Common Cardiac Issues in Pediatrics

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Deepak M. Kamat, MD, PhD, FAAP

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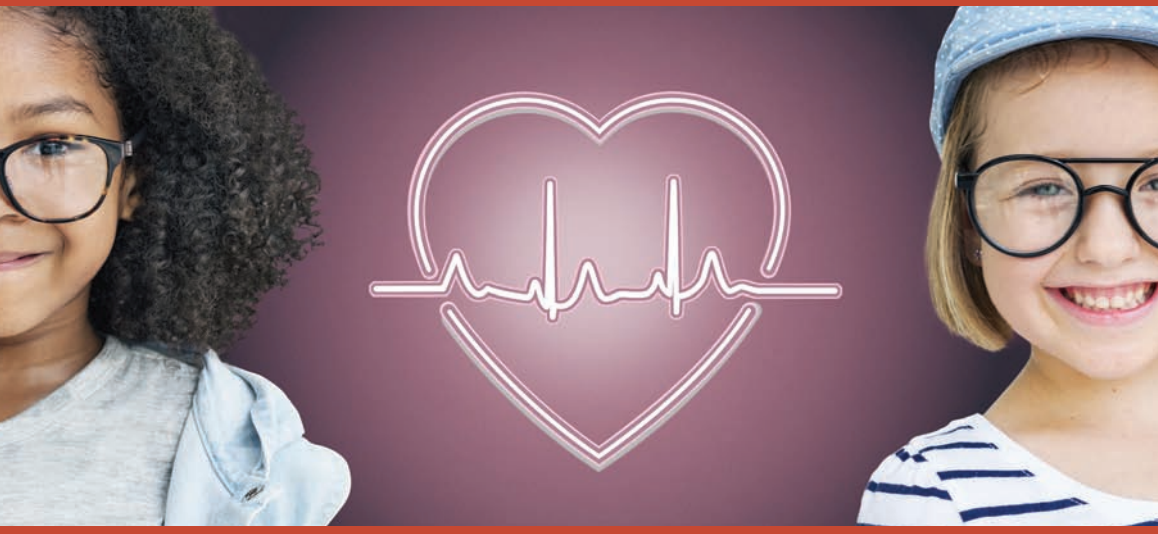

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Introduction

Though heart defects in children had been studied for many years, the field of pediatric cardiology was essentially born in the 1930s, when Robert Gross performed the first surgical ligation of a patent ductus arteriosus. Instead of cardiac defects being a pathologic entity seen at autopsy, they became treatable. Helen Taussig began training pediatricians in the new field of pediatric cardiology in the late 1940s, and pediatric cardiology became the first sub-board of pediatrics in 1961. The Section on Cardiology of the American Academy of Pediatrics (AAP) was formed in 1955.

The field of pediatric cardiology has seen incredible advances in its short period of existence. Cardiopulmonary bypass, used to repair intracardiac defects, was first successfully used in children in 1954 and 1955. The first echocardiograms, initially 1-dimensional, were reported in 1953, but 2-dimensional images of the moving heart were not available until the early 1970s. The arterial switch procedure, now the standard of care for most patients with transposition of the great arteries, was first performed in 1975—only 43 years ago.

As these advances in pediatric cardiac care have occurred, the scope of pediatric primary care exposure to patients with complex cardiac anatomy has increased. This text was created specifically for the pediatric care professional (pediatrician, family physician, nurse practitioner, physician assistant, and all trainees and students) who could see a patient in the inpatient or outpatient setting with heart disease. The content starts with the fundamentals of history and physical examination, followed by the basics of cardiac testing, including electrocardiography, echocardiography, and chest radiography. Specific chapters focus on common symptoms, such as chest pain, syncope, and murmurs. Extensive guidance is provided for each type of congenital lesion a patient may present with in your office—each easily looked up and reviewed quickly just prior to seeing a patient. Acquired diseases such as Kawasaki disease are discussed, as well as infectious diseases, such as rheumatic fever, Lyme disease, endocarditis, pericarditis, and myocarditis. Common preventive cardiology issues are presented, including dyslipidemia, hypertension, sports clearance, screening prior to attention-deficit/hyperactivity disorder medication administration, and adolescent autonomic dysfunction. Finally, a robust discussion of cardiac medications is provided, focused on the pediatric care professional.

In assembling this book, we have capitalized on the rich and unique resources of the American Academy of Pediatrics (AAP), as well as contributions from experts from around the globe. We thank all of our authors for their time and expertise. The contents of this book were reviewed by experts from relevant AAP sections, committees, and councils, including non–cardiology specialty perspectives. The editors and contributing authors are grateful for these reviewers' expertise and generous feedback. Special thanks are due to the cardiologists

and cardiac surgeons of the Publications and Communications Workgroup of the Section on Cardiology and Cardiac Surgery, who personally reviewed every chapter. We want to give our sincere thanks to Chris Wiberg at the AAP, who was the senior editor for this book. We appreciate his commitment to the project, his editorial prowess, his constant encouragement, and his collaborative spirit. Finally, we want to thank our families for their love, understanding, and support while we took on this amazing endeavor.

This book is focused on the pediatric care professional for a specific reason: You are essential partners with families and cardiology teams in the care of these children. We hope this book will help answer questions that arise on a daily basis and serve as an essential resource in your office, with the ultimate goal of improving care for all children with heart disease.

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PART 1

Evaluation of the Patient

CHAPTER 1

Clinical History and Physical Examination

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Introduction

The history and physical examination are the first and most important steps in the evaluation of any child for whom there is a concern for heart disease. A thorough history is essential when evaluating children who present with common cardiac concerns, such as syncope and chest pain. Often, the history will contain important clues that an astute clinician will use to guide medical decision-making and management. A thorough physical examination can also play a role in helping to establish the correct diagnosis without the need for expensive ancillary tests. This chapter will address the basics of the cardiac history and physical examination in children.

History

A thorough history—including a detailed personal history of the child, history of the mother during pregnancy, the child's birth history, past medical history, review of systems, family history, and social history—should be obtained. The maternal history should include assessment of complications during pregnancy or delivery and assessment of any teratogenic exposures during pregnancy. In all patients, the timing of appearance of signs and symptoms should be clearly delineated, because

this can often help establish a differential diagnosis. Specific details of the personal cardiac history can vary, depending on the age of the child.

Infants

The largest energy expenditure in infants is feeding, and infants with congenital heart disease (CHD) or heart failure commonly present with symptoms during feeding. Infants may ingest less breast milk or formula per feeding than usual, or their symptoms may arise only during feeding, including sweatiness, tachypnea, fussiness, and irritability. In infants with heart failure, the volume of feedings has been shown to be the most sensitive variable historically associated with clinical heart failure. Poor growth may be present, particularly in patients with large left-to-right shunts or heart failure.

Young Children

Symptoms in young children can be subtle, because children can compensate very well despite having clinically significant CHD or poor heart function. Patients and their parents should be asked about any chest pain, palpitations, or fainting spells. A chronic cough may be present if pulmonary congestion or bronchial compression is present. Abdominal pain, nausea, or anorexia may be present in children with heart failure because of poor cardiac output to peripheral tissues. Because parents often find it difficult to know what "normal" exercise ability is for a young child, it can be helpful to ask what his or her ability level is in comparison to other children or older siblings.

Adolescents

Although older children and adolescents may present with symptoms similar to those of younger children, they may also complain of symptoms similar to those of adults. Patients should be asked about any chest pain, palpitations, dizziness or fainting spells, and changes in exercise tolerance. Patients should also be asked about any of these symptoms occurring with exertion. Additionally, patients should be asked about snoring and whether they have any symptoms of orthopnea.

Past Medical History

Obtaining a thorough past medical history is important to ensure that no other medical conditions are present that may place the patient at risk for cardiac disease. Particular attention should be given to any history of genetic syndromes or chromosomal abnormalities. Patients with connective tissue disorders may report a history of pneumothorax, easy joint dislocation, or lens dislocation. Patients should be asked about a history of Kawasaki disease and rheumatic heart fever, particularly if they are from a geographic area or have an ethnic background endemic to either condition. A history of previous fainting episodes

and seizures and a family history should be obtained when there is concern that the patient's syncopal episode may be caused by a genetic mutation, such as long QT syndrome.

Family History

Patients and their parents should be asked about any family history of CHD, cardiomyopathy, hyperlipidemia, hypertension, or early coronary artery disease (manifesting before the age of 50 years). Any history of sudden, unexplained death should be obtained, including unexplained car accidents, drownings, seizures, or sudden infant death syndrome. It is most important to obtain this history for first- and second-degree family members.

History Compilation Techniques

In older children, it is important to engage the patient directly in the history compilation process, since he or she is the one experiencing the symptoms. In a child who has undergone a prior surgical repair of CHD, history compilation is an opportunity to assess the child's knowledge about his or her own health condition.

Precision is also important in the history compilation process. Terms such as "heart attack" and "passing out" need to be clarified to make sure that parents and practitioners are using the same definitions.

Physical Examination

Assessment of Vital Signs

Heart rate, respiratory rate, oxygen saturation, and blood pressure should be assessed at each visit. An abnormal heart rate can alert the clinician to a diagnosis of congestive heart failure or even a noncardiac disorder, such as thyroid dysfunction or Lyme disease with heart block. In addition to physical palpation of the pulse in the upper and lower extremities, blood pressure measurements should be obtained in the upper and lower extremities during the first visit to assess the patient for coarctation of the aorta. Oxygen saturation should be recorded in all patients. In patients who underwent a previous cardiac surgery, oxygen saturation should be followed serially, and results should be compared to those obtained at previous clinical encounters.

It is important to remember that blood pressure should be assessed with the patient calm and sitting down. The use of an inappropriately sized blood pressure cuff remains the most common cause of inaccurate hypertension diagnoses in children. The length of the bladder of the cuff should be 80% of the circumference of the limb being measured, and the width of the cuff should cover two-thirds of the length of the extremity.

Inspection

A considerable degree of information can be obtained with observation. The physician should determine if the child is in any distress, is over- or undernourished, or has any potential genetic syndrome. Cyanosis can be indicative of CHD but can be difficult to detect in patients with anemia or dark skin pigmentation. Special attention should be given to the tongue, nail beds, and conjunctiva. Acrocyanosis may be seen in healthy infants, where the hands and feet have a blue hue but the remainder of the skin is normal. Digital clubbing can be seen in the fingernails and toenails of a patient with prolonged desaturation.

The respiratory rate and presence of any retractions should be noted. The chest should be inspected to look for any pectus abnormalities (excavatum or carinatum) or asymmetry of the chest.

Palpation

Palpation of the peripheral pulse should be performed, including the pulse in both upper and lower extremities. A weak leg pulse relative to that of the arm suggests coarctation of the aorta.

A bounding pulse (widened pulse pressure) may be found in a patient with patent ductus arteriosus, aortic regurgitation, or arteriovenous fistula.

Palpation of the chest should be performed to identify the apical impulse and to look for evidence of a hyperactive precordium and thrills. After the age of 10 years, the apical impulse is typically located at the midclavicular line in the fifth intercostal space. An apical impulse that is displaced laterally or downward suggests cardiomegaly. A hyperactive precordium is associated with large left-to-right shunts. Thrills are palpable vibratory sensations associated with harsh murmurs. Thrills may be felt on the chest wall or in the suprasternal notch in the setting of clinically significant aortic stenosis.

Palpation of the abdomen should be performed to assess the size and texture or consistency of the liver and spleen. With increased venous pressure or increasing shunt volume, the liver will become enlarged. Percussion of the liver may be performed in older children and adolescents.

Auscultation

To diagnose a cardiac murmur, it is necessary to understand and describe the heart sound in the context of the cardiac cycle. The cardiac cycle can be broken down into systole and diastole (Figure 1-1). Systole is the period of ventricular contraction during which blood is ejected from the right ventricles (RVs) and left ventricles (LVs) into the main pulmonary artery and aorta, respectively. Diastole is the period of ventricular relaxation during which tricuspid and mitral valves are open and the ventricles fill with blood from the atria. Most of the blood enters the ventricles passively, but a small portion is ejected during atrial contraction that occurs in late diastole. At the onset of systole, the ventricles

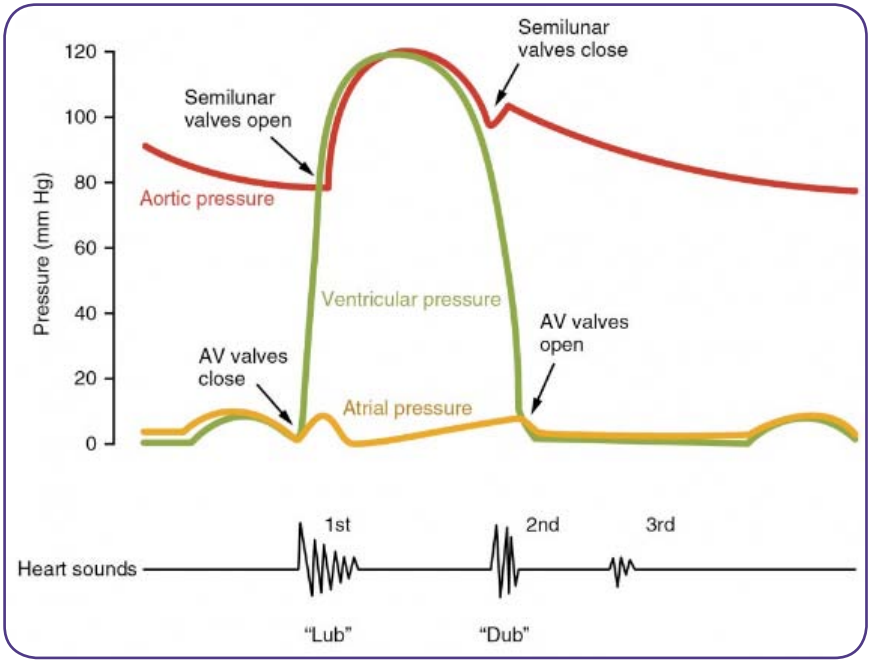

FIGURE 1-1. The cardiac cycle. AV = atrioventricular. From https://courses.lumenlearning.com/ap2/chapter/cardiac-cycle/.

contract, causing a rapid increase in ventricular pressure that soon exceeds the atrial pressure. Once the pressure within the ventricles exceeds that of the atria, the tricuspid and mitral valves are forced closed. Closure of these valves produces the first heart sound (S_1). The ventricular pressures continue to rise with contraction during a phase called *isovolumetric contraction* until they exceed that of the pulmonary artery and aorta. This results in opening of the pulmonic and aortic valves and ejection of blood from the ventricles (ejection phase). As the ejection of blood is completed, the ventricular pressure begins to decrease. Once ventricular pressure falls below that of the main pulmonary artery and aorta, the pulmonic and aortic valves are forced closed, producing the second heart sound (S_2). The ventricular pressure decreases rapidly as a result of the ventricular relaxation during a phase called *isovolumetric relaxation*. Once the ventricular pressure falls below that of the atria, the tricuspid and mitral valves open, allowing passive filling of blood into the ventricles, and the cycle repeats.

Being able to accurately identify the first and the second heart sounds (S_1 and S_2, respectively) will allow for categorization of murmurs and extra heart sounds on the basis of the phase of the cardiac cycle within which they occur (systole or diastole). Both S_1 and S_2 are composed of 2 components, each of which can be heard by the trained ear. The mitral valve closes slightly before the tricuspid

valve because of normal nonsynchronous contraction of the ventricles, with the LV beginning and ending the contraction prior to the RV. The splitting of S_1 is generally not appreciated because of the minimal time difference between when the mitral and tricuspid valves close. S_2 is composed of P_2 and A_2, which represent closure of the pulmonic and aortic valves, respectively. Normally, the aortic valve closes slightly before the pulmonic valve, but because it is in close proximity, it can be difficult to differentiate the 2 sounds. Physiological splitting of S_2 occurs throughout the respiratory cycle. With inspiration, there is increased blood return to the right atrium and therefore the RV. This increased blood volume results in an increase in the ejection time from the RV. Therefore, the pulmonic valve remains open longer and accentuates the time difference between closure of the aortic and pulmonic valves, which is heard as splitting of the S_2. During expiration, A_2 and P_2 occur almost simultaneously, as mentioned earlier, and are heard as a single S_2.

In addition to S_1 and S_2, on occasion 2 other sounds, called S_3 and S_4, can be heard (audio available at www.youtube.com/watch?v=DxMnm5C5PW8&index=1&list=PLKCIeugVenPTLW-nspw_wUkXxO2IIiesu)( ). Both of these sounds occur during diastole. S_3 occurs during early diastole as a result of rapid filling of the ventricle after opening of the tricuspid and mitral valves. This is generally a normal sound in children but can indicate volume overload. S_4 occurs during late diastole and coincides with atrial contraction. When the atria contract and eject blood into a stiffened ventricle, S_4 is produced.

Physiology of Cardiac Murmurs

There are 4 main locations that are used for auscultation, which correspond to the 4 cardiac valves (Figure 1-2). The aortic area is located at the second to third intercostal space to the right of the sternum (right upper sternal border). The pulmonic area is located at the second to third intercostal space to the left of the sternum (left upper sternal border). The tricuspid area is at the left lower sternal border, and the mitral area is located at the apex of the heart. As described previously, S_1 is the closure of the tricuspid and mitral valves, and it can be best heard at the apex. S_2 and splitting of the S_2 are best heard at the left upper sternal border in the pulmonic area. S_3 and S_4 are best heard at the apex or the left lower sternal border, depending on whether they are originating from the LV or the RV.

When a murmur is heard, it is important to be able to localize the sound to a specific area of the heart and to characterize the murmur. The key components of characterizing a murmur are timing, intensity, pitch, quality, location, radiation, positional variation, and presence of extra heart sounds. The first step is to identify S_1 and S_2. Once these are identified, the timing of the murmur in the cardiac cycle can be defined as ejection systolic, holosystolic, diastolic, or continuous. Systolic murmurs occur after S_1, and diastolic murmurs occur

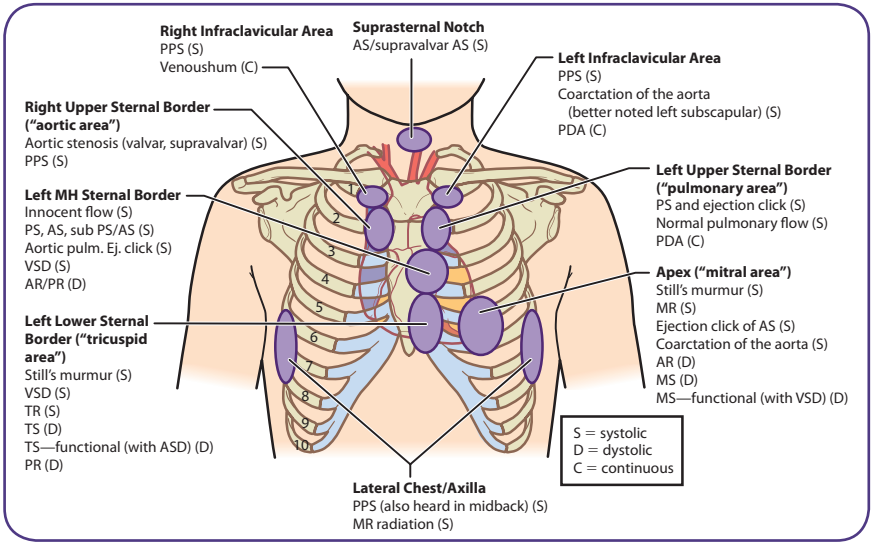

FIGURE 1-2. Typical location or listening areas for murmurs. AR = aortic regurgitation, AS = aortic stenosis, ASD = atrial septal defect, MR = mitral regurgitation, MS = mitral stenosis, PDA = patent ductus arteriosus, PPS = peripheral pulmonic stenosis, PR = pulmonary regurgitation, PS = pulmonary stenosis, TR = tricuspid regurgitation, TS = tricuspid stenosis, VSD = ventricular septal defect. From Koenig P, Hijazi Z, Zimmerman F. *Essential Pediatric Cardiology*. New York, NY: McGraw Hill; 2004.

after S_2. Holosystolic murmurs occur at the onset of systole and result in the obliteration of S_1. The presence of a holosystolic murmur indicates that blood is escaping from the ventricle as soon as ventricular contraction begins. This can be caused by a ventricular septal defect or tricuspid or mitral valve regurgitation. An ejection systolic murmur, however, begins after S_1 (during the ejection phase) and is usually caused by ventricular outflow tract obstruction. With an ejection systolic murmur, S_1 can be defined, whereas with a holosystolic murmur, S_1 is incorporated into the murmur and cannot be defined. Continuous murmurs are present throughout systole and diastole. (See the list of Audio Recordings of heart murmurs at the end of this chapter.)(👂)

Systolic and diastolic murmurs each have their own intensity classification. Systolic murmurs are graded I to VI, whereas diastolic murmurs are graded I to IV. The initial classification of systolic heart murmurs was the Levine system. Owing to the subjective nature of this system for murmurs graded III or VI or less, a variation may be used in which the patient's own heart sounds are used as an internal reference for grading.[1] By using this system, grade I murmurs are less intense than the normal heart sounds (S_1 and S_2). Grade II murmurs are equal in intensity to S_1 and S_2, and grade III murmurs are more intense than S_1 and S_2. Grade IV murmurs include the presence of a thrill. Grade V murmurs can be heard with only the edge of the stethoscope on the patient's chest, and grade VI murmurs can be heard with the stethoscope removed from the chest

wall. Grading diastolic murmurs is subjective and listener dependent. Grade I murmurs are barely audible. Grade II murmurs are faint but audible. Grade III murmurs are easily heard, and grade IV murmurs are loud.

The next step is to describe the pitch of the murmur, which is related to the frequency of the sound and reflects the velocity of the turbulent blood flow; higher pitch represents higher velocity of flow. The pitch of the murmur, therefore, provides more information about the potential cause and severity of the lesion. High-frequency murmurs are produced when there are large (or high) pressure gradients between 2 chambers, whereas low-frequency murmurs are produced when there is a smaller (or low) pressure gradient.

Knowing where on the chest wall to auscultate each of the 4 valves, as described previously, is important to localize the examination. Where on the chest wall the murmur is heard the loudest should be specified. In addition, where else the murmur can be heard or where it radiates to needs to be determined. Radiation of the murmur follows the course in which the blood flows. For example, murmurs that can be heard in the back and axilla are following the course of the pulmonary blood flow. In general, the sound of innocent murmurs will not radiate, so the presence of this finding should prompt the question of a need for further evaluation.

Certain types of murmurs can be altered by positional changes, respiration, or maneuvers. This can help to differentiate similar murmurs from each other. In general, innocent murmurs will vary in intensity, depending on the patient's position during auscultation. Innocent murmurs are usually loudest when lying supine and diminish in intensity or disappear when sitting upright or standing. One exception to this is a venous hum, which resolves or diminishes when lying supine. A lack of change in murmur intensity with position change should raise suspicion for a pathologic murmur. Squatting is another positional change that can be helpful when evaluating a murmur. In patients with hypertrophic cardiomyopathy, the murmur intensity decreases with squatting and increases with standing.

The Stethoscope

The bell of the stethoscope is best suited to hear low-frequency events, while the diaphragm of the stethoscope selectively picks up higher-frequency sounds. Using only the diaphragm of the stethoscope may result in missing some low-pitched sounds, such as a diastolic rumble, pulmonary regurgitation, or Still murmur (also commonly referred to as Still's murmur). Innocent or functional murmurs are common in children. They occur in the absence of anatomic anomalies. Examples include vibratory murmurs, pulmonary flow murmurs, venous hums, and carotid bruits.

Key Points

- History and physical examination remain important tools in the evaluation of children with murmurs and other common cardiac complaints.
- A thorough history should contain a detailed personal history of the child, maternal history during pregnancy, the child's birth history, past medical history, review of systems, family history, and social history.
- In adolescent patients, it is important to obtain the history directly from them and assess the role psychosocial stress may play in their symptoms.
- Heart rate, respiratory rate, oxygen saturation, and blood pressure should be assessed at each visit.
- In addition to physical palpation of the pulse in the upper and lower extremities, blood pressure measurements should be obtained in the upper and lower extremities during the first visit to assess the patient for coarctation of the aorta.

Audio Recordings (👂)

- Normal Third Heart Sound (S3) (Willam Buck Kyle, MD). www.youtube.com/watch?v=DxMnm5C5PW8&index=1&list=PLKCIeugVenPTLW-nspw_wUkXxO2IIiesu
- Bicuspid Aortic Valve and Systolic Click—normal speed (Willam Buck Kyle, MD). www.youtube.com/watch?v=zsAj2xGNGs4&index=3&list=PLKCIeugVenPTLW-nspw_wUkXxO2IIiesu
- Bicuspid Aortic Valve and Mild Stenosis—normal speed (Willam Buck Kyle, MD). www.youtube.com/watch?v=opyMtEHhyLE&index=4&list=PLKCIeugVenPTLW-nspw_wUkXxO2IIiesu
- Bicuspid Aortic Valve and Mild Stenosis—slow (Willam Buck Kyle, MD). www.youtube.com/watch?v=dsoG-OX_Wv8&index=5&list=PLKCIeugVenPTLW-nspw_wUkXxO2IIiesu
- Small Muscular Ventricular Septal Defect—normal speed (Willam Buck Kyle, MD). www.youtube.com/watch?v=VLD0ao6lQ3M&index=8&list=PLKCIeugVenPTLW-nspw_wUkXxO2IIiesu
- Small Muscular Ventricular Septal Defect—slow (Willam Buck Kyle, MD). www.youtube.com/watch?v=BJ-1c4JFY9Y&index=9&list=PLKCIeugVenPTLW-nspw_wUkXxO2IIiesu
- Restrictive Membranous Ventricular Septal Defect—normal speed (Willam Buck Kyle, MD). www.youtube.com/watch?v=dnzZDGKMW5I&index=10&list=PLKCIeugVenPTLW-nspw_wUkXxO2IIiesu
- Restrictive Membranous Ventricular Septal Defect—slow (Willam Buck Kyle, MD). www.youtube.com/watch?v=bpEKVgXQPhY&index=11&list=PLKCIeugVenPTLW-nspw_wUkXxO2IIiesu

- Restrictive Muscular Ventricular Septal Defect (Willam Buck Kyle, MD). www.youtube.com/watch?v=jXvE1otbxac&index=12&list=PLKCIeugVenPTLW-nspw_wUkXxO2IIiesu
- Restrictive High Muscular Ventricular Septal Defect (Willam Buck Kyle, MD). www.youtube.com/watch?v=l-pLcHQVjQA&index=13&list=PLKCIeugVenPTLW-nspw_wUkXxO2IIiesu
- Restrictive Mid Muscular Ventricular Septal Defect—slow (Willam Buck Kyle, MD). www.youtube.com/watch?v=7YeB7wbVFNc&index=14&list=PLKCIeugVenPTLW-nspw_wUkXxO2IIiesu
- Restrictive Apical Muscular Ventricular Septal Defect—slow (Willam Buck Kyle, MD). www.youtube.com/watch?v=nKlCgY_XtjM&index=15&list=PLKCIeugVenPTLW-nspw_wUkXxO2IIiesu
- Pulmonary Stenosis—mild (Willam Buck Kyle, MD). www.youtube.com/watch?v=8uk8iKxu5HY&index=17&list=PLKCIeugVenPTLW-nspw_wUkXxO2IIiesu
- Aortic Regurgitation and Mild Stenosis (Willam Buck Kyle, MD). www.youtube.com/watch?v=54BhXSnaXk4&index=19&list=PLKCIeugVenPTLW-nspw_wUkXxO2IIiesu

Reference

1) Keren R, Tereschuk M, Luan X. Evaluation of a novel method for grading heart murmur intensity. *Arch Pediatr Adolesc Med*. 2005;159(4):329–334

CHAPTER 2

Electrocardiography

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Introduction

Electrocardiography (ECG) provides information regarding the conduction of electricity throughout the heart. Standard ECG in children has either 12 or 15 leads (see Figure 2-1). Each lead provides information from a particular vantage point on the chest, like an "electrical camera." ECG tends to be useful for determining rhythm but can also provide insights into structural or systemic disease and cardiomyopathies. The limb leads (I, II, III, aVR, aVF, aVL) provide information about how electricity moves in the superior-inferior and left-right directions. These leads are useful for helping to determine rhythm, atrial enlargement, and overall direction of net electrical force (axis). The precordial leads provide information about the movement of electrical activity in an anterior-posterior and left-right direction (Figure 2-1). This can be helpful for determining ventricular hypertrophy. Both precordial and limb leads can provide information about conduction delays and blocks, ischemia, myocardial disease, and structural abnormalities of the heart.

Standard ECG (Figure 2-2) captures 10 seconds of data. While the limb and precordial leads can be configured to display in numerous ways, typically, the left portion of the ECG tracing contains the limb leads, and the right portion contains the precordial leads. Usually, 1 or more rhythm strips (RSs) of a continuous lead tracing are recorded at the bottom, with lead II often being used because most ventricular forces are frequently directed toward this lead. The upper portion of the ECG tracing contains demographic data, computer-calculated intervals, and a computer-determined analysis of the ECG tracing.

The tracing is made on a background of 1-mm × 1-mm boxes. On the x-axis, time is typically noted, so each small box is 40 ms, and each larger box (consisting of 5 smaller boxes) is 200 ms. The paper speed is 25 mm/s (for a 10-second ECG, there will be 250 small boxes representing 40 ms per box); it can be changed but

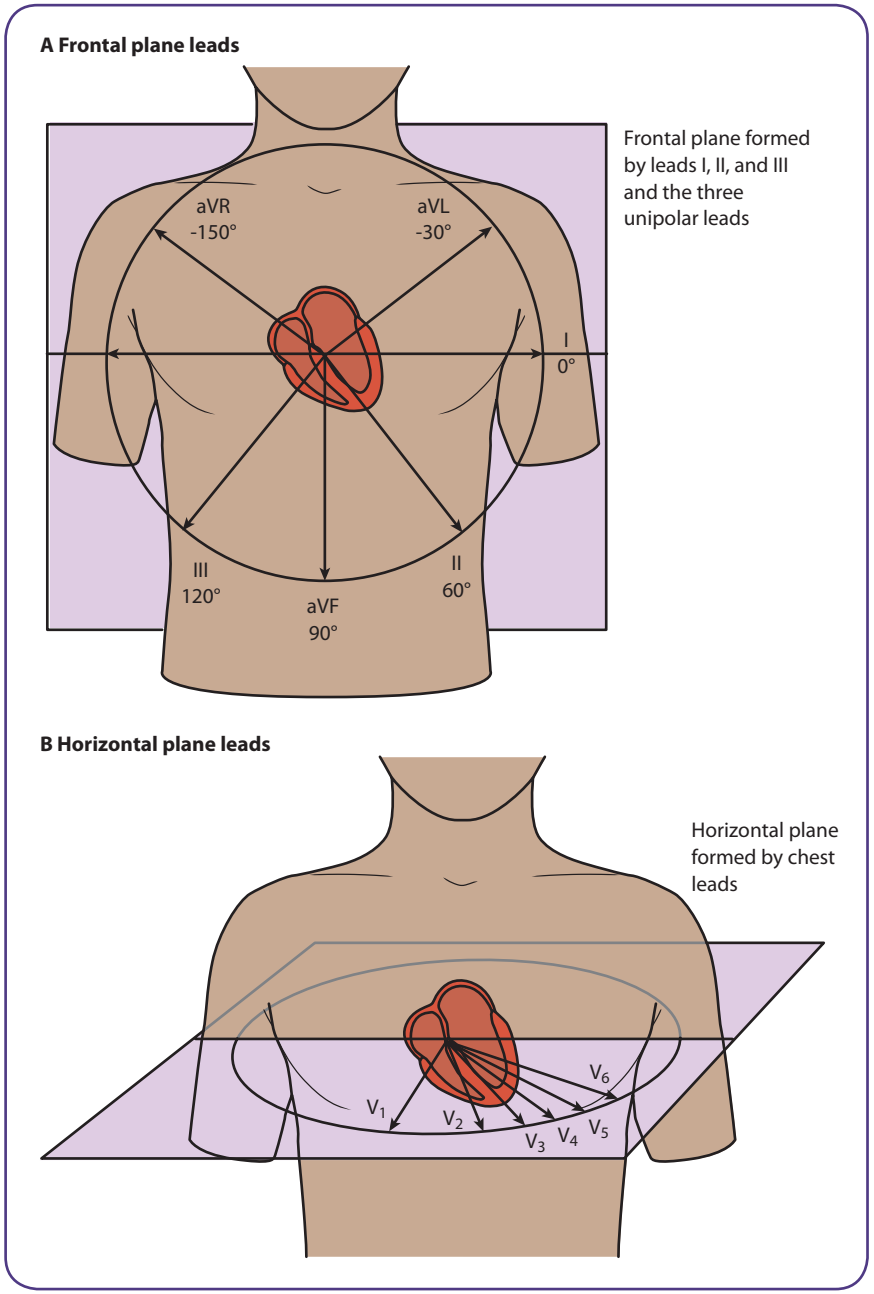

FIGURE 2-1. Electrocardiographic lead orientation and limb lead vectors. **A.** The frontal plane leads with the negative electrodes aligned to a central point. The approximate location of the heart is shown for reference. The leads are described relative to lead I. *Counterclockwise* is defined as the negative direction, and *clockwise* is the positive direction. **B.** The horizontal or precordial plane and the relative position of the chest leads. From Kusumoto FM. *Cardiovascular Pathophysiology.* Raleigh, NC: Hayes Barton Press; 1999. Reprinted with permission from Hayes Barton Press.

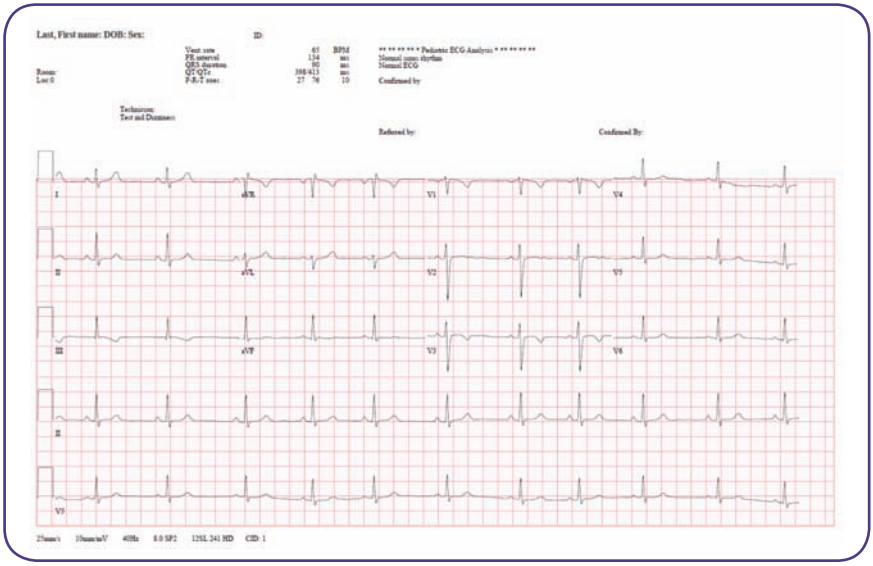

FIGURE 2-2. Standard 12-lead pediatric electrocardiogram.

typically is not. The paper speed is shown at the bottom of the ECG tracing. The y-axis, also called the *amplitude*, is simply discussed in boxes or millimeters. At the far left of the strip, the vertical standardization is conveyed by the height of the squared-off column. Standard vertical calibration will include 10 small boxes. Both the limb and precordial leads can have their vertical calibration changed separately. When the ECG is "half standard," the ECG amplitudes will display at one-half the usual height and, as such, all amplitude measures will need to be multiplied by 2 for making interpretive decisions. Conversely, if displayed as "double standard," all ECG amplitudes will be twice the usual height and should be halved for interpretation. Change in standardization is often done when the R and/or S waves are overlapping each other, making differentiation difficult.

By convention, electrical depolarization forces toward a lead will be displayed as a positive deflection above the baseline, and forces away from the lead will be displayed as a negative deflection. Each lead will show the heartbeat from a different vantage point on the chest. The limb leads can be used to describe the electrical direction in 360° or around a "clockface." Information about superior-inferior and left-right forces can be determined. Leads I, II, and III are situated 60° apart from each other, between 0° and 120°. For most children and adults, most electrical forces of the heart come from the ventricles. Typically, these forces will be most directed in the range of 0° to 120°. aVF, aVL, and aVR are augmented leads (meaning they are not directly placed on the patient but are virtually determined via vectoral calculation). These leads are at 120° separations

from each other and, in conjunction with leads I, II, and III, can help show a perspective of the heart's electrical activity from around the entire 360° clockface.

The precordial leads are situated on the chest, with V_1 (V_3R, V_4R) starting just to the right side of the sternum, overlying the right ventricle (RV), and progressively moving leftward through V_6 (V_7), overlying the left ventricle (LV). The quantitative measurements most often used in precordial lead interpretation are the R and S wave heights and ratios that can be used for determination of ventricular hypertrophy.

Interpretation Method

While there is no one correct method for ECG interpretation, essential components must be addressed each time for complete and accurate interpretation. The order in which they are addressed is less important than ensuring that each task is addressed each time. Failure to address any of the following components will result in an incomplete assessment of the ECG results.

- Rate
- Rhythm
- Axis
- Atrial enlargement
- Ventricular hypertrophy
- Conduction delays
- Interval assessment
- Segment assessment
- Corrected QT interval (QTc) calculation and assessment
- Repolarization (T wave assessment)

The interpretive method used here will be based on the understanding of the physiology of a normal sinus heartbeat as it occurs through the cardiac cycle. Understanding how the surface ECG correlates with the cardiac cycle allows ECG interpretation to be more deeply linked to underlying cardiac physiological processes.

The Normal Heartbeat

Each normal heartbeat (see Figure 2-3) should involve atrial depolarization (from the sinus node), conduction to the atrioventricular (AV) node, depolarization of the ventricles in a nearly synchronous fashion through the His-Purkinje system, atrial repolarization, and, finally, ventricular repolarization. Except for atrial repolarization (which gets lost in the QRS deflections), each of these events is represented on the surface ECG (Table 2-1). Each may appear differently, depending on which electrical vantage point or lead is viewed. Unlike adults, the normative values for many ECG parameters will vary in children, depending on age. Sources may differ slightly in the upper limits of