

(see chapter 37, Table 37.5). This system is a fairly subjective measure of disability but more realistically estimates a patient's functional improvement or decline

over time and also the effects of therapy. A similar system, the Canadian Cardiovascular Society Criteria, rates disability from angina (Table 30.3).

## CHAPTER 31 CARDIOVASCULAR EXAMINATION

### Arterial Pulse

Arterial pulse (Figure 31.1) is best evaluated by palpating the brachial or, less preferably, the radial artery to determine the rate and rhythm. All accessible arterial pulses should be felt and compared bilaterally; normal arterial pulses are symmetric. As the closest accessible arteries to the aortic valve, the carotids should be palpated, one at a time, for assessing pulse volume and contour, which accurately reflect LV ejection velocity, stroke volume, and aortic valve function. The normal carotid, felt as a gentle tap, has a rapid upstroke. Distal

pulses, with their greater amplitude and velocity of flow, can mask diagnostic clues.

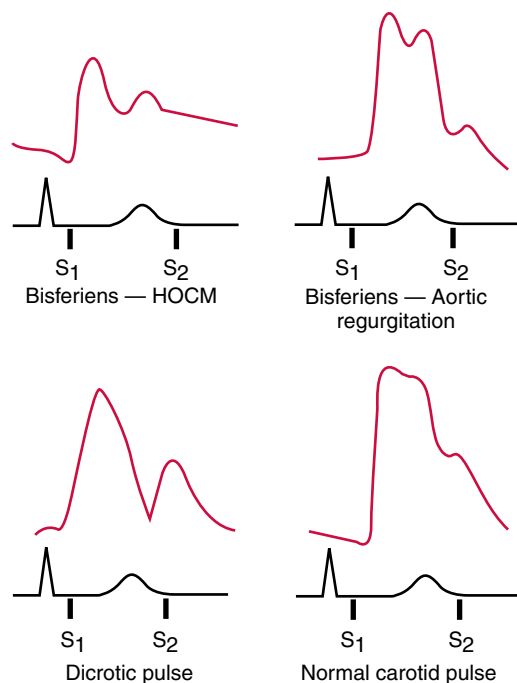
A **high-volume (amplitude) pulse**, or bounding pulse, corresponds to increased pulse pressure (systolic minus diastolic pressure) that follows rapid ejection of a large stroke volume into the aorta against a low systemic vascular resistance. It occurs in hyperkinetic states (anxiety, fever, thyrotoxicosis, anemia), aortic regurgitation, patent ductus arteriosus, arteriovenous fistula, bradycardia, and arteriosclerosis. A **small-volume pulse** corresponds to decreased pulse pressure that follows slow ejection of a normal or low stroke volume; it may also occur when the systemic vascular resistance is high. The pulse is weak and thready in left heart failure and shock. In severe aortic valvular stenosis, the pulse is typically low volume, with a slowly rising upstroke and a delayed peak (*pulsus parvus et tardus*; *parvus* means “slow rising” and *tardus* means “late”). In the elderly with aortic valve stenosis and hypertension or arteriosclerosis, these features may be absent.

In **pulsus bisferiens** (*bis* means “twice” and *feriens* means “beating”), the pulse has two strong systolic peaks; the percussion and tidal waves occur before the diastolic wave. Pulsus bisferiens is seen in severe aortic regurgitation, aortic regurgitation with stenosis, and hypertrophic obstructive cardiomyopathy.

In a **dicrotic pulse**, the second palpable peak occurs in diastole and is an exaggerated dicrotic wave following the dicrotic notch (aortic valve closure). Associated with a low cardiac output and low peripheral resistance, dicrotic pulse is typically seen in heart failure, hypovolemic shock, and young patients with fever. It is **rare** in patients older than 45 years.

Pulsus alternans indicates alternately strong and weak pulses, occurring at regular intervals or sometimes detected in the brachial or radial pulses by palpation, it usually requires a blood pressure cuff for detection (manifesting as a sudden doubling of rate as the blood pressure cuff is slowly deflated). Pulsus alternans is produced when high and low stroke volumes are ejected alternately from larger and smaller end-diastolic volumes of the LV. Its presence always indicates severe LV dysfunction.

Pulsus paradoxus is an excessive drop (>10 mm Hg) in systolic pressure during inspiration. In normal sub-



**FIGURE 31.1. Carotid pulse tracings. The bisferiens pulse contains two systolic peaks that occur in conditions with rapid ejection of blood through the aortic valve. In the dicrotic pulse, the second palpable wave is diastolic and is due to an exaggerated dicrotic wave following the dicrotic notch (aortic valve closure). A normal carotid pulse is shown for comparison. HOCM = hypertrophic (obstructive) cardiomyopathy.**

jects, systolic pressure (measured with a blood pressure cuff) falls 3–10 mm Hg during inspiration. When the paradox exceeds 20 mm Hg, it is usually specific for cardiac tamponade due to pericardial effusion. In pericardial tamponade, right ventricular (RV) filling increases during inspiration, causing a larger RV volume and a raised intrapericardial pressure; this lowers inspiratory LV filling and leads to a diminished inspiratory LV stroke volume and pulse amplitude. Pulsus paradoxus may be noted in other conditions such as acute severe asthma, severe cardiomegaly, marked obesity, and massive pulmonary embolism.

### Venous (Jugular) Pulse

#### Bedside evaluation

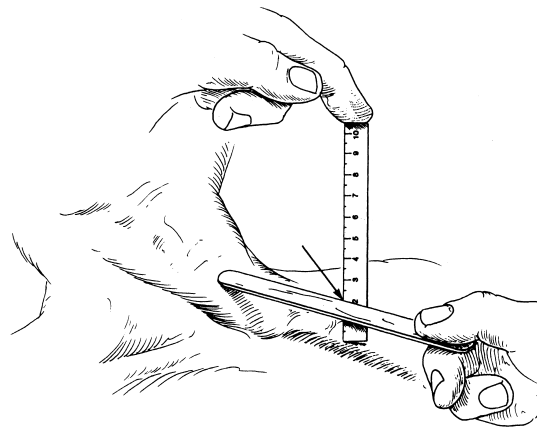
Bedside examination of the jugular venous pulse allows evaluation of the right atrial pressure and the morphology of the venous pulse waveforms. Examining the right internal jugular vein is preferred to the external jugular vein for pulse wave analysis because the right vein is in a direct line with the superior vena cava and, thus, better reflects right atrial events.

The patient should rest quietly in bed with his or her neck muscles relaxed and trunk elevated to a sufficient angle—usually 30–45 degrees—to allow the venous pulse to be visible. Venous pulses are often best seen with tangential lighting. To estimate jugular venous pressure, the vertical distance from the sternal angle to the top of the oscillating venous column is measured (Figure 31.2). This distance is ordinarily less than 3 cm. Because the sternal angle is approximately 5 cm above the mid-right atrium, regardless of body position, the jugular venous pressure would equal 5 cm plus 3 cm (the height of the oscillating venous column), or 8 cm H<sub>2</sub>O. This value can be converted to mm Hg by dividing it by 1.3 (1 mm Hg = 1.3 cm of blood); thus, the upper limit of normal jugular venous pressure is 6 mm Hg.

Jugular venous pressure reflects the mean right atrial or central venous pressure. The most common cause of an elevated right atrial pressure is **RV failure**. Other causes include tricuspid regurgitation and abnormalities of RV filling (pericardial tamponade, constrictive pericarditis, or rarely, tricuspid stenosis) (Figure 31.3). The normal jugular venous pressure decreases with inspiration and increases with expiration. In superior vena cava obstruction, the venous pressure is elevated but the venous waves and respiratory fluctuation are absent.

#### Hepatojugular reflux

In incipient or early right heart failure when the venous pressure is normal, a positive **hepatojugular reflux** may be elicited. When steady pressure is applied to the abdomen (liver) for 15–20 seconds (ensuring that the patient does not perform a Valsalva maneuver) in



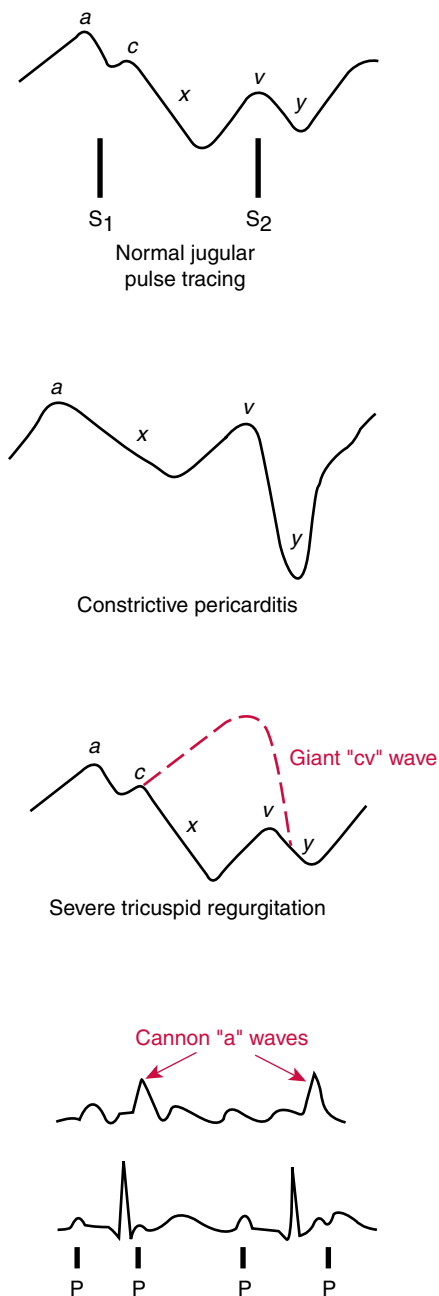
**FIGURE 31.2. Measuring the jugular venous pressure.** (From: Adair OV, Havranck EP (eds.). *Cardiology Secrets*. Philadelphia: Henley & Belfus, Inc., 1995, p. 17. Used with permission.)

normal individuals, the venous column rises transiently but returns to normal, despite continued abdominal compression. A positive hepatojugular reflux is one where the venous column increases more than 1 cm and remains high throughout the period of compression and gradually declines only after compression is relieved. The hepatojugular reflux occurs as the abnormal RV is unable to handle the increased venous return during abdominal compression.

#### Jugular venous pulse waves

The normal jugular venous pulse has two visible peaks (the *a* and *v* waves) and two troughs (the *x* and *y* descents) (see Figure 31.3). Another positive deflection after the *a* wave, the *c* wave, is not palpable but can be recorded; however, it is of little clinical importance.

- The *a wave* is produced by retrograde blood flow in the jugular veins caused by right atrial contraction. The *a* wave immediately precedes the carotid upstroke and the first heart sound, S<sub>1</sub>.
- The *x descent* reflects a fall in right atrial pressure that occurs with right atrial relaxation and the downward movement of the tricuspid valve ring owing to RV systole. The *x* descent is often the most easily detected motion in the jugular pulse; it occurs during systole and ends just prior to S<sub>2</sub>.
- The *v wave* results from venous inflow to the right atrium during ventricular systole while the tricuspid valve is closed. It occurs roughly coincident with the carotid pulse and the S<sub>2</sub>.
- The *y descent* results from the fall in atrial pressure that occurs as the tricuspid valve opens.



**FIGURE 31.3. Jugular venous pulse tracings. Normal jugular venous pressure**—The *a* wave and *x* descent are normally more prominent than the *v* wave and *y* descent. The *c* wave may be recorded but is not palpable on examination. **Constrictive pericarditis**—The steep *y* descent (diastolic ventricular filling) results from elevated venous pressures and rapid early diastolic filling. As the rigid pericardium suddenly limits further inflow, the pressure rapidly rises. **Severe tricuspid regurgitation**—A giant *cv* wave of tricuspid regurgitation is shown. **Cannon *a* waves** in complete heart block—Note the large *a* waves in the venous pulse, which occur due to atrial contraction against a closed tricuspid valve during ventricular systole.

Normally, the *a* wave is more visible than the *v* wave, and the *x* descent is more prominent than the *y* descent. In fact, the *v* wave and *y* descent are often not visible in healthy adults owing to the very compliant normal right atrium.

#### Abnormalities of jugular venous pulse waves

The *a* wave is increased in RV hypertrophy, tricuspid or pulmonic stenosis, or contraction of the atrium against a closed tricuspid valve (so-called cannon *a* wave, see Figure 31.3). Cannon waves occur regularly in junctional rhythm and irregularly in atrioventricular dissociation. The normal *a* wave is absent and the *x* descent less prominent in atrial fibrillation.

The *x* descent becomes deeper in pericardial tamponade and constrictive pericarditis. It is attenuated or disappears with tricuspid regurgitation (being replaced by a *cv* wave) and is attenuated in atrial fibrillation.

The classic cause of an enlarged *v* wave is tricuspid regurgitation. An enlarged *v* wave may also be seen in mitral regurgitation, but only when left atrial pressure recordings are made from a wedged pulmonary artery catheter.

A rapid, deep *y* descent is seen in tricuspid regurgitation, constrictive pericarditis, and severe right heart failure. The *y* descent becomes dominant in atrial fibrillation. A slow *y* descent suggests impeded RV filling and may be seen in tricuspid stenosis or pericardial tamponade.

#### Cyanosis

Cyanosis is a bluish discoloration of the skin and mucous membranes. It is conventionally divided into central and peripheral types. **Central cyanosis** reflects arterial desaturation and is recognized as a blue tinge of the tongue, conjunctivae, lips, nose, ears, and nail beds. It occurs in lung diseases, and congenital heart diseases with right-to-left shunt. In **peripheral cyanosis** the arterial oxygenation is normal; however, an increased extraction of oxygen peripherally by the tissues, due to sluggish blood flow, leads to an excess of reduced hemoglobin. Seen in the ears, nose, lips, nails, and fingertips but not in the tongue or conjunctivae, peripheral cyanosis occurs with low cardiac output states, exposure to cold, and arterial and venous obstruction.

The absolute requirement for cyanosis is only the presence of at least 5 g/dl of reduced hemoglobin in the capillary blood. Thus, in a person with a hemoglobin of 15 g/dl, it reflects an oxygen saturation of 66% or less. However, cyanosis in an anemic patient indicates severe desaturation of hemoglobin (e.g., 50% in a patient with a hemoglobin of 10 g/dl). For the same reasons, cyanosis may appear even with less ominous levels of desaturation in polycythemic states; smaller concentrations of meth-

moglobin and sulfmethemoglobin (1.5 and 0.5 g/dL, respectively) may also evoke central cyanosis.

**Palpation of the Precordium**

The **apical impulse** is located normally in the fourth or fifth left intercostal space, at or inside the midclavicular line. It can best be felt with the fingertips and is normally produced by the left ventricle. The apical impulse occupies a maximal area of 2–3 cm and should be located no more than 10 cm to the left of the midsternal line. If the apical impulse is not felt with the patient supine (which is common in patients older than 50), one should palpate for it in the left lateral position.

Besides location, one should note the duration, force, and contour of the apical impulse (Table 31.1). Normally, the apical impulse is felt as a brief, gentle tapping motion that ends before midsystole.

Simultaneous auscultation of S<sub>1</sub> and S<sub>2</sub> during precordial palpation allows one to estimate the duration of the apical impulse (Figure 31.4). A **sustained** apical impulse is one that maintains its peak into the second half of systole. It is caused by LV hypertrophy.

| Hyperkinetic              | Sustained                              |
|---------------------------|--|
| Hyperdynamic states       | Normal location (suggests normal LVEF) |
| Exercise                  | Hypertension                           |
| Excitement                | Aortic stenosis                        |
| Thyrotoxicosis            | HOCM                                   |
| Severe anemia             | Lateral displacement                   |
| Volume overload           | Chronic volume overload states         |
| Aortic regurgitation      | LV dilation with decreased LVEF        |
| Mitral regurgitation      |  |
| Ventricular septal defect |  |

LV = left ventricle; LVEF = left ventricular ejection fraction; HOCM = hypertrophic cardiomyopathy.

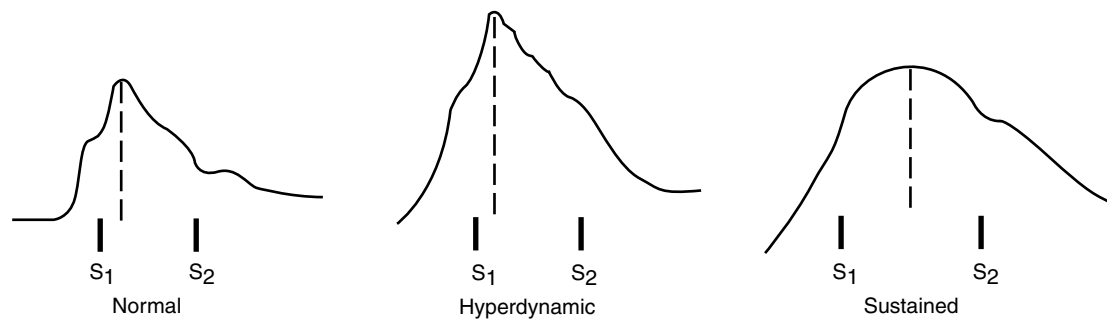
The force of the apical impulse, although subjective, should be determined. A normal impulse is felt as a gentle tap whereas a **hyperkinetic** impulse (exaggeration of the normal contour) may be found in volume overload and other hyperdynamic states.

Other palpable cardiovascular events and structures may be found on examination. Rapid LV distension during early and late diastole may produce distinctly visible and palpable impulses—corresponding in timing to ventricular (S<sub>3</sub>) and atrial (S<sub>4</sub>) gallops, respectively. A systolic bulge, medial to the apical impulse, can be produced by an LV aneurysm. An apical tap in mitral stenosis is a palpable first heart sound. A palpable S<sub>2</sub> in the second left interspace may occur in pulmonary hypertension. **Thrills** are merely murmurs loud enough to be palpable; these are usually low-frequency murmurs. A left parasternal pansystolic impulse (lift) indicates RV dilation and/or hypertrophy. Severe mitral regurgitation may be associated with a late systolic parasternal lift owing to expansion of the left atrium beneath the RV.

**Cardiac Auscultation**

Examination and auscultation are usually best done from the patient’s right side and ideally in quiet surroundings. A good-quality stethoscope, having both a diaphragm and bell, is essential. The diaphragm detects higher frequency sounds (>300 Hz) and is applied with moderate pressure. The bell detects lower frequency sounds (30–150 Hz) and is applied with the least pressure necessary to create a skin seal; excess pressure lessens the transmission of low-frequency sounds.

Auscultation should be done with the patient supine, but also when the patient is sitting and lying on the left side. At times, other positions (e.g., standing and squatting) and maneuvers (e.g., isometric exercise or Valsalva maneuver) that alter loading conditions may provide additional diagnostic information.



**FIGURE 31.4.** Major types of apical impulses. The **normal** apical impulse is felt as a brief tapping sensation, which clearly peaks before mid-systole (simultaneous auscultation of S<sub>1</sub> and S<sub>2</sub> is required). A **hyperdynamic** apical impulse

is an exaggerated form of a normal impulse contour; it is not sustained or late-peaking but simply more forceful. A **sustained** apical impulse is prolonged and late-peaking; the peak of the impulse may still be felt in the latter half of systole.

Four primary precordial areas for cardiac auscultation are as follows:

- Aortic area—the 2nd right and 3rd left intercostal spaces (ICS)
- Pulmonic area—the 2nd left ICS
- Tricuspid area—the 4th and 5th left ICS adjacent to the left sternal border
- Mitral area—the cardiac apex

Each physician should adopt a systematic method of listening, but it is imperative to focus attention on one auscultatory event in the cardiac cycle at a time. Many cardiac and noncardiac variables can affect the intensity of heart sounds and murmurs (Table 31.2).

**First heart sound (S<sub>1</sub>)**

The first heart sound, S<sub>1</sub>, signals the beginning of ventricular systole and is generated by mitral and

tricuspid valve closure (Figure 31.5). Although audible over the entire precordium, it is loudest over the apex and left lower sternal border. Its pitch, although relatively high, is lower than S<sub>2</sub>; therefore, it is heard best by using the diaphragm of the stethoscope.

The intensity of S<sub>1</sub> is determined primarily by valve mobility, force of ventricular contraction, and most important, the velocity of valve closure (Table 31.3). S<sub>1</sub> is louder when atrioventricular valves are widely separated at the onset of ventricular contraction, such as in mitral stenosis with pliable leaflets, a short PR interval, tachycardias, and increased diastolic flow rates. Alternatively, a soft S<sub>1</sub> occurs when the atrioventricular valves are partially closed at the onset of ventricular contraction, as with a long PR interval, acute aortic regurgitation, and decreased flow rates (low cardiac output). Finally, the S<sub>1</sub> may vary in intensity from beat to beat in atrial fibrillation, reflecting variable ventricular filling and thus contractility.

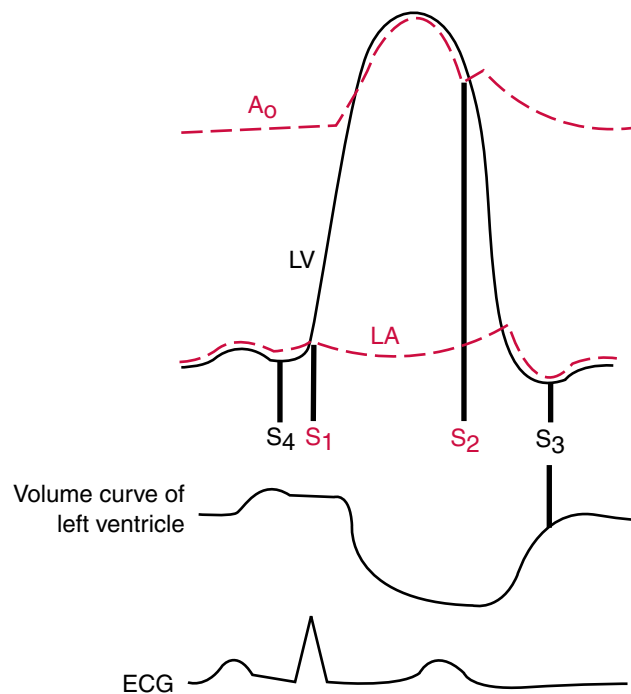
**Second heart sound (S<sub>2</sub>)**

Evaluation of S<sub>2</sub> is a key component of the cardiac physical examination. S<sub>2</sub> is best heard over the base of the heart, especially at the left upper sternal border. The closure of the aortic (A<sub>2</sub>) and pulmonic (P<sub>2</sub>) valves at end-systole generates S<sub>2</sub> (see Figure 31.5). Abnormalities of S<sub>2</sub> relate primarily to alterations in intensity or timing (Table 31.4).

A<sub>2</sub> is the louder component and is audible at all locations on the chest wall. In normal subjects, P<sub>2</sub> is heard

| TABLE 31.2. Conditions Affecting Intensity of Heart Sounds and Murmurs |                       |                     |
|--|-----------------------|---------------------|
|  | Noncardiac Conditions | Cardiac Conditions  |
| Decreased intensity  | Emphysema             | Low cardiac output  |
|  | Obesity               |                     |
|  | Muscular chest wall   |                     |
|  | Pericardial fibrosis  |                     |
| Increased Intensity  | Pericardial effusion  | Hyperdynamic states |
|  | Thin chest            |                     |
|  | Anemia                |                     |

**FIGURE 31.5.** The cardiac cycle and related hemodynamic events. The relationship of intracardiac pressures to the timing and sequence of heart sounds is shown. S<sub>4</sub> is a late diastolic event occurring after atrial contraction, and S<sub>3</sub> is an early diastolic event occurring during the rapid-filling phase of the left ventricular volume curve. A<sub>o</sub>=aortic pressure; LV=left ventricular pressure; LA=left atrial pressure.



**TABLE 31.3. Factors Affecting Intensity of First and Second Heart Sounds**

|           | S <sub>1</sub>  | S <sub>2</sub>   |                                       |
|-----------|---|--|---------------------------------------|
|           |   | A <sub>2</sub>   | P <sub>2</sub>                        |
| Increased | PR <160 ms<br>Mitral stenosis with pliable valve<br>Hyperdynamic states<br>Holosystolic MVP<br>Rapid heart rates  | Systemic HTN<br>Hyperdynamic states<br>Aortic dilation | Pulmonary HTN<br>Atrial septal defect |
| Decreased | PR >200 ms<br>Poor LV systolic function<br>Mitral stenosis with rigid valve<br>LBBB<br>Acute aortic regurgitation | Calcific aortic stenosis<br>Aortic regurgitation       | Pulmonic stenosis                     |

A<sub>2</sub> = aortic component of second heart sound; HTN = hypertension; LBBB = left bundle branch block; LV = left ventricle; MVP = mitral valve prolapse; PR = PR interval; P<sub>2</sub> = pulmonic component of second heart sound.

**TABLE 31.4. Alterations in the Second Heart Sound and Their Causes**

| Single S <sub>2</sub>               | Fixed Splitting     | Paradoxical Splitting  | Wide Splitting                                     |
|-------------------------------------|---------------------|------------------------|--|
| Aging                               | ASD                 | Complete LBBB          | Complete RBBB                                      |
| Severe AS                           | Right heart failure | RV pacing              | Atrial septal defect                               |
| Pulmonic stenosis                   |                     | Ischemic heart disease | Pulmonary HTN with right heart failure             |
| Any cause of delayed A <sub>2</sub> |                     | Aortic stenosis<br>HCM | LV pacing<br>Pulmonic stenosis<br>Severe MR<br>VSD |

AS = aortic stenosis; ASD = atrial septal defect; HCM = hypertrophic cardiomyopathy; HTN = hypertension; LBBB = left bundle branch block; LV = left ventricular; MR = mitral regurgitation; RBBB = right bundle branch block; RV = right ventricular; VSD = ventricular septal defect.

only at the upper left sternal border and is always less audible than A<sub>2</sub> at this location. An audible P<sub>2</sub> at the apex is an abnormal finding and strongly suggests pulmonary hypertension or atrial septal defect (apical impulse is caused by an enlarged RV).

A **single** S<sub>2</sub> results from attenuation of either A<sub>2</sub> or P<sub>2</sub>. A single S<sub>2</sub>, a common finding in older adults, arises from an inaudible P<sub>2</sub> caused by increased anteroposterior chest diameter.

The timing, or **splitting**, of S<sub>2</sub> varies with the phases of respiration. Normally, the split widens during inspiration and narrows during expiration. Slow, regular respirations are best for auscultating S<sub>2</sub>.

- Wide splitting of S<sub>2</sub> during expiration with further widening during inspiration, i.e., a widely split S<sub>2</sub> having normal respiratory variation occurs when P<sub>2</sub> is delayed (e.g., right bundle branch block) or with early A<sub>2</sub> (e.g., mitral regurgitation).
- Fixed splitting of S<sub>2</sub> occurs when the RV cannot augment its stroke volume (e.g., in right heart failure) or when respiration-induced changes in filling, and hence stroke volumes, are similar in both ventricles (e.g., in atrial septal defect).

- Paradoxical splitting of S<sub>2</sub> occurs typically in conditions that delay the onset of LV depolarization and, thus, LV ejection (e.g., in left bundle branch block) or those that delay aortic valve closure (e.g., in severe aortic stenosis). In paradoxical splitting of S<sub>2</sub>, A<sub>2</sub> follows P<sub>2</sub> during expiration and coincides with P<sub>2</sub> during inspiration.

**Diastolic sounds**

A **ventricular gallop** (S<sub>3</sub>) sound occurs in early diastole, 140–160 ms after S<sub>2</sub> as the active ventricular relaxation ends. It corresponds to the end of the rapid filling phase on the LV volume curve (see Figure 31.5). It is a low-frequency sound, best heard with the bell of the stethoscope lightly applied to the apex (LV S<sub>3</sub>) or left lower sternal border (RV S<sub>3</sub>). Often, gallops are heard only in the left lateral position.

The S<sub>3</sub> is caused by an interplay between ventricular filling and ventricular compliance (Table 31.5). An S<sub>3</sub> will be intensified by maneuvers that enhance ventricular filling and lessened by maneuvers that diminish venous return. Although an S<sub>3</sub> occurs in many conditions, most commonly, a nonphysiologic S<sub>3</sub> is associated with

**TABLE 31.5. Causes of Left Ventricular S<sub>3</sub> and S<sub>4</sub> Gallops**

|               | S <sub>3</sub>  | S <sub>4</sub>   |
|---------------|---|--|
| Physiological | Children and young adults<br>Common during pregnancy<br>Hyperkinetic states<br>Rare after age 40 years  | Rarely a normal finding  |
| Pathological  | High diastolic flow<br>Mitral regurgitation<br>Ventricular septal defect<br>Aortic regurgitation<br>Systolic dysfunction<br>Diastolic dysfunction<br>Hypertrophic cardiomyopathy<br>Restrictive cardiomyopathy<br>Constrictive pericarditis (knock) | Coronary artery disease<br>Hypertension<br>Aortic stenosis<br>Hypertrophic cardiomyopathy<br>Acute mitral regurgitation<br>Dilated cardiomyopathies<br>Hyperkinetic states |

abnormally high LV filling pressures, low cardiac output, and a dilated, poorly contractile LV. An S<sub>3</sub> is not heard when significant mitral stenosis is present. A “pericardial knock,” a higher pitched diastolic sound that occurs earlier (closer to A<sub>2</sub>) than the usual S<sub>3</sub>, is heard in patients with constrictive pericarditis.

**Atrial gallop (S<sub>4</sub>)** is a dull, low frequency sound that precedes S<sub>1</sub> and is best heard over the apical impulse (left-sided S<sub>4</sub>) or left lower sternal border (right-sided S<sub>4</sub>). The techniques used to elicit an S<sub>3</sub> sound also apply for the S<sub>4</sub>. The S<sub>4</sub> is attributed to forceful atrial contraction to fill a noncompliant or stiff ventricle (see Table 31.5). An S<sub>4</sub>, although abnormal, is quite common in older adults. It implies a less serious alteration of overall LV function and better prognosis than a pathologic S<sub>3</sub>. The S<sub>4</sub> disappears in atrial fibrillation.

A **summation gallop** occurs in the presence of tachycardia in a patient who has both an S<sub>3</sub> and an S<sub>4</sub>. During tachycardia, the shortened diastole forces the S<sub>4</sub> and S<sub>3</sub> into a loud, single diastolic sound.

**Mitral opening snap (OS)** is a sharp, high-frequency sound heard best over the left lower sternal border in patients with mitral valve stenosis. It is attributed to the sudden arrest of a rapidly opening mitral valve that is stenotic but pliable. Immobility or calcification of the mitral leaflets causes softening or disappearance of the OS. The A<sub>2</sub>-OS interval correlates inversely with the severity of the mitral stenosis; because left atrial pressure increases with the progression of severe mitral stenosis, the OS occurs earlier, resulting in a narrow A<sub>2</sub>-OS interval.

### Systolic sounds

**Ejection click (EC)** is a sharp, high-frequency sound audible immediately after S<sub>1</sub>. They occur in aortic and pulmonary valvular stenoses as well as in dilation of the ascending aorta and pulmonary artery. An aortic EC is audible over the entire precordium and varies little with respiration. A pulmonary EC is most audible

along the left upper sternal border, becoming louder during expiration and less audible or absent during inspiration. ECs occur owing to abrupt cessation of the systolic motion of the dome-shaped, stenotic aortic and pulmonary valves. As the valves become stiff and calcified in aortic and pulmonary valvular stenosis, the EC disappears.

Nonejection clicks are associated with mitral valve prolapse. They are high-frequency sharp clicks occurring over the apex or left lower sternal border. These may occur as isolated findings or be followed by late systolic murmurs. Maneuvers such as squatting and gripping one’s hand move a midsystolic click toward S<sub>2</sub> whereas standing and performing the Valsalva maneuver move the click toward S<sub>1</sub>. A midsystolic click arises from the sudden tension on the chordae tendineae or from the sudden halt of the prolapsing mitral valve leaflet during ventricular systole.

### Prosthetic valve sounds

A ball-in-cage prosthesis (Starr-Edwards) has loud, metallic opening and closing sounds. Disc valves produce distinct closing sounds but usually no audible opening sounds. Porcine valves in the aortic position usually generate no abnormal sounds, but when placed in the mitral position, they may produce an opening snap followed by a diastolic rumble.

A pressure gradient exists across all prosthetic valves because the valve area of a prosthesis is typically smaller than that of a normal native valve. This causes a barely audible systolic murmur across prosthetic aortic valves and a soft diastolic rumble across prosthetic mitral valves.

### Heart murmurs

A prolonged series of audible vibrations constitute a heart murmur. Heart murmurs are traditionally classified as systolic, diastolic, or continuous (Table 31.6). Dur-



ing auscultation of a murmur, its timing in the cardiac cycle in relation to  $S_1$  and  $S_2$ , intensity, quality (e.g., blowing, harsh, rumbling), duration, and radiation (e.g., to the neck, axilla, or back) should be defined. Additionally, if a patient with a systolic murmur has a coexistent arrhythmia, the effect of varying diastolic filling periods on the intensity of the murmur should be noted. Commonly, the intensity of outflow murmurs (e.g., aortic sclerosis or stenosis) will clearly increase in intensity in the beat following a longer diastole whereas murmurs owing to mitral regurgitation will remain unchanged.

The grading system for murmur intensity is described in Table 31.7. Most innocent murmurs are grades I or II. With a grade III murmur, one should initiate a search for pathology. Grade IV to VI murmurs are uncommon to rare. Whereas some systolic murmurs may be innocent, all diastolic and continuous murmurs are abnormal and pathological.

*Systolic murmurs*

Systolic murmurs are categorized according to their duration and relationship to  $S_1$  and  $S_2$ . The most important point to ascertain is whether the systolic

TABLE 31.7. Grading System for Murmur Intensity

| Grade  | Description   |
|--------|---|
| I/VI   | Very faint; barely audible  |
| II/VI  | Soft but readily audible  |
| III/VI | Moderately loud; no thrill  |
| IV/VI  | Very loud; thrill present   |
| V/VI   | Louder; thrill present; still requires a stethoscope on the chest to be heard |
| VI/VI  | Audible with stethoscope close to, but not touching, chest; thrill present    |

murmur extends to  $S_2$ . Ejection murmurs usually are crescendo-decrescendo in shape and end prior to  $S_2$ . Regurgitant or pansystolic murmurs are usually of more uniform intensity and extend to or even through  $S_2$ .

A midsystolic **ejection** murmur occupies only the ejection portion of systole. Ejection (and thus the murmur) begins at the time of semilunar valve opening, following  $S_1$ . Because the pressure gradient and blood flow markedly diminish before semilunar valve closure ( $S_2$ ), these murmurs usually end prior to  $S_2$ . These murmurs are classically diamond-shaped (crescendo-decrescendo). However, determining whether a systolic murmur extends to  $S_2$  is more important than determining its shape.

The most common type of midsystolic ejection murmur is the flow murmur, which arises owing to the normal turbulence of aortic blood flow during systolic ejection. Most pathologic midsystolic ejection murmurs originate at the semilunar valve; the classic example is aortic stenosis (Figure 31.6).

**Regurgitant or pansystolic** murmurs begin with  $S_1$  and extend to  $S_2$ . A truly pansystolic murmur implies a large and continuous pressure differential between two chambers, causing a high-velocity stream of flow throughout systole. The resulting murmur is classically high-frequency, blowing in quality (not harsh), and relatively uniform in intensity (Figure 31.7). All pansystolic murmurs are pathologic.

**Late systolic** murmurs occur in the latter part of systole, well after  $S_1$  and end in or after  $A_2$ . If caused by mitral valve prolapse, a midsystolic click may precede the murmur. All late systolic murmurs are pathologic.

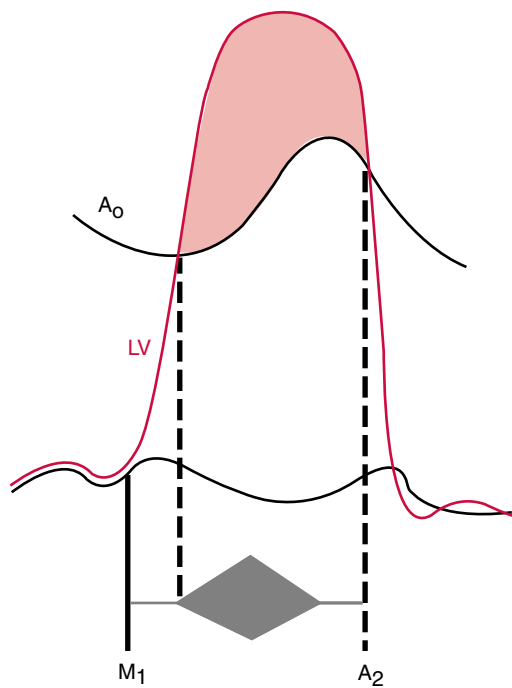
*Diastolic murmurs*

**Early diastolic** regurgitant murmurs are heard with aortic and pulmonic regurgitation. Because the pressure difference and thus regurgitant flow between the aorta and left ventricle (or pulmonary artery and right ventricle) decrease throughout diastole, these murmurs are classically decrescendo (Figure 31.8). Unless it is specifically sought, the murmur of aortic regurgitation can be easily missed on auscultation. It is best heard with

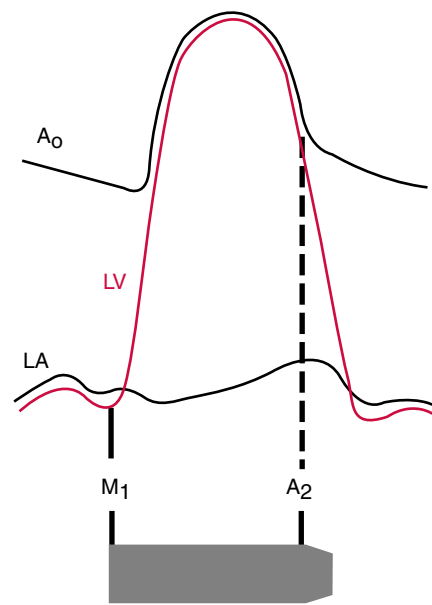
TABLE 31.6. Classification and Causes of Heart Murmurs

|   |
|---|
| INNOCENT MURMURS  |
| SYSTOLIC MURMURS  |
| Ejection (midsystolic; crescendo–decrescendo)   |
| Subvalvular, valvular, and supra- valvular aortic or pulmonic stenosis  |
| Malformed but nonstenotic aortic valve (aortic sclerosis)   |
| Dilation of aortic or pulmonary artery  |
| Increased systolic flow—e.g., aortic regurgitation, atrial septal defect  |
| Regurgitant (pansystolic; constant amplitude)   |
| Mitral and tricuspid regurgitation  |
| Ventricular septal defect   |
| Late systolic (onset well after $S_1$ ; end in $A_2$ )  |
| Mitral regurgitation—papillary muscle dysfunction or mitral valve prolapse  |
| DIASTOLIC MURMURS   |
| EARLY (onset with $A_2$ or $P_2$ ; decrescendo; high-pitched)   |
| Aortic and pulmonic regurgitation   |
| MID-DIASTOLIC (begin after $S_2$ ; low-pitched rumble)  |
| Mitral or tricuspid stenosis  |
| Increased atrioventricular valve flow without stenosis—mitral and tricuspid regurgitation, atrial and ventricular septal defects, aortic regurgitation (Austin Flint) |
| CONTINUOUS MURMURS (systolic/early diastolic; peak late systole)  |
| Patent ductus arteriosus  |
| Ruptured aneurysm of sinus of Valsalva  |
| Coronary arteriovenous fistula  |



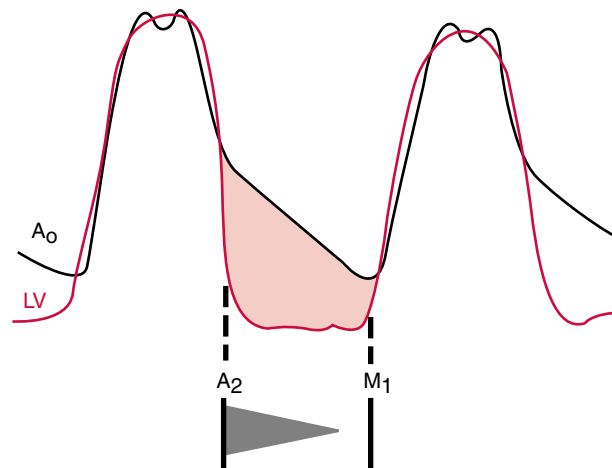


**FIGURE 31.6.** Midsystolic murmur in aortic stenosis. Left ventricular (LV) and aortic (Ao) pressure tracings are shown. The hatched area is the LV-Ao pressure gradient across the stenosis. Note the diamond-shaped midsystolic murmur begins after the mitral component of the first heart sound ( $M_1$ ), as the aortic valve is not yet open. The murmur ends before aortic valve closure ( $A_2$ ).



**FIGURE 31.7.** Pansystolic murmur in mitral regurgitation. Left ventricular (LV), aortic (Ao), and left atrial (LA) pressure tracings in typical mitral regurgitation are shown. The classic holosystolic murmur of mitral regurgitation begins with and may replace the first heart sound ( $M_1$ ). The murmur continues up to and even through the second heart sound ( $A_2$ ) since, at that time, left ventricular pressure still exceeds left atrial pressure and thus the pressure gradient causing the regurgitant flow continues to exist.

**FIGURE 31.8.** Early diastolic murmur in chronic aortic regurgitation. Left ventricular (LV) and aortic (Ao) pressure curves in chronic aortic regurgitation are shown. Note the low aortic diastolic pressure. The hatched area is the LV-Ao diastolic pressure gradient driving the AR flow. The murmur of aortic regurgitation begins with the second heart sound ( $A_2$ ). Since the gradient between the aorta and LV is maximal almost instantaneously and then slowly decreases, the murmur has a high-pitched, slow-decrescendo character.



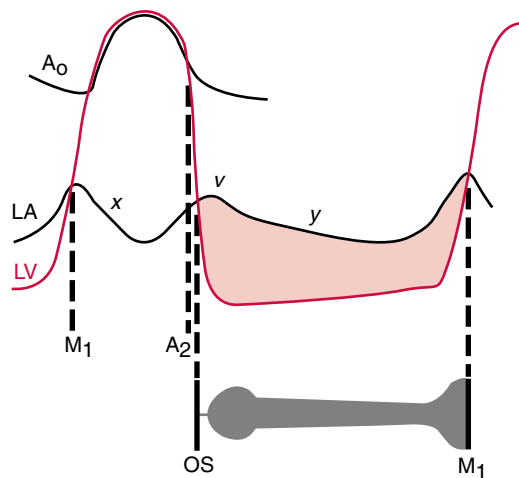
the patient sitting up and leaning forward, with breath held in expiration. The murmur of pulmonary regurgitation owing to pulmonary hypertension is termed a Graham Steell murmur.

**Mid-diastolic** murmurs are caused by increased diastolic flow across normal mitral and tricuspid valves

or normal diastolic flow across stenosed or distorted mitral and tricuspid valves. Classically, the murmur of mitral stenosis follows the opening snap of the mitral valve and then diminishes in intensity, only to increase again at the end of diastole (presystolic accentuation, Figure 31.9). These murmurs are of low frequency, heard

best with the bell of the stethoscope with the patient in the left lateral position. They too can be easily missed unless carefully sought.

A diastolic rumbling murmur may also be heard in patients with severe aortic regurgitation (Austin Flint



**FIGURE 31.9.** Mid-diastolic murmur in mitral stenosis. Left ventricular (LV), left atrial (LA), and aortic (Ao) pressure curves in mitral stenosis are shown. Note the elevated LA pressure. The pink-shaded area represents the LA-LV diastolic pressure gradient, showing the late diastolic rise in gradient associated with atrial contraction. The low-pitched diastolic rumble of mitral stenosis is immediately preceded by the high-pitched mitral opening snap (OS).

murmur). This murmur has been ascribed to vibrations of the anterior mitral leaflet sandwiched between the aortic regurgitant stream on one side and blood across the mitral orifice on the other.

*Continuous murmurs*

Continuous murmurs occur when a large and persistent pressure difference throughout the cardiac cycle exists. This may occur between two communicating chambers or vessels with no intervening valve or across a severely stenosed segment of an artery. Typically, a continuous murmur begins in systole and continues without interruption or change into diastole. These murmurs usually peak at S<sub>2</sub>.

*Pericardial friction rub*

A pericardial friction rub is a superficial scratching sound heard over the precordium in acute pericarditis. Faint rubs are best heard with the patient sitting up and leaning forward; they are notorious for their evanescence. Friction rubs may be triphasic with systolic, diastolic, and presystolic components. The systolic component is loudest and virtually always present. All three components are present in only about 50% of cases.

*Effects of physical maneuvers on murmurs*

Certain physical maneuvers can profoundly affect murmur intensity by acutely changing loading conditions (Table 31.8). These maneuvers can augment the intensity of soft murmurs or gallops and aid in the differential diagnosis of various murmurs.

| Maneuver                          | Technique  | Pathophysiology  | Effect   |
|-----------------------------------|--|--|--|
| Valsalva                          | Place hand on patient's abdomen and exert downward pressure as patient exerts outward pressure | During strain phase:<br>↓ venous return/<br>cardiac output;<br>↓ LV chamber size | ↓ Most heart sounds and murmurs except:<br>• ↑ in HCM<br>• MVP, earlier click and/or murmur  |
| Rapid standing                    | Abrupt assumption of upright posture   | Abrupt ↓ in venous return  | Augments in HCM and MVP<br>↓ in aortic stenosis, mitral and tricuspid insufficiency          |
| Respiration                       | Normal respiration   | Inspiration: ↑ venous return   | Inspiration ↑ right heart murmurs and gallops  |
| Isometric handgrip                | Sustained handgrip for 20–30 sec   | ↑ systemic resistance and arterial pressure                                      | Augments aortic and mitral insufficiency, ventricular septal defect<br>Decreases HCM and MVP |
| Post PVC or prolonged RR interval | Auscultate murmur in beat following a longer RR interval                                       | ↑ ventricular filling<br>↑ contractility   | Augments aortic stenosis<br>No change in mitral insufficiency                                |

HCM = hypertrophic cardiomyopathy; LV = left ventricle; MVP = mitral valve prolapse; PVC = premature ventricular contraction.