CVS:

1. **Intermittent chest pain:**

Angina:

- **Stable**: (caused by chronic narrowing in one or more coronary arteries), episodes of pain are precipitated by exertion and may occur more readily when walking in cold or windy weather, after a large meal or while carrying a heavy load; the pain is promptly relieved by rest and/or sublingual glyceryl nitrate (GTN) spray, and typically lasts for less than 10 minutes.
- **unstable** angina (caused by a sudden severe narrowing in a coronary artery), there is usually an abrupt onset or worsening of chest pain episodes that may occur on minimal exertion or at rest.

- Retrosternal/ Progressive onset/ increase in intensity over 1–2 minutes/ Constricting, heavy/ Sometimes arm(s), neck, epigastrium/ Associated with breathlessness/ Intermittent, with episodes lasting 2–10 minutes/ Triggered by emotion, exertion, especially if cold, windy/ Relieved by rest, nitrates Mild to moderate.
- Aggravated by thyroxine or drug-induced anemia, e.g. aspirin or NSAIDs

Esophageal:

- Retrosternal or epigastric/ Over 1–2 minutes; can be sudden (spasm)/ C: Gripping, tight or burning/ R: Often to back, sometimes to arms/ A: Heartburn, acid reflux/ T: Intermittent, often at night-time; variable duration/ Lying flat/some foods may trigger/ Not relieved by rest; nitrates sometimes relieve/ Usually mild but esophageal spasm can mimic myocardial infarction.

2. **Acute chest pain:**

MI:

- SOCRATES: Retrosternal/ Rapid over a few minutes/ Constricting, heavy/ Often to arm(s), neck, jaw, sometimes epigastrium/ Sweating, nausea, vomiting, breathlessness, feeling of impending death (angor animi)/ Acute presentation; prolonged duration/ 'Stress' and exercise rare triggers, usually spontaneous/ Not relieved by rest or nitrates/ Usually severe.
- Fever may occur after MI (and with infections = infective endocarditis and epicarditis).
- May cause abdominal pain (non-alimentary cause): epigastric pain without tenderness.

- MI with pulmonary edema: acute sudden breathlessness with crushing central chest pain and crackles in the chest (if normal breathing sound = large PE)
- History of MI increases the risk of arrhythmias and IHD
- The presence of xanthelasma is an independent risk factor for coronary heart disease and myocardial infarction.
- Apex beat may be diffusely displaced inferiorly and laterally in left ventricular dilatation (such as after myocardial infarction or in dilated cardiomyopathy), severe aortic regurgitation or decompensated aortic stenosis.
- Pericardial rub is most often heard in acute pericarditis or a few days after an extensive myocardial infarction.
- The differential diagnosis of a murmur heard after myocardial infarction includes acute mitral regurgitation due to papillary muscle rupture, functional mitral regurgitation caused by left ventricular dilatation, and a pericardial rub.

Pericardial chest pain:

- SOCRATES: Retrosternal or left-sided/ Gradual; postural change may/ suddenly aggravate/ Sharp, 'stabbing', pleuritic/ Left shoulder, or back/ Flu-like prodrome, breathlessness, fever/ Acute presentation; variable duration/ NSAIDs help, exacerbated by inspiration or lying down, and relieved by sitting forwards/

Everything about "**carditis**":

- Infective endocarditis:
  ** Microbial infection of a heart valve, frequently presents with non-specific symptoms, including weight loss, tiredness, fever, and night sweats.
  ** May cause a vegetation on the mitral valve. (Causes valvular diseases)
  ** Peripheral signs: Janeway lesions on the hypothenar eminence, Splinter hemorrhages, Osler's nodes, Roth's spot on fundoscopy, Petechial hemorrhages on the conjunctiva, and Clubbing,
  ** Can cause rapidly changing murmurs
  ** May affect the kidney (hematuria)
  ** May cause splenomegaly

- Constrictive pericarditis: may cause edema + may cause HF (impaired ventricular filling). And it's an uncommon cause of ascites (hence: edema)
- Pericarditis: most common cause of pericardial rub

Aortic dissection:

- SOCRATES: Interscapular/retrosternal Very sudden Tearing or ripping Back, between shoulders/ Sweating, syncope, focal neurological signs, signs of limb ischemia, mesenteric ischemia/ Acute presentation; prolonged duration/ Spontaneous/ No maneuvers relieve pain/ very severe. predisposing: Marfan's

- If the tear involves the cranial or upper limb arteries, there may be associated syncope, stroke, or upper limb pulse asymmetry.
- Horner's syndrome may be caused by Carotid artery dissection

3. **HF:** A bad consequence of many diseases (end-organ damage) (Decreased CO)

- The most common CV cause of both acute and chronic dyspnea.
- Baseline symptoms: Dyspnea, fatigue, ankle swelling Record usual functional status.
- Major events: Hospitalization for decompensated heart failure and Ventricular arrhythmias.
- Patients with acute heart failure and pulmonary oedema (accumulation of fluid in the alveoli) usually prefer to be upright, while patients with massive pulmonary embolism are often more comfortable lying flat (if upright >> syncope).
- Exertional dyspnea is the symptomatic hallmark of chronic heart failure.
- Dyspnea caused by myocardial ischemia is known as 'angina equivalent'. It has identical precipitants to angina and may be relieved by GTN.

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)

- Orthopnea = advanced disease + the same mechanism as PND.
- PND = anytime during sleep / Asthma = at dawn/ HF = produce frothy white or blood-stained sputum = to differentiate from asthma.

- Bilateral lower limb edema (unilateral = DVT).
- Advanced heart failure may result in either abdominal distension due to ascites, or weight loss and muscle wasting ('cardiac cachexia') due to a prolonged catabolic state.
- JVP: Elevation, sustained abdominojugular reflux > 10 seconds
- Pulsus alternans, beat-to-beat variation in pulse volume with a normal rhythm, may occur in advanced heart failure.
- In adults, coarctation (congenital aortic narrowing that causes radio-femoral delay) usually presents with hypertension and heart failure.
- Low pulse volume may result from severe heart failure and conditions associated with inadequate ventricular filling such as hypovolemia, cardiac tamponade, and mitral stenosis.
- Apex beat may be diffusely displaced inferiorly and laterally in left ventricular dilatation, such as after myocardial infarction or in dilated cardiomyopathy. + in aortic stenosis and regurgitation.
- If sufficient (with pulmonary edema and congestion) >>> central cyanosis.
- In heart failure, S3 occurs with a tachycardia, referred to as a 'gallop' rhythm, and S1 and S2 are quiet.

4. Palpitations:

Ectopic beats (extrasystoles):

- A benign cause of palpitation at rest and are abolished by exercise. They come as premature ectopic beats followed by a forceful contraction with the next beat.
- They have normal rhythm but irregular pulse. Accordingly, patients often describe 'missed beat' followed by 'jump beat'.
- Table: Sudden/ associated with nil/ Fatigue, caffeine, alcohol may trigger/ Often relieved by walking (increases sinus rate)/ mild.
- Regularly irregular = intermittent extrasystoles or second-degree atrioventricular block.

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 with variable response

- Variable abnormalities of the first heart sound: Extrasystoles, complete heart block, and A.fib.

Supraventricular tachycardia:

- sudden paroxysms of rapid, regular palpitation that can sometimes be terminated with vagal stimulation using Valsalva breathing maneuvers or carotid sinus pressure. It often affects young patients with no other underlying cardiac disease. Associated with Polyuria, lightheadedness, chest tightness. Minutes to hours/ Moderate to severe.
- JVP: Regular cannon waves (giant a waves) occur during junctional rhythm and with some ventricular and supraventricular tachycardias.

Ventricular tachycardia:

- more commonly associated with presyncope or syncope and tends to affect patients with cardiomyopathy or previous myocardial infarction.
- Severe/ triggered by exercise.

Atrial fibrillation:

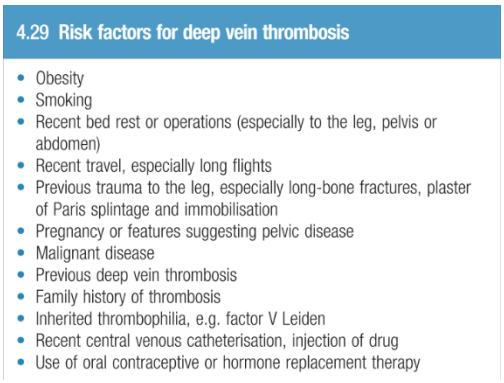
- Sudden, Irregular, usually fast; slower in elderly, Polyuria, breathlessness, Syncope uncommon, variable duration, exercise, or alcohol may trigger; often spontaneous, very variable severity, may be asymptomatic.
- Alcohol can cause atrial fibrillation.
- Irregularly irregular pulse. The rate in atrial fibrillation depends on the number of beats conducted by the atrioventricular node. Untreated, the ventricular rate may be very fast (up to 200 bpm).
- JVP: absent a wave.
- Variable intensity of the first heart sound. The forth sound cannot be heard.
- Thromboembolism that causes acute limb ischemia is usually from left atrium in association with heart failure or atrial fibrillation.
- Hyperthyroidism may be associated with atrial fibrillation.

Sinus tachycardia:

- The most common cause of tachycardia.
- Gradual onset/ regular fast character (pounding)/ persists for few minutes/ associated with anxiety/ exacerbated by exercise/ moderate to severe.

5. **Hypertrophic cardiomyopathy:**
    - May cause syncope and angina
    - It increases the likelihood of life-threatening arrhythmia.
    - May lead to HF by impairing the ventricular filling.
    - A double apical impulse is characteristic of hypertrophic cardiomyopathy.
    - Reverse splitting of S2: Aortic stenosis/ Hypertrophic cardiomyopathy/ Left bundle branch block/ Ventricular pacing.
    - An S4 is most often heard with left ventricular hypertrophy (due to hypertension, aortic stenosis, or hypertrophic cardiomyopathy).
    - Ejection systolic murmur.
6. **Dilated cardiomyopathy:**
    - May lead to HF by decreasing ventricular contractility.
7. **Restrictive cardiomyopathy:**
    - Kussmaul's sign (a paradoxical rise of JVP on inspiration) is seen (+ in pericardial constriction, severe right ventricular failure).
    - May lead to HF by impairing ventricular filling.
    - Apex beat is diffusely displaced inferiorly and laterally.

8. **DVT:**
    - unilateral lower limb edema.
    - Elevated JVP suggests cardiogenic cause of edema (>>> not DVT)
    - Once established, DVT causes pain and tenderness in the affected part.
    - Life-threatening DVT may be asymptomatic, while apparently trivial varicose veins may be associated with significant symptoms.



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

9. **Premature CA disease:**
    - Family history (first degree relatives): males < 65 / females < 55
    - The patient him/herself = males < 55 / females < 45
    - Hypercholesterolemia is associated with premature coronary artery disease.

10. Heart block:

- First-degree: quiet S1
- Complete heart block: variable intensity of S1 // JVP: irregular cannon waves
- Regularly irregular pulse: 2nd degree AV block or extrasystoles.

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11. Aortic regurgitation:

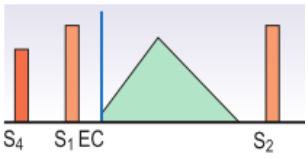
- A pathological cause of increased pulse volume.
- Collapsing pulse (exaggerated by raising the patient's arm above the level of the heart) >>> wide pulse pressure.
- Pulsus bisferiens, an increased pulse with a double systolic peak separated by a distinct mid-systolic dip, is classically produced by concomitant aortic stenosis and regurgitation.
- Apex beat is diffusely displaced inferiorly and laterally >> severe aortic regurgitation or decompensated aortic stenosis.
- The aortic component of S2 is sometimes quiet or absent in calcific aortic stenosis and reduced in aortic regurgitation
- High-pitched murmurs often correspond to high-pressure gradients, so the diastolic murmur of aortic regurgitation is higher-pitched than that of mitral stenosis.
- Location helps to differentiate diastolic murmurs (mitral stenosis at the apex, aortic regurgitation at the left sternal edge) but is less helpful with systolic murmurs, which are often audible across the precordium.
- Early diastolic murmurs usually last throughout diastole but are loudest in early diastole, so the term 'early diastolic murmur' is misleading. The murmur is typically caused by aortic regurgitation and is best heard at the left sternal edge with the patient leaning forwards in held expiration.
- The duration of the aortic regurgitation murmur is inversely proportional to severity.
- An associated systolic murmur is common because of the increased flow through the aortic valve in systole.

- An Austin Flint murmur is a mid-diastolic murmur that accompanies aortic regurgitation. It is caused by the regurgitant jet striking the anterior leaflet of the mitral valve, restricting inflow to the left ventricle.

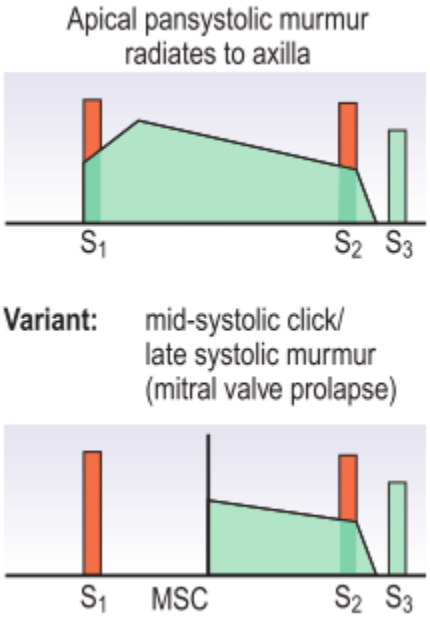
12. Aortic stenosis:

- Prolonged capillary refill time may occur in shock, or chronic conditions associated with a low cardiac output state (as in severe aortic stenosis, mitral stenosis, or pulmonary hypertension).
- May cause angina and syncope or presyncope.
- A slow-rising pulse has a gradual upstroke with a reduced peak occurring late in systole and is a feature of severe aortic stenosis >>> narrow pulse pressure.
- Radiation of ejection systolic murmur of aortic stenosis >>> carotid arteries.
- Pulsus bisferiens (when concomitant with aortic regurgitation)
- Decompensated aortic stenosis >>> apex beat diffusely displaced inferiorly and laterally.
- Left ventricular hypertrophy, as in hypertension or severe aortic stenosis, produces a forceful but undisplaced apical impulse. This thrusting apical 'heave' is quite different from the diffuse impulse of left ventricular dilatation.
- The murmur of aortic stenosis is often audible all over the precordium. It is harsh, high-pitched, and musical.
- The most common thrill is that of aortic stenosis, which is usually palpable over the upper right sternal border.
- The aortic component of S2 is sometimes quiet or absent in calcific aortic stenosis and reduced in aortic regurgitation
- Reversed splitting that's increased by expiration.
- An S4 is most often heard with left ventricular hypertrophy (due to hypertension, aortic stenosis, or hypertrophic cardiomyopathy). It cannot occur when there is atrial fibrillation.
- Ejection clicks are high-pitched sounds best heard with the diaphragm. They occur early in systole just after the S1, in patients with congenital pulmonary or aortic stenosis.
- Ejection clicks do not occur in calcific aortic stenosis because the cusps are rigid.

Lean patient forward with breath held in expiration to feel thrill and hear murmur best



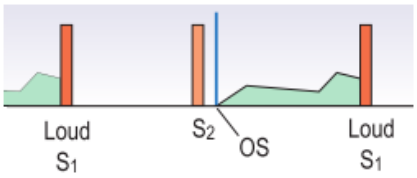
13. Mitral regurgitation:



- Apex >>>> Axilla
- Radiation of the pansystolic murmur of mitral regurgitation >>> left axilla.
- The most common causes of S3 are left ventricular failure, when it is an early sign, and mitral regurgitation, due to volume loading of the ventricle.
- Quiet S1 in rheumatic mitral regurgitation.
- Mitral regurgitation is caused by dilatation of the left ventricle and failure of leaflets to co-apt.
- The murmur begins at the moment of valve closure and may obscure the first heart sound. It varies little in intensity throughout systole.
- In mitral valve prolapse the murmur begins in mid- or late systole and there is often a mid-systolic click (MSC).
- The differential diagnosis of a murmur heard after myocardial infarction includes acute mitral regurgitation due to papillary muscle rupture, functional mitral regurgitation caused by left ventricular dilatation, and a pericardial rub.

14. Mitral stenosis:

- Low pulse volume may result from severe heart failure and conditions associated with inadequate ventricular filling such as hypovolemia, cardiac tamponade, and mitral stenosis.



- Mid diastolic murmur, low pitched, may follow an opening snap, accentuated by exercise.
- The 'tapping' apex beat in mitral stenosis represents a palpable first heart sound and is not usually displaced.
- In mitral stenosis the intensity of S1 is increased due to elevated left atrial pressure >>> loud S1.
- The murmur is heard best with the bell at the apex and the patient rolled on to their left-hand side.

15. Tricuspid regurgitation:

- Lower left sternal border.
- JVP: Giant v or cv wave + there may be an associated pulsatile liver.

Pansystolic murmurs

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

Late systolic murmurs

- Mitral valve prolapse

16. Tricuspid stenosis:

- Large, prominent "a" wave (not giant) caused by delayed right ventricular filling.

17. Pulmonary stenosis:

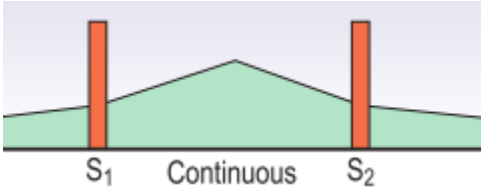
- Ejection systolic murmur.
- Persistent S2 splitting (enhanced by inspiration >>> physiological)

18. Pulmonary regurgitation:

- very uncommon.
- It may be caused by pulmonary artery dilatation in pulmonary hypertension (Graham Steell murmur) or a congenital defect of the pulmonary valve.

19. PDA:

- Below left clavicle (at the upper left sternal border) and radiates over the left scapula: Continuous 'machinery' murmur of a persistent PDA.



- A continuous 'machinery' murmur is heard because aortic pressure always exceeds pulmonary arterial pressure, resulting in continuous ductal flow. The pressure difference is greatest in systole, producing a louder systolic component to the murmur.

20. ASD:

- Ejection systolic murmur (not pansystolic) (pulmonary flow murmur) with fixed splitting of S2.

Ejection systolic murmur (pulmonary flow murmur) with fixed splitting of second sound

21. VSD:

- The murmur of ventricular septal defect is pansystolic, heard at the lower left sternal border, radiates towards the right sternal edge, and often associated with a thrill.
- Rupture of the interventricular septum can complicate myocardial infarction, producing a harsh pansystolic murmur.
- The thrill caused by a ventricular septal defect is best felt at the left and right sternal edges.

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

22. Cardiac tamponade:

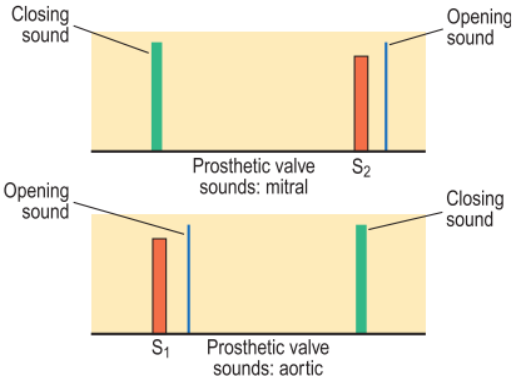
- Pulsus paradoxus is an exaggeration of the normal variability of pulse volume with breathing. Pulse volume normally increases in expiration and decreases during inspiration due to intrathoracic pressure changes affecting venous return to the heart. This variability is exaggerated when ventricular diastolic filling is impeded by elevated intrapericardial pressure. This is usually due to accumulation of pericardial fluid (cardiac tamponade) but can occur to a lesser extent with pericardial constriction and in acute severe asthma. If suspected, pulsus paradoxus can be confirmed using a blood pressure cuff; a fall of > 10 mmHg between the cuff pressure at which Korotkoff sounds appear in expiration only and the cuff pressure at which Korotkoff sounds persist throughout the respiratory cycle is diagnostic.
- JVP is increased in any event that leads to high ventricular filling pressure including cardiac tamponade.

Extra:

- **Familial hypercholesterolemia**: associated with tendon xanthoma// **Hyperlipidemia**: xanthelasmata and corneal arcus// The presence of xanthelasma is an independent risk factor for coronary heart disease and myocardial infarction.

- The most common causes of bradycardia are medication, athletic conditioning and sinoatrial or atrioventricular node dysfunction.
- Mechanical obstruction of the superior vena cava (most often caused by lung cancer) may cause extreme, non-pulsatile elevation of the JVP >>> the abdominojugular test will be negative.
- Coarctation is a congenital narrowing of the aorta, usually distal to the left subclavian artery; it may produce reduced-volume lower limb pulses, which are also delayed relative to the upper limb pulses (radiofemoral delay). In adults, coarctation usually presents with hypertension and heart failure.
- Different BP in each arm: a difference of > 10 mmHg on repeated measurements suggests the presence of aortic or subclavian artery disease.
- Pectus excavatum (funnel chest), a posterior displacement of the lower sternum, and pectus carinatum (pigeon chest) may displace the heart and affect palpation and auscultation.
- The apex beat may be impalpable in overweight or muscular people, or in patients with asthma or emphysema because the lungs are hyperinflated.
- Normally, S2 is louder and higher pitched than the S1, and the aortic component is normally louder than the pulmonary component. Physiological splitting of S2 occurs because left ventricular contraction slightly precedes that of the right ventricle so that the aortic valve closes before the pulmonary valve. This splitting increases at end-inspiration because increased venous filling of the right ventricle further delays pulmonary valve closure. The separation disappears on expiration.
- The aortic component of S2 is loud in systemic hypertension, and the pulmonary component is increased in pulmonary hypertension.
- Wide splitting of S2, but with normal respiratory variation, occurs in conditions that delay right ventricular emptying, such as right bundle branch block or pulmonary hypertension.
- S3 is a normal physiological finding in children, young adults, and febrile patients, and during pregnancy, but is usually pathological after the age of 40 years.
- A pleuropericardial rub occurs in time with the cardiac cycle (like pericardial rub) but is also influenced by respiration and is pleural in origin. Occasionally, a ‘crunching’ noise can be heard, caused by gas in the pericardium (pneumopericardium).

- Mechanical heart valves can make a sound when they close or open. The closure sound is normally louder, especially with modern valves. The sounds are high-pitched, 'metallic' and often palpable, and may even be heard without a stethoscope. A mechanical mitral valve replacement makes a metallic S1 and a sound like a loud opening snap early in diastole. Mechanical aortic valves have a loud, metallic S2 and an opening sound like an ejection click at the start of systole. They are normally associated with a flow murmur.



- 'Innocent' murmurs are caused by increased velocity of flow through a normal valve and occur when stroke volume is increased, as in pregnant women, athletes with resting bradycardia or patients with fever.

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

4.14 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)

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~~ flattened 'y' descent
Pericardial constriction	Elevation, Kussmaul's sign, prominent 'y' descent 'x' 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

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