Second 2009
VIVA 6

This viva will test your knowledge about the use of diuretics, pharmacokinetics and dynamics. And Use of drugs during a cardiac arrest. Candidates were asked to explain mechanism of action of diuretics, in particular frusemide. Candidates were also tested upon the basis that various vasoactive drugs (eg adrenaline and vasopressin) are used in cardiac arrest, in particular their mechanism of action and routes of administration.

"How do diuretics work?"

decrease sodium reabsorption along the nepron,
increasing urinary sodium and water loss
potency of action is determined by the site of action

"Discuss the pharmacology of frusemide"

it is a loop diuretic
it is available as both an oral formulation and in parenterally as a clear colourless liquid
its mechanism of action; blocks Na.K.2Cl symporter, decreasing Na reabsorption and disrupting CCM
effects; prompt diuresis,
side effects include; hypokalaemia, hyponatraemia, hyperchloraemic acidosis
it has bioavailability of 50%
onset of diuresis is 30-60 mins PO, and 5 mins IV
it is extensively protien bound (up to 95%), and crosses the placenta
metabolism is hepatic via gluronidation, and it is dependent on hepatic blood flow
excretion is via the urine and faeces

"What drugs are used in the cardiac arrest protocol?"

the new guideline for advanced adult life support recommend only adrenaline and amiodarone
shockable rhythm
- 1mg adrenaline should be given after the second shock and second loop after
- 300mg amiodarone can be given after the third shock

non shockable rhythm
- 1mg adrenaline immediately then every second loop

"Discuss the pharmacology of adrenaline"

is a naturally occuring catecholamine
it used for iontropic support, in anaphylaxis and with local anaesthetics to cause vasoconstriction
it is presented as a clear colourless solution, in minijets 1mg in 10ml or undiluted 1mg/ml in vials
its mechanism of action is via the alpha and beta receptors of the SNS
at lower doses it has vasodilatory effects and bronchial dilatation via beta 2 receptors
at moderate doses it has effects on beta 1 receptors causing increase ionotropy and chronotropy
at high doses it acts as a vasoconstrictor
its side effects include severe hypertension, arrhythmias, deranged metabolic states
increased gluconeogensis, glycogenolysis, insulin increases then decreases with increasing dose
it has is generally delivered IV, but can be given IM or SC as well as inhaled
it has a rapid onset of action within seconds - minutes
it does not cross the BBB
metabolised by COMT and MAO
its half life is around 2 minutes
excretion is via urine to inactive metabolites