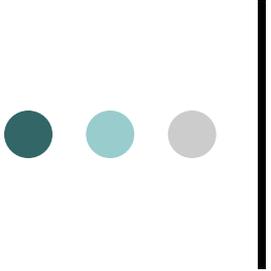


Chest Pain

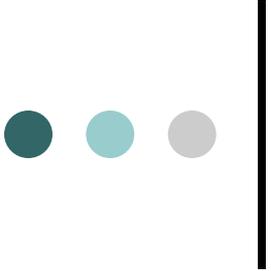
Dr Bhakti Hansoti

October 2008



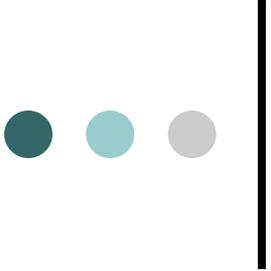
S.O.C.R.A.T.E.S

- S = Site
- O = Onset
- C = Character
- R = Radiation
- A = Association
- T = Timing
- E = Exacerbating / Relieving factors
- S = Severity



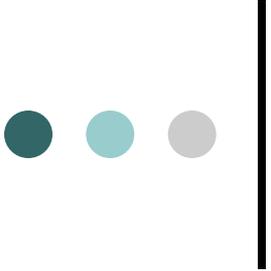
Differentials

- Retrosternal:
 - Angina Pectoris: Crushing pain on exercise, relieved by rest, radiating to jaw or arms
 - Myocardial infarction: more severe, occurs at rest, lasts longer
 - Pericarditis: Sharp pain aggravated by movement, respiration and change in posture
 - Aortic dissection: Severe tearing chest pain radiating to the back
 - Reflux Oesophagitis: Occurs at night and when bending/lying down, may radiate to the neck



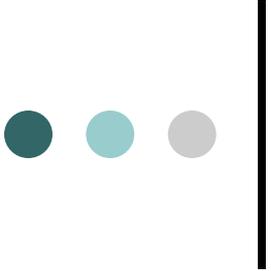
Differentials

- Other sites: Usually Lateral
 - Pulmonary infarct, Pneumonia, Pneumothorax
 - -> pleuritic pain, sharp well-localized, aggravated by coughing, inspiration and movement
 - Fractured rib, costochondritis
 - -> musculoskeletal pain is usually sharp, well-localized pain with a tender area on palpation



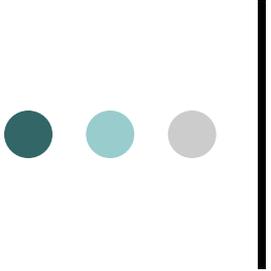
Myocardial Infarction

- Most common cause of death in developed countries
- Secondary to rupture of an atherosclerotic plaque -> development of thrombosis and total occlusion of the artery



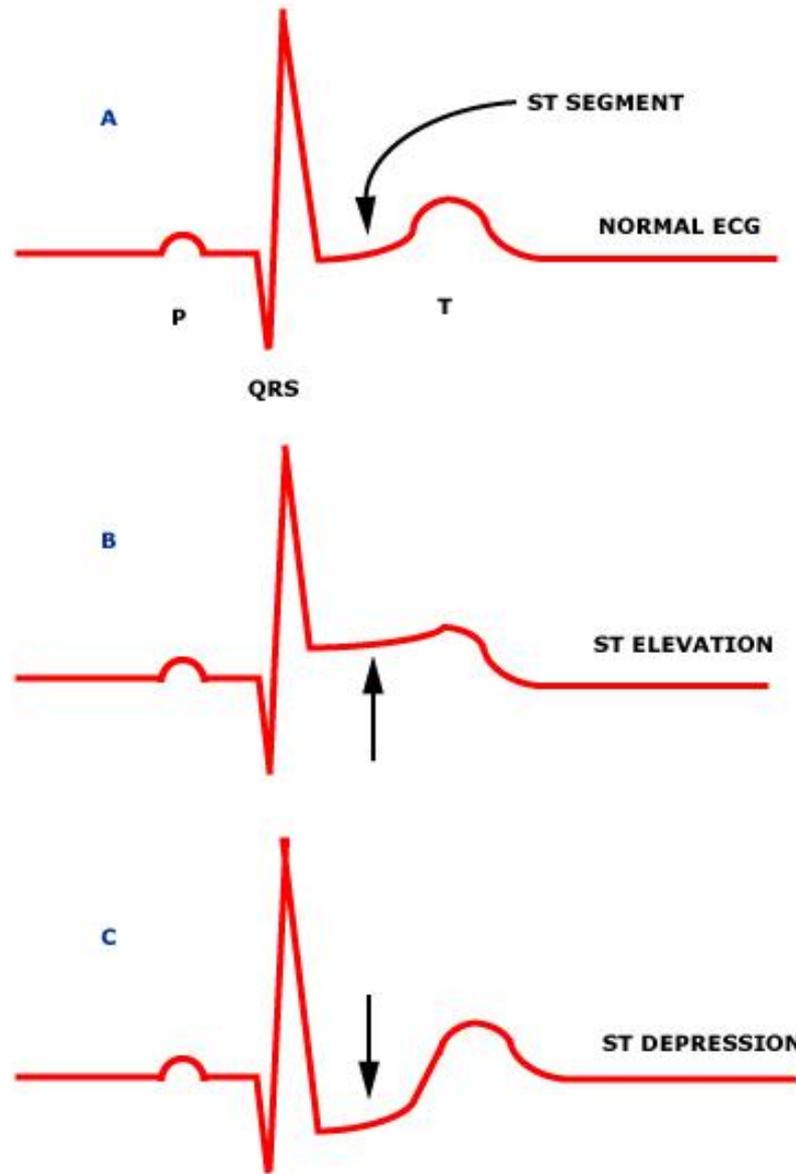
MI – clinical features

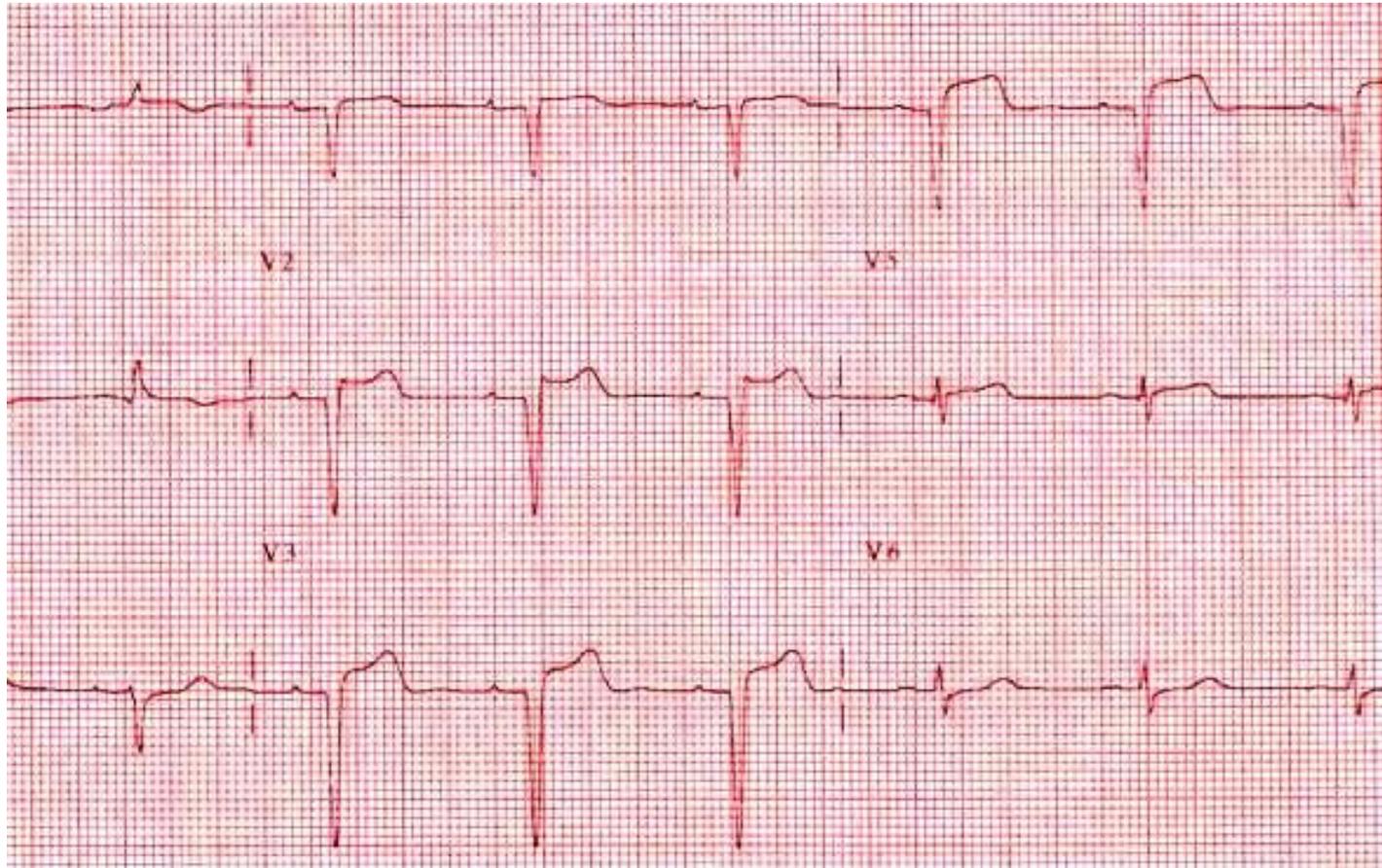
- Chest pain at rest, severe and lasts for hours
- Associated with sweating, nausea, vomiting, breathlessness and restlessness
- Patient appears pale, sweaty and grey
- NOTE: 20% silent MI!!!!!!!!!!!!!!!!!!!!!!

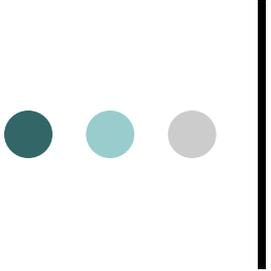


MI - Investigations

- Diagnosis from clinical history
- ECG :
 - ST elevation ($>1\text{mm}$ in two or more contiguous leads) followed by t-wave flattening/inversion
 - Pathological Q-waves are broad ($>1\text{mm}$) and deep ($>2\text{mm}$), in full thickness MI
 - Infarcted muscle is electrically silent so that the recording leads 'look through' the infarcted area



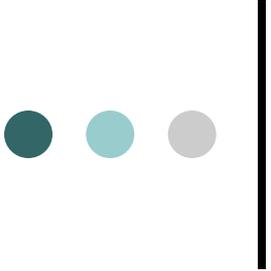




MI - investigations

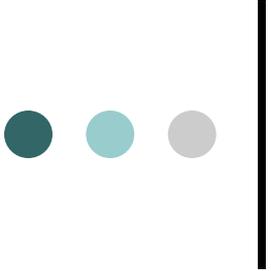
- Cardiac markers

- Troponin T & I are regulatory proteins, highly specific, peak at 12 hours
- Creatine Kinase (CK) also produced by damaged skeletal muscle and brain. MB isoenzyme is specific for heart muscle damage, size of rise is proportional to infarct size
- Aspartate aminotransferase (AST) and lactic dehydrogenase (LDH), rarely used remains elevated for 10 days



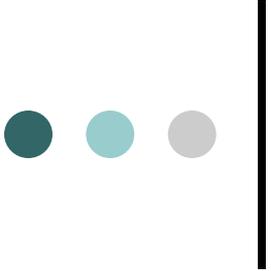
MI - management

1. PCI door to needle time 30mins!
2. Thrombolytic therapy if ST-elevation MI, reperfusion in 50-70% -> Streptokinase or recomb. TPa
3. M.O.N.A
4. GTN infusion 2-10mg/h titrated to response, maintain SBP >90mmHg
5. If blood glucose >11mmol/L, aim for BM 7-10mm
6. ol/L



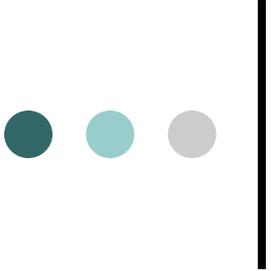
M.O.N.A

- M = Morphine
- O = Oxygen
- N = Nitrates
- A = Asprin



MI - Complications

- Cardiac failure
- Thromboembolism
- Pericarditis



MI - Prognosis

- Depends on age, size of infarct, previous MI
- 50% of patients die in the acute event
- 10% die in hospital during acute admission
- 10% die in the next two years