

Acute Coronary Syndromes

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Objectives

- Definitions of the acute coronary syndromes
- The coronary arteries
- The disease process which gives rise to acute coronary syndromes
- How to differentiate between the acute coronary syndromes.
- Treatment and Intervention

The Acute Coronary Syndromes

- Unstable angina
- Non-Q wave myocardial infarction
- Q wave myocardial infarction

Prevalence of Coronary artery disease

- CAD is the biggest killer in the UK
- More than 1.4million suffer from angina
- 300,000 have have attacks every year
- 110,000 die of heart problems in England every year

(Figures taken from the national surface framework standards 2000)

Definition

Coronary artery disease is a process that leads to a build up of fatty deposits in the arteries. This can develop into partial or total blockage of arteries that supply blood to the heart.

Narrowed arteries reduce blood flow to the heart (ischaemia). Total blockage results in death of heart tissue (myocardial infarction)

Risk factors

- Family History
- Sex (pre-menopausal, non smoking women are at lower risk than males)
- Hyperlipidaemia/cholestreamia
- Obesity
- Stress
- Smoking
- Diabetes
- High blood pressure
- Sedentary lifestyle

Angina

- Angina is a symptom of reduced blood flow to the heart. Angina is often described as chest tightness, heavy sensation, ache or pain which may radiate to the arm and jaw.
- Many people perceive it as a ‘discomfort’ rather than pain.
- Angina provoked only by exercise and which settles when exercise ceases is referred to as **stable angina**
- An exercise test can be a useful diagnostic tool to assess ischaemia.

Unstable angina

- Increased frequency and severity of angina
- Episodes of angina occurring recurrently and unpredictably, without the specific provocation of exercise.
- Pain becoming less tolerant to GTN spray

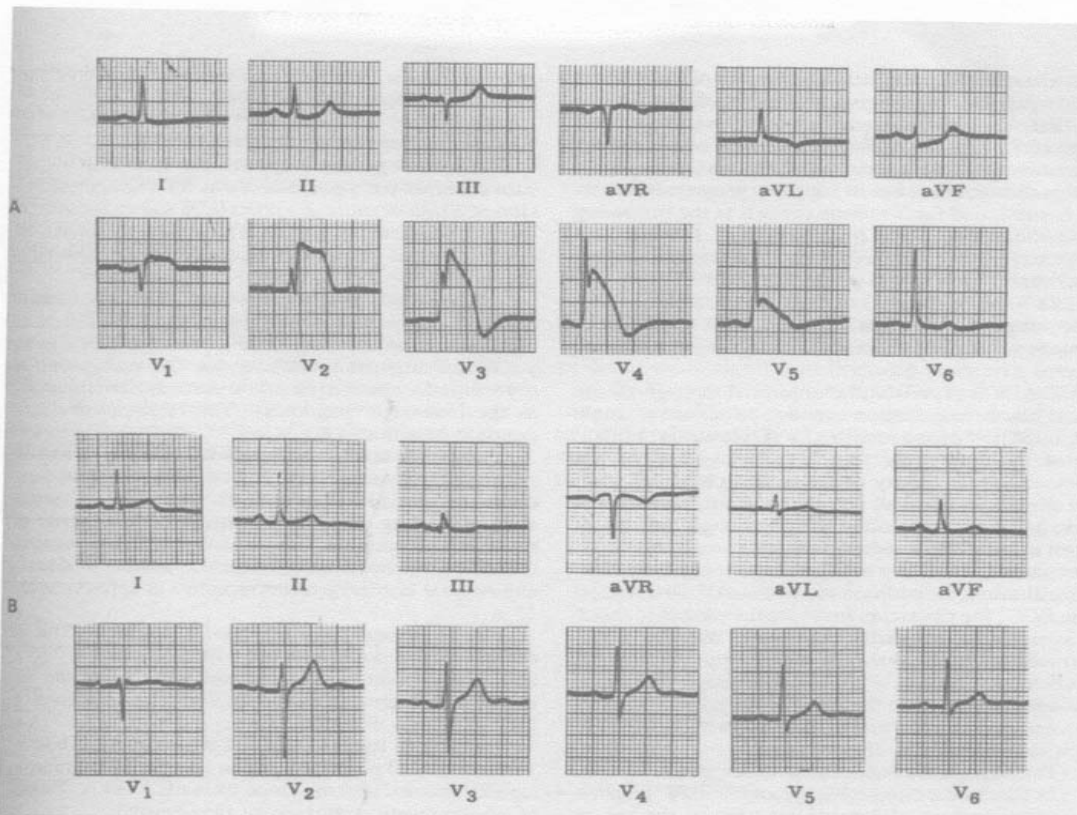
Other types of angina

Prinzmetal angina is a form of angina that can mimic an MI. It is associated with pain and ST elevation. This type of angina is generally more severe in the nature and duration of pain. The ST segment normalizes once the pain subsides.

Prinzmetal Angina

A. This ECG was taken during an episode of anginal pain that occurred whilst the patient was at bed rest in the hospital. There is marked ST elevation in leads V2-V5 with some ST depression in aVF

B. This tracing was taken 30 minutes later. The patient was pain free. The ECG shows isometric ST segments and is now normal. Serial enzyme measurements and ECG's showed no evidence of MI. Although trace A is quite typical of an early MI, the rapid disappearance of the ST elevation and absence of other indicators of MI indicate that this is Prinzmetal angina.



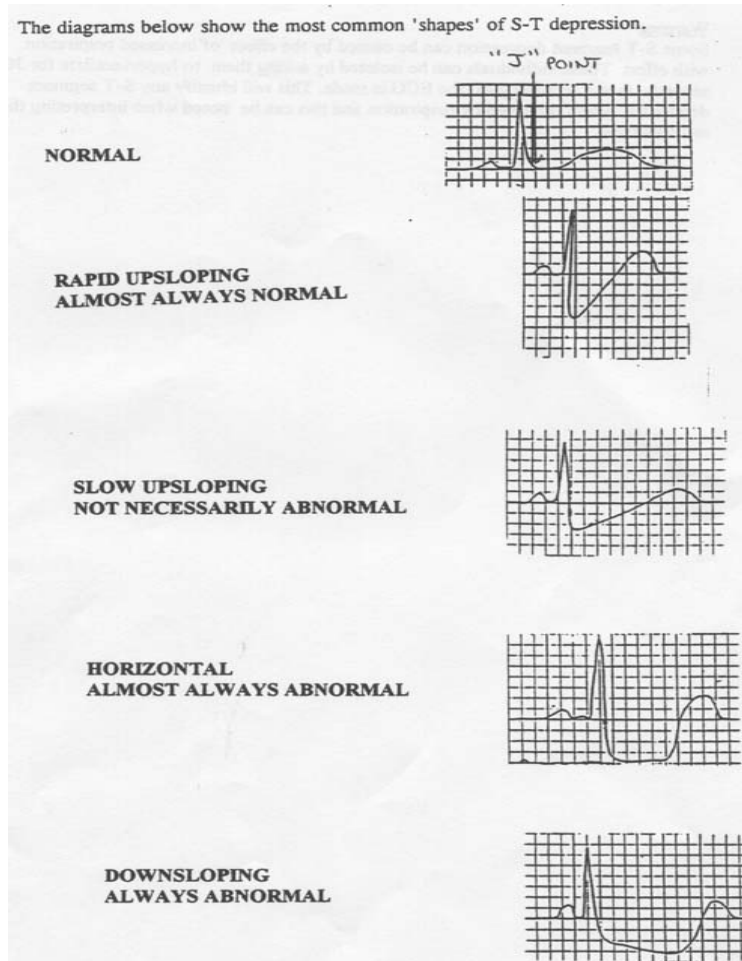
Case study 1

- Gentleman attended the Cardio-Respiratory Department on the 11/07/02 for an exercise test.
- History of ischaemic heart disease and has had a previous MI. Test requested to see how well he has recovered 6 weeks post MI.
- Comment on the results

Results of the exercise Test

- Resting Q wave in lead III
- 4mm ST depression in V2-V4 after 1.49mins.
- 2mm ST depression in V5-V6 after 1.49mins
- Some ST changes seen in leads II,III,aVF and lead
- Asymptomatic during test
- Slow recovery of the ST segment in recovery.
- Extensive anterior ischaemia. Suggests disease in the left anterior descending artery but probable multi-vessel disease.

Types of ST depression



Myocardial infarction

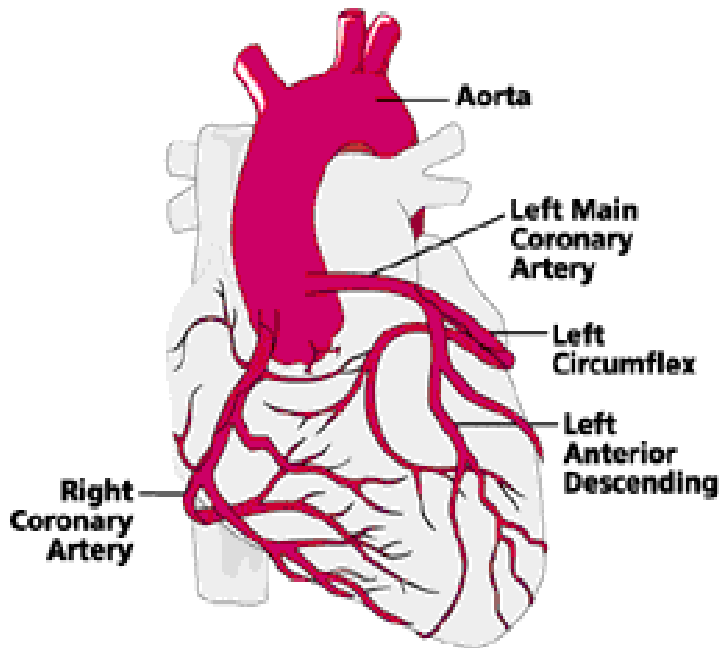
- **Myocardial infarction** is the ‘death of heart muscle (myocardium)’.
- Death of tissue results from a **total blockage** in **the artery**, preventing blood flow to the myocardium it supplies.
- **Tissue damage produces Q waves** (Q wave MI or ‘acute MI’)
- **Partial tissue damage may not produce Q waves** (non Q wave MI or ‘subendocardial’)

Infarction is a Latin term meaning ‘to stuff’-when the myocardium is viewed under a microscope it becomes stuffed with cells to help heal the damage.

Blood supply to the myocardium

The coronary arteries

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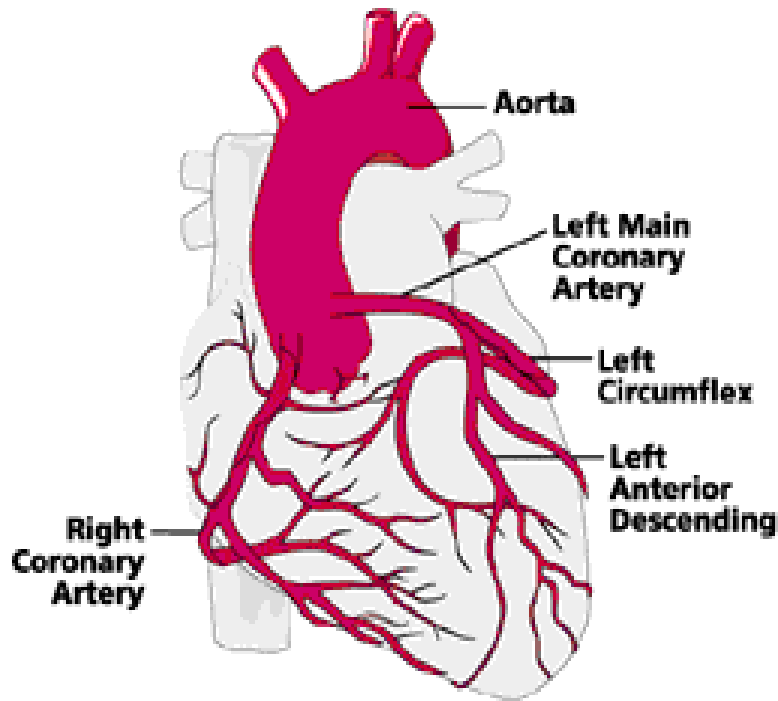


- The heart muscle supplies itself with oxygen and nutrients from coronary arteries which originate from the aorta
- Blockage causes most of the heart disease in this country.
- Heart attacks, heart failure, angina and sudden death occur from blockage to one or all of these arteries

Blood supply to the myocardium

The left coronary artery

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The left main coronary artery branches into the anterior descending (LAD) and the circumflex (Cx).

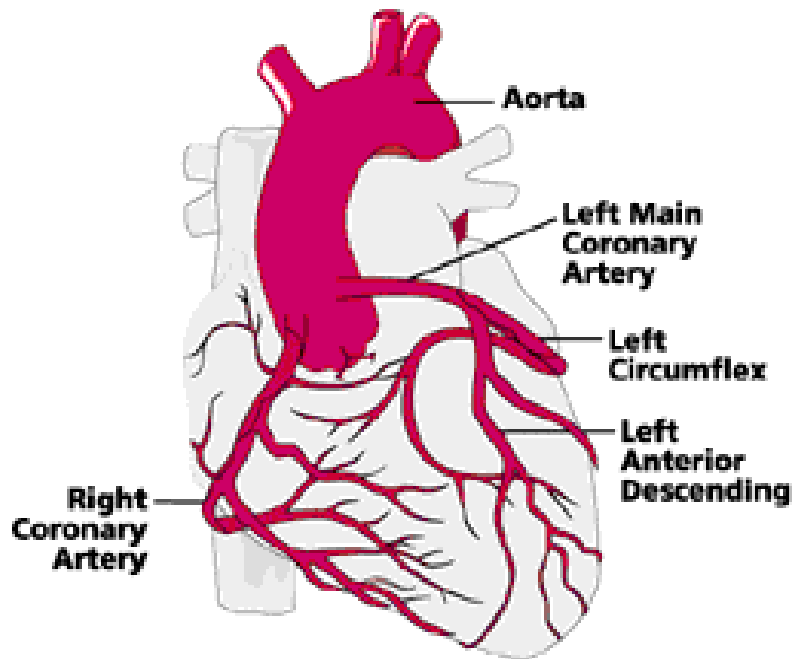
The Cx wraps around the left side and back of the heart-the lateral(side) portion of the left ventricle (V5, V6, I, aVL)

The LAD supplies the front of the heart-anterior portion of the left ventricle(V1-V4)

Blood supply to the myocardium

The right coronary arteries

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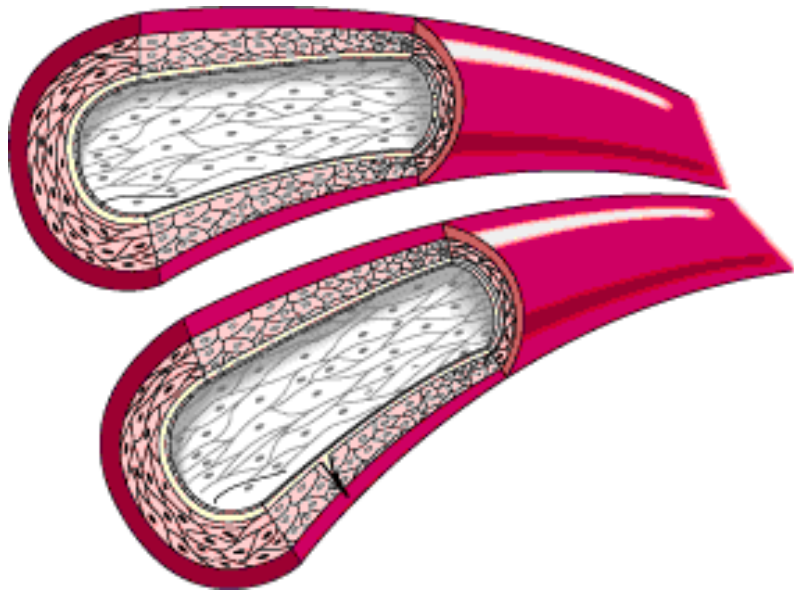
Right coronary supplies the SA node, AV node and the His Bundle!?. And the back (posterior) part of the heart

acute posterior infarcts can therefore lead to dangerous arrhythmias and may need a temporary or permanent pacemaker

Blood supply to the base of the heart

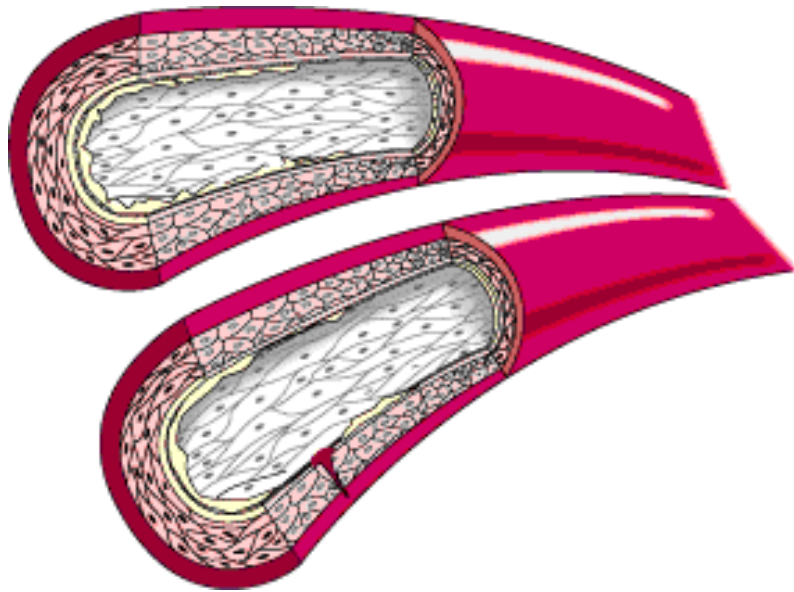
- The base of the heart is supplied by either the LCA or RCA depending on which is dominant, therefore an inferior infarct are caused by occlusion of either vessel
- Inferiors are seen in leads II, III and aVF

Development of coronary artery disease



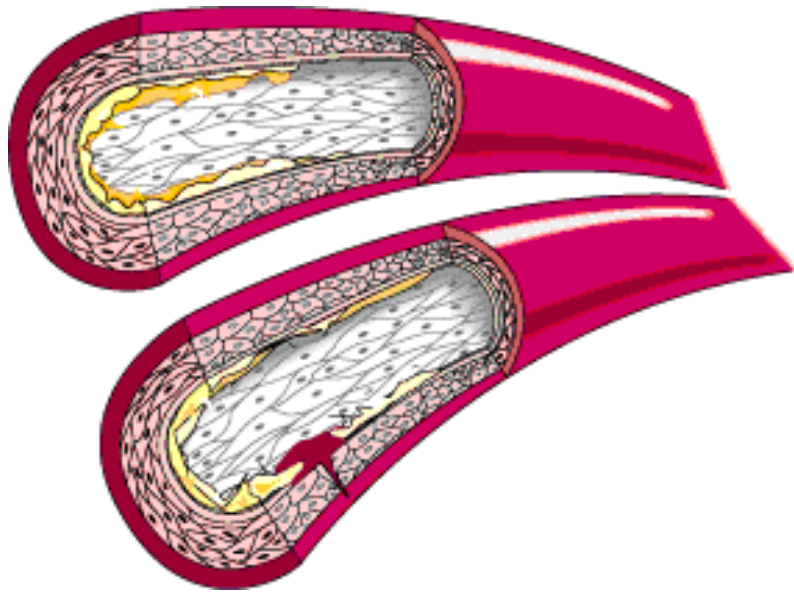
- Deposits of lipids (atheroma) in the arterial wall build up over many years
- Thickening and fibrosis occurs leading to calcification, known as atherosclerosis (hardening of the arteries)

Development Of Coronary Artery Disease (cont..)



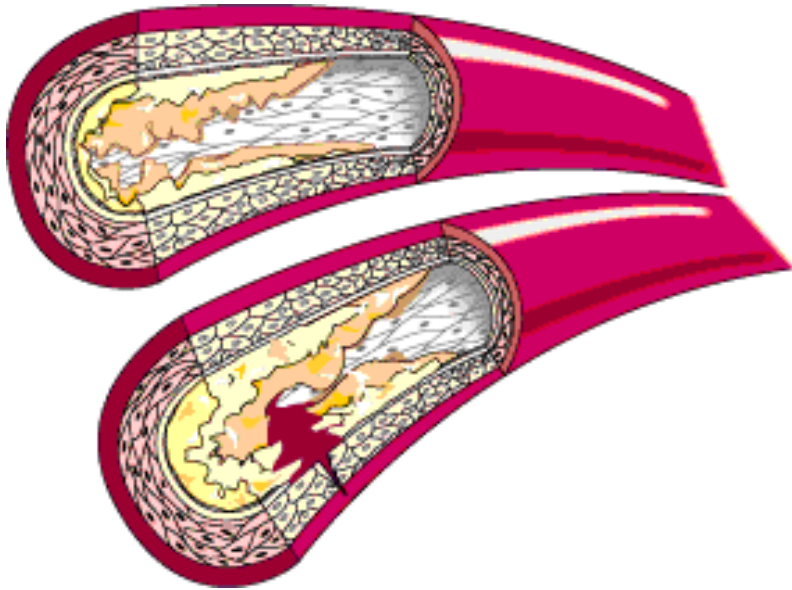
- Fatty deposits over a number of years add stress to the arterial wall causing inflammation
- This begins to eat away at the inside lining of the artery

Development Of Coronary Artery Disease (cont..)



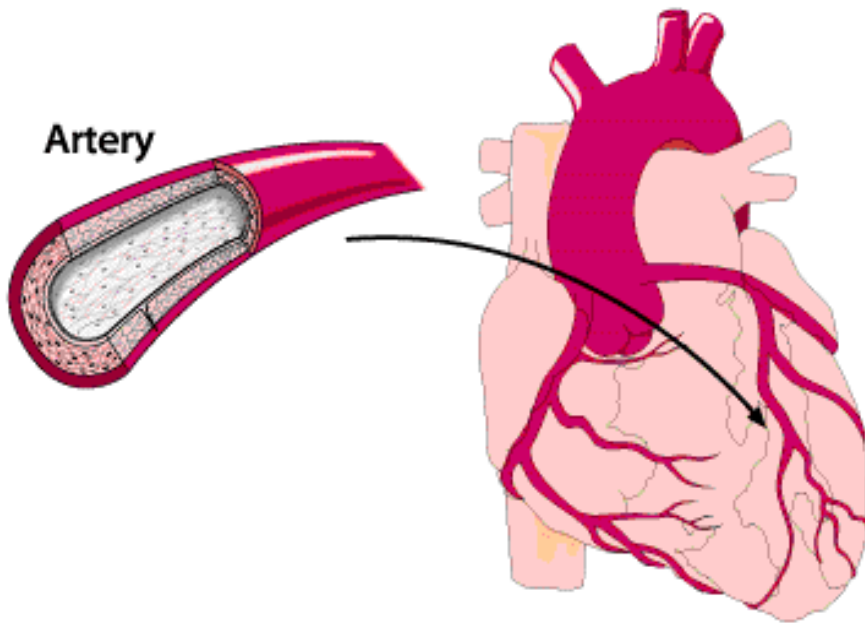
- The fatty plaque eventually becomes exposed to the bloodstream
- This results in clot formation- the bodies natural response to prevent further damage

Development Of Coronary Artery Disease (cont..)



- The clot continues to grow and begins to occlude the artery
- As the artery becomes narrower the part of the heart it is supplying blood with will become 'ischaemic'

Development Of Coronary Artery Disease (cont..)

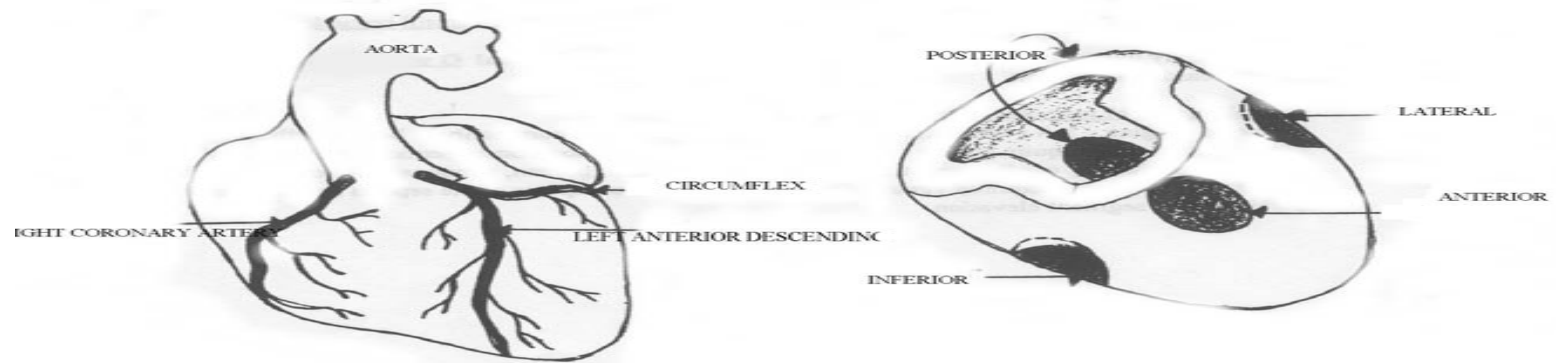


- Sudden blockage of the artery can occur by plaque rupture, leaving its contents open in the blood stream. This clots and totally cuts off the blood supply to the myocardium leading to myocardial infarction.
- Unblocking the arteries is crucial to prevent further myocardial damage

Severity of coronary artery disease

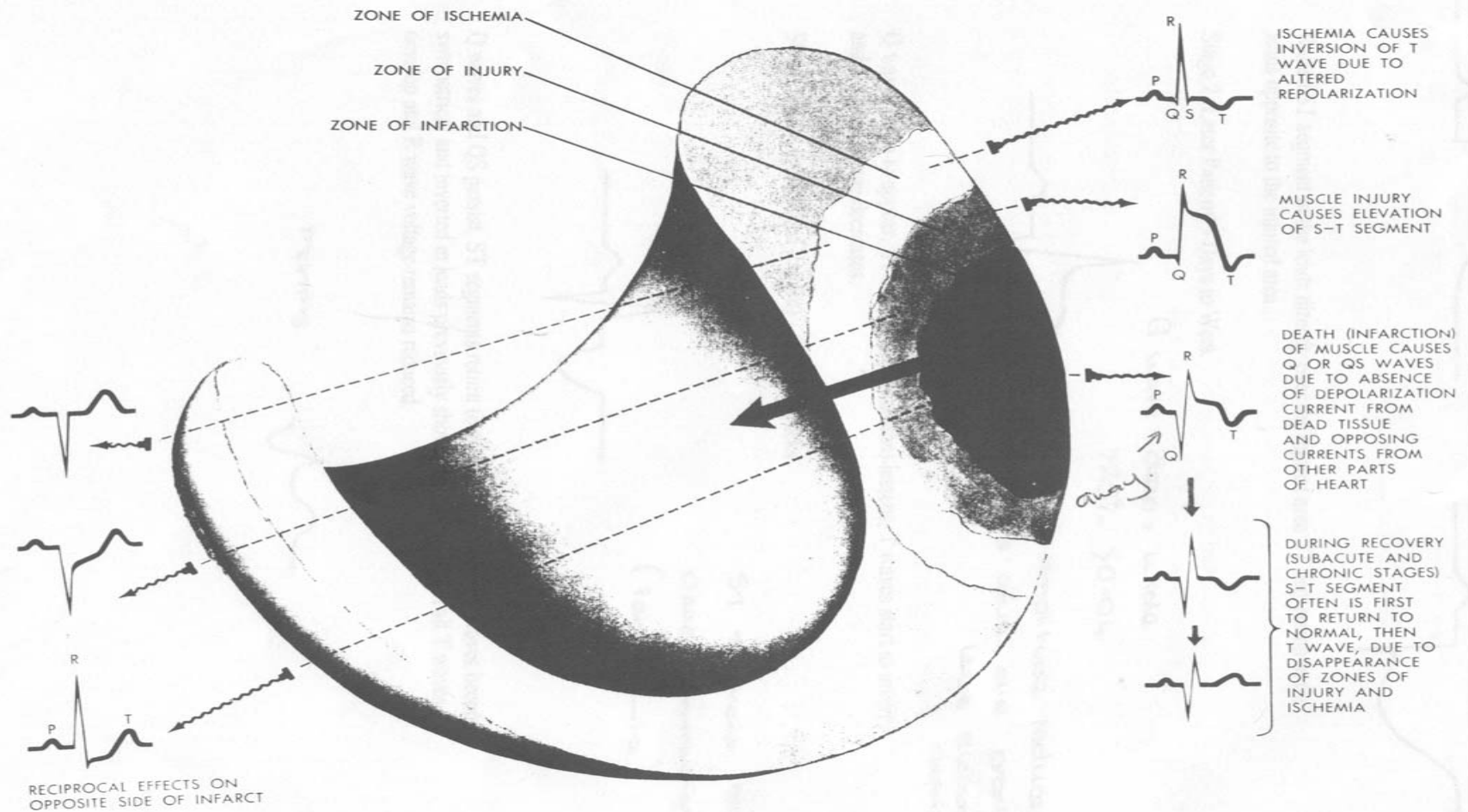
- Coronary arteries can become up to 50% narrowed before symptomatic.
- Mild narrowing- heart muscle is generally healthy at rest but with exercise arteries cannot meet the oxygen demand of the muscle and angina may be experienced.
- 50-90% stenosis. Unstable angina
- 90-99% stenosis. High risk of sudden death from plaque rupture

Location Of Infarction



- The heart has front, back and sides. Surface electrodes can identify which part of the heart has been involved.
- Once you know which part of the heart has been involved this can give you an idea about the extent of disease and which coronary arteries maybe involved
- Anterior: V2, V3, V4
- Extensive anterior: I,aVL,V1-V6
- Inferior: II,III,aVF
- Infer lateral: II,III,aVF, I, aVL,V5-V6
- Posterior V1, V2

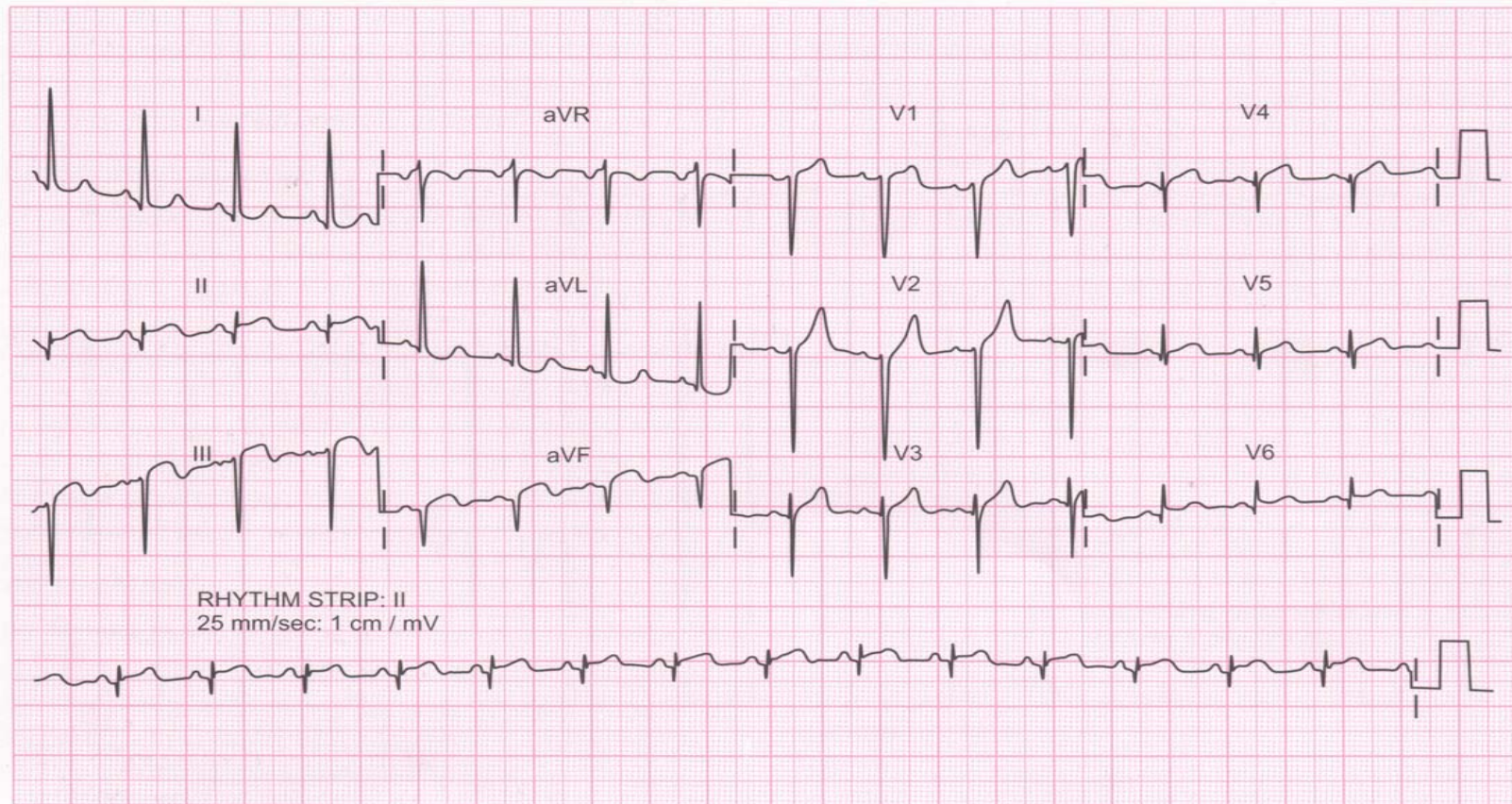
Zones of tissue damage



Localization Of Infarction using the ECG

- Antero-septal: I, aVL, V1-V3
- Antero-Lateral: aVL, II, III, V4-V6
- **Anterior: V2, V3, V4**
- **Extensive anterior: I, aVL, V1-V6**
- Apical: I, aVL, V3, V4
- High lateral: aVL only but if move chest leads 1 or 2 ribs spaces higher may see changes
- **Inferior: II, III, aVF**
- Infer lateral: II, III, aVF, I, aVL, V5-V6
- Inferoseptal: II, III, aVF, V1-V3
- **posterior: V1, V2**
- **Sub-endocardial: any lead usually multiple. T wave inversion or ST depression**

INFERIOR MYOCARDIAL INFARCTION



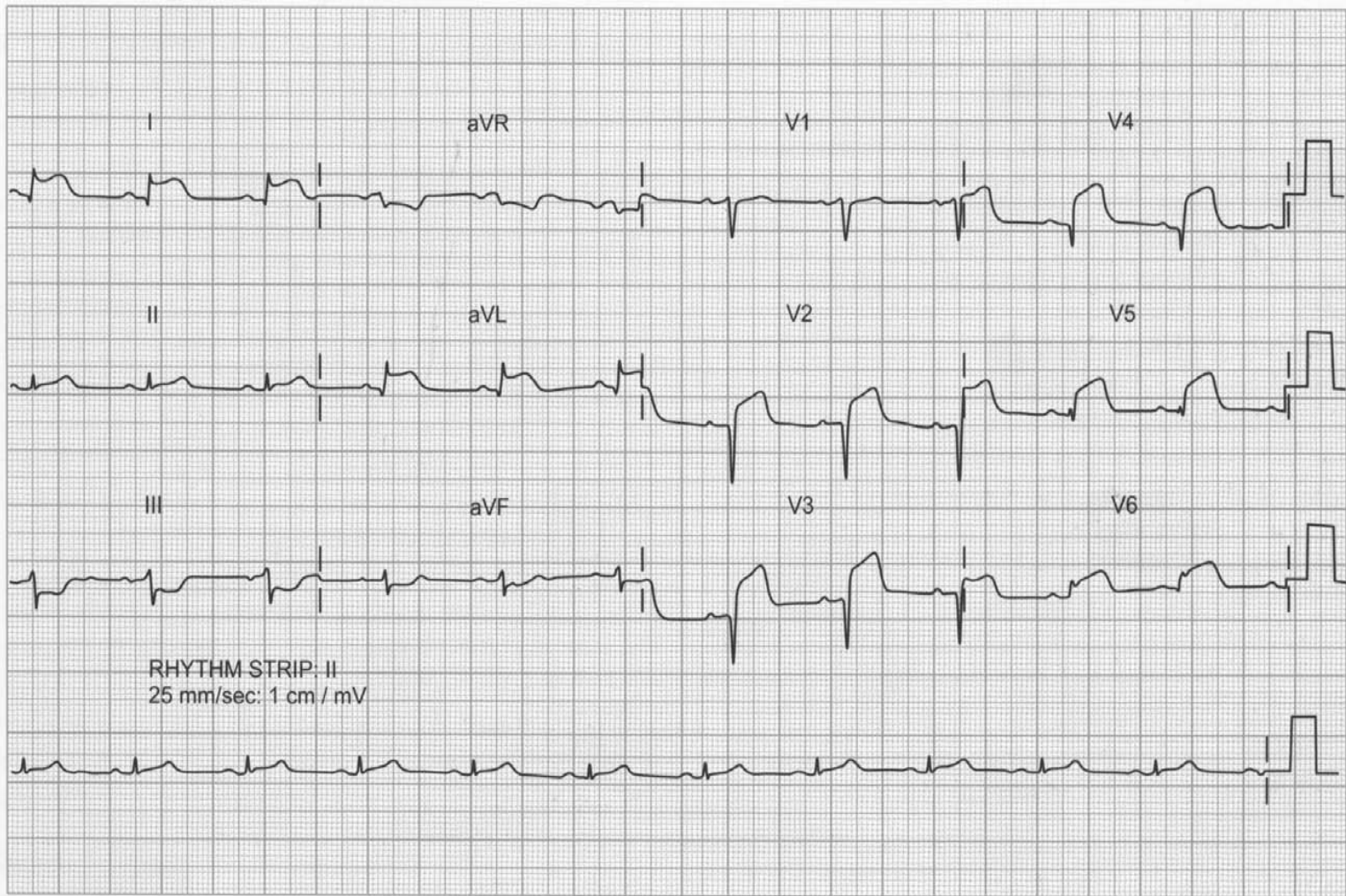


Figure 3.2 12-lead ECG - Anterolateral MI

POSTERIOR MI

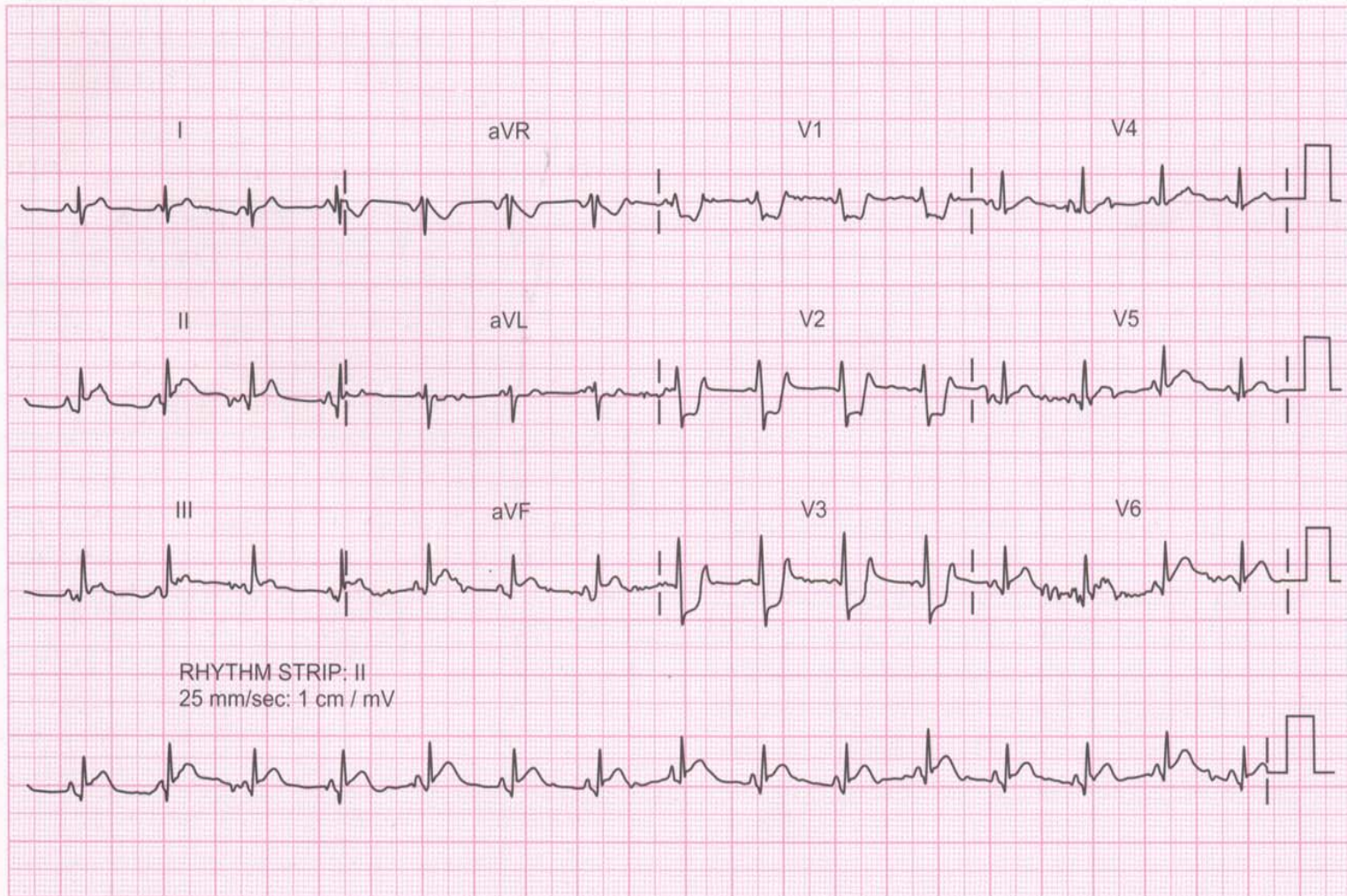
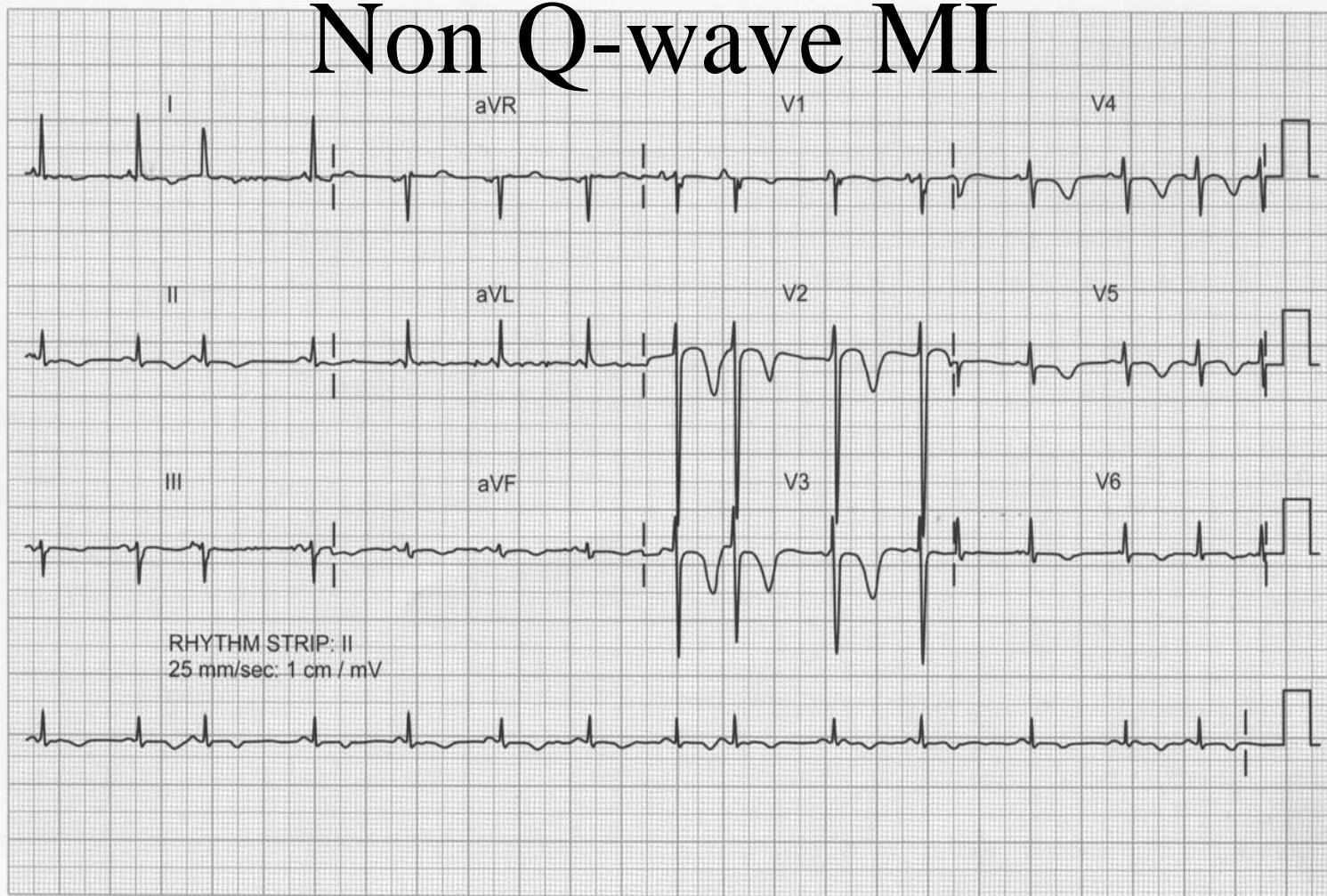


Figure 3.4 12-lead ECG - Posterior MI

Original kindly supplied by Dean Jenkins

Non Q-wave MI



Case study 2

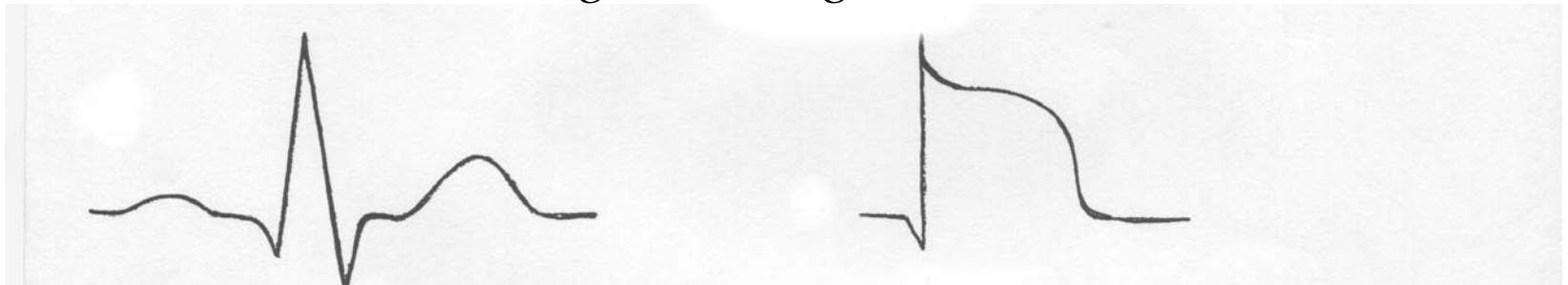
Sequential ECG changes during an acute MI

- *Stage 1*-very early, within hours of onset

Myocardium is not yet dead but will be unless there is intervention.

Leads over this area will show ST elevation and loss of R wave height and loss of t wave. ST depression in the leads opposite the injured area

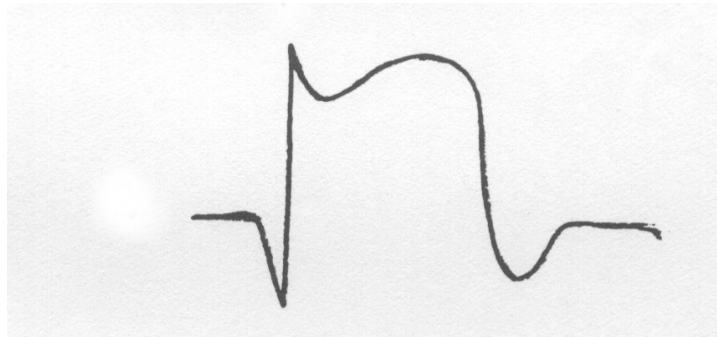
Often associated with crushing central chest pain, clamminess, sweating, vomiting, breathlessness



Sequential ECG changes during an acute MI

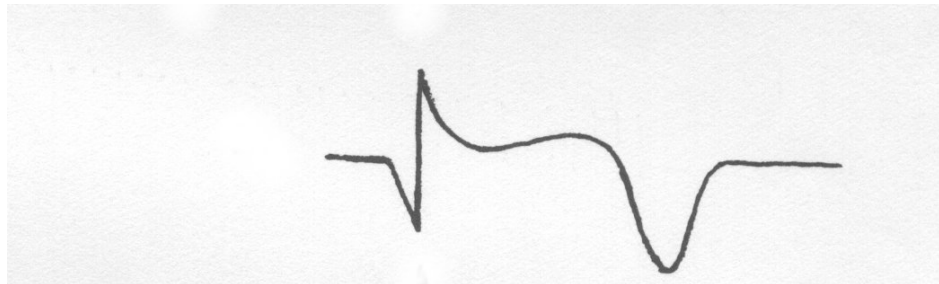
Stage 2-within days of onset

- The injured myocardium starts to necrose and changes to the ECG will prove that infarction has occurred.
- Leads facing the infarct see through to depolarization in the opposite part of the heart-shown as Q waves. Should be $>25\%$ of Rwave size and $>0.04\text{sec}$ wide
- Reduction in ST elevation.
- Reduction in R wave voltage (due to a reduction in myocardium,as some as died)
- T wave inversion due to persistent ischaemia surrounding the infarcted area



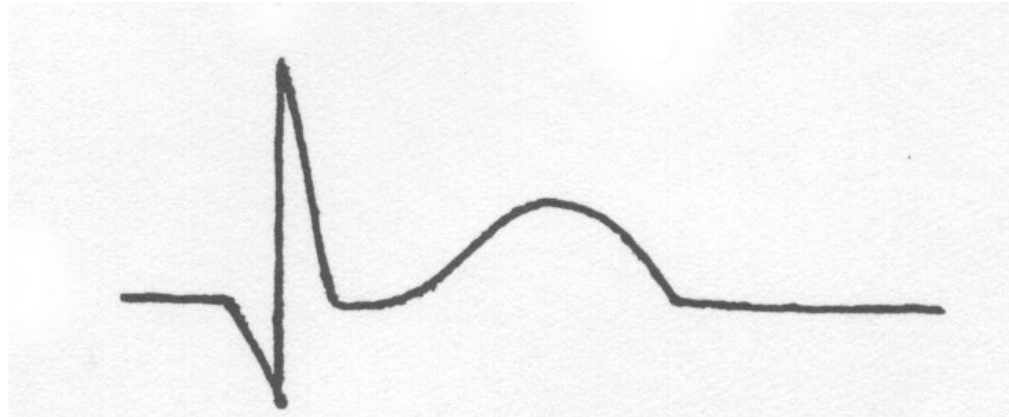
Sequential ECG changes during an acute MI

- Stage 3-within 1-2 weeks
- The zone of injury has evolved into infarcted myocardium.
- Pathological Q waves
- Reduction in R wave height
- QS pattern
- ST segments returned to normal
- T wave inversion persists due to ischaemia in the surrounding area (may never normalize)



Sequential ECG changes during an acute MI

- Stage 4-months to years post infarction
- T waves return to normal as the zone of ischaemia recovers
- Reduction in R wave voltage over the infarcted area.
- Q waves remain as the lifelong fingerprint for myocardial infarction



Complications of an acute MI

- Regurgitation
- Septal rupture
- Tamponade
- Aneurism
- Cardiogenic shock

This will be discussed in a future lecture

Treatment and intervention

- Thrombolysis
- Angioplasty
- Bypass surgery

This will be discussed in a future lecture