Cardiovascular Assessment of Infants and Children

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INTRODUCTION

Before the era of cardiac catheterization, cardiac ultrasound, nuclear studies, and computerization, few aids were available to supplement the eyes, hands, and ears. Today, some patients are examined by ultrasound even before a thorough beside appraisal has been performed. Such misuse of modern technology can escalate medical costs when a patient's problem might be resolved easily at the bedside by a good clinical assessment. A systematic approach will help you develop the skills and confidence that will allow you to make correct decisions for most children without indiscriminate use of high-tech procedures.

CLASSIFACATION OF HEART DISEASE IN CHILDREN

Heart disease in children may be divided into the following categories:

I . Congenital A. Structural cardiac changes present at birth i.e. ventricular septal defect

B. Genetic tendencies which lead to overt changes developing after birth i.e. cardiomyopathy

II Acquired Almost any disease process that affects the adult can occur in the child. Processes such as neoplasia, cardiac infection, metabolic and endocrine abnormalities and auto-immune disorders, may occur in the child. Certain disorders such as rheumatic fever, now uncommon in North America is still prevalent in South America, and occurs more commonly in the child. Thus in the approach to the examination of the child, one must be ever aware not only of the variations of normal, but of the wide spectrum of diseases that may occur in the child.

Where disease states may surface at different times during childhood, and where the cardiovascular system changes with the child's age, the physical examination of the cardiovascular system will be approached for three age groups**; the infant**, the **three year old** and **the teenager**.

EXAMINING THE INFANT AND YOUNG CHILD

The commonest heart lesion that affects the newborn and older infant is congenital heart disease. Other less common causes are persistent pulmonary hypertension, asphyxia and symptomatic cardiac arrhythmia.

Of every 1,000 live births, approximately 13 are babies with a congenital cardiovascular anomaly. Congenital heart lesions may be divided naturally into three groups:

1. obstructive lesions—cause pressure overload (aortic stenosis, coarctation of the aorta, and pulmonary stenosis)

2. left-to-right shunts—cause volume overload (ventricular septal defect, atrial septal defect, and patent ductus arteriosus)

3. cyanotic lesions—produce central cyanosis (tetralogy of Fallot, transposition of the great arteries, and tricuspid atresia)

The three most common clinical presentations are (1) a **murmur**, (2) **cyanosis**, and (3) **respiratory difficulty.**

Always interpret the clinical findings in terms of the underlying hemodynamic disturbance, as illustrated in the following clinical manifestations:

RESPIRATORY DISTRESS

When respiratory distress occurs in a newborn or a young infant, do not assume the underlying problem is primarily respiratory. A child whose problem is primarily cardiac may present with pulmonary infection.

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

Cyanotic heart lesions or lesions associated with low cardiac output may be associated with a compensatory rapid respiratory rate, particularly on exertion, because of **diminished peripheral oxygenation**. When left ventricular failure results in a high end-diastolic pressure in the left ventricle and elevated pulmonary venous pressure, the early clinical manifestations such as easy fatigue result from **low cardiac output**. **Increased pulmonary venous pressure** causes increased stiffness of the pulmonary vessels and transudation of fluid into the interstitial tissue, making the lungs less compliant. The child works harder to breathe. Wet stiff lungs encourage secondary infection; respirations become rapid, the accessory muscles come into use, and subcostal indrawing is observed.

FATIGUE, EXCESSIVE PERSPIRATION, AND POOR WEIGHT GAIN

In young infants, metabolic demands are usually greatest during feedings. The infant with poor peripheral oxygenation due to low cardiac output will tire easily during feeding, the equivalent of exercise in older children. As a result 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, the process is exaggerated by the increased caloric needs of an overworked myocardium. Increased sympathetic activity causes **excessive perspiration**—often a valuable diagnostic feature. Any baby with this clinical presentation has congestive heart failure until proved otherwise. When a young baby tires rapidly, sweats during feedings, and has subcostal indrawing, always think of the possibility of congestive heart failure.

SQUATTING

Parents of 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.

CENTRAL CYANOSIS

Central cyanosis is caused by increased deoxyhemoglobin content (greater than 5 g%), reducing oxygen available for delivery to the tissues. By various compensatory mechanisms, the fetus lives happily in utero, despite a low (65 percent) oxygen saturation. Even when a congenital anomaly such as transposition of the great arteries is present, birth weight is usually normal.

HYPOXIC SPELLS

These typically occur in children with cyanotic congenital heart disease that involves stenosis of the infundibulum of the right ventricular outflow tract and ventricular septal defect, classically known as tetralogy of Fallot. A typical spell is characterized by a sudden increase in intensity of the cyanosis, at times associated with loss of consciousness. This clinical phenomenon is caused by infundibular muscle tissue contraction, further restricting right ventricular outflow and increasing right-to-left shunting.

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

ANGINA

Angina is rare but not unknown in infants and children; it can occur in severe aortic stenosis, or possibly in pulmonary stenosis, due to associated myocardial ischemia. It also may occur with very rapid paroxysmal tachycardias and has been recognized in infants with an aberrant left coronary artery.

PERIPHERAL EDEMA

Infants and young children differ strikingly from adults in the development of peripheral edema in congestive heart failure. Pretibial and presacral edema are late developments in the child's congestive circulatory failure picture, apparently due to difference in tissue turgor. When peripheral edema due to heart failure does develop in an infant, it first appears **periorbitally**, usually preceded by other manifestations such as **tachypnea**, **tachycardia**, **dyspnea**, and **liver enlargement**.

ORTHOPNEA

Unlike 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.

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 a child becomes symptomatic due to congenital heart disease, there is a 95 percent probability that those symptoms will develop **before 3 months of age** and usually before 2 months.

2. Heart failure is **rarely present at birth** because the fetal circulation is in parallel and there are communications between the two sides. When there is obstruction on one side, blood flows easily to the other. As the fetal lungs are collapsed, increased pulmonary blood flow does not occur in utero.

3. Heart failure that develops during the first week of life, especially in the first 3 days, is usually due to an **obstructive lesion or to persistent pulmonary hypertension**.

4. Heart failure that develops at 4 to 6 weeks of age is invariably due to **left-to-right shunting** through a defect (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 bottoms out by 4 weeks of age, allowing left-to-right shunting to reach a maximum.

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

5. If heart failure develops after 3 months of age, look for causes other than anomalies, such as **myocarditis, cardiomyopathy**, or **paroxysmal tachycardia**.

6. Central cyanosis due to congenital heart disease **may be present at birth** or may appear first when the ductus closes off, **usually by 5 days of age** In tetralogy of Fallot, it may develop later (2 months of age or older) when the infundibular stenosis becomes more severe, increasing the volume of right-to-left shunting.

OBTAINING THE HISTORY

FAMILY HISTORY

If one parent has a congenital heart anomaly, the risk of the child having one (frequently the same type) can be as high as 10 percent. When a first cousin has a congenital heart anomaly, the risk of a sibling having one is approximately 2 percent. With no family history of congenital heart disease, if the firstborn has a congenital heart lesion, the risk of a second child having a congenital heart lesion is 2 to 3 percent, slightly higher than the risk for the general population.

PRENATAL HISTORY

Because the etiology of congenital heart disease is multifactorial, known contributory factors should be sought, including a) exposure to drugs (lithium, dilantin, thalidomide), b) excessive alcohol intake, c) possible rubella in the first trimester and rubella immunization status, d) maternal diabetes (which carries an increased risk of congenital heart malformations) and e)

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 from congenital heart disease may be difficult and usually requires cardiac ultrasound. It is important to elicit a history of **prematurity**, as patency of the **ductus arteriosus** is common in the premature baby.

APPROACH TO PHYSICAL EXAMINATION OF THE INFANT.

Where infants and children have an unfortunate habit of not always cooperating ideally, organize a thorough agenda for the cardiovascular examination but stay 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 Ch. 4).

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

The infant is usually most comfortable on the parent's lap. Do not undress the baby right away. Examination of the palmar creases is usually permitted. The nail beds, and muscle tone can be checked without much protest. Then feel the brachial pulses for rate, rhythm, and volume, the last being the most important. Do this on every baby you examine to learn the difference between normal and abnormal. An abnormally full pulse suggests patent ductus or aortic insufficiency; a

shallow slow rising pulse suggests left ventricular outflow tract obstruction. Do not feel for the femoral pulses--- yet.

Where the most pressing problems are congestive circulatory failure and cyanosis, decide early in the examination whether **central cyanosis** is present. Because this is not always easy, an experienced nurse's opinion may be invaluable. Many normal newborns have a deep plethoric appearance due to their transiently high hemoglobin concentrations, particularly if the obstetrician was slow in clamping the umbilical cord. Plethora 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 will not pink up as quickly in central cyanosis. Many normal infants exhibit a generalized mottling, particularly after being bathed (see Fig. 3-2 in Chapter 3 on Examination of the Newborn). This is called **cutis marmorata**, literally "marbled skin." Observe the effect of crying. Invariably, central cyanosis due to cardiac disease increases during crying, but do not make the baby cry until after listening to the heart. It is important to be certain of the presence of cyanosis; this may require a second examination. In the hospital, determining the blood oxygen saturation will help greatly, and the experience of having observed the effect of breathing 100 percent oxygen may be helpful. Finally, remember that cyanosis can be differential: the lower body may be cyanosed while the upper part is pink. This can occur with an aortic preductal coarctation or persistent pulmonary hypertension when there is associated right-to-left shunting through a patent ductus.

CLINICAL MANIFESTATIONS OF HEART FAILURE

When low cardiac output and high pulmonary venous pressure cause sufficient hemodynamic disturbance to produce clinical manifestations, cardiac enlargement invariably is present. Whether the disturbance involves primarily the left or the right ventricle, **the left side of the thorax is prominent anteriorly** (Fig. 9-1). This may not be evident in the first month of life, but it certainly

will be by 3 months of age. When respiratory distress due to heart failure has been present for 2 months or more, the increased 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 movement, increased respiratory rate, and subcostal indrawing.

Remember, young infants normally have **abdominal breathing,** so be certain that it is not simply normal chest-abdomen movement that is present. Also be sure that the indrawing is not restricted to the midline, as occurs in **pectus excavatum**. True subcostal indrawing is abnormal and usually means stiff lungs from either cardiac or pulmonary causes. In contrast to adults, examining the **jugular venous pulse** is useless in young children.

PALPATION

Now lay a prewarmed hand very gently on the chest, remembering the heart may not be in its normal position. With the tips of the right first and second fingers, depress the thorax just left of the xiphoid process (Fig. 9-2). The 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. Do this repeatedly in normal infants and the difference between normal and abnormal will be evident. This maneuver will aid a quick decision about whether the 6-week-old who presents with respiratory distress has a cardiac problem or a respiratory problem.

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

Now **depress the thorax in the apical area**. Prominence of the apical impulse is diagnostically less helpful in infants, except in rare instances such as in tricuspid atresia, when 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. 9-3), searching first for an abnormal pulsation and then for a thrill. Then work in the opposite direction, searching for thrills and palpable sounds. By this time, a reasonable appraisal of cardiac dynamics should have been made.

If increased heart action cannot be palpated and if the pulses are of normal volume, the child does not have a serious hemodynamic disturbance.

LIVER SIZE AND POSITION

Whether you are right- or left-handed, stand or sit on the baby's right side. Use the tip of the right thumb and begin well down in the right lower quadrant of the abdomen, pressing inward and upward (Fig. 9-4). If the baby has just been fed, do not press too deeply. If the liver edge is soft, its margin may be difficult to appreciate; nevertheless, a sense of resistance as the thumb tip moves superiorly should be appreciated if the liver is enlarged. If the edge is indefinite, use soft percussion, tapping the second digit of the left hand with the second digit of the right, beginning low in the right lower quadrant, and placing the second digit of the left hand parallel to the liver edge (Fig. 9-5). The percussion note change signifying the liver edge should be sensed. Except in the presence of pulmonary hyperinflation, the liver edge normally should not be more than 1–2 cm below the costal margin.

If there is heart failure, there will be liver enlargement; therefore, if you find the heart action to be increased and the liver enlarged to palpation, you can be sure that the baby has a serious cardiac problem, even before you have applied the stethoscope.

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

AUSCULTATION

You need all the acoustic help you can get, so be sure to turn off the radio and television, close the door, and get everything and everyone 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. A bottle or pacifier may help. Nursery stethoscopes are frequently of poor quality, so bring a good one. Remember, the two main determinants of auscultatory proficiency are (1) the fit of the ear pieces and (2) the quality of the gray matter between them. Recognition of normal splitting of the second heart sound is often impossible when the heart rate is rapid. It should be possible, however, to assess the *intensity* 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.

Because breath sounds often interfere with the interpretation of heart sounds, remember that most babies cease breathing for a few seconds after a surreptitious puff in the face by the examiner.

The following are a few dogmatic but valuable generalizations concerning auscultatory findings in young infants:

1. **Innocent murmurs are heard less frequently in neonates**, so if a murmur is heard, take it seriously, particularly if it is nonmusical.

2. If a **loud coarse systolic murmur** is noted in the first 3 days of life, the baby has some type of obstruction.

3. The **murmur of a ventricular septal defect** is often not present in the first week of life.

4. Frequently, the **patent ductus murmur is not continuous in the first week of life** and may be loudest at the left sternal border in the third and fourth interspaces—not its point of maximal intensity in later life.

5. Occasionally, a long, high-pitched, blowing, "organic-sounding," systolic murmur is encountered, heard maximally in the axillae. Common in prematures, it also can be heard in full-term babies with an increased stroke volume. This murmur arises in the **peripheral pulmonary arteries** and is usually innocent. If it persists after 2 months of age, call the cardiologist.

6. A murmur that has the same characteristics as the above but is heard only in the left axilla and in the back could well be due to aortic coarctation, so keep looking.

Possibly an example of an experience with a real patient may help in appreciating the importance of these manifestations. When attending a clinic in another hospital, I was asked to see an eight week old infant. The working diagnosis was pneumonia and there was a history of failure to gain in weight. I was alone during the examination. The infant had obvious Down Syndrome. I said to myself. " 70% 88888of patients with Down Syndrome have a congenital heart condition". There was obvious respiratory distress with subcostal indrawing, which could be either due to pneumonia or heart failure. The left side of the precordium was prominent but not beyond normal limits. I placed a pre-warmed hand on the left side of the precordium. There was marked increase in the heart action-----unquestionably abnormal. On palpation of the liver there was resistance down into the pelvis, but on close examination I could palpate the edge, deep in the pelvis. "How could anyone miss this", I said to myself. On auscultation, there were no murmurs! This of course can happen when pressures and resistances in the right side of the heart are increased, as in heart failure, where velocity of left to right shunting, one of the causes of murmurs, is decreased. The absence of heart murmurs possibly was the cause of the missed diagnosis. This infant went on to

have surgical correction of a complete AV canal. The diagnosis of cardiac disease was made by palpating the precordium, and was confirmed by the gross hepatomegally, so gross that it was missed by previous examiners. This case is cited to remind you that simple bedside procedures can be extremely important.

PALPATING THE PULSES

This part of the examination calls for gentleness, persistence, and patience, so get comfortable. First, palpate for femoral pulsations. Remove the diaper. Many babies do not appreciate having their groins manipulated and may cry, urinate, or both. Femoral pulses are particularly difficult to appreciate in obese babies; **do not rush into a diagnosis of coarctation of the aorta** if you have difficulty feeling them. If they are not palpable in an asthenic baby, that is another matter (see Chapter 3, Fig. 3-21). Go back and palpate both brachial pulses. If good brachials are palpated and it is certain that the femorals are absent or greatly depressed, before taking a blood pressure and upsetting the baby, return and listen to the heart. Listen particularly for a high-pitched blowing systolic murmur, best heard anteriorly below the left clavicle and well heard in the left axilla and back, medial to the scapula.

Check also 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 75 percent of cases.

BLOOD PRESSURE

Try to take the blood pressure. This can be difficult and time-consuming, but it is an essential part of the assessment. The normal systolic blood pressure of an infant is about 60 to 80 mmHg in both the arm and the leg (Table 9-1).

The first decision is choosing the correct cuff: too small a cuff will cause artifactual pseudohypertension. The old adage about covering two-thirds of the upper arm is worse than useless, especially in infants and small children. A cuff that covers almost the full length of the upper arm with the forearm bent is usually right. If the arm is obese, use a larger cuff than will cover the entire upper arm. Always have a generous range of cuff sizes available.

Before inflating the cuff, supinate the hand to make the radial artery easily accessible and elevate the arm to prevent the "auscultatory gap" phenomenon (Fig. 9-6).

Frequently, after expanding the cuff to an appropriate level and listening for the first Korotkoff sound as the cuff pressure is being diminished, the first sound may appear, only to disappear, then reappear as the pressure is further decreased. This silent area, or **auscultatory gap,** reflects increased vascular resistance distal to the cuff. **It may be abolished by elevating the arm or opening and closing the hand before expanding the cuff.** The second decision is what method of blood pressure measurement to use. If Doppler equipment (Dynamap) is available, apply the equipment, sit back, and record the pressure repeatedly when the baby is quiet. Otherwise, it must be attempted in the conventional way, listening for the Korotkoff sounds, using phase 1 for the systolic pressure and phase 4 (or 4-5) for the diastolic. Use the diaphragm of the stethoscope because it is difficult to obtain a satisfactory seal with the bell. Position it directly over the radial artery, with the upper edge barely tucked under the cuff. The palpation method will likely fail also because it is difficult to keep the baby's arm quiet long enough to sustain palpability of the radial pulse.

A satisfactory blood pressure recording usually can be obtained by using the flush method, which unfortunately calls for three hands; two to hold the arm pointing skyward while the blood from the arm is expressed ("milked") and one to pump up the sphygmomanometer cuff. As an

alternative, much of the superficial blood from the arm can be expressed by wrapping it in an elastic bandage, starting at the hand.

Whatever method is used, step 2 is to pump up the cuff to a level well above expected systolic pressure and then remove the bandage or release the arm. As one examiner slowly lowers the pressure and watches the manometer, the other examiner signals at the instant the arm flushes, at which point examiner 1 notes the pressure reading. The flush, which provides a sharp endpoint, occurs when the mean pressure is reached. In a normal neonate, this is approximately 50 mmHg. One way or another, a reliable blood pressure measurement must be obtained. If an aortic coarctation is suspected (high arm pressure, absent femorals, murmur), repeat the procedure in the thigh, using a blood pressure cuff of appropriately larger size. In my experience, so-called radial-femoral pulse delay is a useless sign in infants with a rapid heart rate. It is easy to say it is present when one knows in advance that the diagnosis is aortic coarctation. It is better to rely on comparison of pulse volumes and blood pressure measurements.

After listening to the infant's back and finishing the general examination, it may be necessary to make the baby cry, so the parents should be advised ahead of time. 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 any baby becomes centrally cyanosed with prolonged breath holding. If there is purplish discoloration of the mucous membranes inside the mouth while the baby cries 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 potentially lethal disorder if not diagnosed early. The pros should be called in if there are any doubts.

EXAMINING THE 3-TO 5-YEAR-OLD CHILD

By the time a child is 3 to 5 years old, lesions causing cyanosis or congestive heart failure will be revealed. The spectrum of disease in toddlers and young children includes congenital lesions that have been overlooked, such as atrial septal defect, small ventricular septal defect, bicuspid aortic valve, and acquired cardiac disorders. The latter include pericarditis, myocarditis, cardiac manifestations of hereditary muscular and neuromuscular diseases, rhythm disturbances, and other rare disorders. By far the most common problem faced by clinicians is the interpretation of heart sounds and murmurs, especially the systolic murmur.

HEART SOUNDS AND MURMURS

Heart Sounds

Conveniently numbered 1 to 4, the first and second are sounds of valve closure: the first caused by mitral and tricuspid closure, the second by aortic and pulmonary valve closure. The first sound signals the beginning of isovolemic contraction, the second the beginning of isovolemic relaxation. Blood is not moving during these periods in health. Therefore an innocent murmur will not be heard during these periods.

Memorize these two facts:

1. The left-sided valves close before the right.

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 third and fourth sounds is in question. They probably are due to the deceleration of blood at the end of early (third) and late (fourth) rapid filling phases of the ventricles. Although the exact mechanism of the third and fourth sounds is poorly understood, the third sound is usually related to high flows whereas the fourth reflects a poorly compliant ventricle. **Third sounds are normal in children with hyperdynamic circulations** and thin chest walls but are usually abnormal in

patients older than 30 years of age, when the ravages of age lower stroke volume and increase body mass. Audible fourth sounds are always abnormal. The third and fourth 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.

The terms, ***clicks* and *snaps*** are a continual source of confusion. Valve opening is quiet in health and signals the end of the period of isovolemic contraction or relaxation. When a sound is heard at the time of the opening of any heart valve, there is a problem.

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 they are heard loudest over their respective valves, except the aortic click that is usually well heard at the apex. The pulmonary ejection click is unique in that it is loudest during expiration. The only hope for identifying these sounds is a thorough working knowledge of normal and of what to expect to hear in a normal infant or child when the stethoscope is placed on a particular area of the chest. The normal and abnormal sounds for each listening area are shown in Figure 9-7.

It is essential to follow a constant systematic procedure for listening to heart sounds and murmurs in all children.

Auscultating the heart through clothing is an absolute “nono”. The examination is difficult enough to begin with.

First, listen exclusively to the individual heart sounds, knowing in advance what is normal. After this has been done, listen, equally systematically, **to the murmurs**. Here is a brief summary of normal sounds as heard with the child supine:

At the apex, there is a single first, single second, and possibly a third sound. The first and second sounds are high pitched, and the first usually will be loudest. The third sound will be heard best with the bell. **At the tricuspid area**, the first sound may be closely split and the second sound will be single. A third heart sound may be heard. In the **pulmonary area**, the first sound will usually be single. The second sound will be split in inspiration and may be closely split or single in expiration. **At the aortic area**, both the first and second sounds are single, and aortic closure is usually loudest. If sounds other than these are heard, the child may well have a cardiac problem. Remember that these are the findings with the patient supine. The intensity of the first heart sound varies with atrioventricular (AV) conduction time (the interval between onset of P wave and R waves [P-R interval]). When the P-R interval is prolonged, the valve leaflets may have almost closed when the ventricle contracts. Accordingly, the first sound will be faint or absent. This can occur in normal individuals who have a long P-R interval. Usually if the patient stands up, the P-R interval shortens and the first sound increases to fairly normal intensity. There are occasions in which there is beat-to-beat variation in intensity of the first sound. This will occur in AV dissociation, as in complete heart block, where there is beat-to-beat variation in the P-R interval, a useful sign in differentiating complete AV block from sinus bradycardia.

Gallop Rhythm

Gallop rhythm is a nasty term, because *gallop* generally is thought to signify a problem. Not so. It is better to speak of a triple rhythm and of the sound that causes the tripling. There are several types of tripling, only some of which signify a problem. If the triple rhythm is rapid, there is

a gallop cadence, which still should be described as tripling. A first sound, second sound, and prominent third sound would constitute tripling, as would a fourth sound, first sound, and second sound, or a first sound, ejection click, and second sound. The type of tripling commonly called "gallop rhythm" occurs when the third and fourth heart sounds "sum" in the presence of tachycardia, which may or may not be pathologic. In infants with a physiologically long P-R interval and tachycardia, **summation tripling can be normal**; but if there is a pathologic third sound or fourth sound and tachycardia, it would be abnormal. Because tripling can be normal or abnormal, the physician must try to identify the sound that causes the tripling.

Midsystolic Click

The sound that does not fit any of the above descriptions and that is usually best heard in the midcardiac area is the sound of mitral valve prolapse—the midsystolic click. Usually heard in midsystole, this may be single or a series of clicks. It is caused by the mitral valve or portions of it prolapsing into the left atrium. Frequently associated with a deficiency of tone in connective tissue, it occurs most often in tall asthenic individuals, more commonly females. It is best heard with the patient standing and leaning forward. This also can cause a triple rhythm; until this sound is heard two or three times, it may be confusing. Variation in intensity from moment to moment is also characteristic.

Murmurs

Heart murmurs are caused either by **turbulence in blood** or **tissue vibration**. Conventionally, they are classified by their timing as systolic (occurring between the first and second heart sounds), diastolic (between the second sound and the first sound), or continuous (present continuously through the cardiac cycle). The latter term also includes the murmur that begins in systole, passes through the second sound, and ends in diastole.

Systolic Murmurs

Systolic murmurs also are classified by their dynamic mechanism, of which there are four types:

1. Regurgitation (backward flow of blood)

2. Obstruction to forward flow

3. Vibration of tissue occurs in the normal heart when tissue is caused to vibrate by forceful contraction or, in an abnormal heart, when the presence of a substance such as calcium is caused to vibrate, even by normal blood flow

4. Excessive flow implies a volume of blood that is excessive for a normal orifice or vessel. Ask yourself which mechanism is operating whenever you hear a systolic murmur.

Regurgitant Murmurs

There is a general tendency to use the terms "regurgitation" and "insufficiency" synonymously. The term insufficiency is a poor one. For example, the valve may be insufficient in its ability to open properly, thus valve stenosis could be insufficient. Backward blood flow through a valve is regurgitation. Amen.

Blood that regurgitates does not have to wait for the aortic or pulmonary valve to open; thus, turbulence may begin during the period of isovolemic contraction, commencing with the first heart sound and continuing through systole, concluding with the second heart sound. Typically, these murmurs are pansystolic. In each of the three conditions associated with systolic regurgitation, the pressure gradient between the two chambers is high. A high-pressure gradient is associated with a high-velocity jet, which causes shedding of small vortices or eddies. Although the murmur is traditionally described as high pitched, it is in fact medium pitched, in the middle range of our hearing, (400-550 hertz), but it is relatively high pitched as most murmurs go. It **sounds like a**

breath sound and may be blowing or harsh, like tracheal breathing. Neophyte auscultators invariably mistake breath sounds as low pitched because of their soft quality. They are not.

When a murmur sounds like a breath sound, it is not an innocent murmur.

The three hemodynamic disturbances associated with regurgitant systolic murmurs are (1) ventricular septal defect, (2) mitral regurgitation and (3) tricuspid regurgitation.

These abnormalities share a common hemodynamic feature: each is associated with a high systolic pressure gradient. For example, in mitral regurgitation, left ventricular pressure is 100 mm Hg and left atrial pressure only 5 mm Hg. In small ventricular septal defects, the regurgitant murmur may be cut off in late systole as the septum contracts; therefore, the murmur begins with the first sound but ends before the second sound, and is thus **early to mid** systolic in timing. Generally, regurgitant murmurs are heard loudest over the chamber in which they originate. Thus, the murmur of **mitral regurgitation** is heard loudest at the apex and radiates toward the axilla. The murmur of **ventricular septal defect** is heard best along the left sternal border, over the right ventricular area. The murmur of tricuspid regurgitation is unique in that it increases in intensity during inspiration due to increased right ventricular filling. Again, there is just no innocent murmur that sounds like a regurgitant murmur. **If it sounds like a breath sound, harsh or blowing, of any degree of intensity, it is organic and signifies regurgitation of blood.**

Obstructive Murmurs

All obstructive murmurs are organic. The turbulence caused by obstruction has eddies of large but varying size, and vortex shedding is associated with a large amount of energy. Therefore, obstructive murmurs will be coarse and loud. Because the turbulence occurs during forward flow, it must wait for the aortic and pulmonary valve to open, and there will be a pause between the first heart sound and beginning of the murmur. The velocity and volume of blood passing through the

valve is greatest toward the center of systole, and thus the murmur will be loudest at this time, creating a crescendo- decrescendo, "diamond" or "kite-shaped" type of murmur. These loud coarse murmurs generally occur over the pulmonary or aortic valve. Unfortunately, there is the occasional exception. The murmur of aortic coarctation tends to be higher in pitch, but it is heard in a different area, being maximal high in the precordium, in the left axilla, and over the left side of the back. Occasionally, obstructions occur in the midventricle, in which case the murmur also tends to be more highly pitched and may be difficult to differentiate from a regurgitant murmur. Generally speaking, obstructive murmurs are recognized easily as being organic by their intensity and coarseness, and they tend to radiate in the direction of blood flow, where the vortex shedding process is occurring. Hence, the murmur caused by aortic stenosis is well heard over the carotid arteries.

Vibratory Murmurs

Vibratory murmurs are murmurs of musical quality. They have harmonics. The innocent vibratory murmur found in children was described first in 1909 by Still,[1] who likened it to the "twanging of string." Vibratory murmurs arise in tissue, and because tissue vibrates in harmonics, these murmurs are unlike any others. Nevertheless, the musical quality is difficult for some examiners to appreciate. A medium-pitched musical murmur would sound like a hum, whereas those with high-pitched components sound like a seagull's cry. Because vibration occurs in tissue, it often transmits in the same tissue plane. Thus, a vibratory murmur arising in the lef6t ventricular outflow tract will transmit through the left ventricular tissue toward the apex or through the aortic wall up toward the aortic listening area. The vibratory innocent systolic murmur heard commonly in children probably results from a high stroke volume being ejected forcefully, causing tissue in the left ventricle to vibrate. It is heard maximally in expiration and is usually best heard midway

between the left sternal border and the apex. Merely detecting a musical quality of the murmur in children means that the chances of the murmur being innocent are high (Table 9-2). When calcium is deposited in a heart valve, the resultant murmur is not just musical; it has high-pitched components. Occasionally in children who have a perimembranous ventricular septal defect, the murmur may have a similar high-pitched component, possibly caused by vibration of the membranous portion of the septum.

A musical murmur in a child is almost invariably innocent. **If it hums, forget it.**

Flow Murmurs

Flow murmurs are generated by the turbulence associated with an increased stroke volume. Systolic flow murmurs occur in the outflow tract of either the left or right ventricle and accordingly are usually heard maximally at the left or right sternal border in the second interspace. A flow murmur at the left sternal border probably is occurring in the pulmonary artery and is almost never loud enough to be associated with a thrill. Those heard at the right sternal border may be associated with a short coarse low-pitched sound over the carotid artery. Flow murmurs are usually associated with other evidence of a high stroke volume. **Invariably, when the patient is examined in a standing position, the systolic flow murmur greatly diminishes in intensity or totally disappears,** due to the decrease in stroke volume that occurs in the standing position.

The characteristics of the second sound become extremely important in trying to interpret the significance of flow murmurs. Unfortunately, the mechanism of an atrial septal defect murmur, which is actually a flow murmur arising in the right ventricular outflow tract, **is similar to that of the innocent functional flow murmur heard in normal individuals**, and the two murmurs may be indistinguishable on auscultation. The key distinguishing feature is a characteristic fixed splitting of the second sound that occurs with most atrial septal defects. When listening for the

second heart sound, apply the diaphragm over the second interspace at the left sternal border (with the patient supine). In the normal child, the split of the second sound widens with inspiration due to increasing right ventricular stroke volume and longer ventricular contraction. With expiration, the split narrows but may not close entirely. In the common form of atrial septal defect, blood ejected from the right ventricle is constant in volume both in inspiration and in expiration; hence, splitting of the second sound is fixed, meaning it does not change with respiratory phase. If you have difficulty hearing normal movement of the split of the second sound, sit the child up; movement of the split may be sluggish in the supine position. Other features (easily palpable right ventricular impulse, middiastolic murmur in tricuspid area) may help in the diagnosis of atrial septal defect.

A nonmusical ejection systolic murmur in the pulmonary listening area and fixed splitting of the second sound means atrial septal defect. A systolic murmur cannot be ignored until it is certain that the components of the second sound are moving normally.

Innocent murmurs are common. A murmur may be heard on careful auscultation in as many as 40 percent of 3- to 4-year-olds. Such innocent murmurs include the vibratory murmur, the flow murmur with a normal second sound, the carotid bruit, and the venous hum. It is important to know these well, because any health care system can tolerate only a limited number of cardiology consultations for innocent murmurs.

Diastolic Murmurs

All diastolic murmurs are organic, with rare exceptions, such as the mid-diastolic flow murmur that may occur with marked sinus bradycardia. Velocity of flow in diastole differs from systole; it is maximum early in diastole with the opening of the AV valves and then late in diastole with atrial contraction. These flow velocities will influence the timing of diastolic murmurs, but generally speaking, diastolic murmurs are classified similarly to systolic murmurs and may be

early, beginning with the second sound, mid, or mid-late. Yet, when a murmur is only late diastolic in timing, **we term it presystolic**. The term "pandiastolic" is never used. The mechanisms are the same, and the murmurs that are produced are therefore regurgitant, obstructive, flow, or vibratory.

Regurgitant diastolic murmurs imply either aortic or pulmonary valve regurgitation. As with systolic regurgitant murmurs, the murmur begins with the closure of that portion of the second heart sound caused by the closure of either the pulmonary or aortic valve. **The murmur of aortic regurgitation will be high pitched** because of the high-pressure gradient between the aorta and the left ventricle in diastole, and it will be heard maximally along the left sternal border, where the turbulence is occuring. **The murmur of pulmonary regurgitation with normal pulmonary artery pressure is low pitched** because of the low-pressure gradient; it is heard in the same area as the aortic regurgitation murmur. When the child has **pulmonary hypertension**, the murmur, known as the Graham-Steell murmur, is of high pitch, because of the high pressure gradient between the pulmonary artery and the right ventricle in diastole.

Obstructive murmurs are caused by mitral or tricuspid stenosis, uncommon congenital heart lesions. In areas where rheumatic fever is still endemic, this murmur may be encountered when an older child has mitral stenosis associated with **chronic rheumatic carditis**. Decrescendo-crescendo in shape related to flow velocity, the murmur will be low pitched, and it will not begin until the mitral valve opens; therefore, there will be a pause between the second sound and the start of the murmur.

With mitral valve stenosis, the murmur occurs in the left ventricle and is loudest at the apex. Frequently, only the late diastolic portion of the murmur is present, in which case it is presystolic in timing.

Students frequently time this murmur improperly, believing it to be systolic. There is just no systolic murmur maximal at the apex that is low pitched and rumbling.

A **diastolic flow murmur** occurs with lesions such as ventricular septal defect, atrial septal defect, and mitral or tricuspid regurgitation. Its presence indicates that flow volume across the AV valve is at least twice normal. Mid-diastolic in timing, of short duration, and medium pitch, it is heard maximally in either the apical or tricuspid areas, according to which valve generates the turbulence. As noted above, the same murmur is heard in the presence of marked bradycardia, and it is invariably present in complete AV block before the implantation of a pacemaker.

Occasionally, a musical diastolic murmur may be heard. One example is the "cooing", early diastolic murmur of aortic regurgitation that occurs when the regurgitant jet causes the bacterial endocarditis vegetations on the aortic valve to vibrate.

Many non-cardiologists have difficulty eliciting diastolic murmurs, due to inexperience and the relative rarity of diastolic murmurs.

Continuous Murmurs

Of the many causes of continuous murmurs, only two are of major importance. The common one is a normal finding known as the venous hum. In children whose circulation is hyperkinetic, continuous turbulence is audible over the jugular veins, usually loudest in the right supraclavicular fossa. This murmur, **usually heard only in the sitting or upright position**, varies considerably in intensity with movement of the child's head and its intensity may be influenced by light pressure on either jugular vein (Fig. 9-8). The turbulence may also be palpated with light pressure on the jugular vein (Fig. 9-9). With light finger pressure, a thrill also may be palpable. Occasionally, a venous hum is audible when the child is supine and his or her head only slightly elevated.

This murmur, as common as it is, frequently confounds the examiner, who has usually forgotten the basic rule of first listening with the patient in the supine position.

The other important continuous murmur is that of a patent ductus, heard maximally on the left side of the thorax, usually just below the clavicle, or between the left sternal border and the midclavicular line in the second interspace. In the child older than one month of age, whose pulmonary artery pressure is not elevated, the patent ductus murmur has the same continuous timing as the venous hum but peaks in intensity earlier, at the time of the second heart sound, when the pressure gradient between aorta and pulmonary artery is the greatest. In contrast to the venous hum, it is well heard in the supine position. Flow through a patent ductus of average size increases aortic runoff and left ventricular stroke volume. Accordingly, the pulse will be bounding and left ventricular activity (only the left) is readily palpable. If either of these findings is present, be sure to search particularly for a patent ductus murmur. The ductus murmur has been variously described as having a "machinery" or "train in the tunnel" quality. An inexperienced examiner hears only the loudest part of the murmur and often misses its decrescendo diastolic component.

Rarely, a continuous murmur with the characteristics of a patent ductus is heard in another location over the precordium (e.g., in a coronary AV fistula, in which it is best heard low along the left sternal border).

Other Systolic Murmurs

Two murmurs that deserve individual attention are the cardiorespiratory murmur and the murmur of mitral valve prolapse.

The cardiorespiratory murmur is missed frequently because it generally does not occur in the conventional listening stations. It tends to be loudest in the midclavicular line in the third interspace on either side of the chest, more often on the right. Occasionally, it is also heard in the

back. Characteristically, there will be three successive systolic blowing murmurs occurring in the mid and late inspiration phases. These are entirely absent during early inspiration and expiration. When heard loudest at the apex, the cardiorespiratory murmur may be confused with mitral regurgitation. The cardiorespiratory murmur has no clinical significance. It is thought to be generated in a portion of lung that is "trapped" and compressed during inspiration. If the patient is cooperative and can breath hold, the murmur, of course, disappears.

The "whoop" that occurs with mitral valve prolapse is best heard with the patient standing. It occupies the mid-late portion of systole and may be exceedingly loud, sometimes audible without a stethoscope. Whoops are usually evanescent, being loud at one time and absent at another. When a colleague says excitedly, "you just have to come and hear this," it usually turns out to be this whoop. The patient is usually tall asthenic and frequently has a thoracic bony abnormality such as pectus excavatum. No other murmur sounds like it. This clinical tidbit you may find interesting. Mitral valve prolapse may be familial due to a congenital conective tisse defect. . I recall a situation where two sisters had mitral valve prolapse, and at times either sister could have a whoop, loud enough that it could be heard by the other sister across the room. When one sister had the noise, the other sister would say "your whooping!." The mitral regurgitation that may accompany mitral valve prolapse may **not** have a whooping quality, and may have just the blowing quality of mitral regurgitation from any cause. It will be mid-late in timing however, at least in its mild form.

Pericarditis and Mediastinal Emphysema

Two other auscultatory findings of significance are the **pericardial friction rub** of pericarditis and the **mediastinal crunch** of mediastinal emphysema. Pericardial friction occurs most frequently after operation in patients undergoing cardiac surgery, or in the so-called postpericardiotomy syndrome, which characteristically occurs 3 to 6 weeks after operation. In a nonoperative situation,

it may be a sign associated with pericarditis from any cause, but usually viral. Most clinicians identify a friction rub easily because of its characteristic scratchy quality. Pericardial rubs may be heard anywhere on the left side of the chest but are usually best heard along the sternal border. Contrary to earlier teaching, their presence bears little or no relation to the amount of effusion within the pericardium. They generally have three phases, related to atrial filling, ventricular ejection, and the rapid phase of ventricular filling, giving them a characteristic "cha-cha-cha" cadence.

The mediastinal crunch, once heard, is also characteristic. It has much the same quality as pericardial friction, and although it may have a to-and-fro rhythm, it will not have a three-phase cadence. More often, its rhythm will be chaotic, at times systolic and at times phasic with respiration. It may occur independently or after chest injury. Twice, I have elicited this sign in the cardiac intensive care unit, in patients who were being ventilated. In each case, after hearing the crunch and surreptitiously palpating interstitial emphysema in the neck (which is frequently present), I turned to the intensivists and said, "Turn down your pressure, folks." Red faces caused by poor bedside skills.

Auscultation Technique for Murmurs

Having evaluated the heart sounds in each area, now begin to listen to the intervals, starting at the apex with the patient supine. Begin with the stethoscope diaphragm as most troubling murmurs will be in the medium- to high-pitched range. Listen to systole. A murmur in systole at the apex is not necessarily loudest in this area, so track the murmur with the stethoscope to its point of maximum intensity.

1. Over which chamber or vessel is the stethoscope lying?
2. Is the murmur related to the first heart sound? Listen particularly for its quality and pitch.

3. What is the intensity of the murmur (grade I to VI)?

Grade I is the faintest murmur that you can imagine. At grade IV, an associated thrill is felt, whereas a grade VI murmur is so intense it does not require a stethoscope to be heard. A rather neandethral method. but it works.

Let us suppose that the murmur has been described as pansystolic, of 3/6 intensity, high pitched with a harsh blowing quality, and heard maximally at the fifth left interspace in the anterior axillary line. This is the description of mitral regurgitation. Remember that if any apical systolic murmur is pansystolic in timing, it should be identified automatically as organic. Then listen to diastole at the apex. Listen carefully to the "nothings"—the areas initially perceived to be silent.

Then move the stethoscope to the tricuspid area and listen first to systole and then again to diastole. A murmur is heard in systole, but as it is tracked to its point of maximum intensity, it is heard loudest at the left sternal border in the second interspace. Is it regurgitant or ejection? If it is not pansystolic and if it does not sound like a breath sound, it is probably ejection. Is it caused by obstruction, Is it obstruction, flow, or vibration? If it is not low pitched and coarse, it is not obstructive. Listen for a musical component. If it is not present, it is not a vibratory murmur. Thus, by exclusion, it is a flow murmur, either innocent or due to atrial septal defect. Therefore, listen to the second heart sound again. If splitting is "fixed" the child probably has an atrial septal defect. Do not be satisfied with this. Listen to diastole. If it is an atrial septal defect, there probably is a mid-diastolic flow murmur at the left sternal border in the fourth space. If the split in the second sound moves nicely, atrial septal defect is not present. Stand the child up; if the murmur disappears, it can be concluded that the murmur is innocent. In this situation, other signs of a high output state are probably present. In this manner proceed through each listening area, listening to both systole and diastole.

Auscultation of the heart is a difficult skill to acquire. To become proficient requires many hours of listening to hearts and thus every opportunity you have as a student to listen to a heart, do so. Despite best intentions, the student still will usually not hear enough hearts to acquire this skill. You are thus encouraged to use more modern methods to gain this skill. One such method is through the use of interactive cardiac auscultation CD-ROMS (four of which are currently available), where many surrogate patients will be available for your education.

APPROACH TO PHYSICAL EXAMINATION OF THE CHILD

The order of the examination does not differ greatly from that of the infant, but the emphasis on certain aspects will change. The first challenge is persuading your young patient to cooperate. If in doubt, start with the child on the parent's lap.

If the youngster appears likely to cry, auscultate first, even if this is not the ideal way to begin a cardiovascular examination.

If the youngster is happy to lie on an examining table, stand on the child's right side. Observe his or her body habitus, and look closely for dysmorphic features. Does the child have a Marfanoid habitus? Is there pectus excavatum? Is the voice hoarse—could it be Williams' syndrome? Or does the child simply look like a normal healthy active (perhaps physiologically hyperkinetic) 3-year-old?

OBSERVATION

Observe the child's chest. Is the left side abnormally prominent? Are there abnormal pulsations? A safe way to begin the hands-on part of the examination is by gently picking up the child's hand. Are the palmar creases normal? Is there clubbing? Look at the fingertips from the side (Figs. 9-10 and 9-11). Clubbing occasionally can be normal or may occur in noncardiovascular

diseases. Are the fingers of normal length and number? Is there clinodactyly? Dorsiflex the fingers and wrist. Is tone normal?

TAKING THE PULSES

Start with the brachial pulse, not the radial. The closer to the heart the pulse is felt, the truer its quality. Using the first and second digits of your right hand palpate the brachial artery, just above the antecubital fossa (Fig. 9-12). In the older child it may be preferable to support the child's right arm with your left, using your right thumb to palpate the pulse. The important questions are

1. What is the pulse volume (pulse pressure)?

2. Is the rise normal (slow, fast, smooth)?

3. Is the fall-off normal?

4. What is the blood pressure? If the pulse volume seems to have increased, check for a waterhammer pulse by elevating the child's arm and encircling the upper arm with one hand (Fig. 9-13). A pulse of normal volume will not usually be felt with this maneuver. Now "dissect" the pulse by analyzing the upstroke and downstroke.

If the pulse volume is increased, the child has a hyperkinetic circulation, aortic insufficiency, or a patent ductus.

When the child's blood pressure is measured, the pulse pressure will be increased, and when the chest is palpated, the heart action will be increased. The pulse quality tells how blood leaves the heart and the resistance it meets in the periphery. Now palpate the femoral pulse; if it is of good volume, aortic coarctation is not present. If it is absent or is distinctly smaller in volume than the brachial, the blood pressures in the arms and legs must be carefully measured. Again, radiofemoral delay is difficult to elicit (Fig. 9-14), and if aortic regurgitation accompanies coarctation, it will not be present.

Certain clinical conditions (listed below) can be appreciated by alterations in pulse volume.

Pulsus Paradoxus

The normal systolic blood pressure may decrease as much as 8 mm Hg during average inspiration, and more during a deep inspiration. When the decrease is greater than 8 mm Hg during average inspiration, the condition is termed **pulsus paradoxus**. It is an exaggeration of a normal phenomenon and not, as the name suggests, a paradox. Its presence usually indicates that **cardiac tamponade** is present.

Check for pulsus paradoxus as follows:

1. Ask the supine child to breath normally.

2. Elevate the arm (to avoid the auscultatory gap), then inflate the cuff.

3. While observing respiration, gradually decrease the cuff pressure and note the level at which all Korotkoff sounds are heard (point A).

4. Gradually increase the cuff pressure until no Korotkoff sounds are heard (point B).

5. The difference between point A and point B represents the difference in inspiratory and expiratory systolic blood pressure. Excesses over 8 mmHg indicate the level of "paradox."

6. Unfortunately, patients with asthma or emphysema have an increased difference between inspiratory and expiratory systolic blood pressure, so be careful in interpreting this procedure in such patients.

Pulsus Alternans

This sign is seen infrequently in children and, when present, is invariably associated with myocardial failure. It is present when regular alternating pulses have a perceptible difference in volume. The palpating finger cannot perceive a systolic pressure difference of less than 20 mmHg; and careful observations must be made when recording the blood pressure in the patient with this

sign. As the cuff pressure is being decreased, a systolic pressure will be first encountered of only half the Korotkoff sounds. For example, if the blood pressure is 120 mm Hg, systolic with a regular rate of 50 beats/min, as the cuff pressure is lowered further, a regular rate of 100 beats/min will be encountered, at which time the blood pressure will be 95 mm Hg, possibly lower. The presence of left ventricular hypertension (aortic stenosis, systemic hypertension) will increase the likelihood of eliciting this sign.

Pulsus Bisferiens

Pulsus bisferiens is an ancient term describing the perceptible notch in the pulse wave detectable when a child has significant obstruction and regurgitation of the aortic valve.

Sinus Arrhythmia

The normal pulse rate varies with age and activity state of the child. The range of normal for resting pulse rates in children older than 2 years of age is listed in Table 9-3.

It is abnormal for a child ***not*** to have sinus arrhythmia.

At times, sinus arrhythmia may be so marked that it is impossible to differentiate from frequent extrasystoles or atrial fibrillation, and an electrocardiogram may be required.

Bradycardia

If a child's pulse rate is less than 60 beats/min, complete AV block may be present. Differentiation from sinus bradycardia is usually possible at the bedside. Checking the jugular pulse is normally of little value in this age group; however, in the patient with bradycardia and possible AV block, look for **cannon A-waves.** This is done with the child in the sitting or semirecumbent position, head inclined to one side. When auscultating, check for varying intensity of the first heart sound, caused by the varying position of the AV valves at the beginning of ventricular contraction. Observe the effect of exercise on the child's pulse rate. In complete AV block, only a small increase

occurs. An innocent murmur is frequently seen in association with bradycardia, due to the increased stroke volume.

PALPATION OF THE CHEST AND ABDOMEN

A 3-year-old is unlikely to have heart failure; nevertheless, try to identify the liver edge. If the liver edge is 2 cm or more below the costal margin, look for clinical evidence of pulmonary air-trapping, and percuss the top of the liver.

Gently lay a warm hand on the apical area and palpate the apical impulse. It is quick and diffuse, spilling over to the area left of the sternum in the fourth and fifth spaces. You are now palpating the left and right ventricles as they eject increased volumes of blood. If the child is quite active and pulse volume is increased, then this finding is normal. If an apical impulse is palpated that is exclusively apical and the impulse is forceful and sustained, beware. Then palpate the area to the left of the sternum in the third and fourth spaces, searching for a right ventricular impulse. A diffuse quick impulse would be expected in atrial septal defect, for example.

When palpating the thorax for ventricular dynamics, remember:

1. The ventricle that is volume overloaded is easily palpable, and the impulse will be diffuse and abrupt.

2. The pressure-loaded ventricle will be palpable only when the overload is severe (and usually chronic), and the impulse will be forceful and sustained.

Now, palpate the second left interspace at the sternal border, using your first and second digits. If an impulse is present, an organic lesion is probably present and there is pathologic dilation of the pulmonary artery, due to increased flow or pressure. Then palpate the suprasternal notch by inserting one index finger as deeply as possible. If the previous findings suggest a hyperkinetic circulation, an impulse can be normally palpated. If it is marked and visible, there is increased flow

in the aortic arch that is probably organic, and patent ductus or aortic insufficiency should be sought specifically during auscultation.

Now, palpate in the reverse direction, searching for a thrill. Begin in the suprasternal notch. A thrill may be present here even in minor degrees of obstruction in the left ventricular outflow tract. Then, palpate for sound in the conventional areas. Whatever is felt will be heard, only better. However, an appreciation of a thrill does help to classify murmurs. If a thrill is palpated, it is certain that an organic process is present and a loud murmur, grade IV to VI in intensity, will be heard.

AUSCULTATION

After palpation of the chest and abdomen, it is time to auscultate. Organize the approach. Innocent murmurs are a major problem to the physician, partly as a result of murmurs being considered in isolation.

The innocent murmur is almost always encountered in the presence of a hyperkinetic circulation, which is usually physiologic.

The hyperkinesis is not restricted to the cardiovascular system, and the child usually will be quite physically active. The details of this syndrome are listed in Table 9-4. If most of these features are not present in the child diagnosed as having an innocent murmur, question the diagnosis. Unfortunately, innocent and organic murmurs can coexist. Although all the features of a hyperkinetic circulation may be present, if the murmur sounds like a breath sound, it is abnormal. Ejection clicks are organic in any setting.

EXAMINING THE TEENAGER

Organic symptoms originating in the cardiovascular system are uncommon in teenagers who have no pre-existing cardiac disease. **Atypical chest pain** is a common complaint in teenagers. (It is usually of sharp quality, of short duration, and unrelated to exercise). When a parent or close

relative has had angina, or a recent myocardial infarct, the teenager may complain of similar distress. Providing adequate reassurance for the patient and the family usually requires no more than a good history and a physical examination; occasionally it does demand further investigation, such as electrocardiography, exercise testing, or Holter monitoring. Even in the presence of congenital abnormalities of the coronary arteries, **angina is rare**, and a more likely clinical presentation of cardiovascular disease is **syncope** which may or may not have a primary cardiovascular cause**.**

The patient that presents with a **Marfanoid habitus**, or with true Marfan syndrome and chest pain, requires close attention, as such individuals are subject to dilation of the ascending aorta and dissecting aneurysm. Patients with a Marfanoid habitus require chest radiographs and cardiac ultrasound. A family history of sudden death at an early age is also an indication for more thorough investigation, because familial aortic dissection may occur without any obvious connective tissue disorder. Consider **hypertrophic cardiomyopathy** if the history reveals this familial trait.

aortic stenosis (severe)

hypertrophic cardiomyopathy

atrial myxoma

pulmonary hypertension

long Q-T syndromes (familial or nonfamilial)

ventricular ectopic beats and normal Q-T

supraventricular tachycardias with very rapid ventricular rate,

complete AV block (congenital or acquired)

sick sinus syndrome (bradycardia-tachycardia syndrome).

A good history is imperative, because there may be no abnormality found on physical examination. The next episode may be fatal.

A "drop" indicates urgent cardiac consultation, as does a patient with cardiac dysrhythmia and "spells." Exercise and emotional outbursts are common triggering factors. While awaiting cardiac consultation, the most appropriate investigative procedure is 24-hour Holter monitor.

A presentation that is more or less limited to teenagers is something I call the "excel syndrome," observed in adolescents who possess an intense desire to excel. The typical patient is female and 14 to 15 years of age and may complain of easy fatigue, headache, atypical chest pain, or possibly inability to breathe deeply. The cardiovascular findings can be dramatic. Tachycardia and systolic hypertension, possibly as high as 170 mmHg, are present. The brachial pulse is bounding, and the heart action is hyperdynamic. A systolic ejection murmur, frequently coarse and nonmusical, is present in the pulmonary and aortic areas and may be as loud as 3/6 in intensity. A third sound and carotid bruit are frequently present. If an electrocardiogram is performed, nonspecific S-T segment and T-wave changes may be seen, further complicating the picture. Have the patient stand. Invariably, the auscultatory findings disappear. On questioning, it will often be found that academically she is first or second in her class. Ongoing observations of the blood pressure are required but only to convince the patient that no serious problem exists. The normal range of systolic and diastolic blood pressures at different ages is shown in Figure 9-16.

SUMMARY

A good clinical assessment can spare many children with cardiovascular complaints from unnecessary or inappropriate investigative procedures. The key element is a systematic approach

that always interprets each symptom and sign in terms of the underlying hemodynamic disturbance. Recognition of the characteristic manifestations peculiar to congestive heart failure in early infancy is of paramount importance. Thereafter, the first clinical issue is often to decide whether clinical findings are normal or abnormal, hemodynamically significant or otherwise. Of special importance is determining the presence or absence of central cyanosis. As in other types of pediatric physical assessment, a keen observer can learn much from hands-off examination. This chapter has reviewed several tricks of the trade for conducting a successful cardiovascular assessment without antagonizing the child. Some may believe that it is an impossible task to learn much about cardiac auscultation by reading about it; it is not. Before laying a stethoscope on anyone's precordium, the physician must have a crystal-clear concept of what to listen for as well as what each sound means. The importance of listening with the child supine cannot be over- stressed.

Of all cardiovascular problems encountered in children beyond early infancy, by far the most common is the need to assess the clinical significance of a systolic murmur, which will be present in up to 40 percent of preschool children. The overwhelming majority of such murmurs are innocent. Their clinical characteristics (e.g., vibratory, musical quality, and features of a hyperdynamic circulation) are so archetypical that investigative procedures or referral to a cardiologist should be required rarely, and reassurance of the parents should be unequivocal. As stated earlier, once the ear pieces of the stethoscope are firmly in place, it is the material between them that is the key element in accurate pediatric cardiovascular assessment.

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1–12 months Lower Limits of Normal Average Upper Limits of Normal (75–100) (50–70)

9-1 Normal Values of Pulse and Blood Pressure in First Year of Life

Age Group Pulse Rate (beats/min)

Lower Limits of Normal Average Upper Limits of Normal Blood Pressure (mmHg)

Systolic

Diastolic

Premature 80 120 170 60

(50–75) 35

(30–45)

Neonate 80 120 170 75

(60–90) 45

(40–60)

1–12 months 90 120 180 90

(75–100) 60

(50–70)

(From Moller and Neal,[2] with permission.)

9-2 Systolic Murmurs

Abbreviations: VSD, ventricular septal defect; ASD, atrial septal defect.

(Adapted from Roy3 with permission.)

1 mo–30 mo 100–180 80–160 Exercise/Fever

9-3 Acceptable Heart Rates in Infants and Children (beats/min)

Age Resting Pulse Rates

Awake

Asleep

Exercise/Fever

Newborn 100–180 80–160 <220

1 wk–3 mo 100–220 80–200 <220

3 mo–2 yr 80–150 70–120 <200

2–10 yr 70–110 60–90 <200

>10 yr 55–90 50–90 <200

(Adapted from Adams and Emmanoulides,[5] with permission.)

9-4 The Hyperkinetic Circulation

Physically active

Bounding pulses

Supine position

Hyperkinetic precordium

Pulsation in the sternal notch

Wide pulse pressure

Wide but moving split of S2

Third heart sound

Musical systolic ejection murmur, 2–3/6 intensity, left sternal border to apex, loudest in expiration

Nonmusical ejection systolic murmur, second interspace, left or right sternal border

Carotid bruit (Fig. 9-15)

Intracranial bruits

Standing position

Venous hum present

S3 disappears

Nonmusical systolic murmur disappears

Vibratory systolic murmur diminishes

9-1 Prominence of the left side of the chest in a

3-year-old with ventricular septal defect and moderate

left-to-right shunting causing enlargement of both left and right ventricles.

9-2 Press on the precordium to the left of the xiphoid process with first and second digits of right hand to elicit enlargement of the right ventricle.

9-3 The index finger of the right hand is inserted deep in the suprasternal notch, searching first for pulsation and then for a thrill.

9-4 Palpating the liver with movement of the tip of the thumb inward and cephalad, beginning low in the right lower quadrant of the abdomen.

9-5 Percussing for the liver edge, using soft percussion with second digit of right hand on second digit of the left hand, which has been positioned parallel to the liver edge.

9-6 Supinate the hand and elevate the arm before expanding the cuff. This maneuver positions the radial artery properly and eliminates the "auscultatory gap."

9-7 Normal and abnormal sounds in the four conventional auscultation areas. A, aortic; P, pulmonary; T, tricuspid; M, mitral.

9-8 The intensity of a venous hum may be enhanced by lateral positioning of the head.

9-9 Pressure on the jugular vein will influence the intensity of a venous hum, and on very light pressure a thrill may be present.

9-10 Clubbing is best seen with the digit in the lateral projection, with the earliest sign being the diminution of the angle of the nail root and the skin. (From Constant, [4] with permission.)

9-11 Early clubbing in a 7-month-old child with cyanotic congenital heart disease. The nail root-skin angle has been flattened; the tip of the finger is shiny.

9-12 The brachial pulse is used to assess the quality of the pulse, palpated with the first two digits of the right hand.

9-13 To examine for a collapsing pulse, the arm is elevated and the upper arm is encircled by the examining hand. This sign may be present in patent ductus arteriosus, in aortic regurgitation, or in hyperkinetic circulation.

9-14 In coarctation of the aorta, the initial portion (the percussion wave) of the femoral pulse may be absent, causing so-called radiofemoral delay. Search for this sign with the radial and femoral pulses in juxtaposition.

9-15 Eliciting a carotid bruit. Other findings of a hyperkinetic circulation are usually present, and a thrill in the suprasternal notch will not be present. The murmur of aortic stenosis is also well heard over the right carotid artery, but a thrill usually will be present in the suprasternal notch.

9-16 Normal blood pressure level percentiles in children older than the age of 2 years, for **(A)** boys and **(B**) girls. (From The Task Force on Blood Pressure Control in Children, [6] with permission.)