

Acute coronary syndromes in primary care

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Chest pain - history

Stable angina

- Substernal chest discomfort
- Characteristic quality and duration
- Provoked by exertion or emotional stress
- Relieved by rest or nitrates

Unstable angina

- Pain occurring at rest
- Prolonged chest pain >10 min
- No response to GTN
- Change in frequency or severity of pain
- More frequent pain occurring with less exertion (crescendo angina)

Atypical presentations of ACS

More common in women, diabetic or elderly patients

- Epigastric pain
 - “Indigestion”
 - Arm pain
 - Back pain
-
- Sudden onset back pain – consider aortic dissection

Risk stratification

High risk features

- Ongoing/recurrent chest discomfort despite initial Rx
- Elevated cardiac troponin
- New ischaemic ECG changes
- Diaphoresis
- Haemodynamic compromise
- Sustained VT
- Syncope
- Known LV dysfunction LVEF <40%
- Prior AMI, PCI or CABG

Low risk features

- Age <40 years
- Symptoms atypical for angina
- Remains symptom-free
- Absence of known CAD
- Normal troponin level
- Normal ECG

Intermediate risk – neither high or low-risk criteria

Nonanginal or nonischemic chest pain

- None of the characteristics of classic anginal pain
- Highly localised pain
- Pleuritic - pain worsened by inspiration, coughing
- Musculoskeletal – pain worsened by movement

History



Examination

Stabilisation
and
treatment



ECG



Examination

- **Airway – conscious state**
- **Breathing**
 - tachypnoea, respiratory distress, low O₂ sats (?acute pulmonary oedema)
 - crepitations in lung fields
- **Circulation**
 - **Blood pressure**
 - Hypotension, peripheral shutdown (cardiogenic shock)
 - Difference between arms (? Aortic dissection)
 - **Murmur**
 - **Gallop rhythm**

Initial clinical management

- **Oxygen** only if O₂ saturations <93% (target 88-92% in COPD)
- **Pain relief /Pain relief escalation:**
 - Sublingual GTN (tablet/spray) every 5 min for up to 3 doses (in absence of hypotension)
 - Titrated morphine
 - Consider GTN infusion if ongoing pain
- **Aspirin 300mg orally in all patients**
- **ECG within 10minutes**
- **Refer to emergency department**

Chest pain within 24hrs

- Initial clinical assessment including history, examination, ECG and single troponin testing are unable to exclude a diagnosis of ACS by themselves.
- For this reason, patients who present to primary care with chest pain (within 24 hours) and suspected ACS should be referred as soon as possible to the ED or facility capable of definitive risk stratification and diagnosis of ACS.

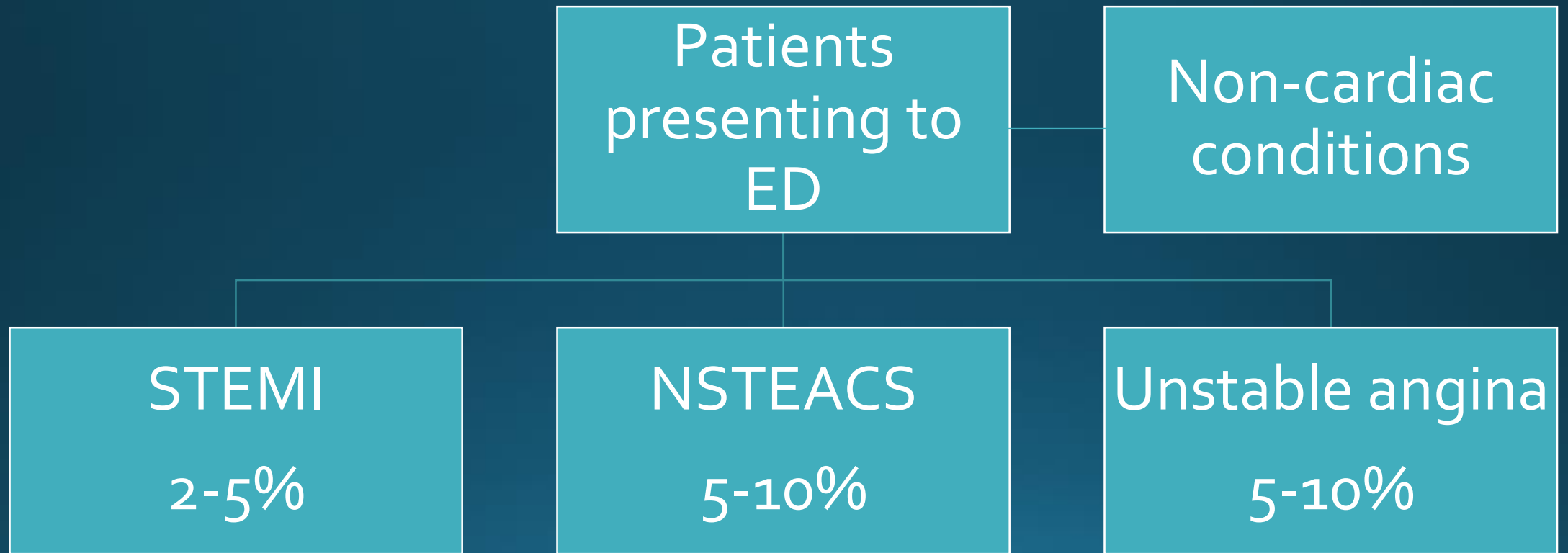


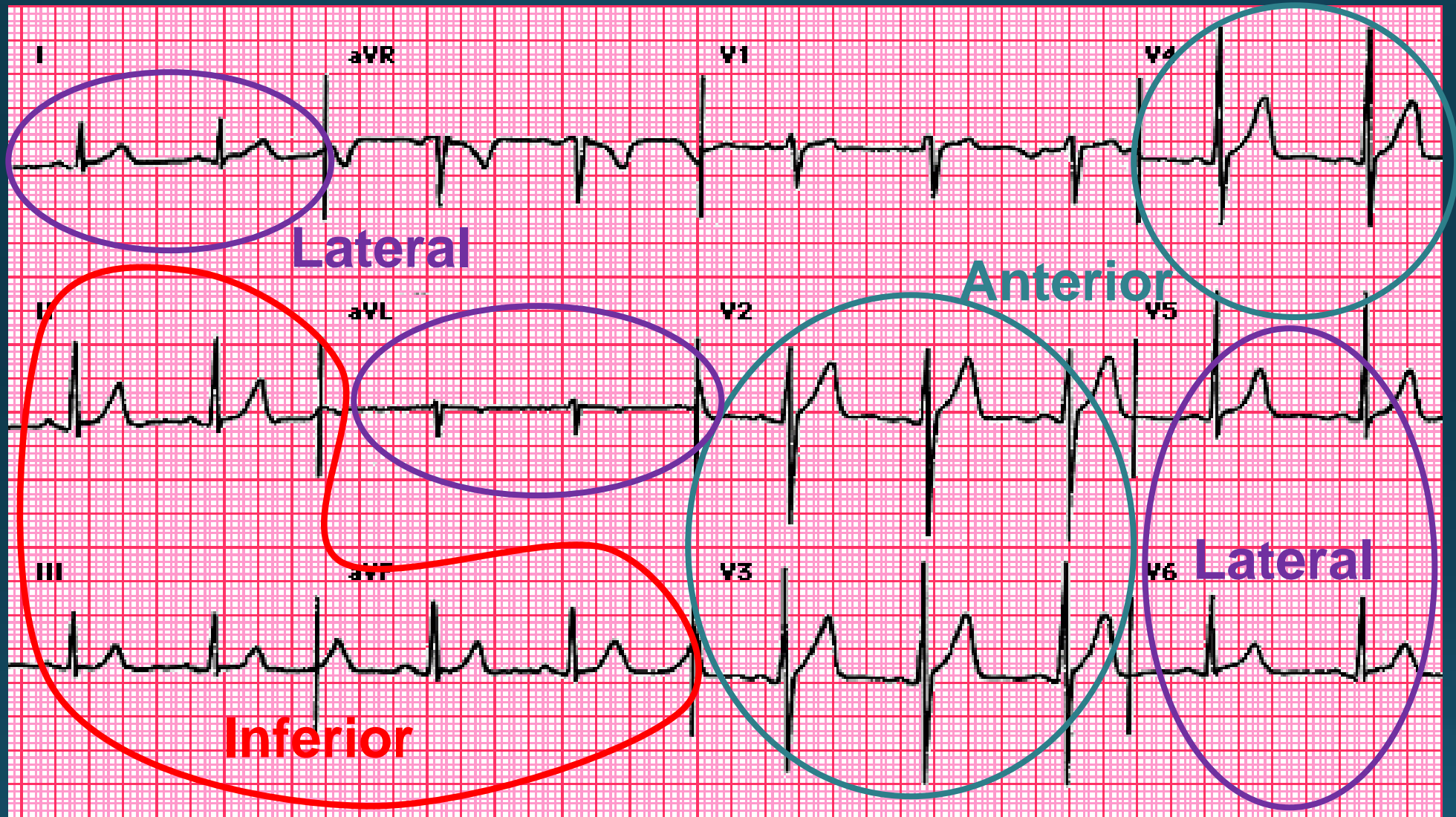
Chest pain within 24hrs

- Patients presenting with high-risk features such as ongoing chest pain, dyspnoea, syncope/presyncope or palpitations should be referred immediately to the ED (goals of management include establishing the diagnosis with an ECG if available, and ensuring immediate access to cardiac defibrillation where possible)
- For this reason, patients should not drive themselves to the ED and transport by emergency medical services is recommended.
- Referral to ED should not depend on troponin testing



Incidence of acute coronary syndromes presenting to ED

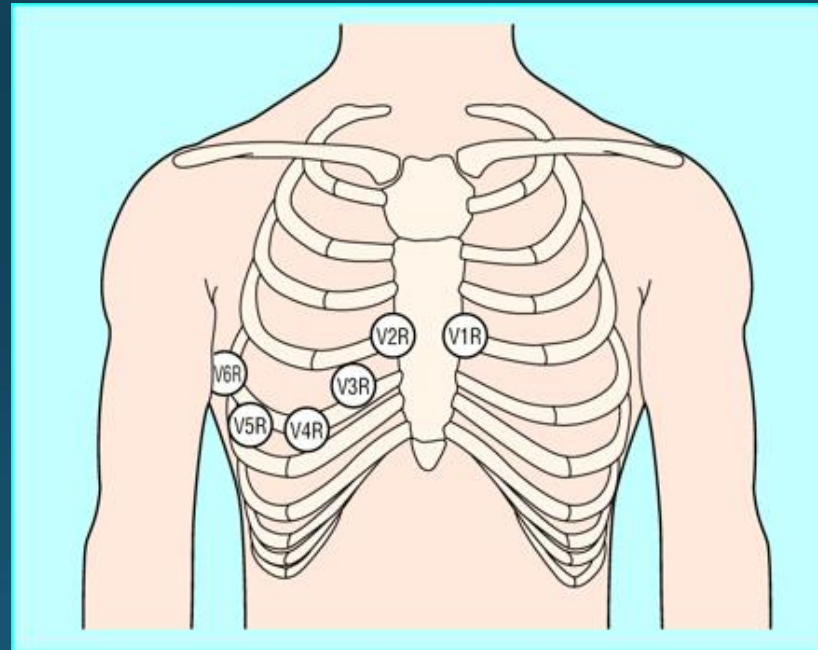




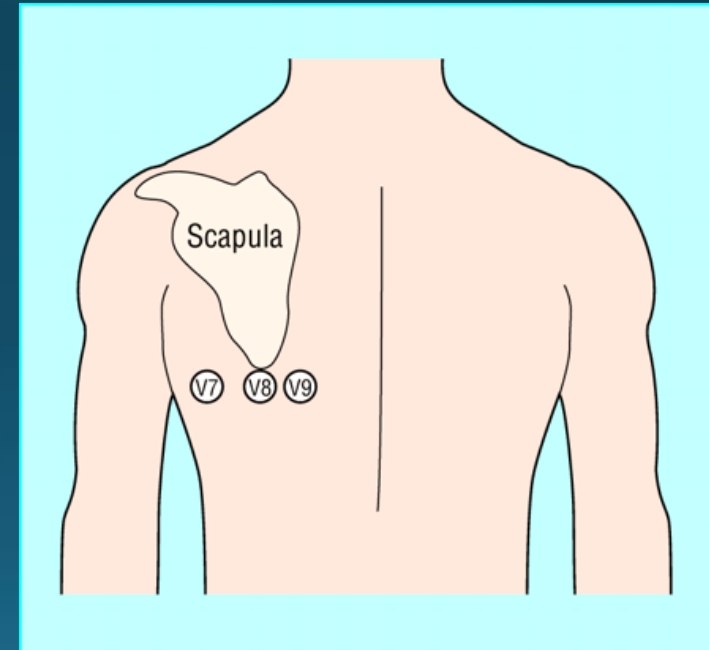
ECG territories

Other territories

- Inferior MI – can have RV involvement
 - RV leads
- Posterior MI
 - V7–V9

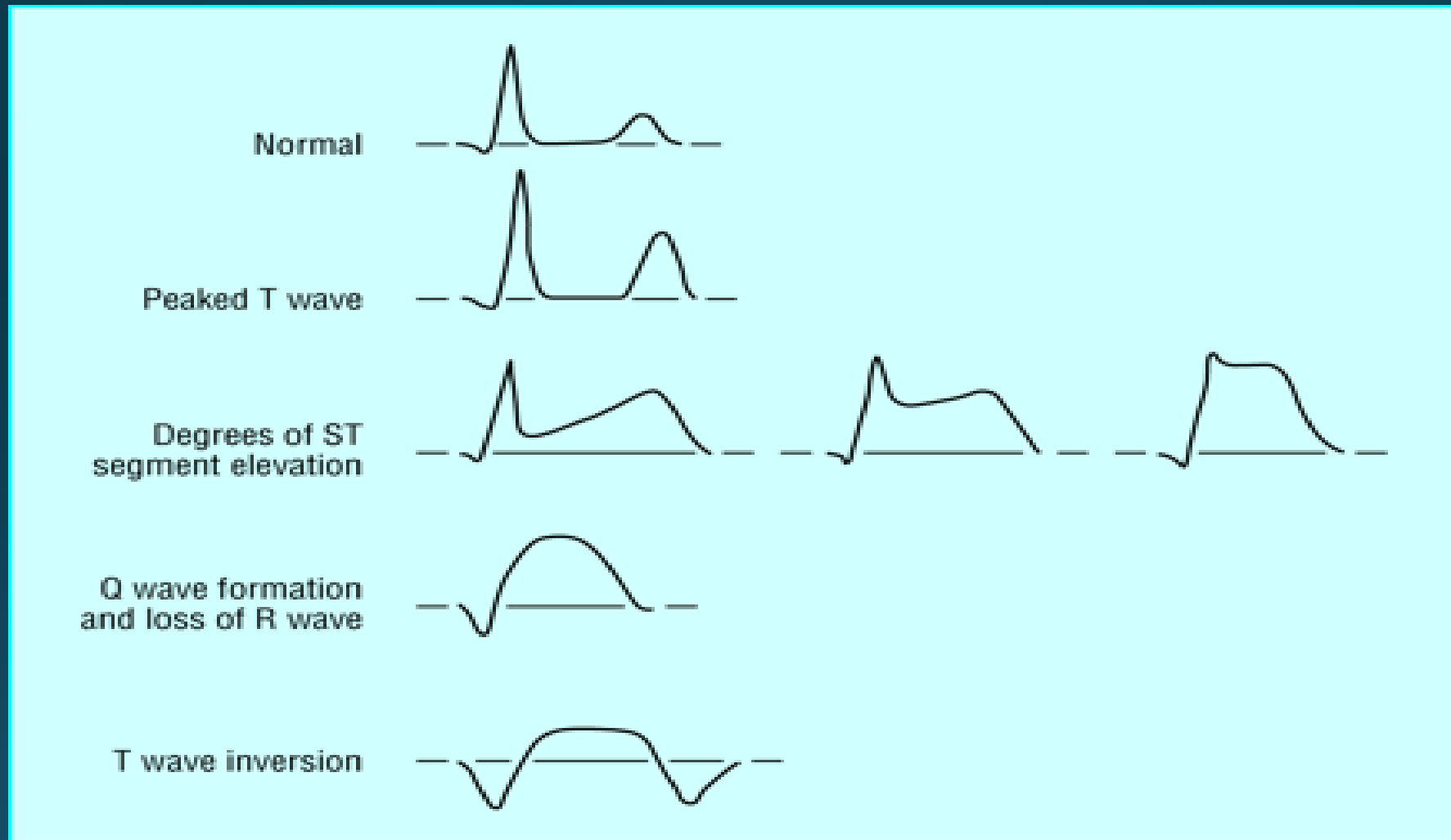


RV lead positioning



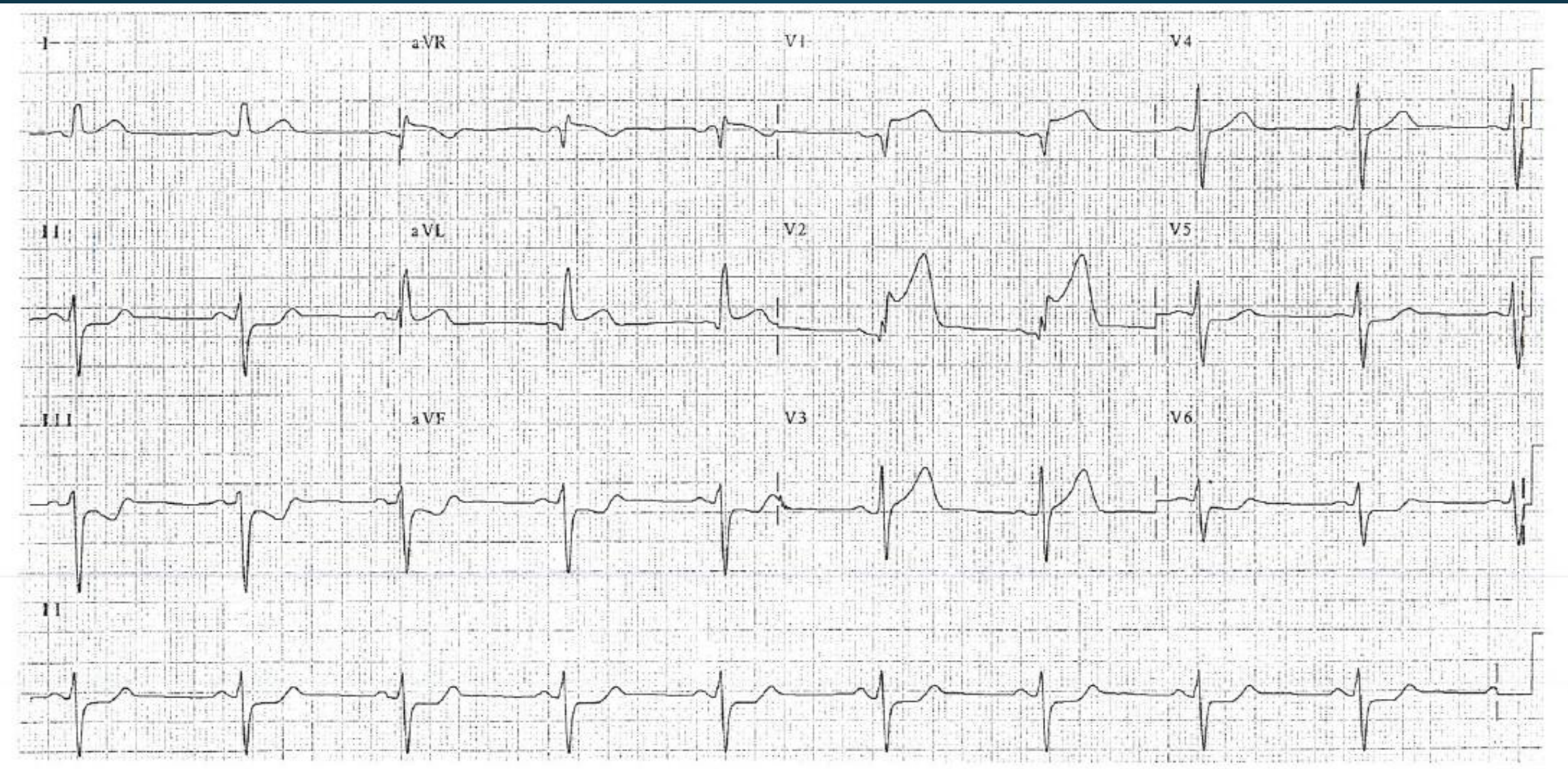
Posterior lead positioning

STEMI - Evolution of ECG changes



Diagnostic criteria for ST-elevation MI

- Typical chest pain
- 12 lead ECG showing
 - ≥ 2 mm ST elevation in precordial leads (V₂-V₃) in men >40 yrs (≥ 2.5 mm in men <40 yrs, ≥ 1.5 mm in women)
 - 1mm ST elevation in other leads
 - New LBBB
- There may be reciprocal changes (ST depression)



I

aVR

V1

V4

II

aVL

V2

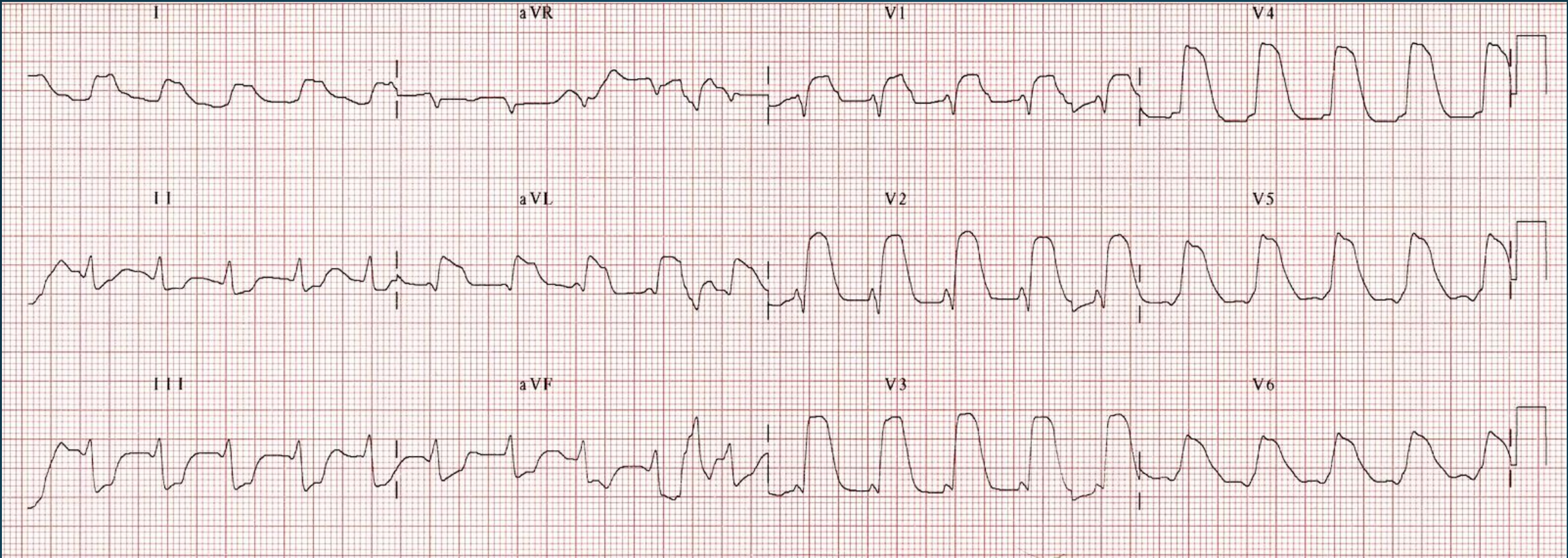
V5

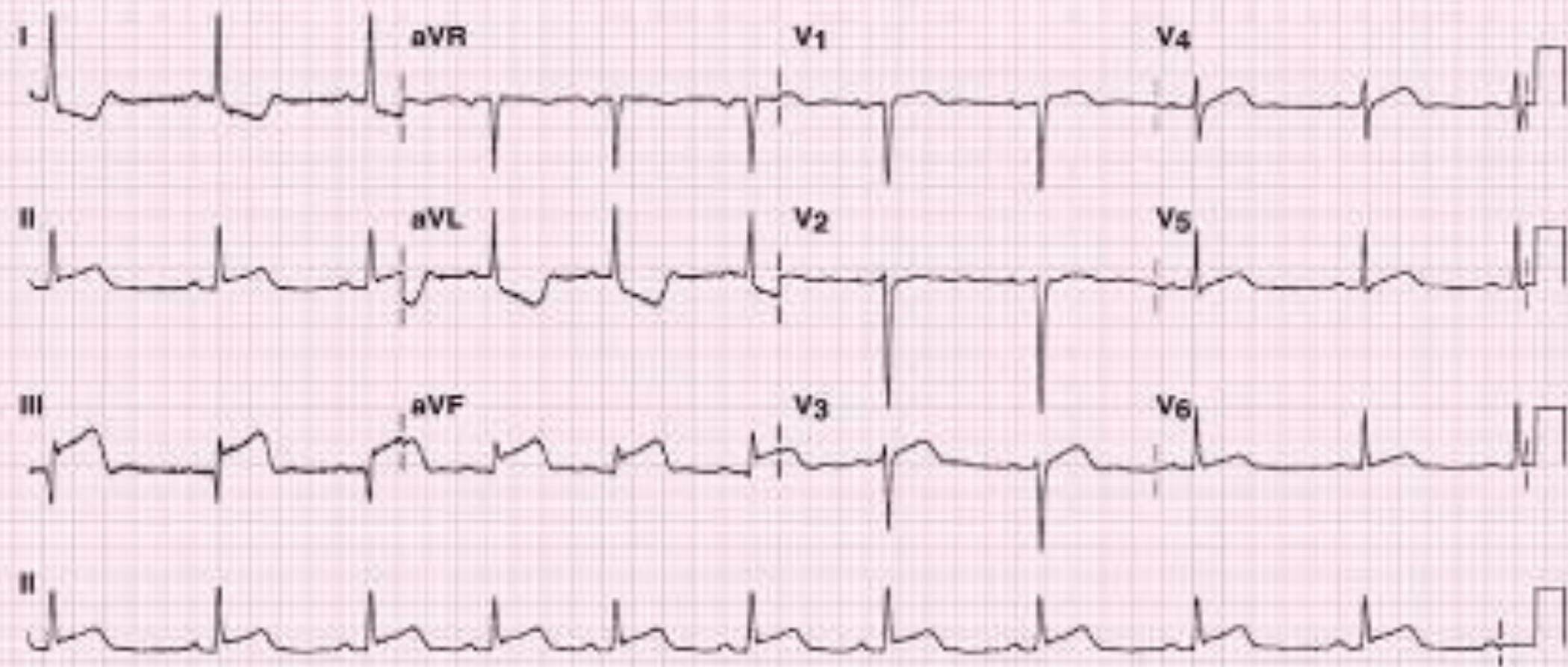
III

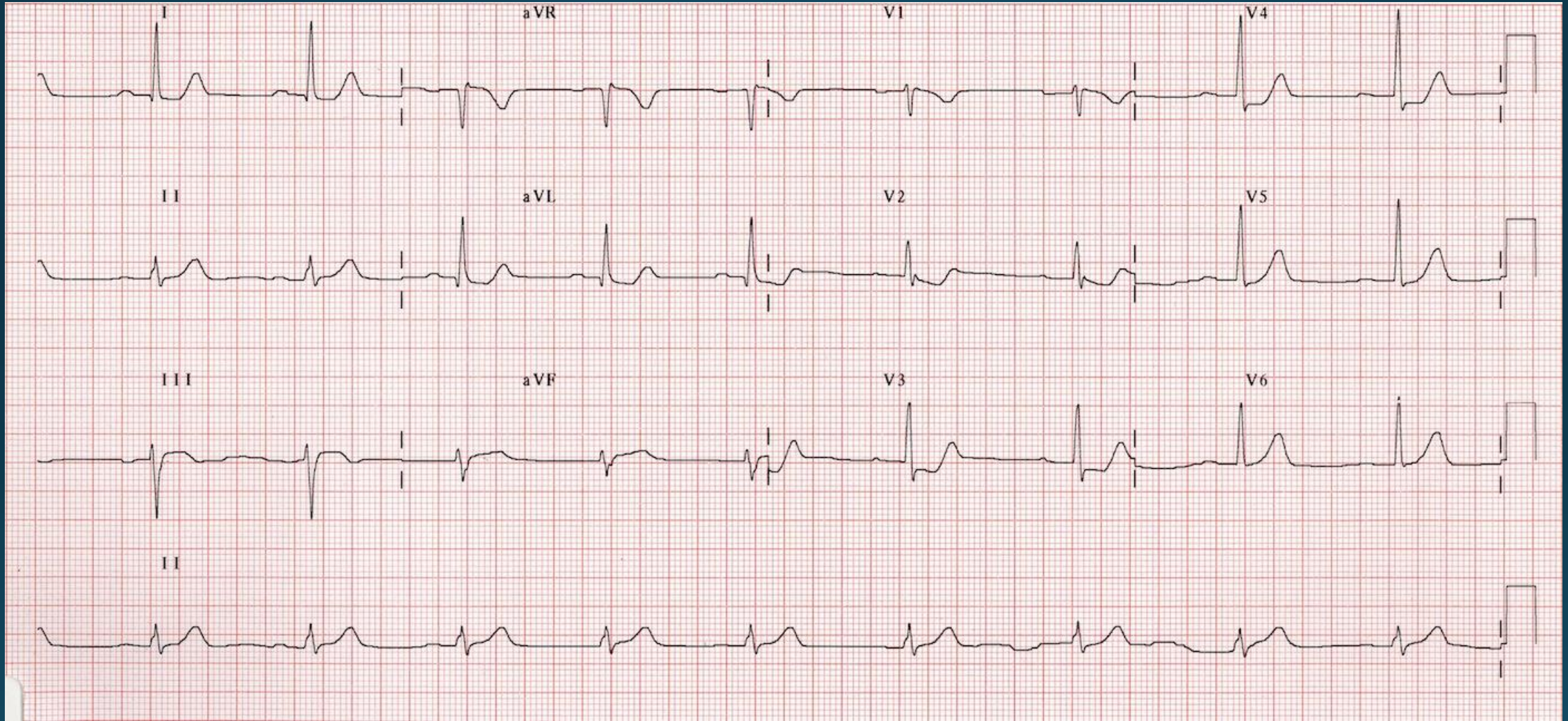
aVF

V3

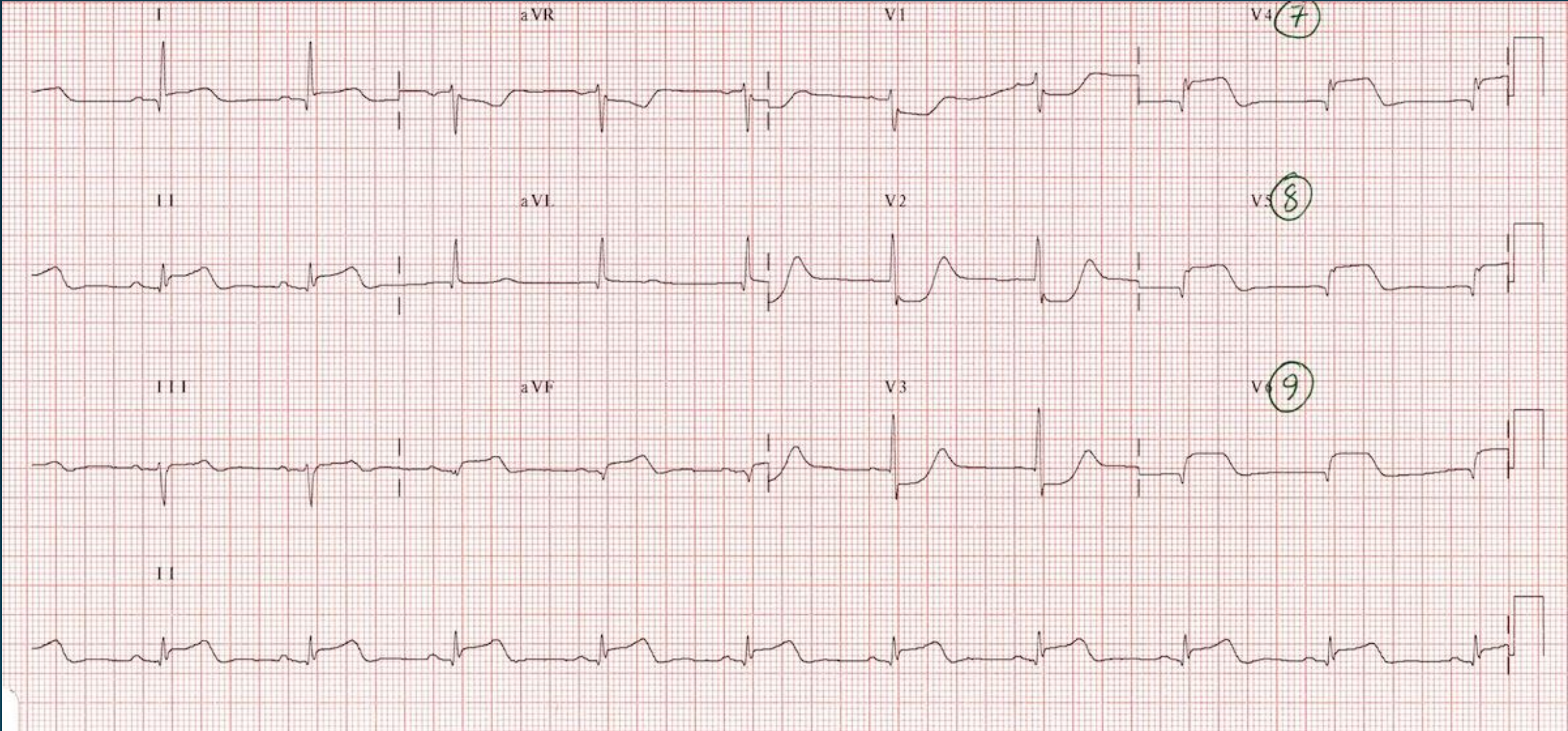
V6





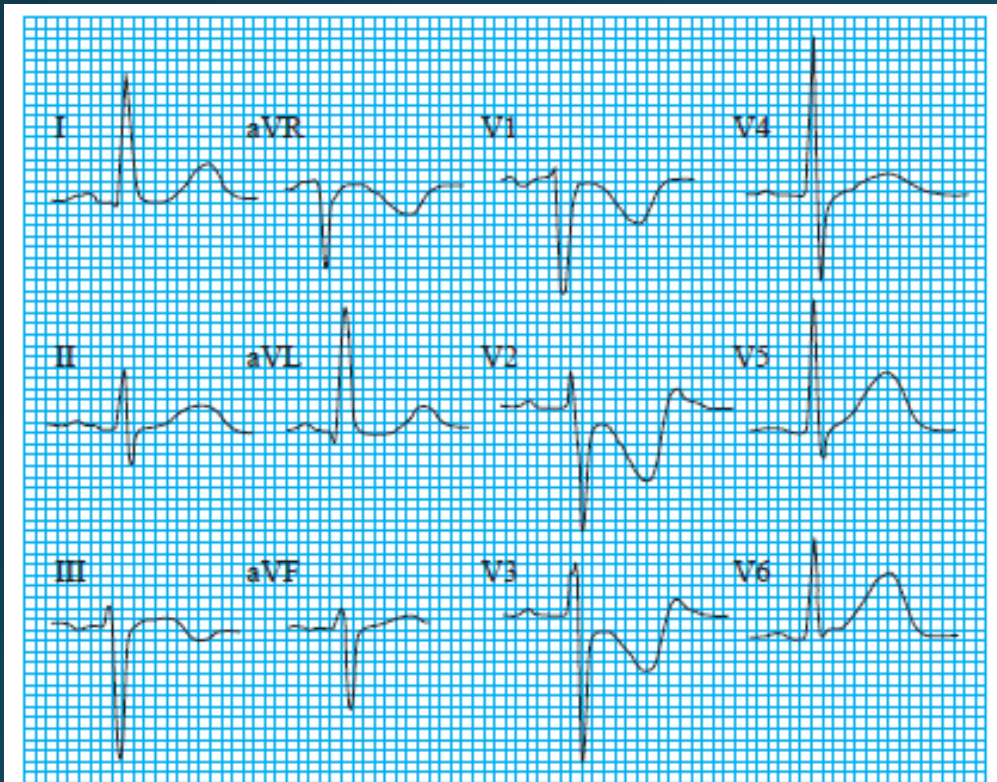


Posterior leads

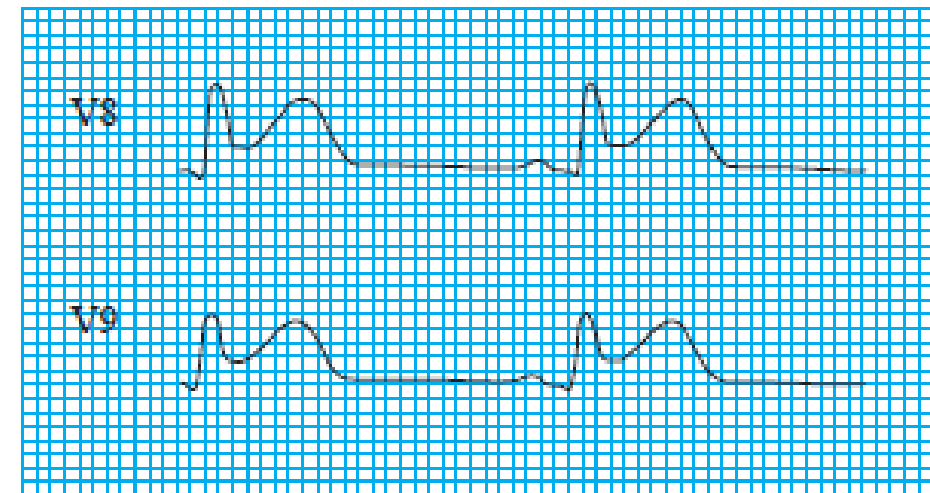


Posterior STEMI

Posterior infarction may be clinically silent on a standard 12-lead ECG
Perform posterior leads if patient has ongoing typical pain with no ECG changes or anterior ST depression



Isolated posterior infarction with no associated inferior changes (note ST segment depression in leads V1 to V3)



ST segment elevation in posterior chest leads V8 and V9

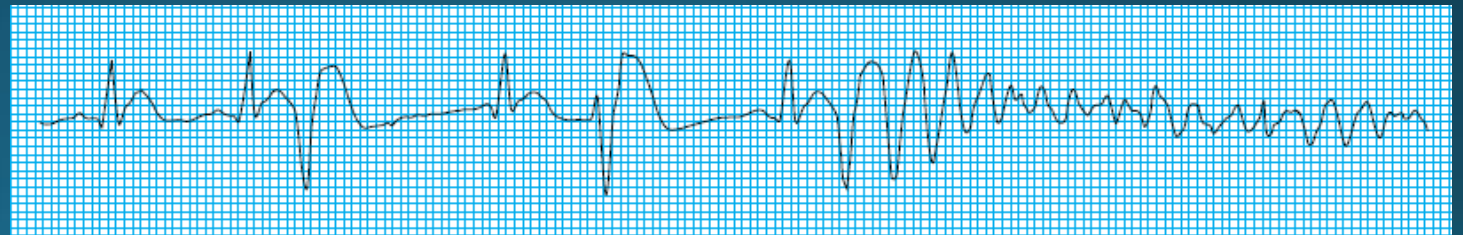
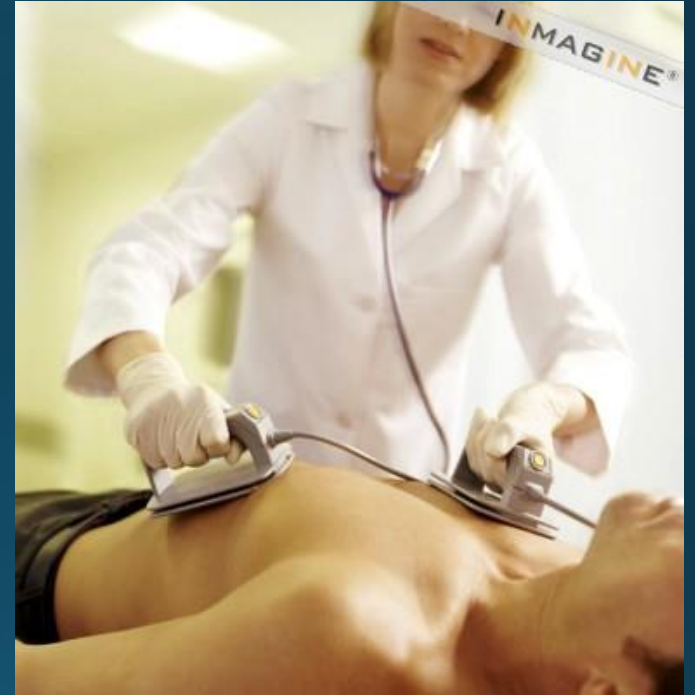
STEMI – “lights and sirens”

- Time critical
- “Time is myocardium”
- Potential for worsening outcomes with time delays
- 12-lead ECG performed in ambulance – if STEMI activate cath lab at hospital in advance to decrease door-to-balloon time



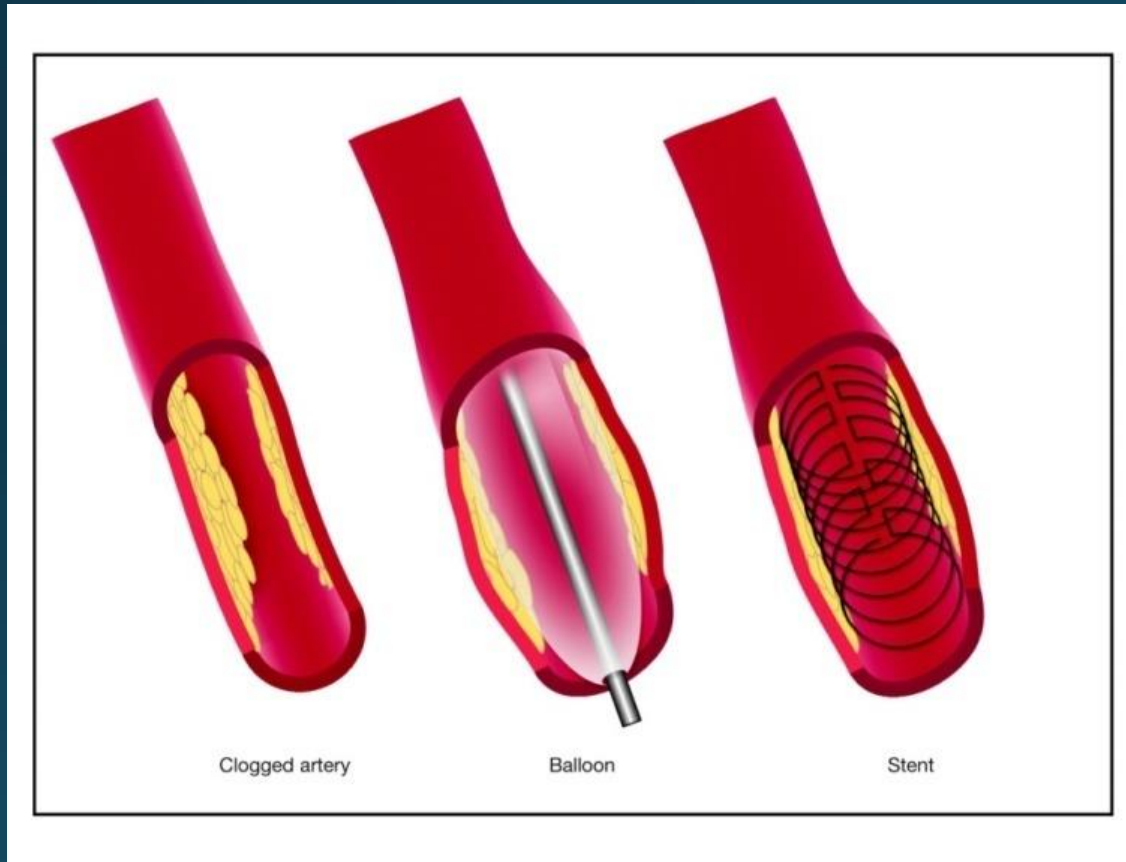
STEMI – Mx while awaiting ambulance/MICA

- Ensure aspirin 300mg given
- Pain relief (GTN/morphine)
- IV access
- Manage arrhythmias
 - Sinus bradycardia/AV block – atropine
 - High risk ECG changes/concerning ventricular arrhythmias – apply defibrillator pads
- Manage acute pulmonary oedema – GTN if SBP > 100mmHg, IV frusemide, IV morphine



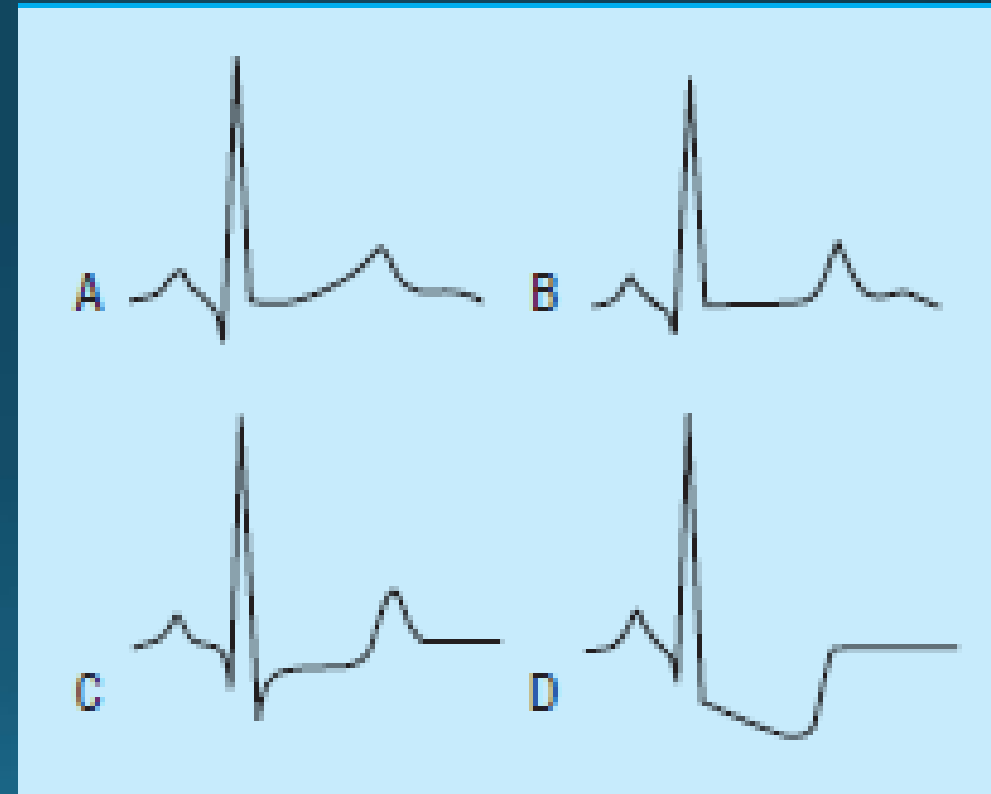
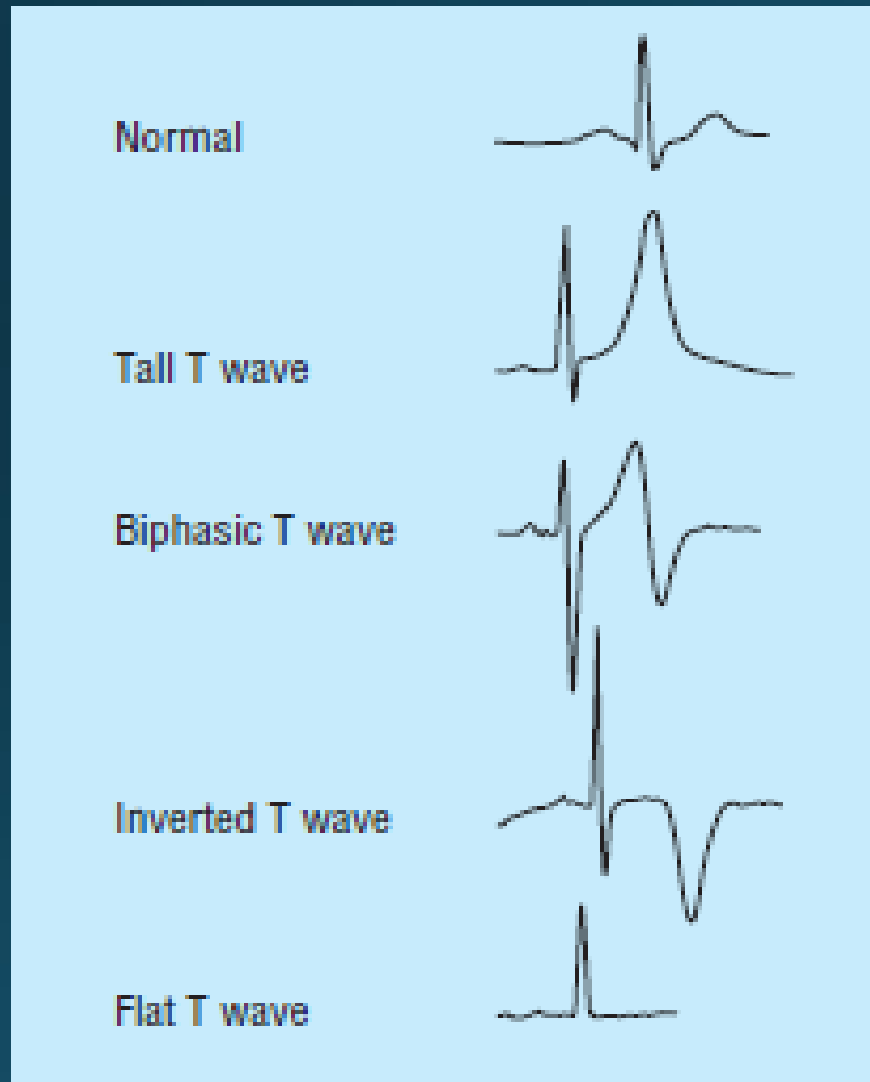
PVCs followed by onset of ventricular fibrillation

Primary PCI vs thrombolysis for STEMI

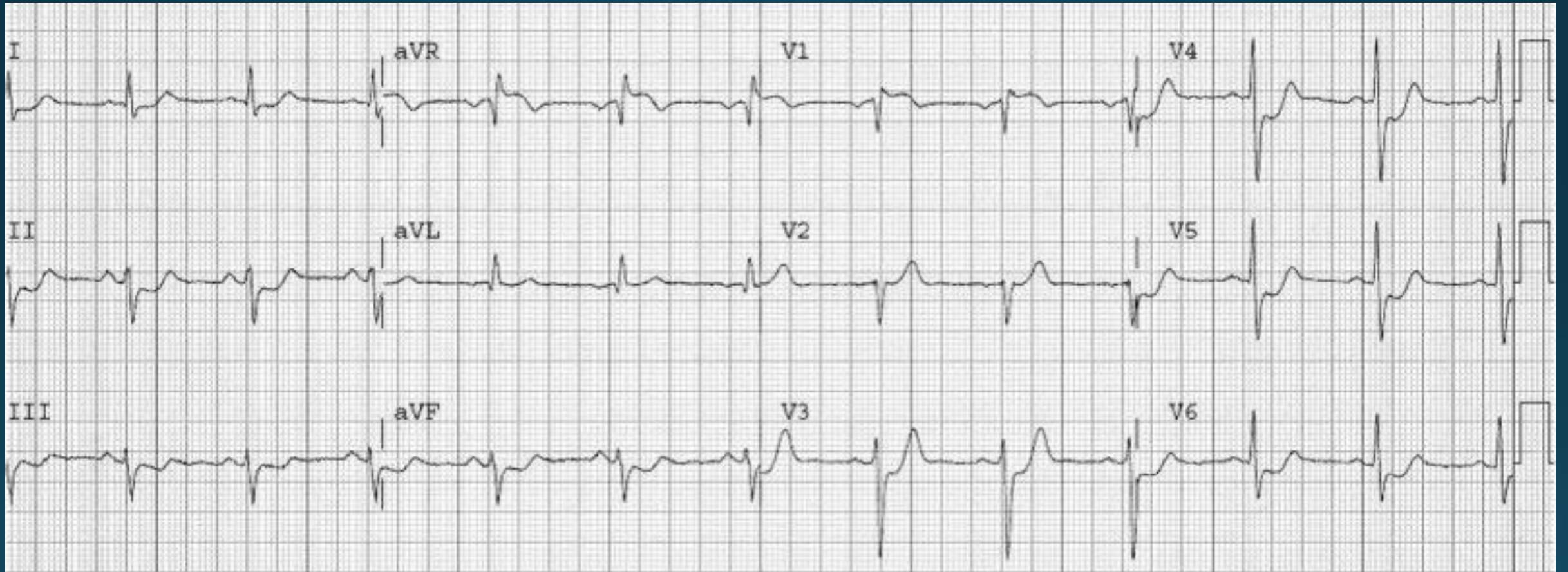


Superior patient outcomes – perform where available

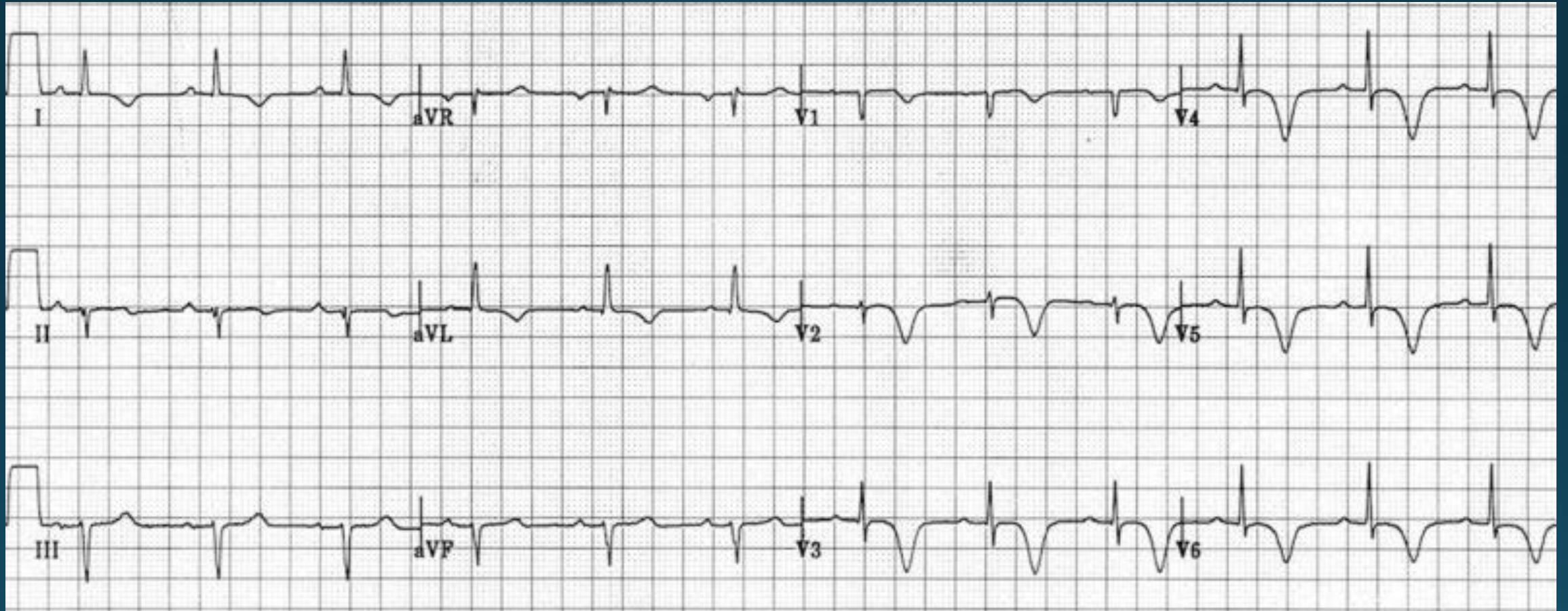
ECG – ischaemic changes (NSTEACS, UA)



High risk ECG – extensive ST depression with ST elevation in aVR

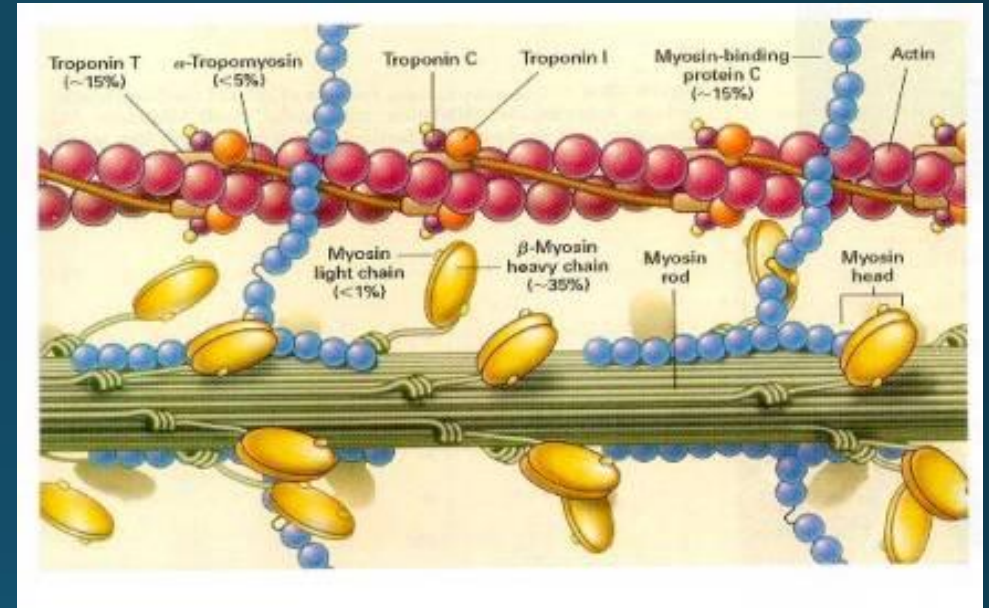


Deep T wave inversion throughout anterolateral leads



Troponin

- Controversial - limited role of troponin testing in primary care
- Guidelines suggest no role if pain within preceding 24hrs
- ? Use if remote history of pain, if clear procedure in place for follow up
- Highly sensitive test for myocardial damage, but may not primarily indicate myocardial ischaemia



Universal classification of MI

- **Type 1: Spontaneous MI**

- Related to atherosclerotic plaque rupture, ulceration, erosion, or dissection with resulting intraluminal thrombus in one or more of the coronary arteries leading to decreased myocardial blood flow or distal platelet emboli with ensuing myocyte necrosis

- **Type 2: MI secondary to an ischaemic imbalance**

- Myocardial injury with necrosis where a condition other than CAD contributes to an imbalance between myocardial oxygen supply and/or demand e.g. coronary endothelial dysfunction, coronary artery spasm, coronary embolism, tachy-/bradyarrhythmias, anaemia, respiratory failure, hypotension, and hypertension with or without LVH

- Type 3 (MI resulting in death), Type 4a (MI related to PCI), Type 4b (MI related to stent thrombosis), Type 5 (MI related to CABG)

Causes of troponin elevation

- Heart failure – acute/chronic
- Tachy/bradyarrhythmias
- Stress cardiomyopathy (Takotsubo)
- Cardiac contusion, surgery, frequent defibrillator shocks
- Aortic valve disease
- Hypertrophic cardiomyopathy
- Infiltrative cardiac disease
- Myocarditis
- Coronary vasculitis
- Rhabdomyolysis with cardiac injury
- **Aortic dissection**
- **Pulmonary embolism, severe pulm HT**
- **Critical illness, esp respiratory failure/sepsis**
- **Hypoxia**
- **Stroke/subarachnoid haemorrhage**
- Renal failure
- Drug toxicity eg anthracyclines
- Burns, esp 30% BSA
- Extreme exertion
- Cross-reacting heterophile antibodies (false positives)

During hospital admission for ACS

- Emergent PCI for STEMI (or thrombolysis in regional areas without access to PCI service)
- Early angiography for NSTEMI/UA
- Monitoring in coronary care
- Medical therapy/RF assessment/smoking cessation
- Echocardiography
- Education
- Referral for rehabilitation



Intermediate risk patients discharged from ED

- Approximately 4% patients with normal Trop/ECG have significant underlying coronary artery disease
- Referred for further risk stratification with
 - Stress echocardiography
 - Myocardial perfusion scan
 - CT coronary angiography
 - (exercise ECG)

? Need for angiography



ACS – update on pharmacotherapy (for further reading)

- All ACS – dual antiplatelet therapy for 12 months (preferably aspirin and ticagrelor)
- Individualised duration of DAPT depending on ischaemic vs bleeding risk
- Statin with target LDL < 1.8 (? Need for ezetimibe/evolocumab)
- Vasodilatory beta blockers if LV dysfunction
- ACE inhibitors

Q & A