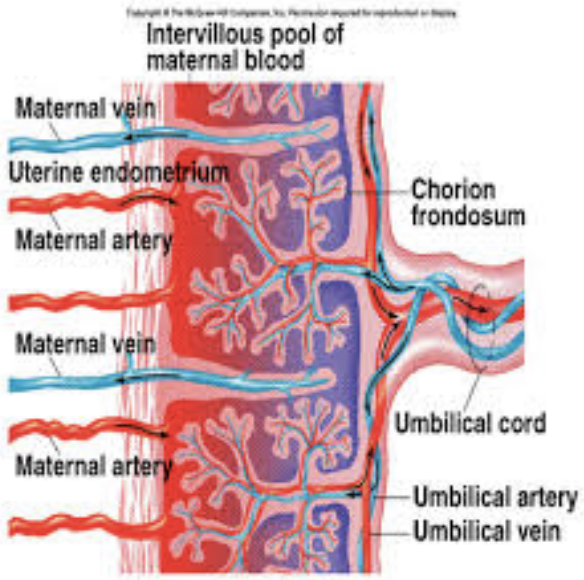
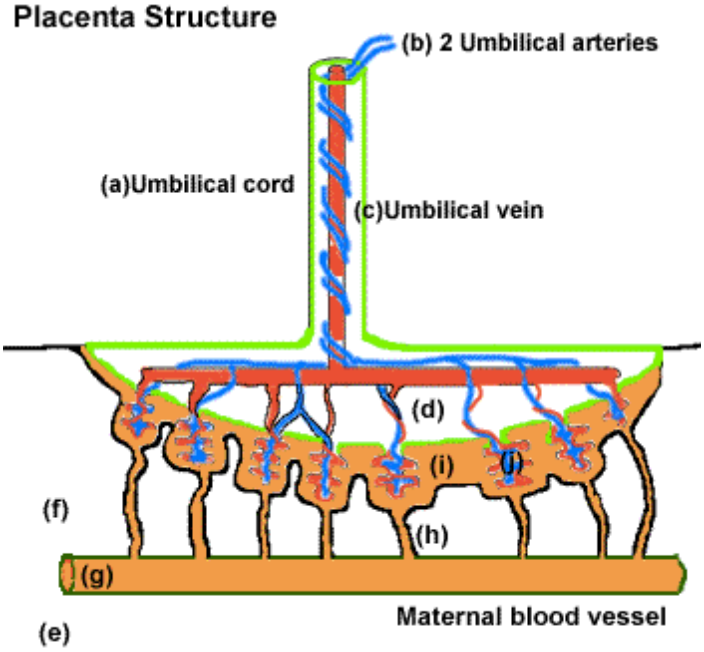
# 2013 September VIVAs

Viva 1
ABG of type 2 respiratory failure.
What types of respiratory failure do you know....what type of respiratory failure is represented by this blood gas?
What would be the bicarbonate in this blood gas?
Is this patient on supplemental oxygen?
How can you tell? The alveolar gas equation
Can you write it please and I will add some values...
Affects of CO2 on the CNS in general (not just CBF). fatigue, confusion, dizziness, blurred vision, decr seizure threshold
Effects on CBF
What is morphine.
Advantages and disadvantages of using morphine in a PCA keo 10-40 minutes
How can we measure GFR?
How can we measure GFR without having to measure the urine volume ad concentrations of things?
Metabolism types in the liver
Aims of metabolism
Examples of drugs that undergo metabolism and get converted to active compounds
Structure of a nerve and label of the nerve components
epineurium>perineurim>endoneurium>axon. Also has blood vesels within
Lipid solubility of local anaesthetics
Types of nerve
Local Anaesthetics MOA
identify structures of Local anaesthetics - prilocaine and lignocaine
Structure activity of catecholamines
What is salmeterol? What things other than a long tail from the amine tail make more B activity?
ß carbon hydroxylation ⇒ NA
bulky amine end = ↑ß activity eg isoprenolol
3,5 –OH aromatic ring = ↑B2 action
α carbon -methylation of ⇒ resist MAOI
Types of nerves and where they are found
sensory
motor
cranial nerves – originate in CNS
spinal nerves – from spinal cord. 31 pairs mixed nerves ⇒ branch into 2 roots 1 sensory & 1 motor

The structure of the placenta




The placental gas exchange
How do drugs go across the placenta
Draw an action potential

Viva two
What is this view of the larynx on laryngoscopy?
Name the structures in this view - names them all but then specifically asked what is the name of the structure between the true and false cords!
What is the nerve supply to the epiglottis?
What is the significance of this for Anaesthetics?
What is the nerve supply to the vocal cords?
What type of epithelium is on the epiglottis? squamous epithelium (resistant to abrasion)
What about the trachea? respiratory epithelium: ciliated columnar cells with goblet cells
What types of flow occur from the nose to the terminal bronchioles?
Why?

What types of cartilage do you know?
elastic, hyaline, fibrocartilage

What types of epithelium do you know?
4 basic tissue types in human: epithelium, CT, muscle, nervous tissue
squamous
cuboidal
columnar
What is the difference in cartilagarytenoif e between the epiglottis and the arytenoids?
epiglottis = elastic (more flexible), arytenoids = hyaline (stronger)
A person you are looking after develops stridor? What is the cause of this?
What can be done to reduce the stridor?
Simple – calming
Pharm – neb adreanaline, steroids
Invasive - +ve pressurve ventilation (NIV) or intubate
Why do you use heliox - reduces density...does it reduce viscosity? I said I didn't think so...only density
What is the most significant way you could reduce stridor - I mentioned elements of hagan posiulle. Increase pressure, increase radius by nebulised adrenaline and steroids, CPAP, slow breathing, intubate
What is pulmonary vascular resistance?
Does that truly represent Ohms law? No because ohms law applies to a direct current whereas the pulmonary circulation is pulsatile.
What are the determinant of PVR?
How does PVR change with volume?
Why does that happen ...why is it that shape? Explain...
What happens to PVR when we give an anaesthetic? I said drugs would decrease cardiac output and thus increase, however this is balanced by the reduction in HPV which would decrease...overall no change
What ways can we reduce PVR?
What drugs can we give to reduce PVR? Mentioned nitrates and prostacyclin inhaled...
Mechanisms if actions of these drugs please...
Draw a washout curve for SEVO...why is it that shape?
What are the main determinates of the initial washout?
Why does it take longer for the second part of the washout curve?
How long does it take for MAC to reduce from 1.0 to 0.5 for sefoflurane?
How does the washout curves of SEVO, nitrous and halothane differ?
Why?

Viva 3
What are the differences between the neonatal and adult respiratory system?
What is surfactant?
How does surfactant work in the neonatal lung?
How does suxamethonium work?
What are the metabolites of sux?
How does it stop working?
What's a phase 1 block?
What's a phase 2 block? Show me the differences you would see on a nerve stimulator?
What is a nerve stimulator?
How does it work? What would you see after a post tetanic count?

What is compliance?
What are the types of compliance?
What are the disadvantages of nitrous oxide?
What types of breathing system would you use for a patient that has hypoxia?
How would you measure the degree of hypoxia in a patient in recovery with hypoxia?
How does a pulse oximetry work?

What is beer and lamberts law?
How does it distinguish between arterial and venous flow?
What are the limitations?
Tell me about scavenging systems...

Opening question – what IV fluids do you commonly use?

- what is in normal saline/hartmanns/plasmalyte
- what happens to lactate
- enzymes involved in conversion of pyruvate to glucose. 11 enzyme catalysed reactions. starts with pyruvate carboxylase & ends with glucose 6 phosphatase
- what happens to pyruvate in CAC – wanted detailed steps of CAC.
    - series of reactions which acetyl Coa is completely oxidised to CO2 & H+
    - acetyl CoA condensed with oxaloacetate ⇒ citrate
    - 4 pairs H atoms transferred to electron transfer chain
    - 12 ATP & 4H2O created
    - major entry points = ß oxidation FA's, glycolysis, ketones, aa (direct)
- effects of glucose on plasma K – cause ↑insulin secretion ⇒ ↑K into cell ⇒ ↓plasma K
- drugs that affect K
    - alkalines ⇒ H out of cell, K in
    - ß2 agonists
    - insulin
    - calcium resonium – chelators
    - IV calcium – antangonise hyperkalaemic arrhythmia risk
    -
- drugs used in cardiac arrest
- what is adrenaline (briefly)
- what is amiodarone – what class of antiarrhythmic, effects of cardiac AP
    - side effects of amiodarone
    - elimination half life – if such a long elimination half life why do we give rapid bolus?
- What is a defibrillator
    - How do we charge a defibrillator with mains current. step up transformer to charge battery, capacitor to store charge before release

2nd examiner

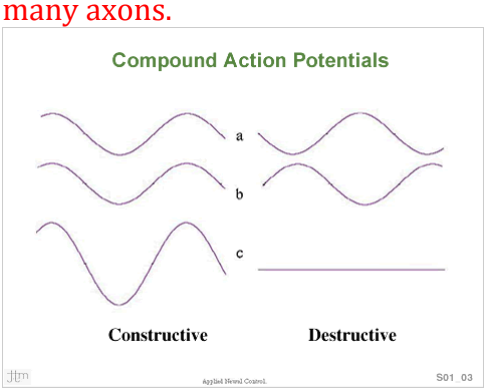
- what is ketamine
- MOA – what is NMDA receptor, what you need for activation
- Dissociative anaesthesia – what would you see
    - catalepsy
    - analgesia
    - amnesia
    - classic droperidol & fentanyl
- Effects on ICP – raised but trial work in trauma dismissed it as significant
- What is Monroe Kellie doctrine
- Draw intracranial elastance curve and explain
- Components of cranial vault

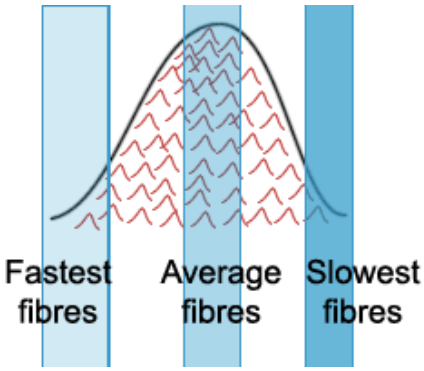
- Formation, pathway and resorption of CSF
- Ways to reduce ICP
  - pain
  - CO2
  - restriction to outflow/30deg head tilt
  - HTN
  - conc saline
  - historically mannitol
- CO2 vs CBF graph
- Effects on volatiles on CBF (flow metabolic uncoupling)

VIVA 2

Opening question – classify antiarrhythmics

- classify calcium channel blockers and give examples
- MOA of verapamil
- How is it metabolised
  - demethylation in liver- norverapamil (active metab with sig antiarhytmic properties)
  - diltiazem active metab des-acetyl-diltiazem
- What is safer – po or IV? Dangers of IV admin. rapid effect site conc ⇒ ↑SEs eg hypotension, neg iontropy
- First pass metabolism
- Drug interactions – wanted digoxin. any AV node blockers!!!!
- Draw SA node AP
  - How does adrenaline vs atropine alter it
- Draw nerve AP
- What is LA, MOA
- Recognise ester vs amide structure – the actual structures were lignocaine and procaine
- Classify peripheral nerves and functions within each group
- Draw compound AP = summation of all action potentials in nerve with many axons.




- Structure-activity relationship for esters vs amides

2nd examiner

- why do we fast patients? For how long? 6 hours light meals, 2 hours clear fluids
- Why do fasted pts have reduced UO
- Talked about increased plasma osmolality

    - Define osmole, osmolality vs osmolarity
    - ADH control system
    - Effects of ADH
    - How to measure osmolarity
        - Osmometers that use Freezing point depression, SVP depression or inc in osmotic pressure
        - How much does freezing point reduce by each mole of substance (1.8 degrees)
    - Uses of vasopressin
    - Other analogues of ADH – desmopressin, uses
- Functions of lower respiratory tract – anything below the trachea FIRMTITS
- Given picture of bronchial tree – point out structures, and terminal bronchiole
- Define laminar vs turbulent flow
    - Hagen pouiseille law
    - Reynolds number
    - Where does true laminar flow occur (terminal bronchioles) vs turbulent.
    - Other types of flow in bronchial tree – mixed, diffusion
    - Where do bronchodilators work – on smooth mm of airways. max 3-6$^{th}$ generation of segmental bronchi

VIVA 3

- Opening question: ABG – pH 7.35, Pco2 60, Po2 90mmHg
- Discuss ABG – what type of disorder (resp acidosis with metabolic compensation) – acute vs chronic (chronic)
- Is this patient on O2 – used alveolar gas equation – yes must be on oxygen
- What is a starlings resistor? Where do starlings resistors occur in the body?
- West zones of lung
- Does zone 1 occur in normal conditions? What is it called? (dead space)
- Shown picture of salmeterol and told it was salmeterol
    - What type of drug is salmeterol
    - What gives it beta selectivity
    - Discussed structure activity relationship of cathecholamines

2$^{nd}$ examiner

- What are some pharmacogenetic causes of variability in drug response. = inherited variation in drug response. aka idio-syncrasy. examples:
    - sux apnoea
    - MH
    - slow/fast acetylators  eg hydralazine
    - hereditary resistance to oral anticoag
    - acute porphyria

  (supersensitivity = receptor up reg from previous occurance)
- Discussed malignant hyperthermia – cause and management
- Asked about genetic causes of different receptor sensitivities in drugs – mentioned opioid but didn't know anymore details

- Why do patients vomit after anaesthetic
    - Patient, surgical, anaesthetic factors
- What anti-emetics do you use – give examples
- How does dexamethasone work?
- Half life of dexamethasone (190mins) vs duration of action – why does DOA outlast half life?
- NNT for vomiting vs nausea. How does that compare with ondansetron
    - ondansetron 5-6 NNT
    - dex 4mg 3.7 NNT
- Physiological effects of 1 dose of dexamethasone
    - supra norm steroid for 72hrs in body
        - CNS – excitation, insomnia, confusion
        - RESP - ↓inflamation/chance of broncoconstriction/upper airway oedema
        - CVS – permissive effect on catecholamines if required
        - M/skel - ↓bone healing/wound healing – controversial
        - Imm - ↑neutrophils, ↓lymphocytes
        - UGS - ↑water retention ???
        - ↑chance of cancer seeding
        - Met - ↑BSL
- Why do you use dexamethasone instead of ondansetron? (ondansetron is good rescue antiemetic)