

The Cardiovascular System

Introductory course , HU

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- The heart comprises **two muscular pumps** working in series,

covered in a serous sac (pericardium) that allows free movement

with each heart beat and respiration.

-**The right heart**(right atrium and ventricle) pumps deoxygenated blood returning from the systemic veins into the pulmonary circulation at relatively low pressures.

-**The left heart** (left atrium and ventricle) receives blood from the lungs and pumps it round the body to the tissues at higher pressures.

- **Atrioventricular valves** (tricuspid on the right side, mitral on the left) separate the atria from the ventricles.

- **The pulmonary valve** on the right side of the heart and the **aortic valve** on the left separate the ventricles from the pulmonary and systemic arterial systems, respectively.

- Cardiac contraction is coordinated by specialised groups of cells. The cells in the **sinoatrial node** normally act as the cardiac pacemaker.
- Subsequent spread of impulses through the heart ensures that atrial contraction is complete before ventricular contraction (**systole**) begins.
- At the end of systole the ventricles relax and the atrioventricular valves open, allowing them to refill with blood from the atria (**diastole**).

How to approach to a patient with a specific complain ?

- History (inquiry about symptoms)
- Physical examination(looking for signs)
- Investigations
- Finally you reach a specific diagnosis

HISTORY

- Cardiovascular diseases may present with a number of diverse symptoms.
- non-cardiac causes must also be considered(**differential diagnosis** for a certain complain)

- **Chest pain**

- Typical angina pain
- Differential diagnosis : cardiac causes vs non-cardiac causes
- Ask about ? (SOCRATES !)

4.3 Cardiovascular causes of chest pain and their characteristics

	Angina	Myocardial infarction	Aortic dissection	Pericardial pain	Ösophageal 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	Stinging, 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 arm(s)
Associated features	Breathlessness	Sweating, nausea, vomiting, breathlessness, feeling of impending death (<i>angor animi</i>)	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, nitrate	'Silent' and exercise rare triggers, usually spontaneous. Not relieved by rest or nitrate	Spontaneous. No manoeuvre relieve pain	Sitting up/lying down may affect intensity. NSAIDs help	Lying flat/some foods may trigger. Not relieved by rest, nitrate 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)	Ösophageal spasm, reflux, hiatal hernia

NSAIDs, see analgesic and inflammatory drugs

- **Dyspnoea (breathlessness)**

- Cardiac (HF , PE, arrhythmias , angina equivalent)

- Vs non-cardiac causes

- **Exertional dyspnoea** is the symptomatic hallmark of chronic





heart failure.

The New York Heart Association grading system is

used to assess the degree of symptomatic limitation caused by

the exertional breathlessness of heart failure

- Orthopnea
- PND (differentiate from respiratory causes ?)

NYHA Class	Level of Clinical Impairment	
I		No limitation of physical activity. Ordinary physical activity does not cause undue breathlessness, fatigue, or palpitations.
II		Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in undue breathlessness, fatigue, or palpitations.
III		Marked limitation of physical activity. Comfortable at rest, but less than ordinary physical activity results in undue breathlessness, fatigue, or palpitations.
IV		Unable to carry on any physical activity without discomfort. Symptoms at rest can be present. If any physical activity is undertaken, discomfort is increased.

- In acute dyspnoea, ask about:

- duration of onset
- background symptoms of exertional dyspnoea and usual exercise tolerance
- associated symptoms: chest pain, syncope, palpitation or respiratory symptoms (such as cough, sputum, wheeze or haemoptysis).

- In patients with chronic symptoms, ask about:

- relationship between symptoms and exertion
- degree of limitation caused by symptoms and their impact on everyday activities
- effect of posture on symptoms and/or episodes of nocturnal breathlessness
- associated symptoms: ankle swelling, cough, wheeze or sputum.

• **Palpitation**

-Palpitation is an unexpected or unpleasant awareness of the heart beating in the chest.

-Detailed history taking can help to distinguish the different types of palpitation ([Box 4.6](#)).

- Ask about:

- nature of the palpitation: is the heart beat rapid, forceful or irregular? Can the patient tap it out?
- timing of symptoms: speed of onset and offset; frequency and duration of episodes
- precipitants for symptoms or relieving factors
- associated symptoms: presyncope, syncope or chest pain
- history of underlying cardiac disease

- **Syncope and presyncope**

- Syncope is a transient loss of consciousness due to transient cerebral hypoperfusion.
- Causes include postural hypotension, neurocardiogenic syncope, arrhythmias and mechanical obstruction to cardiac output, PE , cardiac tumors, valvular diseases
- Non-cardiovascular causes (seizures , cva ...)

- In patients who present with syncope, ask about:
 - circumstances of the event and any preceding symptoms: palpitation, chest pain, lightheadedness, nausea, tinnitus,
 - sweating or visual disturbance
 - duration of loss of consciousness, appearance of the patient while unconscious and any injuries sustained (a detailed witness history is extremely helpful)
 - time to recovery of full consciousness and normal cognition
 - frequency of episodes and impact on lifestyle
 - possible contributing medications, such as antihypertensive agents (Box 4.7).
 - current driving status, including occupational driving.

- **Oedema**

- unilateral vs bilateral
- gravity dependant (ankles , sacrum)
- Heart failure , chronic venous disease, vasodilating calcium channel antagonists (such as amlodipine) and hypoalbuminaemia.
- An elevated jugular venous pressure strongly suggests a cardiogenic cause of oedema.
- Enquire about other symptoms of fluid overload, including dyspnoea, orthopnoea and abdominal distension.

- **Past medical history**

- Obtaining a detailed record of any previous cardiac disease, investigations and interventions is essential .
- conditions associated with increased risk of vascular disease such as hypertension, diabetes mellitus and hyperlipidaemia
- rheumatic fever or heart murmurs during childhood
- potential causes of bacteraemia in patients with suspected infective endocarditis, such as skin infection, recent dental work, intravenous drug use or penetrating trauma
- systemic disorders with cardiovascular manifestations such as connective tissue diseases (pericarditis and Raynaud's phenomenon), Marfan's syndrome (aortic dissection) and
- myotonic dystrophy (atrioventricular block).

- **Drug history**

- Drugs may cause or aggravate symptoms such as breathlessness, chest pain, oedema, palpitation or syncope (see [Box 4.7](#)).
- Ask about ‘over-the-counter’ purchases, such as non-steroidal anti-inflammatory drugs (NSAIDs) and alternative and herbal medicines, as these may have cardiovascular actions.

- **Family history**

- Many cardiac disorders such as cardiomyopathies have a genetic component.
- Ask about **premature coronary artery disease** in first-degree relatives (< 60 years in a female or < 55 years in a male);
- sudden unexplained death at a young age may raise the possibility of a cardiomyopathy or inherited arrhythmia.
- Patients with venous thrombosis may have inherited thrombophilia, such as a factor V Leiden mutation.
- Familial hypercholesterolaemia is associated with premature arterial disease

- **Social history**

- Smoking is the strongest risk factor for coronary and peripheral arterial disease. Take a detailed smoking history.
- Alcohol can induce atrial fibrillation and, in excess, is associated with obesity, hypertension and dilated cardiomyopathy.
- Recreational drugs such as cocaine and amphetamines can cause arrhythmias, chest pain, occlusive and aneurysmal peripheral arterial disease and even myocardial infarction .
- Impact on daily activity and employment .