AMERICAN HEALTH CONSULTANTS® A Medical Economics Company

PEDIATRIC

Emergency Medicine Reports™

The Practical Journal of Pediatric Emergency Medicine

Volume 4, Number 7 July 1999

*The electrocardiogram (ECG) is an important study in the evaluation of many pediatric disease processes. Critical decisions in the care of the child in the emergency department (ED) depend on accurate and timely analysis, yet, reading pediatric ECGs presents a number of challenges to emergency physicians because of the differences between children and adults, variants of normal ECGs, and changes that occur with age. The resulting confusion can delay diagnosis and lead to inappropriate or unnecessary care. This article reviews basic pediatric ECG interpretation and focuses on the accurate identification of rhythm disturbances. The majority of the discussion is organized by presenting symptoms recognizing the problem-focused nature of an ED visit. Extensive tables summarize the important aspects of ECG interpretation, and an ECG supplement provides sample tracings to further enhance the clinical skills of the emergency physician.*

*—The Editor*

# Pediatric ECGs and Rhythm Disturbances

*Authors:* **Sarah W. Alander, MD,** Instructor of Pediatrics, Division of Emergency Medicine, Children's Mercy Hospital; **James Edward Hulse, III, MD,** Associate Professor of Pediatrics, Department of Cardiology, Children's Mercy Hospital; **Jane F. Knapp, MD,** Professor of Pediatrics, Division of Emergency Medicine, Children's Mercy Hospital, Kansas City, MO.

*Peer Reviewer:* **James C. Perry, MD,** Director of Cardiology, Electrophysiology, Children's Hospital San Diego, CA.

## Introduction

Common reasons for obtaining an ECG on a child include: chest pain, irregular or rapid heart beats, seizure, syncope, cyanosis, drug exposure, electrolyte disturbances, and an abnormal cardiac examination.[1]

These indications foster an approach to pediatric ECG interpretation that emphasizes recognition of abnormal findings and identification of disease processes that require a cardiac evaluation. A wide variety of chief complaints will be used to illustrate common and uncommon pediatric ECG abnormalities and rhythm disturbances in addition to emphasizing the relevant differences between children and adults. The major drawback for this model is that there are rhythm disturbances that occur with more than one chief complaint. To eliminate repetition, each major category includes a comprehensive differential and the most important or representative examples for each situation.

## The Basics of the Pediatric ECG

A systematic approach is essential to ECG interpretation. This includes an analysis of rate, rhythm, axis, forces, and repolarization. Textbooks, handbooks, and short reference articles provide excellent and concise reviews of the basics and contain the necessary tables listing normal, age-related values.[2-4] A favorite pediatric ECG text is an essential reference for the ED.

The child's ECG changes over time with the normal growth of the heart. Changes in pulmonary and systemic blood pressures produce corresponding changes in the ECG, such that the characteristics of normal change every few years.[5]

Interpretation begins by establishing the heart rate and determining whether it is normal or abnormal for the age of the patient. Heart rates requiring rapid cardiopulmonary assessment are listed by age in Table 1.

Check that the rhythm originates in the sinoatrial node by noting the axis of the P wave and that the shape of the P wave is

constant. The P wave axis should be 0 to $^{+}90°$ in patients with normal atrial depolarization. This can be checked quickly by noting if the P wave is isoelectric or positive in the frontal leads I and aVF. A P wave axis greater than $^{+}90°$ or less than 0° may be an indication of misplaced limb leads. Each P wave should be followed by a QRS complex and every QRS complex should be preceded by a P wave.

Measure the PR interval from the beginning of the P wave to the beginning of the QRS complex by counting the small boxes and multiplying by 0.04 seconds/box. The PR interval varies by age and heart rate. Pediatric cardiology or ECG textbooks provide normal values for age and heart rate. Usually, the PR interval increases with age and decreases with heart rate. Short PR intervals under 0.10 seconds can be an indication that the patient is at risk for supraventricular tachycardia (SVT). Abnormally long PR intervals are consistent with a diagnosis of first degree heart block.

Next, measure the duration of the QRS complex beginning with the first deflection, the Q wave, to the end of the S wave. QRS duration increases with age. Determine if the QRS interval is prolonged; any QRS interval over 100 milliseconds or 2.5 boxes in length is prolonged.

The corrected QT interval (QTc) should always be calculated. This ratio is obtained by dividing the QT interval in seconds by the square root of the preceding RR interval in seconds. The QTc should be less than a maximum value for age and gender. For children younger than 15 years, the normal value is less than 0.44 seconds, borderline is 0.44-0.46 seconds, and a prolonged value is greater than 0.46.[6] For female patients older than 15 years, borderline values may extend to 0.47 seconds. A long QTc interval is a risk factor for ventricular arrhythmias or may be a marker for the familial long QT syndrome, a condition associated with sudden unexpected death.

The six frontal plane leads are I, II, III, aVR, aVL, and aVF. These will be used to determine the QRS axis. Lead I is examined first. Subtract the number of boxes below the baseline from the number above the baseline. If the number is positive, the range of QRS axis is $^{+}90°$ and $^{-}90°$. Lead aVF is examined in the same manner. If the sum of the boxes is positive, the QRS axis is between 0° and $^{+}180°$. Next, the frontal lead with the most isoelectric QRS complex is identified. The QRS axis will point perpendicular to this lead and lie in the quadrant already identified from determining the vector forces of leads I and aVF.

Many variants of normal may be present in a pediatric ECG. It is normal to have Q waves in leads II, III, and aVF in neonates. Small Q waves in $V_5$ and $V_6$ are expected after 2-3 days of life. Deep Q waves in $V_5$ and $V_6$ can be seen with improperly high lead placement on the chest.

ST segments should not be elevated higher than 1 mm or depressed more than 0.5 mm in any lead in children. *(See Table 2.)* African-American adolescents are an exception. They commonly have a nonpathologic elevation of the ST segment due to early repolarization. Hallmarks of early repolarization (generally benign) are a 1-2 mm elevation at the point where the QRS meets the ST segment (also called the J point). This is usually seen in the inferior and lateral precordial leads, and usually not in I and aVL.

T waves are normally inverted in children older than 4 days of age in leads $V_{4R}$ and $V_1$. It is not uncommon for T waves to be inverted throughout the right side of the chest in infancy. Inverted T waves in leads $V_{4R}$-$V_3$ can persist throughout adolescence. The T wave should always be upright after the first week of life in the inferior (II, III, aVF) and lateral (I, $V_5$, $V_6$) leads. T wave inversion in these leads is always abnormal after the first week of life. The axis of the T wave is normally between 0° and +90° in the frontal plane. If the T wave axis differs by more than 60° to 90° from the QRS axis in the presence of ventricular

## Table 1. Heart Rates Requiring Rapid Cardiopulmonary Assessment

| AGE | HEART RATE |
|---|---|
| Newborn | < 80 bpm or > 200 bpm |
| 0-1 year | < 80 bpm or > 180 bpm |
| 1-8 years | < 80 bpm or > 180 bpm |
| > 8 years | < 60 bpm or > 160 bpm |

Adapted from *Pediatric Advanced Life Support Manual.* Dallas: American Heart Association; 1997.

***Pediatric Emergency Medicine Reports™*** (ISSN 1082-3344) is published monthly by American Health Consultants, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

**Publisher:** Brenda Mooney
**Executive Editor:** Park Morgan
**Managing Editor:** David Davenport
**Associate Managing Editor:** Suzanne Zunic
**Marketing Manager:** Schandale Kornegay

**GST Registration No.:** R128870672

Periodical Postage Pending at Atlanta, GA 30304.

**POSTMASTER:** Send address changes to ***Emergency Medicine Reports***, P.O. Box 740059, Atlanta, GA 30374.



**Back issues: $23.**
Missing issues will be fulfilled by customer service free of charge when contacted within one month of the missing issue's date.

### Accreditation

***Pediatric Emergency Medicine Reports'™*** continuing education materials are sponsored and supervised by American Health Consultants. American Health Consultants designates this continuing education activity as meeting the criteria for 20 credit hours in Category 1 for Education Materials for the Physician's Recognition Award of the American Medical Association, provided it has been completed according to instructions. This CME activity was planned and produced in accordance with the ACCME Essentials. ***Pediatric Emergency Medicine Reports*** is also approved by the American College of Emergency Physicians for 20 hours of ACEP Category 1 credit. This continuing medical education activity has been reviewed by the American Academy of Pediatrics and is acceptable for 20 AAP Credit hours. These credits can be applied toward the PREP Education Award available to Fellows and Candidate Fellows of the American Academy of Pediatrics.

American Health Consultants is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

### Subscriber Information

Customer Service: 1-800-688-2421
**Customer Service E-Mail Address:** customerservice@ahcpub.com
**Editorial E-Mail Address:** david.davenport@medec.com
**World-Wide Web page:** http://www.ahcpub.com

### Subscription Prices

1 year *with* 20 ACEP, AMA, or AAP
Category 1 credits: $329;
1 year without credit: $279;

2 years *with* ACEP, AMA, or AAP
Category 1 credits: $658;
2 years *without* credit: $558;

3 years *with* ACEP, AMA, or AAP
Category 1 credits: $987;
3 years *without* credit: $837.

**Multiple copies:**
One to nine additional copies: **$251 each;**
10 or more additional copies: **$223 each.**

All prices U.S. only. U.S. possessions and Canada, add $30 postage plus applicable GST.
Other international orders, add $30.



### Questions & Comments

Please call **David Davenport,** Managing Editor, at (404) 262-5475 between 8:30 a.m. and 4:30 p.m. ET, Monday-Friday.

### Statement of Financial Disclosure

American Health Consultants does not receive material commercial support for any of its continuing medical education publications. In order to reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Drs. Alander, Hulse, and Knapp (authors) and Dr. Perry (peer reviewer) report no consultant, stockholder, speaker's bureau, research, or other financial relationships with companies having ties to this field of study.

## Table 2. Causes of ST Changes in Children

| | |
|---|---|
| Infarction/ischemia/contusion | Cor pulmonale |
| Myocarditis | Pericarditis |
| Head injury | Digitalis |
| Hyper- or hypokalemia | Emetine intoxication |
| Pneumothorax | Pneumopericardium |
| Duchenne's muscular dystrophy | Myotonic dystrophy |
| Hyperthyroidism | Collagen disease |
| Early ventricular repolarization | Normal atrial repolarization |

## Table 3. Criteria for Ventricular Hypertrophy

RIGHT VENTRICULAR HYPERTROPHY

**Primary Criteria** *(at least one)*

- R in $V_1$ above the 98$^{th}$ percentile for age
- S in $V_6$ above 98$^{th}$ percentile for age
- Upright T in $V_1$ after three days of age until adolescence

**Secondary Criteria** *(support the diagnosis in the presence of primary criteria)*

- R/S ratio in $V_1$ above the 98$^{th}$ percentile for age
- R/S ratio in $V_6$ < 1 after one month of age
- A qR pattern in $V_1$
- Normal duration RSR' in $V_{3R}$ or $V_1$ with R' > 15 mm if , < 1 year, > 10 mm thereafter

LEFT VENTRICULAR HYPERTROPHY

**Primary Criteria**

- R in $V_6$ above 98$^{th}$ percentile for age
- S in $V_1$ above 98$^{th}$ percentile for age
- Inverted T waves in $V_6$

**Secondary Criteria**

- R/S ratio in $V_1$ below 2$^{nd}$ percentile for age
- Q wave > 4 mm in $V_5$ or $V_6$

Adapted from *Harriet Lane Handbook.* 14th ed. St. Louis: Mosby; 1996.

hypertrophy, a strain pattern exists that may be a sign of ischemia.

The criteria for chamber enlargement are age related.[7] *(See Table 3.)* The ECG correlates with chamber size better in children than in adults, but the predictive value is 70% at best. Therefore, an ECG indicative of chamber hypertrophy must be correlated with the clinical situation. Voltage criteria that are used to diagnose ventricular hypertrophy rely on normal conduction. Therefore, when conduction is abnormal, such as in right bundle branch block, ventricular hypertrophy cannot be accurately diagnosed.

Right ventricular hypertrophy (RVH) occurs with atrial septal defect, pulmonary stenosis, tetralogy of Fallot, large ventricular septal defects with pulmonary hypertension, and coarctation of the aorta in the newborn. Left ventricular hypertrophy (LVH) occurs with ventricular septal defect, patent ductus arteriosus, complete AV block, aortic stenosis, and hypertrophic cardiomyopathies (HCM).

The ECG is most effectively used to assess heart rate and rhythm. The limitations of ECG in the presence of structural heart disease must be recognized. The ECG can be confusing and difficult to interpret with malposition of the heart and/or structural defect. Especially in young infants, an abnormal or unusual appearance should heighten the suspicion of congenital heart disease and prompt consideration of performing an echocardiogram.

Recognition of artifact eliminates another source of confusion in ECG interpretation. The most common type of artifact results from incorrect positioning of the limb leads, especially reversing the right arm and the left arm or the right leg and the left leg. When this happens, only the limb lead tracings are affected. Since lead 1 and $V_6$ have similar vectors, if the complexes in lead 1 and $V_6$ are completely different, check to see if the arm leads are reversed. If the chest leads are placed too high, R waves are diminished and S waves are increased in the left chest leads and the reverse if they are too low. A P wave axis of greater than +90° suggests atrial inversion, abnormal atrial depolarization, or misplaced leads.

## Chest Pain

The overwhelming majority of chest pain in children is noncardiac in origin.[8,9] Obtaining an ECG on all children with chest pain is reassuring but offers very low yield and is not cost effective.[10] In most instances a detailed history and physical examination are sufficient to rule out heart disease and determine the cause. A history of exertional chest pain, syncope, presyncope, palpitations, cardiac illness or anomaly, previous cardiac surgery, Kawasaki's disease, street-drug use, or family history of early, sudden, unexpected death in a patient with chest pain suggests the possibility of cardiac origin. In children referred for cardiologic evaluation of chest pain, pericarditis, mitral valve prolapse, myocardial infarction, aortic stenosis, and tachyarrhythmias are the most common cardiac-related diagnoses.[11] In a large study of pediatric emergency department (PED) patients with MI secondary to Kawasaki's disease, chest pain was the most common symptom in children older than 4 years of age.[12] Children younger than 4 years presented with shock, vomiting, and chest pain/crying hard. The ECG is an important component of the diagnostic work-up of children with a suspected cardiac cause of chest pain.

ECG abnormalities in patients with cardiac-related chest pain include: a rapid rate with SVT, abnormalities of the T wave (usually inverted in the inferior and lateral leads) in patients with mitral valve prolapse, LVH in patients with severe aortic stenosis or HCM, RVH in patients with pulmonary stenosis, the presence of a prolonged QT interval, or inverted T waves in myocardial ischemia. *(See Figure 1 in enclosed supplement.)*

**Myocardial infarction.** ECG criteria have been established for the diagnosis of childhood MI.[13] *(See Table 4.)* These include new appearance of wide Q waves (more than 35 ms in duration), increased amplitude or duration of pre-existing Q waves, new onset Q waves in serial tracings, Q wave notching, ST segment elevation greater than 2 mm, and prolonged QTc longer than 0.44 seconds when associated with any other criterion. ST segments are elevated in leads overlying the infarction. There may be reciprocal ST segment depression in leads opposite the infarction. T wave changes are common in normal children and are too nonspecific to be helpful in diagnosing MI.

## Table 4. ECG Criteria for MI in Children

- New appearance of wide Q waves (> 35 ms)
- Increased amplitude or duration (> 35 ms) of pre-existing Q waves
- New onset Q waves in serial tracings
- Q wave notching
- ST segment changes of ≥ 2 mm when associated with other criterion
- Calculated QTc of > 0.44 sec when associated with other criterion

Source: Towbin JA, Bricker JT, Garson A. Electrocardiographic criteria for diagnosis of acute myocardial infarction in childhood. *Amer J Cardiol* 1992;69:1545-1548.

## Table 5. Normal Heart Rates Based on Age

| | RANGES OF NORMAL HEART RATE | | |
|---|---|---|---|
| **Age** | **Awake** | **Sleeping** | **Mean** |
| Newborn to 3 mos. | 82-205 | 80-160 | 140 |
| 3 mos to 2 yr. | 100-190 | 75-160 | 130 |
| 2 yr. to 10 yr. | 60-140 | 60-90 | 80 |
| > 10 yr. | 60-100 | 50-90 | 75 |

Adapted from Pediatric Advanced Life Support Manual. Dallas; American Association: 1997.

Pediatric criteria differ from adult criteria in that there is not a requirement for Q waves in more than one lead, ST segment depression is not included, and T wave inversion is not considered particularly helpful.

**Pericarditis.** Pericarditis is classically characterized by chest pain that is exacerbated by lying down and relieved by sitting up and leaning forward. Pericarditis is the most common pathologic cause of ST segment elevation in children, occurring in more than 90% of patients.[14]

The ECG changes of acute pericarditis occur in four stages, usually following a progression of early, widespread ST segment elevation, and followed over time by T wave inversion. In stage I, there is ST segment elevation most characteristically in leads II, aVF, and $V_3$ to $V_6$. Leads aVR and $V_1$ may show reciprocal ST depression. PR segment elevation greater than 0.8 mm can also be seen during stage 1. Stage 2 shows normalization of ST segments and T wave flattening. *(See Figure 2 in enclosed supplement.)* Stage 3 progresses to T wave inversion in leads II or III, aVF, and $V_3$ to $V_6$. Stage 4 is resolution.

The QRS complex can be unaffected by pericarditis, even in patients with large pericardial effusions.

Pericarditis is differentiated from MI on ECG by the presence of ST elevation in numerous leads. In MI the ST elevation is limited to the leads overlying the infarction. The ST segment elevation in the first stage of pericarditis is usually associated with an upright T wave, whereas beyond the hyperacute phase of MI the T wave is usually inverted.

## Tachyarrhythmias

Supraventricular tachycardia (SVT) is a nonspecific term, referring only to a sustained accelerated rhythm that involves structures above or within the level of the atrioventricular (AV) junction. Further classification of SVT is based on the anatomic site and specific mechanism (automatic or reentry) of the tachycardia.[15,16] There are at least 16 different types of SVT, but they may be grouped into three basic categories: Reentry SVTs that use an accessory pathway, reentry SVTs that do not use an accessory pathway, and automatic tachycardias.[17]

Reentrant SVTs account for 90% of the arrhythmias seen in the pediatric population. The incidence is estimated at 1:250-1000 children.[16] Mechanisms of SVT have age-dependent distributions: Reentry tachycardia using an accessory pathway (i.e., Wolff-Parkinson-White) accounts for 80% of SVT in infants. After infancy, there is a gradual increase of reentry tachycardias that do not use an accessory pathway, so that by adolescence, this mechanism accounts for up to one-third of the episodes of SVT.[18,19] Because the etiology of SVT is variable, obtaining an ECG prior to and after treatment is helpful in delineating the cause and determining subsequent management. A continuous lead II tracing should be obtained during treatment.

Clinical presentation can be similar between sinus tachycardia and SVT; differentiating these conditions may be difficult for the clinician evaluating the child with a narrow complex tachycardia. The clinician must have a thorough understanding of the differences in a child's heart rate based on the age of the child as well as an understanding of the underlying conditions, anxiety, fever, sepsis, or shock, which may enhance sinus node activity. Normal heart rates based on age are shown in Table 5.

**Sinus tachycardia.** Figure 3 in the supplement shows sinus tachycardia. In infants, the heart rate is usually less than or equal to 200 bpm. The P wave is often difficult to identify but when seen as in Figure 3, the morphology is normal and the axis is a normal 0° to +90° (deflection toward leads I and aVF and away from lead aVR). Automatic tachycardia may have beat-to-beat variability in the rate, and the rhythm will gradually slow as the underlying cause is managed.

**Supraventricular tachycardia.** Accessory pathway tachycardia, the most common mechanism of SVT in children, is shown in Figure 4 in the supplement. In SVT, a narrow complex tachycardia, the heart rate is usually greater than 230 bpm in infants, greater than 200 bpm in young children, and greater than 180 bpm in teenagers.[17] If P wave morphology can be identified, it is abnormal. *(See Figure 4 in enclosed supplement.)* While most children have narrow QRS complexes during SVT, it is not uncommon for children younger than 1 year of age to have a wider QRS with a left bundle branch block pattern (wide and positive in $V_6$).[17]

**Wolff-Parkinson-White.** The same patient with a normal sinus rhythm shows the underlying Wolff-Parkinson-White (WPW) pattern in Figure 5 in the supplement. Recall that patients with WPW have an accessory AV connection. This abnormal connection conducts faster than the AV node, so there is abnormally rapid transit of the wave of depolarization from the atria to the ventricles. The "pre-excitation" shortens the PR interval (for age) and causes the initial slurring—the delta wave—of the QRS complex.[20]

**Ventricular tachycardia.** Fortunately, ventricular tachycardia (VT), as seen in Figure 6 in the supplement, is uncommon in the pediatric population. The majority of children presenting

with VT have an underlying cardiac defect. Severe metabolic derangement and certain drug toxicities also can cause VT. Clinical presentation is varied: Infants may be able to tolerate long periods of VT, whereas adolescents tend to present with symptoms similar to adults.[21]

Because VT is rare, it is often misdiagnosed and treated as SVT in children. Wide complex tachycardia should be considered VT until proven otherwise, regardless of symptoms. Especially in infants the "wideness" of the QRS complex may be subtle; a high degree of suspicion and careful attention to measurement of the QRS is important.[21] Figure 6 in the supplement shows sustained VT. At least three consecutive ventricular complexes are necessary for the diagnosis.

**Atrial flutter.** Atrial flutter can be seen in infants with structurally normal hearts and is usually benign. However, it is more commonly seen in post-operative congenital heart disease patients and is discussed under this heading.

**Ventricular fibrillation.** Ventricular fibrillation (VF) is less common in children than in adults. Children are more likely to have bradycardia followed by asystole as a terminal cardiac rhythm, and this may be mistakenly read as fine VF. When VF does occur, it is usually secondary to a preceding VT.[17] VF, as seen in Figure 7 in the supplement, is characterized by rapid irregular rate and rhythm with bizarre QRS complexes of varying size and configuration.

## Syncope

Syncope is defined as a sudden, nontraumatic, reversible loss of consciousness resulting in impairment of cerebral function.[22,23] Exact frequency in the pediatric population is not known, but as many as 15% of children may have an episode before adulthood.[24] Most pediatric syncopal episodes are benign, resulting from autonomic dysfunction (i.e., neurocardiogenic syncope) that leads to a short, reversible period of cerebral hypoxemia.[25] However, syncope can be a marker for serious underlying cardiac pathology in a small group of patients. For this reason, a careful history, physical examination, and the use of a 12-lead ECG provide the complete information necessary to avert a catastrophic cardiac event. Syncope that occurs during exercise is a potentially ominous sign.

Patients with the more common neurocardiogenic syncope have structurally normal hearts and, thus, are expected to have a normal ECG. Patients with cardiac pathology usually have either a tachy- or bradyarrhythmia that leads to syncope. While a child of any age may have SVT heart block or sick sinus syndrome as the underlying cause of syncope, age may be a useful clue for certain diagnoses.[25,26] These causes are listed in Table 6.

**Prolonged QT interval.** Historically, a QTc greater than 0.44 sec is considered to be abnormal.[27] Recent studies show that the QT interval is influenced by gender and age as discussed in "The Basics of the Pediatric ECG" section in this issue. A prolonged QTc *(see Figure 8 in enclosed supplement)* represents abnormal electrical recovery of the ventricle, leaving the myocardium vulnerable to develop ventricular tachyarrhythmias, particularly when provoked by sympathetic stimuli. Patients with syncope as a result of a prolonged QTc will often give a history of pain, physical exertion, or emotional stress provoking the event.[26] Patients with a QTc greater than 0.50 sec appear to be most at risk for sudden cardiac death. Most concerning is the potential for these patients to develop torsade de pointes, a polymorphous form of ventricular tachycardia that is difficult to treat successfully.[25] *(See Figure 9 in enclosed supplement.)*

### Table 6. Cardiac Causes of Syncope Based on Age

| INFANT | CHILD | ADOLESCENT |
|---|---|---|
| Heart block | Heart block | Heart block |
| Sick sinus syndrome | Sick Sinus syndrome | Sick sinus syndrome |
| SVT (WPW) | SVT | SVT |
| Long QT | Long QT | Long QT |
| Tetralogy spell | Cardiomyopathy | Hypertrophic cardiomyopathy |
| Myocardial tumor | Myocarditis | Aortic stenosis |
| | Other ventricular tachycardias | Myocarditis |
| | | Other ventricular tachycardia |
| | | Vasovagal syncope |

Prolonged QTc can be congenital or acquired. The congenital forms are inherited in a dominant mode of transmission. Acquired prolongation of the QTc is associated with metabolic derangements such as hypocalcemia, hypomagnesemia, hypokalemia, and hypothermia. Numerous drugs, such as antiarrhythmics, cisapride, antifungals, and erythromycin, also prolong the QTc .[28]

**Heart block.** A wide range of congenital or acquired etiologies can cause atrioventricular (AV) conduction abnormalities in children. Congenital heart disease (either pre- or postoperative), cardiomyopathy, myocarditis, Lyme carditis, and certain drug ingestions are a few representative examples that may result in a variety of AV blocks.[29] Children without pre-existing risk factors and structurally normal hearts can also have heart block. In the patient presenting with syncope, both Mobitz type II second degree AV block and complete heart block (third-degree block) should be included in the differential diagnosis.[23,26]

Mobitz type II second degree AV block is defined as intermittent loss of AV conduction without a preceding lengthening of the PR interval for at least two consecutive beats.[29] While not very common in children, it causes concern when identified because of its association with Stokes-Adams attacks and sudden cardiac death.[29]

Complete heart block may be congenital, with an incidence of 1 in 22,000 live births. With current monitoring standards during pregnancy and delivery, most cases of congenital heart block are identified early; it would be unusual for this condition to first present in the ED. However, some patients may not be symptomatic until later in childhood, at which time syncope may be the presenting symptom. Heart block may also occur in the setting of structural heart disease, particularly in patients with L-transposition of the great vessels. Surgical repair of a VSD, AV canal, transposition of the great vessels, or the Fontan procedure for single ventricle, may place the child at risk for complete heart block due to anatomical involvement or proximity of the surgical site to the AV node.[29] With complete heart block, the atria and ventricles beat independently of each other. Therefore, the atrial rhythm is regular, as noted by a normal PP interval, and the ventricular rate is also regular as noted by a regular, but much slower RR interval.[2] *(See Figure 10 in*

## Table 7. ECG Findings with Common Ingestions

| DRUG | ECG FINDINGS |
|---|---|
| Antihistamines | Sinus tachycardia, wide QRS |
| Cyclic antidepressants | *Mild:* Sinus tachycardia, long QT<br>*Moderate:* SVT, prolonged QRS<br>*Severe:* BBB, AV block, ventricular bradycardia, asystole |
| Class IA antiarrhythmics (quinidine, procainamide) | Long QT, prolonged QRS, AV block, VT, VF, torsade de pointes |
| Phenothiazines | Long QT, prolonged QRS, AV block, VT, VF, torsade de pointes, widened, flat, or inverted T waves, prominent U wave |
| Digoxin | *At therapeutic doses*<br>Bradycardia, inverted T waves, shortened QT, depressed ST segment<br>*At toxic doses*<br>Prolonged PR, ectopic rhythms, bidirectional VT, SA block |
| Beta blockers | Bradycardia, AV block, ventricular rhythm disturbances |
| Calcium channel blockers | Bradycardia, AV block, ventricular rhythm disturbances |
| Phenytoin | Long QT, prolonged QRS, ventricular rhythm disturbances |

## Table 8. Congenital Heart Repairs Commonly Associated with Post-Operative Rhythm Disturbances

| DEFECT | PERCENT INCIDENCE |
|---|---|
| Transposition of great vessels (intra-atrial repair) | 50-85% |
| Tetralogy of Fallot | 30-60% |
| Fontan repair for single ventricle | 25-40% |

Adapted from Vetter VL. What every pediatrician needs to know about arrhythmias in children who have had cardiac surgery. *Pediatric Annals* 1991;20:378-384.

*enclosed supplement.)*

**Aortic stenosis.** Aortic stenosis is a general term used to describe several lesions of the left ventricular outflow tract. Accounting for 3-8% of all patients with congenital heart disease, these lesions are further categorized anatomically as valvar, fibromuscular subvalvar, and supravalvar aortic stenosis. Clinically, pediatric patients can be divided into in two groups: those who develop critical symptoms of congestive heart failure in infancy (10-15%) and those who develop symptoms in adolescence and young adulthood.

Of particular concern for the emergency physician is the teenager with aortic stenosis. A previously healthy patient may present with exercise-induced syncope or anginal chest pain and be at risk for sudden cardiac death. This patient's ECG will show left ventricular hypertrophy: R is of greater than normal amplitude in leads $V_5$ and $V_6$, and S is greater than normal in $V_1$ and $V_2$. The patient may also show the "strain" pattern: flat or inverted T waves or ST depression in the left precordial leads. *(See Figure 11 in enclosed supplement.)*

## Irregular Heartbeat

**Sinus arrhythmia.** Sinus arrhythmia is most frequently a variation of normal. Sinus arrhythmia is sometimes referred to as respiratory sinus arrhythmia because it represents a cyclic variation of the sinus rhythm with respiration. The rate increases toward end inspiration and decreases toward end expiration. The ECG shows progressive shortening of the PP intervals followed by prolongation of the PP intervals. Sinus arrhythmia is more pronounced with a slow heart rate and tends to disappear with increased heart rate. It is found in virtually all normal school age children during 24-hour electrocardiograms.[30,31] Sinus arrhythmia, though most commonly seen in normal children, occurs in the diseased heart as well.

Sinus pauses averaging more than one second in duration also have been reported in healthy children on 24-hour ECG monitoring.[32]

**Second degree AV block.** An irregular ventricular rhythm occurs with second degree AV block. Mobitz type I and Mobitz type II second degree AV blocks are distinguished by ECG findings. Mobitz type I second degree AV block, also known as Wenkebach, is characterized by progressive lengthening of the PR interval terminating in a blocked P wave. The consequence is a missed QRS complex or dropped beat. The grade of the second degree block is quantified by the ratio of all P waves to conducted P waves. There are at least three P waves in a Wenkebach sequence, two of which are conducted, producing a 3:2 block. Another ECG feature is that the RR interval becomes progressively shorter before the blocked P wave. In most cases, Mobitz type I second degree AV block is a benign, nonprogressive phenomenon in children with otherwise normal hearts.[33]

**Extrasystoles.** Extrasystoles are premature beats and can be either atrial, AV junctional, or ventricular in origin. Most extrasystoles have a uniform shape on the ECG. When more than one pattern is observed, they are termed multiform. Atrial extrasystoles are defined by early P waves. The early P wave frequently looks different than other normally conducted P waves. It may be followed by a normal appearing QRS complex, one of a different shape, or by no QRS. When the abnormal P wave is superimposed on a T wave and the QRS morphology of the extrasystolic beat is a different shape, it can be difficult to differentiate between an atrial and a ventricular extrasystole.

Ventricular extrasystoles are identified by a QRS complex with a different shape from the prevailing rhythm that is not preceded by a P wave. Ventricular extrasystoles may be found on the routine ECG in 1-2% of normal children.[34] Most of them are uniform and infrequent. Multiform ventricular extrasystoles are rare in children and raise the concern of heart disease. The differential for ventricular extrasystoles includes: congenital heart disease, especially after cardiac surgery; undiagnosed myocar-

## Table 9. ECG Changes with Electrolyte Disturbances

| POTASSIUM CHANGES | |
|---|---|
| **Hyperkalemia** | **Hypokalemia** |
| Prolonged QRS | Long QT |
| ST changes | Prolonged QRS |
| AV block | ST changes |
| VT | |
| Peaked T waves | |
| Low-voltage P waves | |
| **CALCIUM** | |
| **Hypercalcemia** | **Hypocalcemia** |
| Short QT | Long QT |
| Sinus bradycardia | Sinus tachycardia |
| AV block | AV block |
| VT | |
| **MAGNESIUM** | |
| **Hypermagnesemia** | **Hypomagnesemia** |
| AV block | Long QT |

dial disease; mitral valve prolapse; Marfan's syndrome; long QT syndrome; drug ingestions; hypoxia; and acidosis. In particular, a healthy patient presenting with ventricular extrasystoles should be carefully screened for prolonged QT syndrome.

## Trauma

Cardiac involvement must be considered in the child who has sustained a serious or focal blunt or penetrating injury to the torso. Although the ECG is frequently abnormal after chest trauma, it is not sufficiently sensitive or specific to confirm the diagnosis of myocardial contusion.

**Myocardial contusion.** Most children will experience symptoms of myocardial contusion soon after injury.[35] The child with myocardial contusion who is conscious and verbal commonly complains of precordial chest pain. The most common ECG finding is a sinus tachycardia, which is excessive for age and unexplained by other factors. The most common rhythm disturbance is frequent, premature ventricular complexes. Signs of myocardial injury similar to an infarction pattern with ST-segment elevation, T wave changes, or presence of abnormal Q waves may be present. *(See Figure 12 in enclosed supplement.)*

**Other chest injury.** A penetrating wound to the heart can produce atrioventricular conduction defects at the level of the injury. Ventricular rhythm disturbances can also occur.

Acute pericarditis and cardiac tamponade can occur with either blunt or penetrating injury to the chest. ECG changes may be seen with the accumulation of fluid under pressure in the pericardium. As previously noted, the QRS complex can be normal or low-voltage. Electrical alternans is an alteration of electrical amplitude of the T wave and QRS complex with each cycle that occurs with large effusions.

Air is a poor conductor of electrical activity. Therefore, in patients with a pneumomediastinum or pneumothorax when air is between the heart and the recording electrode, the amplitudes of the P, QRS, and T waves may be decreased, depending on the location of the air. ST segment changes can also occur.

**Head injury.** Sinus bradycardia occurs as part of Cushing's triad in response to increased intracranial pressure accompanying serious head injury. Patients with head injury may also exhibit ST segment changes and prolongation of the QT interval.

## Ingestions

A wide range of drugs and other substances ingested by children produce cardiac effects. In the previously healthy child who presents with a rhythm disturbances, the ECG can be helpful to formulate a differential diagnosis and to guide management.

Rhythm disturbances from drug effects can be caused by direct or indirect sympathomimetic effects, anticholinergic effects, the effects of altered central nervous system regulation of the peripheral autonomic system, and direct effects on the myocardial membranes.[36-40] Some drugs produce toxicities by more than one of these mechanisms. Clinical management should be based on countering the mechanism of drug action; access to detailed pharmacologic information is essential in the ED. *(See Table 7.)*

## Post-Operative Congenital Heart Disease Rhythm Disturbances

Approximately 25,000 infants are born each year in the United States with congenital heart disease (CHD) and nearly half of them require urgent palliative or corrective surgery.[41] Remarkable advances have occurred in the field of congenital heart surgery in the past generation, resulting in increased lifespan for nearly all types of cardiac defects.[42] While it is still not an everyday occurrence for the pediatric emergency medicine physician to treat children for post-operative rhythm disturbances, a basic understanding of what ECG findings are expected behind the sternotomy scar is important.

The post-operative CHD patient may present to the ED for a problem that may be related to the heart—chest pain, palpitations, chest trauma, syncope, or symptoms related to the use of cardiac medication. The patient may also present for unrelated reasons and be noted to have a coincidental rhythm disturbance. Historical factors, such as the specific defect, pre-existing rhythm disturbance, type of surgical repair performed, and the age at repair, aid in developing an approach to the child. The patient's prior ECG should be obtained if possible.

All patients who have had intracardiac repair are at risk to develop supraventricular rhythm disturbances. Most repairs use a right atrial surgical approach or bypass cannulation, which may result in interference with atrial conduction. Children who undergo surgical repair within the ventricle are of particular concern; they may have intracardiac conduction impairment due to surgical interruption of the specialized conduction system (His-purkinje system). The impairment seen is often a delay in depolarization of the right ventricle, resulting in a right bundle branch block pattern (RBBB) on the ECG. *(See Figure 13 in enclosed supplement.)* The ECG shows permanent widening of the QRS with a wide and slurred S in leads I, $V_5$, and $V_6$.[42] The differentiation of VT from SVT with BBB can be a diagnostic dilemma in the patient with sustained tachycardia. It is prudent to approach all wide complex tachycardia in children as ventricular rhythm disturbance until proven otherwise.

The congenital heart defects commonly associated with post-

operative rhythm disturbances are shown in Table 8.

**Transposition of the Great Vessels.** Until the mid-1970s intra-atrial procedures (Mustard, Senning) were exclusively used to repair a transposition of the great vessels. In the last decade, arterial switch procedures have largely supplanted these procedures in a successful effort to minimize postoperative complications. Intra-atrial procedures involve suture lines close to the SA node, across the arterial supply to the SA node, and across extensive portions of the atrial walls. Sinus node dysfunction and SVT are frequent complications with these procedures. Progressive scarring may interrupt the normal intra-atrial conduction, leaving the heart vulnerable to the development of atrial reentry circuits (atrial flutter) over a variable period of time—up to years later.[41-43] These patients present to the ED with complaints of palpitations or fluttering in the chest, syncope, shortness of breath, chest pain, or dizziness. Figure 14 in the supplement shows an example of atrial flutter. The configuration is seen best in leads $V_1$, II, and III. It is characterized by an atrial rate of about 300, giving the "saw-tooth pattern" and normal QRS complexes with varying degrees of block.[43] Because this rhythm is associated with sudden death, patients who have had intra-atrial procedures with a resting heart rate higher than 100 should be carefully evaluated for atrial flutter. Patients who have undergone arterial switch procedures have a much lower risk of atrial flutter,[43] although they may develop other supraventricular rhythm disturbances.

**Tetralogy of Fallot.** The ECG of uncorrected tetralogy of Fallot will show right ventricular hypertrophy and right axis deviation.[2,42] The infant with a "tet spell" may present to the ED with a cyanotic episode that may be described as a syncopal episode. After surgical correction, the vast majority will have RBBB.[44] Up to two-thirds of the patients will have premature ventricular contractions (PVC).[41] Sudden cardiac death has been observed in 2-5% of these postoperative patients, and is attributed to ventricular rhythm disturbances.[45,46]

**Fontan.** The Fontan-type repair involves directing the systemic venous return from the superior vena cava and inferior vena cava to the pulmonary arteries. These procedures often result in marked atrial dilatation or extensive suture lines with scarring. Given this anatomic alteration, these patients are at high risk for sinus node dysfunction or atrial rhythm disturbance. Because the single ventricle may have had long-standing ventricular volume overload and subsequent fibrosis, these patients are at risk for ventricular disturbances as well.[41-43]

## Critically Ill Infants

**Myocarditis.** Myocarditis can be a severe and rapidly fatal infection in newborns and young infants. In older children, the course varies from clinically minor to fulminant. ECG changes occur in 70% of children with myocarditis and reflect the injury to the myocardial tissue or the conduction system.[14] A sinus tachycardia out of proportion to age or level of fever is a hallmark of myocarditis and should prompt a search for other ECG findings that support the diagnosis. The ECG may reveal low voltage QRS complexes, less than 5 mm total amplitude, in all the limb leads. Low voltages may also be present in the precordial leads. Leads $V_5$ and $V_6$ show low-voltage or slightly inverted T waves and a small or absent Q wave. ST segment depression reflects subendocardial injury. ST segment elevation should alert the clinician to a co-existing pericarditis.

New rhythm disturbances noted after a febrile illness suggest myocarditis. These can range from SVT and uniform PVCs to complete AV block. Prolongation of the PR and QT intervals are nonspecific findings.

## Electrolyte Disturbances and Hypothermia

A number of electrolyte disturbances produce ECG changes. The ECG effects of disturbances in serum potassium, calcium, and magnesium are summarized in Table 9.

Hypothermia produces a J wave at core body temperatures lower than 25°C. The J wave is an additional wave that is found between the QRS complex and the early ST segment. Other changes include prolongation of the PR and QT intervals. Either ST segment elevation or depression may be seen.

## Summary

Proficiency in interpreting pediatric ECGs is important. Equally important is the recognition that an ECG should be performed in a given patient. An approach that combines basic knowledge of pediatric ECGs with symptom-based clinical correlation avoids the common pitfalls associated with pediatric ECG interpretation and leads to accurate diagnosis and timely treatment.

Successful interpretation begins with a systematic approach to the ECG. This includes analysis of rate, rhythm, axis, and forces. The QTc must always be calculated. A key component of interpretation is an understanding of the many normals that are age-related. A reference book, which contains tables of normal age-related values, is indispensable.

In addition to age-related differences in normal values, there are routinely encountered entities in adults that are uncommon in children and whose diagnostic criteria differ (e.g., MI). Children with congenital heart disease or newly repaired congenital heart disease represent a unique group of patients. An ECG in these patients may identify post-operative rhythm disturbances.

A number of illnesses, infections, and ingestions can also affect the child's heart—producing ECG changes. A familiarity with the ECG findings associated with these conditions will facilitate diagnosis and treatment.

Overall, ECGs are infrequently needed in the emergency care of children. When indicated, the accurate and timely diagnosis of ECG abnormalities may be critical for an ill child. The ultimate goal of this review is to aid the clinician in a rapid review of basic ECG skills  pediatric and then highlight the unique aspects of pediatric ECGs through a number of clinical situations. When in doubt, a pediatric cardiologist should always be consulted and may even be a valuable asset in a pediatric ECG quality improvement process.

## References


1. Horton LA, Mosee S, Brenner J. Use of the electrocardiogram in a pediatric emergency department. *Arch Pediatr Adolesc Med* 1994;148:184-188.
2. Park MK, Guntheroth WG. *How To Read Pediatric ECGs*. Chicago: YearBook; 1987.
3. Berman W. *Handbook of Pediatric ECG Interpretation.* St Louis; Mosby-Year Book:1991.
4. Robinson B, Anisman P, Eshaghpour E. A primer on pediatric ECGs. *Contemporary Pediatrics* 1994;11:69-96.
5. Benson DW. The normal electrocardiogram. In: Emmanouilides GC, Riemenschneider TA, Allen HD, Gutgesell HP, eds. *Moss and*

*Adams, Heart Disease Infants, Children and Adolescents.* Baltimore: Williams and Wilkins; 1995:152-165.
6. Moss AJ, Robinson JL. Long QT syndrome. Heart Disease and *Stroke* 1992;1:309-314.
7. Barone MA. *The Harriet Lane Handbook.* 14th ed. St Louis: Mosby;1996.
8. Selbst SM, Ruddy R, Clark BJ. Chest pain in children: Folow-up of patients previously reported. *Clin Pediatr* 1990;29:374.
9. Zavaras-Angelidous KA, Weinhouse E, Nelson DB. Review of 180 episodes of chest pain in 134 children. *Pediatr Emerg Care* 1992;8:189.
10. Selbst SM, Ruddy RM, Clark BJ et al. Pediatric chest pain: A prospective study. *Pediatrics* 1988;82:319-323.
11. Fyfe DA, Moodie DS. Chest pain in pediatric patients presenting to a cardiac clinic. *Clin Pediatr* 1984;23:321.
12. Kato H, Ichinose E, Kawasaki T. Myocardial infarction in Kawasaki disease: Clinical analysis of 195 cases. *J Pediatr* 1986;108:923-927.
13. Towbin JA, Bricker JT, Garson A. electrocardiographic criteria for diagnosis of acute myocardial infarction in childhood. *Amer J Cardiol* 1992;69:1545-1548.
14. Garson AG. Electrocardiography. In: Garson AG, Bricker JT, Fisher DJ, Neish SR, eds. *The Science and Practice of Pediatric Cardiology* 2nd ed. Baltimore: Williams and Wilkins; 1998:735-788.
15. Walsh EP, Saul JP. Cardiac arrhythmias. In: Garson AG, Bricker JT, Fisher DJ, Neish SR, eds. *The Science and Practice of Pediatric Cardiology*, 2nd ed. Baltimore: Williams and Wilkins; 1998:377-433.
16. O'Connor BK, Dick M. What every pediatrician should know about supraventricular tachycardia. *Pediatr Ann* 1991;20:370-376.
17. Perry JC. The diagnosis and emergency management of pediatric tachyarrhythmias. *Ped Emerg Med Rep* 1996;1:65-74.
18. Ko, JK, Deal BJ, Straburger JF et al. Supraventricular tachycardia mechanisms and their age distribution in pediatric patients. *Am J Cardiol* 1992;69:1028-1031.
19. Perry JC. Supraventricular tachycardia. In: Garson AG, Bricker JT, Fisher DJ, Neish SR, eds. *The Science and Practice of Pediatric Cardiology*, 2nd ed. Baltimore: Williams and Wilkins; 1998: 2059 - 2101.
20. Xie B, Thakur RK, Shah CP et al. Clinical differentiation of narrow QRS complex tachycardias. *Emerg Med Clin N Amer* 1998;16: 295-330.
21. Case CL. Diagnosis and treatment of pediatric arrhythmias. *Pediatr Clin N Amer* 1999;46:347-354.
22. Feit LR. Syncope in the pediatric patients: Diagnosis, pathophysiology, and treatment. *Adv in Pediatrics* 1996;43:469-494.
23. Scott WA. Evaluating the child with syncope. *Pediatr Ann* 1991;20:350-359.
24. Gordon TA, Moodie DS, Passalacqua M, et al. A retrospective analysis of the cost-effective workup of syncope in children. *Cleve Clin J Med* 1987;54:391-394.
25. Kanter RJ. Syncope and sudden death. In: Garson AG, Bricker JT, Fisher DJ, Neish SR, eds. *The Science and Practice of Pediatric Cardiology*, 2nd ed. Baltimore: Williams and Wilkins; 1998:2169-2199.
26. Ruckman RN. Cardiac causes of syncope. *Pediatr Rev* 1987;9: 101-108.
27. Bazett HC. An analysis of the time relations of the electrocardiogram. *Heart* 1920;7:353-367.
28. Berger S, Dhala A, Friedberg DZ. Sudden cardiac death in infants, children and adolescents. *Pediatr Clin N Amer* 1999;46;221-234.
29. Ross BA, Trippel DL. Atrioventricular block. In: Garson AG, Bricker JT, Fisher DJ, Neish SR, eds. *The Science and Practice of Pediatric Cardiology*, 2nd ed. Baltimore: Williams and Wilkins; 1998: 2047-2057.
30. Dickinson DF, Scott O. Ambulatory electrocardiographic monitoring in 100 healthy teenage boys. *Br Heart J* 1984;51:179-183.
31. Nagashima M, Matsushima M, Ogawa A et al. Cardiac arrhythmias in healthy children revealed by 24-hour ambulatory ECG monitoring. *Pediatr Cardiol* 1987;8:103-108.
32. Southall DP, Johnston F, Shinebourne EA et al. 24-hour electrocardiographic study of heart rate and rhythm patterns in a population of healthy children. *Br Heart J* 1981;45:281-291.
33. Mehta AV, Sanchez GR, Balian A, et al. Natural history of second-degree atrioventricular (AV) block in children : Clinical and electrophysiologic findings. *Pediatr Cardiol* 1982:351-352. (abstract)
34. Jacobsen JR, Garson A. Irregular heartbeat. In: Garson AG, Bricker JT, Fisher DJ, Neish SR, eds. *The Science and Practice of Pediatric Cardiology.* 2nd ed. Baltimore: Williams and Wilkins; 1998:2225.
35. Dowd MD, Krug S. Pediatric blunt cardiac injury; Epidemiology, clinical features, and diagnosis. *J Trauma; Injury, Infection and Critical Care* 1996;40:61-67.
36. Clancy C. electrocardiographic evaluation of the poisoned or overdosed patient. In: Goldfrank LR, Flomenbaum NE, Lewin NA et al. *Goldfrank's Toxicologic Emergencies.* 6th ed. Stamford CT;Appleton and Lange;1998:105-128.
37. Miller MD. Arrhythmias associated with drug toxicity. *Emerg Med Clinics N Amer* 1998;16:405-417.
38. *Ellenhorn's Medical Toxicology: Diagnosis and Treatment of Human Poisoning.* 2nd ed. Baltimore: Williams and Wilkins; 1997.
39. Hardman JG, Gilman AG, Limbird LE,et al, eds. *Goodman's and Gilman's The Pharmacological Basis of Therapeutics.* New York: McGraw-Hill; 1996.
40. Haddad ML, Winchester JF, eds. *Clinical Management of Poisoning and Drug Overdose.* Philadelphia: Saunders; 1983.
41. Vetter VL. What every pediatrician needs to know about arrhythmias in children who have had cardiac surgery. *Pediatr Ann* 1991;20: 378-384.
42. Vetter VL, Horowitz LN. electrophysiologic residua and sequelas of surgery for congenital heart defects. *Amer J Cardiol* 1982;50: 588-604.
43. Van Hare GF, Porter CJ. Atrial flutter and atrial fibrillation. In: Garson AG, Bricker JT, Fisher DJ, Neish SR, eds. *The Science and*

*Practice of Pediatric Cardiology*. 2nd ed. Baltimore: Williams and Wilkins;1998: 2103-2119.
44. Gelband H, Waldo AL, Kaiser GA et al. Etiology of right bundle branch block in patients undergoing total correction of tetralogy of Fallot. *Circulation* 1971;44:1022-1033.
45. Quattlebaum TG, Varghese PJ, Neill CA, et al. Sudden death among post-operative patients with tetralogy of Fallot. *Circulation* 1976:54:289-293.
46. Gillette PC, Yeoman MA, Mullins et al. Sudden death after repair of tetralogy of Fallot. *Circulation* 1977;56:566-571.

## Physician's CME Questions

1. The most common pathologic cause of ST segment elevation in children is which of the following?
   a. Hyperkalemia
   b. Pericarditis
   c. Myocardial contusion
   d. Tetralogy of Fallot
   e. Romano Ward Syndrome

2. A short PR interval represents a risk factor for which of the following conditions?
   a. SVT
   b. Ventricular tachycardia
   c. Torsade de pointes
   d. First degree heart block
   e. Wenkebach

3. ECG findings consistent with hypokalemia include:
   a. AV block.
   b. tall, peaked T waves.
   c. prolonged QRS.
   d. low-voltage P waves.
   e. sinus bradycardia.

4. The most common ECG finding in children with myocardial contusion is:
   a. sinus tachycardia.
   b. ventricular tachycardia.
   c. wide Q waves.
   d. complete heart block.
   e. inverted T waves.

5. The majority of pediatric patients with syncope have:
   a. a prolonged QTc.
   b. heart block.
   c. a normal ECG.
   d. sick sinus syndrome.
   e. ventricular rhythm disturbance.

6. Which patient with repair for congenital heart disease is most likely to develop atrial flutter?
   a. A 10-year-old patient with repaired VSD
   b. An infant with repaired coarctation of the aorta
   c. A teenager with repaired transposition of the great vessels
   d. An infant with ligation of a patent ductus arteriosus
   e. A toddler with repaired tetralogy of Fallot

7. Drug toxicity causes rhythm disturbances by which mechanism?
   a. Direct effects on myocardial membranes
   b. Sympathomimetic effects
   c. Anticholinergic effects
   d. CNS regulation of peripheral autonomic system
   e. All of the above

8. Which of the following statements is true regarding wide complex tachycardia in children?
   a. It is almost always due to bundle branch block.
   b. It is considered VT until proven otherwise.
   c. Tachycardia results from fever.
   d. It is common in infants.
   e. it is not harmful if the P wave axis is normal.



**Take Your CME Tests Online With *TestWeb***

American Health Consultants' newsletter subscribers can now take their CME tests online. As an alternative to twice-yearly scantron exams, current subscribers can answer their CME questions on a monthly basis and submit answers electronically.

For more information visit the **TestWeb site** at:
**http://www.ahcpub.com/testweb.htmL**
**or call: 1-800-688-2421.**

**In Future Issues:** Pain Management