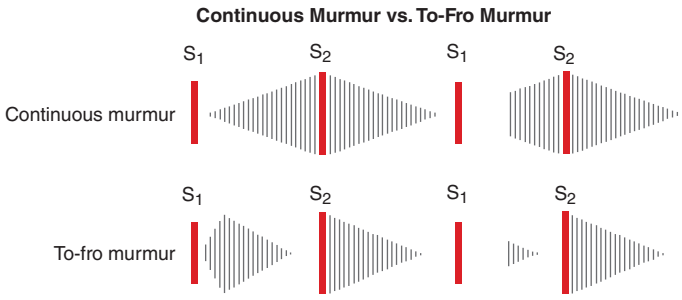

FIGURE 51e-7 Comparison of the continuous murmur and the to-fro murmur. During abnormal communication between high-pressure and low-pressure systems, a large pressure gradient exists throughout the cardiac cycle, producing a continuous murmur. A classic example is patent ductus arteriosus. At times, this type of murmur can be confused with a to-fro murmur, which is a combination of systolic ejection murmur and a murmur of semilunar valve incompetence. A classic example of a to-fro murmur is aortic stenosis and regurgitation. A continuous murmur crescendos to near the second heart sound (S_2), whereas a to-fro murmur has two components. The mid-systolic ejection component decrescendos and disappears as it approaches S_2. *(From JA Shaver, JJ Leonard, DF Leon: Examination of the Heart, Part IV, Auscultation of the Heart. Dallas, American Heart Association, 1990, p 55. Copyright, American Heart Association.)*

movement of the chest tends to obscure the heart sounds. Left-sided murmurs may be best heard at end expiration, when lung volumes are minimized and the heart and great vessels are brought closer to the chest wall. This phenomenon is characteristic of the murmur of AR. Murmurs of right-sided origin, such as tricuspid or pulmonic regurgitation, increase in intensity during inspiration. The intensity of left-sided murmurs either remains constant or decreases with inspiration.

Bedside assessment also should evaluate the behavior of S_2 with respiration and the dynamic relationship between the aortic and pulmonic components (Fig. 51e-8). Reversed splitting can be a feature of severe AS, HOCM, left bundle branch block, right ventricular pacing, or acute myocardial ischemia. Fixed splitting of S_2 in the presence of a grade 2 or 3 mid-systolic murmur at the mid- or upper left sternal border indicates an ASD. Physiologic but wide splitting during the respiratory cycle implies either premature aortic valve closure, as can occur with severe MR, or delayed pulmonic valve closure due to PS or right bundle branch block.

Alterations of Systemic Vascular Resistance Murmurs can change characteristics after maneuvers that alter systemic vascular resistance and left ventricular afterload. The systolic murmurs of MR and VSD become louder during sustained handgrip, simultaneous inflation of blood pressure cuffs on both upper extremities to pressures 20–40 mmHg above systolic pressure for 20 s, or infusion of a vasopressor agent. The murmurs associated with AS or HOCM will become softer or remain unchanged with these maneuvers. The diastolic murmur of AR becomes louder in response to interventions that raise systemic vascular resistance.

Opposite changes in systolic and diastolic murmurs may occur with the use of pharmacologic agents that lower systemic vascular resistance.

TABLE 51e-2 DYNAMIC AUSCULTATION: BEDSIDE MANEUVERS THAT CAN BE USED TO CHANGE THE INTENSITY OF CARDIAC MURMURS (SEE TEXT)

1. Respiration
2. Isometric exercise (handgrip)
3. Transient arterial occlusion
4. Pharmacologic manipulation of preload and/or afterload
5. Valsalva maneuver
6. Rapid standing/squatting
7. Passive leg raising
8. Post-premature beat

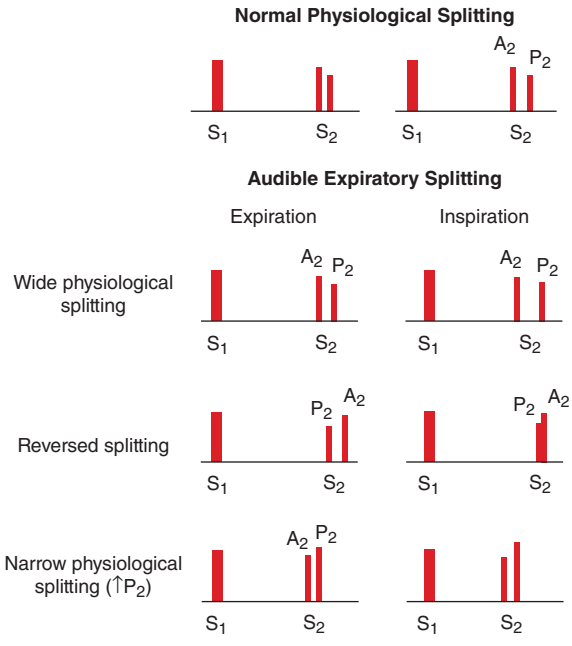

FIGURE 51e-8 ***Top.*** Normal physiologic splitting. During expiration, the aortic (A_2) and pulmonic (P_2) components of the second heart sound are separated by <30 ms and are appreciated as a single sound. During inspiration, the splitting interval widens, and A_2 and P_2 are clearly separated into two distinct sounds. ***Bottom.*** Audible expiratory splitting. Wide physiologic splitting is caused by a delay in P_2. Reversed splitting is caused by a delay in A_2, resulting in paradoxical movement; i.e., with inspiration P_2 moves toward A_2, and the splitting interval narrows. Narrow physiologic splitting occurs in pulmonary hypertension, and both A_2 and P_2 are heard during expiration at a narrow splitting interval because of the increased intensity and high-frequency composition of P_2. *(From JA Shaver, JJ Leonard, DF Leon: Examination of the Heart, Part IV, Auscultation of the Heart. Dallas, American Heart Association, 1990, p 17. Copyright, American Heart Association.)*

Inhaled amyl nitrite is now rarely used for this purpose but can help distinguish the murmur of AS or HOCM from that of either MR or VSD, if necessary. The former two murmurs increase in intensity, whereas the latter two become softer after exposure to amyl nitrite. As noted previously, the Austin Flint murmur of severe AR becomes softer, but the mid-diastolic rumble of MS becomes louder, in response to the abrupt lowering of systemic vascular resistance with amyl nitrite.

Changes in Venous Return The Valsalva maneuver results in an increase in intrathoracic pressure, followed by a decrease in venous return, ventricular filling, and cardiac output. The majority of murmurs decrease in intensity during the strain phase of the maneuver. Two notable exceptions are the murmurs associated with MVP and obstructive HOCM, both of which become louder during the Valsalva maneuver. The murmur of MVP may also become longer as leaflet prolapse occurs earlier in systole at smaller ventricular volumes. These murmurs behave in a similar and parallel fashion with standing. Both the click and the murmur of MVP move closer in timing to S_1 on rapid standing from a squatting position (Fig. 51e-3). The increase in the intensity of the murmur of HOCM is predicated on the augmentation of the dynamic left ventricular outflow tract gradient that occurs with reduced ventricular filling. Squatting results in abrupt increases in both venous return (preload) and left ventricular afterload that increase ventricular volume, changes that predictably cause a decrease in the intensity and duration of the murmurs associated with MVP and HOCM; the click and murmur of MVP move away from S_1 with squatting. Passive leg raising can be used to increase venous return in patients who are unable to squat and stand. This maneuver may lead to a decrease in the intensity of the murmur associated with HOCM but has less effect in patients with MVP.