Auscultation of the Heart: General Principles

I. CHARACTERISTICS OF HEART SOUNDS AND MURMURS

Different heart sounds and murmurs are distinguished by four characteristics:
1. Timing (i.e., systolic or diastolic)
2. Intensity (i.e., loud or soft)
3. Duration (i.e., long or short)
4. Pitch (i.e., low or high frequency)

A fifth characteristic, the sound's quality, is also sometimes included in the descriptions of sounds (e.g., it is described as "musical," a "whoop," or a "honk"). Almost all heart sounds contain a mixture of frequencies (i.e., they are not musical in the acoustic sense but instead are "noise," like the static of a radio). Therefore, the descriptors low-frequency and high-frequency do not indicate that a sound has a pure musical tone of a certain low or high pitch but instead that the bulk of the sound's energy is within the low or high range.

Although the human ear can hear sounds with frequencies from 20 to 20,000 cycles per second (Hz), the principal frequencies of heart sounds and murmurs are at the lower end of this range, from 20 to 500 Hz. Low-frequency sounds, therefore, are those whose dominant frequencies are less than 100 Hz, such as third and fourth heart sounds and the diastolic murmur of mitral stenosis. These sounds are usually difficult to hear because the human ear perceives lower frequencies relatively less well than higher frequencies. The murmur containing the highest-frequency sound is aortic regurgitation, whose dominant frequencies are about 400 Hz. The principal frequencies of other sounds and murmurs are between 100 and 400 Hz.

II. THE STETHOSCOPE

A. BELL AND DIAPHRAGM

The stethoscope has two different heads to receive sound, the bell and the diaphragm. The bell is used to detect low-frequency sounds and the diaphragm to detect high-frequency sounds.

The traditional explanation that the bell selectively transmits low-frequency sounds and the diaphragm selectively filters out low-frequency sounds is probably incorrect. Actually, the bell transmits all frequencies well, but in some patients with high-frequency murmurs (e.g., aortic regurgitation), any additional low-frequency sound masks the high-frequency sound and makes the murmur difficult to detect. The diaphragm does not selectively filter out low-frequency sounds but instead attenuates all frequencies equally, thus dropping the barely audible low-frequency ones below the threshold of human hearing.

B. PERFORMANCE OF DIFFERENT STETHOSCOPE MODELS

Many studies have examined the acoustics of stethoscopes, but the clinical relevance of this research has never been formally tested. In general, these studies show that shallow bells transmit sound as well as deeper bells and that double-tube stethoscopes are equivalent to single-tube models. The optimal internal bore of a stethoscope is somewhere between one-eighth and three-sixteenths of an inch because smaller bores diminish transmission of the higher-frequency sounds. Compared with shorter lengths of stethoscope tubing, longer tubes also impair the conduction of high-frequency sounds.

Most modern stethoscopes, however, transmit sound equally well, the differences among various models for single frequencies being very small. The most important source of poor acoustic performance is an air leak, which typically results from poorly fitted earpieces. Even a tiny air leak with a diameter of only 0.015 inch will diminish transmission of sound by as much as 20 dB, particularly for those sounds of less than 100 Hz.

III. USE OF THE STETHOSCOPE

Between the 1950s and late 1970s, cardiac auscultation was at its peak. During this time, cardiologists perfected their skills by routinely comparing the bedside findings to the patient's phonocardiogram, angiogram, and surgical findings, which allowed clinicians to make precise and accurate diagnoses from bedside findings alone. The principles of bedside diagnosis used by these clinicians are included elsewhere in this book. How these clinicians specifically used the stethoscope to examine the patient is presented below.

A. EXAMINATION ROOM

Many faint heart sounds and murmurs are inaudible unless there is complete silence in the room. The clinician should close the door to the examination room, turn off the television and radio, and ask that all conversation stop.

B. BELL PRESSURE

To detect low-frequency sounds, the stethoscope bell should be applied to the body wall with only enough pressure to create an air seal and exclude ambient noise. Excessive pressure with the bell stretches the skin, which

*Decibels describe relative intensity (or loudness) on a logarithmic scale.

†In the late 1970s, two events initiated the decline of cardiac auscultation: the widespread introduction of echocardiography and the decision by insurance companies to no longer make reimbursements for phonocardiography.
then acts like a diaphragm and makes low-frequency sounds more difficult to hear. By selectively varying the pressure on the stethoscope bell, the clinician can easily distinguish low-frequency from high-frequency sounds. If a sound is audible with the bell using light pressure but disappears with firm pressure, it is a low-frequency sound. This technique is frequently used to confirm that an early diastolic sound is indeed a third heart sound (i.e., third heart sounds are low-frequency sounds, whereas other early diastolic sounds such as the pericardial knock are high-frequency sounds) and to distinguish the combined fourth and first heart sounds \( (S_4 + S_1) \) from the split \( S_1 \). (The \( S_4 \) is a low-frequency sound, but the \( S_1 \) is not; firm pressure renders the \( S_4 + S_1 \) sounds into a single sound but does not affect the double sound of the split \( S_1 \).)

C. PATIENT POSITION

The clinician should listen to the patient's heart with the patient in three positions: supine, left lateral decubitus, and seated upright. The lateral decubitus position is best for detection of the third and fourth heart sounds and the diastolic murmur of mitral stenosis. (To detect these sounds, the clinician places the bell lightly over the apical impulse or just medial to the apical impulse.) The seated upright position is necessary to further evaluate audible expiratory splitting of \( S_2 \) (see Chapter 38) and to detect some pericardial rubs and murmurs of aortic regurgitation (see Chapters 43 and 45).

D. ORDER OF EXAMINATION

Routine auscultation of the heart should include the right upper sternal area, the entire left sternal border, and the apex. Some cardiologists recommend proceeding from base to apex; others from apex to base. The diaphragm of the stethoscope should be applied to all areas, especially at the upper left sternal area to detect \( S_2 \) splitting and at all areas to detect other murmurs and sounds. After using the diaphragm to listen to the lower left sternal area and apex, the bell should also be applied to these areas to detect diastolic filling sounds \( (S_4 + S_1) \) and diastolic rumbling murmurs (e.g., mitral stenosis).

In selected patients, the clinician also should listen over the carotid arteries and axilla (in patients with systolic murmurs, to clarify radiation of the murmur), the lower right sternal area (in patients with the diastolic murmur of aortic regurgitation, to detect aortic root disease), the back (in young patients with hypertension, to detect the continuous murmur of coarctation), or other thoracic sites (in patients with central cyanosis, to detect the continuous murmur of pulmonary arteriovenous fistulas).

E. DESCRIBING THE LOCATION OF SOUNDS

When describing heart sounds and murmurs, the clinician should identify where on the chest wall the sound is loudest. Traditionally, the second right intercostal space next to the sternum is called the aortic area or right base; the second left intercostal space next to the sternum, the pulmonary area or left base; the fourth or fifth left parasternal space, the tricuspid area or left lower sternal border; and the most lateral point of the palpable cardiac impulse, the mitral area or apex (see Fig. 36-1 in Chapter 36). The terms aortic area, pulmonary area, tricuspid area, and mitral area are ambiguous, however, and are best avoided. Many patients with aortic stenosis have murmurs loudest in the mitral area, and some with mitral regurgitation have murmurs in the pulmonary or aortic area. A more precise way to describe the location of sounds is to use the apex and the parasternal areas as reference points, the parasternal location being further specified by the intercostal space (first, second, third, or lower sternal border) and whether it is the right or left edge of the sternum. For example, a sound might be loudest at the "apex," the "second left intercostal space" (i.e., next to the left sternal edge in the second intercostal space), or "between the apex and left lower sternal border."

F. TECHNIQUE OF FOCUSING

The human brain has an uncanny ability to isolate and focus on one type of sensory information, by repressing awareness of all other sensations. A common example of this phenomenon is the person reading a book in a room in which a clock is ticking: The person may read long passages of the book without even hearing the clock but hears the ticking clock immediately after putting the book down. When listening to the heart, the clinician's attention is quickly drawn to the most prominent sounds, but this occurs as the expense of detecting the fainter sounds. To avoid missing these fainter sounds or subtle splitting, therefore, the clinician should concentrate sequentially on each part of the cardiac cycle, asking the following questions at each location:

1. Is \( S_1 \) soft or loud?
2. Is \( S_2 \) split and, if so, how is it split?
3. Are there any extra sounds or murmurs during systole?
4. Are there any extra sounds or murmurs during diastole?

G. IDENTIFYING SYSTOLE AND DIASTOLE

Because all auscultatory findings are characterized by their timing, distinguishing systole from diastole accurately is essential. Three principles help the clinician distinguish these events.

1. Systole Is Shorter Than Diastole

If the heart rate is normal or slow, systole can be easily distinguished from diastole because systole is much shorter. The normal cadence of the heart tones, therefore, is

\[
\text{ lub dup lub dup lub dup lub dup}
\]

(\( S_1 \) is \( S_1 \) and \( S_2 \) is \( S_2 \)). When the heart rate accelerates, however, diastole shortens, and at a rate of 100 beats/min or more, the cadence of \( S_1 \) and \( S_2 \) resembles a tic-toc rhythm:

\[
\text{ lub dup lub dup lub dup lub dup lub dup lub dup lub dup}
\]

In these patients, other techniques are necessary to distinguish systole from diastole.
2. Characteristics of the First and Second Heart Sounds
At the second left intercostal space, S₂ is generally louder, shorter, and sharper than S₁. (S₂ has more high-frequency energy than S₁, which is why "dup," a snappier sound than "lub," is used to characterize S₂.) If the timing of extra heart sounds and murmurs is confusing at the lower sternal edge or apex (as it often is in patients with fast heart rhythms), the clinician can return the stethoscope to the second left intercostal space, identify S₂ by its louder and sharper sound, and then inch slowly back to the area of interest, keeping track of S₂ along the way.

3. Carotid Impulse
The palpable impulse from the carotid usually occurs just after S₁, which the clinician detects by simultaneously listening to the heart tones and palpating the carotid artery. In elderly patients with tachycardia, however, this rule is sometimes misleading because the carotid impulse seems to fall closer to S₂, although even in these patients the carotid impulse still falls between S₁ and S₂.

The references for this chapter can be found on www.expertconsult.com.

THE FIRST HEART SOUND (S₁)

I. THE FINDING
S₁ is heard well across the entire precordium, both with the bell and diaphragm of the stethoscope. It is usually loudest at or near the apex and contains more low-frequency energy than does S₂, which explains why, when mimicking the sound, the term "lub" is used for S₁ and the sharper term "dup" for S₂.

II. PATHOGENESIS
A. CAUSE OF S₁
The precise cause of S₁ has been debated for decades. Although its two recordable components coincide with closure of the mitral and tricuspid valves, the force of valve closure itself is insufficient to generate sound. Instead, their closure probably causes moving columns of blood to abruptly decelerate, which sets up vibrations in the chordae tendineae, ventricles, and blood as a unit (i.e., cardiohemic system).
B. INTENSITY OF $S_1$

The most important abnormalities of $S_1$ relate to its intensity: The sound can be abnormally loud or abnormally faint or can vary in intensity abnormally from beat to beat. The primary variables governing intensity of $S_1$ are the strength of ventricular contraction and the position of the atrioventricular leaflets at the onset of ventricular systole.

1. **Ventricular Contractility**

The stronger the ventricular contraction, the louder the $S_1$. Strong contractions, which have a high dP/dT (i.e., large increase in pressure with respect to time), intensify $S_1$ because the valves close with more force and generate more vibrations in the cardiohemic system.\(^6\)

2. **Position of the Valve Leaflets at Onset of Ventricular Systole**

If the mitral valve is wide open at the onset of ventricular systole, it will take longer to close completely than if it had been barely open. Even this small delay in closure intensifies $S_1$ because closure occurs on a later and steeper portion of the left ventricular pressure curve (i.e., dP/dT is greater).\(^8\)

The PR interval is the main variable determining the position of the valves at the beginning of ventricular systole. If the PR interval is short, ventricular systole immediately follows atrial systole (i.e., the R wave immediately follows the P wave). Because atrial systole kicks the valve open, a short PR guarantees that the valve will be wide open at the onset of ventricular systole. In contrast, a long PR interval allows time for the cusps of the atrioventricular valves to float back together before ventricular systole occurs. Studies show that, with PR intervals less than 0.20 seconds, the intensity of $S_1$ varies inversely with the PR interval (the shorter the PR interval, the louder the sound). With PR intervals greater than 0.20 seconds, $S_1$ is faint or absent.\(^8\)

III. CLINICAL SIGNIFICANCE

A. **LOUD $S_1$**

$S_1$ may be abnormally loud because of unusually vigorous ventricular contractions or because of delayed closure of the mitral valve.

1. **Vigorous Ventricular Contractions**

Vigorous contractions, such as those occurring from fever and sympathetic stimulation (e.g., beta-adrenergic inhalers, thyrotoxicosis), increase dP/dT and intensify $S_1$.\(^6\)

2. **Delayed Closure of the Mitral Valve**

a. **Prolapsed Mitral Valve**

In patients with the murmur of mitral regurgitation, a loud $S_1$ is a clue to the diagnosis of early prolapse of the mitral valve. (Many patients with mitral regurgitation have a normal or soft $S_1$.)\(^10\) $S_1$ is loud in these patients because the prolapsing leaflets stop moving and tense later than normal, when dP/dT in the ventricle is greater.\(^10\)

b. **Mitral Stenosis**

Ninety percent of patients with pure uncomplicated mitral stenosis have a loud $S_1$.\(^12\) Because the murmur of mitral stenosis is often difficult to hear, a traditional teaching is that clinicians should suspect mitral stenosis in any patient with a loud, unexplained $S_1$ and listen carefully for the murmur with the patient lying on the left side.

Mitral stenosis delays closure of the mitral valve because the pressure gradient between the left atrium and left ventricle keeps the leaflets open until the moment of ventricular systole. After successful valvuloplasty, the loud $S_1$ becomes softer.\(^12\)

c. **Left Atrial Myxoma**

Many patients with left atrial myxoma (seven of nine in one series) also have a loud $S_1$ because the tumor falling into the mitral orifice during diastole delays closure of the valve.\(^11\)

B. **FAINT OR ABSENT $S_1$**

$S_1$ is unusually faint if ventricular contractions are weak or if the mitral valve is already closed when ventricular systole occurs.

1. **Weak Ventricular Contractions (Low dP/dT)**

Common examples of weak contractions causing a faint $S_1$ are myocardial infarction and left bundle branch block.\(^14\)

2. **Early Closure of the Mitral Valve**

Common causes of early mitral closure causing the faint $S_1$ include the following:

a. **Long PR Interval (>0.20 Seconds)**

See the section on Intensity of $S_1$.

b. **Acute Aortic Regurgitation**

In patients with the murmur of aortic regurgitation, the faint or absent $S_1$ is an important clue that the regurgitation is acute (e.g., endocarditis) and not chronic. Patients with acute aortic regurgitation have much higher left ventricular end-diastolic pressures than those with chronic regurgitation because the acutely failing valve has not allowed time for the ventricle to enlarge, as it does to compensate for chronic regurgitation. The high pressures in the ventricle eventually exceed diastolic left atrial pressures, closing the mitral valve before ventricular systole and thus making $S_1$ faint or absent.\(^15\)

C. **VARYING INTENSITY OF $S_1$**

If the arterial pulse rhythm is regular but $S_1$ varies in intensity, the only possible explanation is that the PR interval is changing from beat to beat, which means the patient has atrioventricular dissociation. In contrast, in patients with irregular rhythms, a changing intensity of $S_1$ has no diagnostic significance, because ventricular filling and dP/dT—and, therefore, $S_1$ intensity—depend completely on cycle length.
In patients with pacer-induced regular rhythms, an \( S_1 \) that varies in intensity is compelling evidence for atrioventricular dissociation (likelihood ratio \( LR = 24.4 \); EBM Box 38-1). Presumably, the finding is also as accurate in patients with native rhythms. In patients with complete heart block, \( S_1 \) intensity is predictable, varying inversely with the PR interval for intervals of less than 0.2 seconds, becoming inaudible for intervals of 0.2 to 0.5 seconds, and becoming louder again with intervals of more than 0.5 seconds (because the mitral valve reopens).9

### D. PROMINENT SPLITTING OF \( S_1 \)

Any delay in the closure of the tricuspid valve, the second component of \( S_1 \), accentuates splitting of \( S_1 \). This finding therefore occurs in patients with right bundle branch block or in left ventricular eustachian or paced beats, all of which delay the onset of right ventricular systole and also cause wide physiologic splitting of \( S_1 \) (see later).3,23

How to distinguish the split \( S_1 \) from other double sounds occurring around \( S_1 \), such as \( S_4 \) plus \( S_1 \) and \( S_1 \) plus ejection sound, is discussed in Chapter 39.

### THE SECOND HEART SOUND (\( S_2 \))

The most important diagnostic feature of \( S_2 \) is its "splitting," which refers to how the aortic and pulmonic components of \( S_2 \) vary in timing during the respiratory cycle. The intensity of \( S_2 \) has less diagnostic importance. (This contrasts with \( S_1 \), in which intensity is more important than splitting.) Splitting of \( S_2 \) was first recognized by Potain in 1865, and its importance to cardiac auscultation was described by Leatham in the 1950s, who called \( S_2 \) the "key to auscultation of the heart."26,27 The correct explanation for normal splitting—increased "hangout" in the pulmonary circulation—was discovered in the 1970s.28,29

### I. NORMAL SPLITTING OF \( S_2 \)

#### A. THE FINDING

In normal persons, the first component of \( S_2 \) is caused by closure of the aortic valve (\( A_2 \)); the second, by closure of the pulmonic valve (\( P_2 \)). During inspiration, the interval separating \( A_2 \) and \( P_2 \) increases by about 20 to 30 ms (Fig. 38-1).17,27,29

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**EBM BOX 38-1**

**The First and Second Heart Sounds**

<table>
<thead>
<tr>
<th>Finding (Reference)†</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Likelihood Ratio† if Finding Is Present</th>
<th>Absence of palpable ( P_2 ), arguing against pulmonary hypertension</th>
<th>Varying intensity ( S_1 ), detecting AV dissociation if tachycardia</th>
<th>Absence of fixed wide ( S_2 ) splitting, arguing against atrial septal defect</th>
<th>Fixed wide ( S_2 ) splitting, detecting atrial septal defect</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>First Heart Sound</strong></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>VARYING INTENSITY ( S_1 ) Detecting atrioventricular dissociation16</td>
<td>58</td>
<td>98</td>
<td>24.4</td>
<td>0.4</td>
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<tr>
<td><strong>Second Heart Sound</strong></td>
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<tr>
<td>FIXED WIDE SPLITTING Detecting atrial septal defect17</td>
<td>92</td>
<td>65</td>
<td>2.6</td>
<td>0.1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PARADOXIC SPLITTING Detecting significant aortic stenosis18</td>
<td>50</td>
<td>79</td>
<td>NS</td>
<td>NS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LOUD ( P_2 ) Detecting pulmonary hypertension19,20</td>
<td>58-96</td>
<td>19-46</td>
<td>NS</td>
<td>NS</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>PALPABLE ( P_2 ) Detecting pulmonary hypertension19</td>
<td>96</td>
<td>73</td>
<td>3.6</td>
<td>0.05</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>ABSENT OR DIMINISHED ( S_2 ) Detecting significant aortic stenosis in patients with aortic flow murmurs18,21-24</td>
<td>44-90</td>
<td>63-98</td>
<td>3.1</td>
<td>0.4</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

*Diagnostic standard: For atrioventricular dissociation, ventricles were paced independently of atria, for atrial septal defect, right heart catheterization for severe aortic stenosis, aortic valve area <0.75 cm²,12 <0.6 cm²;2peak gradient >50 mm Hg,12,13 or peak velocity of aortic flow >3.6 m/sec12 or 2.4 m/sec11; for pulmonary hypertension, mean pulmonary arterial pressure >50 mm Hg.

†Definition of findings: For loud \( P_2 \), splitting heard with lead second component19 or \( S_2 \) louder at left second interspace than right second interspace25; the figures for fixed splitting of \( S_2 \) apply only to patients having audible expiratory splitting.

‡Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR. NS, not significant.
B. LOCATION OF SOUND

$S_2$ splitting is usually heard only in the second or third intercostal space, next to the left sternum. It is sometimes heard at a slightly lower location, especially in patients with chronic pulmonary disease, and at a slightly higher location in those who are obese. Splitting is not heard at other locations normally because $P_2$ is too faint.

C. TECHNIQUE

It is important that the patient breathe regularly in and out when evaluating $S_2$ splitting because held inspiration or held expiration tends to make the two components drift apart, thus making it impossible to interpret the sound.

D. PHYSIOLOGY OF SPLITTING

The normal delay in $P_2$ results from a long "hangout" interval in the normal pulmonary circulation. (It is not because right ventricular systole ends later than left ventricular systole; they actually end at the same moment; Fig. 38-2.) Hangout means that the pulmonary circulation offers so little resistance to blood flow that flow continues for a short period even after completion of right ventricular mechanical systole. At the aortic valve, there is little hangout, causing flow to cease and the valve to close immediately after completion of left ventricular contraction.

$A_2$ and $P_2$ move apart during inspiration, primarily because inspiration delays $P_2$ even more. About half of the inspiratory augmentation of the $A_2$-$P_2$ interval is due to a further increase in the hangout interval in the pulmonary circulation. About 25% of inspiratory augmentation is due to shortening of right ventricular systole (from increased filling of the right side of the heart during inspiration), and the remaining 25% is due to shortening of left ventricular systole (from a reduction of filling of the left side of the heart during inspiration).

II. ABNORMAL SPLITTING OF $S_2$

A. THE FINDING

There are three abnormalities of $S_2$ splitting (see Fig. 38-1):

1. Wide Physiologic Splitting

Wide physiologic splitting means that splitting occurs during inspiration and expiration, though the $A_2$-$P_2$ interval widens further during inspiration.

2. Wide Fixed Splitting

Wide fixed splitting means that splitting occurs during inspiration and expiration, but the $A_2$-$P_2$ interval remains constant.

3. Paradoxic Splitting (Reversed Splitting)

Paradoxic splitting means that audible expiratory splitting narrows or melds into a single sound during inspiration. Paradoxic splitting occurs because the order of the $S_2$ components has reversed: $A_2$ now follows $P_2$, and as $P_2$ is delayed during inspiration, the sounds move together.

*These two components are very close together, bordering the threshold of being perceived as a single sound. Harvey sugestas mimicking the normal expiratory sound by striking a single knuckle against a tabletop and mimicking inspiratory physiologic splitting by striking two knuckles almost simultaneously.\(^\)\(^\) Constant suggests mimicking inspiratory splitting by rolling the tongue as in a Spanish $tr\ or\ tr$, or saying $s\ p$ as quickly and sharply as possible.
Table 38-1 lists the common causes of abnormal S₂ splitting.

1. **Wide Physiologic Splitting**

   Wide physiologic splitting may result from P₂ appearing too late or A₂ too early (see Table 38-1).17,35 The most common cause is right bundle branch block.

   In pulmonic stenosis, the A₂-P₂ interval correlates well with severity of stenosis (gauged by the right ventricular systolic pressure, \( r = 0.87, p < 0.001 \)).38 Although in many patients the clinician must listen at the third interspace to hear splitting because the murmur is too loud at the second interspace.

   In most patients with pulmonary hypertension, the normal hangout interval disappears and S₂ is single. S₂ becomes wide in these patients only if there is associated severe right ventricular dysfunction and prolonged right ventricular systole.28,29,39 Most patients with pulmonary hypertension and a wide S₂ have either long-standing severe pulmonary hypertension28,29,39 or massive pulmonary embolism. (The wide S₂ of pulmonary embolism is temporary, usually lasting hours to days.)

2. **Wide and Fixed Splitting**

   Patients with atrial septal defect have wide fixed splitting of S₂, although this is true only when their pulse is regular. (If the patient has atrial fibrillation or frequent extrasystoles, the degree of splitting varies directly with the preceding cycle length.)17,41 The reason S₂ is wide is not the same in every
patient: In some patients, hangout is increased; in others, right ventricular mechanical systole is prolonged. S2 is fixed because hangout remains constant during respiration^4^ and because the presence of a common left and right atrial chamber interrupts the normal respiratory variation of right ventricular filling.

In patients with audible expiratory splitting (and regular rhythm), the absence of fixed splitting significantly decreases the probability of atrial septal defect (LR = 0.1; see EBM Box 38-1), whereas the presence of fixed splitting increases the probability of atrial septal defect only modestly (LR = 2.6; see EBM Box 38-1). Patients with false-positive results (i.e., fixed splitting without atrial septal defect) commonly have the combination of right ventricular failure and audible expiratory splitting from bundle branch block or some other cause.

3. Paradoxic Splitting
In elderly adults with aortic flow murmurs, the finding of paradoxic splitting does not distinguish significant aortic stenosis from less severe disease (see EBM Box 38-1).

D. S2 Splitting Versus Other Double Sounds
Other double sounds that mimic S2 splitting include the following (see also Chapter 40):

1. S2-Opening Snap
In contrast to the split S2, the S2-opening snap interval is slightly wider; the opening snap is loudest at the apex, and the opening snap ushers in the diastolic rumble of mitral stenosis at the apex. Patients with S2-opening snap sometimes have a triple sound (split S2 plus opening snap) during inspiration at the upper sternal border.

2. S2-Pericardial Knock
In contrast to the split S2, the S2-knock interval is slightly wider, the pericardial knock is loudest at or near the apex, and the knock is always accompanied by elevated neck veins.

3. S2-Third Heart Sound
In contrast to the split S2, the S1-S2 interval is two to three times wider, and S2 is a low-frequency sound heard best with the bell.

4. Late Systolic Click-S2
Clicks are loudest at or near the apex and are often multiple. Their timing changes with maneuvers (see Chapter 44).

III. INTENSITY OF S2
Despite traditional teachings, no evidence supports a loud P2 as a sign of pulmonary hypertension. Whether defined as an S2 that is louder at the left side of the upper sternum compared with the right side^2^ or as a split S2 with a louder second component,^19^ the finding does not accurately discriminate patients with pulmonary hypertension from those without it (see EBM Box 38-1). Even when A2 and P2 are precisely identified by phonocardiography (e.g., A2 corresponds to aortic incisura on simultaneous aortic pressure tracing), the relative intensities of the two components do not correlate well with pulmonary pressure.^42^ Another suggested sign of pulmonary hypertension is audible splitting at the apex, which is based on the observation that P2 normally is not heard at the apex^30^ and the assumption that splitting at this location therefore indicates that P2 is abnormally loud. But even this finding correlates better with the etiology of heart disease—it is common in atrial septal defect and primary pulmonary hypertension—than it does with measurements of pulmonary pressure.^^39,42^^

Nonetheless, the palpable S2 does accurately detect pulmonary arterial pressures of 50 mm Hg or more in patients with mitral stenosis (positive LR = 3.6, negative LR = 0.05; see EBM Box 38-1). In this study, the palpable P2 was defined as an abrupt tapping sensation coincident with S2 at the second left intercostal space.

In patients with aortic flow murmurs, an absent or diminished S2 increases the probability of significant aortic stenosis (LR = 3.1; see Chapter 42).

The references for this chapter can be found on www.expertconsult.com.
CHAPTER 39 — THE THIRD AND FOURTH HEART SOUNDS

The Third and Fourth Heart Sounds

I. INTRODUCTION

Although the third and fourth heart sounds (S₃ and S₄) are both sounds that originate in the ventricle from rapid diastolic filling, they differ in timing and clinical significance. S₃ appears in early diastole, and if the patient is older than 40 years of age, the sound indicates severe systolic dysfunction or valvular regurgitation. In persons younger than 40 years of age, S₃ may be a normal finding (i.e., the "physiologic S₃"). S₄ appears in late diastole, immediately before S₁, indicating that the patient's ventricle is abnormally stiff from hypertrophy or fibrosis. If discovered in persons of any age, the S₄ is an abnormal finding.

In the late 19th century, the great French clinician Potain accurately described most features of S₃ and S₄, their pathogenesis, and their distinction from other sounds such as the split S₁ or split S₂. In his writings he called them gallops, a term he attributed to his teacher Bouillart.

II. DEFINITIONS

Several different terms have been used to describe these diastolic sounds.

A. GALLOP

A gallop is a triple rhythm with an extra sound in diastole (either S₃ or S₄, or their summation). The term refers only to pathologic sounds (i.e., it excludes physiologic S₃), and despite its connotation, a patient may have a gallop whether the heart rate is fast or slow.

B. THIRD HEART SOUND (S₃)

The third heart sound is sometimes called the ventricular gallop or protodiastolic gallop. It appears in early diastole, 120 to 180 ms after S₂. To mimic the sound, the clinician should first establish the cadence of the normal S₁ (lub) and S₂ (dup):

\[
\text{lub} \quad \text{dup} \quad \text{lub} \quad \text{dup} \quad \text{lub} \quad \text{dup}
\]

and then add an early diastolic sound (hub):

\[
\text{lub} \quad \text{duh} \quad \text{lub} \quad \text{duh} \quad \text{lub} \quad \text{duh}
\]

The overall cadence of the S₃ gallop (lub duh lub duh lub duh) is similar to the cadence of the word Kentucky.

C. FOURTH HEART SOUND (S₄)

The fourth heart sound is sometimes called the atrial gallop or presystolic gallop. To mimic the sound, the clinician establishes the cadence of S₁ and S₃ (lub dup) and then adds a presystolic sound (be):

\[
\text{be lub} \quad \text{dup} \quad \text{be lub} \quad \text{dup} \quad \text{be lub} \quad \text{dup}
\]

The cadence of the S₄ gallop (be lub dup) is similar to the cadence of Tennessee.

D. SUMMATION GALLOP

The summation gallop is a loud gallop that occurs in patients with tachycardia. In fast heart rhythms, diastole shortens, causing the events that produce S₃ (rapid early diastolic filling) to coincide with those producing S₄ (atrial systole). The resulting sound sometimes is louder than the patient's S₁ or S₂.

Not all gallop rhythms in patients with tachycardia are summation gallops. The only way to confirm the finding is to observe the patient after the heart rate slows. (In the past, slowing was often induced by carotid artery massage, although in elderly patients this is no longer recommended; see Chapter 15.) If slowing causes the gallop to disappear or evolve into two distinct but fainter sounds (i.e., S₁ and S₄), it was a genuine summation gallop. If the sound evolves instead into a single S₁ or single S₄, it was not a summation gallop.

E. QUADRUPLE RHYTHM

The quadruple rhythm consists of S₁ and S₂ and both S₃ and S₄. If it is an uncommon finding, usually only evident in patients with slow heart rates. It is sometimes called the train wheel rhythm because the sound resembles that produced by the two pairs of wheels from adjacent train cars as they cross the coupling of a railroad track.

\[
\text{be lub} \quad \text{duh} \quad \text{be lub} \quad \text{duh} \quad \text{be lub} \quad \text{duh}
\]

III. TECHNIQUE

A. LOCATION OF SOUND AND USE OF STETHOSCOPE

S₃ and S₄ are both low-frequency sounds (20 to 70 Hz), bordering on the threshold of hearing. Therefore, they are best heard with the bell of the stethoscope, applied lightly to the body wall with only enough force to...
create an air seal. Gallops that originate in the left ventricle are best heard with the bell over the apical impulse or just medial to it. They are sometimes only audible with the patient lying in the left lateral decubitus position. Gallops from the right ventricle are best heard with the bell over the left lower sternal border or, in patients with chronic lung disease, the subxiphoid area.

B. RIGHT VS. LEFT VENTRICULAR GALLOPS
Aside from their different locations, other distinguishing features of right and left ventricular gallops are their response to respiration and association with other findings in the neck veins and precordium. Right ventricular gallops become louder during inspiration; left ventricular gallops become softer during inspiration. The right ventricular S4 may be associated with giant A waves in the neck veins and sometimes a loud presystolic jugular sound (see Chapter 34). The left ventricular S4 may be associated with a palpable presystolic movement of the apical impulse (see Chapter 36).

C. DISTINGUISHING THE S4-S1 SOUND FROM OTHER SOUNDS
Three combinations of heart sounds produce a double sound around S1:
1. The S4-S1 sound
2. The split S1
3. The S1-ejection sound
The following characteristics distinguish these sounds:

1. Use of the Bell
The S4 is a low-frequency sound, best heard with the bell. Firm pressure with the bell on the skin—which tends to remove low-frequency sounds—will cause the S4-S1 combination to evolve into a single sound, in contrast to the split S1 and the S1-ejection sounds, which remain double.

2. Location
The S4-S1 sound is heard best at the apex, left lower sternal border, or subxiphoid area. (See the section on Location of Sound and Use of Stethoscope.) The split S1 is loudest from the apex to the lower sternal border but sometimes is also heard well over the upper left sternal area. The aortic ejection sound is heard from the apex to the upper right sternal border. The pulmonary ejection sound is restricted to the upper left sternal area.

3. Effect of Respiration
Although the S4 may become louder (right ventricular S4) or softer (left ventricular S4) during inspiration, respiration does not affect the interval between S4 and S1. In contrast, the split S1 interval varies with respiration in up to one-third of patients.

Expiration makes the pulmonary ejection sound louder. The aortic ejection sound does not vary with respiration.

4. Palpation
Only the S4-S1 sound is accompanied by a presystolic apical impulse (see Chapter 36). The intensity of the S1 (i.e., by auscultation) correlates moderately with the amplitude of the presystolic impulse on apexcardiography ($r = 0.46, p < 0.01$); similarly, the palpability of the presystolic impulse correlates roughly with the amplitude of S4 on phonocardiography ($r = 0.52, p < 0.01$).

IV. PATHOGENESIS
A. NORMAL VENTRICULAR FILLING CURVES
Filling of the right and left ventricles during diastole is divided into three distinct phases (Fig. 39-1). The first phase, the rapid filling phase, begins immediately after opening of the atrioventricular valves. During this phase, blood stored in the atria rapidly empties into the ventricles. The second phase, the plateau phase (diastasis), begins at the moment the ventricles are unable to relax passively any further. Very little filling occurs during this phase. The third phase, atrial systole, begins with the atrial contraction, which expands the ventricle further just before the next S1.

B. VENTRICULAR FILLING AND SOUND
Both S1 and S2 occur at those times during diastole when blood flow entering the ventricles temporarily stops, that is, the S1 appears at the end of the rapid filling phase and the S2 toward the peak of atrial systole (see Fig. 39-1). Sounds become audible if the blood decelerates abruptly enough, which transmits sufficient energy to the ventricular walls and causes them to vibrate. (An analogy is the tensing of a handkerchief between two hands: abrupt tensing produces sound, whereas slow tensing is silent.) Two variables govern the suddenness of this deceleration and, therefore, whether or not the gallops become audible:

1. The flow rate during entry
2. The stiffness of the ventricle
The greater the flow rate, the louder the sound. The stiffer the ventricle, the higher the frequency of the sound. Because gallops consist of low frequencies that are difficult to hear (about 20 to 50 Hz), anything increasing its frequency content (i.e., stiff ventricles) makes the sound more likely to be heard.

Even though S1 and S2 both result from rapid flow rates into stiff ventricles, the diseases causing them differ completely.

C. THIRD HEART SOUND (S3)
The S3 gallop appears when early diastolic filling is exaggerated, which occurs in two types of cardiac disorders: congestive heart failure and regurgitation and shunts.
dilated cardiomyopathy and low cardiac output. Although both high atrial pressure (causing rapid flow rates) and cardiomyopathy (causing stiff ventricles) contribute to the sound, atrial pressure is the more important clinical variable because the sound disappears as soon as pressure falls after diuresis.

2. Regurgitation and Shunts
Patients with valvular regurgitation or left-to-right cardiac shunts also may develop an S₃ gallop, whether or not atrial pressure is high because these disorders all cause excess flow over the atrioventricular valves. Patients with mitral regurgitation, ventricular septal defect, or patent ductus arteriosus may develop a left ventricular S₃ from excess diastolic flow over the mitral valve into the left ventricle. (In mitral regurgitation, the excess diastolic flow simply represents the diastolic return of the regurgitant flow.) Patients with atrial septal defect may develop a right ventricular S₃ from excess flow over the tricuspid valve into the right ventricle.

D. FOURTH HEART SOUND (S₄)
The S₄ gallop occurs in patients with hypertension, ischemic cardiomyopathy, hypertrophic cardiomyopathy, or aortic stenosis—all disorders characterized by ventricles stiffened from hypertrophy or fibrosis. Patients with the sound must be in sinus rhythm and have strong atrial contractions, and most have normal atrial pressures, normal cardiac output, and normal ventricular chamber size. Unlike the S₃, the S₄ is a durable finding that does not wax and wane unless the patient develops atrial fibrillation (and thus loses the atrial contraction).

E. SUMMATION GALLOP AND QUADRUPLE RHYTHM
The summation gallop occurs because fast heart rates shorten diastole, primarily by eliminating the plateau phase (see Fig. 39-1), which brings the events causing S₃ close to those causing S₄. Diastolic filling is concentrated into a single moment, thus causing a very loud sound.

The quadruple rhythm typically occurs in patients who have had a long-standing S₃ gallop from ischemic or hypertensive heart disease but who then develop cardiac decompensation, high filling pressures, and an S₄.

Rarely, an intermittent summation gallop may appear in patients with slow heart rates due to complete heart block (or VVI pacing). The gallop appears only during those moments of atrioventricular dissociation when atrial systole and early diastole coincide (i.e., the P wave on the electrocardiogram falls just after the QRS). Although the sound is technically a summation gallop, the clinician perceives what sounds like an intermittent S₃.

F. PHYSIOLOGIC S₃
Persons younger than 40 years of age with normal hearts may also have an S₃ sound (i.e., physiologic S₃) because normal early filling can sometimes be so rapid that it ends abruptly and causes the ventricular walls to vibrate and produce sound. Compared with healthy persons lacking the sound, those
with the physiologic S₃ are leaner and have more rapid early diastolic filling. ¹ The physiologic S₃ disappears by age 40 years because normal aging slows ventricular relaxation and shifts filling to later in diastole, thus diminishing the rate of early diastolic filling and making the sound disappear.²⁷

V. CLINICAL SIGNIFICANCE

A. THIRD HEART SOUND

1. Congestive Heart Failure

EBM Box 39-1 shows that the presence of the S₃ gallop is a significant finding indicating depressed ejection fraction (LR = 3.4 to 4.1), elevated left atrial pressures (LR = 3.9), and elevated B-type natriuretic peptide (BNP) levels (LR = 10.1). Other studies confirm its value as a predictor of poor systolic function.³⁵ ³⁴ The absence of the S₃ gallop argues that the patient's ejection fraction is greater than 30% (i.e., negative LR for ejection fraction <30% is 0.3; see EBM Box 39-1).

In patients with a history of congestive heart failure, the S₃ predicts responsiveness to digoxin³⁵ and overall mortality.⁴⁶

2. Valvular Heart Disease

In patients with mitral regurgitation, the S₃ is a poor predictor of elevated filling pressure (LR not significant) and depressed ejection fraction (LR = 1.9).⁴⁷ Some studies correlate the sound with severity of mitral regurgitation;²⁰ whereas others do not.⁴⁷

In contrast, the S₃ is a helpful finding in patients with aortic valve disease. In patients with aortic stenosis, the S₃ detects both elevated filling pressures (LR = 2.3 for pulmonary capillary wedge pressures ≥12 mm Hg) and depressed ejection fraction (LR = 5.7 for ejection fraction <50%).⁴⁷ In patients with aortic regurgitation, the S₃ detects both severity of regurgitation (LR = 5.9 for regurgitant fraction ≥40%; see Chapter 43) and ejection fraction of less than 50% (LR = 8.3).²⁰

3. Patients with Acute Chest Pain

In patients with acute chest pain presenting to emergency departments, the finding of an S₃ increases the probability of myocardial infarction (LR = 3.2).

4. Preoperative Consultation

During preoperative consultation, the finding of S₃ is ominous, indicating that the patient, without any other intervention, has an increased risk of perioperative pulmonary edema (LR = 14.6) and myocardial infarction or cardiac death (LR = 8).³⁰

B. FOURTH HEART SOUND

The finding of the S₄ gallop has less diagnostic value, simply because the disorders causing stiff ventricles are so diverse and because the S₄ does not predict the patient's hemodynamic findings. The finding does not predict

<table>
<thead>
<tr>
<th>Finding (Reference)</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Likelihood Ratio if Finding Is Present</th>
<th>Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Third Heart Sound</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Detecting ejection fraction &lt;0.5⁵⁰ ³⁶ ³⁷</td>
<td>11-51</td>
<td>85-98</td>
<td>3.4</td>
<td>0.7</td>
</tr>
<tr>
<td>Detecting ejection fraction &lt;0.3³⁶ ³⁷ ³⁹</td>
<td>68-78</td>
<td>80-88</td>
<td>4.1</td>
<td>0.3</td>
</tr>
<tr>
<td>Detecting elevated left heart filling pressures³¹ ³³</td>
<td>12-37</td>
<td>85-96</td>
<td>3.9</td>
<td>0.8</td>
</tr>
<tr>
<td>Detecting elevated BNP level³³ ³⁶</td>
<td>41-65</td>
<td>93-97</td>
<td>10.1</td>
<td>0.5</td>
</tr>
<tr>
<td>Detecting myocardial infarction in patients with acute chest pain³³ ³⁶ ³⁹</td>
<td>16</td>
<td>95</td>
<td>3.2</td>
<td>NS</td>
</tr>
<tr>
<td>Predicting postoperative pulmonary edema³³ ³⁹ ³⁰</td>
<td>17</td>
<td>99</td>
<td>14.6</td>
<td>NS</td>
</tr>
<tr>
<td>Predicting postoperative myocardial infarction or cardiac death³³ ³⁹ ³⁰</td>
<td>11</td>
<td>99</td>
<td>8.0</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Fourth Heart Sound</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Predicting 5-year mortality rate in patients after myocardial infarction³⁰</td>
<td>29</td>
<td>91</td>
<td>3.2</td>
<td>NS</td>
</tr>
<tr>
<td>Detecting elevated left heart filling pressures³¹ ³³ ³⁴</td>
<td>35-71</td>
<td>50-70</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Detecting severe aortic stenosis³³ ³⁴</td>
<td>29-50</td>
<td>57-63</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Diagnostic standards: For ejection fraction, left ventricular ejection fraction <0.5 or <0.3 (as indicated above) by scintigraphy or echocardiogram (see Chapter 46); for elevated left heart filling pressures, pulmonary capillary wedge pressure ≥12 mm Hg⁵¹ ³³ ³⁴ ³⁰; for elevated BNP level, ≥100 pg/ml³³ ³⁴ or ≥1525 pg/ml³³ ³⁴, for myocardial infarction, development of new electrocardiographic Q waves or elevations of CK-MB, or both, for severe aortic stenosis, peak gradient ≥50 mm Hg³² or valve area <0.75 cm³¹ ³⁴.

Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.

BNP, B-type natriuretic peptide; NS, not significant.
the ejection fraction, left heart filling pressures, or postoperative cardiac complications. It also does not predict significant aortic stenosis in elderly patients with aortic flow murmurs, presumably because many patients with mild stenosis have the finding for other reasons, such as ischemic heart disease.

When $S_4$ is detected 1 month after myocardial infarction, it is, nonetheless, a modest predictor of the 5-year cardiac mortality rate (LR = 3.2; see EBM Box 39-1). Experienced auscultators in the past did show that clinical deterioration in patients with ischemic disease caused the $S_4$-$S_1$ interval to widen, which could be recognized at the bedside, but proper interpretation of this finding required knowledge of the patient's PR interval, thus limiting its utility. In patients with chaotic heart rhythms, the finding of an $S_4$ excludes atrial fibrillation and suggests other diagnoses such as multifocal atrial tachycardia.

The $S_4$ is rare in patients with chronic mitral regurgitation because the dilated atrium of these patients cannot contract strongly. Finding an $S_4$ gallop in a patient with mitral regurgitation is therefore an important clue to the diagnosis of acute mitral regurgitation (e.g., ruptured chordae tendinae; see Chapter 44).

The references for this chapter can be found on www.expertconsult.com.
II. CLINICAL SIGNIFICANCE

The presence of the characteristic click or murmur alone is sufficient grounds for the diagnosis of mitral valve prolapse. Chapter 44 discusses these findings further.

OPENING SNAP

I. THE FINDING AND PATHOGENESIS

The opening snap is an early diastolic sound heard in patients with mitral stenosis. The sound occurs because the stenotic mitral leaflets (although fused, they are mobile) billow like a large sail into the ventricle during early diastole but then abruptly decelerate as they meet the limits of movement. The abrupt deceleration causes a loud, medium-frequency to high-frequency sound, which is then followed by the mid-diastolic rumbling murmur of mitral stenosis. The opening snap is best heard between the apex and left lower sternal border.

The clinician can mimic the sound of snap and murmur together by first setting up the cadence of $S_1$, $S_2$, and opening snap (RUP = $S_1$, bu = $S_2$, DUP = opening snap):

\[
\text{RUP bu DUP RUP bu DUP RUP bu DUP}
\]

and then adding the murmur:

\[
\text{RUP bu DUPSSSRRRRRUP bu DUPSSSRRRRRUP bu DUP}
\]

In some patients, the opening snap is so loud it is easily heard at the second left intercostal space, where it then mimics a widely split $S_2$. Careful attention to inspiration in these patients, however, may reveal a triple sound (split $S_2$ and opening snap) at this location, confirming the last sound to be the opening snap.

The opening snap of mitral stenosis was first described by Bouillard in 1835.

II. CLINICAL SIGNIFICANCE

According to traditional teachings, the opening snap is inaudible in patients with mitral stenosis whose valve leaflets have become so thickened and inflexible they cannot create sound. There is an inverse correlation between the opening snap amplitude and degree of calcification of the mitral valve ($r = -0.675$, $p < .01$). The interval between the $A_2$ component of $S_3$ and the opening snap ($A_2$-OS interval) has been used to gauge the severity of mitral stenosis. Patients with more severe obstruction tend to have a narrower $A_2$-OS interval than

*Patients with tricuspid stenosis also may have an opening snap, but all of these patients also have mitral stenosis and the mitral opening snap. Differentiating tricuspid and mitral opening snaps by auscultation alone is difficult.
those with milder disease. This occurs because the mitral valve opens when the pressure in the relaxing ventricle falls below the atrial pressure; the more severe the obstruction, the higher the atrial pressure and the sooner this crossover occurs. Nonetheless, determining the A2-OS interval is primarily a phonocardiographic exercise, not an auscultatory one. Furthermore, the A2-OS interval also depends on variables other than severity of stenosis, such as ventricular relaxation time and heart rate, further complicating the accurate interpretation of the interval at the bedside. The opening snap does indicate that the accompanying diastolic murmur represents mitral stenosis and not a flow rumble from increased flow over a nonstenotic valve. (See Chapter 44 for discussion of flow rumbles.)

PERICARDIAL KNOCK

The pericardial knock is a loud early diastolic sound heard in 28% to 94% of patients with constrictive pericarditis (see Chapter 45). It is heard over a wide area between the apex and the left lower sternal border. Compared with the third heart sound, the pericardial knock is a higher-frequency sound (easily detected with the diaphragm of the stethoscope), appears over a wider area of the precordium, and occurs slightly earlier (although still later than the opening snap or widely split second heart sound).

The pericardial knock results from the sudden deceleration of the filling ventricle as it meets the borders of the rigid pericardial sac. In this way, it is similar to the third heart sound, although the more abrupt deceleration of constriction is what probably makes the pericardial knock higher-pitched and louder than the third heart sound (see Chapter 39).

TUMOR PLOP

The tumor plop is an early diastolic sound representing prolapse of the pedunculated tumor from the atrium over the mitral (or tricuspid) valve into the ventricle. In two large series of patients with myxoma (283 patients), it was detected in 15% to 50% of patients. Characteristically, the intensity and timing of the tumor plop vary between examinations. The plop may occur as early as the timing of an opening snap or as late as that of the third heart sound. It is often associated with a diastolic murmur that mimics the rumbling murmur of mitral stenosis.

PROSTHETIC HEART SOUNDS

1. INTRODUCTION

Abnormal prosthetic heart sounds may be the only clue explaining the patient's dyspnea, syncope, or chest pain. To recognize these abnormal sounds simply and quickly, the clinician must first understand the normal prosthetic heart sounds. This section focuses on rigid mechanical valves, such as caged-ball (Starr-Edward) valves, single tilting-disc (Bjork-Shiley, Medtronic-Hall) valves, and bileaflet tilting-disc (St. Jude Medical) valves.

II. PRINCIPLES

The important observations are

1. Timing and intensity of opening and closing sounds, which typically have a clicking or metallic quality and are often audible without a stethoscope

2. Associated murmurs

Any new or changing sound or murmur requires investigation.

A. OPENING AND CLOSING SOUNDS

In patients with caged-ball valves, the opening sound is louder than the closing sound. In patients with tilting-disc valves (both single-disc and bileaflet valves), the closing sounds are loud and the opening sounds are only faint or inaudible (Fig. 40-2).

1. Caged-Ball Valves

In the aortic position, the caged-ball valve produces a loud opening sound, which is an extra systolic sound occurring just after S1 with timing identical to the aortic ejection sound (i.e., instead of S1 and S2, lub dup... lub dup, the clinician hears ledup dup... ledup dup). Caged-ball valves in the mitral position produce an extra diastolic sound when they open, with timing identical to that of the opening snap (i.e., instead of S1 and S2, lub dup... lub lub, it is lub lubdup... lub lubdup). These opening sounds should always

---

FIGURE 40-2 Prosthetic valve sounds. The normal findings of prosthetic valves, based on references 22 to 24. AC: Closure sound of aortic prosthesis; AO: opening sound of aortic prosthesis; MC: closure sound of mitral prosthesis; MO: opening sound of mitral prosthesis; P2: pulmonary component of second heart sound; S1, first heart sound; S2, second heart sound. See text.