

A
PRIMER
ON
PEDIATRIC
ELECTROCARDIOGRAMS

ver story

A primer on pediatric ECGs

By Bradley Robinson, MD, Paul Anisman, MD, and Eshagh Eshaghpour, MD

If you've ever worried about missing something important on an ECG, this article is for you. The simplified, systematic method it presents will give you all the information you need to interpret ECGs accurately and with confidence.

Do you cringe when asked to read an electrocardiogram, fearing that you may miss a significant finding? Cringe no more. The ECG need not cause Every Clinician to Grimace. The step-by-step approach outlined here will enable you to detect all major ECG abnormalities.

We follow the simplified method of Dr. [Name]. There are only five words you need to memorize:

- Rhythm
- Axes
- Forces
- Repolarization.

Those five words form the basic outline of this primer, as summarized in the checklist on page 70. Before you begin reading, we suggest you review the checklist and the glossary of basic ECG terminology also on page 70. You may also want to look at the summary of normal ECG values at the end of the article. This can be removed and used as a handy reference.



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Reading ECGs: A checklist

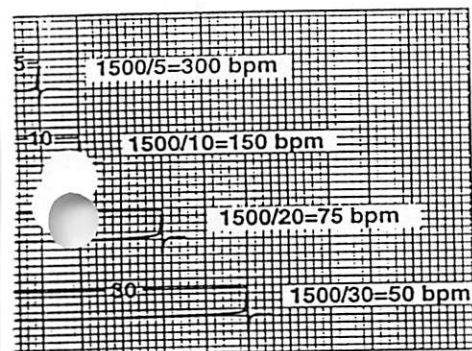
Rate Start by measuring the ventricular rate	Forces/hypertrophy Check for right atrial enlargement (RAE) Check for left atrial enlargement (LAE) Look for right ventricular hypertrophy (RVH) Look for left ventricular hypertrophy (LVH) Look for combined ventricular hypertrophy (CVH) Look for decreased QRS voltages	Repolarization changes/ ischemia* Look for abnormal Q waves Look at the ST segment Look at the T wave Always measure the corrected QT interval (QTc)
Rhythm Check for sinus rhythm Measure the RR interval Measure the QRS duration		
Axes Calculate the QRS axis Calculate the P axis Calculate the T axis		

Rate

Start by measuring the ventricular rate. Count the number of small boxes between two consecutive R waves and divide into 1,500. This will give you the heart rate in beats per minute (bpm). For normal heart rates in infants and children, see page 95.²

If the rhythm is irregular, count six seconds. Use the three-second markers, if present, or count 30 large boxes. Multiply the number of R waves occurring during that time by 10.

Bradycardia is defined as a heart rate below the normal range for age. Common causes of sinus brady-



Basic ECG terminology

The standard surface ECG is run at a paper speed of 25 mm/sec, and 10 mm of height represents 1 millivolt (mV).

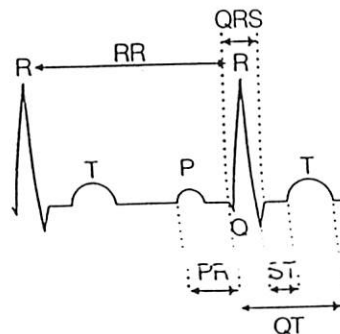
RR interval: The interval from the preceding R wave to the R wave of interest.

PR interval: The interval from the beginning of the P wave to the beginning of the QRS complex.

QRS complex: The interval from the beginning of the Q wave to the end of the S wave. The first deflection, if negative, is the Q wave. The first positive deflection is the R wave.

ST segment: The interval from the end of the S wave to the beginning of the T wave.

QT interval: The interval from the beginning of the Q wave to the end of the T wave.



ECG intervals

ardiac include sleep, sedation, vagal stimulation (such as stooling or coughing), hypothyroidism, and an athletic heart. Pathologic bradycardia may result from second-degree or third-degree atrioventricular (AV) block or junctional rhythm.

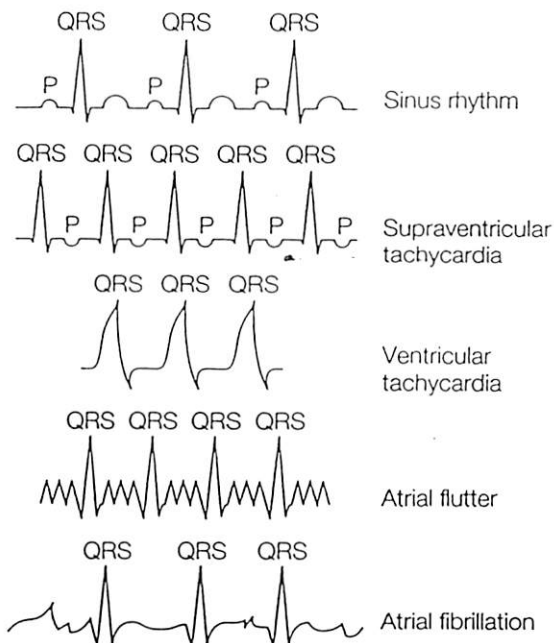
Tachycardia is a heart rate above the normal range for age. Common causes of sinus tachycardia include fever, hypovolemia, sepsis, and medications such as theophylline.³ Other causes include hyperthyroidism and congestive heart failure. You must distinguish sinus tachycardia from pathologic tachycardias, including supraventricular tachycardia, ventricular tachycardia, atrial flutter, and atrial fibrillation. This is done during analysis of rhythm.

Rhythm

Check for *sinus rhythm*. Sinus rhythm is recognized by the following characteristics:

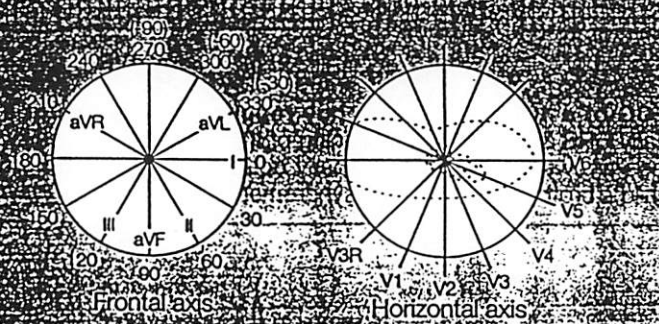
- Every P wave is followed by a QRS complex.
- Every QRS complex is preceded by a P wave.
- The P-wave axis is between 0° and +90°.
- The P-wave morphology is constant.

• In most pathologic tachydysrhythmias, P waves do not precede the QRS complexes and may not be readily visible. When present, they often follow the QRS complex, as in supraventricular tachycardia, or may even be dissociated, as in ventricular tachycardia. Atrial flutter and atrial fibrillation



Q-T interval: The Q-T interval divided by the square root of the preceding RR interval.

ECG leads:
 • Leads I, II, III, aVR, aVL, and aVF (limb leads) are in the frontal plane.
 • Leads II and III are each 60° from the horizontal, respectively.
 • Leads aVR and aVL are 30° from the horizontal.
 • The precordial leads (V₁-V₆) are in the horizontal plane.



Source: Johnson KB (ed): *The Heart Lane Handbook*, ed 13. St. Louis, MO, Mosby, 1993, adapted from Devignon A et al,⁶ used with permission.

are easily distinguished by the presence of flutter and fibrillatory waves.

Measure the PR interval. The PR interval represents conduction time through the atria and the AV node to the His bundle and Purkinje's fibers, immediately up to but not including the myocardium. The PR interval is affected by age and heart rate. An abnormally prolonged PR interval is called first-degree AV block.

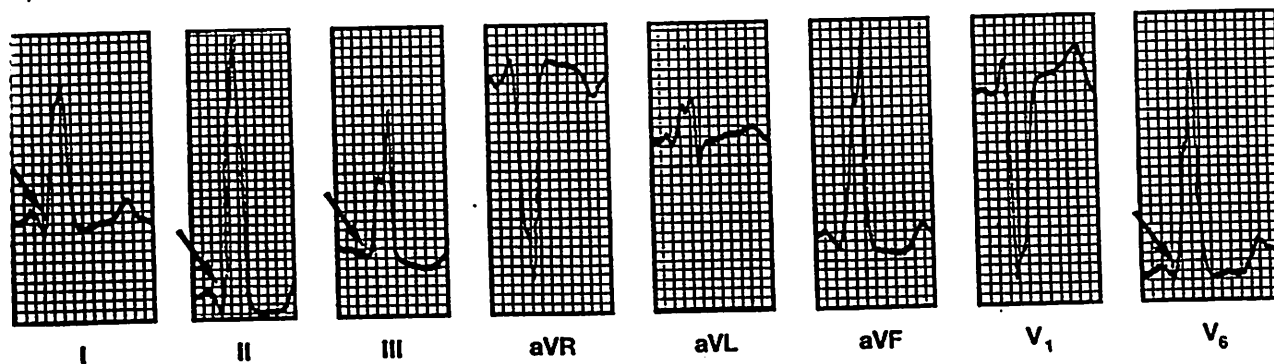
Common reasons for first-degree AV block include:

- Myocarditis
- Digoxin effect
- Hyperkalemia
- Ischemia
- Increased vagal tone
- Hyperthyroidism³

Reasons for an abnormally short PR interval include:

- Ectopic atrial pacemaker
- Preexcitation syndromes such as Wolff-Parkinson-White syndrome (Figure 1) and Lown-Ganong-Levine syndrome
- Glycogen storage diseases.⁴

FIGURE 1.



ECG of a 10-year-old boy reported sensations of a rapid heart beat. Note the shortened PR interval with early QRS activation and widening of the QRS toward the P wave. This upslowed early activation is called a delta wave (see arrows) and represents early activation (preexcitation) of the ventricle in Wolff-Parkinson-White syndrome. The child's brother has the same syndrome!

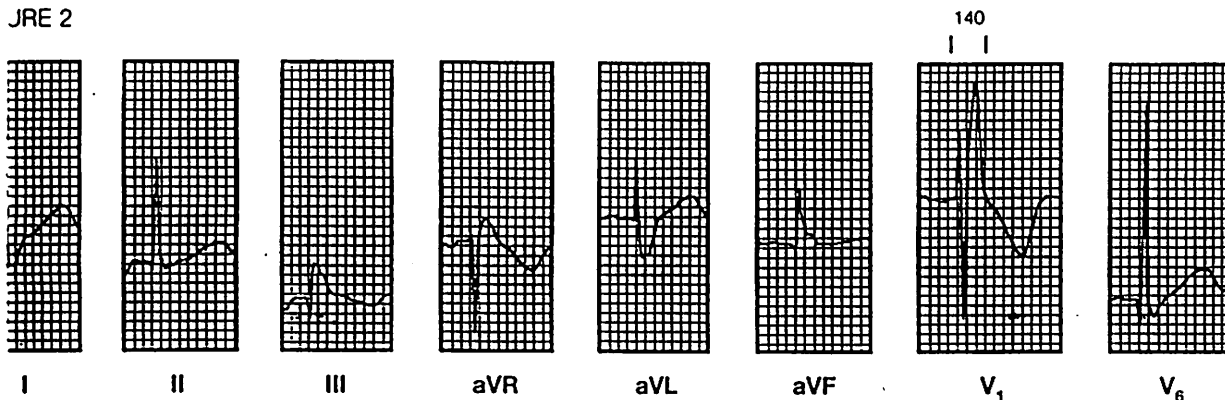
Pearl: A short PR interval may be your clue that the patient is at risk for supraventricular tachycardia.

Measure the QRS duration. The QRS interval represents ventricular conduction time. It is short in

Continued on page 77

at. ... d increases with age.
 The QRS complex may be prolonged in:
 right bundle branch block (RBBB) (Figure 2)
 left bundle branch block (LBBB)
 Wolff-Parkinson-White syndrome
 mechanical pacemaker rhythms
 premature ventricular contractions.⁵

JRE 2



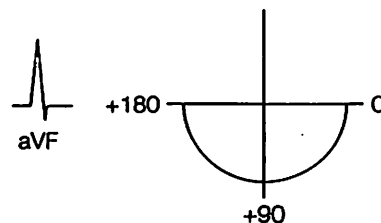
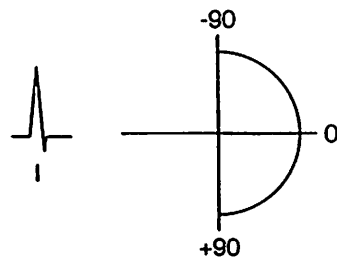
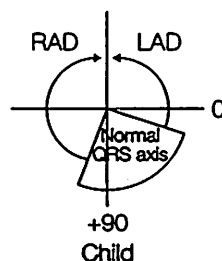
Complete RBBB in a 3-year-old boy after repair of tetralogy of Fallot. The QRS interval is prolonged (140 ms). RBBB is recognized by a prolonged QRS interval with an RSR' pattern in the right precordial lead (V₁) and terminal blunting of the S wave in lead I and the left precordial leads (V₆ above).

... the P axis. Use the procedure described in the following section.

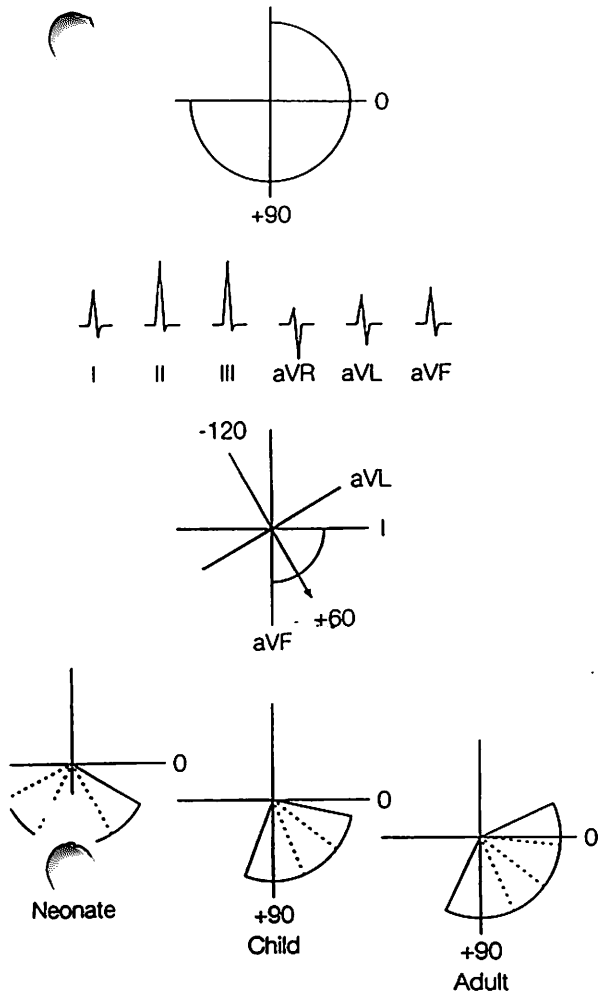
The calculated electrical axis is actually a vector. It is affected by hypertrophy (muscle mass) and location of the heart chambers. It may also be affected by conduction pathway abnormalities. Right axis deviation (RAD) is a QRS axis that lies to the right side the normal range of the axis. Left axis deviation (LAD) is a QRS axis that lies to the left side the normal range of the axis.

Calculate the QRS axis in the frontal plane as follows:

Examine leads I and aVF.
 In lead I, count all the forces above the baseline in boxes (millimeters) and subtract all the forces below the baseline. If this number is positive, the angle of the QRS vector should be between +90° and -90°.
 Do the same in aVF. If this sum is positive, the S wave vector also lies between 0° and +180°.



ADING ECGs



■ Superimpose the ranges. The region of overlap is the quadrant in which the QRS axis will lie.

■ Finally, examine all six leads in the frontal plane (I, II, III, aVR, aVL, aVF).

Find the lead in which the QRS complex is most nearly isoelectric—that is, where positive and negative forces add up to 0 (the aVL lead in this case). The QRS axis will point perpendicular to this lead and lie in the quadrant already identified (+60°).

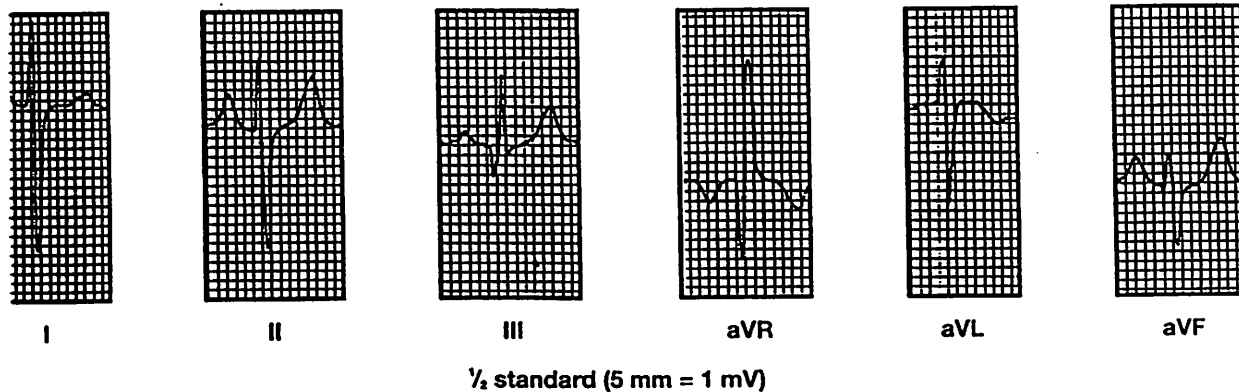
If all leads in the frontal plane are equiphasic, the axis is perpendicular to all leads and perpendicular to that plane. It is directed anteriorly or posteriorly and called indeterminate. This is a nonspecific variation.

The significance of the QRS axis is that it generally reflects muscle mass. More right ventricular (RV) muscle mass shifts the axis to the right. More left ventricular (LV) mass shifts it to the left. That is why newborns, who have relatively more RV muscle mass than adults, have QRS axes farther to the right than adults. Note how the range of the QRS axis changes with age.

Some causes of right axis deviation include:

- Severe pulmonary stenosis with right ventricular hypertrophy (RVH)
- Pulmonary hypertension (Figure 3)
- Conduction disturbances (typically RBBB).⁵

FIGURE 3



ECG of a 5-year-old with severe pulmonary hypertension caused by an unrepaired ventricular septal defect and dilatation of the aorta. The QRS axis at +205° is outside the normal range for age and toward the right according to reference values. Note that this tracing is recorded at half standard sensitivity (5 mm = 1 mV). Therefore when you attempt to assess forces to compare to reference values, you must double all height measurements.

Pearl: Left axis deviation with RvH is highly suggestive of atrioventricular canal malformation (Figure 4). Consider this possibility, especially in patients with Down syndrome.

JRE 4

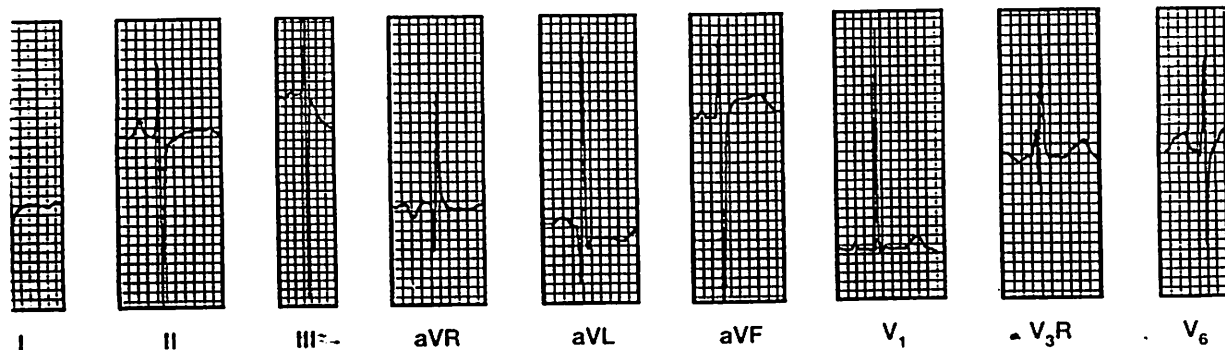


Figure 4: ECG in a 5-week-old infant with atrioventricular canal defect. Note the QRS axis of -70° representing right axis deviation. There is also right ventricular hypertrophy.

UF

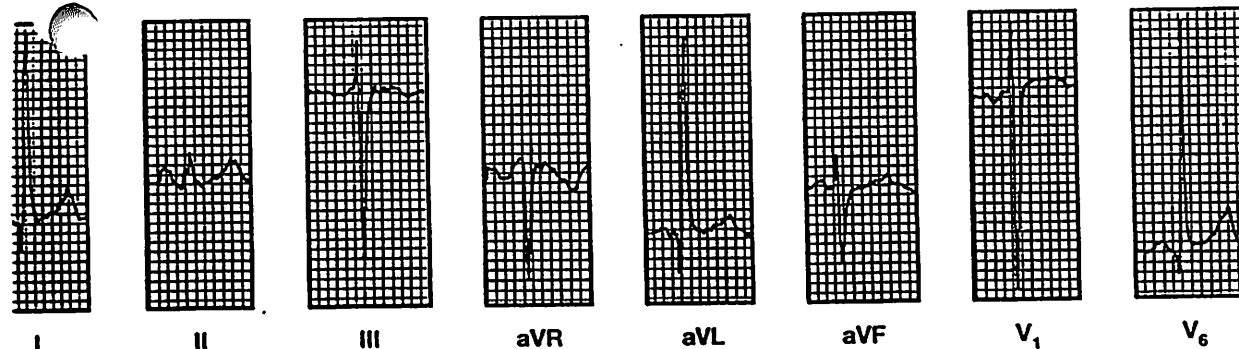
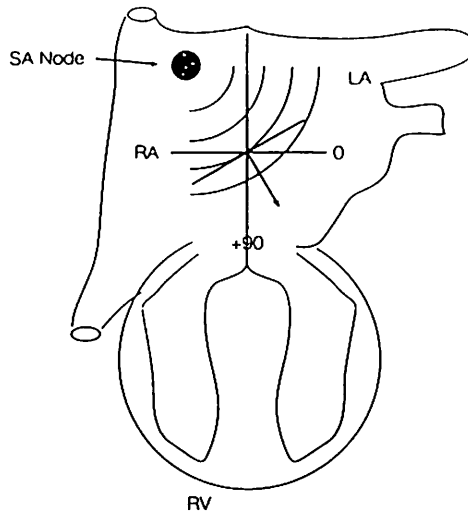


Figure 5: ECG in a 6-week-old child with aortic stenosis. Left axis deviation to -20° and left ventricular hypertrophy are indicated.

Pearl: Mild left axis deviation in a cyanotic infant suggests aortic stenosis (Figure 5).

Calculate the P axis. This calculation is performed during rhythm analysis. A normal P axis (0° to $+90^\circ$) both defines sinus rhythm and describes normally-related atria (atrial situs solitus). That is, regardless of the position of the heart in the chest, the morphologic right atrium (RA) is to the right of the morphologic left atrium (LA). The atrial electrical forces emanating from the sinus node tend to spread from the right upper quadrant toward the left lower quadrant, and the sum of

se. The vector is between 0° and $+90^\circ$. P axis of 0° to -90° may result from an ectopic right atrial pacemaker, which in the absence of SA node dysfunction is not clinically significant. Changing P-wave morphology (changing P-wave axis), called a wandering atrial pacemaker, represents different points of origin of atrial activation. When the heart is structurally normal, this dysrhythmia is considered minor and of no clinical importance.

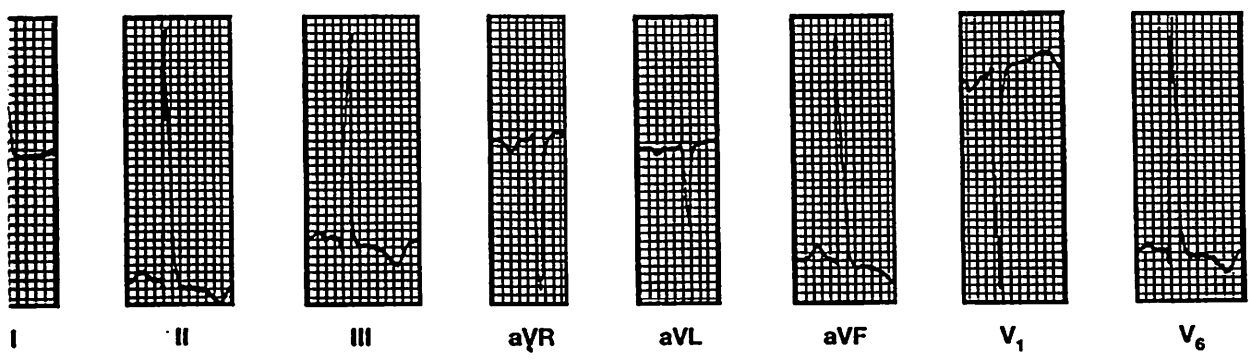


Superimposed frontal P axis showing propagation of sinus beat

Early A P axis greater than $+90^\circ$ strongly suggests atrial inversion or misplaced leads. If the chest X-ray demonstrates normally located abdominal viscera (visceral sinus collitis), the limb leads are most likely misplaced.

Calculate the T axis. A rough estimation of T axis is important to exclude serious myocardial disease. Normally, it lies between 0° and $+90^\circ$ in the frontal plane. If the T-wave axis differs by more than 60° from the QRS axis in the presence of ventricular hypertrophy (see below in the section on chest/hypertrophy), it is called a strain pattern and may be a sign of ischemia. If "strain" is present, examine in particular the left precordial leads (I, II, III, aVR, aVL, aVF, V₁, V₆) for abnormal repolarization, indicated by T wave inversion (Figure 6).

FIGURE 6



ECG of a 19-year-old with aortic stenosis and left ventricular hypertrophy. Note the inverted T waves in the anterior leads (II, III, aVF) and V₆, representing "strain."

Early ECG with strain in patients with aortic stenosis or hypertrophic cardiomyopathy is an ominous finding indicating severe disease and must not be ignored.

Forces/hypertrophy

When rhythm is normal, both atrial and ventricular forces are measured by estimating electrical voltages or forces. Strictly speaking, the area under the wave or complex (the integral) should be measured, but with a normal narrow wave, the height measurement is usually sufficient.

FIGURE 7

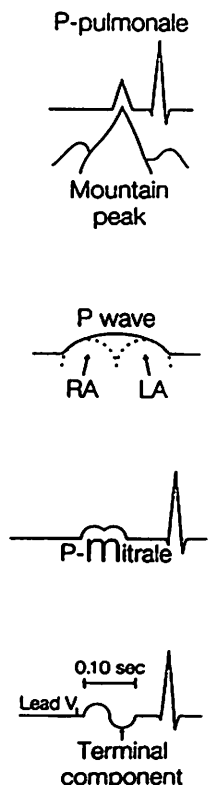


Peaked P waves (arrows) in this ECG indicated right atrial enlargement.

Check for right atrial enlargement (RAE). RAE is defined by a peaked P wave higher than 2.5 mm (2.5 small boxes) in children over 6 months of age. It is usually best seen in the inferior lead II and the anterior precordial lead V_1 (Figure 7). In the first 6 months of life, however, the P wave must reach 3 mm (3 small boxes) for RAE to be present.⁶

Historically called P-pulmonale, tall peaked P waves occur in cor pulmonale because pulmonary hypertension and RVH result in a poorly compliant right ventricle and enlarged right atrium. Another cause of RAE in children is the left-to-right shunt from an anomalous pulmonary venous connection or, less commonly, from a large atrial septal defect.

Pearl: Think of a steep mountain peak and remember peaked P or P-pulmonale.



Check for left atrial enlargement (LAE). LAE is identified by a widened (prolonged) P wave, which may be notched in lead II, or have deep terminal inversion best seen in lead V_1 . Normally the LA depolarizes somewhat later than the RA, so that a large LA component may prolong this complex.⁷ A P-wave duration greater than 0.08 sec (80 ms) signifies LAE in children under 12 months of age. In children over 12 months, a P-wave duration greater than 0.10 sec (100 ms) indicates LAE.⁵

Historically, a notched P wave with an m-shaped deformity was called P-mitrale, signifying mitral stenosis. In children, however, ventricular septal defect and patent ductus arteriosus are more common causes of LAE than mitral stenosis.

Terminal inversion of the P wave alone, without increased duration, is not sufficient to diagnose LAE. When LAE is present, however, the wider and deeper the terminal component of the P wave, the more severe the enlargement.⁸

A diagnosis of combined atrial enlargement, or biatrial enlargement, requires criteria sufficient for both RAE and LAE. That is, there must be a tall

ced wave that is also wide or has associated terminal inversion.⁹

for RVH. Any of the following findings is sufficient to diagnose RVH, and the more criteria that present, the more likely the accuracy of the diagnosis by ECG (RAD supports the diagnosis but not definitive):

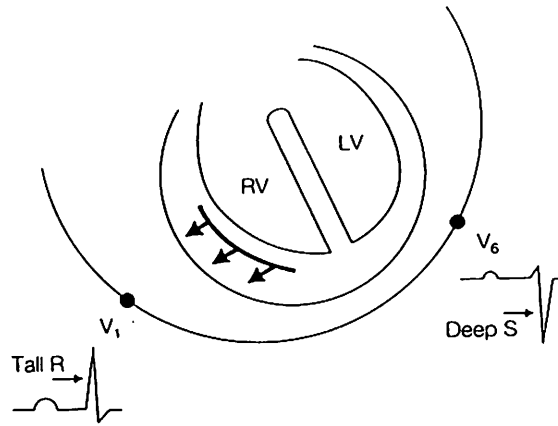
1. An R wave greater than the 98th percentile in V₁ (right precordium) or an S wave greater than the 98th percentile in lead I or V₆ (left precordium). The hypertrophied RV has increased muscle mass and exerts more electrical force toward the right anterior chest wall. This is recorded as a large positive force toward the right seen in V₁ (Tall R) and a strong force away from the left chest (Deep S in V₆).

2. An increased R/S ratio in V₁ (Figure 8) or decreased R/S ratio in V₆.

3. An RSR' wave pattern in V₁ or V_{3R} in the absence of complete RBBB. An RSR' pattern with R' greater than 15 mm in children under 1 year of age or R' greater than 10 mm in children over 1 year is characteristic of RVH caused by right ventricular overload (Figure 4).³ In newborns, a pure R wave greater than 10 mm indicates pressure-overloaded RV.⁵

4. An upright T wave in V₁ in patients older than 3 yrs.¹⁰ The upright T wave adult pattern may, however, occur as early as 6 years of age.⁵

5. A qR pattern of the Q wave in V₁ suggests severe RVH.¹¹ This pattern may be seen in 10% of



Horizontal plane with precordial leads showing RV forces.

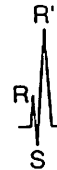
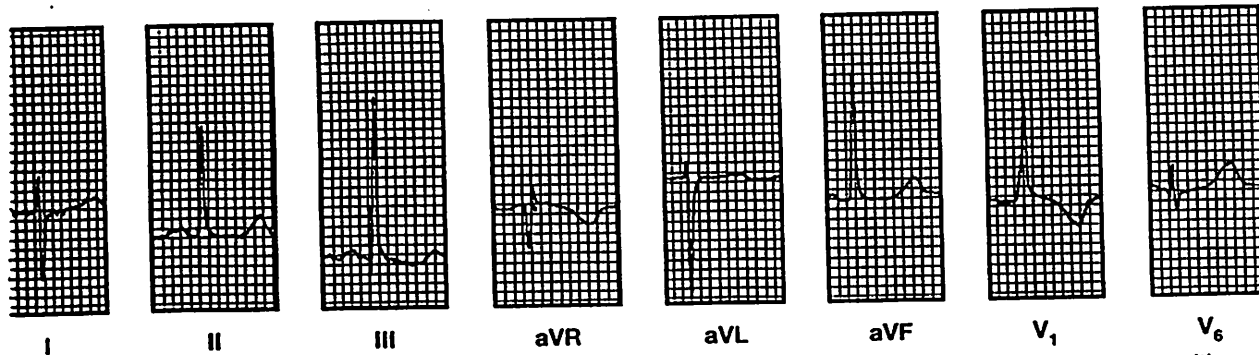


FIGURE 8



This ECG shows a pure R wave representing an increased R/S ratio in V₁ in an 8-year-old with transposition of the great arteries after the intra-atrial baffle procedure (Mustard operation).

normal newborns and in children with complex structural heart disease with ventricular inversion. Usually, when a qR pattern occurs in V_1 and truly represents RVH, additional findings supportive of RVH are present.⁵

Causes of RVH include:

- RV volume overload lesions (atrial septal defect, anomalous pulmonary venous connection)
- RV pressure overload lesions (pulmonary stenosis, tetralogy of Fallot)
- Lesions that increase pulmonary vascular resistance (large ventricular septal defect with pulmonary hypertension)
- Coarctation of the aorta in the newborn.¹²

Look for LVH. Any of the following findings is sufficient to diagnose LVH, but the more criteria that are present, the more likely the accuracy of the diagnosis by ECG:

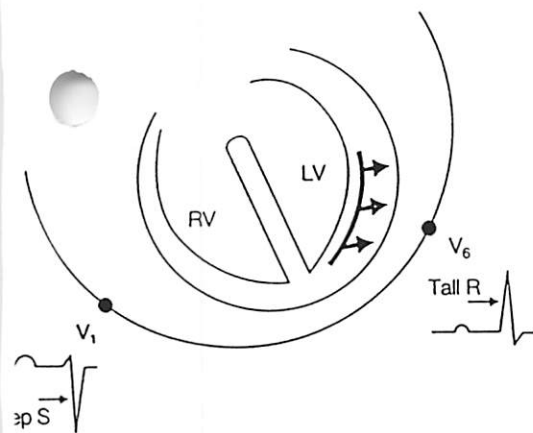
- R wave greater than 98th percentile in V_6 (left precordium)
- S wave greater than 98th percentile in V_1 (right precordium)
- Increased R/S ratio in V_6 or decreased R/S ratio in V_1
- Q wave 5 mm or higher in V_6 with peaked T waves. This occurs with LV diastolic overload and denotes septal hypertrophy.¹³

Flat or inverted T waves in lead I or V_6 in the presence of LVH suggests severe LVH ("strain" pattern). Excessive LAD for age supports a diagnosis of LVH but is not sufficient in itself to make the diagnosis.

Causes of LVH include:

- LV volume overload (ventricular septal defect, patent ductus arteriosus, anemia, complete AV block)
- LV pressure overload (aortic stenosis, systemic hypertension)
- Cardiomyopathies (obstructive and nonobstructive hypertrophic cardiomyopathies).¹²

Look for combined ventricular hypertrophy (CVH). In the presence of RVH, the dominant RV forces usually diminish the apparent LV forces, causing lower LV voltage (small R in V_6 and small S in V_1). If criteria for RVH exist and the LV forces exceed normal *mean* values for age, the patient has CVH. If LVH is present, similar reasoning may apply to diagnose associated RVH.³



horizontal plane with precordial leads showing increased forces away from V_1 and toward V_6 LVH.



Large equiphasic voltages in the limb leads (frontal plane) and midprecordial leads (horizontal plane) are called Katz-Wachtel phenomenon and suggest biventricular hypertrophy.¹⁴ This finding may be subtle because of large but equal left and right ventricle forces, which appear to cancel each other out.

Causes of CVH include left-to-right shunts with pulmonary hypertension (as with a large ventricular septal defect) and complex structural heart disease.¹²

Pearl: It is not possible to accurately diagnose ventricular hypertrophy by ECG in the absence of normal conduction (i.e., with RBBB, LBBB, or preexcitation syndromes) because voltage criteria rely on the normal timing of conduction.

Look for decreased QRS voltages (QRS height of 5 mm or less in limb leads). Causes of decreased QRS voltages include:

- Pericardial effusion
- Myopericarditis
- Hypothyroidism.

Sometimes normal newborn infants have decreased voltages, which do not represent a problem.⁵

Repolarization changes/ischemia

Look for abnormal Q waves. In a heart with normal structure (D-loop ventricle), it is normal to have a small, narrow Q wave in the inferior (II, III, aVF) and leftward leads (I, V₅, V₆). If such a wave is absent in these leads but present in the right precordial leads (V_{3R}, V₁), suspect congenital heart disease with ventricular inversion (L-loop ventricle).

Q waves of new onset greater than 0.035 secs (about one small box) or an increased duration (widening) of previous Q waves, with or without notching of the Q wave, are abnormal and may represent myocardial infarction. Also supportive of myocardial infarction may be ST segment elevation (greater than 2 mm) or prolonged QTc (greater than 440 ms).¹⁵

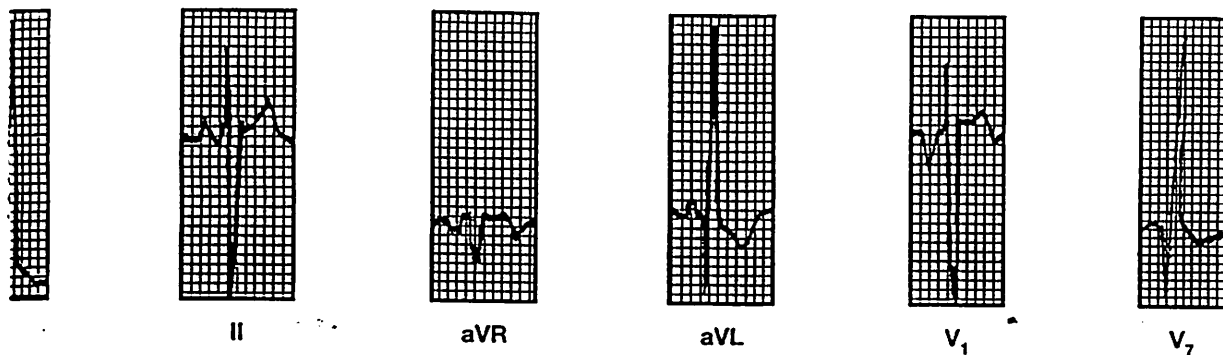
Common causes of ischemia and infarction in children include:

- Anomalous origin of the left coronary artery from the pulmonary artery (Figure 9)
- Coronary artery aneurysm and thrombosis in Kawasaki syndrome

Continued on page 93

cardiomyopathy
 hypertension
 severe aortic stenosis
 myocarditis
 cocaine abuse.¹⁵

FIGURE 9



ECG of a child with an anomalous left coronary artery originating from the pulmonary artery. Note the deep S wave in aVL and the presence of LVH.

A more deep S wave in aVL is a marker for an anomalous origin of the left coronary artery (Figure 9).

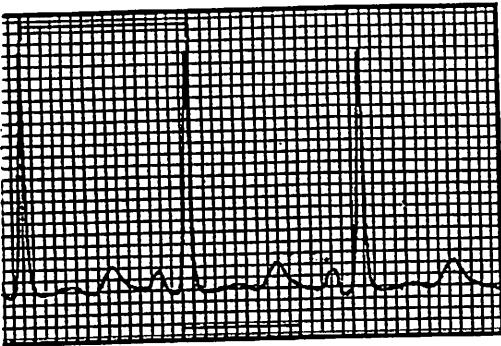
at the ST segment. Pathologic ST-segment changes are defined as a 1-mm displacement of the ST segment in the limb leads or a 2-mm change in precordial leads. ST-T-wave elevation may result from ischemia or from myopericarditis. ST-T-wave depression is consistent with subendocardial ischemia or effects of digoxin.¹

- causes of ST-T-wave abnormalities include:
- myocarditis
 - cor pulmonale
 - myopericardium
 - myothorax
 - myocardial injury
 - abnormal ventricular repolarization and normal atrial repolarization.³

at the T wave. The T wave represents ventricular repolarization. It should always be upright in the inferior (II, III, aVF) and lateral (I, V₅, V₆) leads. T-wave inversion in these leads or a "strain" pattern with LVH is always abnormal. Tall, peaked, pointed T waves may occur with hyperkalemia, LVH, and head injury. Flattened T waves are seen in hypokalemia and hypothyroidism.⁵

FIGURE 10

RR = 0.64



QT = 0.44

Note the long QT interval. QT = 0.44 sec, RR = 0.64 sec, QTc = 0.55.

Always measure the corrected QT interval (QTc). Measure the QT interval in seconds (0.04 secs/box), estimating to the nearest half box. Correct for heart rate by dividing the QT interval by the square root of the preceding RR interval in seconds.

$$QTc = \frac{QT \text{ (secs)}}{\sqrt{RR \text{ (secs)}}}$$

The corrected QT interval is a ratio. Normal corrected ratios are less than 0.45 up to 6 months of age and less than 0.44 in older children (Figure 10).⁶

Common causes of a long QTc interval are:

- Congenital long QT syndrome
- Hypokalemia
- Hypomagnesemia
- Hypocalcemia
- Neurologic injury.⁵

Pearl: A long QTc interval may predispose the patient to ventricular tachycardia and is associated with sudden death. Every patient with syncope should, therefore, be evaluated for congenital long QT syndrome!

Epilogue

Don't expect to absorb all of the information in this article on one reading. Interpreting ECGs takes practice. By following these guidelines and using the reference tables on pages 95 and 96, you will develop confidence. Then the ECG will change from Every Clinician's Grimace to Every Clinician's Gratification. □

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Normal ECG Values

Normal heart rates for infants and children

Age	Heart rate (beats/min)		
	Resting (awake)	Resting (sleeping)	Exercise (fever)
Newborn	100-180	80-160	Up to 220
1wk to 3 mo	100-220	80-200	Up to 220
3 mo to 2 yr	80-150	70-120	Up to 200
2 yr to 10 yr	70-110	60-90	Up to 200
10 yr to adult	55-90	50-90	Up to 200

Source: Gillette PC et al.² p 926, used with permission

RHYTHM PR interval with rate, age, and (upper limits of normal)

Rate	0-1 mo	1-6 mo	6 mo-1 yr	1-3 yr	3-8 yr	8-12 yr	12-16 yr	Adult
<60						0.16 (0.18)	0.16 (0.19)	0.17 (0.21)
60-80					0.15 (0.17)	0.15 (0.17)	0.15 (0.18)	0.16 (0.21)
80-100	0.10 (0.12)				0.14 (0.16)	0.15 (0.16)	0.15 (0.17)	0.15 (0.20)
100-120	0.10 (0.12)			(0.15)	0.13 (0.16)	0.14 (0.15)	0.15 (0.16)	0.15 (0.19)
120-140	0.10 (0.11)	0.11 (0.14)	0.11 (0.14)	0.12 (0.14)	0.13 (0.15)	0.14 (0.15)	^a	0.15 (0.18)
140-160	0.09 (0.11)	0.10 (0.13)	0.11 (0.13)	0.11 (0.14)	0.12 (0.14)			(0.17)
160-180	0.10 (0.11)	0.10 (0.12)	0.10 (0.12)	0.10 (0.12)				
>180	0.09	0.09 (0.11)	0.10 (0.11)					

Lower limits of normal PR interval by age: <3 yr: 0.08 sec 3-16 yr: 0.10 sec >16 yr: 0.12 sec

Source: Park MK, Guntheroth WG.³ as adapted from Guntheroth WG: *Pediatric Electrocardiography*. Philadelphia, WB Saunders Co, 1965, used with permission

RHYTHM QRS duration: Average (and upper limits) for age

	0-1 mo	1-6 mo	6 mo-1 yr	1-3 yr	3-8 yr	8-12 yr	12-16 yr	Adult
Seconds	0.05 (0.065)	0.05 (0.07)	0.05 (0.07)	0.06 (0.07)	0.07 (0.08)	0.07 (0.09)	0.07 (0.10)	0.08 (0.10)

Source: Park MK, Guntheroth WG.³ as adapted from Guntheroth WG: *Pediatric Electrocardiography*. Philadelphia, WB Saunders Co, 1965, used with permission

AXES Frontal QRS axis: Normal values for age

Age	2%	Mean	98%
<1 day	59	137	-167
1-2 days	64	134	-161
3-6 days	77	132	-167
1-3 wk	65	110	161
1-2 mo	31	74	113
3-5 mo	1	60	104
6-11 mo	1	56	99
1-2 yr	1	55	101
3-4 yr	1	55	104
5-7 yr	1	65	143
8-11 yr	1	61	119
12-15 yr	1	59	130

Source: Johnson KB (ed): *The Harriet Lane Handbook*, ed 13, St. Louis, Mosby Yearbook, Inc, 1993, p 102-104, adapted from Davignon A et al.⁶ used with permission

AXES T axis: Normal values for age

Age	V ₁ , V ₂	aVF	I, V ₅ , V ₆
Birth-1 day	±	+	±
1-4 days	±	+	+
4 days-adolescent	-	+	+
Adolescent-adult	+	+	+

+ = T wave positive; - = T wave negative, ± = T wave normally either positive or negative

Source: Johnson KB (ed): *The Harriet Lane Handbook*, ed 13, St. Louis, Mosby Yearbook, Inc, 1993, p 102-104, adapted from Davignon A et al.⁶ used with permission

AXES P axis

Normal frontal P-wave axis in sinus rhythm = 0.90

Continued

FORCES/HYPERTROPHY R and S voltages by lead and age: Mean and (upper limits)*

Lead	voltages							Young Adult
Lead	0-1 mo	1-6 mo	6 mo-1 yr	1-3 yr	3-8 yr	8-12 yr	12-16 yr	Young Adult
LI	4 (8)	7 (13)	8 (16)	8 (16)	7 (15)	7 (15)	6 (13)	6 (13)
I	6 (14)	13 (24)	13 (27)	13 (23)	13 (22)	14 (24)	14 (24)	9 (25)
VR	8 (16)	9 (20)	9 (20)	9 (20)	9 (20)	9 (24)	9 (24)	6 (22)
VL	3 (7)	3 (6)	3 (6)	2 (6)	2 (5)	2 (4)	2 (4)	1 (4)
VF	2 (7)	4 (8)	5 (10)	5 (10)	3 (10)	3 (10)	3 (12)	3 (9)
4R	7 (14)	10 (20)	10 (16)	8 (20)	10 (19)	10 (20)	11 (21)	5 (23)
I	6 (12)	5 (10)	4 (8)	4 (8)	3 (8)	3 (7)	3 (7)	
1	15 (25)	11 (20)	10 (20)	9 (18)	7 (18)	6 (16)	5 (16)	3 (14)
2	21 (30)	21 (30)	19 (28)	16 (25)	13 (28)	10 (22)	9 (19)	6 (21)
5	12 (30)	17 (30)	18 (30)	19 (36)	21 (36)	22 (36)	18 (33)	12 (33)
6	6 (21)	10 (20)	13 (20)	12 (24)	14 (24)	14 (24)	14 (22)	10 (21)

Lead	voltages							Young Adult
Lead	0-1 mo	1-6 mo	6 mo-1 yr	1-3 yr	3-8 yr	8-12 yr	12-16 yr	Young Adult
4R	5 (10)	4 (9)	4 (9)	3 (8)	2 (8)	2 (8)	2 (8)	1 (6)
I	4 (9)	4 (12)	5 (12)	5 (12)	5 (14)	6 (20)	6 (20)	
1	10 (20)	7 (18)	8 (16)	13 (27)	14 (30)	16 (26)	15 (24)	10 (23)
2	20 (35)	16 (30)	17 (30)	21 (34)	23 (38)	23 (38)	23 (48)	14 (36)
5	9 (30)	9 (26)	8 (20)	6 (16)	5 (14)	5 (17)	5 (16)	
6	4 (12)	2 (6)	2 (4)	2 (4)	1 (4)	1 (4)	1 (5)	1 (13)

Voltages are measured in millimeters, when 1 mV = 10 mm paper
 Source: Park MK, Guntheroth WG, as adapted from Guntheroth WG: *Pediatric Electrocardiography*. Philadelphia, WB Saunders Co, 1965, used with permission

FORCES/HYPERTROPHY R/S ratio by age: Mean, lower, and upper limits of normal

Lead	0-1 mo	1-6 mo	6 mo-1 yr	1-3 yr	3-8 yr	8-12 yr	12-16 yr	Adult
LI	0.5	0.3	0.3	0.5	0.1	0.15	0.1	0.0
I	1.5	1.5	1.2	0.8	0.65	0.5	0.3	0.3
ULN	19	S = 0	6	4	2	1	1	1
LLN	0.3	0.3	0.3	0.3	0.05	0.1	0.1	0.1
2 Mean	1	1.2	1	0.8	0.5	0.5	0.5	0.2
ULN	3	4	4	1.5	1.5	1.2	1.2	2.5
LLN	0.1	1.5	2	3	2.5	4	2.5	2.5
6 Mean	2	4	6	20	20	20	10	9
ULN	S = 0	S = 0	S = 0	S = 0	S = 0	S = 0	S = 0	S = 0

Lower limits of normal. †Upper limits of normal.
 Source: Park MK, Guntheroth WG, as adapted from Guntheroth WG: *Pediatric Electrocardiography*. Philadelphia, WB Saunders Co, 1965, used with permission

REPolarization/ISCHEMIA Q voltages by lead and age: Mean and (upper limits)*

Lead	0-1 mo	1-6 mo	6 mo-1 yr	1-3 yr	3-8 yr	8-12 yr	12-16 yr	Adult
I	2 (5)	3 (8)	3 (8)	3 (8)	1.5 (6)	1 (5)	1 (4)	0.5 (4)
VF	2 (4)	2 (5)	2 (6)	1.5 (5)	1 (5)	1 (3)	1 (3)	0.5 (2)
5	1.5 (5)	1.5 (4)	2 (5)	2 (6)	2 (6)	2 (4.5)	1 (4)	0.5 (3.5)
6	1.5 (4)	1.5 (4)	2 (5)	2 (4.5)	1.5 (4.5)	1.5 (4)	1 (2.5)	0.5 (3)

Voltages measured in millimeters when 1 mV = 10 mm paper
 Source: Park MK, Guntheroth WG, as adapted from Guntheroth WG: *Pediatric Electrocardiography*. Philadelphia, WB Saunders Co, 1965, used with permission

REPolarization/ISCHEMIA Normal T waves by lead and age

Lead	<1 yr	>1 yr
5	11 mm	14 mm
6	7 mm	9 mm

Source: Park MK, Guntheroth WG, as adapted from Guntheroth WG: *Pediatric Electrocardiography*. Philadelphia, WB Saunders Co, 1965, used with permission

REPolarization/ISCHEMIA Corrected QT interval (QTc)

$$QTc = \frac{\text{measured QT (sec)}}{\sqrt{\text{RR interval (sec)}}}$$

QTc should not exceed: 0.45 in infants under 6 mo.
 0.44 in children
 0.425 in adolescents and adults