Goals & Objectives: To perform a proper assessment of a child with a murmur and list appropriate steps in the evaluation:
• Understand the components of a complete H&P in a child with a murmur.
• Describe a murmur in terms of location, quality, timing, intensity, and radiation.
• Differentiate pathologic from non-pathologic murmurs, based on the H&P.
• List appropriate initial steps in the evaluation of a child with a pathologic murmur.
• Describe circumstances in which pediatric cardiology consultation is indicated.

Pre-Meeting Preparation:
Please read the following enclosures:
• “Evaluation and Management of Heart Murmurs in Children” (AAFP, 2011)

Conference Agenda:
• Review Murmurs Quiz
• Complete Murmurs Cases
• Optional: Play “Innocent or Not That Innocent” Audio-Clip Game

Post-Conference: Board Review Q&A

Extra-Credit:
• “The Cardiac Murmur: When to Refer” (MD Consult: Pediatrics Clinic of N Am; 1998)
• “Does the Presence of a ‘New’ Murmur Predict Cardiac Pathology in Otherwise Asymptomatic Children and Adolescent” (Gupta & May, unpublished abstract, 2015)
• Modules at www.easyauscultation.com/heart-sounds

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Evaluation and Management of Heart Murmurs in Children

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Heart murmurs are common in healthy infants, children, and adolescents. Although most are not pathologic, a murmur may be the sole manifestation of serious heart disease. Historical elements that suggest pathology include family history of sudden cardiac death or congenital heart disease, in utero exposure to certain medications or alcohol, maternal diabetes mellitus, history of rheumatic fever or Kawasaki disease, and certain genetic disorders. Physical examination should focus on vital signs; age-appropriate exercise capacity; respiratory or gastrointestinal manifestations of congestive heart failure; and a thorough cardiovascular examination, including features of the murmur, assessment of peripheral perfusion, and auscultation over the heart valves. Red flags that increase the likelihood of a pathologic murmur include a holosystolic or diastolic murmur, grade 3 or higher murmur, harsh quality, an abnormal S₂, maximal murmur intensity at the upper left sternal border, a systolic click, or increased intensity when the patient stands. Electrocardiography and chest radiography rarely assist in the diagnosis. Referral to a pediatric cardiologist is recommended for patients with any other abnormal physical examination findings, a history of conditions that increase the likelihood of structural heart disease, symptoms suggesting underlying cardiac disease, or when a specific innocent murmur cannot be identified by the family physician. Echocardiography provides a definitive diagnosis and is recommended for evaluation of any potentially pathologic murmur, and for evaluation of neonatal heart murmurs because these are more likely to be manifestations of structural heart disease. (Am Fam Physician. 2011;84(7):793-800. Copyright © 2011 American Academy of Family Physicians.)
Heart Murmurs in Children

necessary to detect underlying cardiac disease in children who report exertional syncope or chest pain, or who have a family history of hypertrophic cardiomyopathy.¹,¹³,¹⁴

**Physical Examination**

The patient’s vital signs should be compared with age-established norms (available at http://www.cc.nih.gov/ccc/pedweb/pedstaff/age.html), and a focused examination of the respiratory, cardiovascular, and gastrointestinal systems should be performed⁵ (Table 3).²,⁵,⁷,¹⁰,¹³,¹⁶. Congenital anomalies of other organ systems may be associated with CHD in up to 25 percent of patients.⁶ The child’s appearance, activity level, color, and respiratory effort should be assessed, and the neck should be examined for prominent vessels, abnormal pulsations, and bruits.¹ Jugular venous distension is rare in children.⁴ The chest wall should be inspected for abnormalities of the sternum, which can be associated with CHD,¹⁵ and for abnormal cardiac impulses or thrills.¹ The lungs should be auscultated for abnormal breath sounds such as crackles, which may indicate pulmonary congestion, or wheezing, which may indicate cardiac asthma. Abdominal examination should focus on the liver location (seeking abdominal situs) and evaluation for liver

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**Table 1. Historical Findings Suggesting Structural Heart Disease in Children with Heart Murmurs**

<table>
<thead>
<tr>
<th>Historical finding</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Family history</strong></td>
<td></td>
</tr>
<tr>
<td>CHD</td>
<td>More common in children with a first-degree relative who has CHD (three- to 10-fold increased risk); high penetrance with ventricular septal defect and mitral valve prolapse</td>
</tr>
<tr>
<td>Sudden cardiac death or hypertrophic cardiomyopathy</td>
<td>Increased risk of hypertrophic cardiomyopathy (autosomal dominant pattern)</td>
</tr>
<tr>
<td>Sudden infant death syndrome</td>
<td>Can be secondary to undiagnosed CHD lesions⁸</td>
</tr>
<tr>
<td><strong>Personal history</strong></td>
<td></td>
</tr>
<tr>
<td>Conditions that may coexist with CHD:</td>
<td></td>
</tr>
<tr>
<td>Aneuploidy (e.g., trisomy 21, Turner syndrome)</td>
<td>Certain genetic disorders (e.g., DiGeorge syndrome, velo-cardio-facial syndrome) are associated with cardiac malformations</td>
</tr>
<tr>
<td>Connective tissue disorder (e.g., Marfan syndrome)</td>
<td>Trisomy 21 is associated with an increased risk of atrioventricular septal defects, atrial septal defects, ventricular septal defects, patent ductus arteriosus, and tetralogy of Fallot</td>
</tr>
<tr>
<td>Inborn error of metabolism</td>
<td>Turner syndrome is associated with increased risk of coarctation of the aorta, aortic valve stenosis, and left ventricular hypertrophy</td>
</tr>
<tr>
<td>Major congenital defects of other organ systems</td>
<td>Marfan syndrome is associated with mitral valve prolapse, aortic root dilation, and aortic insufficiency</td>
</tr>
<tr>
<td>Syndrome with dysmorphic features</td>
<td>Respiratory symptoms may be attributable to heart disease (i.e., congestive heart failure); enlarged vessels may lead to atelectasis or difficulty clearing respiratory secretions, thereby promoting infection</td>
</tr>
<tr>
<td>Frequent respiratory infections</td>
<td>Leading cause of acquired cardiac disease in children; can cause coronary artery aneurysm and stenosis⁹</td>
</tr>
<tr>
<td>Kawasaki disease</td>
<td>Associated with development of rheumatic heart disease</td>
</tr>
<tr>
<td>Rheumatic fever</td>
<td>Selective serotonin reuptake inhibitor exposure is associated with a small but statistically significant increased risk of mild heart lesions, including ventricular septal defects and bicuspid aortic valve (although not all studies found an increased risk)¹¹</td>
</tr>
<tr>
<td><strong>Prenatal or perinatal history</strong></td>
<td></td>
</tr>
<tr>
<td>In utero exposure to alcohol or other toxins</td>
<td>Fetal alcohol syndrome is associated with an increased risk of atrial and ventricular septal defects, and tetralogy of Fallot¹⁰</td>
</tr>
<tr>
<td>In utero exposure to selective serotonin reuptake inhibitors or other potentially teratogenic medications</td>
<td>Selective serotonin reuptake inhibitor exposure is associated with a small but statistically significant increased risk of mild heart lesions, including ventricular septal defects and bicuspid aortic valve (although not all studies found an increased risk)¹¹</td>
</tr>
<tr>
<td>Intrauterine infection</td>
<td>Lithium exposure is associated with Ebstein anomaly of the tricuspid valve</td>
</tr>
<tr>
<td>Maternal infections may increase risk of structural heart lesions (e.g., maternal rubella infection is associated with patent ductus arteriosus and peripheral pulmonary stenosis)</td>
<td>Valproate (Depacon) exposure is associated with coarctation of the aorta and hypoplastic left heart syndrome</td>
</tr>
<tr>
<td>Maternal diabetes mellitus</td>
<td>Increased risk of CHD, including transient hypertrophic cardiomyopathy, tetralogy of Fallot, truncus arteriosus, and double-outlet right ventricle</td>
</tr>
<tr>
<td>Preterm delivery</td>
<td>CHD is associated with other conditions (e.g., genetic disorders, in utero exposure to toxins) that can result in preterm birth; 50 percent of newborns weighing less than 3 lb, 5 oz (1,500 g) at birth have CHD (most common patent ductus arteriosus)⁷</td>
</tr>
</tbody>
</table>

CHD = congenital heart disease.

Information from references 1, 2, and 4 through 11.
enlargement or ascites, which may signal congestive heart failure.5

The peripheral pulses should be examined for rate, rhythm, volume, and character, and capillary refill time should be less than three seconds.4 The heart should be auscultated over the tricuspid, pulmonary, mitral, and aortic areas with the bell and diaphragm of the stethoscope while the patient is supine, sitting, and standing17 (Figure 1). Innocent murmurs are produced by the normal flow of blood through the heart. Changing the flow by changing the patient’s position (for example, decreasing flow to the heart with the Valsalva maneuver) will change the intensity of the murmur. Young children should be prompted to push out their abdomen against the examiner’s hand.1 The physician should listen for normal S1 and S2; a wide fixed split S2 is characteristic of an atrial septal defect.19 Gallops can be a normal finding in adolescents.1

### Table 2. Symptoms Suggesting Cardiac Disease

<table>
<thead>
<tr>
<th>Symptom or sign</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiovascular</strong></td>
<td></td>
</tr>
<tr>
<td>Chest pain</td>
<td>May be related to aortic stenosis or hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>Cyanosis</td>
<td>Structural heart lesion with restricted pulmonary blood flow</td>
</tr>
<tr>
<td>Dizziness</td>
<td>Multiple potential causes, including hypoxia and CHF</td>
</tr>
<tr>
<td>Near-syncope or syncope</td>
<td>May be related to aortic stenosis or hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>Palpitations</td>
<td>May be related to arrhythmias secondary to structural heart lesions</td>
</tr>
<tr>
<td><strong>Constitutional</strong></td>
<td></td>
</tr>
<tr>
<td>Developmental delay</td>
<td>Congenital heart lesions are more common in children with certain genetic disorders and syndromes</td>
</tr>
<tr>
<td>Diaphoresis</td>
<td>May indicate CHF or poor cardiac fitness</td>
</tr>
<tr>
<td>Easily fatigued</td>
<td>May indicate CHF, hypoxia, or poor cardiac fitness</td>
</tr>
<tr>
<td>Poor exercise tolerance or capacity for play</td>
<td>May indicate CHF, hypoxia, or poor cardiac fitness</td>
</tr>
<tr>
<td>Poor growth or failure to thrive</td>
<td>May indicate CHF, poor cardiac fitness, or a genetic disorder or syndrome; poor weight gain most commonly reflects decreased cardiac output or left-to-right shunts with pulmonary hypertension</td>
</tr>
<tr>
<td><strong>Respiratory</strong></td>
<td></td>
</tr>
<tr>
<td>Asthma-like symptoms</td>
<td>Cardiac asthma resulting from pulmonary congestion</td>
</tr>
<tr>
<td>Chronic cough</td>
<td>Atelectasis or difficulty clearing secretions because of pulmonary vascular congestion</td>
</tr>
<tr>
<td>Dyspnea on exertion</td>
<td>May indicate CHF, hypoxia, or poor cardiac fitness</td>
</tr>
</tbody>
</table>

### Table 3. Physical Examination of Children with Heart Murmurs

<table>
<thead>
<tr>
<th>Finding</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormal growth (height and weight plotted on growth chart)</td>
<td>Feeding difficulties may be a sign of cardiac disease in newborns and infants (decreased exercise capacity)</td>
</tr>
<tr>
<td>Abnormal vital signs (compared with age-adjusted norms)</td>
<td>Arrhythmia, tachycardia, hypoxia, and tachypnea may indicate underlying structural heart disease</td>
</tr>
<tr>
<td>Blood pressure discrepancy between upper and lower limbs may indicate coarctation of the aorta (pressure gradient of &gt; 20 mm Hg with low blood pressure in the lower extremities)</td>
<td></td>
</tr>
<tr>
<td>Adventitial breath sounds (e.g., wheezing, rales, ronchi, pleural rub)</td>
<td>Wheezing may be associated with cardiac asthma; rales may be associated with pulmonary congestion secondary to congestive heart failure</td>
</tr>
<tr>
<td>Chest contour signaling maldevelopment of the sternum19</td>
<td>Defective segmentation of the sternum may occur in children with CHD</td>
</tr>
<tr>
<td>Dysmorphic features</td>
<td>Certain genetic or congenital conditions increase risk of CHD</td>
</tr>
<tr>
<td><strong>Cardiovascular findings</strong></td>
<td></td>
</tr>
<tr>
<td>Abnormal S2</td>
<td>Classic finding of wide split fixed S2 with atrial septal defects; abnormal S2 may be present in other types of CHD</td>
</tr>
<tr>
<td>Capillary refill</td>
<td>Normal peripheral perfusion is less than 2 to 3 seconds; delay may indicate poor perfusion secondary to diminished cardiac output</td>
</tr>
<tr>
<td>Displaced point of maximal impulse; precordial impulses (heaves, lifts, thrills)</td>
<td>Possible structural abnormality or ventricular enlargement</td>
</tr>
<tr>
<td>Edema</td>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>Left-sided precordial bulge</td>
<td>Cardiac enlargement</td>
</tr>
<tr>
<td>S2 or S4</td>
<td>Can indicate structural heart disease; S4 can be a normal finding but usually disappears when patient is upright</td>
</tr>
<tr>
<td>Substernal heave</td>
<td>Right ventricular hypertension</td>
</tr>
<tr>
<td>Systolic ejection click</td>
<td>Semilunar valvular stenosis</td>
</tr>
<tr>
<td>Weak or absent femoral pulses</td>
<td>Coarctation of the aorta</td>
</tr>
<tr>
<td><strong>Gastrointestinal findings</strong></td>
<td></td>
</tr>
<tr>
<td>Ascites</td>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>Hepatomegaly</td>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>Location of liver signals abdominal situs</td>
<td>High rate of CHD with abdominal situs</td>
</tr>
</tbody>
</table>

CHF = congestive heart failure. Information from references 4, 7, and 10.
The heart murmur is characterized by its timing during the cardiac cycle; its location, quality, intensity, and pitch (how it sounds); and the presence or absence of clicks\(^\text{1}\) (Table 4\(^\text{5,7,17}\) and Table 5\(^\text{20-23}\)). The intensity of heart murmurs is graded from 1 to 6. Grade 1 murmurs are barely audible; grade 2 murmurs are faint but can be heard immediately; grade 3 murmurs can be heard easily and are moderately loud; grade 4 murmurs can be heard easily over a wide area but do not have a palpable thrill; grade 5 murmurs are loud and have a precordial thrill; and grade 6 murmurs are loud enough to hear with the stethoscope raised off the chest.\(^\text{17,24}\) Certain characteristics of the murmur may be considered red flags, prompting stronger consideration for structural heart disease. These include a holosystolic murmur (odds ratio [OR] of pathologic murmur = 54), grade 3 or higher (OR = 4.8), harsh quality (OR = 2.4), an abnormal S2 (OR = 4.1), maximal intensity at the upper left sternal border (OR = 4.2), a systolic click (OR = 8.3), diastolic murmur, or increased murmur intensity with standing.\(^\text{6,10,25}\) A decrease or lack

### Table 4. Characteristics of Innocent Heart Murmurs

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
<th>Age at detection</th>
<th>Can sound like</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic systolic murmur</td>
<td>Systolic ejection murmur best heard over the aortic valve</td>
<td>Older childhood into adulthood</td>
<td>—</td>
</tr>
<tr>
<td>Mammary artery soufflé*</td>
<td>High-pitched systolic murmur that can extend into diastole; best heard along the anterior chest wall over the breast</td>
<td>Rare in adolescence</td>
<td>Arteriovenous anastomoses or patent ductus arteriosus</td>
</tr>
<tr>
<td>Peripheral pulmonary stenosis</td>
<td>Grade 1 or 2, low-pitched, early- to mid-systolic ejection murmur heard over axilla or back</td>
<td>&lt; 1 year</td>
<td>Pulmonary artery stenosis or normal breath sounds</td>
</tr>
<tr>
<td>Pulmonary flow murmur</td>
<td>Grade 2 or 3, crescendo-decrescendo, early- to mid-systolic murmur peaking in mid-systole; best heard at the left sternal border between the second and third intercostal spaces; characterized by a rough, dissonant quality; loudest when patient is supine and decreases when patient is upright and holding breath</td>
<td>All</td>
<td>Atrial septal defect or pulmonary valve stenosis</td>
</tr>
<tr>
<td>Still murmur</td>
<td>Grade 1 to 3, early systolic murmur; low to medium pitch with a vibratory or musical quality; best heard at lower left sternal border; loudest when patient is supine and decreases when patient stands</td>
<td>Infancy to adolescence, often 2 to 6 years</td>
<td>Ventricular septal defect or hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>Supravalvular/brachiocephalic systolic murmur</td>
<td>Brief, low-pitched, crescendo-decrescendo murmur heard in the first two-thirds of systole; best heard above clavicles; radiates to neck; diminishes when patient hyperextends shoulders</td>
<td>Childhood to young adulthood</td>
<td>Bicuspid/stenotic aortic valve, pulmonary valve stenosis, or coarctation of the aorta</td>
</tr>
<tr>
<td>Venous hum</td>
<td>Grade 1 to 6 continuous murmur; accentuated in diastole; has a whining, roaring, or whirring quality; best heard over low anterior neck, lateral to the sternocleidomastoid; louder on right; resolves or changes when patient is supine</td>
<td>3 to 8 years</td>
<td>Cervical arteriovenous fistulas or patent ductus arteriosus</td>
</tr>
</tbody>
</table>

*Mammary artery soufflé murmur is caused by blood flow in the arteries and veins leading to and from the breasts.

Information from references 5, 7, and 17.
<table>
<thead>
<tr>
<th>Type of structural heart lesion</th>
<th>Prevalence among children with congenital heart disease (%)</th>
<th>Symptoms and clinical course</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular septal defect</td>
<td>20 to 25</td>
<td>Small defects: usually asymptomatic</td>
<td>Small defects: loud holosystolic murmur at LLSB (may not last throughout systole if defect is very small)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Medium or large defects: CHF, symptoms of bronchial obstruction, frequent respiratory infections</td>
<td>Medium and large defects: increased right-to-left ventricular impulses; thrill at LLSB; split or loud single S₂; holosystolic murmur at LLSB without radiation; grade 2 to S₂; may also hear a grade 1 or 2 mid-diastolic rumble</td>
</tr>
<tr>
<td>Atrial septal defect</td>
<td>8 to 13</td>
<td>Usually asymptomatic and incidentally found on physical examination or echocardiography; large defects can be present in infants with CHF</td>
<td>Grade 2 or 3 systolic ejection murmur best heard at ULSB; wide split fixed S₂; absent thrill; may have a grade 1 or 2 diastolic flow rumble at LLSB</td>
</tr>
<tr>
<td>Patent ductus arteriosus</td>
<td>6 to 11</td>
<td>May be asymptomatic; can cause easy fatigue, CHF, and respiratory symptoms</td>
<td>Continuous murmur (grade 1 to 5) in ULSB (crescendo in systole and decrescendo into diastole); normal S₂; S₂ may be “buried” in the murmur; thrill or hyperdynamic left ventricular impulse may be present</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>10</td>
<td>Onset depends on severity of pulmonary stenosis; cyanosis may appear in infancy (2 to 6 months of age) or in childhood; other symptoms include hypertcyanotic spells or decreased exercise tolerance</td>
<td>Central cyanosis; clubbing of nail beds; grade 3 or 4 long systolic ejection murmur heard at ULSB; may have holosystolic murmur at LLSB; systolic thrill at ULSB; normal to slightly increased S₂; single S₂</td>
</tr>
<tr>
<td>Pulmonary stenosis</td>
<td>7.5 to 9</td>
<td>Usually asymptomatic but may have symptoms secondary to pulmonary congestion</td>
<td>Systolic ejection murmur (grade 2 to 5); heard best at ULSB radiating to infraclavicular regions, axillae, and back; normal or loud S₂; variable S₂; systolic ejection click may be heard at left sternal border and may vary with respiration</td>
</tr>
<tr>
<td>Coarctation of the aorta</td>
<td>5.1 to 8.1</td>
<td>Newborns and infants may present with CHF; older children are usually asymptomatic or may have leg pain or weakness</td>
<td>Systolic ejection murmur best heard over interscapular region; normal S₁ and S₂; decreased or delayed femoral pulse; may have increased left ventricular impulse</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>5 to 6</td>
<td>Usually asymptomatic; symptoms may include dyspnea, easy fatigue, chest pain, or syncope; newborns and infants may present with CHF</td>
<td>Systolic ejection murmur (grade 2 to 5) best heard at upper right sternal border with radiation to carotid arteries; left ventricular heave; thrill at ULSB or suprasternal notch</td>
</tr>
<tr>
<td>Transposition of the great arteries</td>
<td>5</td>
<td>Variable presentation depending on type; may include cyanosis or CHF in first week of life</td>
<td>Cyanosis; clubbing of nail beds; single S₂; murmur may be absent or grade 1 or 2 nonspecific systolic ejection murmur; may have a grade 3 or 4 holosystolic murmur at LLSB and mid-diastolic murmur at apex</td>
</tr>
<tr>
<td>Total anomalous pulmonary venous connection</td>
<td>2 to 3</td>
<td>Onset of CHF at 4 to 6 weeks of age</td>
<td>Grade 2 or 3 systolic ejection murmur at ULSB; grade 1 or 2 mid-diastolic flow rumble at LLSB; wide split fixed S₂</td>
</tr>
<tr>
<td>Tricuspid atresia</td>
<td>1.4</td>
<td>Early-onset cyanosis or CHF within the first month of life</td>
<td>Cyanosis; clubbing of nail beds; normal pulses; single S₂; holosystolic murmur at LLSB or midsternal border; murmur may be absent; mid-diastolic flow murmur at apex may be present</td>
</tr>
<tr>
<td>Hypoplastic left heart syndrome</td>
<td>Rare</td>
<td>May be asymptomatic at birth, with cyanosis and CHF developing with duct closure</td>
<td>Hyperdynamic precordium; single S₂; nonspecific grade 1 or 2 systolic ejection murmur along left sternal border</td>
</tr>
<tr>
<td>Truncus arteriosus</td>
<td>Rare</td>
<td>Onset of CHF in first few weeks of life; minimal cyanosis</td>
<td>Increased cardiac impulses; holosystolic murmur (ventricular septal defect); mid-diastolic rumble</td>
</tr>
</tbody>
</table>

CHF = congestive heart failure; LLSB = lower left sternal border; ULSB = upper left sternal border.
Information from references 20 through 23.
Heart Murmurs in Children

of change in the murmur intensity with passive leg elevation (likelihood ratio [LR] = 8.0) or when the child moves from standing to squatting (LR = 4.5) increases the likelihood of hypertrophic cardiomyopathy.

Characteristics that are more likely to be associated with an innocent murmur include a systolic (rather than diastolic) murmur; soft sound; short duration; musical or low pitch; varying intensity with phases of respiration and posture (louder in supine position); and murmurs that become louder with exercise, anxiety, or fear (Table 6). The most common innocent murmur is a Still murmur, which is characteristically loudest at the lower left sternal border and has a musical or vibratory quality that is thought to represent vibrations of the left outflow tract.

Auscultation may be less accurate in younger patients, when other signs or symptoms of cardiovascular disease are present, and when findings on radiography or electrocardiography (ECG) are abnormal. Online libraries of digital heart sounds are available to familiarize physicians with the characteristics of abnormal heart sounds (Table 7).

Role of Diagnostic Testing

Chest radiography and ECG rarely assist in the diagnosis of a heart murmur. Studies in newborns and children with asymptomatic murmurs have shown that chest radiography does not influence clinical management or assist with diagnosis. A prospective study of 201 newborns who were referred to pediatric cardiologists for evaluation of a heart murmur found that the addition of ECG to clinical assessment did not improve the sensitivity or specificity of detecting structural heart lesions. In a study of 128 infants and children who were evaluated for heart murmurs, the addition of ECG and chest radiography to cardiac auscultation was more likely to mislead than assist the physician in making the correct diagnosis.

In a study of more than 900 children in a pediatric cardiology clinic who had innocent-sounding murmurs, an abnormal finding from the history, physical examination, or diagnostic tests (ECG, chest radiography, or pulse oximetry) was 67 percent sensitive but only 38 percent specific for the presence of a structural heart lesion in infants younger than six weeks, yielding positive and negative LRs very near 1.0 (i.e., no useful diagnostic information). In infants older than six weeks, sensitivity increased to 100 percent, but specificity decreased to 28 percent (positive LR = 1.6; negative LR = 0.026). Thus, this information is helpful for ruling out structural causes of an innocent-sounding murmur in infants and children older than six weeks, but it is not helpful in younger infants.

In two separate populations geographically remote from a pediatric cardiologist, phonocardiography (i.e., digital heart sound recordings reviewed by a pediatric cardiologist) had high sensitivity and specificity, and good intraobserver agreement in distinguishing between innocent murmurs and murmurs that were potentially or probably pathologic and that required echocardiography.

Indications for Referral

In children and adolescents, the diagnosis of an innocent heart murmur can be made if four criteria are met: absence of abnormal
physical examination findings (except for the murmur); a negative review of systems (i.e., child is asymptomatic); a history that is negative for features that increase the risk of structural heart disease; and characteristic auscultatory features of a specific innocent heart murmur.2,5 These criteria are not appropriate for newborns or infants younger than one year because these patients have a higher rate of asymptomatic structural heart disease.39 When an innocent murmur cannot be definitively diagnosed, the child should be referred for echocardiography, to a pediatric cardiologist, or both.

A study in Oman found that the prevalence of abnormal findings on echocardiography was not significantly different between patients referred by pediatric cardiologists and those referred by primary care physicians.37 However, pediatric cardiologists more accurately detect structural heart lesions in newborns and children with heart murmurs,32,38 and can assist family physicians in the assessment of a suspicious murmur. For both innocent and pathologic murmurs, referral to a pediatric cardiologist for confirmation or clarification of the diagnosis is associated with decreased parental anxiety.39

**Neonatal Heart Murmurs**

Newborns are at higher risk of having serious structural heart disease that presents as an asymptomatic murmur.2,5 Approximately 1 percent of newborns have a heart murmur, and 31 to 86 percent of these infants have structural heart disease,40-42 including asymptomatic newborns. Because of the higher likelihood of structural heart disease in asymptomatic newborns and young infants with heart murmurs, referral to a pediatric cardiologist and/or for echocardiography is recommended.28,43 Even potentially life-threatening heart defects may not be associated with any initial signs or symptoms other than a heart murmur.51,42

The reported sensitivity for detection of a pathologic heart murmur in newborns ranges from 80.5 to 94.9 percent among pediatric cardiologists, with specificity ranging from 25 to 92 percent.32,43 These variations are significant because the lowest specificity corresponds to positive and negative LRIs of 1.1 and 0.7, which are uninformative, and the highest specificity corresponds to positive and negative LRIs of 10 and 0.21, which are quite accurate. The ability of a pediatric cardiologist to accurately identify pathologic murmurs depends on multiple factors, including his or her confidence in the diagnosis. Echocardiography may not be required in newborns with a heart murmur if a pediatric cardiologist has diagnosed an innocent murmur with a high degree of confidence32; however, it is important to consider the relatively high prevalence of structural heart disease among asymptomatic newborns with a heart murmur.

The evaluation of newborns for CHD may include pulse oximetry after 24 hours of life. Clinical examination of asymptomatic newborns has a sensitivity of 46 percent for detection of CHD; this sensitivity increases to 77 percent when clinical examination is combined with pulse oximetry (with a cutoff of 94 percent).44

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Heart Murmurs Quiz

1. What are 6 characteristics of murmurs?
   (a) **Timing & Duration** - position in cardiac cycle
   (b) **Intensity**
      - grade 1: heard only with intense concentration
      - grade 2: faint, but heard immediately
      - grade 3: easily heard, of intermediate intensity
      - grade 4: easily heard and with a **thrill** (a palpable vibration on the chest wall)
      - grade 5: very loud, thrill present, and audible with edge of stethoscope on chest wall
      - grade 6: audible with the stethoscope off the chest wall
   (c) **Location** - with regard with PMI and radiation
   (d) **Duration**
   (e) **Configuration** - dynamic shape
   (f) **Pitch** - range of murmur, how it sounds

2. Please match the numbered locations with the murmurs auscultated at these sites.

   1. **RUSB**
      - Aortic Stenosis
      - Venus Hum
      - Aortic Systolic
   2. **LUSB**
      - Pulm Stenosis
      - Pulmonary Flow Murmur
      - ASD
      - PDA
   3. **LLSB**
      - Stills Murmur
      - VSD
      - Tricuspid Regurg
      - HCM
      - Subaortic Stenosis
   4. **Apex**
      - Mitral Valve Regurg

3. Name some key features of Innocent Murmurs:
   (a) Sensitive - changes with position or respirations
   (b) Short Duration - not holosystolic
   (c) Single - no clicks or gallops
   (d) Small - nonradiation, limited area
   (e) Soft - ≤ Grade 3
   (f) Sweet - not harsh
   (g) Systolic - limited to systole
Heart Murmur Cases

You are working in continuity clinic and will be seeing the Thomas family, including David, a 1.5 month-old for early 2mo well and Valerie, a 5 year-old for well visit.

Case 1: David
When asked about any concerns, Mrs. Thomas admits that David has recently had problems breastfeeding and she would like to see a lactation specialist. He seems to now have difficulty latching on and gets “worked up.” Even when he is given expressed breastmilk in a bottle, he seems to have problems with coordination and works so hard that he seems to sweat. Last night, it took him about 1 hour to finish 4 oz.

What additional history would you like to know?
- BHx: Prenatal complications (e.g. GDM, maternal drug use, maternal infections, etc.)
- PMHx: Any previous history of Murmur
- Growth HX: Birth weight, weight gain trend, any concern for FTT
- ROS: Tachypnea, tachycardia, edema/swelling of eyelids, scrotum, etc
- FamHx: CHD or sudden death

Mother’s prenatal history is remarkable for GDMA1. David was born full-term via SVD. “Now that you ask,” Mother reports that she was told David had a murmur on the MICC, but “it disappeared” by his 2-week check. His birth weight was at the 25th percentile. Reviewing his AHLTA growth-chart, you note that his weight has plateaued and is now at the 10th percentile. Mother remarks that he does occasionally have fast-paced breathing “when he gets worked up”. His face also “looks puffy,” but usually after doing tummy-time on his baby-blanket. Mother is unaware of anyone in the family born with heart disease or any unexplained deaths.

On exam, you appreciate a regular heart rhythm with a II/VI blowing holosystolic murmur which is heard best along the LLSB without radiation; there is normal physiological splitting of S2.

What other physical exam findings would be instructive? What additional testing (if any) would you like to perform during this visit?

Exam:
- Tachycardia, gallop, pulse characteristics (weak, thready, etc)
- Tachypnea, Wheezing, Pulmonary Crackles
- Hepatomegaly

Additional Testing: EKG, 4pt Blood pressures, Pulse-ox, CXR (N.B. CXR is not needed for every routine murmur evaluation; in this case, there is some concern for CHF)

What is your presumed diagnosis and plan?
- Concern for CHF due to VSD (medium to large defect).
- Plan for emergent referral to Peds Cardiology and probable admission for acute management.
**Case 2: Valerie**

Valerie proudly tells you that she’s here for her Girl Scout Daisy camp physical. On review of PMHx, Mother reports that “she’s my healthy one”; however, when you check your Autocites (which you haven’t figured out how to shut off), you see “**murmur**” listed. Unfortunately, AHLTA crashes before you are able to further investigate her past encounters.

You proceed with her PE. Examining Valerie in a sitting position, you appreciate a Grade II/VI continuous murmur, mildly louder in diastole with a roaring quality, best heard in the right supraclavicular area. **Is your exam done?**

No—the heart should be auscultated over the tricuspid, pulmonary, mitral, and aortic areas with the bell and diaphragm of the stethoscope while the patient is **supine, sitting, and standing**.

* Also, review VS, neck for JVD, chest wall for sternum abnormalities, lungs for abnormal breath sounds, abdomen for liver location, peripheral pulses and cap refill.

When you ask Valerie to lay down, her initial murmur is no longer audible, but you are now able to appreciate a Grade II/VI early systolic vibratory murmur best heard at the LLSB. **What do you think is going on? How do you counsel this family— Can Valerie be cleared for sports?**

- **1st murmur:** **Venous Hum murmur**- innocent murmur mostly notable when patient sitting up or standing, but resolves or changes when patient is supine. Can also be obliterated by having patient rotate head or by gently occluding the neck veins with fingers.
- **2nd murmur**- **Still murmur**- innocent murmur loudest when patient supine and less notable when patient stands. Intensity can increase during febrile illness, anemic state, after exercise, or during excitement.

Counsel Mother that these are considered “innocent murmurs” and do not require further evaluation at this time. Valerie is cleared for sports *(and selling cookies)! If murmur persists, would obtain EKG.*

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**Faculty—Have residents demonstrate how they explain “innocent murmurs” to parents.**

**Example from [www.kidshealth.org](http://www.kidshealth.org):**

The most common type of heart murmur is called functional or innocent. An innocent murmur is the sound of normal blood moving through a normal heart in a normal way. Just as we can sometimes hear the sound of air moving in an air duct or water flowing through a plumbing pipe, we can often hear the sound of blood moving through the heart even if there is not a heart problem. An innocent murmur can come and go throughout childhood. It usually goes away on its own as the child gets older and doesn't pose any health threat.
Heart Murmurs Audio-Clip Game
Faculty Answer Key

- In this exercise, residents will identify (a) whether murmurs are innocent vs. not innocent; (b) what the specific type of murmur or cardiac lesion is.
- Have residents seated with their backs turned away from the overhead computer screen, so that they do NOT see the answer key links. Be sure to turn the volume of the speakers up to 100%!
- Residents can work individually, filling out their own answer keys, or as a group, filling out a “master” answer key on the board. Faculty may review the answers one-by-one, or after all 10 clips are completed.
- Files are from www.easyauscultation.com and www.cardiologysite.com. All files require Flash Player to work, except for #4. These sites contain additional description, waveform, and anatomy for some of the murmurs. Faculty may also refer to Tables 4&5 for descriptions of each murmur.

1. Ventricular septal defect
2. Atrial septal defect (#1) Atrial septal defect (#2)
3. Venous hum
4. Still/vibratory murmur (mp3)
5. Patent ductus arteriosus
6. Aortic systolic murmur (physiologic)
7. Coarctation of the aorta
8. Aortic stenosis (mild); Aortic stenosis (severe)
9. Pulmonary flow murmur
10. Pulmonary stenosis
Heart Murmurs Board Review

1. Included in your rounds today is a 36-hour-old boy who was born at term by normal, spontaneous vaginal delivery. His respiratory rate is 80 breaths/min and heart rate is 168 beats/min. He has easily palpable, bounding pulses in all four extremities, and his blood pressure is 72/30 mm Hg. Precordial examination reveals a lift and a 3/6 systolic ejection murmur at the upper left sternal border. You also note a murmur over the anterior fontanelle.

Of the following, the MOST likely diagnosis is
A. aortic coarctation with congestive heart failure
B. aortic insufficiency
C. large ventricular septal defect with congestive heart failure
D. left-to-right extracardiac shunting with congestive heart failure
E. right-to-left extracardiac shunting with right heart failure

Systemic arteriovenous malformations constitute an extracardiac left-to-right shunt (system to venous). Blood flow always moves from high to low resistance when given the opportunity. When an arteriovenous communication occurs, as might be seen in the brain or in the liver, blood from the high-pressure, high-resistance systemic circulation can move directly into the low-pressure, low-resistance venous circulation, bypassing the capillary bed of the affected organ.

In so doing, the volume of blood entering the venous system is increased, as is the oxygen content of the blood because the tissues have not been exposed to the oxygenated blood. With the passage of time and decreasing blood viscosity as physiologic anemia of the newborn ensues, the volume of blood “shunted” through the arteriovenous malformation becomes greater. Such excess blood flow “loads” the venous pool, which is delivered to the right atrium.

As a result, the right atrium and right ventricle become dilated, and congestion may occur. If such congestion is significant, jugular venous distension and hepatomegaly may become evident on physical examination. Other signs of the right heart volume overload include a prominent precordial lift on palpation and a systolic ejection murmur or relative pulmonary stenosis as the excess blood from the right heart makes its way across the pulmonary valve. The murmur is termed “relative” because the pulmonary valve annulus does not dilate despite dilation of the right ventricle.

The excess right heart output enters the pulmonary vascular bed, often leading to some level of congestion, with the resulting sign of tachypnea on examination. Similarly, because arterial blood has a “run-off” into the low-pressure veins, there is a pronounced pulse pressure with a typically low diastolic pressure that produces bounding pulses on examination. In some patients, a continuous murmur can be heard over the area of the arteriovenous malformation, such as the fontanelle.

The newborn described in the vignette has physical findings and blood pressure that suggest a run-off lesion from the aorta, which could be significant aortic insufficiency, a large-volume ductus arteriosus, or an arteriovenous malformation. There is no diastolic murmur to suggest aortic insufficiency, and at 36 hours of age, a ductus arteriosus would not be expected to lead to symptoms. Similarly, a large ventricular septal defect might present with a holosystolic murmur and rarely leads to symptoms in the first few days after birth. Coarctation often leads to narrowed blood pressure and is associated with a pressure load on the left ventricle rather than a volume load, as in this patient. Right-to-left extracardiac shunting can occur only when pressure in the venous (right) vessel exceeds that in the arterial (left) vessel. This is a situation that does not exist.
2. You are evaluating a 2-month-old girl as part of a routine health maintenance visit. Her mother tells you that she has no trouble feeding and is gaining weight like her previous children. Her precordial examination demonstrates a mild lift. The first and second heart sounds are normal. There is a systolic click at the upper left sternal border as well as a 3/6 systolic ejection murmur at the upper left sternal border with radiation to the axillae. Diastole is clear, and her pulses are normal in all extremities.

Of the following, the MOST likely cause of this patient’s signs and symptoms is
A. aortic stenosis
B. atrial septal defect
C. patent ductus arteriosus
D. **pulmonary stenosis**
E. ventricular septal defect

The infant described in the vignette has typical findings of **pulmonary stenosis**, which often is associated with a systolic click resulting from the abnormal structure and function of the pulmonary valve. The click is caused by the opening of the thickened valve leaflets during systole. In contrast to the normal thin and flexible semilunar valve leaflets, those of the stenotic pulmonary valve have an accentuated sound that is referred to as an opening click. The murmur of pulmonary stenosis results from systolic blood flow from the right ventricle across the abnormally narrowed orifice of the pulmonary valve. The narrowing yields a diminished valve area through which the stroke volume crosses, creating turbulence. Such turbulence is noted during auscultation as a **systolic ejection murmur** and typically is heard best over the pulmonary valve and main pulmonary artery. On the chest wall, these structures lie beneath the left sterna border, with extension cephalad toward the left clavicle. Frequently, the murmur radiates into the back and the axillae as the sound of turbulence follows the course of the branch pulmonary arteries.

**Aortic stenosis** also is associated with a systolic ejection click that does not change with position, but the accompanying murmur is heard best at the upper right sternal border, with radiation into the neck. The murmur associated with an **atrial septal defect** is not from the blood flow across the atrial septum, which usually is nonturbulent and at low pressure. Rather, the systolic murmur created by an atrial septal defect is the result of a relative pulmonary stenosis as the left-to-right atrial shunt and resulting increased right ventricular volume must cross the pulmonary valve. In contrast to pulmonary valve stenosis, there is no structural abnormality of the pulmonary valve and, thus, no systolic click. **Patent ductus arteriosus** typically produces a continuous murmur characterized as having a “machinery” quality that is usually loudest at the left infraclavicular area. It is continuous because of the constant flow between the systemic and pulmonary circulation resulting from the higher systemic vascular resistance compared with the pulmonary vascular resistance throughout the cardiac cycle and the lack of a valve to separate the two circulations. The murmur of a **ventricular septal defect** typically is holosystolic because the left-to-right shunt at the ventricular level begins with the onset of systole, even before the aortic and pulmonary valves open. When the ventricular septal defect is small, it produces a high-pitched murmur, heard along the sternal border, and a normal second heart sound without a change in its normal physiologic splitting.

3. You are evaluating a 12-year-old girl as part of a sports screening program at the local school. She tells you that she has trouble keeping up with her friends during gym class and on the soccer field. On physical examination, she appears well and is in no distress. Her precordial examination demonstrates a mild lift. The first heart sound is normal, and the second heart sound is prominently split. There is a 3/6 systolic ejection murmur at the upper left sternal border. Diastole is clear, and her pulses are normal in all extremities.
Of the following, the MOST likely cause of this patient's signs and symptoms is
A. aortic stenosis
B. atrial septal defect
C. patent ductus arteriosus
D. pulmonary stenosis
E. ventricular septal defect

Recognition of cardiac anomalies in pediatric patients requires a complete history and physical examination. The timing and severity of the presentation often depends on the severity of the underlying condition, such as the size of a ventricular septal defect, the degree of semilunar valve stenosis, or the extent of obstruction to pulmonary blood flow. Many of the congenital cardiac anomalies lead to turbulent blood flow within the heart or great vessels, which produces a murmur. The loudness, timing, location, radiation, and pitch of the murmur can suggest the cause of the anomaly.

The girl described in the vignette has the typical findings of an atrial septal defect. When the left-to-right shunt at the atrial level is significant, the patient may report a history of decreased exercise tolerance when compared with peers. Such decreased tolerance likely is the result of the dilated right ventricle, which receives the normal blood flow returning to the right atrium from the systemic veins as well as the abnormal blood flow that results from the left-to-right shunt of the atrial septal defect.

The murmur in patients who have atrial septal defects is not from the blood flow across the atrial septum because this flow usually is not turbulent and at low pressure. Rather, the systolic murmur results from a relative pulmonary stenosis because the left-to-right atrial shunt and the subsequent increased right ventricular volume are required to cross the pulmonary valve. The valve annulus does not dilate and, therefore, the amount of blood (stroke volume) crossing the valve is increased. The increased flow across the valve per heart beat necessitates an increase in the velocity of that blood flow, resulting in turbulence. It is this turbulence that creates the murmur that is heard best at the upper left sternal border.

Finally, patients who have atrial septal defects often have a fixed and split second heart sound that most likely results from the relative prolonged time required for the dilated ventricle to empty its contents during systole. In contrast, in the healthy heart, the second heart sound splits variably with respiration. The lack of variation of the split most likely is due to the free communication between the two atria, which allows for equalization of the influence of respiration on both the right and left ventricle.

The murmur of pulmonary stenosis often is associated with a systolic click that results from the abnormal structure and function of the pulmonary valve itself. The normal splitting of the second heart sound occurs because the volume of right ventricular blood and its stroke volume generally are normal. Similarly, aortic stenosis is associated with an ejection click that does not change with position, and the accompanying murmur is heard best at the upper right sternal boarder, with radiation into the neck. Affected patients usually have a normal second heart sound.

The patent ductus arteriosus typically produces a continuous murmur that is characterized as having a "machinery" quality and usually is loudest at the left infraclavicular area. The murmur is continuous because the flow between the systemic and pulmonary circulation is constant due to the higher systemic compared with pulmonary vascular resistance throughout the cardiac cycle and the lack of a valve to separate the two in the structure of the ductus.

The murmur of a ventricular septal defect typically is holosystolic because the left-to-right shunt at the ventricular level begins with the onset of systole, even before the aortic and pulmonary valves open. When the ventricular septal defect is small, it produces a highpitched murmur, heard along the sternal border, and the second heart sound is normal, with no change in its normal physiologic splitting.
4. You are conducting a preparticipation evaluation of a 14-year-old girl who is trying out for her school volleyball program. She is athletic and has never had any health problems. On physical examination, her height is at the 90th percentile and weight is at the 50th percentile for her age. Her lungs are clear, and cardiac examination reveals a systolic click and an apical systolic murmur that is late systolic and graded at 2/6 with radiation to the left axilla.

Of the following, the MOST likely diagnosis is
A. anemia with high-output state
B. aortic valve stenosis
C. atrial septal defect
D. mitral valve prolapse
E. small midmuscular ventricular septal defect

Normally, the mitral valve closes very early in systole as pressure in the ventricle increases with the onset of contraction. The anterior and posterior leaflet of the valve come together to provide complete and straight apposition, thereby protecting the left atrium from the pressure and content of the left ventricle. In mitral valve prolapse (MVP), the mitral valve moves backwards (prolapses) into the left atrium during systole. The anterior or posterior leaflet does not achieve straight apposition; rather, one or both of the leaflets billows into the space of the left atrium as pressure in the left ventricle increases. If there is retrograde leakage of left ventricular blood into the left atrium, the MVP is associated with mitral regurgitation. The prolapse may result from either abnormality of one or both of the mitral valve leaflets (eg, redundancy, “floppy”, myxomatous) or of the supporting apparatus (chordae tendineae, papillary muscles). MVP may occur primarily as an intrinsic abnormality of the mitral valve or its apparatus or it can occur secondarily from acquired disease such as rheumatic heart disease, myocarditis, or cardiomyopathy. Primary MVP is more common in females than males (2:1), and the disorder may be diagnosed in any age group.

The diagnosis of MVP is based on physical findings; patients can present with auscultatory findings that include a mid-systolic click that is believed to occur from the snapping of the mitral valve in the closed position during ventricular systole similarly to a sail catching wind. This timing of the click during systole may change, depending on patient position and the relative volume of the left ventricle during the examination. Squatting fills the left ventricle and may make the click occur later; standing reduces the left ventricular volume and may move the click to an earlier portion of systole. If there is regurgitation across the mitral valve, a late systolic murmur may be audible at the cardiac apex, with radiation to the left axilla.

The patient described in the vignette has physical findings indicative of MVP. The murmur associated with anemia typically is ejection, located along the left sternal boarder with radiation to the base, and not associated with a click. Aortic stenosis is associated with an ejection click that does not change with position, and the accompanying murmur is best heard at the upper right sternal boarder with radiation into the neck. An atrial septal defect typically creates a murmur of relative pulmonary stenosis as the left-to-right atrial shunt leads to increased right ventricular volume that subsequently must cross the pulmonary valve. The murmur is heard best at the upper left sternal border. Because there is no structural abnormality of the valve, no click is appreciated in patients who have atrial septal defects. The murmur of a small muscular ventricular septal defect is high-pitched, heard along the sterna border, and not associated with a systolic click.
5. You are evaluating a 15-year-old boy who will be attending sports camp in the summer. He tells you that he is very athletic, has no trouble keeping up with his peers during physical activities, and, in fact, has less fatigue with activities than most of his friends. On physical examination, he is well-developed and comfortable. The first and second heart sounds are normal. There is a systolic click at the upper right sternal border as well as a 3/6 systolic ejection murmur at the upper right sternal border. There is a thrill in his suprasternal notch. Diastole is clear, and his pulses are normal in all extremities.

Of the following, the MOST likely cause of this patient’s signs and symptoms is
A. aortic stenosis
B. atrial septal defect
C. patent ductus arteriosus
D. pulmonary stenosis
E. ventricular septal defect

The patient described in the vignette has the typical findings of aortic stenosis, which often is associated with a systolic click that results from the abnormal structure and function of the valve. The click occurs with opening of the thickened semilunar valve leaflets during systole. In contrast to the normal thin and flexible valve leaflets, those of the stenotic aortic valve have an accentuated sound that is referred to as an opening click. The murmur of aortic stenosis results from systolic blood flow from the left ventricle across the abnormally narrowed orifice of the aortic valve. The narrowing yields a diminished valve area through which the stroke volume crosses, creating turbulence that is noted during auscultation as a systolic ejection murmur and typically is heard best over the aortic valve and ascending aorta. On the chest wall, these structures lie beneath the right sternal border, with extension up toward the right clavicle. The murmur often radiates into the neck. A thrill may be appreciated in the suprasternal notch, with the turbulent blood flow in the transverse aortic arch being palpable in some patients.

Pulmonary stenosis is associated with a systolic ejection click that does not change with position, but the accompanying murmur is heard best at the upper left sternal border, with radiation into the back and axillae. The murmur associated with an atrial septal defect is not from the blood flow across the atrial septum, which usually is not turbulent and at low pressure. Rather, the systolic murmur created by an atrial septal defect is caused by a relative pulmonary stenosis because the left-to-right atrial shunt and resulting increased right ventricular volume must cross the pulmonary valve. In contrast to pulmonary valve stenosis, there is no structural abnormality of the pulmonary valve and, thus, no systolic click. A patent ductus arteriosus typically produces a continuous murmur that is characterized as having a "machinery" quality and usually is loudest at the left infraclavicular area. It is continuous because of the constant flow between the systemic and pulmonary circulation, with the higher systemic than pulmonary vascular resistance throughout the cardiac cycle and no valve to separate the two in the structure of the ductus. The murmur of a ventricular septal defect is typically holosystolic because the left-to-right shunt at the ventricular level begins with the onset of systole, even before the aortic and pulmonary valves open. When the ventricular septal defect is small, it produces a high-pitched murmur, heard along the sternal border, and the second heart sound is normal, with no change in its normal physiologic splitting.