CORONARY HEART DISEASE

Catherine Heywood
Hospital Teacher Practitioner

Thromboembolic Disease

Arterial Thrombosis

- Acute myocardial infarction (AMI)
- Transient ischaemic attacks (TIA's)
- Cerebral vascular infarcts /accidents (CVA's)

Venous Thrombosis

- Deep vein thrombosis (DVT)
- Pulmonary embolism (PE)

Inherited/Acquired

- Thrombophilia

Arterial Thrombosis

- Occurs as a result of rupture of atherosclerotic plaques
- Platelet deposition & vessel occlusion
- "White thrombi"

Venous Thrombosis

- Often occurs in normal vessels
- Majority deep vein of leg
- "Red thrombi"

Objectives:

- Definition of Coronary Heat Disease (CHD)
- Epidemiology, pathophysiology & aetiology
- Stable Angina & Acute Coronary Syndrome (STEMI/NSTEMI/Unstable Angina)
  - Clinical feature
  - Diagnosis
  - Management

What is CHD?

"CHD is a condition in which the vascular supply to the heart is impeded by atheroma, thrombosis or spasm"

(Walker & Whittlesea[(1)])

- Inadequate blood supply $\Rightarrow \downarrow O_2$ supply to the heart $\Rightarrow$ ischaemic chest pain (IHD= Ischaemic Heart Disease) & depending on extent, can cause:
    - Stable angina
    - Acute Coronary Syndrome (ACS) (MI + Unstable angina)
    - Sudden death

Epidemiology

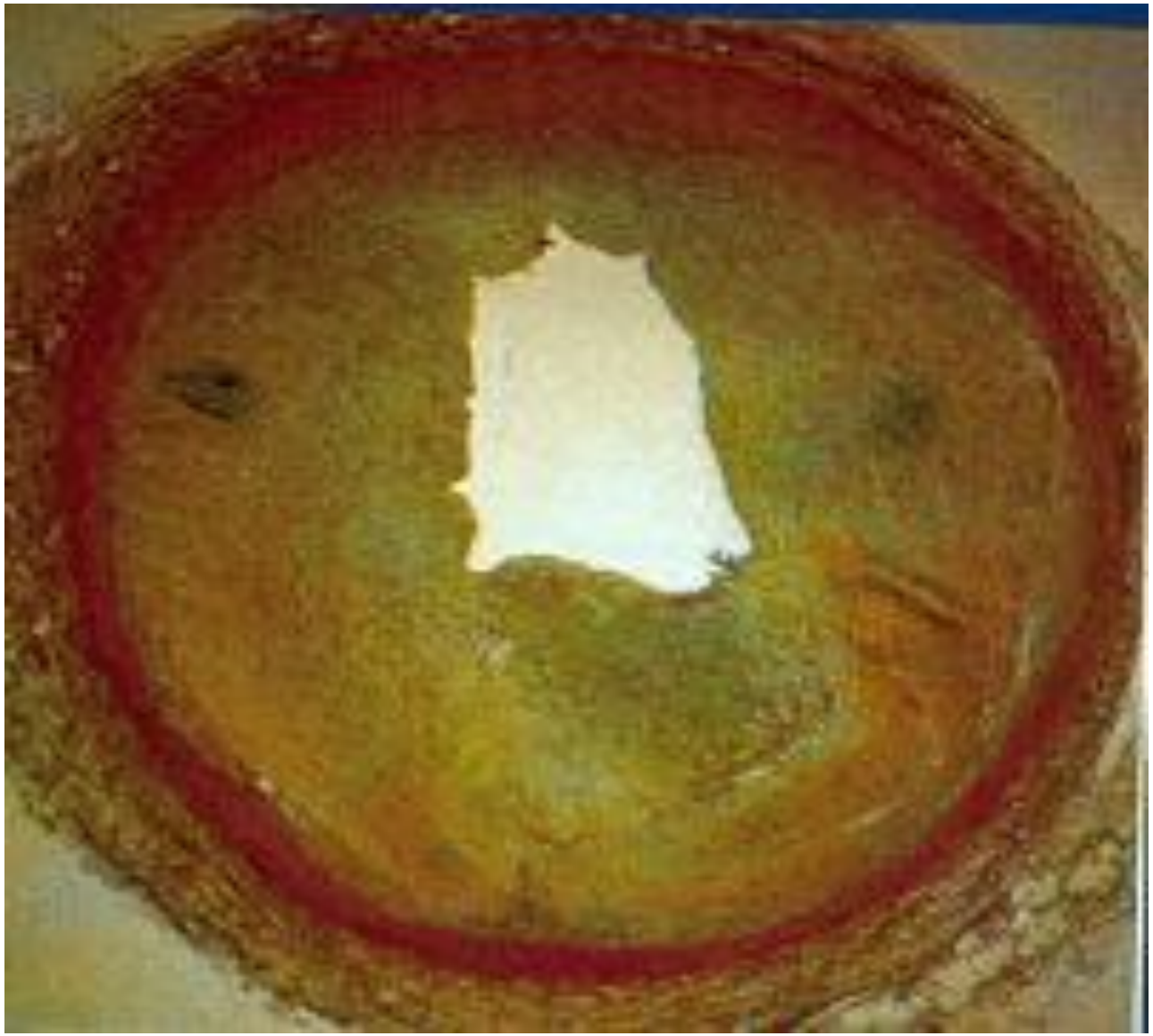
- About 4% UK population have symptoms of CHD
- More common in males (until women reach menopause) & ↑ with age
- About 124,000 AMI / year of which about 15-20% die
- in UK, S.Asians have ~45% ↑ risk of death & Black African Caribbean have ~ 50% ↓ risk

Aetiology

- **<u>Atherosclerosis</u>**

- complex inflammatory process initiated due to ‘injury’ or dysfunction of the endothelium
- $\Rightarrow \uparrow$ permeability to oxidised lipoproteins $\Rightarrow$ macrophages $\Rightarrow$ lipid-laden foam cells
- $\Rightarrow$ “fatty-streaks”

- Smooth muscle cells secrete collagen, proteoglycans, elastin & glycoproteins
- $\Rightarrow$ fibrous cap $\Rightarrow$ plaque
- $\Rightarrow$ narrowing of blood vessels & $\downarrow$ blood flow
- ($\Rightarrow$ rupture of plaque $\Rightarrow$ clot)



Risk factors

- **Main:**
  - Age
  - Gender
  - FH
  - Smoking
  - Diet
  - Obesity
  - HT
  - **Hyperlipidaemia**
- **Others:**
  - DM
  - Sedentary lifestyle
  - Ethnicity
  - Alcohol
  - Stress

Assessment of CV Risk

- For primary prevention
- Treat if >10% (NICE 2014)(2)
- If already have CVD then assessment not applicable ⇒ assume high risk and treat
- CV Risk assessment:
  - QRISK3(www.qrisk.org)

Pathophysiology

- Imbalance between O_2 demand and supply
- O_2 demand
  - HR, contractility & systolic wall tension
- O_2 supply
  - coronary blood flow & O_2 carrying capacity of blood

STABLE ANGINA

Stable angina

- Narrowing of coronary arteries due to atheromatous plaques
- $\Rightarrow$ chest pain typically provoked by exercise, stress, heavy meals or extremes of temperature
- $\Rightarrow$ relieved by rest or s/l GTN

- **Stable angina**
  - "demand ischaemia"
  - narrowed coronary arteries unable to meet ↑ O_2 demand during exercise, stress etc

Clinical symptoms & diagnosis:

- Central crushing chest pain
- May radiate to jaw, neck, back or arms
- “Constricting”, “choking”, “heavy weight”, or “stabbing”, “burning” or “like a knife”
- Induced by exercise etc & relieved by rest/ GTN
- Lack of ECG / cardiac enzymes changes

Management:

- **Symptom control**[NICE, 2011(3)]
  - S/L GTN for acute angina
  - Antianginals:
    - Ist-Line: Beta-blockers, Calcium channel blockers
    - Add-on: Long acting nitrate, ivabradine, ranolazine or nicorandil

- **Secondary prevention**
  - Lifestyle changes (Smoking/weight loss/diet/exercise etc)
  - Antiplatelet (aspirin)
  - Statins

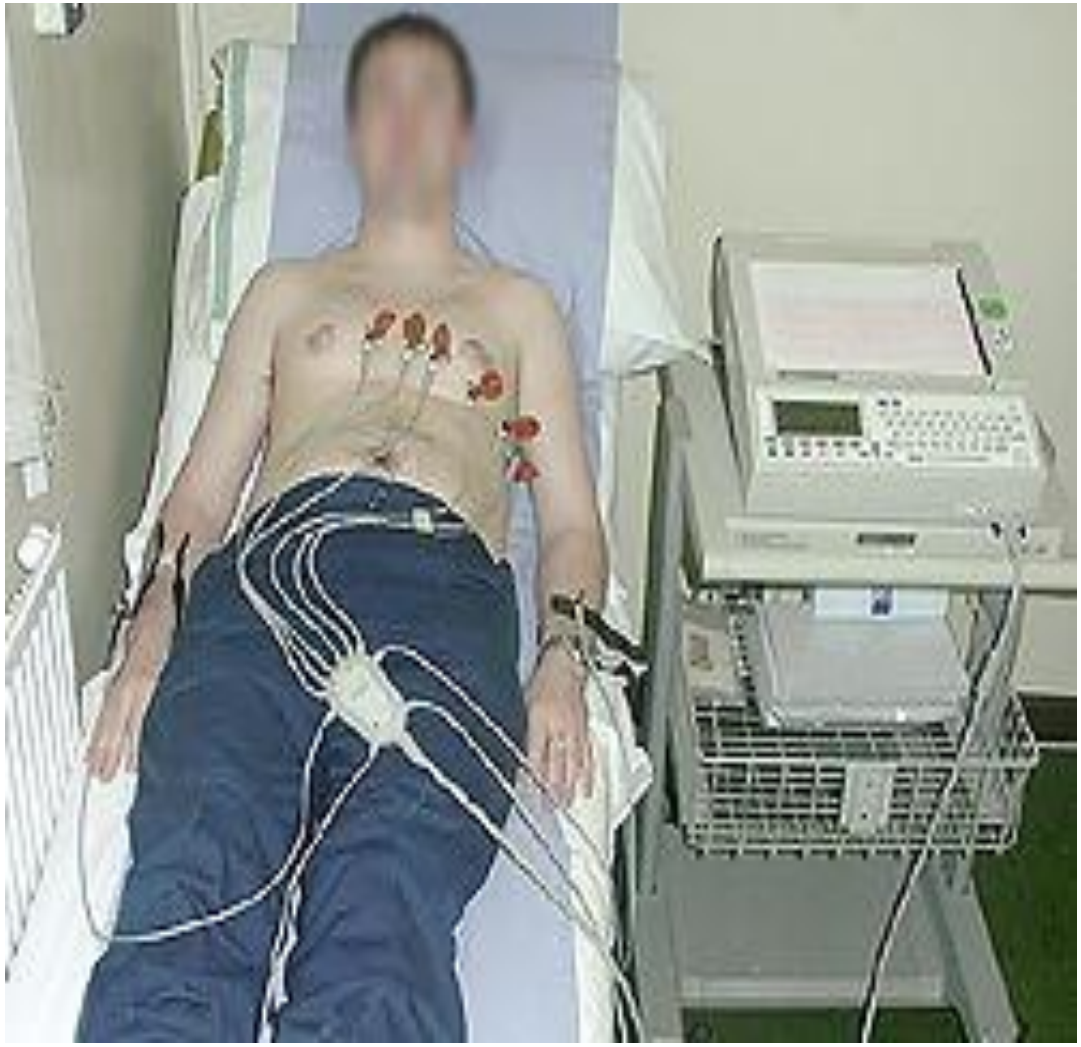
ACUTE CORONARY SYNDROME

- **Acute Coronary Syndrome**:
  - Myocardial Infarction (MI)
    - ST elevated MI (STEMI)
    - Non-ST elevated MI (NSTEMI)
  - Unstable Angina (Troponin Positive ACS)

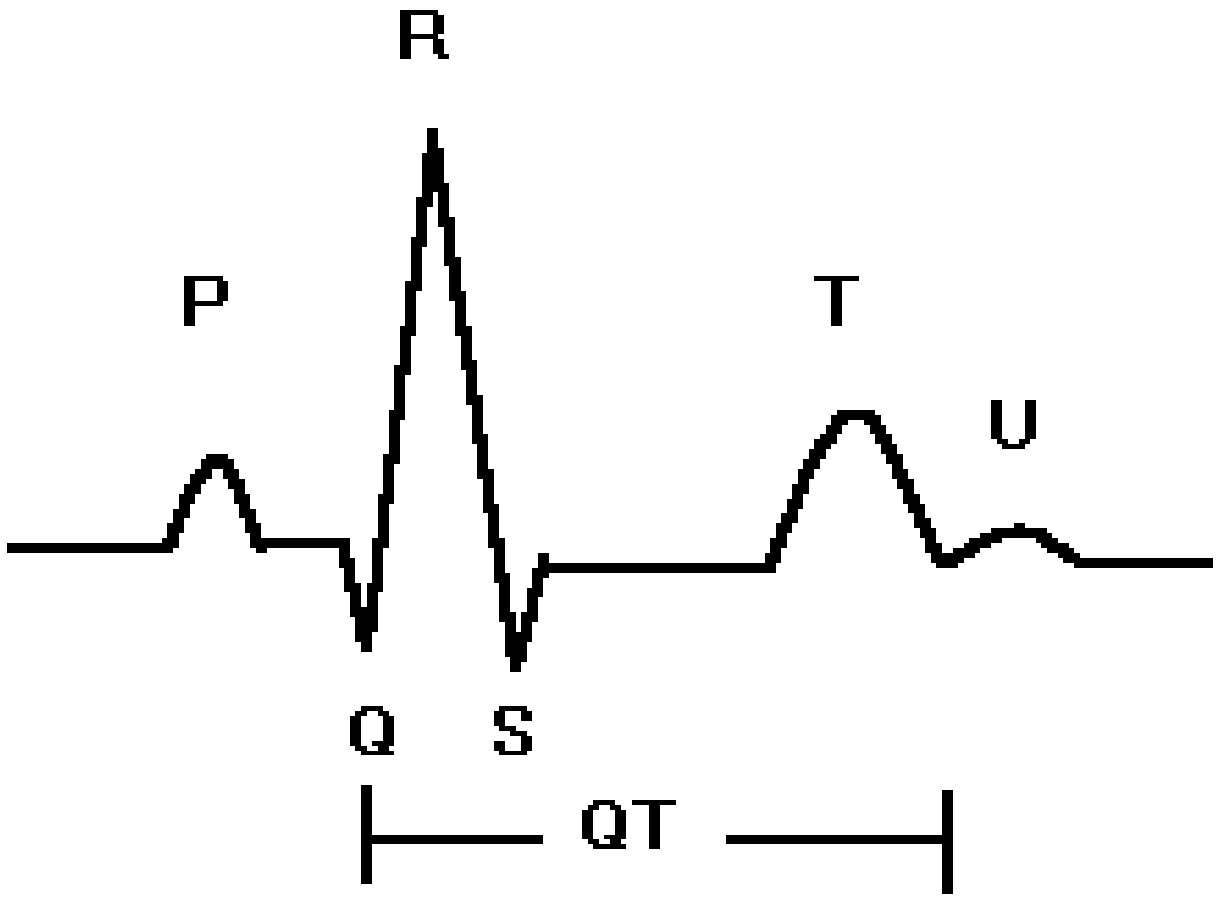
Differential diagnosis:

- History of ischaemic chest pain
- ECG changes
- ↑ cardiac enzymes

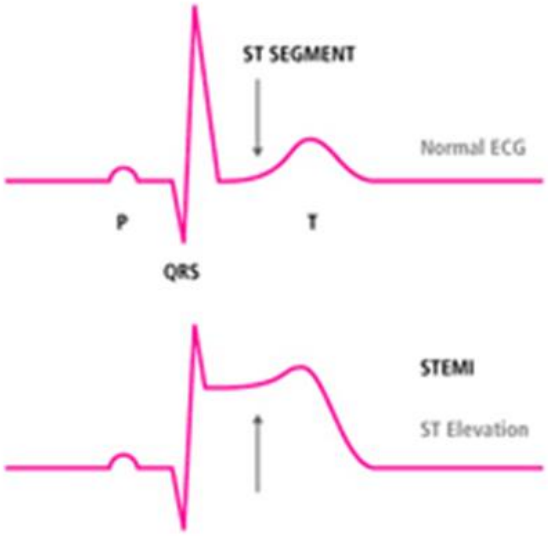
12 lead ECG:

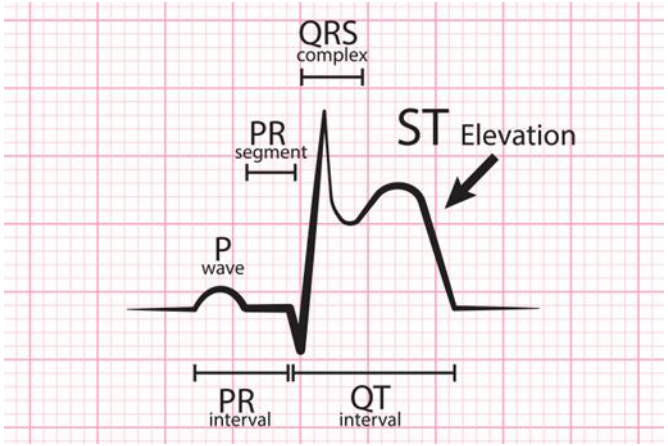


Normal ECG:



ECG changes in STEMI:





- ST elevation is **NOT** seen in:
  - NSTEMI
  - Unstable angina

- Sometimes other ECG changes aid differential diagnosis:

- STEMI:
  - Left Bundle Branch Block (LBBB)

- NSTEMI:
  - T wave inversion/ ST segment depression
  - Q wave changes

Cardiac enzymes:

- **Troponin T & Troponin I (Trop I):**
  - highly specific
  - released after 2-4 hrs, peaks at 12 hrs & can persist up to 7 days
  - Measured on admission
  - Standard Troponin assays – repeated after 10-12 hrs
  - High sensitivity Troponin assays – repeated after 3 hrs (enables early rule out of NSTEMI)(4)

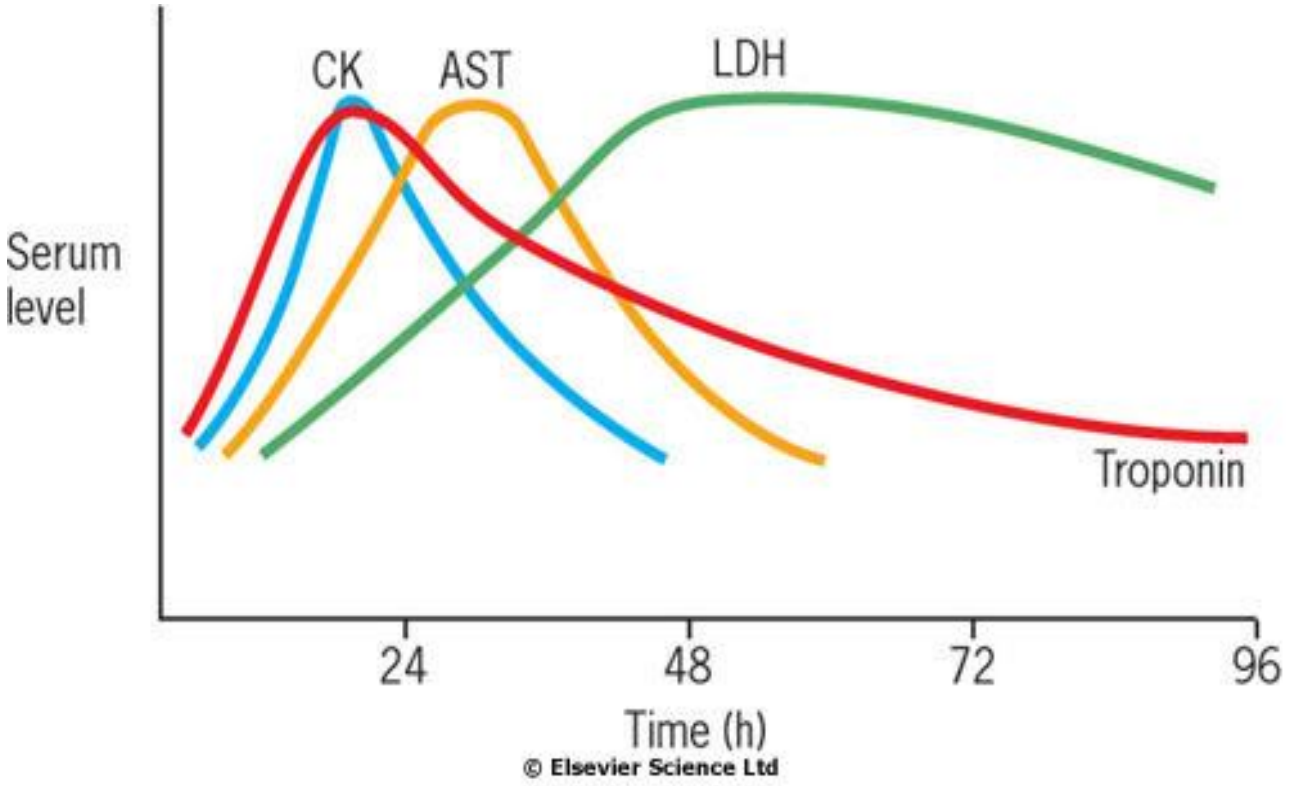
- STEMI/NSTEMI => ↑ Troponin >99th percentile cut-off/upper reference limit (varies according to specific assay used)
- Unstable angina => some change in Troponin level but does not meet criteria for MI
- <0.4ng/ml => ACS unlikely
- **Size of ↑ ≈ size of infarct**

- Other causes of increased Troponin levels:
  - Pulmonary embolism
  - Heart failure
  - Myocarditis
  - CKD
  - Sepsis

Other enzymes which rise in STEMI/NSTEMI:

- **Creatine Kinase (CK):**
  - peaks within 24 hrs
  - normal within 48hrs
  - also in skeletal muscle & brain
  - **CK-MB -** cardiac specific isoform

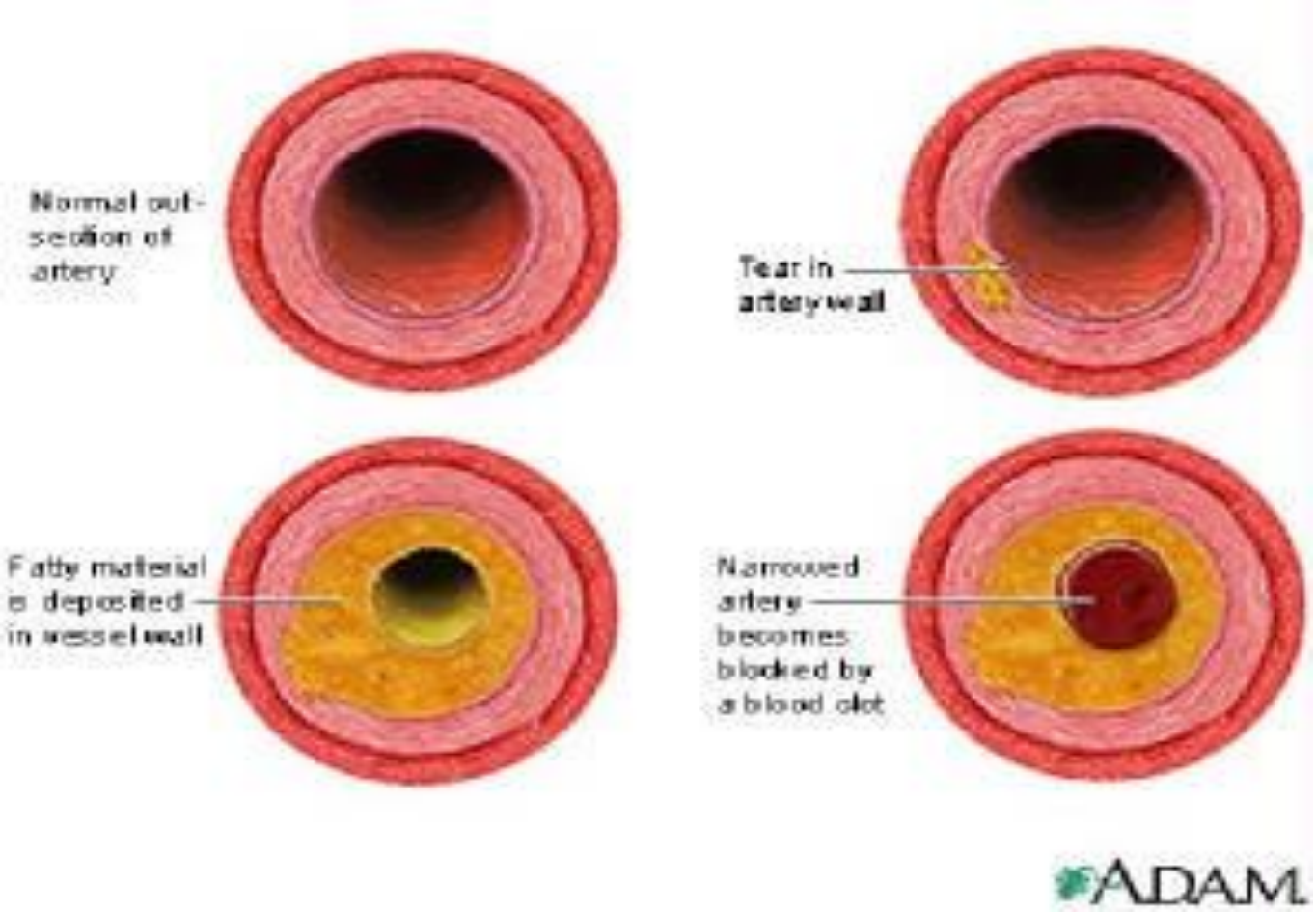
- **Aspartate Transaminase (AST) and Lactate dehydrogenase (LDH):**
  - non-specific
  - released from other parts of body
  - not used routinely
  - LDH peaks at 3-4 days and remains ↑ for up to 10 days ⇒ can be useful in late presentations


CK
AST
LDH
Serum level
Troponin
24
48
72
96
Time (h)
© Elsevier Science Ltd

Myocardial Infarction

- Thrombosis forms at site of rupture of atheromatous plaque
- $\Rightarrow$ severe & prolonged ischaemia
- $\Rightarrow$ death of cardiac muscle cells ($\Rightarrow$ release of enzymes from cells)
- STEMI $\Rightarrow$ damage to full thickness of cardiac muscle
- NSTEMI $\Rightarrow$ damage to partial thickness of cardiac muscle

- **Myocardial infarction & Unstable angina (UA)**
  - "supply ischaemia" (vs "demand ischaemia" of stable angina)
  - ↓ coronary blood flow & ↓ O_2 supply due to thrombus formation
  - ⇒ partial blockage (UA) & complete blockage (STEMI/NSTEMI)
  - Thrombus forms as a result of plaque rupture ⇒ activation & aggregation of platelets


Normal cut-section of artery
Tear in artery wall
Fatty material is deposited in vessel wall
Narrowed artery becomes blocked by a blood clot
ADAM.

STEMI/NSTEMI clinical features:

- Severe chest pain, sudden onset, often at rest and constant
- 20% of AMI $\Rightarrow$ no symptoms
  - **"Silent" MI's** - more common in elderly & DM

Additional symptoms:

- Sweating
- Breathlessness
- Nausea & vomiting
- Restlessness
- Pale
- Grey

Unstable Angina clinical features:

- Sudden deterioration in angina symptoms
- Often at rest, not relieved by rest or s/l GTN

MANAGEMENT OF ACUTE CORONARY SYNDROME(5)

Management of ACS:

- Need to differentially diagnose different types of ACS as acute treatment is different for **STEMI**

STEMI

Management of STEMI:

- **Acute/immediate care**
  - pain relief, thrombolysis/reperfusion & minimisation of infarct size
- **Management of complications**
  - Eg LVF, arrythmias
- **Secondary prevention**
  - drug therapy & lifestyle changes

Immediate:

- **Oxygen** (if indicated)
  - relieves ischaemia
- **Diamorphine**
  - pain relief
  - anxiolytic
  - vasodilatation
  - + antiemetic (eg cyclizine, metoclopramide)
- **Aspirin**
  - 300mg stat ASAP
- **Clopidogrel (or Ticagrelor* or Prasugrel**)**
- **300mg stat (or 180mg* or 60mg**)**

PRIMARY PERCUTANEOUS CORONARY INTERVENTION (PPCI)

See later

OR …..
(if PPCI not available)

- **Thrombolysis:**
  - Reperfusion
  - Eg: Streptokinase, alteplase, tenecteplase, reteplase
  - “call to needle time” 1 hour & “door to needle time” 30 mins
  - C/I: CVA, recent surgery, peptic ulcer, uncontrolled HT, > 6 hrs
  - S/E: haemorrhage, stroke, reperfusion arrythmias, allergy [SK]
- **Heparin** (For first 48 hrs after thrombolysis)

Complications

- Arrhythmias
- Heart failure
- Treat if they occur

► See later screencasts/workshops!

Secondary prevention:

- **Drug therapy:**
  - Antiplatelets (aspirin + clopidogrel/tiacgrelor/prasugrel*)
    - Dual antiplatelet therapy (DAPT) for 12 months, aspirin for life
  - Beta-blocker (review at 12 months – continue if also heart failure)
  - ACEI
  - Statin – Atorvastatin 80mg od
- **Lifestyle changes:** Smoking/weight loss/diet/exercise etc

NSTEMI & UNSTABLE ANGINA

Management of NSTEMI/Unstable Angina:

- **Immediate:**
  - **Oxygen**
  - **Diamorphine**
  - **Aspirin**
  - **Clopidogrel (or Ticagrelor or Prasugrel)**

- **Also:**
  - **Fondaparinux – until stable**

NO THROMBOLYSIS

OR

PPCI

- Further treatment/investigations depends on prediction of 6-mth risk of mortality/further CV events
- Use GRACE (Global Registry of Acute Cardiac Events) Scoring system
- If Intermediate/higher risk >3% => for angio and PCI (see below)
- If low risk <3% => conservative management (no angio/PCI)

Secondary prevention (same as STEMI):

- **Drug therapy:**
  - Antiplatelets (aspirin + clopidogrel/ticagrelor/prasugrel*)
    - Dual antiplatelet therapy (DAPT) for 12 months, aspirin for life
  - Beta-blocker (review at 12 months – continue if also heart failure)
  - ACEI
  - Statin – Atorvastatin 80mg od
- **Lifestyle changes: as before**

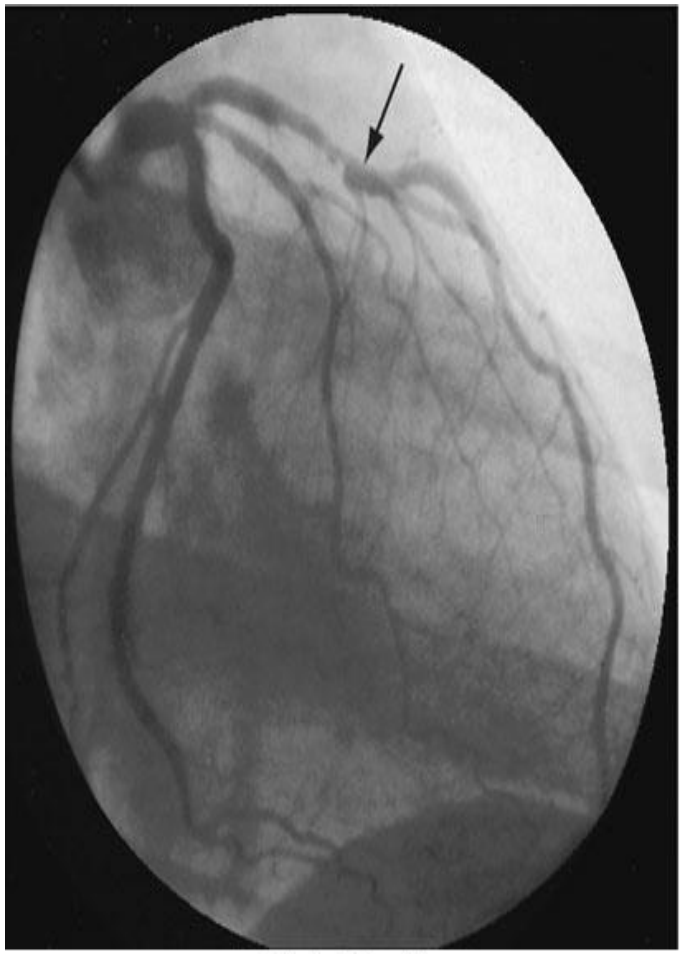
INVESTIGATIONS

&

INTERVENTIONS

Investigations:

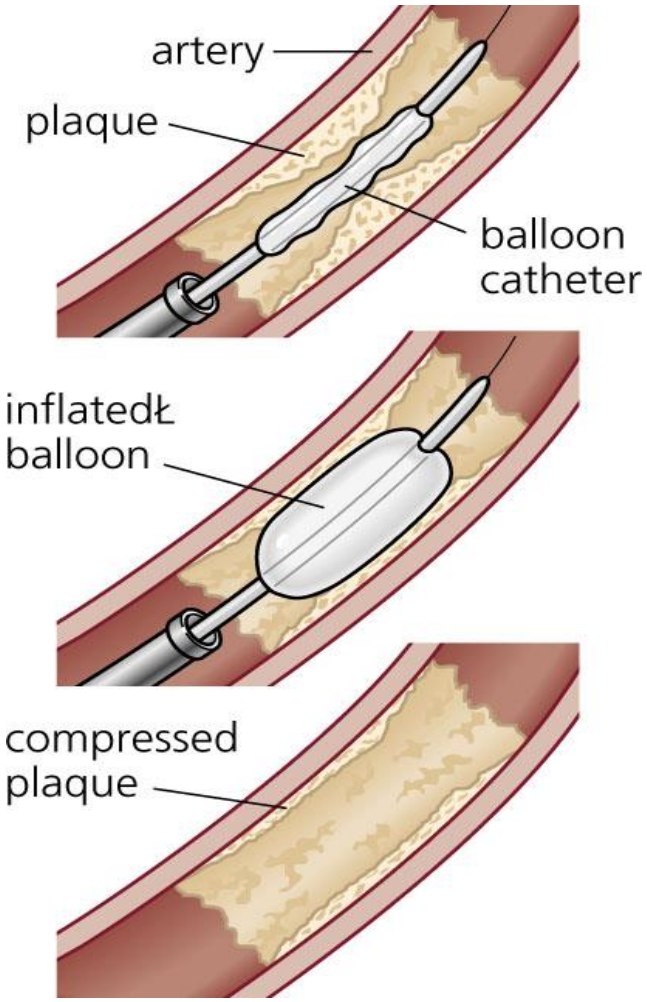
- **Angiography:**
  - thin radiopaque tube (catheter) introduced into coronary circulation
  - X-ray contrast material injected into coronary artery via catheter
  - Allows observation of severity of narrowing (stenosis) due to atherosclerotic plaque)



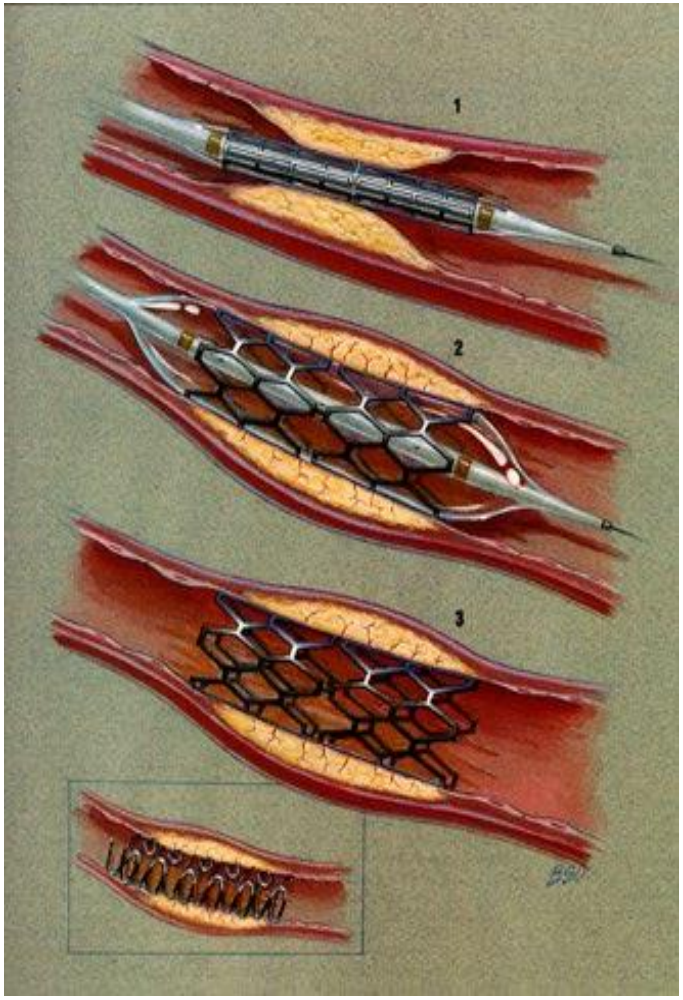
Surgical Interventions:

- **Percutaneous Coronary Intervention (PCI):**
  - Angioplasty
  - Stenting
- **Coronary Artery By-pass Graft (CABG)**

- **Angioplasty:**
  - balloon mounted on tip of very thin catheter inserted through obstruction and inflated
  - restenosis common



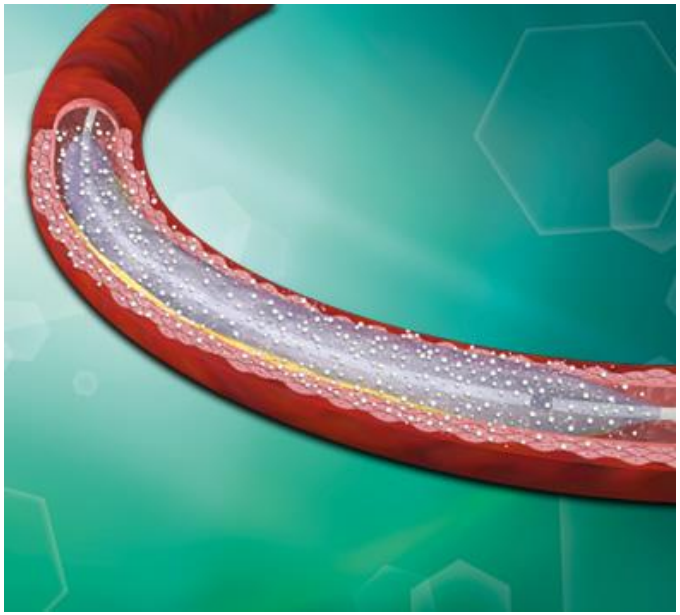
- **Stenting:**
  - wire mesh inserted with balloon to keep stenosis open
- Bare metal stent (BMS) or drug eluting stent (DES)
  - Elutes anti-proliferative drug (E.g. Tacrolimus, Paclitaxel)
  - Stops overgrowth of stent by wall tissue



- Both angioplasty & stents can damage vessel wall $\Rightarrow \uparrow$ clotting (in-stent thrombosis)

- $\Rightarrow$ Long-term aspirin & 12 months Clopidogrel/Ticagrelor/Prasugrel

- **Drug eluting balloon:**
  - Balloon covered with anti-proliferative drug (E.g. Paclitaxel)
  - Released into vessel wall during inflation
  - Lipophilic ► absorbed into vessel wall
  - Reduces restenosis
  - No problem of in-stent thrombosis

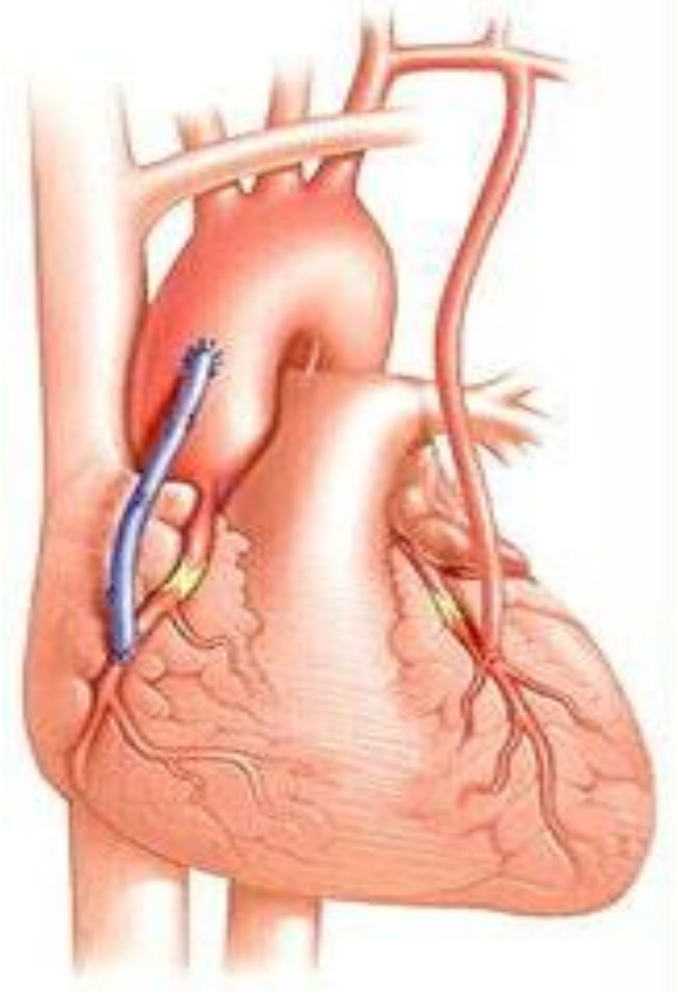


Primary PCI:

- Alternative treatment for STEMI instead of using thrombolysis
- Better outcomes and less people contraindicated compared to thrombolysis
- Patient taken straight to angio suite for angio then angioplasty (with or without stenting)
- Clot is removed during procedure
- “Call to balloon” time 120min
- “Door to balloon” time 30min

Surgical Interventions:

- **Coronary artery by-pass grafting (CABG):**
  - veins grafted to by-pass stenosis in coronary artery



References

- (1) Clinical Pharmacy & Therapeutics, 5th Edn (Walker & Whittlesea)
- (2) Lipid modification, NICE 2014
- (3) Stable Angina, NICE 2011
- (4) High- sensitivity troponin tests for the early rule out of NSTEMI, NICE 2020
- (5) Acute coronary syndromes, NICE 2020