Understanding Troponin & How to avoid Troponin-itis

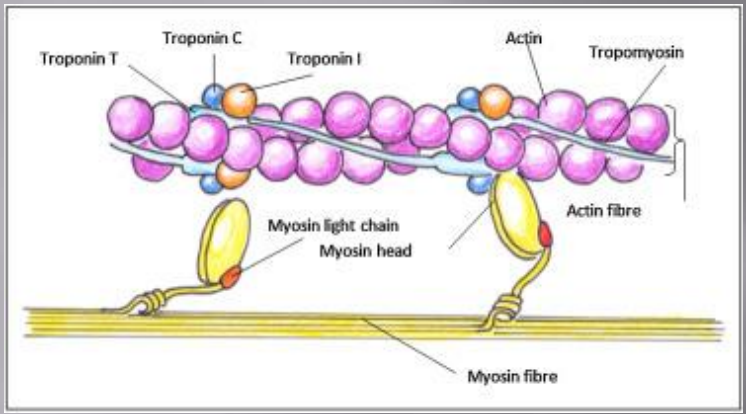
Dr Fahad Farooqi Consultant Cardiologist
BHRUT & Barts

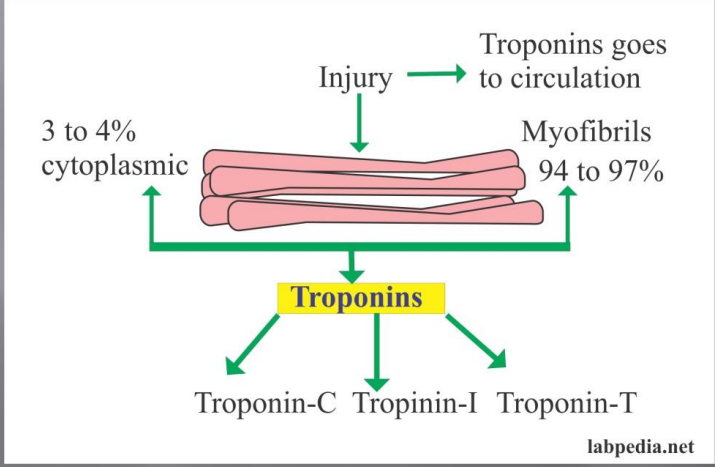
Troponin facts

- 97.5% of Troponins requested DO NOT have an acute MI diagnosis
- Most are ordered inappropriately, despite no clinical suspicion for ACS
- Other than MI, several cardiac conditions and non-cardiac conditions can raise troponin

- Inappropriate use:
  - endangers pt. safety
  - wastes clinical time and resources
  - causes diagnostic confusion
  - delays discharge

What is Troponin (cTn)?



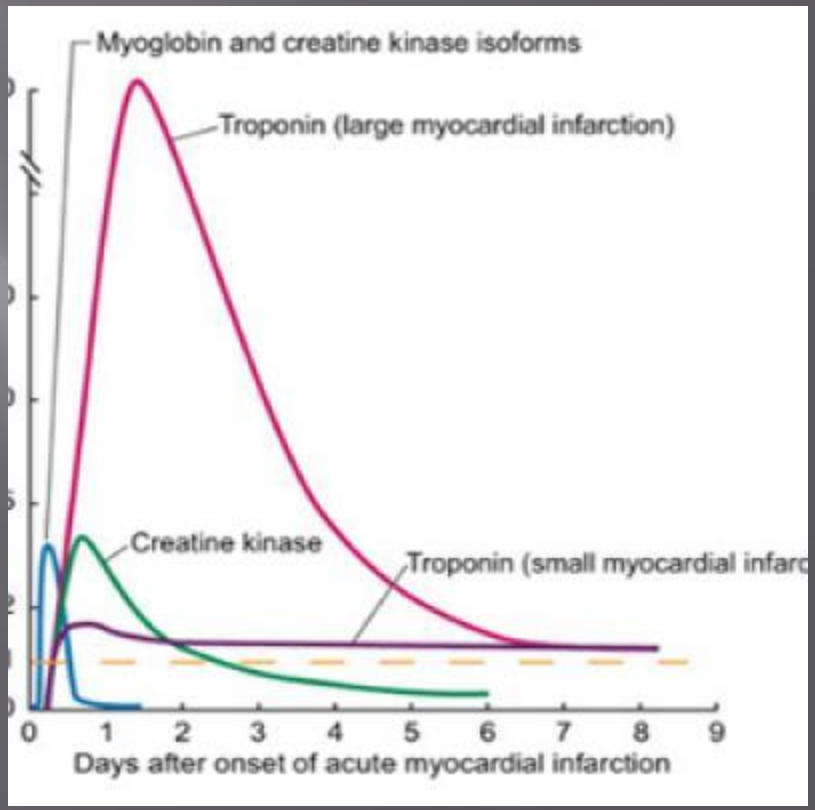


- Trop I & T found *only* in cardiac muscle
- protein component of the cardiac myofibril

- Released into circulation during cardiac myocyte death
- It *does not* indicate the mechanism of the myocyte injury
- As bio-markers hs assay TnI and TnT have **100% sensitivity for AMI**, *but very low specificity*

cTn Levels

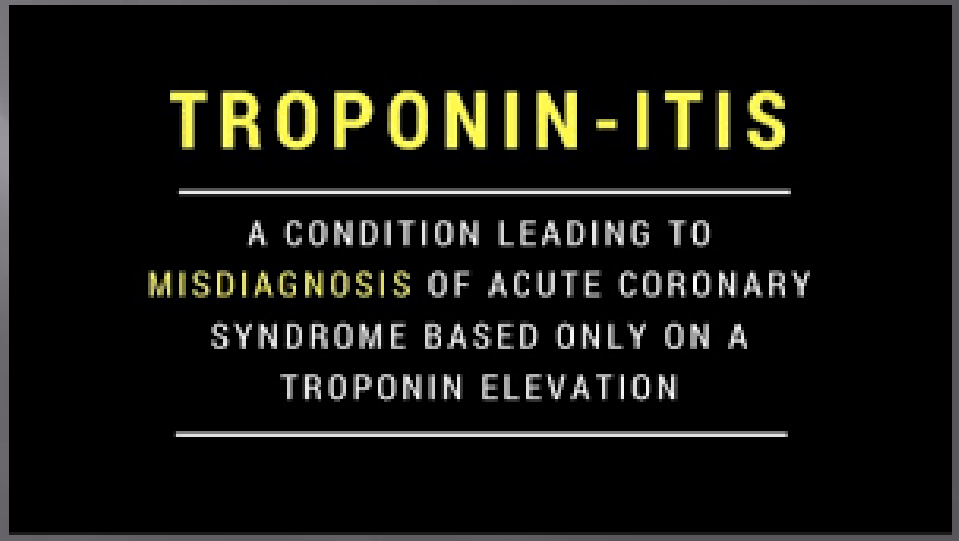
- In healthy people cTn levels are *usually* undetectable
- **'Normal levels'** falls below the *99th percentile* in the blood test. *May be inaccurate observational study of 20K consecutive I/P and O/P showed 5% of hospital patients having routine blood tests (for any reason) have troponin greater than the reference level
- **cTn levels** can elevate within 3-4 hours after the heart has been injured and can remain high for up to 14 days



* Curzen et al, BMJ March 2019

What is Troponin-itis?

- *Clinician believes elevated cTn = AMI… Irrespective of history , clinical exam findings or likely alternative causes*
- **Afflicts only clinicians, never patients**



Emergency Medicine Journal, BMJ.com, 2018

How to avoid Troponin-itis

IT'S ALL ABOUT THE CONTEXT!

How to avoid Troponin-itis?

3 core principles to interpreting 'positive' cTn

- *Should troponin have been sent?*
  - Clinical context – history, clinical exam, ECG(s)
- *Patient's baseline condition*
  - presence or absence of factors that may predict a cTn elevation
- *Rise / fall in serial cTn*
  - differentiate acute rise from chronic cTn elevation

Chest Pain

- **Chest pain** up to 5-8% of all ED visits
- About 50-60% of **chest pain** patients presenting to the ED are hospitalised
- 70% of those patients ***not*** discharged from ED are subsequently shown to ***not*** have acute cardiac disease
- Check hs Tn at 0 and 3 hrs, if being used as rapid rule out

CP- Cardiac vs. non-Cardiac

NON-ISCHAEMIC CP

- **Onset:** sudden or gradual
- **Severity:** any
- **Site:** Localised or diffuse ,Right sided, back, epigastric, interscapular`
- **Characteristics:** sharp / stabbing/ burning/ 'electric shock' dyspepsia
- **Relieving/exacerbating factors:** Worse on twisting, turning, coughing, relief with burping
- **Radiation**- +/-
- **Duration**- Less than 1 minute or continuous for days
- **Triggers:** spontaneous or related to meal **times**
- **Associated symptoms:** dyspepsia, anxiety
- **Relevant Hx**: known GORD/gallstones/anxiety/ arthritis

TYPICAL CARDIAC CP

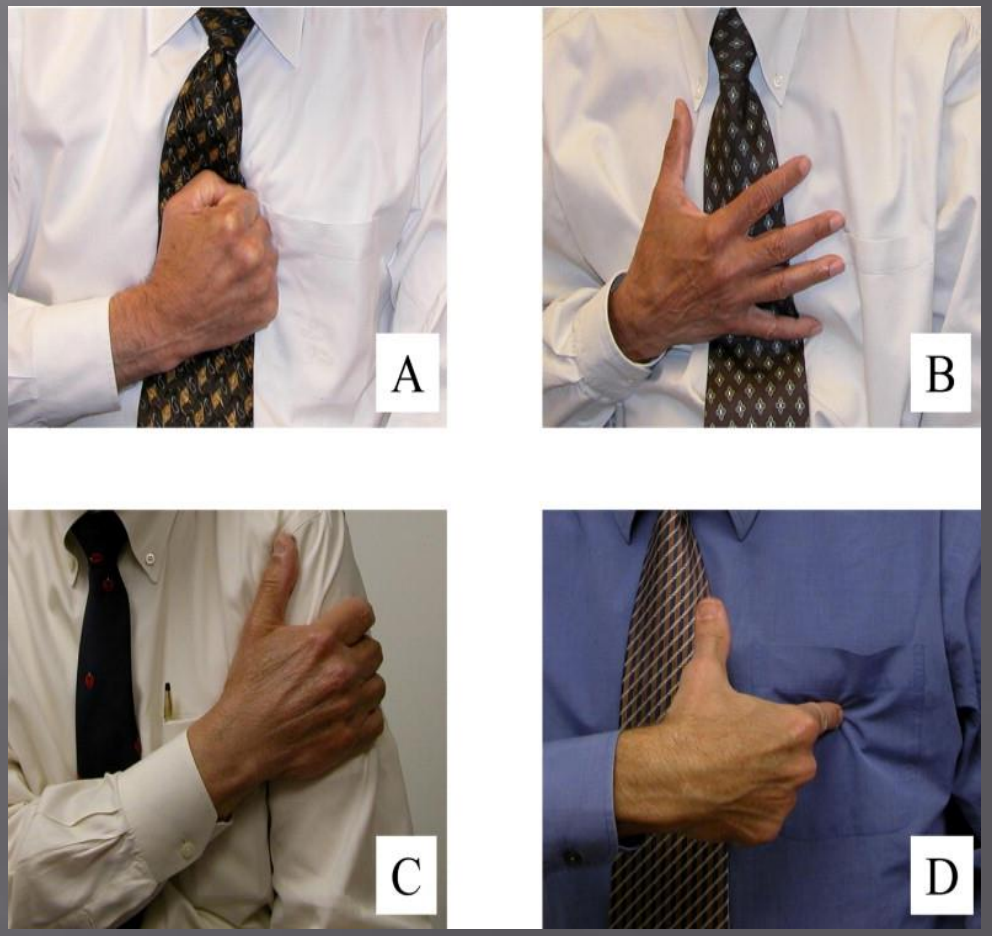
- **Onset** – *gradual*
- **Severity**- *AMI- builds up to severe, UA / SA is mild*
- **Site**- *diffuse, central retrosternal*
- **Characteristics**- *tight band, heavy, squeezing, aching, sometimes burning*
- **Relieving /exacerbating factors**- *usually none but pre-existing exertional pain possible, UA may respond to GTN*
- **Radiation**- *left arm, neck, jaw, epigastrium, sometimes back*
- **Duration**- *UA usually atleast 5 mins. AMI atleast 30 mins. Not continuous for days*
- **Associated symptoms**: *SOB, sweating, nausea, vomiting*
- **Relevant Hx:** *like previous MI, crescendo deterioration in exercise tolerance, multiple CVS risk factors*

- **ACS uncommon in men <30years, women <40 years**

Common causes of Non-ischaemic Chest Pain

- Musculoskeletal
- Anxiety
- Pleurisy
- GORD /Peptic ulcer disease/ indigestion
- Oesophageal spasm – severe pain, responds to GTN
- Gallstones
- Myocarditis/Pericarditis
- **Don't forget life threatening causes**: ***PE ,aortic dissection,, pneumothorax, oesophageal rupture!***

ACS Rx: aspirin / clopidogrel / fondaparinux can risk life threatening bleeding



Classification of ACS

All AMI is ACS, but not all ACS is AMI

Typical cardiac sounding chest pain history

Chest Pain Duration few mins ----------→ hours

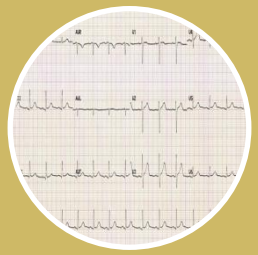
Normal ECG→ ischaemic ECG with no ST Elevation

ST Segment Elevation

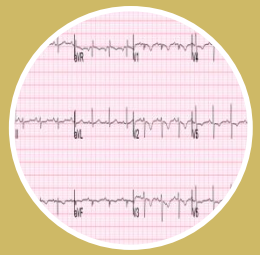
Troponin-ve

Troponin +ve

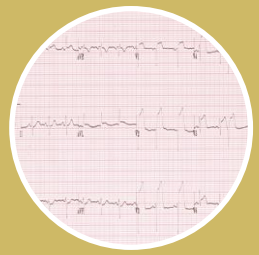
Do NOT test Troponin



Unstable Angina



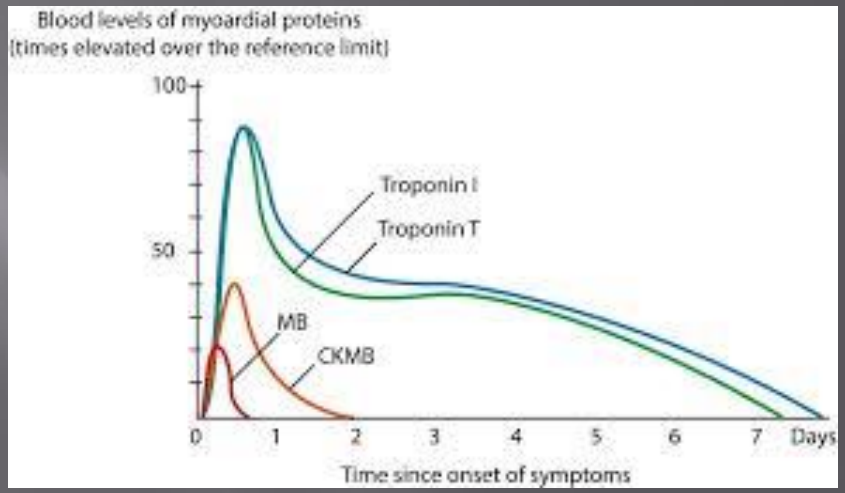
NSTEMI



STEMI

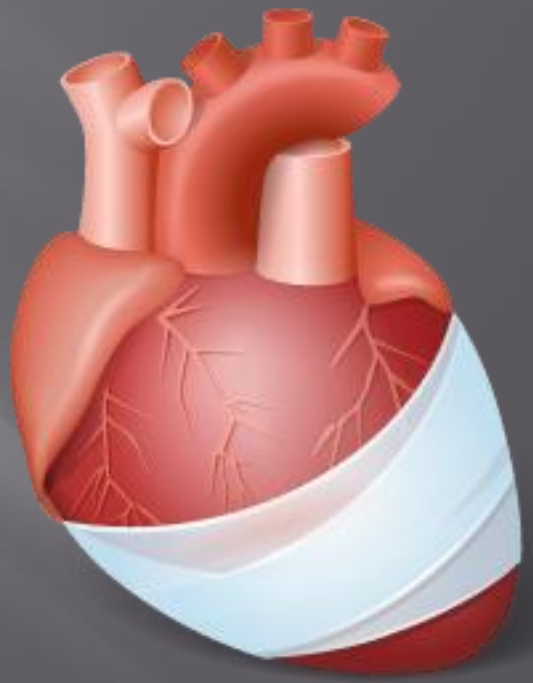
Acute cTn elevation – rise and fall

- marker of acute myocardial injury
  - **does not tell you how the injury was sustained!**
- Myocardial Injury could be due to:
  - Ischaemic (ACS due to obstructive coronary disease)
  - Hypoxia -oxygen demand /perfusion mismatch- with or without underlying IHD
  - Inflammation
  - Trauma – myocardial contusion
  - Drug toxicity



‘Non –MI’ cardiac causes of Myocardial Injury

- All arrhythmias – sinus tachy, AF, Flutter, SVT, VT
- Heart Failure
- Myocarditis (inflammatory myocardial injury)
- Valvular heart disease
- Cardiomyopathy- HOCM/ Takot Subo
- ICD shocks/ Post PCI / Post catheter ablation
- Trauma - Cardiac contusion
- Hypertension/ hypotension

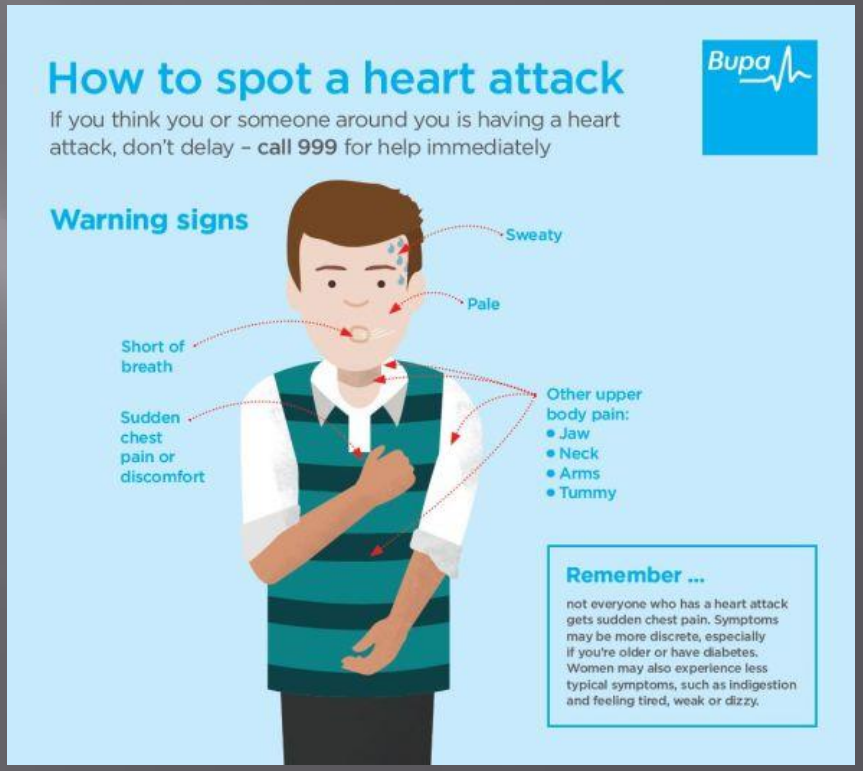


Secondary Myocardial Injury occurs in virtually ALL sick patients

- **All causes of haemodynamic disturbance/shock**: sepsis, haemorrhagic, cardiogenic, arrhythmia
- **All causes of Hypoxia**– Pneumonia, COPD, PE, pulmonary oedema (cardiogenic or non-cardiogenic), pulmonary hypertension
- **All causes of AKI/ CKD**
- **All arrhythmias** – sinus tachy, AF, Flutter, SVT, VT
- **All causes of Cardiac Arrest**
- **All causes of LVH or cardiac infiltration amyloid / sarcoid**
- **Falls**- including severe skeletal muscle injury, rhabdomyolysis
- **Stroke/SAH**
- **Drugs- chemotherapy**
- **Strenuous exercise**

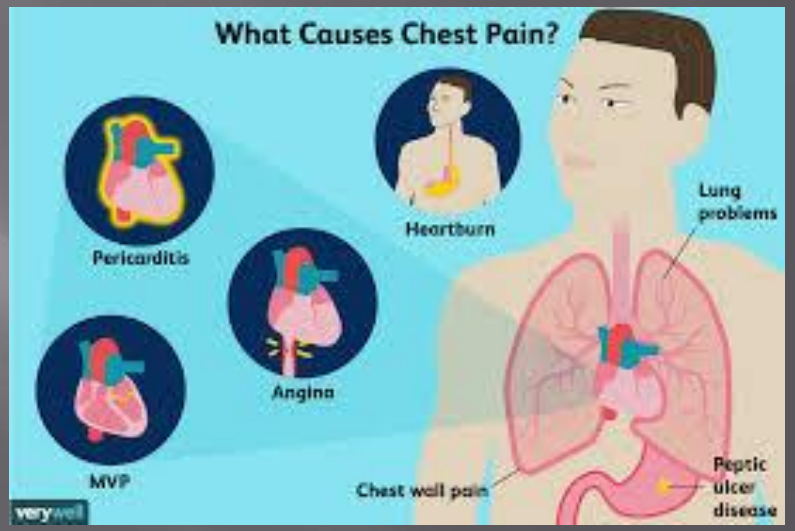
Principles of Chest Pain assessment

- Troponin is **not a screening test**
- **Recognise the characteristics 'cardiac sounding' vs. non cardiac chest pain**
- Clinical assessment should focus on **history, clinical examination** and **ECG – if troponin result does not fit consider alternative diagnosis**
- if cause is unclear, **apply clinical judgement to make a rational differential diagnosis** and investigate accordingly



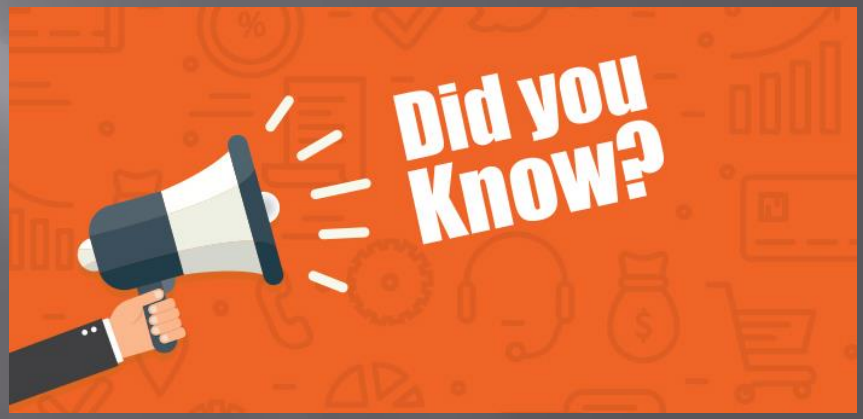
Principles of Chest Pain assessment

- pts. are **unlikely to have more than one cause for acute chest pain**
- **Review and repeat ECG in acute cardiac sounding chest pain** – dynamic ischaemic changes cannot be diagnosed on single ECG
- **Trop –ve, normal ECG and atypical chest pain excludes MI**, but **doesn't rule out UA, PE or aortic dissection**
- **Not all raised troponin pts. require hospitalisation**



Quiz -True or False

- Troponin is only found in the heart?
- All troponin release is due to myocardial injury?
- Negative troponin excludes ACS?
- All patients presenting with cardiac sounding chest pain should have troponin testing?



Quiz -True or False

- Chest pain responsive to GTN indicates ischaemic cardiac cause?
- Chest pain similar to patients previous MI is likely to be recurrent ACS?
- A very high troponin level must be due to AMI?
- Some healthy patients continuously leak troponin?
- ‘Silent MI’ has no symptoms?

Summary

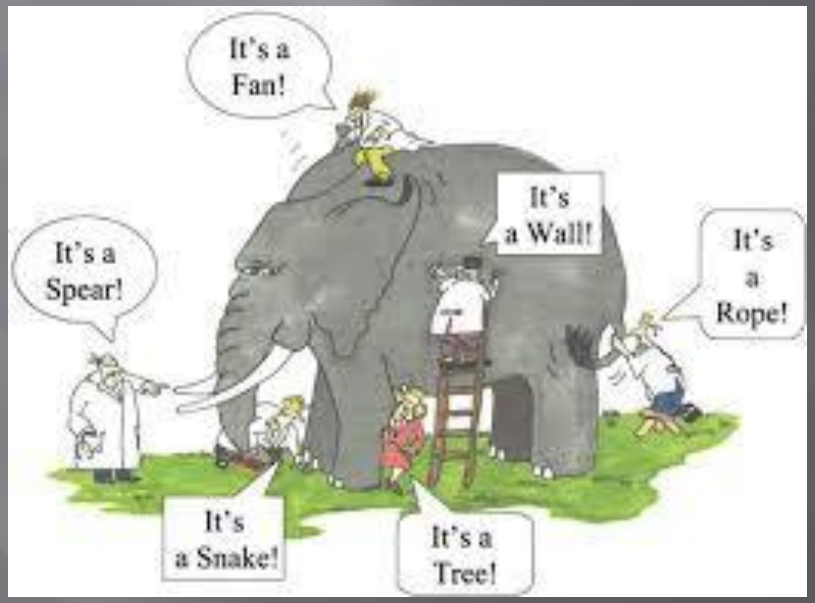
- Think if cTn is required, BEFORE you request it
- Always treat the patient, not the troponin result
- Don't develop troponin-itis!
- Thank you for listening, any Questions?

Types of Myocardial Infarction

Type	Clinical history of the patient
Type 1	MI consequent to a pathologic process in the wall of the coronary artery (eg, plaque erosion/rupture, fissuring, or dissection), resulting in intraluminal thrombus.
Type 2	MI consequent to increased oxygen demand or decreased supply
Type 3	Sudden unexpected cardiac death before blood samples for biomarkers could be drawn or before their appearance in the blood.
Type 4a	MI related to PCI
Type 4b	MI related to stent thrombosis
Type 5	MI related to CABG

For the purposes of this talk we will focus on Type 1,2 and myocardial injury

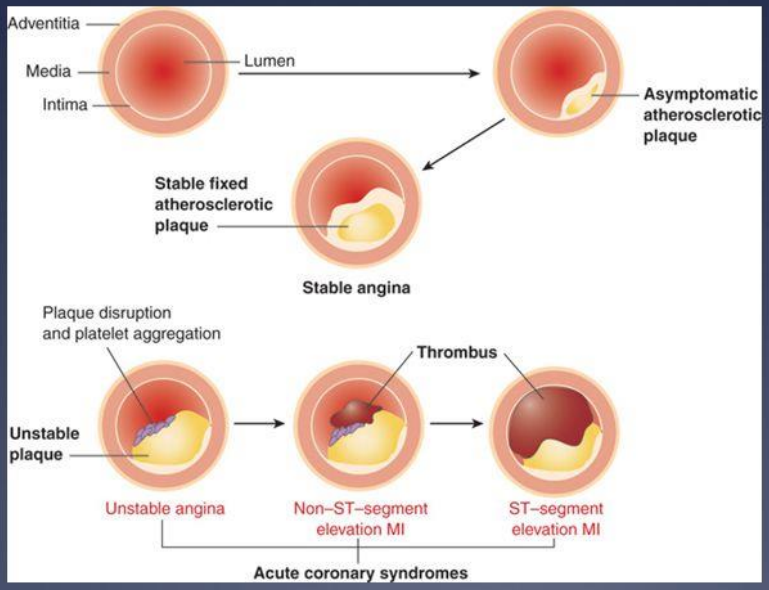
It's all about the CONTEXT



Pathophysiology of AMI

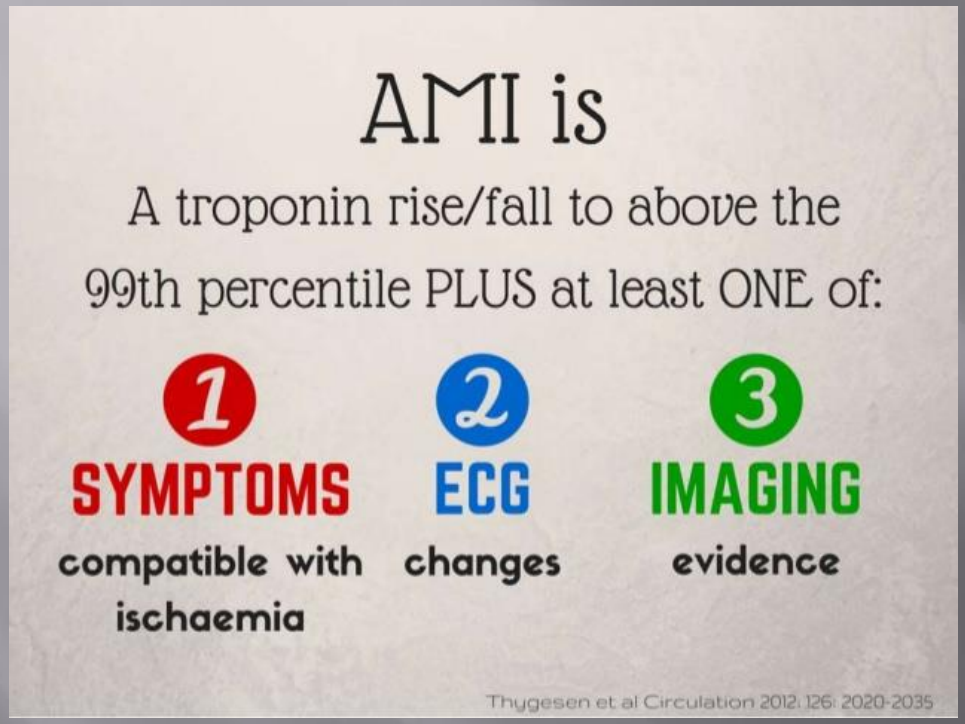
What is Acute Coronary Syndrome?

Stable Angina | Unstable Angina | NSTEMI | STEMI



N.B. Excludes coronary artery spasm, coronary artery dissection, coronary embolism

AMI vs Myocardial Injury



- Myocardial Injury:
  - Very common, due to numerous different causes
  - Only requires Raised cTn biomarker only

Both are associated with adverse prognosis, if underlying cause not treated