JULY 2007
VIVA 6

55 year old male presents with cardiogenic shock
BP 90/60mmHg, CI 1.8L/min/m^2, SVR 5000 dyne.sec.cm-5

What drugs would you use to increase the BP in this situation?

"Please define shock"

Is a pathological state in which there is a mismatch of oxygen supply and demand which results in inadequate end organ perfusion and tissue damage.

"Describe this patients blood pressure"

normal is 120/80, this patient is therefore hypotensive

"What is a normal Cardiac Index"

is the CO/BSA, which is normally 5/1.73, which equals 2.9. this patient is therefore in cardiogenic shock

"What is the normal Systemic Vascular Resistance"

is a measure of peripheral resistance, 80(MAP - MRAP)/CO which equals 1700 dyne.sec.com^{-5}
this patient therefore has increased peripheral resistance

"Describe the physiological state of this patient"

In summary, this patient has partially compensated for the impaired function of the heart by increas ing peripheral resistance.

"How would you manage these physiological parameters"

High flow oxygen therapy, consider early intubation
Consider judicious fluid challenge
Iontropes and Vasoactives

"Could you please give me some examples of inotropes which could be appropriate in this setting"

Ideally an ionodilator would be approriate in this setting

- Milrinone
  - selective phosphodiesterase inhibitor
  - increases intracellular cAMP
  - causes increased ionotropy and arterial and venodilation
  - minimally metabolised, requires dose adjustment in renal failure (HL 2.5hrs)
  - may cause SVT/VT and worsen outcomes in acute on chronic heart failure
- Dobutamine
  - direct acting sythetic catecholamine derivative of isoprenaline
  - direct agonism of B1 - increased ionotropy, chronotropy and MVO2
  - minor vasodilatory effects due to weak B2 actions, overall increased BP
  - may precipitate tachyarrythmias, AFlut/Fib
  - very short half life (2 minutes), metabolised hepatically and in tissues
- Levosemindan
  - dual actions of sensitising myocytes to Ca (at lower doses)
  - phosphodiesterase III inhibition (at higher doses)
  - also causes vasodilation through opening of ATP sensitive K channels
  - limited pharmacokinetic data, half life around an hour
  - interference with K channels leads to increased QTc and risk of arrythmias

Use of noradrenaline and adrenaline may be considered if BP drops further