Myocardial Infarction

Cardiac Cycle + ECG →

- cardiac impulse starts in SAN. As it crosses atria it gives rise to the P wave. Then onto AVN which is not seen on ECG as amount of conducting tissue is small
- Then we have electrical silence (PR interval) bc L+R V contract + give rise to QRS complex. Then we have more silence as heart repolarises ST segment.

ST segment is crucial part of ECG for interpreting myocardial ischaemia

Sensation of MI →

① Silent Radiation → Diffuse, ant. chest, L/forearm, neck.
② Character → Tight, pressure, weight, constriction, dull
③ Triggers (Angina) → exercise, cold, meals, psychostress
④ Relief → rest, GTN
⑤ Duration → <30m is angina, >30m is infarction

Coronary Thrombus →

Atherosclerotic Plaque Rupture → ruptures under stress of blood flow and white platelets (Thrombus) begin to stick to exposed collagen + cholesterol. Is platelet aggregation is key to this event. This then triggers the classic clotting cascade. RBC get trapped in fibrin web.

Atherosclerotic Plaque →

"Soft - hard"

① In intima layer of vessel we have a mix of atherosclerotic. Soft is cholesterol (result from frustrated phagocytosis by macrophages). These macrophages secrete enzymes which break down proteins which degrade the matrix in the fibrous cap. Eventually it is degraded enough to open, platelets think it is an open wound + be in the clotting cascade.

Then a Thrombus forms which if becomes free can form a blood clot.

To stop this → ① STATINS are most effective at preventing this.
② Anti-platelet drugs can be added to aspirin to ↑ the anti-platelet effect.

Main side effect of Thrombolytic drugs is bleeding (commonly into GIT or brain).

Mechanical reperfusion is better than pharmacological. It is faster, higher % reperfusion, less stroke + bleeds, ↓ mortality.

CVD Risk Factors →

Non modifiable → Fitz, age, sex
Modifiable → Smoking, high cholest + BP, Diabetes, obesity / diet / Exercise, Alcohol.

Contraction Cycle →

Troponin is used to diagnose MI. When myocytes exploded, 2 Troponins are released → Troponin T, Troponin I (unique to CM)

I is the inhibitory part which prevents it from interacting w/ actin.

Shortness of Breath → vascular problem.

When a coronary artery (CA) is blocked the heart stops it's next energy intensive form which is contraction. So LA P ↑ so heart TCO though Frank-Starling relationship. But as heart becomes more damaged, the LA P / LV EDP ↓ so CO cannot ↑ to match inflow so LAP becomes even higher as RA is pumping blood in as normal. Fluid is no longer reabsorbed on venous side of arteriole.

So in right heart failure → ankle edema
Left V failure → pulm edema so thickening of alveolar + capillary. So diffusion distance ↓. CO_2 is still quite sol. ∴ is breathed out but cannot intake as much O_2 now.

Chest Pain → People often feel that they vaguely have a deep sensation they can't localise, often interpreted as 'indigestion'.

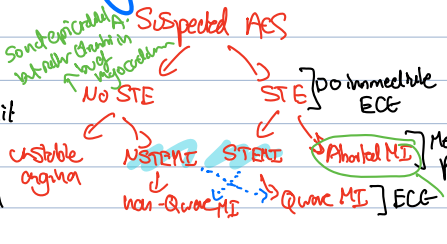
- Sensation of Angina is the same as acute MI
- The pathology is that ADP is not being reabsorbed to ATP and so adenosine is accumulating in the myocardium. Atherosclerotic plaque covered + fibrous cap is safe. Thrombolytic material underneath is protected. A stable plaque can cause angina
- If resting flow is 1, you need more than a 90% stenosis to get the flow down to 1 so heart is very good at maintaining flow. When you get to a 60-80% stenosis you start eating into your flow reserve and that causes angina.

disruption of metabolism causes Depain.

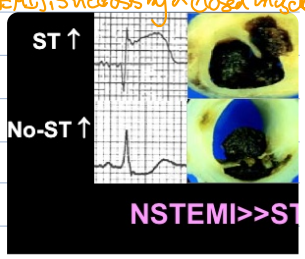
Adenosine build up causes feeling of chest tightness / compression.

Stable angina → Triggered when ↑ demand for blood (↑ exercise). It is not the supply that becomes ↓ but the flow reserve.

Diagnostic Framework →



How you treat suspected ACS is based only on history + ECG. You will have time to measure Troponin, Straight to Cath lab. emergency.



Suspected Acute Coronary Syndrome (ACS) →

10% of all presentations are chest pain.

ST elevated MI (STEMI) is necessarily an occluded A.
Non-STEMI (NSTEMI) is necessarily an occluded muscle

if you don't measure Troponin, Straight to Cath lab. emergency.

- NSTEMI pt's do not need a clot busting drug as the problem is platelets. Need anti-platelets + Heparin. This is NOT ultra-urgent.
- High Troponin tells you your CM has died but not how.

Anti-Anginals → are drugs that improve the Supply-demand imbalance of the heart.

① Beta-AR Blockers ② L-type Ca^{2+} channel blockers ③ Nitrates ④ ATP sensitive K^+ channel openers. ⑤ If channel blockers.

Preventing MI → ↓ cholest, BP

- use ACE-I, Aspirin + ADP ® antagonist, B-blockers, Statins
- Stop Smoking, control diabetes.

Post MI → The heart will dilate and this is accelerated by plaque rupture.

- current passing through AVN is not sensed as current is too small!!
- T wave flipped on ECG = ischaemic
- Troponin T and I IVS are used to monitor MI
- Atherosclerotic plaque rupture → platelet aggregates → thrombus
  - w/ RH failure → ankle edema
    w/ LH failure → ↑LA P so ↑pulm P so pulm edema
  - Angina + MI probability is v. similar.
  - only when stenosis reaches 60-70% inner artery diameter enough blood flow reserve + get angina.
  - STABLE angina is triggered on ↑ demand. Supply is fine but flow reserve ↓
  - ↳ To stop plaque buildups → Statins, anti-platelet drugs, Aspirin,

- STEMI = necrosis by a closed A. = in bw of epicardium
  NSTEMI = necrosis by a closed muscle = in bw of myocardium
  ↳ need anti-platelets + Heparin

Preventing MI → ↓cholest+ BP, ACEi, B-blockers, Statins (↓ cholest+).
(↓cholest+ ↓heart effort)

Anti-anginals → improve Supply-demand imbalance of heart
- ↳ B-blockers
- ↳ L-type Ca^{2+} channel blockers } ↓ contraction
- ↳ Nitrates
- ↳ K^+ channel openers.
- ↳ If channel blockers