Cardiology, to a degree perhaps seen in no other subspecialty, relies heavily on the clinical skills of practitioners to identify disease. This reliance on clinical skills has not changed, even in this age of advancing and readily available technology. Cardiovascular assessment of children requires patience, thoroughness, and flexibility, because often you must adapt to children who are in varying states of arousal and may not be at all ready for you to examine them. It also requires a great deal of compassion, because few things engender as much parental anxiety as the thought that something may be wrong with their child’s heart.

In this chapter, we will review an approach to the history and physical examination of children at different ages. Because disease states may surface at different times during childhood, and because the cardiovascular system changes with age, clinical symptoms to be explored and the approach to the physical examination are described separately for four age groups: the infant, the 3- to 5-year-old child, the school-aged child, and the teenager. A systematic approach to history-taking and physical examination helps you develop the skills and confidence that will allow you to make correct decisions for most children without resorting to indiscriminate use of imaging or other investigations.

Classification of Heart Disease in Children

When taking a history of a presenting complaint with a potential cardiac cause or association, remember that heart disease in children takes several forms. These types of heart disease may be divided into the following categories:

- **Congenital:** This category can be further divided into:
  1. Structural cardiac changes present at birth (e.g., ventricular septal defect [VSD] or bicuspid aortic valve)
  2. Genetic disorders which lead to overt changes or symptoms that develop after birth (e.g., cardiomyopathies or inherited rhythm disorders, such as long QT syndrome)

- **Acquired:** Acquired heart diseases are those not manifested at birth but acquired sometime later. The most common acquired heart diseases in children are Kawasaki disease and rheumatic fever. Although acquired diseases may arise from a combination of genetic and environmental factors, the disease themselves are not present or inevitable when the child is born. Almost any disease process that affects adults can occur in children. However, in children acquired heart disease is much less common than congenital heart disease.

Thus, in approaching the cardiovascular examination of the child, be aware not only of the variations of normal but also of the wide spectrum of diseases that may occur.

**Case History**

Peter is a 3-month-old who is brought to the office by his mother because of new concerns about feeding. In the past 2 weeks, his feeding has decreased significantly. He is irritable and upset a lot of the time and seems to be constantly clammy or sweaty. His mother thinks he also breathes faster than other babies. She has not noticed that he ever looks blue, but her friends comment on how pale he is. Her husband, who works on an oil rig offshore, thinks the baby has colic and that he is overreacting.

The experienced clinician will recognize these symptoms as potentially worrisome and highly suggestive of congestive heart failure. Further history-taking that explores the details regarding his symptoms, any associated symptoms, and the perinatal and family history will help narrow down potential causes. A careful, thorough physical examination will help confirm suspicions and allow appropriate investigation, referral, and timely treatment.

In this case, the timing of the onset of symptoms is slightly later than is usually seen in lesions, such as VSDs or atrioventricular (AV) septal defects (AVSDs).
canals), which are the most common causes of congestive heart failure in infancy. These lesions usually become symptomatic between 4 and 6 weeks of life, as pulmonary vascular resistance falls enough to allow enough left-to-right shunting to cause congestive heart failure. Considering the later onset and the irritability in this child, a more unusual lesion also must be considered—an aberrant left coronary off the pulmonary artery. These lesions "steal" blood away from the left coronary system and result in both a dilated cardiomyopathy and recurrent angina for the baby. Correction of this problem can be accomplished surgically but is much more urgent than correction of a ventricular level shunt.

The assessment of infants and children who present with symptoms suggestive of heart disease is described in this chapter. Tips for separating "functional" or benign symptoms or physical examination findings from more serious ones also are provided.

History of Present Illness and Cardiac Functional Inquiry in the Infant and Young Child

Of every 1000 live births, approximately 13 infants are born with a congenital cardiovascular anomaly. Hence, when evaluating newborns and infants for potential cardiac problems, congenital heart disease should be at the top of your differential diagnoses. Less common conditions that may arise are persistent pulmonary hypertension, asphyxia, and symptomatic cardiac arrhythmias.

Congenital heart lesions can be divided into three groups:
- Obstructive lesions causing pressure overload (e.g., aortic stenosis, coarctation of the aorta, and pulmonary stenosis)
- Left-to-right shunts causing volume overload (e.g., VSD, and patent ductus arteriosus [PDA])
- Cyanotic lesions producing central cyanosis (e.g., tetralogy of Fallot, transposition of the great arteries, and tricuspid atresia)

The three most common clinical presentations of heart disease in a newborn or infant are (1) a murmur, (2) cyanosis, and (3) respiratory difficulty.

To help identify the underlying pathology, always interpret clinical findings in terms of the underlying hemodynamic disturbance, as described in the following discussions of clinical manifestations.

RESPIRATORY DISTRESS

When a newborn or a young infant is in respiratory distress, do not assume that the underlying problem is primarily respiratory. A child whose problem is primarily cardiac may present with pulmonary symptoms or secondary pulmonary complications, such as an infection.

For our purposes, two types of respiratory distress can be defined: (1) tachypnea, or abnormally rapid respirations, and (2) dyspnea, or difficulty breathing.

Cyanotic heart lesions or lesions involving low cardiac output may be associated with a compensatory rapid respiratory rate, particularly on exertion, because of diminished peripheral oxygenation. Children with cyanotic lesions often are tachypneic without displaying increased work of breathing (i.e., dyspnea).

Left ventricular (LV) failure from any cause results in a high end-diastolic pressure in the left ventricle and elevated pulmonary venous pressure. This in turn causes a higher back pressure in the pulmonary vessels and transudation of fluid into the pulmonary interstitial spaces, making the lungs less compliant. The child then works harder to breathe and becomes dyspneic. To assist with the increased work required for breathing, the accessory muscles come into use, and subcostal indrawing is observed. Respirations become rapid. The child's wet, stiff lungs are very susceptible to secondary infection, which may be the reason medical attention initially is sought for the patient.

FATIGUE, EXCESSIVE PERSPIRATION, AND POOR WEIGHT GAIN

In young infants, metabolic demands are usually greatest during feedings. Thus the infant with poor peripheral oxygenation as a result of low cardiac output tires easily during feeding (the equivalent of exercise in older children). Because of fatigue, the infant is unable to take a full feeding. In addition, rapid respiration diminishes the time available for swallowing. This combination of factors results in failure to gain weight. In the baby with a large left-to-right shunt, such as a VSD, the process is exaggerated by the higher caloric needs of an overloaded myocardium. Increased sympathetic activity causes excessive perspiration, which often is a valuable diagnostic feature.

It also is important to ask about the duration and quantity of feedings. Feeding usually is accomplished within 20 minutes, which means that the mother's breast usually should feel empty by then or the bottle should be finished. In a bottle-fed baby, the amount of formula taken per feed and how many times per day the baby feeds are important details that may help you understand the baby's difficulties and can be used in guiding future treatment.

KEY POINT

When a young baby tires rapidly, sweats during feedings, and has subcostal indrawing, always consider the possibility of congestive heart failure.

SQUATTING

Parents of older children with certain cyanotic heart defects, especially tetralogy of Fallot, may offer the observation that when their youngster tires, he or she assumes a squatting position. Squatting helps increase systemic oxygen saturation by decreasing the amount of right-to-left shunting. These days squatting is a very uncommon symptom/sign in the developed world because most children with conditions for which squatting helps have their lesions fully repaired long before they are able to walk.
CYANOSIS
Parents frequently report that their children turn blue. On further inquiry, this phenomenon turns out to be blueness of the hands and feet only, or blueness around the lips. If the lips themselves remain pink, all of these events represent peripheral cyanosis or acrocyanosis. In some disease states, this phenomenon can represent a condition in which there is decreased peripheral circulation as a result of poor ventricular function, although these children generally look unwell in other ways. Much more commonly, acrocyanosis represents immaturity of the autonomic nervous system, causing peripheral vasoconstriction and slower peripheral circulation as a result. Consequently, acrocyanosis in a child who is well should be considered benign.

In contrast, central cyanosis occurs when more than 5 g/dl of deoxygenated hemoglobin is present in the blood. This condition is most commonly manifested as blueness or sometimes as excessive redness or as a purple tinge of the lips and tongue. Although central cyanosis can come from a variety of cardiac, respiratory, neurologic (hypoventilation), or hematologic causes, its presence is always abnormal.

**KEY POINT**
Not all instances of central cyanosis are attributable to the heart; it also be seen in certain types of pulmonary disease, when abnormal hemoglobin is present at birth, or with acute methemoglobinemia at any age.

HYPERCYANOTIC SPELLS
In some forms of congenital heart disease, the degree of cyanosis worsens periodically and can be quite profound, to the point of causing loss of consciousness. These episodes are known as hypercyanotic spells and occur classically in children with cyanotic congenital heart disease that involves narrowing of the infundibulum (the subpulmonary outflow tract) and a VSD, classically tetralogy of Fallot. This clinical phenomenon is caused by either: (1) an increase in pulmonary resistance (resulting from either a subpulmonary infundibular muscle spasm or an increase in peripheral pulmonary vascular resistance) or (2) a fall in systemic vascular resistance.

Both mechanisms result in an increase in the restriction of blood flow out of the right ventricular outflow and a subsequent increase in right-to-left shunting across the VSD. Spells often are precipitated by crying. After patients with hypercyanotic spells have been stabilized, their condition should always be discussed with a pediatric cardiologist before they are discharged from the hospital or emergency department.

**KEY POINT**
Hypercyanotic spells are a medical emergency and often affect the treatment algorithm. Cases of patients with hypercyanotic spells should be discussed with a cardiologist before the patient is discharged from the hospital or emergency department.

ANGINA
Angina is rare but not unknown in infants and children; it can occur in persons with severe aortic stenosis or possibly pulmonary stenosis because of associated myocardial ischemia. It also may occur in persons with certain forms of congenital heart disease after surgical repair that involved manipulation of the coronary arteries. Angina may be seen in patients with Kawasaki disease in whom coronary stenoses or thromboses develop within coronary aneurysms. Angina has been recognized in infants with very rapid paroxysmal tachycardias and in those with an aberrant left coronary artery. Rarely, children with homozygous forms of familial hypercholesterolemia can have angina as a result of atherosclerotic coronary narrowing. Chest pain in this rare group of patients always must be taken seriously.

The manifestations of angina in young children are varied but may include classic chest pain or simply periodic irritability associated with sweating or pallor. When chest pain in children is invariably associated with exercise, angina should be a consideration.

PERIPHERAL EDEMA
Infants and young children differ strikingly from adults regarding the development of peripheral edema in the presence of congestive heart failure. Pretibial and presacral edema are late developments in a child with congestive circulatory failure. This phenomenon is believed to be due to a difference in tissue turgor. When peripheral edema resulting from heart failure does develop in an infant, it first appears periorbitally and usually is preceded by other manifestations, such as tachypnea, tachycardia, dyspnea, and liver enlargement.

ORTHOPNEA
Unlike in adults, orthopnea is not obvious in the infant with heart failure, even when tachypnea, dyspnea, hepatomegaly, and the radiographic findings of pulmonary edema are present. In the adult, orthopnea is a symptom; in the infant, it is a sign.

AGE OF ONSET
Significance of the Age of Onset of Congestive Heart Failure
The clinical significance of the age of onset of congestive heart failure is as follows:

1. If symptoms develop in a child because of congenital heart disease, there is a 95% probability that the symptoms will develop before the age of 3 months and usually before 2 months.
2. Heart failure is rarely present at birth, because the fetal circulation is in parallel and communications occur between the two sides; thus when there is obstruction on one side, blood flows easily to the other side. Because the fetal lungs are collapsed, they have a high resistance, so increased pulmonary blood flow does not occur in utero.
3. When heart failure is present right at birth, myocardial dysfunction (either resulting from a cardiomyopathy or related to in utero hypoxia) or severe AV valve...
regurgitation should be the primary considerations. Large systemic arteriovenous fistulae also can cause very early heart failure.

4. Heart failure that develops during the first week of life, and especially in the first 3 days, usually is due to an obstructive lesion, commonly hypoplastic left heart syndrome, or to persistent pulmonary hypertension.

5. Heart failure that develops 4 to 6 weeks after birth almost invariably is due to left-to-right shunting through a defect distal to the tricuspid valve (i.e., beyond the atrial level). These lesions cause LV volume overload. Pulmonary resistance is high at birth and, although a communication may exist between the two circulations, little left-to-right shunting occurs. Pulmonary resistance usually reaches its nadir by 4 to 6 weeks of age, allowing left-to-right shunting to reach a maximum.

Although atrial septal defects also cause a left-to-right shunt, they result in volume loading of the right ventricle and do not cause heart failure in children unless associated with other LV inflow obstructions.

6. If heart failure develops after the age of 3 months, look for causes other than shunts, such as myocarditis, anomalous coronary artery from the pulmonary artery, cardiomyopathy, and paroxysmal tachycardia.

**KEY POINT**

When an infant presents at age 6 weeks with respiratory distress, it may not be caused by pneumonia.

**Significance of Age of Onset of Cyanosis**

Central cyanosis resulting from congenital heart disease may be present at birth or may first appear when the ductus arteriosus closes off, usually by 5 days after birth. In persons with tetralogy of Fallot, it may develop later when the infundibular stenosis becomes more severe, increasing the volume of right-to-left shunting. Because the murmur of infundibular pulmonary stenosis greatly resembles that of a small VSD, the newborn infant presenting with what appears to be VSD could have tetralogy of Fallot.

**FAMILY HISTORY**

Genetic factors are increasingly recognized to be important in the etiology of congenital heart disease. Reports exist of as many as three children of the same parents being affected with VSD, PDA, and aortic stenosis. These are exceptions, however, because the tendency for congenital heart disease to occur in a child when one parent has a congenital heart lesion is only mildly increased (2% to 4% in most cases, compared with about 1% in the general population). The genetic tendency for a congenital heart lesion also exists in siblings, particularly for left-sided lesions such as bicuspid aortic valve. In certain syndromes or hereditary disorders with cardiac implications, however, the genetic tendency for recurrence is high. For example, Marfan syndrome is inherited as an autosomal dominant gene, and thus there is a 50% chance of recurrence. The same is true in 22q.11 deletion syndromes, which are highly associated with conotruncal cardiac defects. Consanguinity is a significant causative factor in congenital heart disease, but the tendency varies between families and with the type of heart lesion.

In summary, a search for evidence of congenital heart disease in the family is important. However, when there is a history of congenital heart disease in one parent or in a previous child, counseling regarding the risk of recurrence is best done by a geneticist.

**PRENATAL HISTORY**

Because the cause of congenital heart disease is multifactorial, known contributory factors should be sought in the prenatal history, including:

1. Exposure to drugs (e.g., lithium, hydantoin, and thalidomide)
2. Alcohol intake
3. Possible rubella in the first trimester (check the mother’s rubella immunization status)
4. Maternal diabetes (which bestows a higher risk of congenital heart malformations)
5. Exposure to radiation

In most instances, however, no specific contributory factors can be identified.

**HISTORY OF DELIVERY**

An important but infrequent cardiovascular problem in newborns is persistent pulmonary hypertension, which may cause central cyanosis, myocardial dysfunction, or both. This condition often is preceded by a difficult delivery and meconium aspiration. It is unlikely to occur after an uncomplicated delivery. Clinical differentiation of pulmonary hypertension from congenital heart disease may be difficult and usually requires echocardiography.

Determining the gestational age of a newborn also is very important because persistent patency of the ductus arteriosus is common in premature infants.

**Approach to Cardiovascular Examination of the Infant**

Because infants and children have an unfortunate habit of not always cooperating, you need to have an organized approach to the cardiovascular examination, while also staying flexible. Do what can be done when the opportunity arises. Begin by assessing the child’s physical development and looking for dysmorphic features, using a systematic approach (see Chapter 5).

Five percent of congenital heart lesions are associated with a chromosomal disorder, and many nonchromosomal dysmorphic syndromes have an associated cardiac lesion. A child with a cleft palate, for example, has a 20% possibility of having a congenital heart lesion.

Infants and toddlers usually are most comfortable on the parent’s lap during the examination. Taking a minute to play a game such as peek-a-boo at the start of the examination of a toddler often goes a long way toward winning the trust of the child, and it pays dividends in the cooperation you get later during the examination.

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INITIAL ASSESSMENT

When you start the examination, don't undress the baby right away. Begin with something relatively non-threatening, such as an examination of the hands. The baby usually allows you to examine the palmar creases and check the nail beds and muscle tone without much protest. Then feel the brachial pulses for rate, rhythm, and volume, the last being the most important. Feel this pulse in every baby you examine to learn the difference between a normal and abnormal brachial pulse. An abnormally “full” pulse suggests a PDA or aortic insufficiency. In premature infants, palpable palmar pulses (from the palmar arch) indicate the same thing. A shallow, slow-rising brachial pulse suggests LV outflow tract obstruction. Do not feel for the femoral pulses yet.

LOOKING FOR CENTRAL CYANOSIS

Because the most pressing clinical problems are congestive heart failure and cyanosis, decide early in the examination whether central cyanosis is present. Because this determination is not always easy, you may find an experienced nurse’s opinion invaluable. Many normal newborns have a deep plethoric appearance as a result of a transiently high hemoglobin concentration, particularly if there was a delay in clamping the umbilical cord. Pethora is not as obvious in the mucous membranes, so look carefully in the baby’s mouth. Deep pressure on the skin may help, because the blanched area does not pink up as quickly in infants with central cyanosis. Many normal infants exhibit a generalized mottling, particularly after being bathed (see Fig. 4–2); this condition is called cutis marmorata (literally, marbled skin) and is not pathologic. It also is common in children with Down syndrome. Observe the effect of crying. Invariably, central cyanosis resulting from cardiac disease increases during crying, but do not make the baby cry until after you have listened to the heart.

It is important to be certain of the presence of cyanosis; this determination may require a second examination and often is not at all obvious. When you are examining a child in the hospital, measuring the blood oxygen saturation will help greatly, as may observing the effect of having the child breathe 100% oxygen. Finally, remember that cyanosis may occur in only one part of the body; for example, the lower part of the body may show cyanosis while the upper part remains pink. This condition can occur with an aortic coarctation or persistent pulmonary hypertension when there is associated right-to-left shunting through a PDA.

CLINICAL MANIFESTATIONS OF HEART FAILURE

When low cardiac output and high pulmonary venous pressure cause sufficient hemodynamic disturbance to produce clinical manifestations, cardiac enlargement is invariably present. Whether the disturbance involves primarily the left or the right ventricle, the left side of the thorax becomes prominent anteriorly (Fig. 10–1). Although this prominence may not be evident in the first month of life, it certainly will be evident by the age of 3 months. When respiratory distress due to heart failure has been present for 2 months or longer, the greater diaphragmatic contractions during respiration may produce a sulcus in the lower thorax, with outward flaring of the inferior rib cage edge. Therefore, look for a sulcus, left-sided chest prominence, abnormal chest or abdominal movement, an increased respiratory rate, and subcostal indrawing.

KEY POINT

Remember that young infants are normally abdominal breathers, so you must be certain that you are not simply observing normal chest-abdominal movement. Also, be sure that the indrawing is not restricted to the midline, as occurs in persons with pectus excavatum. True subcostal indrawing is abnormal and usually signifies stiffness of the lungs from either cardiac or pulmonary causes. In contrast to adults, in young children examination of the jugular venous pulse for evidence of volume overload is useless.

PALPATION

Now lay your prewarmed hand very gently on the infant’s chest, remembering that the heart may not be in its normal position. With the tips of the first and second fingers of your right hand, depress the thorax just left of the xiphoid process (Fig. 10–2). Your fingertips are now lying on the right ventricle. A faint impulse is allowable, but if the heart is enlarged, a definite forceful movement will be present. When you perform this maneuver repeatedly in normal infants, you soon will be able to tell the difference between normal and abnormal findings. This distinction will help you make a quick decision about whether the 6-week-old baby who presents with respiratory distress has a cardiac or a respiratory problem.
LIVER SIZE AND POSITION

Whether you are right- or left-handed, stand or sit on the baby’s right side to palpate for the liver. Use the tip of your right thumb and begin well down in the right lower quadrant of the abdomen, pressing inward and upward (Fig. 10–4). If the baby has just been fed, do not press very deeply. If the edge of the liver is soft, its margin may be difficult to detect; nevertheless, if the liver is enlarged, you should appreciate a sense of resistance as your thumb tip moves superiorly. If the edge is difficult to feel, use soft percussion, tapping the second digit of your left hand with the second digit of your right hand, beginning low in the right lower quadrant and placing the second digit of your left hand parallel to the liver edge (Fig. 10–5). You should be able to sense the change in the percussion note signifying the liver’s edge. Except in the presence of pulmonary hyperinflation, the edge of the liver normally should not be more than 1 to 2 cm below the costal margin.

KEY POINT

Except in the rare instance in which the baby has a dilated cardiomyopathy, if the respiratory distress is due to heart failure, a prominent precordial pulsation is evident. It is that simple.

Now depress the thorax in the apical area. Prominence of the apical impulse is diagnostically less helpful in infants than in older children, except in rare instances, such as in tricuspid atresia in which the right ventricle is hypoplastic. Then palpate in the second interspace at the left sternal border, where a prominent pulmonary artery pulsation may be elicited. Finally, place one index finger carefully in the suprasternal notch (Fig. 10–3), searching first for an abnormal pulsation and then for a thrill. Then work in the opposite direction, searching for thrills and palpable sounds. At this point, you should have made a reasonable appraisal of the child’s cardiac dynamics.

FIGURE 10–2 Press on the precordium to the left of the xiphoid process with the first and second digits of your right hand to detect enlargement of the right ventricle.

FIGURE 10–3 Insert the index finger of your right hand deep in the suprasternal notch, searching first for pulsation and then for a thrill.

FIGURE 10–4 Palpate the liver with movement of the tip of the thumb inward and cephalad, beginning low in the right lower quadrant of the abdomen.

FIGURE 10–5 Percuss for the liver edge, using soft percussion with the second digit of your right hand on the second digit of your left hand, which has been positioned parallel to the liver edge.

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KEY POINT

In the presence of heart failure, the liver will be enlarged; therefore, if you find that the heart action is increased and the liver is enlarged upon palpation, you can be sure that the baby has a serious cardiac problem, even before you have used the stethoscope.

Finally, remember that the liver can be ectopic (i.e., on the left side or up in the thorax).

AUSSCULATION

You need all the acoustic help you can get during cardiac auscultation, so be sure to turn off radios and televisions, close the door, and have everything and everyone be as quiet as possible. Cardiac auscultation is not easy, even in older cooperative patients, but coping with a restless baby with rapid cardiac and respiratory rates in a noisy nursery is a real trial. Having the child eat or giving him or her a pacifier may help. Hospital stethoscopes frequently are of poor quality, so find and use a good one.

Remember the two main determinants of auscultatory proficiency are the fit of the earpieces and the quality (or at least the education!) of the gray matter between them.

Recognition of normal splitting of the second heart sound often is impossible when the heart rate is rapid. However, you should be able to assess the intensity or loudness of the second sound. Its intensity increases in the presence of pulmonary hypertension or when the aorta is anteriorly placed, as in transposition of the great vessels. Occasionally an ejection sound can be appreciated, which is an abnormal finding. Listen over the back for the murmur of coarctation and to both sides of the skull for the bruit of an intracranial arteriovenous malformation.

KEY POINT

Breath sounds often interfere with the interpretation of heart sounds; however, most babies will stop breathing for a couple of seconds after you administer a surreptitious puff of air in the face or briefly and gently pinch the nose.

A detailed description of cardiac auscultation and the types of findings possible is contained in a subsequent section of this chapter. The following list contains a few dogmatic but valuable generalizations concerning auscultatory findings in young infants:

1. Innocent murmurs are not commonly heard in neonates, so if you hear a murmur, take it seriously, particularly if it is not musical.
2. If you note a loud, coarse systolic murmur in the first 3 days of life, the baby has some type of obstruction.
3. The murmur of a VSD often is not present in the first week of life.
4. The murmur of the PDA usually is not continuous in the first week of life and may be loudest at the left sternal border in the third and fourth interspaces, which is not its point of maximal intensity later in life.
5. Occasionally, a long, high-pitched, blowing, organic-sounding systolic murmur is encountered, heard maximally in the axillae. This murmur, which is common in premature infants, also can be heard in full-term babies who have an increased stroke volume. It arises in the peripheral pulmonary arteries and is usually innocent. This murmur is believed to be the result of a relatively more acute angle of takeoff of the branch pulmonary arteries in the newborn, as compared with later life. If such a murmur persists in an infant older than 6 months or is associated with other abnormal findings, consult a cardiologist.
6. A murmur that has the same characteristics as described in point No. 5 but is heard only in the left axilla and in the back could well be due to aortic coarctation. Verification of normal femoral pulses and blood pressures is important in such a case, and, if there is doubt, a cardiologist should be consulted.

PALPATING THE FEMORAL PULSES

Palpation of the pulses calls for gentleness, persistence, and patience, so make yourself comfortable before you begin. First, remove the baby’s diaper and palpate for femoral pulsations. Many babies do not appreciate having people poke around in their groins and may cry, urinate, or both. Femoral pulses are particularly difficult to appreciate in obese babies; thus, do not rush into a diagnosis of coarctation of the aorta if you have difficulty feeling them. If the femoral pulses are not palpable in an asthenic baby, however, there is cause for concern (see Fig. 4–20). Now palpate both brachial pulses again. If you can detect good brachial pulses and are certain that the femoral pulses are absent or greatly depressed, listen to the heart before measuring the blood pressure and upsetting the baby. Listen particularly for a high-pitched blowing systolic murmur, which is best heard anteriorly below the left clavicle and can be heard clearly in the left axilla and back, medial to the scapula.

KEY POINT

Check for wide splitting of the first heart sound at the apex; the second component of the split sound probably indicates the presence of a bicuspid aortic valve, which accompanies aortic coarctation in up to 85% of cases.

BLOOD PRESSURE

Although measuring the systemic blood pressure can be a difficult task, you should try to do so. The normal systolic blood pressure of an infant is between 60 and 80 mm Hg in both the arm and the leg (Table 10–1). The four methods of measuring blood pressure are (1) auscultatory, (2) palpatory, (3) visual (flush), and (4) Doppler.

All methods require the use of an inflatable cuff. The first decision is to choose the size of the cuff; the size is of great importance because the measuring the pressure involves occlusion of the arterial pulse (or the brachial arterial pulse when the arm is used). If the cuff used is too small, greater pressure must be used to obliterate the
pulse, and the blood pressure measured will be artificially high. Use a cuff that covers almost the full extent of the upper arm, with the elbow bent. Always have a full selection of cuff sizes available.

For all methods of measuring blood pressure, first supinate the child’s arm to make the radial artery easily accessible. Apply the cuff, elevate the arm, and then inflate the cuff. Prior elevation of the arm (or opening and closing the hand) prevents the auscultatory gap phenomenon (Fig. 10–6). If this procedure is not followed, when you inflate the cuff and listen for Korotkoff sounds, as you decrease the pressure in the cuff you may hear a sound appear, then disappear, and then reappear as the pressure is further decreased. This phenomenon, known as the auscultatory gap, occurs because of increased vascular resistance distal to the cuff.

The conventional method of measuring blood pressure is the auscultatory method. The edge of the diaphragm of a warm stethoscope is placed under the inferior edge of the cuff, and the cuff is inflated. Listen for the Korotkoff sounds as the cuff pressure is decreased, watching the mercury level (or the needle in an aneroid sphygmomanometer) as you listen. The first sound you hear denotes the systolic level. As the cuff pressure is decreased further, an abrupt change in the intensity of the sound may be heard. If this change is detected, it is recorded as the diastolic level. Continue decreasing the cuff pressure, recording the disappearance of Korotkoff sounds, which is recorded as the diastolic level if no intensity change has been detected. Usually two or three recordings are made, elevating the arm before each attempt.

It may be impossible to measure the blood pressure by the auscultatory method, particularly when a baby does not cooperate. The palpatory method of measurement should then be attempted. Prior elevation of the arm is not required. The radial artery pulse is palpated, and the cuff is elevated until the pulse disappears. The cuff pressure is then decreased, and the level of systolic pressure is estimated by the time of the reappearance of the pulse. Only the systolic pressure can be measured by the palpatory method. A variation of this method involves detection of the appearance of the pulse with a manual Doppler probe held on the patient’s radial or brachial pulse. This method is the one we prefer using for infants, in whom cooperation for auscultation is so frequently limited.

The flush method also measures systolic pressure only, and unfortunately, it requires two persons. Using both hands on the child’s upraised arm, express most of the blood from the arm. This maneuver sometimes can be accomplished by wrapping the arm tightly from the fingers to the cuff level with a tensor bandage. The second person should then inflate the cuff and unwrap the bandage. The arm should look relatively pale. One person watches the arm, and the other watches the sphygmomanometer. As the cuff pressure is decreased, the person watching the arm indicates verbally the moment at which the flush occurs while the other person records the manometer level.

Blood pressure measurement with Doppler equipment is much easier than with the other methods, because no auscultation is required. Unfortunately, the equipment is not always available, blood pressures obtained are sometimes questionable, and the choice of cuffs may be limited.

One way or another, a reliable blood pressure measurement must be obtained. If you suspect an aortic coarctation (i.e., if there is high pressure in the arm, an absence of femoral pulses, and a murmur), repeat the procedure in the thigh, using a blood pressure cuff of an appropriately larger size. Again, the manual Doppler method with the probe held on the posterior tibial or popliteal pulse is

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Respiratory difficulty that occurs in older children as opposed to infants usually is precipitated by activity rather than by feeding. Respiratory symptoms are closely related to exercise intolerance, which is the primary manner in which respiratory symptoms manifest in this group. Eliciting the exact nature of the respiratory symptoms that are being experienced is important. For example, stridor on exertion has a different differential diagnosis than does dyspnea on exertion. In the former, a vascular ring would be an important diagnosis to exclude, particularly if the child has concomitant swallowing difficulties. Shortness of breath on exertion is a common finding in older children with heart disease, but it usually is not described that way. Instead, children may complain that they are not able to keep up with their same-age peers in gym class. They may stop before other children at play when participating in activities such as bike riding or running. You can inquire about this phenomenon in a very concrete way by asking about common childhood games, such as tag. For example, you can ask children if they are always “it” when playing tag or if they often finish last in races. Try to quantify exercise tolerance by asking the number of stairs the patient can go up without becoming tired or whether it is necessary to pause halfway up the stairs to take a rest before continuing. Ask children who cannot climb any stairs how far they are able to walk on a level surface. Other (usually older) children have a feeling of fatigue in the absence of activity.

**KEY POINT**

When exercise tolerance is reduced, it is important to inquire about the time course of its development. Cardiomyopathies and myocarditis often present with new onset of exercise intolerance, while congenital problems may cause a more insidious reduction in functional capacity.

**POOR WEIGHT GAIN**

Like babies with heart disease, older children with serious heart disease often do not grow very well. This finding is most pronounced in children with congestive heart failure and is caused by the increased energy needs of the body created by the heart failure. These increased metabolic demands use up the energy that otherwise would be used for growth. Compounding the problem, these children often have poor food intake, either because they tire easily when eating or because they cannot tolerate food due to intestinal edema related to the heart failure. A former teacher used to enjoy asking students and cardiology fellows about this phenomenon at rounds. He would say, “If you were trapped on a desert island with only one instrument to look after children with heart disease, what would it be?” Almost invariably, the uninitiated would say, “A stethoscope.” However, the answer he was looking for was “a scale,” because children with serious heart disease just do not grow properly.

**CYANOSIS**

 Cyanosis is distinctly uncommon in older children presenting for the first time with possible heart disease because most cyanotic defects have been repaired or palliated by the time children reach 5 years of age. It is still worth asking about, though, because occasionally a patient with

TABLE 10-2  ACCEPTABLE HEART RATES IN INFANTS AND CHILDREN

<table>
<thead>
<tr>
<th>Age</th>
<th>Resting Pulse Rates (Beats/Min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Awake</td>
</tr>
<tr>
<td>Newborn</td>
<td>100-180</td>
</tr>
<tr>
<td>1 wk–3 mo</td>
<td>100-220</td>
</tr>
<tr>
<td>3 mo–2 yr</td>
<td>80-150</td>
</tr>
<tr>
<td>2–10 yr</td>
<td>70–110</td>
</tr>
<tr>
<td>&gt;10 yr</td>
<td>55–90</td>
</tr>
</tbody>
</table>

Table 10-2: Acceptable heart rates in infants and children. The normal range of systolic and diastolic blood pressures for older children is shown in Table 10-2. In our experience, the so-called radial-femoral pulse delay is a useless sign in infants with a rapid heart rate. It is easy to say that such a delay is present when one knows in advance that the diagnosis is aortic coarctation. It is better to rely on a comparison of pulse volumes and blood pressure measurements.

**ADDITIONAL MANEUVERS**

After listening to the infant’s back and finishing the general examination, you may have to make the baby cry, so advise the parents of your intention. Gently flicking the bottom of the foot usually does the trick, but at times a surreptitious pinch or two of the big toe (the dermal compression test) is required. While the baby cries, look for central cyanosis. Remember that central cyanosis occurs in any baby who holds his or her breath for a prolonged period. If the mucus membranes inside the mouth exhibit a purplish discoloration while the baby is crying vigorously, the infant probably has a serious problem. Discoloration of the buccal mucous membranes may be the one and only clinical finding in transposition of the great arteries, a disorder that is potentially lethal if not diagnosed early. If you have any doubts about this diagnosis, consult a cardiologist as soon as possible.

**Approach to the Cardiovascular Assessment of the Older Child**

**HISTORY-TAKING**

Although many of the symptoms sought in infants and older children are similar, the cardiovascular history of the older child differs somewhat from that of the infant, because the diseases seen are different and children are becoming more active. It is therefore important to modify the cardiac functional enquiry accordingly for older children. For example, respiratory symptoms that previously were precipitated by feeding may be brought on with exercise or play. These modifications are discussed in the following sections.

**RESPIRATORY SYMPTOMS AND EXERCISE INTOLERANCE**

Respiratory difficulty that occurs in older children as opposed to infants usually is precipitated by activity rather than by feeding. Respiratory symptoms are closely related to exercise intolerance, which is the primary manner in which respiratory symptoms manifest in this group. Eliciting the exact nature of the respiratory symptoms that are being experienced is important. For example, stridor on exertion has a different differential diagnosis than does dyspnea on exertion. In the former, a vascular ring would be an important diagnosis to exclude, particularly if the child has concomitant swallowing difficulties. Shortness of breath on exertion is a common finding in older children with heart disease, but it usually is not described that way. Instead, children may complain that they are not able to keep up with their same-age peers in gym class. They may stop before other children at play when participating in activities such as bike riding or running. You can inquire about this phenomenon in a very concrete way by asking about common childhood games, such as tag. For example, you can ask children if they are always “it” when playing tag or if they often finish last in races. Try to quantify exercise tolerance by asking the number of stairs the patient can go up without becoming tired or whether it is necessary to pause halfway up the stairs to take a rest before continuing. Ask children who cannot climb any stairs how far they are able to walk on a level surface. Other (usually older) children have a feeling of fatigue in the absence of activity.

**KEY POINT**

When exercise tolerance is reduced, it is important to inquire about the time course of its development. Cardiomyopathies and myocarditis often present with new onset of exercise intolerance, while congenital problems may cause a more insidious reduction in functional capacity.

**POOR WEIGHT GAIN**

Like babies with heart disease, older children with serious heart disease often do not grow very well. This finding is most pronounced in children with congestive heart failure and is caused by the increased energy needs of the body created by the heart failure. These increased metabolic demands use up the energy that otherwise would be used for growth. Compounding the problem, these children often have poor food intake, either because they tire easily when eating or because they cannot tolerate food due to intestinal edema related to the heart failure. A former teacher used to enjoy asking students and cardiology fellows about this phenomenon at rounds. He would say, “If you were trapped on a desert island with only one instrument to look after children with heart disease, what would it be?” Almost invariably, the uninitiated would say, “A stethoscope.” However, the answer he was looking for was “a scale,” because children with serious heart disease just do not grow properly.

**CYANOSIS**

Cyanosis is distinctly uncommon in older children presenting for the first time with possible heart disease because most cyanotic defects have been repaired or palliated by the time children reach 5 years of age. It is still worth asking about, though, because occasionally a patient with
pulmonary hypertension or an undiagnosed cyanotic lesion turns up, and the presence of cyanosis may be an important clue to the diagnosis.

**PALPITATIONS**

Palpitations are uncommon, but they can occur in very young children. Palpitations are discussed in detail in the section on teenagers (pages xxx-xxx). In young children, it is important to remember that palpitations may be reported as “chest pain” or simply as an uncomfortable feeling in the throat, the latter being particularly common in the presence of supraventricular tachycardia (SVT).

**SYNCOPE**

Although true cardiac syncope is uncommon in young children, the practice of holding one’s breath is not uncommon. As described for older patients later in this chapter, the history is the key to the diagnosis. Breath-holding typically is practiced by children between the ages of 6 months and 6 years and rarely occurs beyond 8 years of age. Episodes may be associated with cyanosis or pallor. In contrast to most cases of cardiac syncope (which usually occur out of the blue), breath-holding episodes are almost always triggered by an injury or anger-inducing event, though such triggers may be relatively minor. As with older children, exploration of the family history is important (see the section on teenagers), with consideration of a wider differential when there is a history of sudden or unexplained death. When the description of the events suggests cardiac syncope, patients should be referred to a pediatric cardiologist. A more detailed description of syncope is included in the section on teenagers.

A summary of points to include in the cardiac functional inquiry at different ages is provided in Table 10–3.

**MEDICAL HISTORY AND MEDICATION USE**

The medical history also is important in older children because the presence of certain syndromes, which may have been undiagnosed in younger children, may lead you to suspect a cardiac problem. For example, a child known to have a history of tracheal-esophageal fistula repair might have VACTERL syndrome, a condition commonly associated with congenital heart defects such as VSDs. Similarly, in a girl complaining of short stature, subsequent examination might reveal findings of Turner syndrome. Some children, such as recent immigrants or patients who recently have moved from other cities, may have a previously unknown cardiac condition or have had previous cardiac surgery. Eliciting this information is important, because it clearly would affect your interpretation of other symptoms.

Medication use, including the dose, frequency, and duration of use, should always be explored carefully. Ask specifically about over-the-counter medications and herbal remedies, because they often are overlooked but may have cardiac effects. Medication use may be solely or largely responsible for some symptoms, notably palpitations. In other cases, a medication may be contraindicated in a particular heart condition, such as erythromycin in patients with long QT syndrome. Other aspects of the history are similar to those described for younger and older children.

**Approach to the Cardiovascular Examination of the 3- to 5-Year-Old Child**

**GENERAL EXAMINATION, INSPECTION, AND PALPATION**

The general examination, inspection, and palpation portions of the examination of a 3- to 5-year-old are similar to those for the infant and toddler. The decision to examine the child while he or she is on an examination table or in the parent’s lap must be individualized; often it is useful to keep the patient in the parent’s lap. Both the parent and child should face you and sit with the child facing forward. As with younger children, you should start by examining the hands and brachial pulses. The transition to a supine position is easy with a child who is facing forward—just raise the legs and pull forward slightly. This position, with the majority of the child’s body still on the parent’s lap, keeps the child comfortable while giving you easy access to the precordium for palpation and auscultation and to the legs for checking the femoral pulses. For children inclined to kick, you can maintain control of the legs by holding them close to your body underneath your arms. These positions are pictured in Figure 10–7.

By the time a child is 3 to 5 years old, lesions causing cyanosis or congestive heart failure will have been revealed. The spectrum of disease in toddlers and young children includes congenital lesions that have been overlooked or cause few symptoms, such as atrial septal defects (ASDs), small VSDs, bicuspid aortic valve, and acquired cardiac disorders, such as pericarditis, myocarditis, cardiac manifestations of hereditary muscular and neuromuscular diseases, rhythm disturbances, and other rare disorders. By far, the most common problem clinicians face in this age group is the interpretation of heart sounds and murmurs, especially the systolic murmur. Consequently, we will concentrate on this issue.

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but the murmur of obstruction or excessive flow begins after the preceding sound. If you can comprehend this process, you are well on the way to understanding cardiac auscultation.

Memorize these two facts:
1. The left-sided valves close before the right-sided valves.
2. Left-sided valve closures are much louder. The right-sided valve can be heard closing only when the stethoscope is positioned directly over it on the chest.

The mechanism of production of the \( S_1 \) and \( S_2 \) heart sounds is in question. They probably are a result of the deceleration of blood at the end of early (\( S_3 \)) and late (\( S_4 \)) rapid filling phases of the ventricles. Although the exact mechanism of the third and fourth heart sounds is poorly understood, \( S_3 \) usually is related to high flows and \( S_4 \) reflects a poorly compliant ventricle. \( S_4 \) sounds are normal in children with hyperdynamic circulations and thin chest walls but are usually abnormal in patients older than 30 years, when the effects of age have lowered stroke volume and increased body mass. Audible \( S_4 \) sounds are always abnormal. \( S_3 \) and \( S_4 \) sounds occur in the ventricles and are low-pitched. They are heard loudest over the ventricle in which they occur and are best heard with the bell of the stethoscope.

Use of the terms clicks and snaps is a continual source of confusion. Valve opening is quiet in healthy persons and signals the end of the period of isovolumic contraction, or relaxation.

**KEY POINT**

A problem exists when a sound is heard at the time of the opening of any heart valve.

A sound heard at the time of opening of the pulmonary or aortic valve is called an ejection click; when mitral or tricuspid opening is heard, the term opening snap is used. The clicks signal the beginning of ejection into a dilated great vessel; the snaps signal the commencement of diastolic flow into the ventricle. Both are always high-pitched and are loudest over their respective valves, except the aortic click, which is usually heard clearly at the apex. The pulmonary ejection click is unique in that it is loudest or sometimes only heard during expiration. The only hope of identifying these sounds is a thorough working knowledge of what is normal and of what you would expect to hear in a normal infant or child when you place a stethoscope on a particular area of the chest. Normal and abnormal sounds for each listening area are shown in Figure 10–8.

**KEY POINT**

It is essential to follow a constant, systematic procedure for listening to heart sounds and murmurs in all children. Never auscultate the heart through clothing. The examination is difficult enough without such interference.

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