Cardiac auscultation

- It is an important aspect of the clinical cardiovascular examination, but auscultatory skills are decreasing with the almost universal availability of echocardiography.

- Auscultation requires a stethoscope equipped with a bell and a diaphragm. The bell emphasizes low-pitched sounds such as the murmur of mitral stenosis.

- The diaphragm filters out these sounds and helps to identify high-pitched sounds such as normal heart sounds and most systolic murmurs.
You must know:

the surface anatomy of the heart to understand how and where the sounds and murmurs radiate and

basic cardiac physiology to appreciate their timings.
Surface Projections of the Heart and Great Vessels

The right ventricle occupies most of the anterior cardiac surface and to the left of the sternum.

The left ventricle, behind the right ventricle and to the left, forms the left lateral margin of the heart. Its tapered inferior tip is often termed the cardiac “apex.”
The cardiac cycle
Augmented ventricular filling due to atrial contraction
Technique of cardiac auscultation

1. Where to listen - areas of auscultation
2. What and how to identify and describe by listening:
   - the first and second heart sounds
   - extra heart sounds (third and fourth heard in diastole)
   - additional sounds, e.g. clicks and snaps
   - pericardial rubs
   - murmurs in systole and/or diastole.
Where to listen-areas of auscultation

1. **Mitral**: at and around the cardiac apex. Sounds and murmurs arising from the mitral valve are usually heard best here.

2. **Tricuspid**: 5th intercostal space at the left sternal edge. Sounds and murmurs originating in the tricuspid valve are heard best here.

3. **Pulmonary**: 2nd intercostal space at the left sternal edge.

4. **Aortic**: 2nd intercostal space at the right sternal edge.

5. **Erb-Botkin**: 3rd intercostal space at the left sternal edge, for listening the murmur in aortic insufficiency.

These areas do not relate exactly to the anatomical position of the valves but are the areas at which the sound of each valve can be best heard.
Practice is needed here and many hearts should be listened to in order to be familiar with the normal sounds.

Make sure the **room is quiet** when auscultating.

**Position of the patient:**

The patient lying at approximately 45°.

Left lateral decubitus (bringing the left ventricle close to the chest wall) for listening murmur of mitral stenosis, leftsided S₃ and S₄

Sitting, leaning forward, exhaling completely and withholding breath for listening aortic regurgitation.

Standing up for listening mitral valve prolapse

Ask the patient to briefly stop breathing while you are listening.

Normal heart valves make a sound when they close (lub-dub) but not when they open.
Use a regular routine for auscultation.
Sequence of auscultation is not important, one can start either at the apex or at the base, just do not leave out any of the areas of auscultation.
You can then “go back” and concentrate on any abnormalities.
First step should be identifying the two heart sounds.
Simultaneously feel the carotid pulse with your thumb to time the sounds and murmurs. The carotid pulsation occurs with $S_1$.
Another way to recognize sounds is the duration of the pause: between sounds: the sound which appears after a longer pause (diastole) is $S_1$.
Listen with the diaphragm at each area and then repeat using the bell.
Listen over the carotid arteries and in the left axilla.
At each site:
- Identify the first and second heart sounds
- Assess their character and intensity
- Note any splitting (relation of splitting to respiratory phases, wideness of splitting)
- Concentrate in turn on systole (the interval between S1 and S2) and diastole (the interval between the S2 and S1).
- Listen for added sounds and then for murmurs (note location, timing, intensity, pitch, effect of respiration)
- Use special positions as mentioned above.
- Use inspiratory or expiratory apneea
- Note the character and intensity of any murmur heard.
- Sometimes listen after exercise.
Effects of respiration on auscultation

- If a murmur or sound is made louder by inspiration it is nearly always right sided since right heart blood flow is increased during inspiration.

- If a murmur is made louder by expiration it may be left sided, but this is not definite since expelling air from the lungs decreases the amount of air between the heart and chest wall and may increase the intensity of any event whether its source is right or left sided.
Normal heart sounds

Normal findings

$S_1$ and $S_2$ are usually the only heart sounds heard on auscultation of a normal heart, although in young and athletic subjects a soft third sound $S_3$ may be present.

The first heart sound ($S_1$)

Is caused by the closure of the mitral and tricuspid valves at onset of ventricular systole and

It is best heard at the apex (mitral area)

It has low intensity, it is low pitched and longer duration than $S_2$. 
The second heart sound (S2)

- Is caused by closure of the pulmonary and aortic valves at the end of ventricular systole, and
- Is best heard at the basis of the heart.
- The second sound is louder and higher pitched than the first sound, has a shorter duration and normally the aortic component is louder than the pulmonary one.
Physiological splitting of the second heart sound occurs because contraction of the left ventricle slightly precedes that of the right ventricle so that the pulmonary valve closes after the aortic valve.

This splitting increases at end-inspiration because the increased venous filling of the right ventricle further delays pulmonary valve closure.

This separation disappears on expiration.

Splitting of the second sound is best heard at the left sternal edge using the diaphragm.

On auscultation, the clinician hears 'lub d/dub' (inspiration) 'lub-dub' (expiration).
Heart sounds other than S1 and S2 are usually abnormal, but S3 and an ejection sound and very rarely S4 can occur in normal subjects.

A third heart sound (S3)

Is a low-pitched early diastolic sound best heard with the bell at the apex.

It coincides with rapid ventricular filling immediately after opening of the atrioventricular valves.

A third heart sound is therefore heard after the second as 'lub-dub-dum'.

A third heart sound is a normal finding in children, young adults and during pregnancy.
**Ejection sound:** an ejection sound occurs as the aortic or pulmonary valve opens and is close to S1.

Ejections sounds are sometimes heard in normal subjects but the most common cause in an asymptomatic patient is a bicuspid aortic valve.

S4 also called atrial sound is exceptionally rare in normal subjects.
Abnormalities of the heart sounds

- Intensity of heart sounds can be influenced by:
  - Thickness and elasticity of the chest wall
  - Elasticity and density of the lungs
  - Phases of respiration
  - Ventricle contractility and output
  - Distance from which valves are closing
  - Speed at which valves are closing
  - The consistency of the valves
  - Duration of PR interval
Pathological third heart sound
This is a **low frequency** (can just be heard with the bell) sound occurring just **after $S_2$** at the end of rapid ventricular filling, early in diastole and is caused by tautening of the papillary muscles or ventricular distension.

It can be heard as “Da-da-dum” or “ken-tuck-y”.

Is **usually pathological after the age of 40 years**

Combined with the normal heart sounds produces a **“triple rhythm“ or “gallop” rhythm** as it resembles galloping horses-protodiastolic or ventricular gallop

Left sided $S_3$ is best heard at the apex in the left lateral position during expiration

Right sided $S_3$ is best heard along the lower left sternal border or below the xifoid, in supine position, during inspiration
Clinical implication of a pathological S₃: LV impairment (decreased compliance) or increased filling.

The commonest causes are:
- left ventricular failure (IHD, hypertensive heart disease, MI, severe myocarditis)
- dilated cardiomyopathy,
- mitral regurgitation
- tricuspid regurgitation
- aortic regurgitation.

In cardiac failure S₃ is usually accompanied by a tachycardia and S₁ and S₂ are quiet.
4th heart sound (atrial sound or atrial gallop or pre-systolic gallop) occurs just before S1 in the late diastole.

**Clinical implication:** left ventricular impairment or hypertrophy.

It is soft and low pitched, best heard with the bell of the stethoscope at the apex.

It is caused by decreased compliance or increased stiffness of the ventricular myocardium.

Can be heard “Da-lub dub” or “Ten-ne-ssee”.
Coincides with abnormally forceful atrial contraction and raised end diastolic pressure in the left ventricle.

- It is almost always pathological.
- **Causes of left sided S4** (heard at the apex in left lateral position)
  - hypertrophic cardiomyopathy
  - aortic stenosis and
  - systemic hypertension
  - ischaemic heart disease
Causes of right sided S4 (best heard along the lower left sternal border or below the xifoid):

- Pulmonary hypertension
- Pulmonic stenosis
- Occasionally, a patient has both an S3 and an S4, producing a quadruple rhythm of four heart sounds.
- At rapid heart rates the S3 and S4 may merge into one loud extra heart sound, called a summation gallop.
- Additional or extra heart sounds
- Extra heart sounds in systole:
  - Ejection clicks
  - Mid-systolic clicks
- Extra heart sounds in diastole:
  - Opening snap
  - Pericardial knock
- Mechanical prosthetic valves
Extra heart sounds in systole:

Ejection click occur early in systole just after the first heart sound, caused by the opening of stiffened but not too calcified semilunar (aortic, pulmonary) valves or by systemic or pulmonary hypertension.

This is a high-pitched sound, sharp, clicking quality, which can be heard with the diaphragm of the stethoscope at the aortic or pulmonary areas and down the left sternal edge.

Clinical significance: indicate cardiovascular disease.

Causes:

Over the aortic area: aortic stenosis, bicuspid aortic valve, systemic hypertension

Over the pulmonary area: pulmonic stenosis, pulmonary hypertension
- **Midsystolic clicks**
- Occur in mitral valve prolapse (abnormal systolic ballooning of part of the mitral valve into the left atrium)
- The prolapsing mitral valve tenses in mid/late systole and this produces single or multiple clicks.
- May be associated with a late systolic murmur.
- They are high pitched, clicking in quality and best heard at or medial to the apex with the diaphragm.
- Squatting delays the murmur, standing moves it closer to S1.
Extra heart sounds in diastole:

- **Opening snap**
  
  An opening snap is commonly heard in mitral (rarely tricuspid) stenosis (if the valves are not calcified and almost immobile)
  
  The mitral valve normally opens immediately after $S_2$.
  
  In mitral stenosis, sudden opening of the stiffened valve, due to high atrial pressure can cause an audible **high-pitched snap, early in diastole, just after the second heart sound**. May be followed by the murmur of mitral stenosis.
  
  The opening snap of mitral stenosis is best heard at the apex and over the left sternal edge in 4-5 th ICS, with the diaphragm.
Pericardial knock:

- May be heard in early diastole.
- Appears in constrictive pericarditis.
- Is due to the high pressure atrium rapidly decompressing into a restricted LV producing an audible reverberation.
Extra heart sounds in systole and diastole: **pericardial friction rub**

- It is a superficial scratching, **high pitched** sound, comparable with creaking leather, best heard with the diaphragm of the stethoscope.
- Often has **systolic and diastolic** components.
- It may be audible over any part of the precordium, usually heard best in the **3rd interspace to the left of the sternum** (Erb Botkin) and is often very **localized**, does not radiate.
- **Intensity** varies over time, **increases** when the patient leans forward and during expiration.
- It is most often heard in acute **viral pericarditis** and sometimes **24-72 hours after myocardial infarction**.
MURMURS

Murmurs are audible vibrations produced by turbulent flow through the heart, across an abnormal valve, septal defect or outflow obstruction, or by increased volume or velocity of blood flow through a normal valve.

Murmurs are differentiated from heart sounds by their longer duration.

*Whatever constricts an orifice, whatever dilates a cavity, whatever establishes an orifice or cavity where none shall be, will disturb the even flow of blood and produce vibrations and a murmur.*

—Samuel Jones Gee (1839–1911)
Clinically murmurs can be:

- **Innocent murmurs**
- **Functional or relative murmurs**
- **Organic murmurs**

**Innocent murmurs**: no anatomic or physiologic abnormality exists

Occur in a healthy heart in situations where the circulation is hyperdinamic, e.g. normal children, during pregnancy, fever, anaemia and hyperthyroidism.

They are always systolic, usually soft or moderate in intensity and with musical quality.

It may require an echocardiogram to be sure that murmurs are innocent.
- **Functional or relative murmurs:**
  - Without anatomical lesions of the valves
  - They reflect cardiac diseases
  - Can be caused by:
    - Dilatation of great vessels
    - Dilatation of valvular orificies (secondary to left or right ventricle dilatation)
    - Papillary muscle dysfunction
    - Congenital heart disease (ASD, VSD)

- **Organic murmurs:** are produced by anatomical lesions of the valves, leading to stenosis or incompetence (insufficiency)
For each murmur heard, you should determine:

1. **Timing**: relation to cardiac cycle (systolic, diastolic, systolo-diastolic)
2. **Pattern** (shape)
3. **Intensity** (loudness)
4. **Location of Maximal Intensity**
5. **Radiation**
6. **Character and pitch**
7. **Variation of the murmur**: with position, with respiration, with exercise
1. **Timing**: relation to cardiac cycle (systolic, diastolic, systolo-diastolic)

Decide if you are hearing a **systolic murmur**, falling between S₁ and S₂, or a **diastolic murmur**.

Murmurs that coincide with the carotid upstroke are systolic.

Within systole or diastole they can be heard:
- At the beginning-early or proto-systolic, early or proto-diastolic
- In the middle: mid-systolic, mid-diastolic
- At the end: late systolic or telesystolic, late-diastolic or tele-diastolic or pre-systolic.

Throughout the systole: pansystolic or holosystolic.
2. Pattern (shape)

- The shape of a murmur is determined by its intensity over time.
- A crescendo murmur grows louder: MS presystolic (only in sinus rhythm)
- A decrescendo murmur grows softer: AI
- A crescendo–descrescendo murmur first rises in intensity, then falls: AS
- A plateau murmur has the same intensity throughout: MI
- Continuous: PDA
3. Intensity (loudness)

Based on Levine 6 grade classification expressed as a fraction (1/6).

**Grade 1** Very faint murmur, heard by an expert in optimum conditions.

**Grade 2** Soft, heard by a non-expert in optimum conditions.

**Grade 3** Easily heard, moderately loud, without an associated thrill (palpable murmur)

**Grade 4** Loud murmur with barely palpable thrill

**Grade 5** Loud murmur with easily palpable thrill. May be heard when the stethoscope is partly off the chest.

**Grade 6** Loud murmur with thrill and audible with stethoscope removed from chest wall.
4. Location of Maximal Intensity

Where the murmur is best heard
This is determined by the site where the murmur originates.
For example, a murmur best heard in the 2nd right interspace usually originates at or near the aortic valve.

5. Radiation—precardial and other (e.g. carotids) radiation of the murmur.
Murmurs radiate in the direction of the blood flow causing the murmur to specific sites outwith the precordium.
The radiation of cardiac murmurs is complex and any cardiac murmur from any structure can be heard anywhere in the chest.
There are, however some typical radiations.

The pansystolic murmur of mitral regurgitation radiates towards the left axilla.

The systolic murmur of ventriculoseptal defect towards the right sternal edge.

The ejection systolic murmur of aortic stenosis to the aortic area and the carotid arteries.
6. Character and pitch

The quality of murmurs is hard to define.

Terms such as harsh, blowing, musical, rumbling, high or low pitched are used.

Some examples:

blowing—MI

musical MI of ischaemic cause (papillary muscle dysfunction -Möweschrei), calcified mitral or aortic valves (degenerative mitral regurgitation or aortic stenosis)

rumbling—MS

sighing- AI

Harsh AS

machinery--PDA

High-pitched murmurs often correspond with high-pressure gradients, so the diastolic murmur of aortic incompetence is higher pitched than that of mitral stenosis.
7. Variation of the murmur:

- With position
- With respiration
- With exercise

Variation with position:

- Some murmurs will become louder if you position the patient so as to let gravity aid the flow of blood creating the sound.
- **Aortic regurgitation** is heard louder if you ask the patient to sit up, leaning forwards, and listen at the left sternal edge.
- **Mitral stenosis** is louder if you ask the patient to lie on their left-hand side (listen with the bell at the apex).
Variation with respiration

- Ask the patient to breathe deeply whilst you listen.
- Right-sided murmurs (e.g. pulmonary stenosis) tend to be louder during inspiration and quieter during expiration (because of increased venous return).
- Left-sided murmurs are louder during expiration.

Variation with exercise:

- Murmurs are louder after exercise
- Description of a murmur: a medium pitched, grade 3/6, blowing holosystolic murmur, best heard at the apex, radiating to the left axilla (mitral regurgitation)
SYSTOLIC MURMURS

1. Ejection systolic murmur
2. Pansystolic murmurs
3. Late systolic murmurs
A midsystolic murmur begins after \( S_1 \) and stops before \( S_2 \). Brief gaps are audible between the murmur and the heart sounds. Listen carefully for the gap just before \( S_2 \). It is heard more easily and, if present, usually confirms the murmur as midsystolic, not pansystolic.

A pansystolic (holosystolic) murmur starts with \( S_1 \) and stops at \( S_2 \), without a gap between murmur and heart sounds.

A late systolic murmur usually starts in mid- or late systole and persists up to \( S_2 \).
1. Causes of ejection systolic or myd-systolic murmurs (S1 and S2 can be heard clearly)

- *Increased flow through normal valves*
- 'Innocent systolic murmur':
  - fever
  - athletes (bradycardia, large stroke volume)
  - pregnancy (cardiac output maximum at 15 weeks)
- Atrial septal defect (causing high pulmonary flow)
- Severe anaemia
Normal or reduced flow through stenotic valve

- Aortic stenosis
- Pulmonary stenosis

Other causes of flow murmurs

- Hypertrophic obstructive cardiomyopathy (obstruction at subvalvular level)
- Aortic regurgitation (aortic flow murmur, relative stenosis)
- Aortic sclerosis

Physiopathology of ejection murmurs

- The flow of blood through a pathological structure generates the murmur and this flow is determined by the pressure difference on opposite sides of the responsible pathology (abnormal valve, septal defect, coarctation etc.)
The sound generated (murmur) is louder when the pressure difference is greater.

The murmur does not start until ejection begins and peaks when blood flow is greatest.

The murmur stops before S2 since ejection is finished.

Thus the murmur has a crescendo/decrescendo character.

The murmur is flow dependent, meaning, that it gets softer and may disappear if transvalvular flow starts to fall when a lesion is very severe and causes heart failure.
2. Causes of pansystolic or holosystolic murmurs

This is a murmur that lasts for the whole of systole, S₁ and S₂ cannot be heard in all areas.

All caused by a systolic leak from a high to a lower pressure chamber (regurgitant murmurs)

- Mitral regurgitation
- Tricuspid regurgitation
- Ventricular septal defect
- Leaking mitral or tricuspid prosthesis

Regurgitant systolic murmurs through the AV valves, e.g. mitral regurgitation, may start immediately isovolumic contraction begins, before ejection, since the leak occurs as soon as the pressure rises in the ventricle and continues up until S₂ or slightly beyond.
This is because there is a continuing pressure difference between the LV and the LA during this period of time.

Often the S2 is swamped by the end of the murmur.

3. Late systolic murmurs

There is an audible gap between S1 and the start of the murmur which then continues until S2.

Typically due to tricuspid or mitral regurgitation through a prolapsing valve.

In these cases the valve does not become incompetent until it has prolapsed and the murmur begins in mid- or even late systole and then continues up to and slightly beyond the second heart sound.

Late systolic murmur may have a crescendo rather similar to an ejection systolic murmur but are much later in the cycle.
DIASTOLIC MURMURS

1. Early diastolic murmur
2. Mid-diastolic murmur

Diastolic murmurs can be extremely difficult to hear.

They are often very low pitched and rumbling.

Almost always they are produced by a heart disease.
1. Early diastolic murmur

Early diastolic murmurs occur from regurgitation through incompetent aortic or pulmonary valves.

They are descrescendo and follow the S2. This is because the biggest pressure difference between the outflow vessel and the ventricle is at the beginning of the diastole.

They are heard at the left sternal edge (occasionally louder at the right sternal edge) and are most obvious in expiration with the patient leaning forward. (brings the heart closer to the chest wall)
Since the regurgitant blood volume must be ejected during the subsequent systole, significant aortic regurgitation leads to increased stroke volume and is almost always associated with a systolic flow murmur (relative stenosis).

Pulmonary regurgitation is uncommon. It may be caused by pulmonary artery dilatation in pulmonary hypertension (Graham Steel murmur) or to a congenital defect of the pulmonary valve.
2. **Mid-diastolic murmur**

A mid-diastolic murmur is usually caused by **mitral stenosis**.

This is a **low-pitched, rumbling** sound which may follow an opening snap.

It is best heard with the bell of the stethoscope at the **apex with the patient rolled to the left side**.

The murmum can be accentuated by listening **after exercise**.

The whole cadence sounds like 'lup-ta-ta-roo' where 'lup' is the loud first heart sound, 'ta-ta' the second sound and opening snap and 'roo' the mid-diastolic murmur.
If the patient is in sinus rhythm, left atrial contraction increases the blood flow across the stenosed valve leading to **presystolic accentuation** of the murmur. The murmur of tricuspid stenosis is similar but rare.

An **Austin Flint murmur** is a mid-diastolic murmur caused by the vibration of the mitral valve during diastole as it is hit by flow of blood due to **severe aortic regurgitation**.

**Carey –Coombs murmur**: occurs in patients with mitral valvulitis and secondary valve incompetence due to acute rheumatic fever.

It is a short, mid-diastolic rumble best heard at the apex, which disappears as the valvulitis improves.

Can be distinguished from the diastolic murmur of mitral stenosis by the absence of an opening snap before the murmur.

The murmur is caused by an increased blood flow across a thickened mitral valve. (relative stenosis)
Continuous murmurs are rare in adults. These are murmurs heard throughout both systole and diastole – murmur begins in the systole and continues through the second sound into all or part of diastole. The systolic component is usually louder than the diastolic component.

Causes:

1. Patent arterial duct
2. Shunt-acute communication between the right and the left side
3. Pericardial friction rub
4. Venous hum
1. Patent arterial duct, which connects the upper descending aorta and pulmonary artery in the fetus and normally closes just after birth.

The murmur is best heard at the upper left sternal border and radiates over the left scapula.

It is continuous and harsh described as 'machinery-like as it sounds like heavy machinery working in the background.

2. Most commonly in adults continuous murmurs are due to some acute communication developing between the right and the left side of the heart through which flow occurs both in systole and diastole. The commonest situation is a ruptured sinus of Valsalva, but infective endocarditis can lead to arteriovenosus and right/left heart communication.
3. Pericardial friction rub

- It is a superficial scratching, **high pitched** sound, comparable with creaking leather, best heard with the diaphragm of the stethoscope.
- Often has **systolic and diastolic** components.
- It may be audible over any part of the precordium, usually heard best in the 3rd interspace to the left of the sternum (Erb Botkin) and is often very **localized**, does not radiate.
Remember

- Auscultation remains an important clinical skill despite the ready availability of echocardiography.
- You must be able to detect abnormal signs to prompt appropriate investigation.
- Furthermore, certain auscultatory signs, such as the third or fourth heart sounds and pericardial friction, have no direct equivalent on echocardiography but are helpful prognostically.