

# 28

## Ejection Clicks

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### Definition

*Ejection clicks* are high-pitched sounds that occur at the moment of maximal opening of the aortic or pulmonary valves. They are heard just after the first heart sound. The sounds occur in the presence of a dilated aorta or pulmonary artery or in the presence of a bicuspid or flexible stenotic aortic or pulmonary valve (Figure 28.1). Ejection clicks may also be called *ejection sounds*. The diastolic correlate of the ejection click is the opening snap, which occurs at maximal opening of a flexibly stenotic mitral or tricuspid valve.

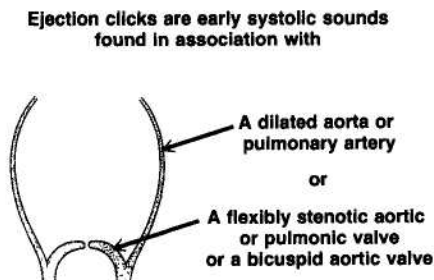
### Technique

Clicks are heard through the stethoscope during auscultation of the heart. Because of their high frequency, clicks are best heard with the diaphragm of the stethoscope. Aortic and pulmonary clicks are most prominent along the upper right and left sternal border. Mitral and tricuspid clicks are loudest along the lower left sternal border and at the apex. The first step in identifying a click is to distinguish it from normal heart sounds and determine its timing in the cardiac cycle. This is best accomplished by simultaneously auscultating the heart and palpating the carotid artery pulse to clearly identify the first ( $S_1$ ) and second ( $S_2$ ) heart sounds. Systolic clicks may be further characterized by their location in systole, that is, early, mid, or late systolic clicks.

Once the timing of the click is ascertained, its response to respiration, postural change, Valsalva maneuver, or various pharmacologic agents, such as amyl nitrite or phenylephrine, should be evaluated.

### Basic Science

Systolic clicks are classified as ejection or nonejection clicks. Systolic ejection clicks occur in early systole and may result from either the abrupt opening of the semilunar valves or the rapid distention of the proximal aorta or pulmonary



**Figure 28.1**  
The ejection click occurs at the moment of maximal opening of the valve.

artery at the onset of ejection. Systolic ejection clicks may be aortic or pulmonary in origin. The two are difficult to differentiate. In fact, it is frequently difficult even to separate a systolic ejection click from a split first heart sound or an atrial gallop sound ( $S_4$ ) followed closely by  $S_1$ .

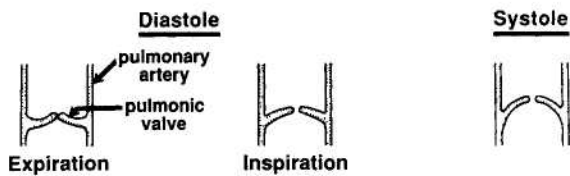
Aortic ejection clicks are usually best heard with the diaphragm in the second right intercostal space or at the apex, whereas pulmonary ejection clicks are maximal in the second left intercostal space and at the left sternal border. Pulmonary ejection clicks frequently decrease in intensity or merge with the first heart sound during inspiration, whereas aortic ejection clicks are not affected by respiration. Both aortic and pulmonary ejection clicks usually occur immediately before or coincident with the initial carotid upstroke; however, a significant delay in the onset of the click suggests a pulmonary origin.

Systolic nonejection clicks are most commonly produced by the mitral or tricuspid valve apparatus. These clicks usually occur in mid to late systole and appear to be related to tensing of the chordae tendineae or valve leaflets when mitral or tricuspid valve prolapse is present. These clicks are often multiple and are quite variable in their timing, being very responsive to changes in ventricular volume induced by posture or pharmacologic agents. Originally these nonejection clicks were thought to be extracardiac, but Barlow and subsequently others have clearly demonstrated their cardiac origin. Mitral and tricuspid clicks usually move to an earlier position in systole in response to factors that decrease left ventricular volume, such as standing or administration of amyl nitrite. They move to a later position in systole in response to maneuvers such as squatting or to agents, such as phenylephrine, which increase ventricular volume.

### Clinical Significance

When *right-sided ejection clicks* occur in the presence of a normal pulmonary valve, they are loudest at the upper left sternal border and are usually accompanied by an enlarged pulmonary artery on chest x-ray. Ejection clicks associated with a dilated pulmonary artery and normal pulmonary valve have no consistent respiratory variation. Their origin is unclear; the occurrence at the maximal opening of the pulmonary valve may be coincidental or may mean that there are changes in, or tensions on, the valve cusps or annulus related to the generation of the click.

Right-sided ejection clicks may also be heard in association with a flexibly stenotic pulmonary valve. These ejection clicks are also heard at the upper left sternal border; with mild or moderate stenosis, there is respiratory variation. In contrast to other right-sided acoustic events (e.g., the murmur of tricuspid regurgitation), the ejection click introducing the murmur of pulmonary stenosis becomes softer during inspiration. Pulmonary stenosis evokes hy-

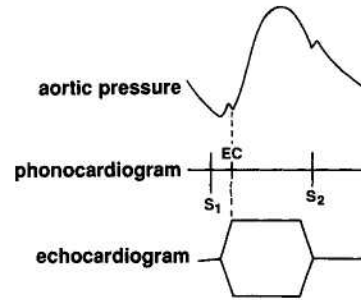


**Figure 28.2**  
Mechanism of inspiratory softening of the pulmonary ejection click.

pertrophy of the right ventricle, stiffening that chamber. When blood is drawn into the right ventricle during inspiration, the thickened right ventricle does not distend normally, so pressure in that chamber rises, and the pulmonary valve is partially opened. Because the pulmonary valve is partially open when the right ventricle contracts, its excursion is less, and the ejection click associated with its maximal opening is softer and earlier (Figure 28.2). With severe valvular pulmonary stenosis, the right ventricle may be so stiff and atrial contraction so vigorous that right atrial contraction actually opens the pulmonary valve completely and produces a click in late diastole. When a phonocardiogram is recorded along with the pulmonary arterial pressure tracing, the ejection click associated with pulmonary stenosis may be seen to correspond with a notch in the upstroke of the pulmonary arterial pressure tracing; this low-frequency mechanical event also occurs at the maximal opening of the pulmonary valve.

*Left-sided ejection clicks* occur in the presence of a dilated aorta or aortic valve abnormalities. When the aortic valve is normal, left-sided ejection clicks seem to be related to dilation of the aortic root, even though the ejection click occurs at maximal opening of the normal aortic valve. Ejection clicks associated with a dilated aortic root are best heard at the aortic area and are poorly transmitted to the apex. Aortic valvular ejection clicks, in contrast, are loudest at the apex. When there is aortic stenosis, the aortic ejection click just precedes the murmur. Ejection clicks occur with flexibly stenotic aortic valves and with nonstenotic bicuspid aortic valves. It may be difficult or impossible to distinguish an aortic ejection click from the tricuspid component of a widely split  $S_1$ . The tricuspid component of  $S_1$  is best heard at the left lower sternal border and becomes louder with inspiration. The combination of  $S_4$  and  $S_1$  may also be mistaken for an  $S_1$  ejection click. The  $S_4$  (atrial gallop) is best heard with the bell lightly applied to the cardiac apex and is associated with a presystolic apical impulse.

Like right-sided ejection clicks, the aortic ejection click may correspond to a notch in the upstroke of the aortic pressure tracing or the carotid pulse tracing (Figure 28.3). This notch apparently results from maximal opening and abrupt deceleration of the flexibly stenotic aortic valve. When the aortic valve is immobilized by heavy calcification, this sudden deceleration of cusp motion cannot occur, so there is no ejection sound; the aortic component of the second



**Figure 28.3**  
Timing of the aortic ejection click.

heart sound is soft or absent. When an ejection click is associated with a nonstenotic bicuspid aortic valve, the ejection click is loud and widely separated from the first heart sound.

Other causes of systolic clicks have been described: mitral valve prolapse, the pseudoejection sound of idiopathic hypertrophic subaortic stenosis, aortic dissection, and ventricular septal aneurysm. Correct identification of an ejection click prevents confusion with other heart sounds, localizes ventricular outflow obstruction to the valvular level, or suggests the presence of a bicuspid aortic valve or dilation of the aorta or pulmonary artery.

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