PR murmurs also occur early in diastole. They are usually medium pitched but may be high pitched if pulmonary hypertension is present. They are best heard at the third left intercostal space and radiate along the left sternal border. These murmurs are associated with postoperative TOF (because of surgically induced PR), pulmonary hypertension, postoperative pulmonary valvotomy or post-balloon valvuloplasty for PS, and mild isolated deformity of the pulmonary valve.

2. **Mid-diastolic murmurs** start with a loud S3 and are heard in early or mid-diastole but are not temporally midway through diastole (see Fig. 2–13). These murmurs are always low pitched and best heard with the bell of the stethoscope applied lightly to the chest. These murmurs are caused by turbulence in mitral or tricuspid flow secondary to anatomic stenosis or relative stenosis of these valves.

Mitral mid-diastolic murmurs are best heard at the apex and are often referred to as an **apical rumble**, although frequently they sound more like a hum than a rumble. These murmurs are associated with MS or a large left-to-right shunt VSD or PDA, which produces relative MS secondary to a large flow across the normal-sized mitral valve.

Tricuspid mid-diastolic murmurs are best heard along the lower left sternal border. These murmurs are associated with ASD, PAPVR, TAPVR, and ECD because they all result in relative tricuspid stenosis (TS). Anatomic stenosis of the tricuspid valve is also associated with these murmurs, but such cases are rare.

3. **Presystolic (or late diastolic) murmurs** are also caused by flow through the AV valves during ventricular diastole. They result from active atrial contraction that ejects blood into the ventricle rather than a passive pressure difference between the atrium and ventricle. These low-frequency murmurs occur late in diastole or just before the onset of systole (see Fig. 2–13) and are found with anatomic stenosis of the mitral or tricuspid valve.

**Continuous Murmurs.** Continuous murmurs begin in systole and continue without interruption through the S2 into all or part of diastole (see Fig. 2–13). Continuous murmurs are caused by the following:

1. Aortopulmonary or arteriovenous connection (e.g., PDA, arteriovenous fistula, after systemic-to-PA shunt surgery, persistent truncus arteriosus, rarely)
2. Disturbances of flow patterns in veins (e.g., venous hum)
3. Disturbance of flow pattern in arteries (e.g., COA, PA stenosis)

The murmur of PDA has a machinery-like quality, becoming louder during systole (crescendo), peaking at the S2, and diminishing in diastole (decrecendo). This murmur is maximally heard in the left infraclavicular area or along the upper left sternal border. With pulmonary hypertension, only the systolic portion can be heard, but it is crescendic during systole.

Venous hum is a common innocent murmur that is audible in the upright position, in the infraclavicular region, unilaterally or bilaterally. The murmur’s intensity also changes with the position of the neck. When the child lies supine, the murmur usually disappears. It is usually heard better on the right side.

Less common continuous murmurs of severe COA may be heard over the intercostal collaterals. The continuous murmurs of PA stenosis may be heard over the right and left anterior chest, the sides of the chest, and in the back.

The combination of a systolic murmur (e.g., VSD, AS, or PS) and a diastolic murmur (e.g., AR or PR) is referred to as a **to-and-fro murmur** to distinguish it from a machinery-like continuous murmur.

**Innocent Heart Murmurs**

Innocent heart murmurs, also called **functional murmurs**, arise from cardiovascular structures in the absence of anatomic abnormalities. Innocent heart murmurs are common in children. More than 80% of children have innocent murmurs of one type or another sometime during childhood. All innocent heart murmurs (as well as pathologic murmurs) are accentuated or brought out in a high-output state, usually during a febrile illness.
Probably the only way a physician can recognize an innocent heart murmur is to become familiar with the more common forms of these murmurs by auscultating under the supervision of pediatric cardiologists. All innocent heart murmurs are associated with normal ECG and x-ray findings. When one or more of the following are present, the murmur is more likely pathologic and requires cardiac consultation:

1. Symptoms
2. Abnormal cardiac size or silhouette or abnormal pulmonary vascularity on chest roentgenograms
3. Abnormal ECG
4. Diastolic murmur
5. A systolic murmur that is loud (i.e., grade 3/6 or with a thrill), long in duration, and transmits well to other parts of the body
6. Cyanosis
7. Abnormally strong or weak pulses
8. Abnormal heart sounds

**Classic Vibratory Murmur.** This is the most common innocent murmur in children, first described by Still in 1909. Most vibratory murmurs are detected between 3 and 6 years of age, but the same murmur may be present in neonates and infants as well as adolescents. It is maximally audible at the mid-left sternal border or over the mid-precordium (between the lower left sternal border and the apex). It is generally of low frequency and best heard with the bell of the stethoscope with the patient in the supine position. The murmur is midsystolic (i.e., not regurgitant) in timing and of grade 2 to 3/6 in intensity. This murmur is not accompanied by a thrill or ejection click. It has a distinctive quality, described as a “twanging string,” groaning, squeaking, or musical sound, giving a pleasing musical character to the murmur. The murmur is generally loudest in the supine position and often changes in character, pitch, and intensity with upright positioning. The vibratory quality may disappear and the murmur may become softer when the bell is pressed harder, thereby proving its low frequency. The intensity of the murmur increases during febrile illness or excitement, after exercise, or in anemic states. The murmur may disappear briefly at a maximum Valsalva maneuver. The ECG and chest x-ray films are normal (Table 2–9; Fig. 2–14).

<table>
<thead>
<tr>
<th>Table 2–9. <strong>Common Innocent Heart Murmurs</strong></th>
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<tbody>
<tr>
<td><strong>Type (Timing)</strong></td>
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<tr>
<td>Classic vibratory murmur (Still’s murmur) (systolic)</td>
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<tr>
<td>Pulmonary ejection murmur (systolic)</td>
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<tr>
<td>Pulmonary flow murmur of newborn (systolic)</td>
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<tr>
<td>Venous hum (continuous)</td>
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<td>Carotid bruit (systolic)</td>
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LLSB, lower left sternal border; MLSB, mid-left sternal border; ULSB, upper left sternal border.
An inexperienced examiner may confuse this murmur with the murmur of a VSD. The murmur of a VSD is usually harsh, grade 2 to 3/6 in intensity, holosystolic starting with the S1 rather than midsystolic, and often accompanied by a palpable thrill. The ECG and x-ray films are often abnormal.

The origin of the murmur remains obscure. It is believed to be generated by low-frequency vibrations of normal pulmonary leaflets at their attachments during systole or periodic vibrations of a left ventricular false tendon.

**Pulmonary Ejection Murmur (Pulmonary Flow Murmur) of Childhood.** It is common in children between 8 and 14 years of age but is most frequent in adolescents. The murmur is maximally audible at the upper left sternal border. This murmur represents an exaggeration of normal ejection vibrations within the pulmonary trunk. The murmur is exaggerated by the presence of pectus excavatum, straight back, or kyphoscoliosis. The murmur is midsystolic in timing and slightly grating (rather than vibratory) in quality, with relatively little radiation. The intensity of the murmur is usually a grade 1 to 3/6. The S2 is normal, and there is no associated thrill or ejection click (see Table 2–9; Fig. 2–14). The ECG and chest x-ray films are normal.

This murmur may be confused with the murmur of pulmonary valve stenosis or an ASD. In pulmonary valve stenosis, there may be an ejection click, systolic thrill, widely split S2, right ventricular hypertrophy (RVH) on ECG, and poststenotic dilatation of the main PA segment on chest x-ray films. Important differential points of ASD include a widely split and fixed S2, a mid-diastolic murmur of relative TS audible at the lower left sternal border if the shunt is large, RBBB or mild RVH on ECG manifested by rsR′ in V1, and chest x-ray films revealing increased pulmonary vascular markings and enlargement of the right atrium, RV, and main PA.

**Pulmonary Flow Murmur of Newborns.** This murmur is commonly present in newborns, especially those with low birth weight. The murmur usually disappears by 3 to 6 months of age. If it persists beyond this age, a structural narrowing of the pulmonary arterial tree (i.e., PA stenosis) should be suspected. It is best audible at the upper left sternal border. Although the murmur is only a grade 1 to 2/6 in intensity, it transmits impressively to the right and left chest, both axillae, and the back. There is no ejection click. The ECG and chest x-ray film are normal (see Table 2–9; Fig. 2–14).

In the fetus, the main PA trunk is large but the branches of the pulmonary artery are relatively hypoplastic because they receive a small amount of blood flow during fetal life (only 15% of combined ventricular output goes to these vessels). When the ductus closes after birth, the large dome-shaped main pulmonary artery trunk gives off two small branch pulmonary arteries. The flow through these small vessels produces turbulence with a faster flow velocity and the turbulence is transmitted along the smaller branches of the PAs. Therefore, this murmur is heard well around the chest wall. The murmur is louder in small preterm babies than the larger full-term neonates.

The murmur resembles the murmur of organic PA stenosis, which may be seen as a component of rubella syndrome, Williams’ syndrome, or Alagille’s syndrome.
Characteristic noncardiac findings in children with these syndromes lead physicians to suspect that the PA stenosis murmur has an organic cause. Organic PA stenosis is frequently associated with other cardiac defects (e.g., VSD and pulmonary valve stenosis), is at the site of a previous Blalock-Taussig shunt, or is seen occasionally as an isolated anomaly. The heart murmur of organic PA stenosis persists beyond infancy, and the ECG may show RVH if the stenosis is severe.

**Venous Hum.** This murmur is commonly audible in children between the ages of 3 and 6 years. It originates from turbulence in the jugular venous system. This is a continuous murmur in which the diastolic component is louder than the systolic component. The murmur is maximally audible at the right and/or left infraclavicular and supraclavicular areas (see Table 2–9; Fig. 2–14). The venous hum is heard only in the upright position and disappears in the supine position. It can be obliterated by rotating the head or by gently occluding the neck veins with the fingers.

It is important to differentiate a venous hum from the continuous murmur of a PDA. The murmur of a PDA is loudest at the upper left sternal border or left infraclavicular area and may be associated with bounding peripheral pulses and wide pulse pressure if the shunt is large. The systolic component is louder than the diastolic component. The x-ray films show increased pulmonary vascular markings and cardiac enlargement. The ECG may be normal (with a small shunt) or show left ventricular hypertrophy or combined ventricular hypertrophy (with a large shunt).

**Carotid Bruit (or Supraclavicular Systolic Murmur).** This is an early systolic ejection murmur, best heard in the supraclavicular fossa or over the carotid arteries (see Table 2–9; Fig. 2–14). It is produced by turbulence in the brachiocephalic or carotid arteries. The murmur is a grade 2 to 3/6 in intensity. Although it rarely occurs, a faint thrill is palpable over a carotid artery. This bruit may be found in children of any age.

The murmur of AS often transmits well to the carotid arteries with a palpable thrill, requiring differentiation from carotid bruises. In AS, the murmur is louder at the upper right sternal border, and a systolic thrill is often present in the upper right sternal border and suprasternal notch as well as over the carotid artery. An ejection click is often present in aortic valve stenosis. The ECG and chest x-ray film may appear abnormal.

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**Some Special Features of the Cardiac Examination of Neonates**

The following section briefly summarizes some unique aspects of normal and abnormal physical findings in the newborn, which are different from those in older infants and children. The difference is caused by the normal RV dominance and elevated pulmonary vascular resistance seen in the early neonatal period. Premature infants in general have less RV dominance and lower pulmonary vascular resistance than full-term neonates, adding variability to this generalization.

**NORMAL PHYSICAL FINDINGS OF NEONATES**

The following are normal cardiovascular findings in newborn infants:

1. The heart rate generally is faster in newborns than in older children and adults (the newborn rate is usually 100 beats/minute, with a normal range of 70 to 180 beats/minute).

2. A varying degree of acrocyanosis is the rule rather than the exception.

3. Mild arterial desaturation with arterial partial pressure of oxygen (P_{O_2}) as low as 60 mm Hg is not unusual in an otherwise normal neonate. This may be caused by an intrapulmonary shunt through an as yet unexpanded portion of the lungs or by a right atrium–to–left atrium shunt through a patent foramen ovale.

4. The RV is relatively hyperactive, with the point of maximal impulse at the lower left sternal border rather than at the apex.

5. The S2 may be single in the first days of life.

6. An ejection click (representing pulmonary hypertension) is occasionally heard in the first hours of life.