However, it is more apparent in conditions that delay closure of the tricuspid valve, including right bundle branch block and Ebstein's anomaly (Audio Clip 3-1, Ebstein Abnormalities).

S_2 can be accentuated in the presence of hypertension, when the aortic component will be louder, or in pulmonary hypertension, when the pulmonic component will be enhanced. In the setting of severe aortic or pulmonic stenosis, leaflet excursion of the respective valves is reduced and the intensity of S_2 is significantly diminished. It may become absent altogether if the accompanying murmur obscures what remains of S_2.

There are several patterns of abnormal splitting of S_2. S_2 can remain single throughout respiration if either A_2 or P_2 is not present or if they occur simultaneously. A_2 can be absent, as previously mentioned, with severe aortic stenosis. P_2 can be absent with a number of congenital abnormalities of the pulmonic valve. Splitting may be persistent throughout the respiratory cycle if A_2 occurs early or if P_2 is delayed, as in the presence of right bundle branch block. In that case, splitting is always present but the interval between A_2 and P_2 varies somewhat. In fixed splitting, the interval between A_2 and P_2 is consistently wide and unaffected by respiration. This finding is observed in the presence of an ostium secundum atrial septal defect or right ventricular failure. Paradoxical splitting of S_2 occurs when P_2 precedes A_2. This leads to splitting with expiration and a single S_2 with inspiration. It is commonly found in situations of delayed electrical activation of the left ventricle, as in patients with left bundle branch block or right ventricular pacing. It can also be seen with prolonged mechanical contraction of the left ventricle, as in patients with aortic stenosis or hypertrophic cardiomyopathy.

TABLE 3-5 ABNORMAL INTENSITY OF HEART SOUNDS

	S_1	A_2	P_2
Loud	Short PR interval Mitral stenosis with pliable valve	Systemic hypertension Aortic dilation Coarctation of the aorta	Pulmonary hypertension Thin chest wall
Soft	Long PR interval Mitral regurgitation Poor left ventricular function Mitral stenosis with rigid valve Thick chest wall	Calcific aortic stenosis Aortic regurgitation	Valvular or subvalvular pulmonic stenosis
Varying	Atrial fibrillation Heart block	—	—

A_2, Component of second heart sound caused by closure of aortic valve; P_2, component of second heart sound caused by closure of pulmonic valve; S_1, first heart sound.

The third heart sound, S_3, is a low-pitched sound heard best at the apex in mid diastole. Because it is low pitched, it is best recognized with use of the bell on the stethoscope. As stated previously, S_3 can be physiologic in children but is pathologic in older individuals and often associated with underlying cardiac disease. An S_3 occurs during the rapid filling phase of diastole and is thought to indicate a sudden limitation of the expansion of the left ventricle. This can be seen in cases of volume overload or tachycardia. Maneuvers that increase venous return accentuate an S_3, whereas those that reduce venous return diminish the intensity. The fourth heart sound, S_4, is also a low-frequency sound, but in contrast to S_3, it is heard in late diastole, just before S_1. The S_4 gallop occurs as a result of active ejection of blood into a noncompliant left ventricle. Therefore, when atrial contraction is absent, such as in atrial fibrillation, an S_4 cannot be heard. This heart sound is also best recognized with the use of a bell at the apex. It can be heard in patients with left ventricular hypertrophy, acute myocardial infarction, or hyperdynamic left ventricle. At times, an S_3 and an S_4 can be heard in the same patient. In tachycardic states, the two sounds can fuse in mid diastole to form a summation gallop.

As stated earlier, S_3 and S_4 gallops are heard in mid diastole and late diastole, respectively. There are other abnormal sounds that can be heard during systole and early diastole. *Ejection sounds* are typically heard in early systole and involve the aortic and pulmonic valves. These are high-frequency sounds that can be heard with a diaphragm shortly after S_1. Ejection sounds are caused by the opening of abnormal valves to their full extent, such as with a bicuspid aortic valve or congenital pulmonic stenosis. They are frequently followed by a typical ejection murmur of aortic or pulmonic stenosis. Ejection sounds can also be heard with systemic or pulmonary hypertension, in which case the exact mechanism is not clear.

Midsystolic to late systolic sounds are called *ejection clicks*. They are most commonly associated with mitral valve prolapse. They are also high pitched and easily auscultated with the diaphragm. The click occurs because of maximal displacement of the prolapsed mitral leaflet into the left atrium and resultant tensing of chordae and redundant leaflets (Audio Clip 3-2, MVP). The click is usually followed by a typical murmur of mitral regurgitation. Any maneuver that decreases venous return will cause the click to occur earlier in systole, whereas increasing ventricular volume will delay the click (see Table 3-4).

The opening of abnormal mitral or tricuspid valves can be heard in early diastole. This *opening snap* is most frequently associated with rheumatic mitral stenosis. It is heard if the valve leaflets remain pliable and is generated when the leaflets abruptly

TABLE 3-6 ABNORMAL SPLITTING OF S_2

SINGLE S_2	WIDELY SPLIT S_2 WITH NORMAL RESPIRATORY VARIATION	FIXED SPLIT S_2	PARADOXICALLY SPLIT S_2
Pulmonic stenosis Systemic hypertension Coronary artery disease Any condition that can lead to paradoxical splitting of S_2	Right bundle branch block Left ventricular pacing Pulmonic stenosis Pulmonary embolism Idiopathic dilation of the pulmonary artery Mitral regurgitation Ventricular septal defect	Atrial septal defect Severe right ventricular dysfunction	Left bundle branch block Right ventricular pacing Angina, myocardial infarction Aortic stenosis Hypertrophic cardiomyopathy Aortic regurgitation

S_2, Second heart sound.