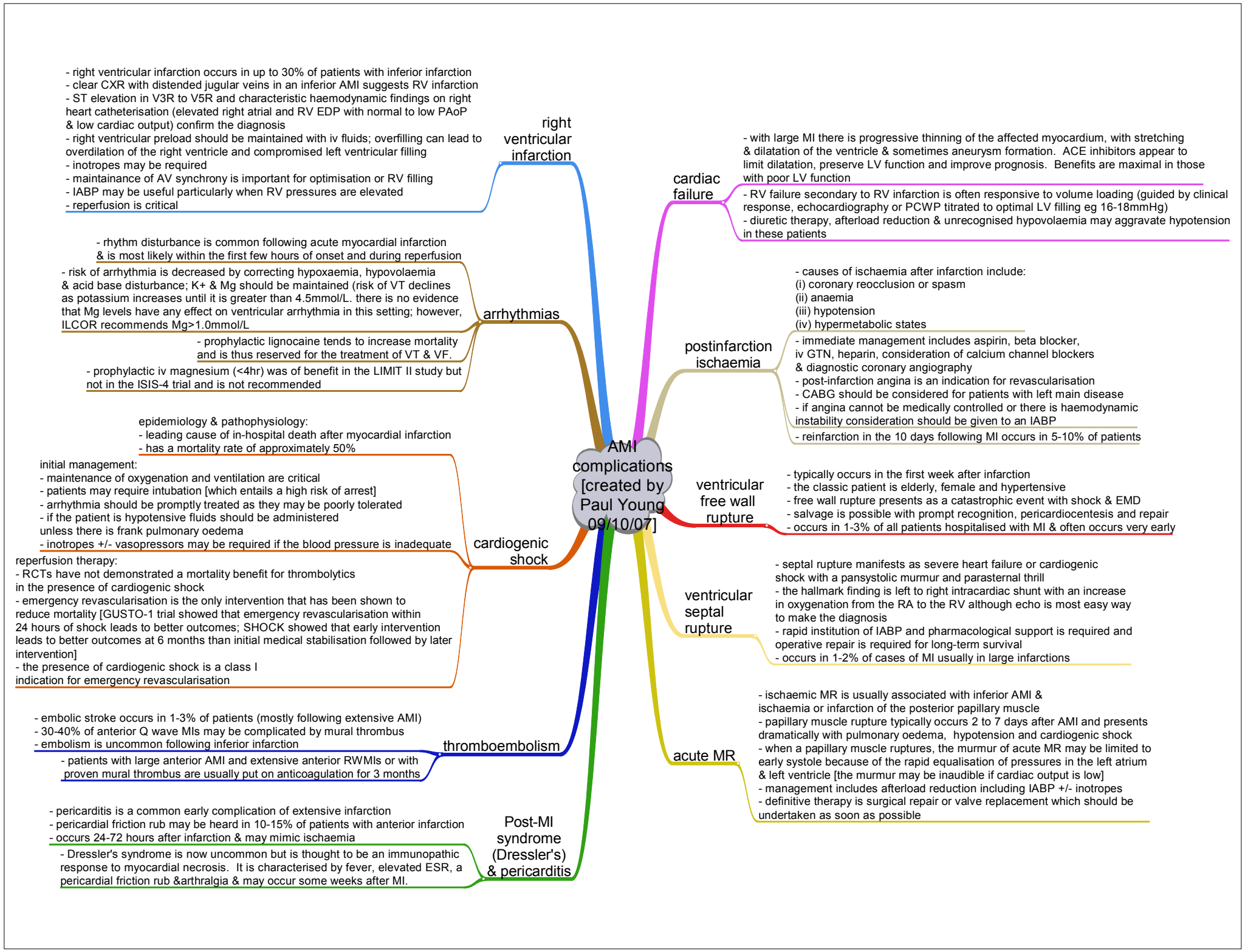
AMI complications [created by Paul Young 09/10/07]
right ventricular infarction
- right ventricular infarction occurs in up to 30% of patients with inferior infarction
- clear CXR with distended jugular veins in an inferior AMI suggests RV infarction
- ST elevation in V3R to V5R and characteristic haemodynamic findings on right heart catheterisation (elevated right atrial and RV EDP with normal to low PAoP & low cardiac output) confirm the diagnosis
- right ventricular preload should be maintained with iv fluids; overfilling can lead to overdilation of the right ventricle and compromised left ventricular filling
- inotropes may be required
- maintainance of AV synchrony is important for optimisation or RV filling
- IABP may be useful particularly when RV pressures are elevated
- reperfusion is critical
arrhythmias
- 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 lignocaine tends to increase mortality and is thus reserved for the treatment of VT & VF.
- prophylactic iv magnesium (<4hr) was of benefit in the LIMIT II study but not in the ISIS-4 trial and is not recommended
cardiogenic shock
epidemiology & pathophysiology:
- leading cause of in-hospital death after myocardial infarction
- has a mortality rate of approximately 50%
initial management:
- maintenance of oxygenation and ventilation are critical
- patients may require intubation [which entails a high risk of arrest]
- arrhythmia should be promptly treated as they may be poorly tolerated
- if the patient is hypotensive fluids should be administered unless there is frank pulmonary oedema
- inotropes +/- vasopressors may be required if the blood pressure is inadequate
reperfusion therapy:
- RCTs have not demonstrated a mortality benefit for thrombolytics in the presence of cardiogenic shock
- emergency revascularisation is the only intervention that has been shown to reduce mortality [GUSTO-1 trial showed that emergency revascularisation within 24 hours of shock leads to better outcomes; SHOCK showed that early intervention leads to better outcomes at 6 months than initial medical stabilisation followed by later intervention]
- the presence of cardiogenic shock is a class I indication for emergency revascularisation
thromboembolism
- 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
- patients with large anterior AMI and extensive anterior RWMIs or with proven mural thrombus are usually put on anticoagulation for 3 months
Post-MI syndrome (Dressler's) & pericarditis
- 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.
cardiac failure
- with large MI there is progressive thinning of the affected myocardium, with stretching & dilatation of the ventricle & sometimes aneurysm formation. ACE inhibitors appear to limit dilatation, preserve LV function and improve prognosis. Benefits are maximal in those with poor LV function
- RV failure secondary to RV infarction is often responsive to volume loading (guided by clinical response, echocardiography or PCWP titrated to optimal LV filling eg 16-18mmHg)
- diuretic therapy, afterload reduction & unrecognised hypovolaemia may aggravate hypotension in these patients
postinfarction ischaemia
- 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
- reinfarction in the 10 days following MI occurs in 5-10% of patients
ventricular free wall rupture
- 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
- occurs in 1-3% of all patients hospitalised with MI & often occurs very early
ventricular septal rupture
- 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
acute MR
- 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