Cardiac diseases often cause abnormal findings on physical examination, including pathologic heart sounds and murmurs. These findings are clues to the underlying pathophysiology, and proper interpretation is essential for successful diagnosis and disease management. This chapter describes heart sounds in the context of the normal cardiac cycle and then focuses on the origins of pathologic heart sounds and murmurs.

Many cardiac diseases are mentioned briefly in this chapter as examples of abnormal heart sounds and murmurs. Because each of these conditions is described in greater detail later in the book, it is not necessary to memorize the examples presented here. Rather, it is preferable to understand the mechanisms by which the abnormal sounds are produced, so that their descriptions will make sense in later chapters.

**CARDIAC CYCLE**

**HEART SOUNDS**
- First Heart Sound (S₁)
- Second Heart Sound (S₂)
- Extra Systolic Heart Sounds
- Extra Diastolic Heart Sounds

**MURMURS**
- Systolic Murmurs
- Diastolic Murmurs
- Continuous Murmurs

**CARDIAC CYCLE**

The cardiac cycle consists of precisely timed electrical and mechanical events that are responsible for rhythmic atrial and ventricular contractions. Figure 2.1 displays the pressure relationships between the left-sided cardiac chambers during the normal cardiac cycle and serves as a platform for describing key events. Mechanical **systole** refers to ventricular contraction, and **diastole** to ventricular relaxation and filling. Throughout the cardiac cycle, the right and left atria accept blood returning to the heart from the systemic veins and from the pulmonary veins, respectively. During diastole, blood passes from the atria into the ventricles across the open tricuspid and mitral valves, causing a gradual increase in ventricular diastolic pressures. In late diastole, atrial contraction propels a final bolus of blood into
each ventricle, an action that produces a brief further rise in atrial and ventricle pressures, termed the a wave (see Fig. 2.1).

Contraction of the ventricles follows, signaling the onset of mechanical systole. As the ventricles start to contract, the pressures within them rapidly exceed atrial pressures. This results in the forced closure of the tricuspid and mitral valves, which produces the first heart sound, termed S₁. This sound has two nearly superimposed components: the mitral component slightly precedes that of the tricuspid valve because of the earlier electrical stimulation of left ventricular contraction (see Chapter 4).

As the right and left ventricular pressures rapidly rise further, they soon exceed the diastolic pressures within the pulmonary artery and aorta, forcing the pulmonic and aortic valves to open, and blood is ejected into the pulmonary and systemic circulations. The ventricular pressures continue to increase during contraction, and because the pulmonic and aortic valves are open, the aortic and pulmonary artery pressures rise, parallel to those of the corresponding ventricles.

At the conclusion of ventricular ejection, the ventricular pressures fall below those of the pulmonary artery and aorta (the pulmonary artery and aorta are elastic structures that maintain their pressures longer), such that the pulmonic and aortic valves are forced to close, producing the second heart sound, S₂. Like the first heart sound (S₁), this sound consists of two parts: the aortic (A₂) component normally precedes the pulmonic (P₂) because the diastolic pressure gradient between the aorta and left ventricle is greater than that between the pulmonary artery and right ventricle, forcing the aortic valve to shut more readily. The ventricular pressures fall rapidly during the subsequent relaxation phase. As they drop below the pressures in the right and left atria, the tricuspid and mitral valves open, followed by diastolic ventricular filling and repetition of this cycle.

Notice in Figure 2.1 that in addition to the a wave, the atrial pressure curve displays two other positive deflections during the cardiac cycle: The c wave represents a small rise in atrial pressure as the tricuspid and mitral valves close and bulge toward the atrium, and the v wave is the result of passive filling of the LA from the pulmonary veins during systole, when the MV is closed.

Figure 2.1. The normal cardiac cycle, showing pressure relationships between the left-sided heart chambers. During diastole, the mitral valve (MV) is open, so that the left atrial (LA) and left ventricular (LV) pressures are equal. In late diastole, LA contraction causes a small rise in pressure in both the LA and LV (the a wave). During systolic contraction, the LV pressure rises; when it exceeds the LA pressure, the MV closes, contributing to the first heart sound (S₁). As LV pressure rises above the aortic pressure, the aortic valve (AV) opens, which is a silent event. As the ventricle begins to relax and its pressure falls below that of the aorta, the AV closes, contributing to the second heart sound (S₂). As LV pressure falls further, below that of the LA, the MV opens, which is silent in the normal heart. In addition to the a wave, the LA pressure curve displays two positive deflections: the c wave represents a small rise in LA pressure as the MV closes and bulges toward the atrium, and the v wave is the result of passive filling of the LA from the pulmonary veins during systole, when the MV is closed.
ration of systole remains constant from beat to beat, the length of diastole varies with the heart rate: the faster the heart rate, the shorter the diastolic phase. The main sounds, $S_1$ and $S_2$, provide a framework from which all other heart sounds and murmurs can be timed.

The pressure relationships and events depicted in Figure 2.1 are those that occur in the left side of the heart. Equivalent events occur simultaneously in the right side of the heart in the right atrium, right ventricle, and pulmonary artery. At the bedside, clues to right-heart function can be ascertained by examining the jugular venous pulse, which is representative of the right atrial pressure (see Box 2.1).

**Box 2.1 Jugular Venous Pulsations and Assessment of Right-Heart Function**

Bedside observation of jugular venous pulsations in the neck is a vital part of the cardiovascular examination. With no structures impeding blood flow between the internal jugular (IJ) veins and the superior vena cava and right atrium (RA), the height of the IJ venous column (termed the jugular venous pressure, or JVP) is an accurate representation of the RA pressure. Thus, the JVP provides an easily obtainable measure of right-heart function.

Typical fluctuations in the jugular venous pulse during the cardiac cycle, manifested by oscillations in the overlying skin, are shown in the figure (notice the similarity to the left atrial pressure tracing in Fig. 2.1). There are two major upward components, the $a$ and $v$ waves, followed by two descents, termed $x$ and $y$.

The $x$ descent, which represents the pressure decline following the $a$ wave, may be interrupted by a small upward deflection (the $c$ wave, denoted in the figure by the arrow) at the time of tricuspid valve closure, but that is usually not distinguishable in the JVP. The $a$ wave represents transient venous distension caused by back pressure from RA contraction. The $v$ wave corresponds to passive filling of the RA from the systemic veins during systole, when the tricuspid valve is closed. Opening of the tricuspid valve in early diastole allows blood to rapidly empty from the RA into the right ventricle; that fall in RA pressure corresponds to the $y$ descent.

Conditions that abnormally raise right-sided cardiac pressures (e.g., heart failure, tricuspid valve disease, pulmonic stenosis, pericardial diseases) elevate the JVP, while reduced intravascular volume (e.g., dehydration) decreases it. In addition, specific disease states can influence the individual components of the JVP, examples of which are listed here for reference and explained in subsequent chapters:

- **Prominent $a$**: right ventricular hypertrophy, tricuspid stenosis
- **Prominent $v$**: tricuspid regurgitation
- **Prominent $y$**: constrictive pericarditis

**Technique of Measurement**

The JVP is measured as the maximum vertical height of the internal jugular vein (in cm) above the center of the right atrium, and in a normal person is $\leq 9$ cm. Because the sternal angle is located approximately 5 cm above the center of the RA, the JVP is calculated...
at the bedside by adding 5 cm to the vertical height of the top of the IJ venous column above the sternal angle.

The right IJ vein is usually the easiest to evaluate because it extends directly upward from the RA and superior vena cava. First, observe the pulsations in the skin overlying the IJ with the patient supine and the head of the bed at about a 45° angle. Shining a light obliquely across the neck helps to visualize the pulsations. Be sure to examine the IJ, not the external jugular vein. The former is medial to, or behind, the sternocleidomastoid muscle, while the external jugular is usually more lateral. Although the external jugular is typically easier to see, it does not accurately reflect RA pressure because it contains valves that interfere with venous return to the heart.

If the top of the IJ column is not visible at 45°, the column of blood is either too low (below the clavicle) or too high (above the jaw) to be measured in that position. In such situations, the head of the bed must be lowered or raised, respectively, so that the top of the column becomes visible. As long as the top can be ascertained, the vertical height of the JVP above the sternal angle will accurately reflect RA pressure, no matter the angle of the head of the bed.

Sometimes it can be difficult to distinguish the jugular venous pulsations from the neighboring carotid artery. Unlike the carotid, the JVP is usually not palpable, it has a double rather than a single upstroke, and it declines in most patients by assuming the seated position or during inspiration.

HEART SOUNDS

Typical stethoscopes contain two chest pieces for auscultation of the heart. The concave “bell” chest piece, meant to be applied lightly to the skin, accentuates low-frequency sounds. Conversely, the flat “diaphragm” chest piece should be pressed firmly against the skin to eliminate low frequencies and therefore accentuate high-frequency sounds and murmurs. Some modern stethoscopes incorporate both the bell and diaphragm functions into a single chest piece; in these models, placing the piece lightly on the skin brings out the low-frequency sounds, while firm pressure accentuates the high-frequency ones.

First Heart Sound (S₁)

S₁ is produced by closure of the mitral and tricuspid valves in early systole and is loudest near the apex of the heart (Fig. 2.2). It is a high-frequency sound, best heard with the diaphragm of the stethoscope. Although mitral closure usually precedes tricuspid closure, they are separated by only about 0.01 sec, such that the human ear appreciates only a single sound. An exception occurs in patients with right bundle branch block (see Chapter 4), in whom these components may be audibly split because of delayed closure of the tricuspid valve.

Three factors determine the intensity of S₁: (1) the distance separating the leaflets of the open valves at the onset of ventricular contraction; (2) the mobility of the leaflets (normal, or rigid because of stenosis); and (3) the rate of rise of ventricular pressure (Table 2.1).
The Cardiac Cycle: Mechanisms of Heart Sounds and Murmurs

The distance between the open valve leaflets at the onset of ventricular contraction relates to the electrocardiographic PR interval (see Chapter 4), the period between the onset of atrial and ventricular activation. Atrial contraction at the end of diastole forces the tricuspid and mitral valve leaflets apart. As they start to drift back together, ventricular contraction forces them shut, from whatever position they are at, as soon as the ventricular pressure exceeds that in the atrium. An accentuated S₁ results when the PR interval is shorter than normal because the valve leaflets do not have sufficient time to drift back together and are therefore forced shut from a relatively wide distance.

Similarly, in mild mitral stenosis (see Chapter 8) a prolonged diastolic pressure gradient exists between the left atrium and ventricle, which keeps the mobile portions of the mitral leaflets farther apart than normal during diastole. Because the leaflets are relatively wide apart at the onset of systole, they are forced shut loudly when the left ventricle contracts.

S₁ also may be accentuated when the heart rate is more rapid than normal (i.e., tachycardia) because diastole is shortened and the leaflets have insufficient time to drift back together before the ventricles contract.

Conditions that reduce the intensity of S₁ are also listed in Table 2.1. In first-degree atrioventricular (AV) block (see Chapter 12), a diminished S₁ results from an abnormally prolonged PR interval, which delays the onset of ventricular contraction. Following atrial contraction, the mitral and tricuspid valves have additional time to float back together so that the leaflets are forced closed from only a small distance apart.

In patients with mitral regurgitation (see Chapter 8), S₁ is often diminished in intensity because the mitral leaflets may not come into full contact with one another as they close. In severe mitral stenosis, the leaflets are nearly fixed in position throughout the cardiac cycle, and that reduced movement lessens the intensity of S₁.

In patients with a “stiffened” left ventricle (e.g., a hypertrophied chamber), atrial contraction results in a higher-than-normal pressure at the end of diastole. This greater pressure causes the mitral leaflets to drift together more rapidly, forcing them closed from a smaller-than-normal distance when ventricular contraction begins and thus reducing the intensity of S₁.

**Second Heart Sound (S₂)**

The second heart sound results from the closure of the aortic and pulmonic valves and therefore has aortic (A₂) and pulmonic (P₂) components. Unlike S₁, which is usually heard as a single sound, the components of S₂ vary with the respiratory cycle: they are normally fused as one sound during expiration but become audibly separated during inspiration, a situation termed normal or physiologic splitting (Fig. 2.3).

One explanation for normal splitting of S₂ is as follows. Expansion of the chest during inspiration causes the intrathoracic pressure to become more negative. The negative pressure transiently increases the capacitance (and reduces the impedance) of the intrathoracic pulmonary vessels. As a result, there is a temporary delay in the diastolic “back pressure” of the pulmonary artery responsible for closure of the pulmonic valve. Thus, P₂ is delayed; that is, it occurs later during inspiration than during expiration.

Inspiration has the opposite effect on A₂. Because the capacity of the intrathoracic

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**TABLE 2.1. Causes of Altered Intensity of First Heart Sound (S₁)**

<table>
<thead>
<tr>
<th>Accentuated S₁</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Shortened PR interval</td>
<td></td>
</tr>
<tr>
<td>2. Mild mitral stenosis</td>
<td></td>
</tr>
<tr>
<td>3. High cardiac output states or tachycardia (e.g., exercise or anemia)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Diminished S₁</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Lengthened PR interval: first-degree AV nodal block</td>
<td></td>
</tr>
<tr>
<td>2. Mitral regurgitation</td>
<td></td>
</tr>
<tr>
<td>3. Severe mitral stenosis</td>
<td></td>
</tr>
<tr>
<td>4. “Stiff” left ventricle (e.g., systemic hypertension)</td>
<td></td>
</tr>
</tbody>
</table>

AV, atrioventricular.
Chapter Two

Figure 2.3. Splitting patterns of the second heart sound ($S_2$). $A_2$, aortic component; $P_2$, pulmonic component of $S_2$; $S_1$, first heart sound.

A. Physiologic (normal) splitting

Expiration

In expiration, $A_2$ and $P_2$ fuse as one sound

Inspiration

B. Widened splitting

Expiration

- Right bundle branch block
- Pulmonic stenosis

Inspiration

C. Fixed splitting

Expiration

- Atrial septal defect

Inspiration

D. Paradoxical splitting

Expiration

- Left bundle branch block
- Advanced aortic stenosis

Inspiration

(Note reversed position of $A_2$ and $P_2$)
pulmonary veins is increased by the negative pressure generated by inspiration, the venous return to the left atrium and ventricle temporarily decreases. Reduced filling of the LV causes a reduced stroke volume during the next systolic contraction and therefore shortens the time required for LV emptying. Therefore, aortic valve closure ($A_2$) occurs slightly earlier in inspiration than during expiration. The combination of an earlier $A_2$ and delayed $P_2$ during inspiration causes audible separation of the two components. Since these components are high-frequency sounds, they are best heard with the diaphragm of the stethoscope, and splitting of $S_2$ is usually most easily appreciated near the second left intercostal space next to the sternum (the pulmonic area).

Abnormalities of $S_2$ include alterations in its intensity and changes in the pattern of splitting. The intensity of $S_2$ depends on the velocity of blood coursing back toward the valves from the aorta and pulmonary artery after the completion of ventricular contraction, and the suddenness with which that motion is arrested by the closing valves. In systemic hypertension or pulmonary arterial hypertension, the diastolic pressure in the respective great artery is higher than normal, such that the velocity of the blood surging toward the valve is elevated and $S_2$ is accentuated. Conversely, in severe aortic or pulmonic valve stenosis, the valve commissures are nearly fixed in position, such that the contribution of the stenotic valve to $S_2$ is diminished.

**Widened splitting** of $S_2$ refers to an increase in the time interval between $A_2$ and $P_2$, such that the two components are audibly separated even during expiration and become more widely separated in inspiration (see Fig. 2.3). This pattern is usually the result of delayed closure of the pulmonic valve, which occurs in right bundle branch block and pulmonic valve stenosis.

**Fixed splitting** of $S_2$ is an abnormally widened interval between $A_2$ and $P_2$ that persists unchanged through the respiratory cycle (see Fig. 2.3). The most common abnormality that causes fixed splitting of $S_2$ is an atrial septal defect (see Chapter 16). In that condition, chronic volume overload of the right-sided circulation results in a high-capacitance, low-resistance pulmonary vascular system. This alteration in pulmonary artery hemodynamics delays the back pressure responsible for closure of the pulmonic valve. Thus, $P_2$ occurs later than normal, even during expiration, such that there is wider than normal separation of $A_2$ and $P_2$. The pattern of splitting does not change (i.e., it is fixed) during the respiratory cycle because (1) inspiration does not substantially increase further the already elevated pulmonary vascular capacitance, and (2) augmented filling of the right atrium from the systemic veins during inspiration is counterbalanced by a reciprocal decrease in the left-to-right transatrial shunt, eliminating respiratory variations in right ventricular filling.

**Paradoxical splitting** (or reversed splitting) refers to audible separation of $A_2$ and $P_2$ during expiration that disappears on inspiration, the opposite of the normal situation. It reflects an abnormal delay in the closure of the aortic valve such that $P_2$ precedes $A_2$. In adults, the most common cause is left bundle branch block (LBBB). In LBBB, the spread of electrical activity through the left ventricle is impaired, resulting in delayed ventricular contraction and late closure of the aortic valve such that it follows $P_2$. During inspiration, as in the normal case, the pulmonic valve closure sound is delayed and the aortic valve closure sound moves earlier. This results in narrowing and often superimposition of the two sounds; thus, there is no apparent split at the height of inspiration (see Fig. 2.3). In addition to LBBB, paradoxical splitting may be observed under circumstances in which left ventricular ejection is greatly prolonged, such as aortic stenosis.

**Extra Systolic Heart Sounds**

Extra systolic heart sounds may occur in early, mid-, or late systole.

**Early Extra Systolic Heart Sounds**

Abnormal early systolic sounds, or ejection clicks, occur shortly after $S_1$ and coincide with
the opening of the aortic or pulmonic valves (Fig. 2.4). These sounds have a sharp, high-pitched quality, so they are heard best with the diaphragm of the stethoscope placed over the aortic and pulmonic areas. Ejection clicks indicate the presence of aortic or pulmonic valve stenosis or dilatation of the pulmonary artery or aorta. In stenosis of the aortic or pulmonic valve, the sound occurs as the valve leaflets reach their maximal level of ascent into the great artery, just prior to blood ejection. At that moment, the rapidly ascending valve reaches its elastic limit and decelerates abruptly, an action thought to result in the sound generation. In dilatation of the root of the aorta or pulmonary artery, the sound is associated with sudden tensing of the aortic or pulmonic root with the onset of blood flow into the vessel. The aortic ejection click is heard at both the base and the apex of the heart and does not vary with respiration. In contrast, the pulmonic ejection click is heard only at the base and its intensity diminishes during inspiration (see Chapter 16).

Mid- or Late Extra Systolic Heart Sounds
Clicks occurring in mid- or late systole are usually the result of systolic prolapse of the mitral or tricuspid valves, in which the leaflets bulge abnormally from the ventricular side of the atrioventricular junction into the atrium during ventricular contraction, often accompanied by valvular regurgitation. They are loudest over the mitral or tricuspid auscultatory regions, respectively.

Extra Diastolic Heart Sounds
Extra heart sounds in diastole include the opening snap (OS), the third heart sound (S₃), the fourth heart sound (S₄), and the pericardial knock.

Opening Snap
Opening of the mitral and tricuspid valves is normally silent, but mitral or tricuspid valvular stenosis (usually the result of rheumatic heart disease; see Chapter 8) produces a sound, termed a snap, when the affected valve opens. It is a sharp, high-pitched sound, and its timing does not vary significantly with respiration. In mitral stenosis (which is much more common than tricuspid valve stenosis), the OS is heard best between the apex and the left sternal border, just after the aortic closure sound (A₂), when the left ventricular pressure falls below that of the left atrium (see Fig. 2.4).

Because of its proximity to A₂, the A₂–OS sequence can be confused with a widely split second heart sound. However, careful auscultation at the pulmonic area during inspiration reveals three sounds occurring in rapid succession (Fig. 2.5), which correspond to aortic closure (A₂), pulmonic clo-
The Cardiac Cycle: Mechanisms of Heart Sounds and Murmurs

mitral stenosis, the opening snap is widely separated from \( A_2 \), whereas in more severe stenosis, the \( A_2 \)-OS interval is narrower.

Third Heart Sound (\( S_3 \))

When present, an \( S_3 \) occurs in early diastole, following the opening of the atrioventricular valves, during the ventricular rapid filling phase (see Fig. 2.4). It is a dull, low-pitched sound best heard with the bell of the stethoscope. A left-sided \( S_3 \) is typically loudest over the cardiac apex while the patient lies in the left lateral decubitus position. A right-sided \( S_3 \) is better appreciated at the lower-left sternal border. Production of the \( S_3 \) appears to result from tensing of the chordae tendineae during rapid filling and expansion of the ventricle.

A third heart sound is a normal finding in children and young adults. In these groups, an \( S_3 \) implies the presence of a supple ventricle capable of normal rapid expansion in early diastole. Conversely, when heard in middle-aged or older adults, an \( S_3 \) is often a sign of disease, indicating volume overload owing to congestive heart failure, or the increased transvalvular flow that accompanies advanced mitral or tricuspid regurgitation. A pathologic \( S_3 \) is sometimes referred to as a ventricular gallop.

Fourth Heart Sound (\( S_4 \))

When an \( S_4 \) is present, it occurs in late diastole and coincides with contraction of the atria (see Fig. 2.4). This sound is generated by the left (or right) atrium vigorously contracting against a stiffened ventricle. Thus, an \( S_4 \) usually indicates the presence of cardiac disease—specifically, a decrease in ventricular compliance typically resulting from ventricular hypertrophy or myocardial ischemia. Like an \( S_3 \), the \( S_4 \) is a dull, low-pitched sound and is best heard with the bell of the stethoscope. In the case of the more common left-sided \( S_4 \), the sound is loudest at the apex, with the patient lying in the left lateral decubitus position. \( S_4 \) is sometimes referred to as an atrial gallop.
Quadruple Rhythm or Summation Gallop

In a patient with both an S\textsubscript{3} and S\textsubscript{4}, those sounds, in conjunction with S\textsubscript{1} and S\textsubscript{2}, produce a quadruple beat. If a patient with a quadruple rhythm develops tachycardia, diastole becomes shorter in duration, the S\textsubscript{3} and S\textsubscript{4} coalesce, and a summation gallop results. The summation of S\textsubscript{3} and S\textsubscript{4} is heard as a long middiastolic, low-pitched sound, often louder than S\textsubscript{1} and S\textsubscript{2}.

Pericardial Knock

A pericardial knock is an uncommon, high-pitched sound that occurs in patients with severe constrictive pericarditis (see Chapter 14). It appears early in diastole soon after S\textsubscript{2} and can be confused with an opening snap or an S\textsubscript{3}. However, the knock appears slightly later in diastole than the timing of an opening snap and is louder and occurs earlier than the ventricular gallop. It results from the abrupt cessation of ventricular filling in early diastole, which is the hallmark of constrictive pericarditis.

MURMURS

A murmur is the sound generated by turbulent blood flow. Under normal conditions, the movement of blood through the vascular bed is laminar, smooth and silent. However, as a result of hemodynamic and/or structural changes, laminar flow can become disturbed and produce an audible noise. Murmurs result from any of the following mechanisms:

1. Flow across a partial obstruction (e.g., aortic stenosis)
2. Increased flow through normal structures (e.g., aortic systolic murmur associated with a high-output state, such as anemia)
3. Ejection into a dilated chamber (e.g., aortic systolic murmur associated with aneurysmal dilatation of the aorta)
4. Regurgitant flow across an incompetent valve (e.g., mitral regurgitation)
5. Abnormal shunting of blood from one vascular chamber to a lower-pressure chamber (e.g., ventricular septal defect)

Murmurs are described by their timing, intensity, pitch, shape, location, radiation, and response to maneuvers. **Timing** refers to whether the murmur occurs during systole or diastole or is continuous (i.e., begins in systole and continues into diastole). The **intensity** of the murmur is typically quantified by a grading system. In the case of **systolic murmurs**:

<table>
<thead>
<tr>
<th>Grade 1/6 (or I/VI):</th>
<th>Barely audible (i.e., medical students may not hear it!)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 2/6 (or II/VI):</td>
<td>Faint but immediately audible</td>
</tr>
<tr>
<td>Grade 3/6 (or III/VI):</td>
<td>Easily heard</td>
</tr>
<tr>
<td>Grade 4/6 (or IV/VI):</td>
<td>Easily heard and associated with a palpable thrill</td>
</tr>
<tr>
<td>Grade 5/6 (or V/VI):</td>
<td>Very loud; heard with stethoscope lightly on chest</td>
</tr>
<tr>
<td>Grade 6/6 (or VI/VI):</td>
<td>Audible without the stethoscope directly on the chest wall</td>
</tr>
</tbody>
</table>

And in the case of **diastolic murmurs**:

<table>
<thead>
<tr>
<th>Grade 1/4 (or I/IV):</th>
<th>Barely audible</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 2/4 (or II/IV):</td>
<td>Faint but immediately audible</td>
</tr>
<tr>
<td>Grade 3/4 (or III/IV):</td>
<td>Easily heard</td>
</tr>
<tr>
<td>Grade 4/4 (or IV/IV):</td>
<td>Very loud</td>
</tr>
</tbody>
</table>

**Pitch** refers to the frequency of the murmur, ranging from high to low. High-frequency murmurs are caused by large pressure gradients between chambers (e.g., aortic stenosis) and are best appreciated using the diaphragm chest piece of the stethoscope. Low-frequency murmurs imply less of a pressure gradient between chambers (e.g., mitral stenosis) and are best heard using the stethoscope’s bell piece.

**Shape** describes how the murmur changes in intensity from its onset to its completion. For example, a crescendo–decrescendo
The Cardiac Cycle: Mechanisms of Heart Sounds and Murmurs

(or “diamond-shaped”) murmur first rises and then falls off in intensity. Other shapes include decrescendo (i.e., the murmur begins at its maximum intensity and grows softer) and uniform (the intensity of the murmur does not change).

Location refers to the murmur’s region of maximum intensity and is usually described in terms of specific auscultatory areas (see Fig. 2.2):

| Aortic area: | Second to third right intercostal spaces, next to sternum |
| Pulmonic area: | Second to third left intercostal spaces, next to sternum |
| Tricuspid area: | Lower-left sternal border |
| Mitral area: | Cardiac apex |

From their primary locations, murmurs are often heard to radiate to other areas of the chest, and such patterns of transmission relate to the direction of the turbulent flow. Finally, similar types of murmurs can be distinguished from one another by simple bedside maneuvers, such as standing up-right, Valsalva (forceful expiration against a closed airway), or clenching of the fists, each of which alters the heart’s loading conditions and can affect the intensity of many murmurs. Examples of the effects of maneuvers on specific murmurs are presented in Chapter 8.

When reporting a murmur, some or all of these descriptors are mentioned. For example, you might describe a particular patient’s murmur of aortic stenosis as “A grade III/VI high-pitched, crescendo–decrescendo systolic murmur, heard best at the upper-right sternal border, radiating toward the neck.”

**Systolic Murmurs**

Systolic murmurs are subdivided into systolic ejection murmurs, pansystolic murmurs, and late systolic murmurs (Fig. 2.6). A systolic ejection murmur is typical of aortic or pulmonic valve stenosis. It begins after the first heart sound and terminates before or during $S_2$, depending on its severity and whether the obstruction is of the aortic or pulmonic valve. The shape of the murmur is of the crescendo–decrescendo type (i.e., its intensity rises and then falls).

**Examples**

- **A. Ejection type**
  - $S_1$
  - $S_2$
  - Aortic stenosis
  - Pulmonic stenosis

- **B. Pansystolic (holosystolic)**
  - $S_1$
  - $S_2$
  - Mitral regurgitation
  - Tricuspid regurgitation
  - Ventricular septal defect

- **C. Late systolic**
  - $S_1$
  - $S_2$
  - Mitral valve prolapse

**Figure 2.6. Classification of systolic murmurs.** Ejection murmurs are crescendo–decrescendo in configuration, whereas pansystolic murmurs are uniform throughout systole. A late systolic murmur often follows a midsystolic click and suggests mitral (or tricuspid) valve prolapse.
The ejection murmur of aortic stenosis begins in systole after \( S_1 \), from which it is separated by a short audible gap (Fig. 2.7). This gap corresponds to the period of isovolumetric contraction of the left ventricle (the period after the mitral valve has closed but before the aortic valve has opened). The murmur becomes more intense as flow increases across the aortic valve during the rise in left ventricular pressure (crescendo). Then, as the ventricle relaxes, forward flow decreases, and the murmur lessens in intensity (decrescendo) and finally ends prior to the aortic component of \( S_2 \). The murmur may be immediately preceded by an ejection click, especially in mild forms of aortic stenosis.

Although the intensity of the murmur does not correlate well with the severity of aortic stenosis, other features do. For example, the more severe the stenosis, the longer it takes to force blood across the valve, and the later the murmur peaks in systole (Fig. 2.8). Also, as shown in Figure 2.8, as the severity of stenosis increases, the aortic component of \( S_2 \) softens because the leaflets become more rigidly fixed in place.

Aortic stenosis causes a high-frequency murmur, reflecting the sizable pressure gradient across the valve. It is best heard in the “aortic area” at the second and third right intercostal spaces close to the sternum. The murmur typically radiates toward the neck (the direction of turbulent blood flow) but...
often can be heard in a wide distribution, including the cardiac apex.

The murmur of pulmonic stenosis also begins after \( S_2 \), it may be preceded by an ejection click, but unlike aortic stenosis, it may extend beyond \( A_2 \). That is, if the stenosis is severe, it will result in a very prolonged right ventricular ejection time, elongating the murmur, which will continue beyond \( A_2 \) and end just before closure of the pulmonic valve (\( P_2 \)). Pulmonic stenosis is usually loudest at the second to third intercostal spaces close to the sternum. It does not radiate as widely as aortic stenosis, but sometimes it is transmitted to the neck or left shoulder.

Young adults often have benign systolic ejection murmurs owing to increased systolic flow across normal aortic and pulmonic valves. This type of murmur often becomes softer or disappears when the patient sits upright.

Pansystolic (also termed holosystolic) murmurs are caused by regurgitation of blood across an incompetent mitral or tricuspid valve or through a ventricular septal defect (VSD; see Fig. 2.6). These murmurs are characterized by a uniform intensity throughout systole. In mitral and tricuspid valve regurgitation, as soon as ventricular pressure exceeds atrial pressure (i.e., when \( S_1 \) occurs), there is immediate retrograde flow across the regurgitant valve. Thus, there is no gap between \( S_1 \) and the onset of these pansystolic murmurs, in contrast to the systolic ejection murmurs discussed earlier. Similarly, there is no significant gap between \( S_1 \) and the onset of the systolic murmur of a VSD, because left ventricular systolic pressure exceeds right ventricular systolic pressure (and flow occurs) quickly after the onset of contraction.

The pansystolic murmur of advanced mitral regurgitation continues through the aortic closure sound because left ventricular pressure remains greater than that in the left atrium at the time of aortic closure. The murmur is heard best at the apex, is high pitched and “blowing” in quality, and often radiates toward the left axilla; its intensity does not change with respiration.

Tricuspid valve regurgitation is best heard along the left lower sternal border. It generally radiates to the right of the sternum and is high pitched and blowing in quality. The intensity of the murmur increases with inspiration because the negative intrathoracic pressure induced during inspiration enhances venous return to the heart. The latter augments right ventricular stroke volume, thereby increasing the amount of regurgitated blood.

The murmur of a ventricular septal defect is heard best at the fourth to sixth left intercostal spaces, is high pitched, and may be associated with a palpable thrill. The intensity of the murmur does not increase with inspiration, nor does it radiate to the axilla, which helps distinguish it from tricuspid and mitral regurgitation, respectively. Of note, the smaller the VSD, the greater the turbulence of blood flow between the left and right ventricles and the louder the murmur. Some of the loudest murmurs ever heard are those associated with small VSDs.

Late systolic murmurs begin in mid-to-late systole and continue to the end of systole. The most common example is mitral regurgitation caused by mitral valve prolapse—bowing of abnormally redundant and elongated valve leaflets into the left atrium during ventricular contraction (see Fig. 2.6). This murmur is usually preceded by a midsystolic click and is described further in Chapter 8.

**Diastolic Murmurs**

Diastolic murmurs are divided into early decrescendo murmurs and mid-to-late rumbling murmurs (Fig. 2.9). Early diastolic murmurs result from regurgitant flow through either the aortic or pulmonic valve, with the former being much more common in adults. If produced by aortic valve regurgitation, the murmur begins at \( A_2 \) and has a decrescendo shape, terminates before the next \( S_1 \). Because diastolic relaxation of the left ventricle is rapid, a pressure gradient develops immediately between the aorta and lower-pressured left ventricle in aortic regurgitation, and the murmur therefore dis-
plays its maximum intensity at its onset. Thereafter in diastole, as the aortic pressure falls and the LV pressure increases (as blood fills the ventricle), the gradient between the two chambers diminishes and the murmur decreases in intensity. Aortic regurgitation is a high-pitched murmur, best heard using the diaphragm of the stethoscope along the left sternal border with the patient sitting, leaning forward, and exhaling.

*Pulmonic regurgitation* in adults is usually owing to the presence of pulmonary arterial hypertension. It has an early diastolic decrescendo murmur profile similar to that of aortic regurgitation, but it is best heard in the pulmonic area and its intensity may increase with inspiration.

**Mid-to-late diastolic murmurs** result from either turbulent flow across a *stenotic mitral or tricuspid valve* or less commonly from abnormally increased flow across a normal mitral or tricuspid valve (see Fig. 2.9). If resulting from stenosis, the murmur begins after $S_2$ and is preceded by an opening snap. The shape of this murmur is unique. Following the opening snap, the murmur is at its loudest because the pressure gradient between the atrium and ventricle is at its maximum. The murmur then decrescendos or disappears totally during diastole as the transvalvular gradient decreases. The degree to which the murmur fades depends on the severity of the stenosis. If the stenosis is severe, the murmur is prolonged; if the stenosis is mild, the murmur disappears in mid-to-late diastole.

Whether the stenosis is mild or severe, the murmur intensifies at the end of diastole in patients in normal sinus rhythm, when atrial contraction augments flow across the valve (see Fig. 2.9). The murmur of mitral stenosis is low pitched and is heard best with the bell of the stethoscope at the apex, while the patient lies in the left lateral decubitus position. The much less common murmur of tricuspid stenosis is better aus-
cultivated at the lower sternum, near the xiphoid process.

Hyperdynamic states such as fever, anemia, hyperthyroidism, and exercise cause increased flow across the normal tricuspid and mitral valves and can therefore result in a diastolic murmur. In patients with advanced mitral regurgitation, the expected systolic murmur can be accompanied by an additional diastolic murmur owing to the increased volume of blood that must return across the valve to the left ventricle in diastole. Similarly, patients with either tricuspid regurgitation or an atrial septal defect (see Chapter 16) may display a diastolic flow murmur across the tricuspid valve.

**Continuous Murmurs**

Continuous murmurs are heard throughout the cardiac cycle without an audible hiatus between systole and diastole. Such murmurs result from conditions in which there is a persistent pressure gradient between two structures during systole and diastole. An example is the murmur of patent ductus arteriosus, in which there is an abnormal communication between the aorta and pulmonary artery (see Chapter 16). During systole, blood flows from the high-pressure ascending aorta through the ductus into the lower-pressure pulmonary artery. During diastole, the aortic pressure remains greater than that in the pulmonary artery and flow continues across the ductus. This murmur begins in early systole, crescendos to its maximum at $S_2$, then decrescendos until the next $S_1$ (Fig. 2.10).

The “to-and-fro” combined murmur in a patient with both aortic stenosis and aortic regurgitation could be mistaken for a continuous murmur (see Fig. 2.10). During systole, there is a diamond-shaped ejection murmur, and during diastole a decrescendo murmur. However, in the case of a to-and-fro murmur, the sound does not extend through $S_2$ because it has discrete systolic and diastolic components.

**SUMMARY**

Abnormal heart sounds and murmurs are common in acquired and congenital heart disease and can be predicted by the underlying pathology. Although it may seem difficult to remember even the basic features presented here, it will become easier as you learn more about the pathophysiology of these conditions, and as your experience in physical diagnoses grows. For now, just remember that the information is here, and refer to it as needed. Tables 2.2 and 2.3 and Figure 2.11 summarize features of the heart sounds and murmurs described in this chapter.

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**Figure 2.10.** A continuous murmur peaks at, and extends through, the second heart sound ($S_2$). A to-and-fro murmur is not continuous; rather, there is a systolic component and a distinct diastolic component, separated by $S_2$. $S_1$, first heart sound.
### TABLE 2.2. Common Heart Sounds

<table>
<thead>
<tr>
<th>Sound</th>
<th>Location</th>
<th>Pitch</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>S₁</td>
<td>Apex</td>
<td>High</td>
<td>Normal closure of mitral and tricuspid valves</td>
</tr>
<tr>
<td>S₂</td>
<td>Base</td>
<td>High</td>
<td>Normal closure of aortic (A₂) and pulmonic (P₂) valves</td>
</tr>
<tr>
<td>Extra systolic sounds</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ejection clicks</td>
<td>Aortic: apex and base</td>
<td>High</td>
<td>Aortic or pulmonic stenosis, or dilatation of aortic root or pulmonary artery</td>
</tr>
<tr>
<td>Mid-to-late click</td>
<td>Mitral: apex</td>
<td>High</td>
<td>Mitral or tricuspid valve prolapse</td>
</tr>
<tr>
<td>Extra diastolic sounds</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Opening snap</td>
<td>Apex</td>
<td>High</td>
<td>Mitral stenosis</td>
</tr>
<tr>
<td>S₃</td>
<td>Left-sided: apex</td>
<td>Low</td>
<td>Normal in children</td>
</tr>
<tr>
<td>S₄</td>
<td>Left-sided: apex</td>
<td>Low</td>
<td>Abnormal in adults: indicates heart failure or volume overload state</td>
</tr>
</tbody>
</table>

LLSB, lower left sternal border.

### TABLE 2.3. Common Murmurs

<table>
<thead>
<tr>
<th>Murmur Type</th>
<th>Example</th>
<th>Location and Radiation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic ejection</td>
<td>Aortic stenosis</td>
<td>2nd right intercostal space → neck (but may radiate widely)</td>
</tr>
<tr>
<td></td>
<td>Pulmonic stenosis</td>
<td>2nd–3rd left intercostal spaces</td>
</tr>
<tr>
<td>Pansystolic</td>
<td>Mitral regurgitation</td>
<td>Apex → axilla</td>
</tr>
<tr>
<td></td>
<td>Tricuspid regurgitation</td>
<td>Left lower sternal border → right lower sternal border</td>
</tr>
<tr>
<td>Late systolic</td>
<td>Mitral valve prolapse</td>
<td>Apex → axilla</td>
</tr>
<tr>
<td>Early diastolic</td>
<td>Aortic regurgitation</td>
<td>Along left side of sternum</td>
</tr>
<tr>
<td></td>
<td>Pulmonic regurgitation</td>
<td>Upper left side of sternum</td>
</tr>
<tr>
<td>Mid- or late diastolic</td>
<td>Mitral stenosis</td>
<td>Apex</td>
</tr>
</tbody>
</table>
The Cardiac Cycle: Mechanisms of Heart Sounds and Murmurs

Additional Reading


Figure 2.11. Locations of maximum intensity of common murmurs.