Cardiac auscultation: normal and abnormal

The pivotal role that cardiac auscultation plays in facilitating clinical diagnosis was first documented by Hippocrates (460–370 BC). Point of care ultrasound is increasingly being used to provide highly detailed images, such that the work of the ears is being bypassed for that of the eyes, yet clinical assessment remains gatekeeper to these tests. The importance of cardiac auscultation is still reflected in postgraduate medical and surgical examinations, which necessarily demand a high level of skill.

The heart sounds

Heart sounds are the normal audible reverberations generated during the closure of the cardiac valves, the character of which is governed by chamber architecture, blood pressure, valvular orifice size and electrical propagation.

The first heart sound, S_1 ('lub'), is the sound of both atrioventricular valves closing which occurs when ventricular pressures exceeds atrial pressure at the start of ventricular systole. The mitral component occurs first (M_1), quickly followed by the tricuspid component (T_1). The second heart sound, S_2 ('dub'), is the sound of both semilunar valves closing. This occurs when the pressure in the pulmonary artery and aorta exceed the ventricular pressure at the start of ventricular diastole. The aortic component occurs first (A_2), quickly followed by the pulmonary component (P_2). The first two heart sounds are physiologically normal and the components of S_1 and S_2 are not usually well differentiated because they occur almost simultaneously.

Less commonly heard are third and fourth heart sounds. A third heart sound S_3 (lub-de-dub) may be heard. S_3 reflects rapid ventricular filling during early diastole, immediately after S_2. It can be normal in isolation in the young or athletes, but is pathological in association with a fourth heart sound. A fourth heart sound S_4 (le-lub-dub) is always pathological, occurring in late diastole immediately before S_1, as a result of atrial contraction forcing blood into an abnormally stiff ventricle. Common causes of an S_4 include cardiomyopathies or increased cardiac afterload. The presence of all four heart sounds is known as a gallop rhythm (le-lub-de-dub), rather like the hooves of a trotting horse, and is a feature of acute heart failure.

Heart sound intensity

The intensity of S_1 is dependent upon body habitus, PR interval, atrioventricular valvular mobility and left ventricular contraction velocity. Thus S_1 is commonly quieter in the presence of obesity, a long PR interval or hypodynamic left ventricle. The intensity of S_2 is dependent upon ventriculo-arterial valvular mobility and so the A_2 component can be quiet or even absent in patients with severe aortic stenosis.

Splitting of the second heart sounds

The second heart sounds can be split in four ways: physiological, reversed (or paradoxical), persistent and fixed (*Figure 1*).

Physiological splitting

Physiological splitting of S_2 refers to A_2 occurring before P_2 during inspiration, so that both are individually audible. Inspiration increases right heart venous return, thus prolonging right ventricular systole, relative to the left ventricle, and so the pulmonary valve closes after the aortic valve. This disappears during expiration.

Reversed (or paradoxical) splitting

Reversed (or paradoxical) splitting refers to when the split is heard during expiration, not inspiration. Any process that prolongs left ventricular systole and/or aortic valve closure can cause this. Examples include aortic stenosis, hypertrophic cardiomyopathy or left bundle–branch block. The P_2 component is heard first, then A_2.

Persistent splitting

Persistent splitting of S_2 refers to when A_2 and P_2 are audible separately throughout the respiratory cycle, but the interval prolongs with inspiration. It occurs secondary to processes which prolong right ventricular systole and/or pulmonary valve closure, e.g. right bundle–branch block, pulmonary hypertension or pulmonary stenosis, or processes which hastens left ventricular systole and/or aortic valve closure, e.g. mitral regurgitation or a ventriculoseptal defect.

Fixed splitting

Fixed splitting refers to splitting with a constant closure interval without respiratory

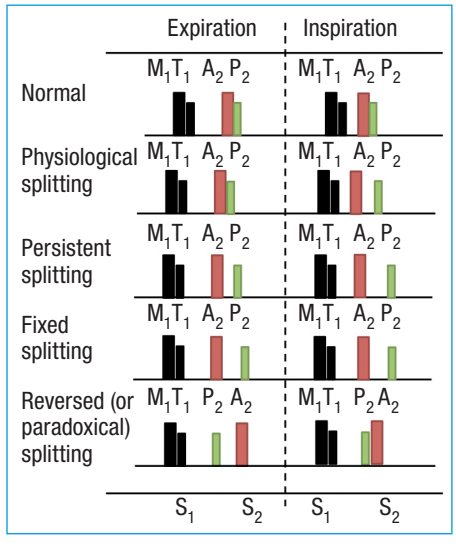

Figure 1. Splitting of the second heart sound.

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variation. This is usually the result of the presence of an atrial septal defect, which abnormally loads the right ventricle (left to right shunt), meaning the right ventricular volume continually exceeds the left ventricle, thus right ventricular systole and pulmonary valve opening are prolonged.

Extra heart sounds: clicks, snaps, knocks and plops

Extra heart sounds tend to be named onomatopoeically, for example, a tumour 'plop' is an early diastolic low-pitched sound just after S_2. This rare but characteristic sound occurs in atrial myxoma, if the tumour is large enough and its stalk long enough to allow it to move through the atrioventricular valve (typically the mitral valve). A mammary 'soufflé' is a rarely heard vascular bruit (systolic and diastolic components) with a blowing quality heard during pregnancy and until the end of lactation, radiating from the vascular breast tissue. Rarely, an early systolic ejection 'click' is caused by thickened aortic valve leaflets in aortic stenosis as opposed to an opening 'snap' which is caused by thickened valve leaflets, typically in mitral stenosis, early in diastole. A pericardial knock is heard during early diastole in constrictive pericarditis, a variant of S_3, as a result of rapid ventricular filling abruptly halted by the taut pericardium, preventing full diastole. Finally, in acute pericarditis, a friction rub is commonly audible which is said to resemble a crunch, like treading in fresh snow.

Flow murmur

Flow murmurs are also known as functional, physiological or benign murmurs. They arise as a result of increased flow across the cardiac valves, high output states, tachycardia, increased venous return or reduced systemic vascular resistance. Examples include pyrexia, anaemia, pregnancy or hyperthyroidism. They are typically soft, systolic, position dependent and without an accompanying thrill, in the absence of structural heart disease.

What are heart murmurs?

Normal blood flow is laminar and therefore inaudible. Blood flow becomes audible when laminar flow breaks down into disturbed or turbulent flow. This may occur for one of two reasons: increased flow across a normal valve or structure, i.e. a flow murmur, or normal flow across an abnormal structure. These two states may co-exist. While murmurs are important clinical signs, they should be interpreted in the context of the remainder of the clinical examination. In an undergraduate assessment, it is usually sufficient to detect a murmur and to formulate a list of likely differential diagnoses but in postgraduate assessment, one will be expected to look for evidence of aetiological factors, markers of severity, complications and decompensation.

Classification

When a murmur is detected, it should be systematically classified according to timing in the cardiac cycle, phonology, location, radiation, intensity, respiratory variation and tonal quality (*Figure 2*).

Timing

This is best measured relative to the carotid or subclavian pulse, which should be palpated while auscultation is being performed. Note whether the murmur occurs during systole or diastole, whether it occurs early, late or fills the whole of the phase.

Phonological shape

This refers to the intensity of the murmur over time – crescendo (increasing), decrescendo (decreasing) or crescendo-decrescendo (increasing then decreasing).

Location and radiation

In which valve area is the murmur heard loudest and which direction does it propagate? Murmurs radiate in the direction of the blood flow. For example, aortic stenosis radiates towards the carotids and mitral regurgitation towards the axilla.

Intensity

This refers to the amplitude of the murmur. It is graded according to the Levine scale (*Table 1*). Amplitude often correlates with the echocardiographic severity of valve disease but this is not always the case. In end-stage aortic stenosis, with left ventricular failure, reduced transvalvular flow causes a reduction in murmur volume despite worsening valve disease.

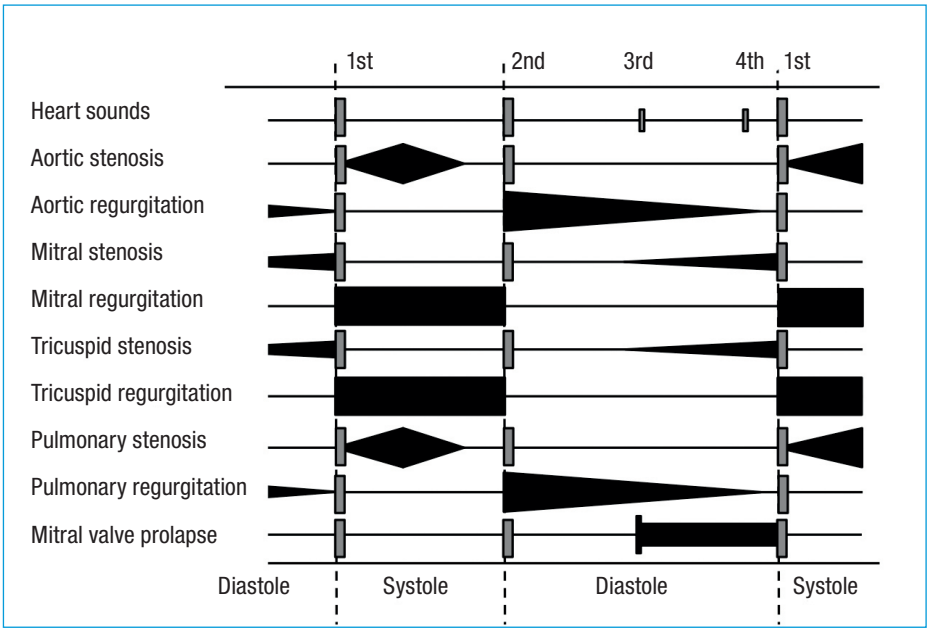


Figure 2. The phonology of heart sounds and associated murmurs.

Table 1. Levine scale of murmur intensity

1	The murmur is only audible upon considered, lengthy auscultation
2	The murmur is immediately audible upon auscultation, but faint
3	The murmur is loud upon auscultation, no palpable thrill
4	A loud murmur with a palpable thrill (palpable vibration on the chest wall)
5	A loud murmur audible with only superficial auscultation necessary, strong thrill
6	A loud murmur audible without auscultation with the stethoscope, strong thrill

From Freeman and Levine (1933)

Respiration

Does the murmur intensity vary with ventilation? Right heart flow increases on inspiration and through the left heart on expiration. Murmur amplitude rises and falls accordingly. This can be used to deduce if the murmur arises from the left or the right heart.

Quality

Additional, defining components should be noted. Does the murmur sound harsh, high- or low-pitched, rumbling, squeaky or blowing?

How to auscultate the heart sounds

Like all components of cardiovascular examination, auscultation should be interpreted within the wider clinical context of the patient's presentation. The examining doctor should be able to tailor his/her approach according to his/her findings. Undergraduates must first learn the physical steps of examination which, after practice, become second nature. At this point, the examining doctor focuses less on what to do and more on what signs he/she is eliciting. With more experience, the mind begins to interpret the signs and synthesize a list of possible and likely diagnoses. The following is one way of approaching cardiac auscultation; with practice and experience, doctors develop their own format and style.

Auscultate each valve area (*Figure 3*) with the stethoscope's diaphragm: mitral, tricuspid, aortic and then pulmonary. Listen during passive inspiration and expiration. By this point you should already have a good idea what the diagnosis might be. Next, do a second 'lap', this time using manoeuvres to amplify murmurs and to either consolidate or discount your working diagnosis, e.g.

1. Mitral region (fifth left intercostal space, mid-clavicular line): roll the patient on to his/her left side and listen on full expiration with the diaphragm and bell (for the low pitched mitral stenosis murmur)
2. Tricuspid region (lower left sternal edge): listen with the diaphragm with the patient leaning forward on expiration
3. Aortic region (right upper sternal edge, second intercostal space): listen with the diaphragm on expiration. If a murmur is heard, does it radiate towards the mitral region or into the carotids?
4. Pulmonic region (left upper sternal edge, second intercostal space)
5. Also listen to the point half way between the mitral and aortic regions (mid-left sternal edge) with the patient leaning forward on expiration (a common point at which aortic regurgitation can be heard).

Systolic murmurs

Aortic stenosis

In aortic stenosis an ejection systolic murmur is heard which is loudest at the right upper sternal border, and loudest when the patient leans forward and fully exhales. This characteristically radiates to the carotids. Slow rising carotid pulse and quiet A_2 are markers of severity.

Pulmonary stenosis

An ejection systolic murmur, loudest during inspiration at the left upper sternal border, is heard in pulmonary stenosis.

Mitral regurgitation

In mitral regurgitation there is a pan-systolic murmur, which is loudest at the mitral region. This can be accentuated by the patient lying on his/her left (which brings the apex towards the chest wall and stethoscope).

Tricuspid regurgitation

A pan-systolic murmur is heard in tricuspid regurgitation, which is loudest at the left lower sternal border, with radiation towards the left upper sternal border.

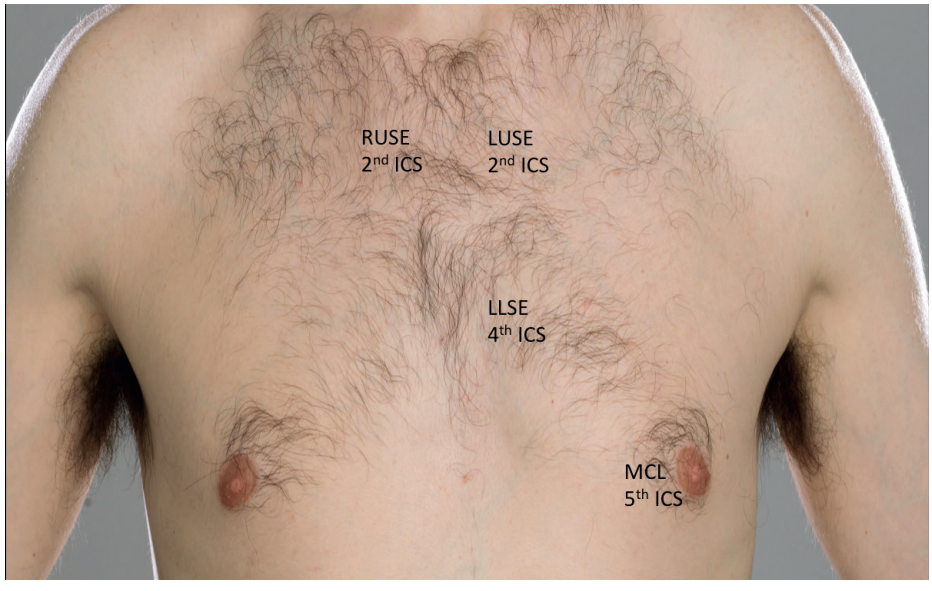
Atrial septal defect

A flow murmur can sometimes be heard in a patient with an atrial septal defect, loudest at the left upper sternal border, as a result of the increased volume of blood from left atrium to right atrium then flowing via the pulmonary valve.

Ventricular septal defect

In a patient with a ventricular septal defect, a pan-systolic murmur is heard which is loudest at the lower sternal border as a result of blood flow from the left ventricle to the right ventricle.

Figure 3. Picture of the praecordium with cardiac auscultation areas. ICS = intercostal space; LLSE = left lower sternal edge; LUSE = left upper sternal edge; MCL = mid-clavicular line; RUSE = right upper sternal edge.



Diastolic murmurs

Aortic regurgitation

In cases of aortic regurgitation an early diastolic, decrescendo murmur is heard. This is loudest at the mid-lower left sternal edge when the patient is leaning forward on expiration.

Pulmonary regurgitation

An early diastolic, decrescendo murmur is heard in patients with pulmonary regurgitation, loudest at the pulmonary area.

Mitral stenosis

In mitral stenosis there is a diastolic, low-pitched (hence using the bell of the stethoscope), rumbling murmur at the apex, amplified when the patient lies on his/her left side during expiration. The left ventricle must achieve a greater pressure to exceed the increased left atrial pressure in mitral stenosis,

this causes a delayed S_1 (closure of the mitral valve), because it takes longer to achieve that pressure, as well as a pre-systolic accentuation. There may also be an opening snap.

Tricuspid stenosis

This causes a rare, diastolic, decrescendo murmur, loudest at the left lower sternal border.

Table 2. Common causes of systolic cardiac murmurs

Disease	Aetiology
Aortic stenosis	Bicuspid aortic valve
	Calcific degeneration
	Congenital aortic stenosis
	Radiotherapy
	Rheumatic heart disease
	Sub-aortic membrane
	Williams syndrome
Mitral regurgitation	Chronic atrial fibrillation
	Endocarditis
	Left ventricle dilation
	Marfan syndrome
	Papillary muscle rupture
	Mitral valve prolapse
	Rheumatic heart disease
Pulmonary stenosis	Congenital pulmonary stenosis
	Carcinoid syndrome
	Tetralogy of Fallot
	Noonan syndrome
	Williams syndrome
	Subvalvar membrane
	Supravalvar membrane
Tricuspid regurgitation	Chronic atrial flutter
	Carcinoid syndrome
	Ebstein anomaly
	Tricuspid valve endocarditis
	Right ventricle dilation
	Myocardial infarction
	Pulmonary embolus

Additional murmurs

Patent ductus arteriosus

In patients with patent ductus arteriosus there is a continuous (throughout systole and diastole) machine-like murmur, which is loudest immediately inferior to the left clavicle, radiating to the back.

Table 3. Common causes of diastolic cardiac murmurs

Disease	Aetiology
Aortic regurgitation	Aortitis or arteritis
	Aortic valve endocarditis
	Ankylosing spondylitis
	Aortic dilation
	Aortic dissection
	Bicuspid aortic valve
	Calcific degeneration
Mitral stenosis	Atrial myxoma
	Cor triatriatum
	Double orifice mitral valve
	Mucopolysaccharidoses
	Mitral atresia
	Radiotherapy
	Rheumatic heart disease
Pulmonary regurgitation	Absent valve
	Carcinoid syndrome
	Pulmonary valve endocarditis
	Tetralogy of Fallot
	Pulmonary hypertension
	Prosthetic valve
	Pulmonary valve valvuloplasty
Tricuspid stenosis	Atrial myxoma
	Carcinoid syndrome
	Cardiac surgery
	Lupus
	Radiotherapy
	Rheumatic heart disease
	Triscupid atresia

KEY POINTS

- Auscultation should first consider the heart sounds.
- A pathological heart murmur is usually caused by either an incompetent or stenotic valve.
- The most commonly examined murmurs are aortic stenosis and mitral regurgitation.
- It is important to appreciate and analyse murmurs fully, not just detect them.

Coarctation of the aorta

A continuous machinery murmur is head in coarctation of the aorta, which is loudest during systole and best heard in the infraclavicular region.

Investigation

After a 12-lead electrocardiogram, a transthoracic echocardiogram should be performed. This assesses myocardial and valvular structure and function and will often reveal the underlying aetiology, such as a bicuspid aortic valve leading to aortic stenosis or papillary muscle dysfunction following a myocardial infarction leading to secondary mitral regurgitation (*Tables 2* and *3*). This will guide subsequent investigation, such as cardiac magnetic resonance imaging, transoesophageal echocardiogram or invasive cardiac catheterization.

Conclusions

Cardiac auscultation remains a key skill for all doctors, to corroborate the working diagnosis considered in the wider context of the patient's presentation. It is an oft-examined part of postgraduate qualifications, requiring not only a structured approach but also the use of manoeuvres to exploit differences in murmur characteristics. A useful free resource for readers can be found at https://www.easyauscultation.com/cases-anatomy?coursecaseorder=10&courseid=31 where different heart sounds can be heard. BJHM

Conflict of interest: none.

Freeman AR, Levine SA. The clinical significance of the systolic murmur: a study of 1000 consecutive "non-cardiac" cases. Ann Intern Med. 1933;6(11):1371–1385. https://doi.org/10.7326/0003-4819-6-11-1371