Made by : Mahmoud Alhalawani

Cardio- vascular system

* chapter 4:- The cardiovascular system.

HEART

4.1:- Anatomy & physiology (Fig 4.1, 4.2)

- 2 muscular pump + pericardium:- allows movement with each heart beat & Respiration.
- Rt. heart:- pump deoxygenated blood from systemic veins to pulmonary circulation, ↓pressure.
- Lt. heart:- from Lungs to body tissue, ↑pressure.
- AV valves (tricuspid & mitral):- separates Atria & ventricles
- SL valves (pulmonary & Aortic):- separates ventricles from pulmonary & systemic arterial system.
- SA node:- specialised group of cells, cardiac pacemaker.
- diastole → AV close → systole → AV open → diastole

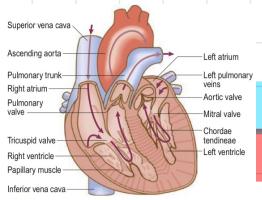
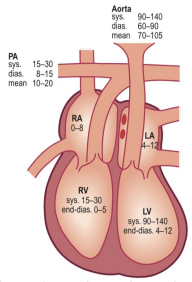


Fig. 4.1 The heart chambers and valves.



4.3 Cardiovascular causes of chest pain and their characteristics

	Angina	Myocardial infarction	Aortic dissection	Pericardial pain	Oesophageal pain
Site	Retrosternal	Retrosternal	Interscapular/retrosternal	Retrosternal or left-sided	Retrosternal or epigastric
Onset	Progressive increase in intensity over 1–2 minutes	Rapid over a few minutes	Very sudden	Gradual; postural change may suddenly aggravate	Over 1–2 minutes; can be sudden (spasm)
Character	Constricting, heavy	Constricting, heavy	Tearing or ripping	Sharp, 'stabbing', pleuritic	Gripping, tight or burning
Radiation	Sometimes arm(s), neck, epigastrium	Often to arm(s), neck, jaw, sometimes epigastrium	Back, between shoulders	Left shoulder or back	Often to back, sometimes to arms
Associated features	Breathlessness	Sweating, nausea, vomiting, breathlessness, feeling of impending death (angor animi)	Sweating, syncope, focal neurological signs, signs of limb ischaemia, mesenteric ischaemia	Flu-like prodrome, breathlessness, fever	Heartburn, acid reflux
Timing	Intermittent, with episodes lasting 2–10 minutes	Acute presentation; prolonged duration	Acute presentation; prolonged duration	Acute presentation; variable duration	Intermittent, often at night-time; variable duration
Exacerbating/relieving factors	Triggered by emotion, exertion, especially if cold, windy Relieved by rest, nitrates	'Stress' and exercise rare triggers, usually spontaneous Not relieved by rest or nitrates	Spontaneous No manoeuvres relieve pain	Sitting up/lying down may affect intensity NSAIDs help	Lying flat/some foods may trigger Not relieved by rest; nitrates sometimes relieve
Severity	Mild to moderate	Usually severe	Very severe	Can be severe	Usually mild but oesophageal spasm can mimic myocardial infarction
Cause	Coronary atherosclerosis, aortic stenosis, hypertrophic cardiomyopathy	Plaque rupture and coronary artery occlusion	Thoracic aortic dissection rupture	Pericarditis (usually viral, also post myocardial infarction)	Oesophageal spasm, reflux, hiatus hernia

4.1 Common symptoms of heart disease

Symptom	Cardiovascular causes	Other causes
Chest discomfort	Myocardial infarction Angina Pericarditis Aortic dissection	Oesophageal spasm Pneumothorax Musculoskeletal pain
Breathlessness	Heart failure Valvular disease Angina Pulmonary embolism Pulmonary hypertension	Respiratory disease Anaemia Obesity Anxiety
Palpitation	Tachyarrhythmias Ectopic beats	Anxiety Hyperthyroidism Drugs
Syncope/presyncope	Arrhythmias Postural hypotension Aortic stenosis Hypertrophic cardiomyopathy Atrial myxoma	Simple faints Epilepsy Anxiety
Oedema	Heart failure Constrictive pericarditis Venous stasis Lymphoedema	Nephrotic syndrome Liver disease Drugs Immobility

4.2 Canadian Cardiovascular Society: functional classification of stable angina

Grade	Description
1	Ordinary physical activity, such as walking and climbing stairs, does not cause angina. Angina with strenuous, rapid or prolonged exertion at work or during recreation
2	Slight limitation of ordinary activity. Walking or climbing stairs rapidly, walking uphill, walking or climbing stairs after meals, in cold, in wind, or when under emotional stress, or only during the few hours after awakening
3	Marked limitation of ordinary physical activity. Walking 1–2 blocks on the level and climbing less than one flight in normal conditions
4	Inability to carry on any physical activity without discomfort; angina may be present at rest

4.2:- The history

*4.2.1:- Common presenting symptoms.

1 Chest pain (Socrates)

- intermittent chest pain:- due to angina pectoris:- dull, band-like, anterior chest wall, radiate to arms & Jaw.
  - stable angina:- ↑in walking, cold & meal, relieved by GTN, 1o min (Box 4.2)
  - unstable angina:- at rest, more acute (<6w)
  - non-cardiac (oesophageal spasms):- normal cardiac enzymes & ECG.
- Acute chest pain:- MI, inflammation of pericardium, CT disease, after surgery, cath or radiotherapy.
  - MI:- + Restlessness, breathlessness, angor animi (feeling of impending death), Autonomic stimulation
  - Aortic dissection:- abrupt, tering pain, to back, + Autonomic stimulation (+ cranial artery → syncope, stroke, pulse asymetry) HTN, Marfan
  - pericardial pain:- radiate to shoulders, sharp or stabbing, ↑:- inspiration or Laying down, ↓:- sitting forwards, NSAID.
    Desler syndrom (after cath).

2 Dyspnoea (breathlessness) (Box 4.4)

- Heart failure:- most common, acute or chronic, prefear upright position.
- pulmonary embolism:- comfortable lying flat, may faint in upright position.
- Arrhythmias

4.4 Some mechanisms and causes of heart failure

Mechanism	Cause
Reduced ventricular contractility (systolic dysfunction)	Myocardial infarction Dilated cardiomyopathy, e.g. genetic, idiopathic, alcohol excess, cytotoxic drugs, peripartum cardiomyopathy Myocarditis
Impaired ventricular filling (diastolic dysfunction)	Left ventricular hypertrophy Constrictive pericarditis Hypertrophic or restrictive cardiomyopathy
Increased metabolic and cardiac demand (rare)	Thyrotoxicosis Arteriovenous fistulae Paget's disease
Valvular or congenital lesions	Mitral and/or aortic valve disease Tricuspid and/or pulmonary valve disease (rare) Ventricular septal defect Patent ductus arteriosus

4.5 New York Heart Association classification of heart failure symptom severity

Class	Description
I	No limitations. Ordinary physical activity does not cause undue fatigue, dyspnoea or palpitation (asymptomatic left ventricular dysfunction)
II	Slight limitation of physical activity. Such patients are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnoea or angina pectoris (symptomatically 'mild' heart failure)
III	Marked limitation of physical activity. Less than ordinary physical activity will lead to symptoms (symptomatically 'moderate' heart failure)
IV	Symptoms of congestive heart failure are present, even at rest. With any physical activity, increased discomfort is experienced symptomatically 'severe' heart failure)

- Types
  - Exertional dyspnoea:- CHF (Box 4.5)
  - angina equivalent:- Myocardial ischemia, + chest discomfort (Age or DM or Female), Same as angina.
  - orthopnoea:- dyspnoea on Lying flat (HF) → ↑VR → pulmonary edema, severity graded by number of pillows. (worse)
  - paroxysmal nocturnal dyspnoea:- Same mechanism, wakes the patient from sleep, DDx:- Asthma, Frothy or Blood-stained sputum.
- Acute Q:- duration, exertional symptoms, Associated symptoms (pain, Syncope, palpitation, Respiratory symptoms).
- chronic Q:- exertional dyspnea, degree of Limitation, posture effect, episodes, associated (ankle swelling, cough, sputum

3 palpitation:- unexpected or unplesant awareness of the heart beating in the chest.

- Questions
  - Nature:- Rapid, forceful, irrugular, can tap it out?.
  - timing:- speed, frequency & duration.
  - precipitants for symptoms or relieving factors.
  - associated symptoms:- presyncope, syncope or chest pain
  - History of underlying cardiac disease.

High risk features for life-threatening arrhythmia
1. Previous MI or cardiac surgery
2. Associated syncope or severe chest pain
3. Family hx of sudden death
4. Wolff-Parkinson-white syndrome
5. Significant structural heart disease (HCM, AS)

4.6 Descriptions of arrhythmias

	Extrasystoles	Sinus tachycardia	Supraventricular tachycardia	Atrial fibrillation	Ventricular tachycardia
Site	–	–	–	–	–
Onset	Sudden	Gradual	Sudden, with 'jump'	Sudden	Sudden
Character	'Jump', missed beat or flutter	Regular, fast, 'pounding'	Regular, fast	Irregular, usually fast; slower in elderly	Regular, fast
Radiation	–	–	–	–	–
Associated features	Nil	Anxiety	Polyuria, lightheadedness, chest tightness	Polyuria, breathlessness Syncope uncommon	Presyncope, syncope, chest tightness
Timing	Brief	A few minutes	Minutes to hours	Variable	Variable
Exacerbating/relieving factors	Fatigue, caffeine, alcohol may trigger Often relieved by walking (increases sinus rate)	Exercise or anxiety may trigger	Usually at rest, trivial movements, e.g. bending, may trigger Vagal manoeuvres may relieve	Exercise or alcohol may trigger; often spontaneous	Exercise may trigger; often spontaneous
Severity	Mild (usually)	Mild to moderate	Moderate to severe	Very variable, may be asymptomatic	Often severe

- Healthy people:- aware of their normal sinus rhythm (after exercise, stressful situation)
- Ectopic beats (extrasystoles):- at Rest & ↓by exercise, missed beat (incomplete filling) → Jolt/thump beat (overfilling).
- Supraventricular tachycardia:- Sudden paroxysms of rapid, regular palpitation, terminated with vagal stimulation (valsalva breathing maneuvres or carotid sinus pressure), young patient + No cardic disease.
- ventricular tachycardia:- + with presyncope/syncope, + cardiomyopathy or previous MI (family history, wolff-parkinson-white-syndrom

4 Syncope & presyncope

- Syncope :- transient loss of consciousness due to cerebral hypotention
  - causes :- postural hypotension, neurocardiogenic syncope, arrhythmias, mechanical obstruction of CO, PE
  - Questions
    - circumstance :- palpaitation, chest pain, Lightheadedness, nausea, tinnitus, sweating or visual disturbance
    - duration :- any injury ?
    - time of recovery.
    - driving state (occupational).
- presyncope :- sensation of Lightheadendness & impending loss of conciousness (not actual).
  - Questions
    - nature & associated symptoms.
    - preciptants :- postural changes, prolonged standing, intense emotion or exertion
    - frequency of episodes & impact on lifestyle.
    - Drugs (Anti-HTN) (Box 4.7).

★causes★

- postural hypotension :- ↓>20 mmHg in systolic BP on standing, +tachycardia, caused by
  - Hypovolaemia
  - drugs
  - Autonomic neuropathy

  , 1/3 of >65y.
- neurocardiogenic (hypersensitivity carotid sinus syndrome, vasovagal).
  - abnormal autonomic reflex → ↓HR (bradycardia) ± vasodilation
  - ↑standing in warm or painful, emotional stimuli.
  - slides to floor → Aid cerebral circulation → wake up (flushing or vomiting).
  - Held upright → cerebral hypoprefusion → seizure or mistake dx of epilepsy.
- Arrtyhmias :- SA disease (AV block), Drugs, Afib (rare), supraventricular tachyarrthmias.
- mechanical obstruction of Lt.V outflow :- aortic stenosis, hypertropic (exertion).
- pulmonary embolism :- obstructing Rt.V outflow, +acute dyspnoea, chest pain & hypoxia.
- cardiac tumors (myxoma, thrombosis, ↓prosthetic heart valve).

5 Oedema :- ↑fliud in interstiail space, gravity-dependent, in ankles or over sacrum.

- uniLateral :- DVT.
- BiLateral :- HF (mostly), chronic venous disease, Drugs (ca⁺ antagonist), ↓albumin, ↑JVP

4.7 Symptoms related to medication

Symptom	Medication
Angina	Aggravated by thyroxine or drug-induced anaemia, e.g. aspirin or NSAIDs
Dyspnoea	Beta-blockers in patients with asthma Exacerbation of heart failure by beta-blockers, some calcium channel antagonists (verapamil, diltiazem), NSAIDs
Palpitation	Tachycardia and/or arrhythmia from thyroxine, β2 stimulants, e.g. salbutamol, digoxin toxicity, hypokalaemia from diuretics, tricyclic antidepressants
Syncope/ presyncope	Vasodilators, e.g. nitrates, alpha-blockers, ACE inhibitors and angiotensin II receptor antagonists Bradycardia from rate-limiting agents, e.g. beta-blockers, some calcium channel antagonists (verapamil, diltiazem), digoxin, amiodarone
Oedema	Glucocorticoids, NSAIDs, some calcium channel antagonists, e.g. nifedipine, amlodipine

6 other symptoms of cardiac disease

- endocarditis :- ↓weight, tiredness, fever, night sweet (dentist).
- vegetations & atria myxoma :- stroke, acute Limb ischemia, acute mesentric ischemia.
- Advanced HF :- abdominal distention (ascites), ↓weight, muscle wasting (cardiac cachexia :- ↑catebolic state).

★4.2.2 :- past medical History

- Questions
  - conditions associated with ↑RisK :- HTN, DM, ↑Lipids.
  - RF or Heart murmurs during childhood.
  - Bacteremia causes (endocarditis) :- skin infection, dental, IV drug, penetrating trauma.
  - Systemic disorders
    - CT disease :- pericarditis & Raynaud's phenomenon.
    - Marfan syndrom :- aortic dissection.
    - myotonic dystrophy :- AV block.

4.8 Key elements of the past cardiac history

	Ischaemic heart disease	Heart failure	Valvular disease
Baseline symptoms	Exertional angina? If so, ascertain functional limitation (see Box 4.2)/response to GTN spray	Dyspnoea, fatigue, ankle swelling Record usual functional status (see Box 4.5)	Often asymptomatic Exertional dyspnoea (common), chest pain or syncope
Major events	Previous myocardial infarction/unstable angina	Hospitalisation for decompensated heart failure Ventricular arrhythmias	Infective endocarditis Previous rheumatic fever
Investigations	Coronary angiography (invasive or computed tomography): presence, extent and severity of coronary artery disease Exercise electrocardiogram (or other stress test): evidence of inducible ischaemia? Exercise capacity and symptoms	Echocardiogram (± cardiac magnetic resonance imaging): left ventricular size, wall thickness and systolic function; valvular disease; right ventricular function	Echocardiogram (transthoracic ± transoesophageal): nature and severity of valve lesion; ventricular size and function
Procedures	Percutaneous coronary intervention (angioplasty and stenting) Coronary artery bypass graft surgery	Implantable cardioverter–defibrillator Cardiac resynchronisation therapy	Surgical valve repair or replacement (note whether mechanical or bioprosthetic) Transcatheter valve procedures

★4.2.3 :- family history (Box 4.7).

- cardiomyopathies (genitic)
- Coronary Artery disease - <65y ♀, <55y male (estrogen dilate the Artery).
- venous thrombosis :- inhereited thrombophilia (factor v Leiden mutation).
- inheritied arrthymia.
- familial hypercholesterolaemia :- premature arterial disease (<55y ♀, <45 male).

★4.2.4 :- Social history

- smoking :- strongest factor for coronary & peripheral Arterial disease.
- Alchol :- Afib, HTN & dilated cardiomyopathy
- Recreational drugs (cocaine & amphetamines) :- arrthythmias, chest pain, MI, occlustive & Aneurysmal peripheral.
- Commerical drivers & pilots :- IHD & arrthmias.

●4.3 :- The physical examination

★4.3.1 :- General examination

- apperance :- unwell, distress, breathlessness, cynosis, Pweigh, cachectic, Marfan, down, Turner, ankylosing spondylitis
- skin :- petechiae, Temp, preforming urinalysis (Heamatourea (endocarditis, vasculitis), glucosuria (DM), proteinuria (HTN, Renal

1 Hands

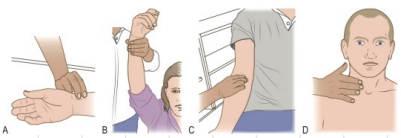
- Temp :- endocarditis & pericarditis (Normal warm) → cold + ↑time :- ↓prefusion (shock, ↓CO)
- Capillary refill time. (normal 2 sec)
- tobacco staining.
- skin crease pellor :- anemia.
- pheripheral cynosis
- Nails :- clubbing, splinter hemorrhages (Red-brown mark along axis). → normal, trauma, endocarditis, vasculitis
- extensor surface :- tendon xanthomata :- hard, slightly, yellow masses, Lipid deposition → familal ↑lipid.
- palmar aspect
  - Janeway Lesion :- painless, blanching red macules on thenar/hypothenar eminence.
  - osler's node :- painful raised erthyromatous lesion on fingers pads. → rare in endocarditis
- IV/Tremor.

2 Face

- mouth :- central cynosis → HF, CHD
- eyelid :- Xanthelasmata (yellow plagues)
- iris :- Corneal arcus (yellow discoloration) → ↑lipid → MI
- fundi :- HTN, DM, Roth's spots (endocarditis). By fundoscopy exam.
- cheeks :- malar flush (mitral stenosis).

★4.3.2 :- Arterial pressure (RRCCV)

- Rate :- number of pulses/min → Radial Artery
- Rhythm :- pattern or regularity of pulses
- Volume :- perceived degree of pulses
- Chercter :- impression of pulse waveform or shape. → bracial, carotid, femoral
- compressability.
- Radial pulse
  - place pads → 15 secx4
  - collapsing pulse → Fell pulse → Raise arm (A. Regurtiation).
  - palpate both Rt & Lt.
  - •3 tests → Radio-Radical. / Radio-femoral. / pulse deficent :- >10 → AFib



- Bracial pulse :- in antecubital fossa, medial to biceps tendon.
- carotid pulse :- Between Larynx & anterior sternocleidomastoid M, never simultaneous (vagal Reflux), BRUIT test.

1 Rate & Rhythm :- normally 60-100 bmp (consider clinical contex)

- Bradycardia (<60 bpm)
  - Drugs (B-blockers).
  - athletic condition
  - SA node or AV node dysfunction
- tachycardia (>100 bpm)
  - Drugs (sympathomimeitcs)
  - sinus tachycardia
  - fever, pain excercise
  - hyperthyroidsm
- Regular :- Sinus rhythm
- iregular :- extrasystoles, 2° AV block, Afib (depends on AV, up to 200 bpm)
  - ↳ regular irregular
  - ↳ irregularly irregular.

4.9 Causes of abnormal pulse rate or rhythm

Abnormality	Sinus rhythm	Arrhythmia
Fast rate (tachycardia, >100 bpm)	Exercise Pain Excitement/anxiety Fever Hyperthyroidism Medication: Sympathomimetics, e.g. salbutamol Vasodilators	Atrial fibrillation Atrial flutter Supraventricular tachycardia Ventricular tachycardia
Slow rate (bradycardia, <60 bpm)	Sleep Athletic training Hypothyroidism Medication: Beta-blockers Digoxin Verapamil, diltiazem	Carotid sinus hypersensitivity Sick sinus syndrome Second-degree heart block Complete heart block
Irregular pulse	Sinus arrhythmia Atrial extrasystoles Ventricular extrasystoles	Atrial fibrillation Atrial flutter with variable response Second-degree heart block

4.11 Common causes of atrial fibrillation

- Hypertension
- Heart failure
- Myocardial infarction
- Thyrotoxicosis
- Alcohol-related heart disease
- Mitral valve disease
- Infection, e.g. respiratory, urinary
- Following surgery, especially cardiothoracic surgery

- inspiration:- ↓parasymp tone, ↑HR
- expiration:- ↓HR

4.10 Haemodynamic effects of respiration

	Inspiration	Expiration
Pulse/heart rate	Accelerates	Slows
Systolic blood pressure	Falls (up to 10 mmHg)	Rises
Jugular venous pressure	Falls	Rises
Second heart sound	Splits	Fuses

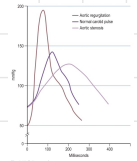
4.12 Causes of increased pulse volume

Physiological
- Exercise
- Pregnancy
- Advanced age
- Increased environmental temperature

Pathological
- Hypertension
- Fever
- Thyrotoxicosis
- Anaemia
- Aortic regurgitation
- Paget's disease of bone
- Peripheral atrioventricular shunt

2 Volume & charcter :- ↑diastolic intervals → ↑SV → ↑pulse volume on examination.

- ↑pulse volume :- physiological or pathological
- ↓pulse volume → inadequate ventricullar filling :- hypovolemia, cardiac tamponade, mitral stenosis
- Asymmetric pulses :- occlusive peripheral arterial disease, stenoss, rarly aortic dissection
- coarctation :- congenital aorta narrowing, Radiofemoral delay (upper Limb > Lower), HTN & HF.
- slow-rising pulse :- gradual upstroke + ↓peak in Late systole :- sever aortic stenosis
- collapsing pulse :- early peak followed by rapid fall (systolic BP - diastolic BP > 80 mmHg) :- sever aortic regurgitation
- pulsus bisferiens :- double systolic pead seperated by systolic dip :- aortic stenosis & regurigitation, HCOM.
- pulsus alternans :- beat-to-beat variation in volume + normal rhthym :- HF
- pulsus paradoxus :- exaggeration of normal variabilly of volume with breathing :- ↑intrapericardial pressure (tamponade or asthma)
  - a fall of >10 mmHg in inspiration



★4.3.3 :- Blood pressure :- pressure that the circulating exerts against arterial wall.

- systolic pressure :- max pressure during ventricular contraction
- dystolic pressure :- Lowest value of pressure during ventricular filling.
- measured → by :- 1) sphygmomanometer, 2) intra-artrial catheter + pressure sensor (ICU).
  - systolic pressure / diastolic pressure, where, how.

4.13 British Hypertension Society classification of blood pressure (BP) levels

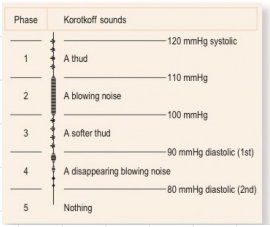
BP	Systolic BP (mmHg)	Diastolic BP (mmHg)
Optimal	<120	<80
Normal	<130	<85
High normal	130–139	85–89
Hypertension		
Grade 1 (mild)	140–159	90–99
Grade 2 (moderate)	160–179	100–109
Grade 3 (severe)	>180	>110
Isolated systolic hypertension		
Grade 1	140–159	<90
Grade 2	>160	<90

4.14 Clinical clues to secondary hypertension

Clinical feature	Cause
Widespread vascular disease Renal bruit	Renovascular disease, including renal artery stenosis
Episodes of sweating, headache and palpitation	Phaeochromocytoma
Hypokalaemia	Primary aldosteronism
Cushingoid facies, central obesity, abdominal striae, proximal muscle weakness Chronic glucocorticoid use	Cushing's syndrome
Low-volume femoral pulses with radiofemoral delay	Coarctation of the aorta
Bilateral palpable kidneys	Adult polycystic kidney disease (p. 243)

1 HTN :- systolic ≥140 mmHg ± diastolic ≥90 mmHg

- associated with :- HF, CAD, cerebrovercular disease, CKD.
- Asymptomatic HTN :- mostly.
- symptomatic HTN :- in sever HTN :- headaches & visual disturbances
- essential HTN :- most, no identifable cause.
- secondary HTN :- <1% (rare) (Box 4.14).
- white-coat HTN :- ↑BP in healthcare setting.
- End-organ damge
  - cardiac :- HF
  - Renal :- CKD, proteinurea.
  - eye :- hypertensive retinopathy.



2 KorotKoff sounds :- cuff pressure is between systolic & diastolic because the artery collapse completely & reopens with each Heart beat, producing a snapping or Knocking sound.

- phase 1 :- first apperance, systole → phase 4 :- muffle → phase 5 :- disapper

3 examination sequence

- Rest the patient for 5min.
- measure BP in both brachial arteries, higher → Tx.
- support the arm, with no tight clothing
- Good cuff size in centre of bladder over brachial Artery.
- palpate bracial pulse.
- inflate the cuff → impable → note pressure on manometer → systolic pressure.
- inflate +30mmHg → Listen, placed on brachial Artery.
- Deflate slouly (2-3 mmHg) → regular tapping sound (I) → systolic pressure.
- Deflate slowly → sound disappear (V) → diastolic
- if muffled sounds persist (IV) & don't disappear → diastolic.

4 Common problems in BP measurement

- Different BP in each arm :- >10 differ :- Aortic or subclavian disease.
- wrong cuff size:- standart cuff bladder (13 x30 cm), obese → ↑size , child → ↓size.
- Auscultatory gap:- in elder HTN , Korotkoff sound dissapear → palpate systolic first.
- patient arm at wrong LvL:- the elbow should be at LvL of heart.
- postural change .
- Atrial fibrillation:- SV & BP vary from Beat to Beat → more slowly + more than time.

★4.3.4 :- Jugular venous pressure (JVP) & waveform.

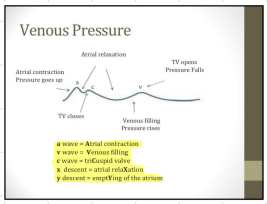
4.15 Differences between carotid artery and jugular venous pulsation

Carotid	Jugular
Rapid outward movement	Rapid inward movement
One peak per heart beat	Two peaks per heart beat (in sinus rhythm)
Palpable	Impalpable
Pulsation unaffected by pressure at the root of the neck	Pulsation diminished by pressure at the root of the neck
Independent of respiration	Height of pulsation varies with respiration
Independent of the position of the patient	Varies with the position of the patient
Independent of abdominal pressure	Rises with abdominal pressure

- JVP :- LvL of pulsation in internal Jugular vein = Rt.A.pressure (< 7mmHg, 9 cmH_2O).
  - ≤ 4 cm above sternal angle (45°) → ↓Rt.A.pressure :- Lie flat
    - ↑Rt.A.pressure :- stand.
- waveform :- 2 main peaks/cycle , distinguish carotid arterial pulse.
- Examination
  - Rt side , 45°dgree, resting in pillow & turned to Left (skin are relaxed).
  - identify the jugular vein pulsation behind the sternocleidomastoid muscle.
  - identify the timing & wave form.
  - waveform
    - a wave :- Rt.A contraction (Afib → No wave).
    - v wave :- atrial filling (v.systole , T.closed).
    - c wave (rare) :- closure of tricusped valve.
    - x & y descent.
  - Abdominojugular test :- press over abdomen (Liver) → ↑VR → ↑JVP.
  - changes with Respiration :- inspiration → ↓intrathoracic pressure → ↓JVP.
  - changes in position :- Flat → ↑JVP , sitting → ↓JVP.
  - occlusion test :- in neck Base → ↓JVP.
  - measure JVP heigh :- +5cm .

4.16 Abnormalities of the jugular venous pulse

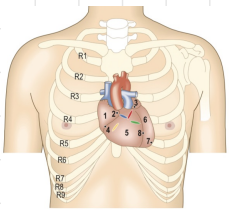
Condition	Abnormalities
Heart failure	Elevation, sustained abdominojugular reflux >10 seconds
Pulmonary embolism, tamponade	Elevation
Pericardial effusion	Elevation, ~~prominent~~ 'y' descent
Pericardial constriction	Elevation, Kussmaul's sign, prominent 'y' descent
Superior vena cava obstruction	Elevation, loss of pulsation
Atrial fibrillation	Absent 'a' waves
Tricuspid stenosis	~~Giant~~ 'a' waves Large, prominent
Tricuspid regurgitation	Giant 'v' or 'cv' waves
Complete heart block	'Cannon' waves

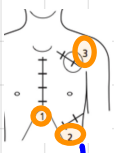


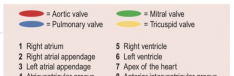
- ↑JVP :- fluid overload (HF), pulmonary edema & effusions, ascites, periphral edema, ↑Rt.v.filling pressure (pulmonary embolism & pulmonary HTN & cardiac tamponade & pericardial constriction), panncost tumor, Kussmal's sign.
- abnormal a wave
  - prominent a wave :- delay or restricted Rt.v.filling (pulmonary HTN & tricuspid stenosis)
  - cannon waves :- Rt.A contracts aganist a closed tricuspid valve.
    - irregular :- complete Heart block, Atrioventricular dissociation.
    - regular :- Junctional rhythm, ventricular & supraventricular tachycardia.
- ★Kusmmal sign & svc obstruction slides. (Last 2).
- prominant systolic v wave (cv) :- Tricuspid regurgitation , pulsatile Liver.

★4.3.5 :- precordium :- anterior chest surface overlying the heart & great vessels

- The optimal sites of auscultation don't correspond with location but sounds are best heard there





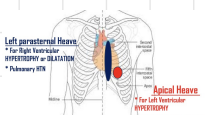


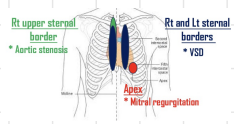
1 Inspection

- pectus excavetum & carinatum :- displace the heart.
- 1 midline sternomy scar :- valve replacment & coronary A bypass.
- 2 Lt.Submammary scar :- mitral valvotomy or transapical transcatheter aortic valve implantation.
- 3 intraclavicular scar :- pacemaker or defibrillator implatation.

2 palpation

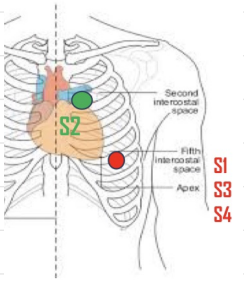
- Apex beat :- 5t intercostal, mid-clavicular
- Heave :- palpable impulse → Lift your hand.
  - Rt :- at Lt.parasternal :- Rt.V. hypertrophy (pulmonary HTN).
  - Lt :- at apex :- Lt.V.hypertrophy (HTN, A.stenosis).
- thrill :- tactile equivalant of murmurs (palpable vibration) above 4G
  - apex :- M. Ryurgitation.
  - lt.parasternal :- VSD
  - Rt.parasternal :- VSD
  - Rt.upper sternal :- A.S





- Abnormal Apex beat
  - impalpable :- overweight, muscular, asthma, emphysema
  - displaced :- Lt.V.dilation (MI, cardiomyopathy, aortic regurgitation or stenosis).
  - on Rt.side :- dextrocardia (rare).
  - forceful undisplaced :- HTN, aortic stenosis.
- .Tabbing :- mitral stenosis
- .double :- hypertrophic

3 Auscultation (8 diaphragm, 2 bell).



- Stethoscope
  - Diaphragm :- 1st & 2nd, ↑pitch (early diastolic murmur of aortic regurgitation, friction rub).
  - bell :- 3rd & 4th, diastolic murmur of mitral stenosis.
- Examination
  - carotid A :- ejection systolic murmur (Aortic stenosis & carotid bruits).
  - Lt. axilla :- pansystolic murmur of mitral regurgitation.
  - S_1 & S_2 (charcter, intesity, splitting, intervals).
  - Lt. Roll apex :- mid-diastolic murmurs of Mitral stenosis.
  - sit + Lean + breathout → Rt. 2nd intercostal :- aortic regurgitation.
- Heart Sounds
  - 1st heart sound (Lub)
    - caused by :- closure of AV valves, ventricular systole.
    - Best heard :- apex (mitral valve)
    - abnormality :- ↑intesity in mitral stenosis

4.18 Abnormalities of intensity of the first heart sound

Quiet	
• Low cardiac output • Poor left ventricular function • Rheumatic mitral regurgitation	• Long P–R interval (first-degree heart block)
Loud	
• Increased cardiac output • Large stroke volume	• Mitral stenosis • Short P–R interval • Atrial myxoma (rare)
Variable	
• Atrial fibrillation • Extrasystoles	• Complete heart block

  - 2nd heart sound (Dub)
    - caused by :- closure of SL valves, end of V.systole.
    - Best heard :- Lt. sternal edge (pulmonary valve)
    - Louder & ↑pitched than S_1.
    - splitting
      - physiological :- aortic close faster.
        - end-inspiration :- ↑ due to ↑venous filling of Rt.V delay pulmonary closure (Lub-d-dub)
        - expiration :- dissapear (Lub-dub)
      - Abs :- clacific aortic stenosis & ↓ in aortic regurgitation.
      - ↑ :- HTN, Pulmonary HTN.
      - wide splitting :- Delay Rt.V emptying (Rt. Bundle branch block or p HTN, VSD, Pulmonary stenosis)
      - Fixed splitting :- ASD.
      - reversed splitting :- Lt.V empting delay (Lt. BBB & Lt.V. outflow obstruction). A. stenosis, HCOM

4.19 Abnormalities of the second heart sound

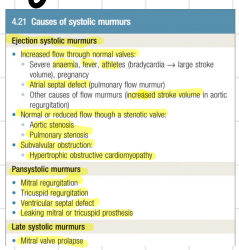
Quiet
• Low cardiac output • Calcific aortic stenosis • Aortic regurgitation
Loud
• Systemic hypertension (aortic component) • Pulmonary hypertension (pulmonary component)
Split
Widens in inspiration (enhanced physiological splitting) • Right bundle branch block • Pulmonary stenosis • Pulmonary hypertension • Ventricular septal defect
Fixed splitting (unaffected by respiration) • Atrial septal defect
Widens in expiration (reversed splitting) • Aortic stenosis • Hypertrophic cardiomyopathy • Left bundle branch block • Ventricular pacing

  - 3rd heart sound (S_3)
    - caused by :- Rapid V.filling. (Lub-dub-dum), in early diastole
    - Best heard :- bell at apex.
    - ↓pitched early diastolic
    - normal in :- children, febrile, pregrency, young adults
    - abnormal in :- after MOI
      - Lt.V failure (+tachycardia) → gallop rhythm (Lub-da-dub)
      - mitral regurgitation.
  - 4th heart sound (S_4)
    - caused by (pathological) :- forceful atrial contraction against non-compliant ventricle
    - Best heard :- Bell at apex.
    - soft & ↓pitched, Before S_1 (da-Lub-dub)
    - pathological :- Lt.V. hypertrophy :- HTN, Aortic stenosis, HCOM
    - cannot occur :- AFib (no atrial contraction).
  - Added Sounds
    - opening snap
      - in :- mitral stenosis
      - from :- sudden opening of valve in early diastole, after S_2.
      - best heard :- diaphragm at apex (mitral valve).
    - Ejection clicks
      - ↑pitched, diaphragm, after S_1 (early systole).
      - in :- Congenital pulmonary or aortic stenosis.
      - don't :- calcific aortic stenosis (cups are rigid).
    - mid-systolic clicks
      - ↑pitched, apex with diaphragm.
      - in :- mitral valve prolapse. (+ Late systolic murmur).
    - mechanical valve (prosthetic valve)
      - ↑pitched, palpable, may heard without stethoscope (during systole).
      - metalic S_1 :- Loud early diastole. mitral valve closure → span opening
      - metalic S_2 :- like ejection click, start of systole. a. closure → EC
    - pericardial Rub (friction)
      - best heard :- holding expiration
      - Localised, varying in intesity
      - in :- acute pericarditis or post MI.
      - pleuropericardial Rub, pneumopericardium → crunching.

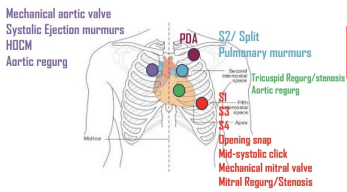
- Murmurs :- Turbulent flow across abnormal valve, septal defect or obstruction
  - innocent :- ↑velocity (↑SV) :- pregnant, athletes, fever, always systolic
  - Examination
    - timing
      - Systolic :- S_1, AV closure
      - Diastolic :- between S_2 & S_1
        - Early :- isovolumic relaxation.
        - mid :- early v.filling S_3, AV stenosis → SL regurgitation
        - presystole :- S_4
    - Duration
      - AV regurgitation :- S_1 → muffing or obscuring → throughout systole (pansystolic).
      - mitral prolapse :- Late systolic
      - SL stenosis :- after S_1 → Max mid-systole → fades before S_2.
    - character & pitch
      - ↑ pitched :- ↑ pressure gradient.
      - harsh, blowing, musical, rumbling
    - intensity
      - Diastolic > 3
      - intesity ≠ valve dysfunction
      - Rapidly changing :- infective endocarditis.
    - Location → Helpfull in diastolic murmurs (Mitral stenosis → apex, Aortic regurgitation → Lt.sternal edge)
    - Radiation
      - pansystolic of mitral regurgitation :- Lt.axilla
      - VSD :- Rt.sternal edge
      - Aortic stenosis :- Suprasternal notch & carotid Artery.

4.20 Grades of intensity of murmur	
Grade	Description
1	Heard by an expert in optimum conditions
2	Heard by a non-expert in optimum conditions
3	Easily heard; no thrill
4	A loud murmur, with a thrill
5	Very loud, often heard over a wide area, with thrill
6	Extremely loud, heard without a stethoscope

  - Systolic murmurs
    - ↑flow, turbelent flow,
    - aortic stenosis :- All over precordium, harsh, ↑pitched, musical, radiates, Load, thrills.
    - pansystolic
      - M.regurgitation (Loud, bowling, apex, Lt.axilla).
      - T.regurgitation (Lower sternal edge, prominant v, pulsatile Liver).
      - VSD :- Left sternal border, Radiates to Rt, thrills
    - Late systolic :- mitral valve prolapse
    - Ejection systolic :- HCOM, A.stenosis & Regugation, pulmonary stenosis, ASD, Asthma, Athlete

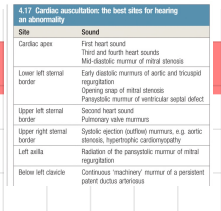


  - Diastolic murmurs
    - Early diastolic :- aortic regurgitation (Erbs area (Lt.3rd) Lean + held expiration)
    - Graham steell :- P.regurgitation (P.Artery dilation in P.HTN or Congenital defect).
    - mid-diastolic :- M.stenosis (↓pitched, rumble, open-snap, bell at apex, patient to Lt.side (Lub-ta-ta-ma)
      - Austin flint :- A.regurgitation (restricting the inflow to Lt.V).
  - continous murmurs → patent ductus arteriouses :- fetus Aorta + pulmonary Artery (upper Lt.sternal border & radiates over Lt.scapula, manchinery-like). at Lt.infraclaveclular.
  - crackles, ascites, Bruit, Lower Limb & sacral edema



★4.4 :- Interpresenting of findings

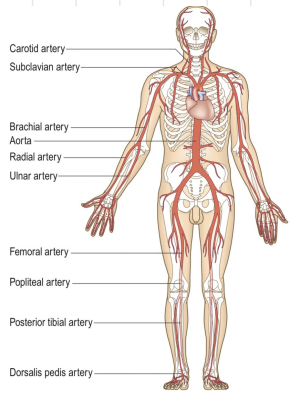
- Auscultation is most important.
- S_3 & S_4 & pericardial Rubs → dx.
- rheumatic heart disease :- multiple valve defect → stenosis (S_1) or regutitation (S_3).



★4.5 :- Investigations

- Haematology & clinical chemistry :- anemia, CBC, ESR, serology, urea, glucose, Lipid, troponin
- ECG
  - Ambulatory (holter - monitor)
  - Exercise.
- Ambulatory BP monitoring :- HTN.
- chest x-Ray :- cardiothoracic ratio <50%, pulmonary edema, Kerley B Lines, wided mediastinum.
- Echocardiography
- Redionuclide studies :- Technetium-99 IV, Thallium & sestambi.
- cardiac catheterisation
- computed tomography & magnetic resonance imaging.

PERIPHERAL ARTERIAL SYSTEM.



★4.1:- Anatomy & physiology (Fig 4.31)

★4.2:- The History

• 4.2.1:- common presenting symptoms

Signs of lower limb PAD

- ❑ Absence of hair.
- ❑ Thin skin.
- ❑ Brittle nails.

1 Leg pain

→ Asymptomatic ischemia

- PAD due to atherosclerosis in Large & medium vessels.
- Mostly Asymptomatic(extensive atherosclerosis)
- ankle-to-Bracial pressure index of <0.9 at rest.
- PAD on Legs x3 then on arms

4.22 Fontaine classification of lower limb ischaemia

Stage	Description
I	Asymptomatic
II (Ai:>200 Bi:<200)	Intermittent claudication
III	Night/rest pain
IV	Tissue loss (ulceration/gangrene)

→ intermittent claudication

- pain felt in Legs on walking due arterial insufficiency (most common symptom of PAD)
- tighness or cramp-like after constant distance
- The pain disappears completely in Rest & recurs on walking.
- claudication distance:- how far can walk before pain comes on.
- total walking distance:-how far can walk before pain → stop.
- The calf muscles is most affected:-Femoropopliteal disease.
- pain in thigh or botlock :- common femoral or aortoiliac obstruction.
- Leriche's syndrome:- male,bikteral common iliac or Internal iliac obstruction.
- occupation,absolute distance are important
- Questions
  - can you walk to the clinic from parking without stoping?
  - can you do your own shooping?
  - what are you unable to do because of pain?

4.23 The clinical features of arterial, neurogenic and venous claudication

	Arterial	Neurogenic	Venous
Pathology	Stenosis or occlusion of major lower limb arteries	Lumbar nerve root or cauda equina compression (spinal stenosis)	Obstruction to the venous outflow of the leg due to iliofemoral venous occlusion
Site of pain	Muscles, usually the calf but may involve thigh and buttocks	Ill-defined Whole leg May be associated with numbness and tingling	Whole leg 'Bursting' in nature
Laterality	Unilateral or bilateral	Often bilateral	Nearly always unilateral
Onset	Gradual after walking the 'claudication distance'	Often immediate on walking or standing up	Gradual, from the moment walking starts
Relieving features	On stopping walking, the pain disappears completely in 1–2 minutes	Bending forwards and stopping walking Patient may sit down for full relief	Leg elevation
Colour	Normal or pale	Normal	Cyanosed Often visible varicose veins
Temperature	Normal or cool	Normal	Normal or increased
Oedema	Absent	Absent	Always present
Pulses	Reduced or absent	Normal	Present but may be difficult to feel owing to oedema
Straight-leg raising	Normal	May be limited	Normal

→ Night pain

- wake up from pain in foot (instep)
- due to:- poor prefusion (↓gravity ,↓HR,↓Co,↓BP)
- Relive:- getting up & walking → edema → ↓tissue perfusion.

→ Rest pain

- occurs when:- Blood flow is insufficient to meet the demands of tissue.
- critical ischemia
  - Rest pain >2w.
  - tissue Loss+ ankle pressure <50mmHg or toe pressure <30mmHg.
- faliure to revascularisre → tissue Loss (gangrene, ulceration) & amputation.
- DDx:- diapetic neurophathy (not confined pain ,don't relived by dependency).

→ Tissue loss

- sever Lower Limb PAD,grandullay spreading proximally.
- progress rapidly ,without revarcularisation → amputation ± death.

→ Acute Limb ischemia <6h

4.24 Signs of acute limb ischaemia

- Pallor
- Pulselessness
- Perishing cold
- Paraesthesia
- Pain (worse when muscle squeezed)
- Paralysis

- paralysis:-anable to move toes/fingers.
- paraesthesia:- numbness or tingling over the forefoot or dorsum.
- Muscle tenderness:- impending muscle infraction.
- irreversible damged unless the circulation is restored.
- causes
  - Thromboembolism:- from Lt.A (Afib,MI),no history of claudication.
  - Thrombosis:- past history of claudication.

4.25 Acute limb ischaemia: embolus versus thrombosis in situ

	Embolus	Thrombosis
Onset and severity	Acute (seconds or minutes), ischaemia profound (no pre-existing collaterals)	Insidious (hours or days), ischaemia less severe (pre-existing collaterals)
Embolic source	Present	Absent
Previous claudication	Absent	Present
Pulses in contralateral leg	Present	Often absent, reflecting widespread peripheral arterial disease
Diagnosis	Clinical	Angiography
Treatment	Embolectomy and anticoagulation	Medical, bypass surgery, catheter-directed thrombolysis

→ Compartment syndrome

- ↑pressure within fasical compartments of the Limb that compromises the prefusion & viability of muscle & Nerves(Relaved by opioids ,↑:-by movement).
- most Common:- calfs
- causes:- Trauma , reperfusion injury following Tx of Lower Limb ischaemia.

2 Abdominal pain

- mesentric ischemia
  - Arteries:- coeliac trunk, superior & inferior mesentric A:- stenosed or occuled
  - site & timing:- central abdominal, 10-15 min after eating (weight)
  - Acute:- surgical emergency, embolus from heart, bloody diarrhoea.
- Abdominal aortic aneurysm (AAA)
  - 150% dilation, CT scan, 5% of men >65y (x3M>F)
  - Back pain, wave sign (pulsation), syncope (mostly asymptomatic)
  - Risk factors:- Smoking, HTN, genetics.

3 Digital ischaemia

- Blue toes
  - atheroembolism from AAA or alternative proximal embolic source
  - ↓emboli → ↑emboli → Acute Limb ischemia → Limb Loss.
- Vasospastic symptoms:- Raynaud's phenomenon
  - digital ischemia induced by cold & emotion.
  - phases
    - pallor:- digital A spasm or obstruction.
    - cynosis:- deoxygenation of static venous blood.
    - redness:- Reactive hyperaemia.
  - primary:- idiopathic.
  - secondary:- Drugs, CTD, hyperviscosity or use of power tools.
- CREST syndrome
  - Calcinosis
  - Raynaud's phenomenon
  - Esophageal dysfunction
  - Sclerodactyly.
  - Telangiectasias.

- 4.2.2:- past medical history
  - investigations, operations
  - Atherosclerosis, CAD, cerebrovascular ?
  - HTN, DM, ↑Lipid.

- 4.2.3:- Drug history
  - antiplatelet, Lipid-Lowering, anti: HTN, DM.
  - Vasoactive drugs & cardiac medications (↓BP, ⊖inotropic).

- 4.2.4:- Family history:- premature coronary or vascular disease

- 4.2.5:- Social history:- smoking, occupation, Lifestyle.

6.38 Diseases associated with secondary Raynaud's syndrome

- Connective tissue syndromes, e.g. systemic sclerosis, CREST (calcinosis, Raynaud's phenomenon, oesophageal dysfunction, sclerodactyly, telangiectasia) and systemic lupus erythematosus
- Atherosclerosis/embolism from proximal source, e.g. subclavian artery aneurysm
- Drug-related, e.g. nicotine, beta-blockers, ergot
- Thoracic outlet syndrome
- Malignancy
- Hyperviscosity syndromes, e.g. Waldenström's macroglobulinaemia, polycythaemia
- Vibration-induced disorders (power tools)
- Cold agglutinin disorders

★4.3:- physical examination (Box 4.26).

- anemia, cynosis, HF signs, PAD.
- Arms
  - Radial & brachial pulses.
  - BP in Both arms.
- Neck:- carotid pressure.
- Abdomen
  - inspect obvious pulsation
  - palpate Abdominal aorta.
  - thin → exansile
  - Below umbilicus → iliac aneurysm.
- Legs
  - ischaemia:- Temp, color, thin skin, brittle skin, abs of hair.
  - Scars (surgery), ulceration (position, margin, depth & color).
  - Look between toes for ulcers → ischemic (pressure sores).
- Femoral pulse → Lie down, femoral A (bruits, radiofemoral delay).
- popliteal pulse → flex Knee 30° → hard to feel (anyrsm).
- posterior tibial pulse → 2cm below & 2cm behind the medial malleolus.
- Dorsalis pedis pulse → felt → walk until pain
  - PAD → pulse will disappear.
  - APBI → will fall.

4.26 Signs suggesting vascular disease

Sign	Implication
Hands and arms	
Tobacco stains	Smoking
Purple discoloration of the fingertips	Atheroembolism from a proximal subclavian aneurysm
Pits and healed scars in the finger pulps	Secondary Raynaud's syndrome
Calcinosis and visible nail-fold capillary loops	Systemic sclerosis and CREST (calcinosis, Raynaud's phenomenon, oesophageal dysfunction, sclerodactyly, telangiectasia)
Wasting of the small muscles of the hand	Thoracic outlet syndrome
Face and neck	
Corneal arcus and xanthelasma	Hypercholesterolaemia
Horner's syndrome	Carotid artery dissection or aneurysm
Hoarseness of the voice and 'bovine' cough	Recurrent laryngeal nerve palsy from a thoracic aortic aneurysm
Prominent veins in the neck, shoulder and anterior chest	Axillary/subclavian vein occlusion
Abdomen	
Epigastric/umbilical pulsation	Aortoiliac aneurysm
Mottling of the abdomen	Ruptured abdominal aortic aneurysm or saddle embolism occluding aortic bifurcation
Evidence of weight loss	Visceral ischaemia

- Buergers test
  - Lying, Raise Leg 45° for 2-3 min.
  - watch for pallor with emptying & guttering of superficial veins.
  - sit up & hang their Legs over the edge.
  - watch for:- Reactive hyperaemia → Dilayed venous filling.
- Ankle/bracial pressure index
  - $\frac{\text{Ankle systolic BP}}{\text{Bracial systolic BP}}$
  - hold the probe over posterior tibial A at 45°.
  - inflate BP cuff round the ankle.
  - systolic pressure:- Doppler signal diappears → Repeat on dorsalis pedis A.
  - brachial BP in both arms.
  - normal:- ABPI > 1.0
  - intermeritent claudication:- <0.9
  - critical Limb ischemia:- <0.4
  - DM (ncompressibality & clacified vessels) :- falsly reassuring.

★4.4:- investigations (Box 4.27)

- unilateral:- Duplex ultrasound.
- bilateral:- CT or MR angiogram.

4.27 Investigations in peripheral arterial disease

Investigation	Indication/comment
Duplex ultrasound	Carotid artery stenosis, abdominal aortic aneurysm surveillance, peripheral arterial disease
Computed tomography	Abdominal aortic aneurysm, peripheral arterial disease, carotid artery stenosis
Magnetic resonance imaging	Peripheral arterial disease, carotid artery stenosis, arteriovenous malformations
Angiography	Acute and chronic limb ischaemia, carotid artery stenosis Invasive angiography has largely been replaced by computed tomography/magnetic resonance angiography as a diagnostic test

PERIPHERAL venous system.

★4.1:- Anatomy & physiology.

- 90% Deep, 10% superficial veins.
- passively from Head & neck. Legs → calf muscle pump & valves (Reflux), pressure of foot sole.
- Ambulatory venous pressure:- <20 mmHg.
- venous Reflux (valvler insufingy)
  - primary:- faliure of muscle pump, venous HTN, thrombotic.
  - secondary:- post DVT
- Deep venous obstruction + Reflux = post-thrombotic syndrome (pain, claudication, blue color, swelling, ulceration).
- Long (great) saphenous.
- short (Lesser) saphenous.
  - + perforator or communicating veins.
- The venous anatomy of Lower Limb is highly variable.

★4.2:- The History

•4.2.1: common presenting symptoms

- 4 ways
  - varicose vein.
  - DVT.
  - chronic venous insufficiency & ulceration.
  - superficial thrombophlebitis.
- The severity of symptoms & signs may bear little relationship to the severity of the underlying pathology & the physical signs.

4.28 Clinical features of venous and arterial ulceration

Clinical feature	Venous ulceration	Arterial ulceration	Neuropathic ulceration
Sex	More common in women	More common in men	Equal in men and women
Risk factors	Thrombophilia, family history, previous deep vein thrombosis, varicose veins	Known peripheral vascular disease or risk factors for atherosclerotic disease, e.g. smoking, diabetes, dyslipidaemia, hypertension	Diabetes or other peripheral neuropathy (loss of sensation, loss of intrinsic foot muscle function, autonomic dysregulation)
Pain	Often painless but some patients have some pain that improves with elevating the leg	Severe pain, except in diabetics with neuropathy; improves on dependency	Painless or neuropathic pain
Site	Gaiter areas: 80% medial (long saphenous vein), 20% lateral (short saphenous vein)	Pressure areas (malleoli, heel, fifth metatarsal base, metatarsal heads and toes)	Pressure areas, sole of foot, tips of toes
Appearance	Shallow, irregular margin Slough on granulating base	Regular, 'punched out' Sloughy or necrotic base	Macerated, moist white skin surrounded by callus, often on load-bearing aspects (motor neuropathy)
Surrounding skin	Lipodermatosclerosis always present Oedema	Shiny, hairless, trophic changes	Dry due to reduced sweating (autonomic neuropathy)
Veins	Full and usually varicose	Empty with 'guttering' on elevation	Normal
Temperature	Warm Palpable pulses	Cold Absent pulses	Warm or cold due to autonomic neuropathy Palpable pulses

1 pain

- uncomplicated varicose (dilated, tortuous, superficial)
  - aching Leg discomfort, itching & swelling
  - ↑ in:- prolonged standing.
- DVT:- pain + tenderness in calfs.
- superficial thrombophlebitis:- Red + painfull, palpable as tender cord.
- varicose ulceration:- painless or pain.

2 Limb swelling

- upper limb DVT:- swollen + cynosed & mottled
  - ↑ by:- activity, arm overhead.
  - may complicate indwelling subclavian/Jugular cath.

3 Skin changes

- chronic venous insufficiency:- bluish, distally.
- varicose eczema:- Red, itchy, dry.
- venous HTN:- Brown (hemosidrin deposition), Lower ⅓.
- Lipodermatosclerosis:- Red/purple, inflammatory to haemosiderin.
- Atropine blanche:- white, multiple, small scarred area.
- inverted champagne bottle:- thicked, fibrotic skin → tight band.
- chronic venous ulceration
  - causes → 70-80% Lower limb ulceration
    - others:- pyoderma, gangrenosum, syphills, TB, Leprosy, sickle, tropically.
  - Area:- medial aspect of the calf.
  - ulcers → shallow + pink (granulation tissue).
    - yellow/green (slough)
    - irregular margin.
  - Associated with:- varicose eczema, Lipodermatosclerosis.

4 Superficial venous thrombophlebitis

- 1o%, ↑ in pregrency
- associated with:- underlying malignancy.
- propagete:- → DVT → PE.

- 4.2.2 :- past History :- venous vein surgery & DVT Risk factors.

★4.3 :- The physical examination.

- standing → lying.
- color, swelling & dilation or tortuosity.
- Temp, pitting edema, cheak JVP → ↑ → cardiac or P. HTN.

★4.4 :- investigation

- tourniquet & Trendelenburg :- saphenofemoral incompetence, Replaced.
- hand-held Doppler

4.29 Risk factors for deep vein thrombosis

- Obesity
- Smoking
- Recent bed rest or operations (especially to the leg, pelvis or abdomen)
- Recent travel, especially long flights
- Previous trauma to the leg, especially long-bone fractures, plaster of Paris splintage and immobilisation
- Pregnancy or features suggesting pelvic disease
- Malignant disease
- Previous deep vein thrombosis
- Family history of thrombosis
- Inherited thrombophilia, e.g. factor V Leiden
- Recent central venous catheterisation, injection of drug
- Use of oral contraceptive or hormone replacement therapy

6.42 Features of deep vein thrombosis of the lower limb

Clinical feature	Non-occlusive thrombus	Occlusive thrombus
Pain	Often absent	Usually present
Calf tenderness	Often absent	Usually present
Swelling	Absent	Present
Temperature	Normal or slightly increased	Increased
Superficial veins	Normal	Distended
Pulmonary embolism	High risk	Low risk

6.41 Clinical features of venous and arterial ulceration

Clinical feature	Venous ulceration	Arterial ulceration
Age	Develops at age 40–45 but may not present for years; multiple recurrences common	First presents in over-60s
Sex	More common in women	More common in men
Past medical history	Deep vein thrombosis (DVT) or suggestive of occult DVT, i.e. leg swelling after childbirth, hip/knee replacement or long bone fracture	Peripheral arterial disease, cardio- and cerebrovascular disease
Risk factors	Thrombophilia, family history, previous DVT	Smoking, diabetes, hypercholesterolaemia and hypertension
Pain	One-third have pain (not usually severe) that improves with elevating the leg	Severe pain, except in diabetics with neuropathy; improves on dependency
Site	Gaiter areas; usually medial to long saphenous vein; 20% are lateral to short saphenous vein	Pressure areas (malleoli, heel, fifth metatarsal base, metatarsal heads and toes)
Margin	Irregular, often with neoepithelium (appears whiter than mature skin)	Regular, indolent, 'punched out'
Base	Often pink and granulating under green slough	Sloughy (green) or necrotic (black), with no granulation
Surrounding skin	Lipodermatosclerosis always present	No venous skin changes
Veins	Full and usually varicose	Empty with 'guttering' on elevation
Swelling (oedema)	Usually present	Absent
Temperature	Warm	Cold
Pulses	Present, but may be difficult to feel	Absent

cardiac cycle

- Atrial systole :- 0.1 sec (not important)
- Atial diastole :- 0.7 sec
- ventricular systole :- 0.3 sec :- isovolumic contration , Rapid & slow ejection
- ventricular diastole :- 0.5 sec :- isovolumic Relaxation , Rapid filling , slow filling (diastasis), Atria contraction.
- stroke volume :- EDV - ESV
  - preload :- passive tension (EDV) . Frank-starling Law.
  - contractility :- inotropic effect , ↑EF → ↑SV
  - afterLoad :- pressure of Aorta during diastole (to open SL valve)
- phases
  - ventricular filling :- 70-80% passively , AV open , SL close , P wave , AP > VP < Aortic pressure
  - Isovolumtric contraction :- Both AV & SL closed , AP < VP < Aortic pressure , S_1 (Lub) , QRS comp
  - ventricular systole / ejection :- AV closed , SL open , VP > Aortic pressure > AP , QRS complex.
  - Isovolumtric relaxation :- Backflow of aorta & pulmonery closes SL , AV closed , Aortic > VP > AP
    t wave , S_2 (Dub) , Dicrotic notch (incisura) :- Breaf raise of Aortic pressure caused by blood backflow.
- cardiac output :- stroke volume (SV) x Heart Rate (HR).
- Ejection fraction :- SV / EDV , normally 60%
- Artiral pressure
  - A wave :- atrial systole (↑mitral stenosis , P wave).
  - C wave :- ventricular systole (AV closure , QRS).
  - V wave :- ventricular diastole (AV opening , T wave).
- Heart sounds
  - S_1 (Lub) :- closing AV valves (QRS)
  - S_2 (Dub) :- closing SL valves (T)
  - S_3 :- Rapid filling
  - S_4 :- Atrial systole

Lub —v.systole→ Dub —v.diastole→ Lub

systolic murmur

diastolic murmur

- cardiac reserve :- Max CO - Resting CO (SL)
- Left ventricle pressure / volume Loop
  - phase I :- blood filling in ventricle.
  - phase II :- Isovolumic contraction.
  - phase III :- period of ejection.
  - phase IV :- Isovolumic Relaxation.

- A :- AV opens (ESV)
- B :- AV close (S_1 , EDV)
- C :- SL open
- D :- SL close (S_2)
- potential energy :- we can use it to ↑SV