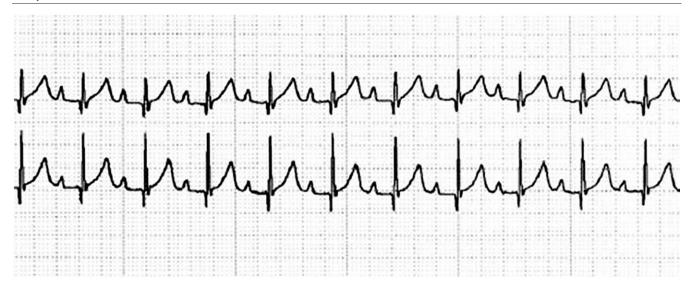


Fig. 5 First degree AV block in a young boy undergoing Holtex monitoring. Note the markedly prolonged PR interval.



**Fig. 6** Type I second degree AV block from a Holter recording in a 7 year old male with congenital completeheart block. Note the progressive prologation of the PR interval during the first two beats.

normal to have mild prolongation of the QRS interval without clinical correlate; these may be labeled "intraventricular conduction delay." Conditions in which pathologic QRS prolongation may be encountered include bundle branch blocks and ventricular preexcitation syndromes, which are discussed further in this series.

The QT interval, measured from the beginning of the QRS complex to the termination of the T wave, represents

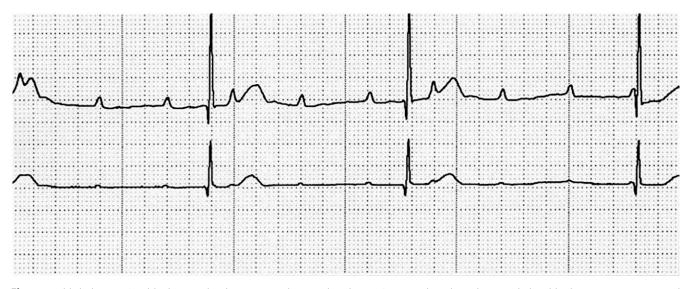
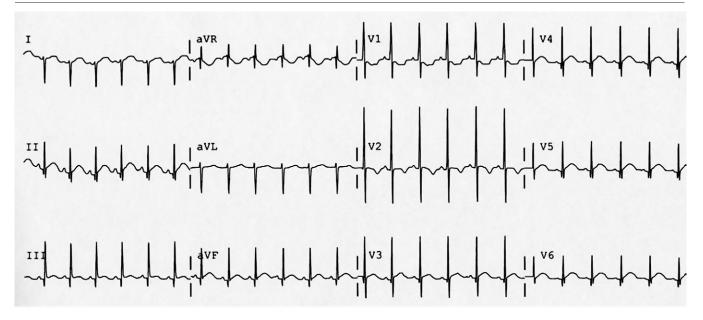


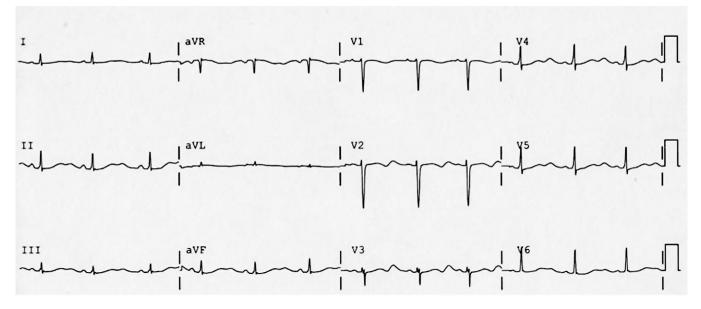
Fig. 7 Third degree AV block seen in the same patient as in Figure 6. Note that there is no relationship between p waves and QRS complexes.



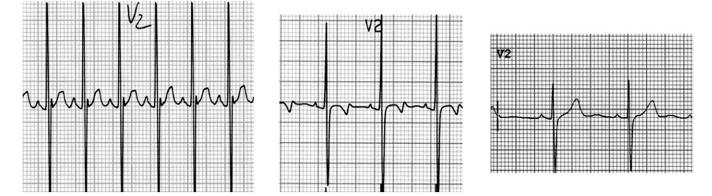
**Fig. 8** Normal neonatal ECG in a one week old infant. QRS axis is 125 degrees, which would be considered right axis deviation in an adult. Note the tall R waves in V1, V2, and V3, which are normal.

ventricular repolarization. As with the other ECG parameters discussed above, it is age dependent, with a QTc of 490 milliseconds being the upper limit of normal for infants younger than 6 months and 440 milliseconds being the upper limit of normal for other age groups. Because the QT interval is heart rate dependent, measurements of the interval must be corrected for the heart rate to obtain the QTc. Numerous methods are available, but the most commonly used is the Bazett's formula, which divides the measured QT interval by the square root of the preceding R-R interval in which the QT is being measured. Specific causes of QT interval prolongation in pediatrics germane to the emergency provider are detailed further in this series. (Refer to Fig. 9 for an example of a prolonged QT interval.)

Although the T wave is of significant interest in adult patients, particularly in ischemic heart disease, its analysis in pediatric patients is of limited value outside the setting of electrolyte abnormalities. It is useful to be aware of the expected changes seen in T-wave morphology throughout childhood (Fig. 10). During the first 7 days of life, the T wave



**Fig. 9** Prolonged QT interval in an 18-year-old female with familial long QT syndrome and a history of cardiac arrest. The QT interval calculated by Bazett's formula is 506 milliseconds.

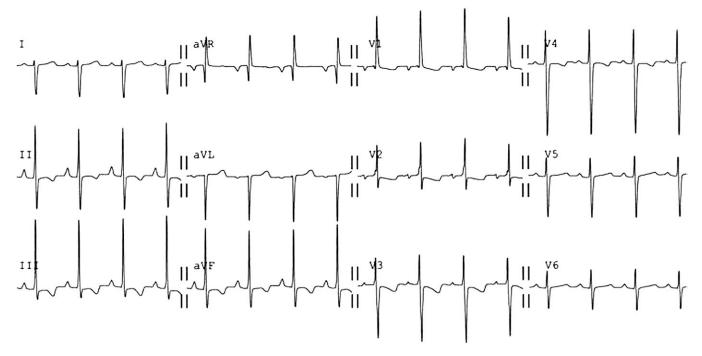


**Fig. 10** T-wave changes in a single patient over childhood. The first panel shows upright T waves in  $V_2$  during the first month of life. The second panel shows inverted T waves in  $V_2$  at 3 years of age. Third panel shows upright T waves at 22 years of age.

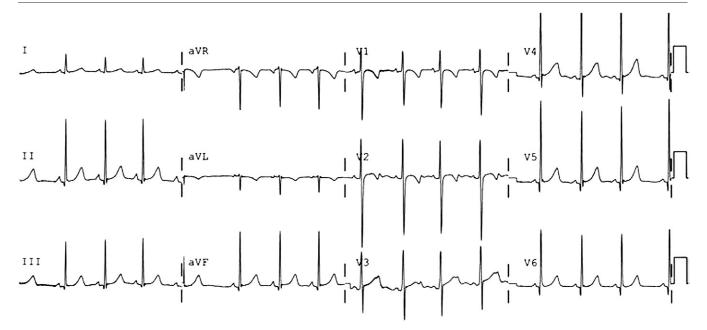
is typically upright in most leads and then develops a downward deflection in most leads until adolescence, when the typical pattern of upright deflection in most leads becomes established. In childhood, upright T waves may be an indicator of right ventricular hypertrophy (RVH) (see below).

One of the more confusing aspects of interpreting the pediatric ECG is proper evaluation of ventricular hypertrophy. Once again, an understanding of developmental changes in the ECG as well as clinical parameters will assist in correlating ECG findings with the patient. For example, the suggestion of RVH via ECG in a 2-day-old newborn or in a young child with unrepaired tetralogy of Fallot would be considered a normal finding; in fact, determination of RVH in infants is difficult, if not impossible, based solely on ECG criteria. Precordial leads are used for the evaluation of ventricular hypertrophy.

A constellation of findings suggests RVH (Fig. 11). Commonly accepted criteria include (1) R-wave height greater than the 98th percentile for age on lead  $V_1$ , (2) S-wave depth greater than the 98th percentile for age in lead  $V_6$ , (3) R/S ratio inappropriately high for  $V_1$  or low for  $V_6$ , (4) T-wave deflection abnormality (see above), and (5) persistence of neonatal pattern of R-wave progression in children in adolescence in which there are tall R waves in the right precordial leads ( $V_1$ ,  $V_2$ ,  $V_3$ ) with small S waves that progress to small R waves and large S waves in the left precordial leads ( $V_4$ ,  $V_5$ ,  $V_6$ ). One caveat should be mentioned. Right axis deviation alone should not be used



**Fig. 11** Right ventricular hypertrophy in a 10-year-old male with primary pulmonary hypertension. Note the tall R waves in  $V_1$  and  $V_2$  and the deep S waves in  $V_5$  and  $V_6$ . Right axis deviation is also present.



**Fig. 12** Left ventricular hypertrophy in a 3-year-old boy with unrepaired coarctation of the aorta. Note the deep S waves in  $V_1$  and  $V_2$  with tall R waves in  $V_4$  and  $V_5$ .

as a criterion for RVH, particularly in infants and young children. Right ventricular hypertrophy may be seen in a number of conditions in children, including congenital lesions such as pulmonic valve stenosis and tetralogy of Fallot, as well as acquired conditions such as primary pulmonary hypertension, which may present in adolescence or late childhood.

Left ventricular hypertrophy (LVH) (Fig. 12) is similarly defined by several criteria, although no set number of criteria must be met to make the diagnosis. Voltage criteria for LVH in children consist of an R-wave height greater than the 98th percentile for age in lead V<sub>6</sub> and an S-wave depth greater than the 98th percentile for age in lead  $V_1$ . The R-wave progression pattern across the precordial leads may also provide helpful information. In newborns, one expects large R waves and small S waves in the right precordial leads. Presence of the inverse pattern (adult-type R-wave progression) is indicative of LVH. T-wave abnormalities are sensitive markers for LVH. Inversion of T waves in leads II, III, aVF, V<sub>4</sub>, V<sub>5</sub>, and V<sub>6</sub> is commonly called a "strain" pattern and is highly suggestive of LVH. Additional electrocardiographic markers of LVH include tall R waves in aVF, left axis deviation, and Q waves in leads V<sub>4</sub>, V<sub>5</sub>, and  $V_6$ .

In summary, the ECG is an inexpensive noninvasive test that provides much information regarding suspected cardiac pathology in both children and adults. In infants and children, appreciation for the rapidly changing parameters within the ECG will aid the clinician in formulating the correct diagnosis.

#### References

- Horton LA, Mosee S, Brenner J. Use of the electrocardiogram in a pediatric emergency department. Arch Pediatr Adolesc Med 1994;148: 184-8.
- [2] Wathen JE, Rewers AB, Yetman AT, et al. Accuracy of ECG interpretation in the pediatric emergency department. Ann Emerg Med 2005;46:507-11.
- [3] Sekiguchi K, Kanda T, Osada M, et al. Comparative accuracy of automated computer analysis versus physicians in training in the interpretation of electrocardiograms. J Med 1999;30:75-81.
- [4] Davignon A, Rautaharju P, Boiseelle E, et al. Normal ECG standards for infants and children. Pediatr Cardiol 1979;1:123-52.
- [5] Allen HD, Gutgesell HP, Clark EB, et al, editors. Moss and Adams' heart disease in infants, children, and adolescents, including the fetus and young adult. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 129-37.
- [6] Park MK, Gunteroth WG. How to read pediatric ECGs. St Louis: Mosby; 1992.
- [7] Chan TC, Brady WJ, Harrigan RA, et al, editors. ECG in emergency medicine and acute care. Philadelphia: Elsevier/Mosby; 2005. p. 37-42.
- [8] Garson Jr A, Bricker JT, Fisher DJ, editors. The science and practice of pediatric cardiology. Philadelphia: Lippincott Williams & Wilkins; 1998. p. 89-101.
- [9] Perloff J, Roberts N, Cabeen Jr W. Left axis deviation: a reassessment. Circulation 1979;60:12-21.