- right ventricular infarction occurs in up to 30% of patients with inferior infarction
- clear CXR with distended jugular veins in an inferior AMI suggests RV infarction
- IAPB may be useful particularly when RV pressures are elevated
- reperfusion is critical
- rhythm disturbance is common following acute myocardial infarction & is most likely within the first few hours of onset and during reperfusion
- risk of arrhythmia is decreased by correcting hypoxaemia, hypovolaemia & acid base disturbance; K+ & Mg should be maintained (risk of VT declines as potassium increases until it is greater than 4.5mmol/L, there is no evidence that Mg levels have any effect on ventricular arrhythmia in this setting; however, ILCOR recommends Mg>1.0mmol/L)
- prophylactic iv magnesium (4hr) was of benefit in the LIMIT II study but not in the ISIS-4 trial and is not recommended
- prophylactic lignocaine tends to increase mortality and is thus reserved for the treatment of VT & VF.
- causes of ischaemia after infarction include:
  (i) coronary reocclusion or spasm
  (ii) anaemia
  (iii) hypotension
  (iv) hypermetabolic states
- immediate management includes aspirin, beta blocker, iv GTN, heparin, consideration of calcium channel blockers & diagnostic coronary angiography
- post-infarction angina is an indication for revascularisation
- CABG should be considered for patients with left main disease
- if angina cannot be medically controlled or there is haemodynamic instability consideration should be given to an IABP
- reinfection in the 10 days following MI occurs in 5-10% of patients
- typically occurs in the first week after infarction
- the classic patient is elderly, female and hypertensive
- free wall rupture presents as a catastrophic event with shock & EMD
- salvage is possible with prompt recognition, pericardiocentesis and repair
- if the patient is hypotensive fluids should be administered
- septal rupture manifests as severe heart failure or cardiogenic shock with a pansystolic murmur and parasternal thrill
- the hallmark finding is left to right intracardiac shunt with an increase in oxygenation from the RA to the RV although echo is most easy way to make the diagnosis
- rapid institution of IABP and pharmacological support is required and operative repair is required for long-term survival
- occurs in 1-2% of cases of MI usually in large infarctions
- ischaemic MR is usually associated with inferior AMI & ischaemia or infarction of the posterior papillary muscle
- papillary muscle rupture typically occurs 2 to 7 days after AMI and presents dramatically with pulmonary oedema, hypotension and cardiogenic shock
- when a papillary muscle ruptures the murmur of acute MR may be limited to early systole because of the rapid equalisation of pressures in the left atrium & left ventricle [the murmur may be inaudible if cardiac output is low]
- management includes afterload reduction including IABP +/- inotropes
- definitive therapy is surgical repair or valve replacement which should be undertaken as soon as possible
- embolic stroke occurs in 1-3% of patients (mostly following extensive AMI)
- 30-40% of anterior Q wave MIs may be complicated by mural thrombus
- embolism is uncommon following inferior infarction
- pericarditis is a common early complication of extensive infarction
- pericardial friction rub may be heard in 10-15% of patients with anterior infarction
- occurs 24-72 hours after infarction & may mimic ischaemia
- Dressler's syndrome is now uncommon but is thought to be an immunopathic response to myocardial necrosis. It is characterised by fever, elevated ESR, a pericardial friction rub &arthralgia & may occur some weeks after MI.