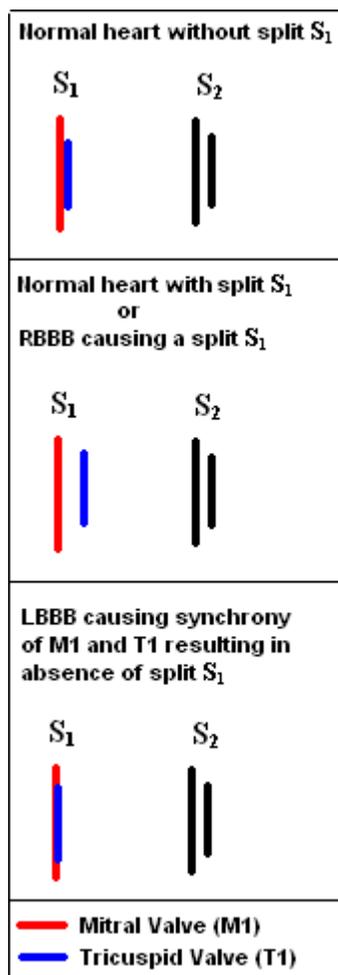


Heart Sounds

The first heart sound results from the closing of the mitral and tricuspid valves. The sound produced by the closure of the mitral valve is termed M1 and the sound produced by closure of the tricuspid valve is termed T1. The M1 sound is much louder than the T1 sound due to higher pressures in the left side of the heart, thus M1 radiated to all cardiac listening posts (loudest at the apex) and T1 is usually only heard at the left lower sternal border. The M1 sound is thus the main component of S1.

CLINICAL PEARL: A split S1 heart sound is best heard at the tricuspid listening post since T1 is much softer than M1.

The M1 sound occurs slightly before T1. Since the mitral and tricuspid valves normally close almost simultaneously, only one single heart sound is usually heard. However, in about 40% of normal individuals, as well as in certain cardiac conditions, a "split S1" sound can be appreciated. This occurs when the mitral valve closes *significantly* before the tricuspid valve allowing both valves to make an audible, separate sound. Inspiration delays the closure of the tricuspid valve in a normal person (due to increased venous return), thus enhancing the splitting of the S1 sound. Also, a split S1 sound is common in the setting of a right bundle branch block (RBBB). A RBBB causes the electrical impulse to reach the left ventricle before the right ventricle. Dysynchrony then occurs resulting in the left ventricle contracting before the right ventricle, thus the pressures in the left ventricle rises before that of the right ventricle. This increased pressure forces the mitral valve closed before the tricuspid valve resulting in a split S1 sound. A left bundle branch block (LBBB) has the opposite effect on S1. In this setting, the electrical impulse reaches the right ventricle before the left ventricle, thus the pressure in the right ventricle rises before that of the left ventricle. This forces the tricuspid valve closed earlier resulting in complete *overlap* of M1 and T1 and thus no audible split S1 sound.





CLINICAL PEARL: A RBBB causes a widened split S1 while a LBBB results in the absence of a split S1.

Three factors affect the intensity of the first heart sound. Since the M1 portion of S1 is much louder than T1, it is only important to discuss what affects the intensity of M1. The greater the distance separating the leaflets of the mitral valve at the beginning of systole, the louder the S1. This is affected by the duration of the PR interval on the EKG. Remember that the PR interval represents part of diastole, so a longer PR interval would result in a longer diastolic filling time. As the left ventricle fills, the pressure gradually increases. This gradual increase in pressure causes the mitral valve leaflets to slowly drift together. Therefore, when ventricular systole occurs in the setting of a long PR interval, the leaflets will be separated by a smaller distance and the S1 sound will be softer. The converse is also true. A short PR interval results in an accentuated S1 since the mitral valve leaflets will be further apart at the onset of ventricular systole.

CLINICAL PEARL: A short PR interval results in an accentuated S1 while a long PR interval results in a soft S1.

The mobility of the valve leaflets in the second factor influencing the intensity of M1. In mild to moderate mitral stenosis, the increased left atrial pressure causes the mobile portions of the mitral valve leaflets to be more widely separated, thus resulting in an *accentuated* M1 sound. In severe to critical mitral stenosis, the valve leaflets are so calcified and immobile that the M1 sound is diminished or absent.

CLINICAL PEARL: Mild to moderate mitral stenosis results in a loud S1 while severe to critical mitral stenosis results in a soft S1.

The rate of ventricular contraction also affects the intensity of S1. The faster the heart rate and the faster the rise in ventricular pressure, the louder the S1. Thus, high flow states such as anemia, thyrotoxicosis, or sepsis results in an accentuated S1. Also, during exercise or any other setting of tachycardia, the S1 will be accentuated.

The second heart sound (S2)

The second heart sound is produced by the closure of the aortic and pulmonic valves. The sound produced by the closure of the aortic valve is termed A2 and the sound produced by the closure of the pulmonic valve is termed P2. The A2 sound is normally much louder than the P2 due to higher pressures in the left side of the heart, thus A2 radiates to all cardiac listening posts (loudest at the *right* upper sternal border) and P2 is usually only heard at the *left* upper sternal border. The A2 sound is thus the main component of S2.

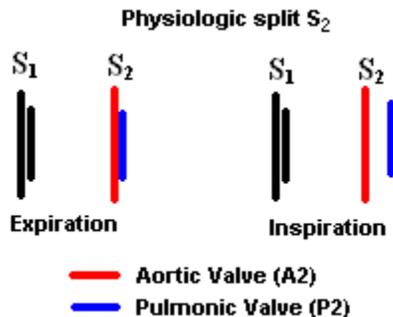
CLINICAL PEARL: A split S₂ is best heard at the pulmonic valve listening post since P₂ is much softer than A₂.

Like the S₁ heart sound, the S₂ sound is described regarding splitting and intensity. S₂ is physiologically split in about 90% of people. The S₂ heart sound can exhibit persistent (widened) splitting, fixed splitting, paradoxical (reversed) splitting, or the absence of splitting.

CLINICAL PEARL: In severe hypertension, a loud S₂ may be prolonged and slurred falsely mimicking a split S₂.

Physiologic split S₂

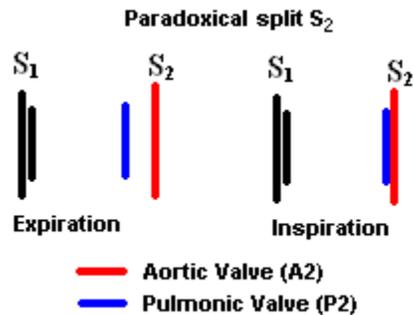
Normally, A₂ occurs just before P₂ and the combination of these sounds make up S₂. A physiologic split S₂ occurs when the A₂ sound precedes P₂ by a great enough distance to allow both sounds to be heard separately. This happens during inspiration when increased venous return to the right side of the heart delays the closure of the pulmonic valve (major effect) and decreased return to the left side of the heart hastens the closure of the aortic valve (minor effect), thus further separating A₂ and P₂. During expiration, the distance narrows and the split S₂ is no longer audible.



Paradoxical split S₂

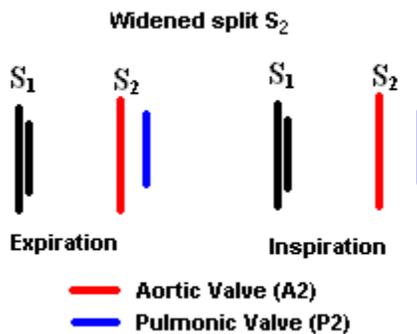
A paradoxical split S₂ heart sound occurs when the splitting is heard during *expiration* and disappears during inspiration, the opposite of the physiologic split S₂. A paradoxical split S₂ occurs in any setting that delays the closure of the aortic valve, such as severe aortic stenosis, hypertrophic obstructive cardiomyopathy (HOCM) or in the setting of a left bundle branch block (LBBB).

CLINICAL PEARL: A paradoxical split S₂ is heard in AS, HOCM, or in the presence of a LBBB.



Persistent (widened) split S₂

Persistent (widened) splitting occurs when both A2 and P2 are audible (split) during the *entire respiratory cycle*, however the *splitting becomes greater with inspiration* (due to increased venous return) and less prominent with expiration. This differs from a fixed split S₂ which exhibits the *same amount* of splitting throughout the entire respiratory cycle (see below).

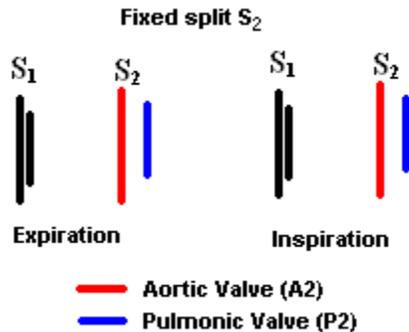


Any condition that causes a non-fixed delay in the closure of the pulmonic valve or early closure of the aortic valve will result in a wide split S₂. Thus, a persistently split S₂ occurs in the setting of a RBBB or severe mitral regurgitation. A RBBB causes a delay in the closure of the pulmonic valve and this a delay in P2 without any effect on A2. In severe mitral regurgitation (MR), the A2 occurs early secondary to a large proportion of the left ventricular stroke volume entering the left atrium, thus causing the left ventricular pressure to decrease faster. The P2 is not affected in severe MR.

CLINICAL PEARL: A persistently (widened) split S₂ occurs in the setting of a RBBB or severe MR.

Fixed split S₂

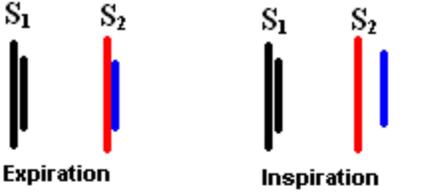
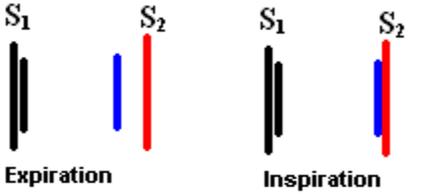
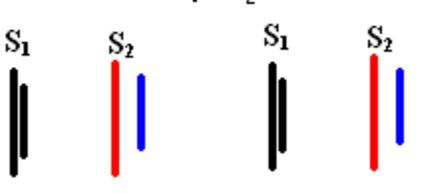
A fixed split S₂ is a rare finding on cardiac exam, however, when found it almost always indicated the presence of an atrial septal defect (ASD). A fixed split S₂ occurs when there is always a delay in the closure of the pulmonic valve and there is *no further delay* with inspiration (compare this to a widened split S₂ as described above).



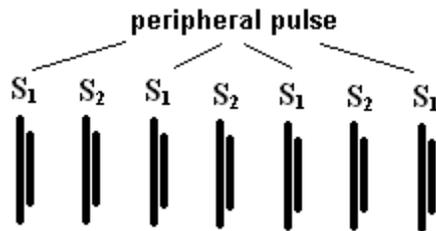
To explore why an ASD results in a fixed split S₂, we must consider the altered cardiac hemodynamics present in this situation which result in a fixed delay in PV closure. During inspiration, as usual there is an increase in venous return to the right side of the heart and thus increased flow through the PV delaying its closure. Where the alteration occurs in a person with an ASD is during expiration. As the person expires, the pressure in the right atrium decreases (since there is less venous return). The decreased pressure allows more blood to flow abnormally through the ASD from the high pressured left atrium to the right atrium ultimately resulting again in increased flow through the pulmonic valve again delaying its closure.

CLINICAL PEARL: A fixed split S₂ is pathognomonic for the presence of an atrial septal defect (ASD).

Now compare all of the splitting patterns of S2

Splitting pattern of S2	<u>Etiology</u>
<p>Physiologic split S₂</p>  <p>Expiration Inspiration</p>	<p>Normal</p>
<p>Paradoxical split S₂</p>  <p>Expiration Inspiration</p>	<p>Aortic stenosis HOCM LBBB</p>
<p>Widened split S₂</p>  <p>Expiration Inspiration</p>	<p>RBBB Severe MR Pulmonic stenosis</p>
<p>Fixed split S₂</p>  <p>Expiration Inspiration</p>	<p>Atrial septal defect</p>
<p>— Aortic Valve (A2) — Pulmonic Valve (P2)</p>	

When a patient is tachycardic, it is sometimes difficult to distinguish S1 from S2. Therefore, it is important to remember that the peripheral pulse corresponds to the S1 heart sound. So with simultaneous cardiac auscultation and palpation of the radial pulse, S1 can be distinguished from S2.

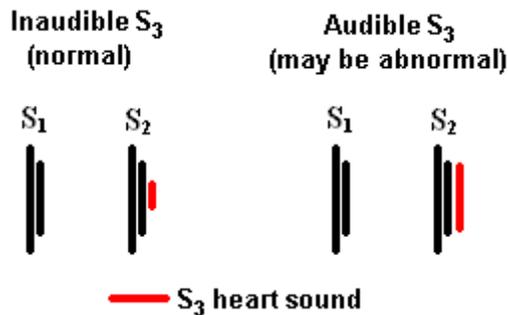


The third heart sound (S3)

The third heart sound (S3), also known as the "ventricular gallop", occurs just after S2 when the mitral valve opens allowing passive filling of the left ventricle. The S3 sound is actually produced by the large amount of blood striking a very compliant left ventricle.

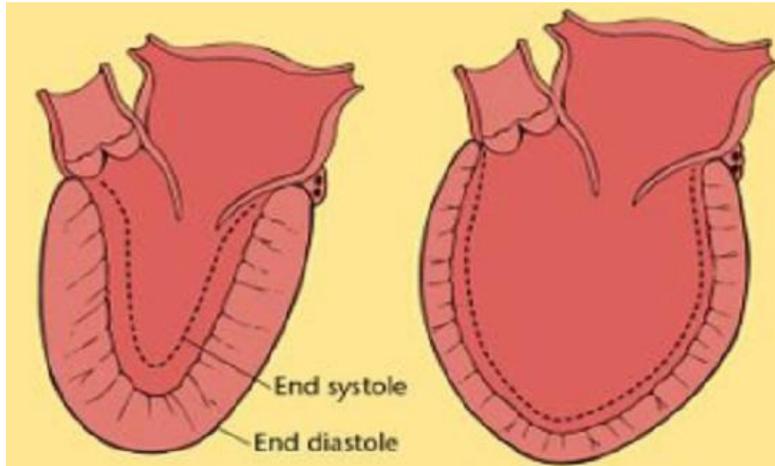
CLINICAL PEARL: A S3 heart sound is produced during passive LV filling when blood strikes a compliant LV.

If the LV is not overly compliant (as is most adults), a S3 will not be loud enough to be auscultated. A S3 can be a normal finding in children, pregnant females, and well trained athletes, however a S4 heart sound is almost never normal.



A S3 can be an important sign of systolic heart failure, since in this setting the myocardium is usually overly compliant resulting in a dilated LV (see image below).

CLINICAL PEARL: A S3 heart sound is often a sign of systolic heart failure, however it may sometimes be a normal finding.



Normal LV

Dilated LV (S3 present)

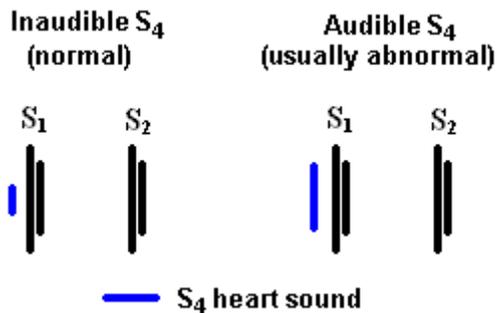
S3 is a low pitched sound. This is helpful to distinguish a S3 from a split S2 which is high pitched. A S3 heart sound should disappear when the diaphragm of the stethoscope is used and should be present while using the bell. The opposite is true for a split S2. Also, the S3 sound is heard best at the cardiac apex while a split S2 is best heard at the pulmonic listening post (left upper sternal border). To best hear a S3, the patient should be in the left lateral decubitus position.

The fourth heart sound (S4)

The fourth heart sound (S4), also known as the "atrial gallop", occurs just before S1 when the atria contract to force blood into the LV. If the LV is non-compliant and atrial contraction forces blood through the AV valves, an S4 is produced by the blood striking the LV.

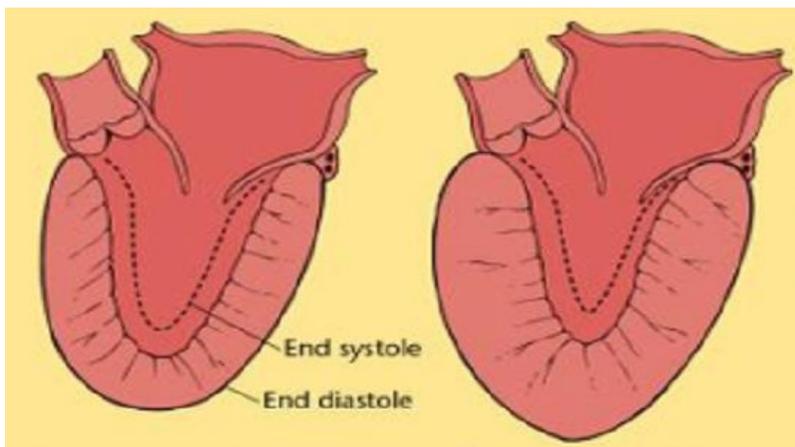
CLINICAL PEARL: A S4 heart sound occurs during active LV filling when atrial contraction forces blood into a non-compliant LV.

Therefore any condition that creates a non-compliant LV will produce a S4, while any condition that creates an overly compliant LV will produce a S3 (as described above).



A S₄ heart sound can be an important sign of diastolic heart failure or active ischemia and is rarely a normal finding. Diastolic heart failure frequently results from severe left ventricular hypertrophy (LVH) resulting in impaired relaxation (compliance) of the LV. In this setting, a S₄ is often heard. Also, if a person is actively having myocardial ischemia, adequate ATP can't be synthesized to allow for the release of myosin from actin, thus the myocardium is not able to relax and a S₄ will be present.

CLINICAL PEARL: A S₄ heart sound is often a sign of diastolic heart failure and it is rarely a normal finding (unlike a S₃).



Normal LV

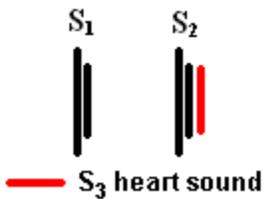
**Hypertrophied LV
(S₄ present)**

Like S₃, the S₄ sound is low pitched and best heard at the apex with the patient in the left lateral decubitus position.

Comparing S3 and S4

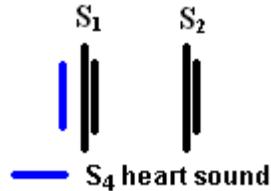
S3 - "ventricular gallop"

Occurs in early diastole
 Occurs during passive LV filling
 May be normal at times
 Requires a very compliant LV
 Can be a sign of systolic CHF



S4 - "atrial gallop"

Occurs in late diastole
 Occurs during active LV filling
 Almost always abnormal
 Requires a non-compliant LV
 Can be a sign of diastolic CHF



Extra systolic sounds

There are a few common extra-systolic sounds that the clinician may encounter. These include ejection sounds that occur with pulmonic or aortic valve stenosis and are heard in early systole, and the classic "clicks" that are heard in mitral or tricuspid valve prolapse and occur later in systole. These sounds are high pitched and result from the sudden tensing of the valves as they reach their elastic limit. They are often difficult, if not impossible, to distinguish from fixed or widened split S2.