

Additional Heart Sounds–Part 2 (Clicks, Opening Snap and More)

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Abstract

Keywords

- ▶ ejection click
- ▶ nonejection clicks
- ▶ mitral valve prolapse
- ▶ opening snap
- ▶ pericardial friction rub
- ▶ Hammam's crunch
- ▶ prosthetic sounds

Systolic clicks are high-pitched sharp sounds. They are classified as ejection and nonejection clicks. Ejections clicks commonly occur at the aortic and pulmonary valve, while nonejection clicks occur at the mitral and tricuspid valve.

Opening snap is an additional sound heard in the diastole. It is described as an early diastolic, high-pitched sound, which is associated with opening of the mitral and/or tricuspid valve.

Pericardial knock is a high-pitched early diastolic sound, which is characteristic of constrictive pericarditis.

The opening and closing of prosthetic valves produce sounds which may vary in intensity and timing according to the type and design of the valve, patient's rhythm, and hemodynamic status.

Systolic Clicks

Systolic clicks are high-pitched sharp sounds.¹⁻⁴ They are classified as ejection and nonejection clicks. Ejections clicks (ECs) commonly occur at the aortic and pulmonary valve, while non-ejection clicks occur at the mitral and tricuspid valve (▶ Fig. 1).

Technique of auscultation and detection of systolic click:

Clicks are best heard with the diaphragm of the stethoscope and are loudest at their respective auscultatory areas (▶ Fig. 2).

They are identified as sharp additional sound in the systole by simultaneous auscultation of the heart and carotid artery palpation to rightly identify S1 and S2. They are further identified as early, mid, and late systolic clicks and evaluated in relation to dynamic maneuvers.

Ejection Clicks (ECs)

They are sharp high-pitched sounds heard in early systole just after the first heart sound (▶ Fig. 3).

ECs occur at the maximal opening of the semilunar valves. Being high frequency sounds, they are best heard with the diaphragm. ECs result from opening of the stenotic/diseased aortic and pulmonary valves or from normal valves in the presence of dilated aorta or pulmonary artery.

Types of ECs: ECs are classified as valvular or vascular.

Valvular clicks: They result from abnormal aortic and pulmonary valves.

Mechanism: Abrupt opening of aortic and pulmonary valves generate ECs. The abrupt halting movement of the diseased valves produces this audible sound (▶ Fig. 4).

A. Aortic valvular click.

- a. Valvular aortic stenosis (AS).
- b. Congenital bicuspid aortic valve.
- c. Congenital quadricuspid aortic valve as in truncus arteriosus.

B. Pulmonary valvular click.

- a. Valvular pulmonary stenosis.

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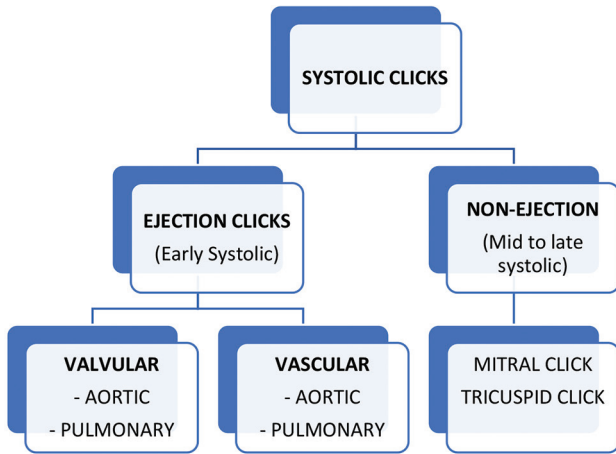


Fig. 1 Classification of systolic clicks.

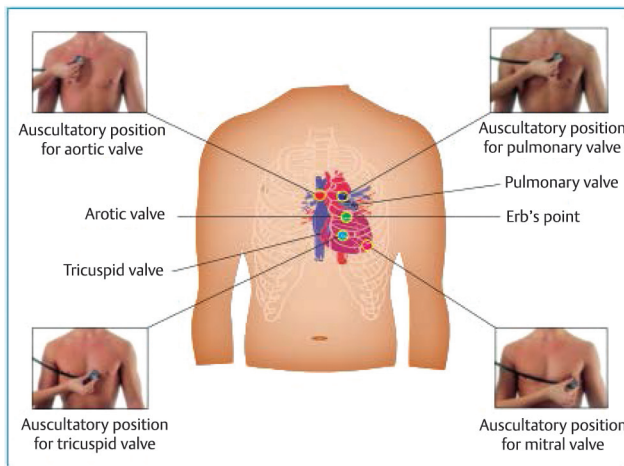


Fig. 2 Auscultation sites on precordium.

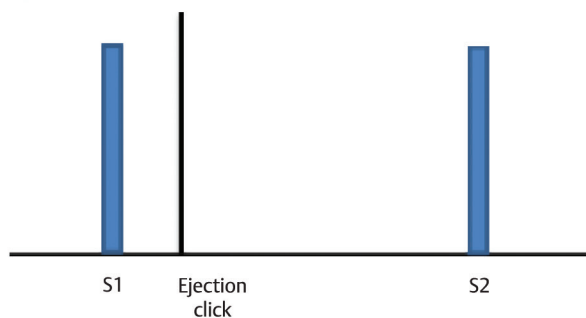


Fig. 3 Systolic ejection click.

Clinical characteristics of aortic and pulmonary valvular clicks are mentioned below (►Table 1).

Clinical Significance

Ejection Click of Aortic Stenosis (AS)^{5,6}

- Diagnostic of valvular AS and is absent in supra- or subvalvular AS.
- It corresponds to the notch in the upstroke of the aortic pressure tracing or the carotid pulse tracing (►Fig. 6).

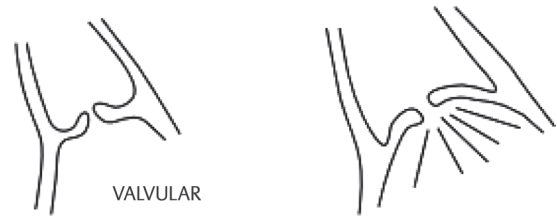


Fig. 4 Mechanism of valvular ejection click.

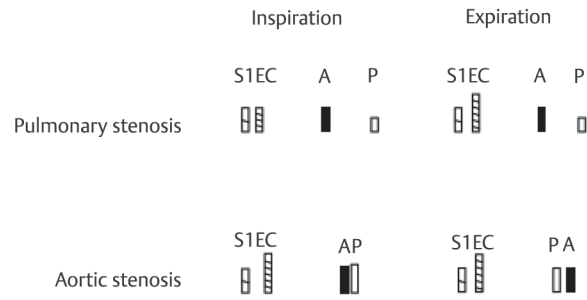


Fig. 5 Effect of respiration on pulmonary and aortic valvular click.

Table 1. Clinical characteristics of aortic and pulmonary valvular click

	Aortic valvular click	Pulmonary valvular click
Site of best audibility	Widely heard Best heard at the apex followed by right second intercostal space	Localized Best heard in the second left intercostal space
Respiration (Fig. 5)	Not affected	Decrease in intensity and occurs early/ merge with S1 in inspiration
Carotid upstroke	Immediately before or coincident with initial carotid upstroke	Usually before or with the initial carotid upstroke, however a delay in onset favors pulmonary origin
Relation with Q wave on ECG	Occurs 120 to 140 milliseconds after onset of Q wave on ECG	Occurs 90 to 110 milliseconds after onset of Q wave on ECG

This notch apparently results from maximal opening and abrupt deceleration of the flexibly stenotic aortic valve.

- Intensity of the sound decreases with calcification. Seen only with pliable valves and is absent in severe calcific AS.
- Calcification of the aortic valve is a rule beyond the age of 40 years. Hence, presence of aortic ejection click in adults suggests either mild AS or presence of congenital bicuspid aortic valve.
- Presence of an aortic EC in the child is diagnostic of congenital bicuspid aortic valve.
- Absence of other signs of AS in the presence of aortic EC strongly suggests the presence of bicuspid aortic valve.

Ejection Click (EC) of Pulmonary Stenosis (PS) ^{7,8}

- Diagnostic of valvular PS.
- It is the only right-sided acoustic event that softens (diminishes) with inspiration.
- Mechanism of inspiratory decrease in the pulmonary EC.
 - In PS, the right ventricle (RV) is stiff and hypertrophied.
 - During inspiration, when more blood enters the RV, it fails to distend normally. As a result, the RV pressure increases, and exceeds that of the pulmonary artery pressure, which falls slightly. The pulmonary valve is partially open (domed or tensed position) prior to onset of ventricular contraction (► Fig. 7a). When the RV contracts, excursion of the partially opened pulmonary valve is reduced and therefore the EC is softer and occurs earlier.
 - During expiration, venous return to the right heart is decreased. At the same time, the pulmonary artery pressure also rises slightly. As the pulmonary artery end-diastolic pressure exceeds the RV pressure, the pulmonary valve leaflets are in a closed or slack position (► Fig. 7b). With ventricular systole, the valve opens quickly and then abruptly halts, producing the ejection sound.

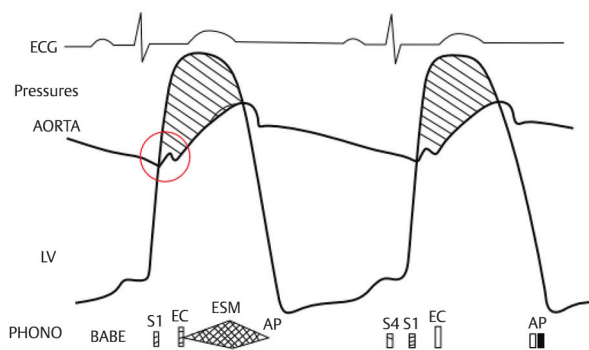


Fig. 6 Physiological basis of ejection click in aortic stenosis.

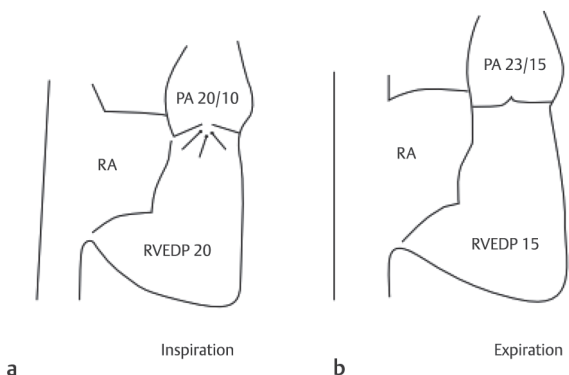


Fig. 7 (a and b) Respiratory variation of pulmonary ejection click.

The S1–pulmonary EC interval is directly related to RV isovolumic contraction time. With increasing severity of PS, the isovolumic systolic interval shortens resulting in early onset of EC.

- Mild-to-moderate PS–EC becomes soft and occurs earlier.
- Severe PS–the RV may become so stiff that a vigorous atrial contraction opens the pulmonary valve completely, producing a click in late diastole.
- Very severe PS–the EC fuses with S1 and may not be identified

Vascular clicks⁹: They occur in the presence of normal aortic and pulmonary valves. They result from rapid distension of the dilated proximal aorta or pulmonary artery at the onset of ejection.

Mechanism: Vascular clicks result from increased pressure, increased flow, and dilatation of the great vessels (► Fig. 8). Causes:

- A. Aortic vascular click.
 - a. Systemic hypertension.
 - b. Aneurysm of the ascending aorta.
 - c. Aortic regurgitation.
 - d. Tetralogy of Fallot–anterior dilated aorta.
 - e. Anemia and thyrotoxicosis–hyperkinetic circulatory states.
- B. Pulmonary vascular click.
 - a. Pulmonary hypertension.
 - b. Idiopathic dilatation of the pulmonary artery.
 - c. Left-to-right shunts.¹⁰
 - d. Hyperkinetic circulatory states.

Clinical characteristics of vascular and valvular clicks¹¹ are shown in ►Table 2.

Aortic vascular and valvular clicks can be identified, based on associated clinical features (►Table 3).

Pulmonary clicks can be differentiated, based on respiratory variation and associated clinical features (►Table 4).

Significance

Aortic vascular click is associated with aortic root dilation in the form of aortic sclerosis with tortuous aortic root, systemic hypertension, ascending aortic aneurysm and aortic regurgitation due to aortic root pathology.

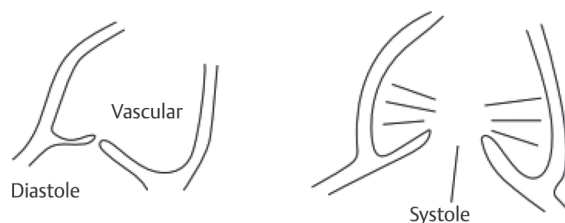


Fig. 8 Mechanism of vascular clicks.

Table 2. Clinical characteristics of vascular and valvular clicks

	Vascular clicks	Valvular clicks
S2 intensity	Loud	Normal or diminished
Split S2	Normal/close/single	Wide/reverse
Palpable artery beyond valve	Palpable	impalpable

Table 3. Aortic vascular versus valvular click

	Aortic vascular click	Aortic valvular click
Etiology	Normal aortic valve. Dilated aortic root	Stenotic aortic valve and non-stenosed Bicuspid aortic valve
Site of best audibility	Best heard at the aortic area (second right intercostal space). Poorly transmitted to the apex	Best heard at the apex

Table 4. Pulmonary vascular versus valvular clicks

	Pulmonary vascular click	Pulmonary valvular click
Etiology	Dilated main pulmonary artery and normal pulmonary valve	Pulmonary stenosis
Respiration	No consistent respiratory variation	Respiratory variation seen. The ejection click becomes softer and occurs earlier with inspiration

Pulmonary vascular click is associated with pulmonary hypertension and pulmonary arterial dilatation.

Differential diagnosis: ECs may be confused with a split S1 or a S4 followed by an early S1¹¹ (► **Tables 5 and 6**).

Nonejection Sounds¹⁻⁴

High-frequency sounds are best heard with the diaphragm of the stethoscope, and usually occur much later after the first heart sound (► **Fig. 9**). They are commonly heard across the atrioventricular valves in prolapse (myxomatous degeneration) of the mitral and tricuspid valves.

Causes:

1. Mitral valve prolapse (MVP)
2. Tricuspid valve prolapse (TVP).
3. Ebstein's anomaly
4. Aneurysm of the membranous septum with ventricular septal defect (VSD).

Characteristic features:

- Sharp, high-frequency, click-like sound
- Heard best at the apex (MVP) or at the left lower sternal border (LLSB) TVP.
- Usually single, but may be multiple
- Heard in mid-to-late systole

Table 5. S1–EC versus split S1

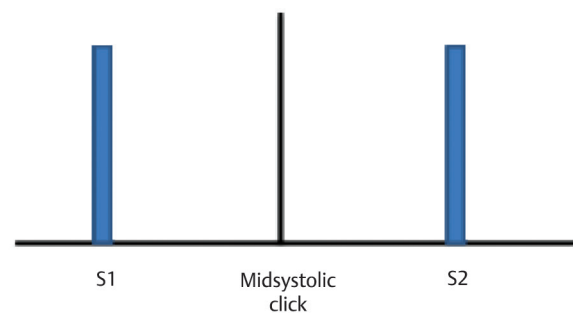
	S1–EC	Split S1
Quality of sounds	Both are high-pitched sounds	Second sound (tricuspid sound) is a low-pitch sound
Site of audibility	Best heard at the base of the heart and apex	Best heard at the lower sternal border
Respiration	No variation in case of aortic ejection click. Pulmonary ejection click softens with inspiration	Tricuspid sound becomes louder on inspiration

Abbreviation: EC, ejection click.

Table 6. S1–EC versus S4 with early S1

	S1–EC	S4–S1
Quality of sound	High-pitched–better heard with diaphragm	S4 is a low-pitched sound–better heard with bell
Site of audibility	Best heard at aortic area and apex	Localized to the apex
Associated features	–	Associated with presystolic impulse

Abbreviation: EC, ejection click.

**Fig. 9** Nonejection click/midsystolic clicks.

- Vary in timing with respiration, posture, and other dynamic maneuvers.

Systolic clicks in MVP^{12,13}: Midsystolic click is a characteristic auscultatory finding of MVP. It is the result of sudden tensing of the mitral valve apparatus, as the leaflets prolapse into the left atrium in systole.

Mechanism: Normally, with the onset of ventricular systole, when the left ventricular (LV) pressure exceeds that of the left atrium, the prolapse of mitral leaflets into the left atrium is prevented by the mitral cusps, chordae tendineae, and papillary muscle contraction (mitral valve apparatus). In MVP, there is myxomatous degeneration and elongation of the mitral leaflets and chordae tendineae. The click results from sudden snapping/halting of the prolapsed mitral leaflets into left atrium after the onset of ventricular contraction.

As different parts of the mitral leaflets prolapse at different times of systole, multiple clicks can be heard (►Fig. 10).

The click is mobile, which means it varies in timing and loudness with change in ventricular volumes and contractility (►Table 7).

Prolapse does not occur immediately on the onset of ventricular systole. Prolapse starts when the LV size reaches a critical point and persists after that (►Fig. 11). Click occurs at the onset of that prolapse, followed by the murmur, which persists throughout the systole.

Any maneuver that decreases the LV volume (↓) (►Fig. 12a) causes the prolapse to occur earlier in systole and brings the click closer to S1. Decrease in LV volume is the result of the following:

- Decreased impedance to LV outflow.
- Decreased venous return.
- Increased contractility.
- Tachycardia.

Any maneuver that increases the LV volume (↑) (►Fig. 12b) causes the prolapse to occur latter and click is delayed.

- Increase in LV volume is seen in conditions of-
- Increased impedance to LV outflow.
- Increased venous return.
- Increased contractility.
- Bradycardia.

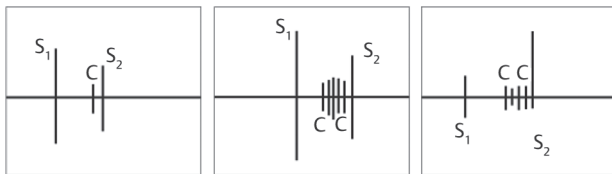


Fig. 10 Multiple clicks in mitral valve prolapse.

Table 7. Physiological maneuvers and their effect on midsystolic click in MVP

Maneuver	MVP click (timing, intensity, duration)
Positional changes	
Standing	Earlier and louder
Supine/squatting/leg raising	Delayed and softer, may disappear
Post-PVC/ premature contraction of AF	Delayed and soft
Valsalva	
Strain phase	Earlier and loud
Overshoot phase	Delayed and soft
Amyl nitrate inhalation/nitroglycerine	Earlier and louder
Isometric exercise	Delayed and soft
Phenylephrine/maneuvers which increased blood pressure	Delayed and soft

Abbreviations: AF, atrial fibrillation; MVP, mitral valve prolapse; PVC, premature ventricular contraction.

Differential diagnosis: Click of MVP must be distinguished from an aortic ejection click (►Table 8).

Systolic clicks of TVP: Isolated TVP is a rare occurrence and is associated with a nonejection systolic click of right heart origin like as in MVP.

Systolic click of Ebstein’s anomaly: An early sharp sound is heard in early systole, designated as the “sail sound” of Ebstein’s anomaly. This occurs when the large sail-like anterior tricuspid valve leaflet has reached the maximum limit of systolic excursion. It occurs just after the peak of the C wave in the jugular venous pressure (JVP).

Opening Snap (OS)

It is an additional sound heard in the diastole¹⁻⁴ (►Fig. 13). It is described as an early diastolic, high-pitched sound, which is associated with opening of the mitral and/or tricuspid valve.

History^{14,15}: OS was first described in mitral stenosis (MS) patients by Bouillaud (1835) and later by Durozier (1862) as asynchronism of pulmonary and aortic valve closure, who compared this to the syllables “ffou-ta-ta-rou.” Guttman in 1872 and later Sansom (1881) suggested that this sound was due to vibration at the stenosed mitral valve itself. Finally, it was Rouches in 1888 who introduced the present nomenclature of “claquement d’ouverture de la mitrale” or OS of the mitral valve to describe this sound. According to him, this sound was produced by sudden tensing of the stenosed mitral valve by the blood passing from the left atrium to the left ventricle. Although other hypothesis have been proposed, it was finally concluded to be the mechanism by Margolies and Wolferth (1932) after a detailed clinical and phonocardiographic study of this sound.

Mechanism of OS: OS results from abnormal audible opening of diseased mitral or tricuspid valves. Abrupt opening of the valves to its maximum results in sudden tensing of the abnormal leaflets and subvalvular apparatus, producing this crisp sound. This is seen in early diastole when the ventricle is relaxed sufficiently to allow the atrial pressure to be effective. Mobility of the valve contributes to its genesis. An OS is absent when the valve is heavily calcified and immobile. An OS snap can also be produced in the absence of diseased valves, due to increased blood flow, especially across the mitral valve into normal LV.

Conditions with OS

1. Diseased atrioventricular valves.
 - a. MS
 - b. Tricuspid stenosis (TS)
2. Normal atrioventricular valves, but excess blood flow from mitral valve into LV
 - a. VSD
 - b. Patent ductus arteriosus (PDA)
 - c. Mitral regurgitation (MR)

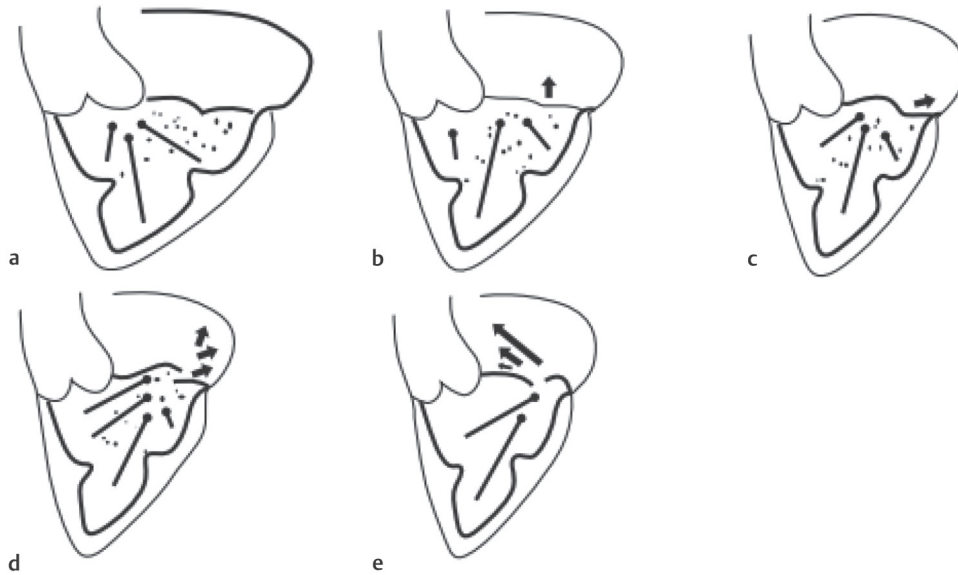


Fig. 11 Relation between LV size/volume and degree of MVP. (a). Prolapse of mitral leaflets does not occur at the onset of systole. (b–c). Onset of prolapse of leaflets. (d). Occurrence of click at the critical point of LV size where there is no coaptation of leaflets and prolapse is to the maximum. (e) Click is followed by murmur. Abbreviations: LV, left ventricle; MVP, mitral valve prolapse.

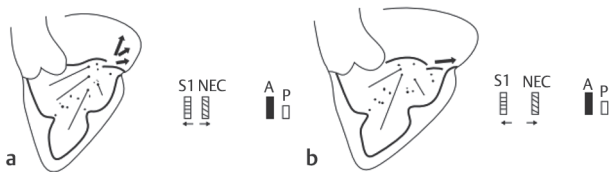


Fig. 12 Relation between LV volume and occurrence of midsystolic click in MVP. (a): Above figure shows early occurrence of ejection click, close to S1 when LV volume is small. (b): Figure below shows late occurrence of ejection click when LV volume is increased. Abbreviations: LV, left ventricle; MVP, mitral valve prolapse.

Table 8. MVP click versus aortic ejection click

	Click of MVP	Aortic ejection click
Timing	Mid to late systole	Early systole
Relation to carotid pulse	Happens after the beginning of the upstroke	Occurs with the beginning of the carotid upstroke

Abbreviation: MVP, mitral valve prolapse.

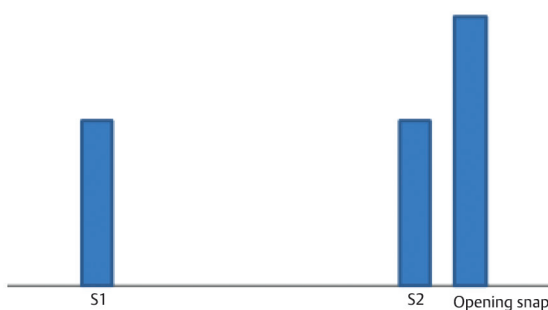


Fig. 13 Opening snap.

3. Rare conditions wherein a mitral OS is heard in the presence of a normal mitral valve are¹⁶:

- Tricuspid atresia with a large atrial septal defect (ASD).
- Idiopathic second- and third-degree heart block.
- Thyrotoxicosis.
- Tetralogy of Fallot occurring after a Blalock–Taussig procedure.

Characteristic Features of OS

- Sharp, high-pitched, crisp sound heard with the diaphragm of the stethoscope.
- Best heard in the supramammary area between the apex and the LLSB, but it has a wider area of radiation. It is loudest in the fourth left interspace, occasionally in the third, and slightly above and to the right of the area in which the diastolic murmur is best heard (i.e., the supramammary area). It is next best heard at the apex, left sternal border, and at the base of the heart in the aortic position, with the least common location being the pulmonary area.
- Usually heard 40 to 120 milliseconds after the second heart sound, it precedes the mid-diastolic murmur by a short interval. The S2–OS interval is influenced by the heart rate (HR) and tends to shorten with tachycardia. During atrial fibrillation (AF) and sinus arrhythmias, the interval duration depends on the length of the preceding cardiac cycle.

Best audible in recumbent position. Exercise can augment its audibility by increasing the HR as OS is louder at higher rates.

Clinical Significance

Mitral stenosis (MS)^{17–19}:

- Mitral OS followed by a mid-diastolic murmur is characteristic of MS and not a flow rumble across a nonstenotic valve.

- Presence of a crisp OS suggests that the mitral valve leaflets are mobile and pliable.
- It softens or disappears as the leaflets become ore calcific and immobile.
- The S2–OS interval helps in assessment of the left atrial pressure and severity of MS.
 - In severe MS, because of higher left atrial pressure, the transmitral gradient develops immediately after mitral valve opening (MO), resulting in an OS almost just after S2.
 - In mild mitral stenosis, the OS occurs later in diastole due to delayed transmitral gradient.
 - In addition, in severe MS, the OS is followed by a longer murmur, as it takes more time for blood to pass through the stenotic mitral valve and for the pressure gradient to dissipate once the mitral valve opens.
 - Intervals < 70 milliseconds suggest severe MS, and > 100 milliseconds are compatible with mild obstruction.
 - An A2–OS interval of ≥ 80 milliseconds indicates the left atrial mean pressure is normal (9 to 12 mm Hg).
 - An A2–OS interval of < 60 milliseconds indicates a markedly elevated left atrial mean pressure (25 to 36 mm Hg).
 - When the A2–OS interval was 60 to 80 milliseconds, there was an overlap of mean pressure value (12 to 25 mm Hg).²⁰
 - Reduction of < 20% of the interval on exercise suggests an increase of < 35% left atrial mean pressure.
 - Whereas a decrease of the A2–OS interval > 30% is associated with > 40% increase of the left atrial mean pressure.

OS is a useful diagnostic sign in MS, especially when other signs have been missed on casual examination. Its presence prompts for a more thorough search for the latent mid-diastolic murmur. Absence of OS in a confirmed case of MS indicates either

- a. mild MS; b. calcified immobile valve c. associated significant MR.

Tricuspid Stenosis (TS): OS in TS was first described by Rivero Carvallo (1952–53)²¹ as a sound from the diseased tricuspid valve and defined as “chasquido de apertura de la

trictspide.” The sound is similar in almost all respects to the OS of the mitral valve except for its area of audibility. It is shorter in duration, occurs earlier and relatively lower pitch than the mitral OS, and best heard over the xiphisternum and right lower end of the sternum²²

Differential diagnosis: OS could be confused for split second heart sound or third heart sound (►Table 9, ►Fig. 14).

The time intervals of opening snap and its mimics play an important role in identifying its character and is depicted in ►Fig. 14.

The sequence of events would be split S2, OS, and then S3 in view of occurrence and intervals (►Fig. 15).

The OS and second component of split-second sound can be differentiated clinically, based on the best audible site and interval. Deep breathing technique is of great value, especially since both the sounds are heard. Differentiation of opening snap from S3 rarely poses a dilemma in clinical reality.

►Fig. 16 summaries the confusion among the additional sounds around first and second heart sound in practice.

Pericardial Knock¹⁻⁴

- It is a high-pitched early diastolic sound, characteristic of constrictive pericarditis. Previously referred to a pericardial protodiastolic sound.
- It is an early sharp S3, which occurs 100 to 120 milliseconds after S2 (►Fig. 17).
- Best heard with the diaphragm of the stethoscope at the LLSB.
- It is rarely palpable.
- It occurs at the trough of Y descent of JVP.

Mechanism²³: Pericardial knock is heard when the stiff and thickened pericardium causes sudden cessation of ventricular filling during early diastole. When the expanding ventricles achieve the limits imposed by the nondistensible thickened pericardium, there is a sudden deceleration of diastolic filling. This produces a sharp sound known as the pericardial knock. The LV filling pressure curve showed a rapid filling period lasting for 30 to 40% of total diastole, followed by a sudden and abrupt plateau, which persisted up to the end of the diastole. Two hypotheses for genesis of the sound has been suggested.

Table 9. Differential diagnosis of opening snap

Clinical feature	OS	Split S2	Third heart sound
Interval between the sound	Longer (40–120 milliseconds after A2)	Shorter (30–50 milliseconds)	Much longer (160–240 milliseconds after A2)
Area best heard	Between the LLSB and apex	Pulmonary area	A little internal to mitral area
Character	High-pitch–loud and sharp	Softer	Low-pitched
Effect of respiration	None on interval. Intensity diminished in inspiration	Split widens in inspiration	None on interval
Conditions	MS/TS	Physiological	Physiological/MR/LVF

Abbreviation: LLSB, left lower sternal border; LVF, left ventricular function; MR, mitral regurgitation; MS, mitral stenosis; OS, opening snap; TS, tricuspid stenosis.

Condition	S1	Interval (mSec)
Normal S2 split (Inap.)	A p	40-50
Normal S2 split (Exp.)	A p	<30
S2 wide split	A p	60-120
Pathological S2-S3 interval	A p S3	120-160
Phyalological S2-S3 interval	A p S3	120-200
S2-Pericardial knock	A p PK	100-120
S2-OS interval	A p OS	40-120

Fig. 14 Occurrence of time intervals of opening snap and its mimics.

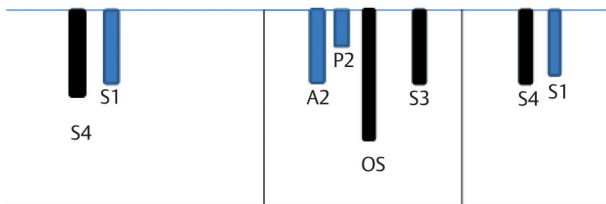


Fig. 15 Sequence of events in relation with opening snap.

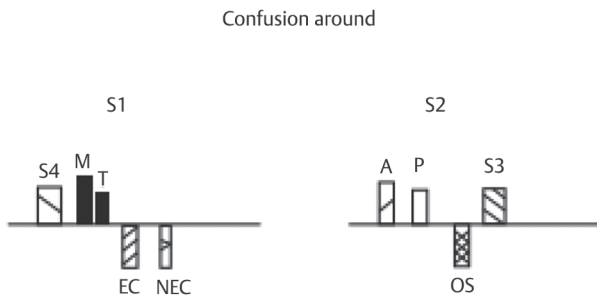


Fig. 16 Additional sounds causing confusion around S1 and S2.

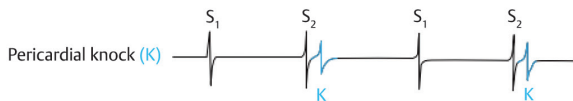


Fig. 17 Pericardial knock.

a. Water hammer effect–sudden deceleration of rapid filling blood into the ventricle sets into vibration the chamber blood, ventricular walls, valves, and pericardium (like ventricular S3 gallop).

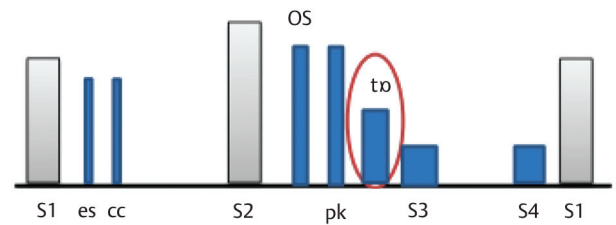


Fig. 18 Tumour plop.

b. Sail-flop phenomenon–Sudden tautening of the pericardium sets into vibration the chamber blood, walls and valves, producing the pericardial knock (like OS of MS or EC of AS).

Differential Diagnosis

S3 gallop is the most common differential diagnosis. A pericardial knock occurs closer to the S2 than S3 gallop, which occurs 100 to 200 milliseconds after S2. Pericardial knock is of higher pitch and louder than S3.

Tumour Plop¹⁻⁴

Early diastolic low-pitched sound heard just after S2 (► Fig. 18). It is heard 80 to 150 milliseconds after the second heart sound. It may or may not be followed by a diastolic murmur. It is heard in left atrial myxoma.

Differential Diagnosis

- S3 gallop–timing and frequency may be indistinguishable.
- OS of MS–OS is a high-pitched sound in contrast to the low-pitched tumor plop.
- Split S2–single S2 followed by a tumor plop may mimic a split S2. However, tumor plop is a transient diastolic sound which disappears as one moves the stethoscope to the pulmonic area. If two sounds are heard at the pulmonic area, it is split S2.

Mechanism²⁴: When the phonocardiographic timings of the tumor plop was correlated with the echocardiographic motion pattern of the cardiac mass, it was noticed that the sound occurred when the diastolic forward motion of the mass was halted into the ventricle. Various hypotheses for generation of the sound have been suggested.

- It is the result of quick diastolic movement of the mass into the ventricle and its impact on the ventricular septum and ventricular wall.
- Tumour obstruction of the mitral orifice along with high velocity across it causes the sound
- Sudden tensing of the tumor stalk and impact on the interventricular septum and right ventricular posterior wall.

Occurrence of tumor plop requires presence of a large mass and/or a long tumor stalk. The mass can move into the ventricle in two stages–early diastolic passive filling stage and late atrial contraction stage; hence, two sounds

can also be heard, that is, early diastolic and late diastolic sounds.

Other Diastolic Sounds

1. Vegetation plop²⁵: an early diastolic sound occasionally heard in endocarditis. This sound is produced when a large vegetation over the mitral valve moves into the LV during early diastole.
2. High-frequency early diastolic sounds are occasionally heard in the following:
 - a. MVP–sound is related to rapid movement of the prolapsed MV into LV before opening of the mitral valve.
 - b. Hypertrophic cardiomyopathy–sound is related to contact of the anterior mitral leaflet (AML) with the interventricular septum (IVS) in early diastole, especially in patients with small LV cavity.
 - c. Severe MR due to ruptured chordae.

Pericardial Friction Rub¹⁻⁴

Superficial, scratch or grating sound is a hallmark of pericardial inflammation. It is heard in acute viral pericarditis and Dressler syndrome (24–72 hours after acute myocardial infarction [MI]).

Mechanism: It is the result of friction between the parietal and visceral layers of the inflamed pericardium. It occurs during the maximal movement of the heart within the pericardial sac.

Characteristic Features²⁶⁻³⁰

- Scratchy/leathery/grating sound–like the two pieces of sandpaper rubbing together. Loud enough to mask other cardiac murmurs.
- May present as a soft blowing murmur and confuse with other cardiac murmurs.
- Best heard with the diaphragm of the stethoscope.
- Best heard with firm pressure of the diaphragm during held inspiration and in leaning forward position or leaning over position (►Fig. 19)
- May be localized or widespread and best heard at the lower sternal border.
- Although synchronizes with the heartbeat, it does not strictly correlate with the systole or diastole.
- It has three components–atrial systole, ventricular systole, and rapid filling phase of the ventricle (►Fig. 20)
 - However, it may present as one component or two.
 - The order of the most audible component is ventricular systole > atrial systole > rapid filling phase.
 - The three-component sound is heard in less than 50% cases.
 - Usually a to-and-fro rub is heard representing the ventricular systole and atrial systole.
 - Single component is rare and heard in myocarditis, following transmural MI, resolving stage of pericarditis, and in the presence of atrial fibrillation.

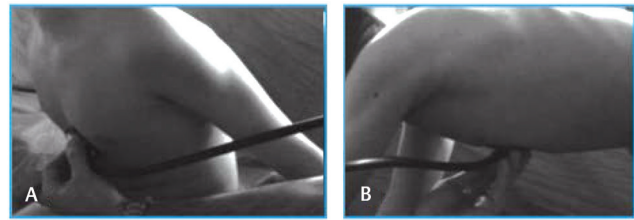


Fig. 19 Best position to auscultate for pericardial friction rub.

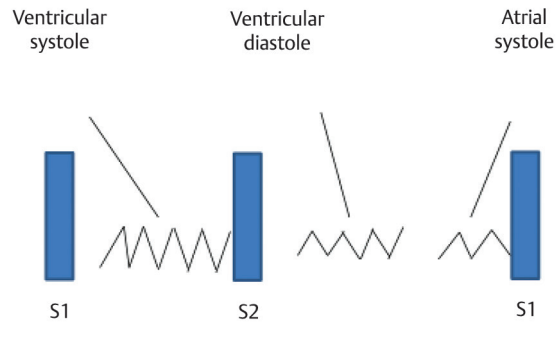


Fig. 20 Components of friction rub.

Inconsistency is a striking feature of pericardial rub. It is usually transitory or evanescent, persisting for few hours or days. Its intensity, character, duration, and site of audibility also varies over a period.

Differential Diagnosis

1. Pleural rub (►Table 10).
2. Systolic or to-and-fro murmur (►Table 11).
3. Means–Lerman scratch in thyrotoxicosis–a to-and-fro high-pitched sound heard over the left second space
4. Hamman's sign/mediastinal crunch–acute mediastinal emphysema, usually postopen heart surgery.
5. Sail sound in Ebsteins's anomaly.
6. Pacemaker heart sound–twitching of the intercostal muscles or the diaphragm during artificial pacing.
7. Pleuropericardial rub: A pleuropericardial rub, resulting from friction between the inflamed pleura and parietal pericardium, can often be confused for a pericardial rub. This happens in dry pleurisy when the anterior margin of the lung is affected. The adjacent heart is involved, and the friction rub now confronts cardiac rhythm rather than the respiratory movements. This movement occurs with beating of the heart and disappears with deep respiration or cessation of breathing.
8. Movement of transvenous pacing lead or balloon floatation catheter across the tricuspid valve produces an early systolic superficial scratching sound that may be confused for a single component friction rub. These sounds usually disappear with alteration of the patient's position
9. Inadvertent entry of air into the RV cavity and its movement with RV systole and diastole produces a slushing

or crunching sound (mill wheel murmur) which mimics pericardial rub.

- Swallowing sounds produced by vibration of vocal cords during swallowing.

Clinical Significance³¹⁻³⁵

- Acute MI—a pericardial rub after acute MI suggests a transmural infarction and a contraindication to anticoagulation. It is responsible for persistent pain after MI and a reason for pyrexia after acute MI. Prompt relief is seen with steroids.
- Rheumatic carditis—suggests pan carditis and response with steroids.
- Postheart surgery—suggests pericardiotomy pericarditis.
- Chronic renal failure- intensification of dialysis
- Infective endocarditis—myocardial abscess/ring abscess.
- Liver abscess—extension into the pericardium.

Table 10. Differentiating features between pleural and pericardial rub

Pleural rub	Pericardial rub
Correlates with respiratory movements	Does not correlate with respiratory movements
Grossly affected by respiration and cessation of breathing—heard in inspiration. Disappears with expiration and breath-holding	Not affected—heard in both phases of respiration and even after cessation of breathing
Independent of cardiac rhythm	Correlated with cardiac rhythm
Pain on the lateral aspect of chest wall	Pain usually in the central location of rib cage
Intensity increased with pressing of diaphragm of stethoscope over the affected chest wall	No such variation or accentuation seen

Table 11. Differentiating features between pericardial rub and cardiac murmur

	Pericardial rub	Cardiac murmur
Quality	Scratch/grating/leathery	Blowing/rumbling/musical
Timings	Systolic or diastolic	Systole–diastole
Site of audibility	LLSB	Typical auscultatory areas
Radiation	Nil	Yes
Constancy	No	Yes
Duration	Evanescent	persistent
Firm pressure with diaphragm	Accentuates	No change
Effect of respiration	Accentuated with-held inspiration	Not much except right sided events
Position	Accentuate with leaning forward posture	Does not vary

Abbreviation: LLSB, lower left sternal border.

7. Malignancy–pericardial involvement.
8. Patients in shock heart failure—look for cardiac tamponade.
9. Fever with septic foci–pyogenic pericarditis.
10. Fever of unknown origin–tuberculosis.

Hamman’s crunch^{36,37}—Multiple crunchy sounds heard when the heart beats against the air-filled tissues. These precordial crackles correlate with the heartbeat and not respiration.

They are seen in the following:

Pneumopericardium—postcardiac surgery, postresuscitation, or postpericardiocentesis

Pneumomediastinum—spontaneous mediastinum emphysema or Boerhaave syndrome.

Prosthetic Sounds¹⁻⁴

The opening and closing of prosthetic valves produce sounds which may vary in intensity and timing according to the type and design of the valve, patient’s rhythm, and hemodynamic status. Prosthetic sounds are the result of valve component motion or abnormal flow patterns. These sounds are louder, of higher frequency than normal sounds, and usually described as “clicky” quality. They may be single or multiple.

Characteristics Features

- A metallic click before the carotid pulse (occurs in diastole after S2) is an auscultatory sign of mitral valve replacement, and a click just after the carotid pulse (occurs in systole, after S1) is heard in aortic valve replacement (► Fig. 21).
- A mitral prosthetic sound is heard louder in the mitral area and an aortic prosthetic sound over the aortic area.

Classical click—Loud, high-frequency metallic closing sounds are typically heard with tilting disc and bileaflet

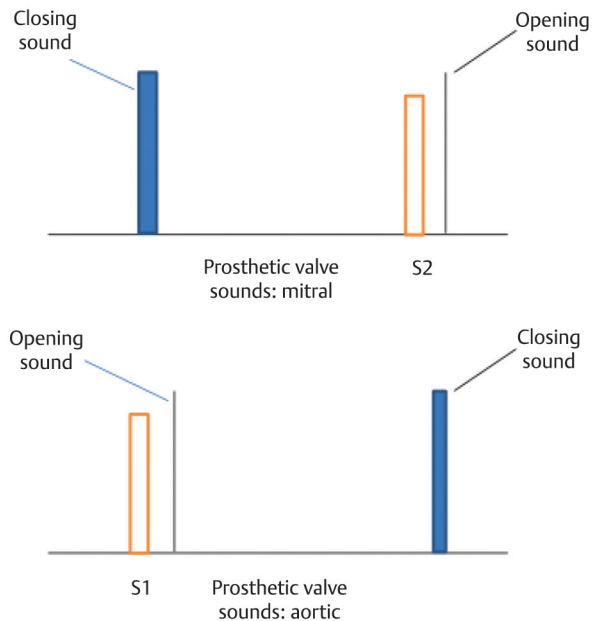


Fig. 21 Mitral and aortic prosthetic sounds.

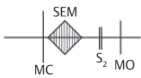
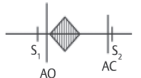
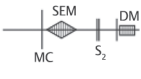
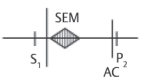
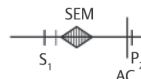
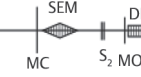
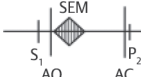
valves. They can be heard even without a stethoscope. A soft opening sound may also be heard.

Ball and caged valves (Star-Edwards)–They have easily audible opening and closing sounds. These sounds are distinct, high-pitched prominent sounds (relatively low-pitched compared with disk and leaflet valves) which can be easily distinguished from normal heart sounds. Both the opening and closing sounds are of equal intensity. The sounds can be very loud, depending on the prosthetic model and composition of the ball. The sounds correspond to the maximal excursion of the ball and its subsequent seating.

Classical clicks are not heard with tissue valves; instead, low-pitched sounds different from the normal heart sounds are heard

Auscultatory features: Auscultatory features³⁸⁻⁴⁰ of different prosthetic valve in mitral and aortic position are mentioned in ► **Table 12.**

Table 12. Auscultatory features of different prosthetic valves

Type and design of valve	Mitral area	Aortic area
Ball valves (e.g., Starr-Edwards)	Opening sound (MO) heard in diastole MO louder than closing sound (MC) A2–MO interval 70–110 milliseconds 	Opening sound (AO) heard in systole AO louder than closing sound (AC) S1–AO is 70 milliseconds 
Tilting disc valves (e.g., Medtronic-Hall)	MO is soft and may not be audible. A2–MO 50–90 milliseconds 	AO is soft and may not be audible S1–AO is 40 milliseconds AC is louder than AO 
Bileaflet valves (e.g., St. Jude)	–	
Bioprosthetic valves	MC louder than MO MO audible in 50% A2–MO interval 100 milliseconds 	AC louder than AO AO may be absent S1–AO interval 30–80 milliseconds 

Abbreviations: AC, aortic closing; AO, aortic opening; MC, mitral closing; MO, mitral opening.

Note: TTK Chitra⁴¹ is a low-cost, high-quality single leaflet tilting disc. In contrast to other disk valves, valve closing sounds are soft and dull due to the use of ultrahigh-molecular weight polyethylene disc; hence, less disturbing in a lean person

Of the commonly used mechanical valves, the sounds associated with Duromedics Edwards (33.5 dB) and Björk-Shiley Monostrut valves (31 dB) are significantly louder than St Jude Medical (24 dB) and Carbomedics (25 dB) valves.^{42,43}

Clinical Significance

Muffled or absent normal prosthetic sound is a clue to valve failure or thrombosis.

Abnormal Prosthetic Sounds^{1-4,44-46}

Ball and caged valves: Both the opening and closing sounds are loud and easily audible. Multiple sounds are frequently heard in a normally functional ball valve.

Abnormal mitral prosthetic sounds–A normal mitral ball valve produces a very prominent opening click, corresponding to the nadir or 0 point of apexcardiogram. It follows the A2 by 0.07 to 0.11 seconds.

1. A2–mitral opening (MO) interval differs minimally from beat to beat even in AF.
 - a. A2–MO interval < 0.05 seconds suggests prosthetic valve obstruction or severe MR.
 - b. A2–MO interval > 0.17 seconds or a beat to beat alteration of the interval suggests abnormal interference with the poppet excursion–stuck valve.
2. Diminished mitral closing (MC) sound in conditions where the poppet has already assumed a closed position before the onset of ventricular isovolumic systole.
 - a. First-degree heart block.
 - b. Long R–R intervals.
3. Single prosthetic closing sound (MC) without and opening sound (MO)–
 - a. Premature ventricular beat–The poppet fails to open after a premature ventricular contraction (PVC), drifts to open position slowly during next diastole and is forcibly closed by the next ventricular contraction, producing a single closure sound.

Abnormal aortic prosthetic sounds:Normally, the ball proceeds to the apex of the cage at the point of LV–aortic opening (AO) pressure crossover. This produces a loud opening click which occurs approximately 0.07 seconds after S1. The ball may remain at the apex or midcage for the entire systole, resulting in production of multiple systolic clicks.

1. Increased intensity of both AO and aortic closing (AC) sounds–
 - a. Tachycardia.
 - b. Anemia.
2. Diminished intensity of both AO and AC sounds–
 - a. Low-cardiac output state
3. Diminished AO (opening sound)–Normally, the amplitude ratio of AO to AC is > 0.5. Reduction of this ratio or absence of appreciable AO is a sign of ball valve dysfunction.

4. An early ventricular beat may be associated with an absent AO, whereas a late ventricular beat may produce both AO and AC sounds.

Tilting disc valves: Normally, distinct closing sounds are heard. These sounds are high-pitched click-like sounds corresponding to the onset of valve closure. However, the opening sound may not be produced as the lightweight disk swings open without forcibly striking any resonant structure

Abnormal mitral prosthetic sound: Prosthetic opening sound (MO), corresponding to disk motion, is rarely heard, but a prominent MC is heard

Absent or diminished intensity of MC–

- a. When disk seats early.
 - i. LV dysfunction.
 - ii. First-degree atrioventricular (AV) block
- b. When disc movement is hindered.
 - i. Fibrosis.
 - ii. thrombosis.

Abnormal aortic prosthetic sounds: Distinct closing sound is heard. Diminution or absence of it is indication of prosthetic valve fibrosis, thrombosis or LV dysfunction.

Bileaflet Valve (St. Jude)

Abnormal aortic prosthetic sounds: Distinct opening sound (AO) is heard after the maximal opening of the valve leaflets, suggesting its origin to be due to turbulent flow and not the leaflets. In contrast, a prominent AC is heard corresponding to the leaflet closure.

Diminished or absent closing sound is a sign of valve dysfunction.

Acoustic characteristics in mitral position have not been described.

Tissue valves (Hancock, Carpenter–Edwards): Both closing and opening sounds are heard, much less prominent than with mechanical valves. Opening sound corresponds to abrupt halting of the opening motion of the leaflets. Closing sounds correspond to deceleration of blood column or due to leaflet contact. Closing sounds are much louder than opening sounds with bioprosthetic valves.

Diminished or Absent Closing Sound is a Sign of Malfunction

Ball variance is a term used to describe the physical changes in a ball valve, which results in changes in the intensity of the opening and closing sounds. It is related to specific model of the ball valve, which is rarely in use nowadays.

Conclusion

Change in the normal prosthetic sound is a sign of valve malfunction. Irrespective of the type of prosthetic valve, the closing sound is usually louder. A decreased intensity of this closing sound should suggest malfunction of the valve. However, malfunction can exist despite the presence of a normal sound. In such cases, Doppler echocardiography

and cardiac catheterization are required to establish the diagnosis.

Conflict of Interest

None.

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