Q1 Describe the pharmacology of suxamethonium (Sept 2013, Q6 March 2011)

Suxamethonium is the dicholine ester of succinic acid which acts as an ultrashort acting depolarising muscle relaxant. It is used in rapid sequence induction and to modify seizures caused by ECT.

PHARMACEUTICAL – structurally is composed of two acetylcholine molecules joined via acetate methyl groups. It is presented as a clear solution 50mg/ml

PHARMACODYNAMIC
MECHANISM OF ACTION → mimics the actions of Ach by binding to the nicotinic Ach receptor and causing membrane depolarization. However, because its hydrolyzing enzyme is not present at the NMJ, the effect lasts longer than for Ach. The persistent depolarization renders the voltage sensitive Na channels inactive within 1-2 mins. This prevents the transmission of further APs. Initially causes fasciculations, then muscle relaxation.

SIDE EFFECTS →

- GIT - Intragastric pressure increases and LOS falls (risks of aspiration)
- Metabolic - Rise of serum K by 0.2-0.4mmol/L (more marked in patients with burns, muscle denervation eg SCI, renal failure),
- CVS effects – bradycardia / ventricular arrhythmias
- CNS – increase in IOP (significant in the presence of globe perforation)
- Other - anaphylaxis, myalgias, malignant hyperthermia in genetically susceptible individuals. Abnormal plasma cholinesterase genes comprise <0.03% of the population and result in reduced plasma cholinesterase and prolonged (10 min) sux apnoea.

PHARMACOKINETICS
ADMINISTRATION
Route - IV
Dose - 0.5-2mg/kg
Time to onset - within 30 sec (action indicated by the presence of fasciculations)
Duration 3-5 min

DISTRIBUTION
Vd – unknown. Crosses the placenta
Protein binding - unknown

METABOLISM - rapid hydrolysis by plasma cholinesterase to choline and succinylmonocholine (relatively inactive), such that only 20% of the administered dose reaches the NMJ.

ELIMINATION - 2-10% excreted unchanged in urine. Half life 2-5 mins