

# acute coronary syndromes [created by Paul Young 06/10/07]

**general** - coronary artery disease accounts for over 30% of deaths in Western countries.

## classification

- Unstable angina:**  
 - ischaemic chest pain with is recent in origin, is more frequent, severe, or prolonged than the patient's usual angina; is more difficult to control with drugs; or is occurring at rest or with minimal exertion  
 - cardiac biomarkers are not elevated
- Myocardial infarction:**  
 - ischaemic symptoms with raised cardiac biomarkers  
 - STEMI: ST elevation  
 - NSTEMI: no ST elevation

## risk factors

- modifiable:**  
 (i) by life-style  
 - smoking  
 - obesity  
 - physical inactivity  
 (ii) by pharmacotherapy or lifestyle  
 - hypertension  
 - dyslipidaemia  
 - diabetes  
 - hyperhomocysteinaemia
- non-modifiable:**  
 - increasing age  
 - male gender  
 - family history

## ECG changes in AMI

- hyperacute (0-20 minutes)**  
 - tall peaking T waves & progressive upward curving & elevation of ST segments
- acute (minutes - hours)**  
 - persistent ST elevation with gradual loss of R wave in the infarcted area.  
 - ST segments begin to fall & there is progressive inversion of T waves
- early (hours to days)**  
 - loss of R wave and development of pathological Q waves in the area of ischaemia.  
 - Return of ST segments to baseline with persistence of T wave inversion
- indeterminate (days to weeks)**  
 - pathological Q waves with persisting T wave inversion.  
 - ST segments normalise (unless there is aneurysm)
- old (weeks to months)**  
 - persisting deep Q waves with normalised ST segments

## criteria for AMI in LBBB

- (i) new LBBB  
 (ii) concordant ST elevation of >1mm  
 (iii) concordant ST depression of >1mm in V1, V2 or V3  
 (iv) discordant ST elevation of >5mm

## thrombolysis contraindications

- absolute contraindications:**  
 (i) active bleeding or bleeding diathesis (excluding menses)  
 (ii) significant closed head injury or facial trauma within 3 months  
 (iii) suspected aortic dissection  
 (iv) risk of intracranial haemorrhage (any prior ICH, ischaemic stroke within 3 months, cerebral vascular lesion, brain tumour)
- relative contraindications:**  
 - risk of bleeding  
 (i) current use of anticoagulants (the higher the INR the higher the risk)  
 (ii) non-compressible vascular punctures  
 (iii) recent major surgery  
 (iv) prolonged CPR >10 minutes  
 (v) internal bleeding within 4 weeks  
 (vi) active peptic ulcer  
 - risk of ICH  
 (i) history of chronic, severe, poorly controlled hypertension  
 (ii) severe uncontrolled HTN on presentation (>180mmHg systolic; or >110mmHg diastolic)  
 (iii) ischaemic stroke more than 3 months previously  
 - other  
 (i) pregnancy

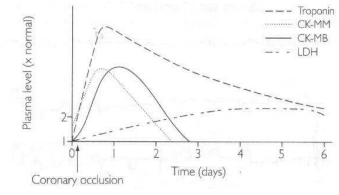
## anatomical patterns of myocardial injury

Location of injury	affected Leads	Infarct-related artery
Anterior/Septal	V <sub>1</sub> , V <sub>3</sub> , V <sub>4</sub>	Mid LAD or Diagonal branch of LAD
Inferior	II, III, aVF	RCA or posterolateral branch of Cx
Lateral	I, aVL, V <sub>5</sub> , V <sub>6</sub>	Cx or LV branch of Cx
True Posterior*	V <sub>1</sub> and V <sub>2</sub>	Posterolateral branch of Cx or Posterior Descending Branch of RCA
Anterolateral	I, aVL, V <sub>2</sub> - V <sub>6</sub>	Proximal LAD
Inferolateral	II, III, aVF	aVL, V <sub>5</sub> , V <sub>6</sub>
Right Ventricular	V <sub>1</sub> R, V <sub>4</sub> R	RCA

- 8% of patients with MI will only display ST elevation in posterior or right precordial leads

## biomarkers in ACS

- Troponin I or T:**  
 - troponin rise indicates myonecrosis & is a high risk feature in non ST elevation acute coronary syndrome  
 - troponin remains elevated for 5-14 days and therefore may not be useful for identifying early reinfarction  
 - troponin elevation is often delayed by 4-6 hours after infarction
- CK:**  
 - should be monitored for 48 hours serially & can be measured subsequently if there is suspected reinfarction
- CK-MB:**  
 - more specific than CK for myocardial infarction & may be used to confirm a reinfarction  
 - earliest rise of CK & CK-MB occurs at 3-4 hours with a peak at 12-24 hours and normalisation by 48 hours



## management of ST elevation AMI

- reperfusion therapy:**  
 - reperfusion can be obtained with fibrinolytic therapy or PCI  
 - a combination of fibrinolysis and PCI can also be used  
 - CABG surgery may occasionally be more appropriate with particular anatomy & may be considered as rescue therapy in patients who fail revascularisation  
 - PCI is the best available treatment; however, benefit depends on prompt access to service and if delay is longer than 90 minutes until balloon inflation thrombolysis should be administered.  
 - **PCI is clearly better in the presence of cardiogenic shock**
- antiplatelet therapy:**  
 - aspirin 300mg should be given to all patients with STEMI unless contraindicated  
 - both the VA Cooperative Study Group and the Canadian Multicentre Trial showed that aspirin reduces the risk of death or myocardial infarction by 50% in patients with unstable angina or non-Q wave infarction  
 - clopidogrel should be given as a load 600mg to all patients who require a stent & should be continued for at least 12 months; clopidogrel should be given to selected patients given fibrinolysis. If urgent CABG is likely, clopidogrel should be withheld  
 - in the CURE trial, clopidogrel given in addition to aspirin within 24hrs of unstable angina symptoms led to significantly reduced of cardiovascular death from 11.4% to 9.3% but was associated with a 1% absolute increase in major, non life threatening bleeds as well as a 2.8% increase in major bleeds associated with CABG within 5 days  
 - ticlopidine & clopidogrel (thienopyridines) are second generation platelet inhibitors acting independently & theoretically synergistically with aspirin
- antithrombin therapy:**  
 (i) with PCI: unfractionated heparin should be administered with dose dependent or whether IIb/IIIa inhibitors are used; the role of enoxaparin in acute STEMI following PCI remains to be determined  
 (ii) with fibrinolysis: heparin or enoxaparin should be used fibrin-specific fibrinolytic agents. The use of antithrombin therapy in conjunction with streptokinase is optional.
- glycoprotein IIb/IIIa inhibitors:**  
 - reasonable to use post primary PCI although data are conflicting regarding efficacy. They reduce mortality the 30-day risk of non-fatal AMI by 38% in NSTEMI in patients undergoing PCI. They have not been shown to be beneficial in the routine management of medically treated patients (GUSTO-IV-ACS)  
 - there are two classes of glycoprotein IIb/IIIa inhibitors  
 (i) murine monoclonal (eg abciximab)  
 (ii) 'small molecule' inhibitors (eg tirofiban & eptibatide)  
 - should be avoided with fibrinolytic therapy because of risk of bleeding; platelet infusion may treat significant bleeding in patients receiving abciximab but not in those receiving tirofiban or eptibatide)
- nitrates:**  
 - reduce myocardial oxygen demand through afterload reduction and may on improve myocardial oxygen delivery through coronary vasodilation  
 - may lead to dramatic resolution of ischaemia in coronary vasospasm  
 - GISSI-3 and ISIS-4 trials failed to demonstrate mortality reduction from acute or chronic nitrates; nevertheless, they remain first line therapies for symptomatic angina and when myocardial infarction is complicated by CCF
- beta blockers:**  
 - iv beta blockers should be considered for patients with tachycardia or hypertension post infarct in the acute setting  
 - oral beta blockers decrease mortality after myocardial infarction and should be administered to all patients who can tolerate them
- ACEIs:**  
 - SAVE trial showed that captopril in patients with EF<20% post AMI lead to a 21% reduction in mortality  
 - ISIS-4 showed a smaller reduction in mortality for all patients treated with captopril post AMI  
 - HOPE showed patients with vascular disease or high risk of atherosclerosis benefited from ramipril
- statins:**  
 - decrease risk of adverse ischaemic events in patients with CAD

Feature	Inferior	Anterior
Onset	Slow (usually via Mobitz I)	Sudden (usually via Mobitz II)
QRS complex	Narrow	Wide
Ventricular rate	>45 bpm	<45 bpm (often 20-30 bpm)
Escape pacemaker	Stable	Unstable
Drug response (e.g. atropine)	Yes	No
Haemodynamic effects	No (usually)	Yes
Permanent pacing	No (usually)	Yes (if high degree A-V block persists)
Prognosis	Good	Very poor

## heart block in AMI

## risk stratification of non ST elevation acute coronary syndromes

- (i) high risk consists of clinical features of ACS with any of the following:  
 - repetitive or prolonged (>10mins) ongoing CP  
 - elevated cardiac biomarkers  
 - persistent or dynamic ECG changes (ST depression or TWI)  
 - transient ST elevation  
 - cardiogenic shock  
 - sustained VT  
 - syncope  
 - EF<40%  
 - prior CABG  
 - percutaneous coronary intervention within 6 months  
 - presence of known diabetes with typical ACS features  
 - chronic renal failure with typical ACS features
- (ii) intermediate risk consists of clinical features with any of the following:  
 - resolved chest pain that occurred at rest or was repetitive or prolonged  
 - age >65  
 - known CAD  
 - two or more of the following risk factors (hypertension, family history, active smoking or hyperlipidaemia)  
 - presence of known diabetes mellitus with atypical ACS features  
 - presence of chronic renal failure with atypical ACS features  
 - prior aspirin use
- (iii) low risk  
 - presentation with clinical features of an acute coronary syndrome without intermediate or high risk features

## management of non ST elevation acute coronary syndromes

- high risk patients require aggressive medical management and coronary angiography  
 - intermediate risk patients require inpatient monitoring and investigation and provocative testing  
 - low risk patients can be discharged with follow-up  
 - ESSENCE trial showed that low molecular weight heparin (enoxaparin) reduced the combined end point of death, MI or recurrent ischaemia at both 14 & 30 days when compared with heparin