

Pharmacology Multiple Choice Question Bank

Primary Exam for FANZCA - July 2001 Update [MCQPharmJul2001v3.doc] (v3.02)

[1] Queensland Anaesthesia Website: <http://www.qldanaesthesia.com>

- All these questions are also available on the website and may be printed from the separate web pages (File->Print on your browser) or downloaded as a complete file (THIS document).
- Please **re-format** the file as required before you print. The MCQs are in 10 point size for easier reading. Change this to whatever you want. Also adjust the file so that individual questions don't print over 2 pages.

[2] Some Answer Comments are available on the site

Answer commentaries and/or references for some of these questions are available on the site. There is also a form where you can submit your comments about any question & this will be posted on the website for the use of all. Thanks in advance for contributing in this way.

[3] Separate Physiology & Pharmacology files

The MCQs have been split into these 2 sections. This is the Pharmacology file. This decreases the size of the file to minimise download time which can be long with large attachments if you have a slow connection.

[4] Why .DOC files OR .RTF files?

The files can be downloaded from the site in several formats: either .DOC (MS Word format) or as .RTF files. Download the type you require. Please read the details on the site about why you may prefer the .RTF format.

[5] Marker Questions

Questions that have a lot of symbols (meaning they have been asked multiple times) are probably all 'Marker Questions' - The score from these questions are used to do a comparison between the groups sitting different papers. These questions are more likely to be on the paper you sit so it is worth your while to know these well.

[6] Thank your colleagues

This collection has been made possible by the efforts of your colleagues who have sat the exam & have written down the questions they have been able to recall. Please thank them for their efforts and please assist by sending along the questions you remember after your paper. (Email is the preferred method for submission of questions)

[7] Many questions are incomplete

In some the question wording may be misleading. In any case the examiners are prone to change some of the options at different exams. SO: The best strategy is to read around the topics suggested by the questions and not try to rote learn answers. A substantial number of these questions will definitely appear on your paper.

[8] Contribute yourself

If you find this collection useful & would like to help in improving this 'Memory Bank' of *Actual Primary MCQs*, could you please send along to me the questions that you can remember after you sit your exam. The question codes remain the same so just use the Question Code to indicate the repeat questions.

[9] Primary Email List

This collection gets updated usually after each exam (ie at least twice per year) as I receive new questions or other collections. If you would like to receive these updates, contact me with your email address and I'll add you to the Mailing List for Primary Updates

[10] FREE

There is *no charge* for this collection. This is a group effort which I am happy to coordinate. Please copy & distribute to assist other registrars with their primary study.

[11] "The Physiology Viva: Questions & Answers"

This book is currently out of print: sold out!. A second edition should be available by 2002. This book was written especially for the Primary ANZCA exam. Contact me for further details or queries if required.

**Thanks, Best wishes with the exam,
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Please copy this collection & distribute to your colleagues

Pharmacology - Question Classification

GP	General Pharmacology
IN	General Anaesthetics - Inhalational
IV	General Anaesthetics - Intravenous
LA	Local Anaesthetics
MR	Muscle Relaxants & Antagonists
OP	Major Analgesics / Opioids
AC	Anticholinergics/Antimuscarinics
PS	Psychotherapeutic Drugs
CD	Cardiovascular Drugs
EN	Endocrine Drugs
MD	Miscellaneous Drugs
ST	Statistics

Coding Letters

The letters (from a to k) within the square brackets [] after the question code indicate which paper(s) the question was on. The key is:

a = Mar 96 paper	b = Jul 96 paper
c = Mar 97 paper	d = Jul 97 paper
e = Mar 98 paper	f = Jul 98 paper
g = Mar 99 paper	h = Jul 99 paper
i = Feb 00 paper	j = Jul 00 paper
k = Apr 01 paper	l = Jul 01 paper

Eg: question CV01 [adgi] . . . was on the papers in Mar 96 (indicated by the 'a'), Jul 97 ('d'), Mar 99 ('g') & Feb 00 ('i')

General Pharmacology

GP01 [a] A drug is given at a dose of 50 mg/kg to a 70 kg man. The plasma concentration after giving it is 10 mg/ml. The elimination half-life is 8 hours. Clearance would be:

- A. 1.3 l/h
- B. 3 l/hr – The answer, based on the above should be 30.3 mL/h
- C. ?
- D. 125 l/hr

$$\frac{CL}{V_d} = k_{el} = \frac{\ln 2}{t_{1/2}}$$
$$CL = \frac{\ln 2 \times V_d}{t_{1/2}} = \frac{0.693 \times \left(\frac{50 \times 70}{10} \right)}{8} = 30.3 \text{ ml/h}$$

GP02 [a] A drug is given orally and 95% absorbed. Only 25% reaches the general circulation due to hepatic first pass metabolism. If hepatic blood flow is 1500 mls/min, the hepatic clearance is:

- A. 400 mls/min
- B. ?
- C. 1100 mls/min – Correct $((0.95-0.25)/0.95 = 1^{\text{st}}$ pass metabolism * hepatic flow rate = 1105 ml/min)
- D. ?
- E. 1425 mls/min

GP03 [d] Histamine release
(no other details)

GP04 [d] Rectal administration of drugs:

- A. Gives predictable blood levels – No, inherently unpredictable
- B. From lower 1/3rd avoids first pass & upper 2/3rds doesn't – Well, that is the theory, but in reality??
- C. None undergoes first pass metabolism – No, never say never...
- D. All of it undergoes first pass metabolism – No, not always

GP05 [gjk] LD50 is:

- A. Median lethal dose - Yes

- B. Determined in phase I clinical trial – No, this involves human volunteers
- C. Determined from log-dose response curve – No, it is determined from (dose)/(quantal response) curves – not LOG dose.
- D: Dose causing death in 50% of animals within 1/4 hours - No
- E. Half the mean lethal dose – No, it is the median lethal dose

GP06 [gi] Which of the following crosses the blood-brain barrier?

- A. GABA – No, but GABA-pentin does...
- B. Propranolol – Yes, side effects include sleepiness & depression
- C. Suxamethonium - No
- D. Edrophonium - No
- E. Dopamine - No

GP07 [fhk] With regard to drug-receptor binding:

- A. A competitive antagonist has no intrinsic activity – True, it shouldn't
- B. A partial agonist has less receptor affinity than a full agonist – No, not necessarily. It may have more affinity for the receptor but less INTRINSIC ACTIVITY... therefore less effect
- C. KD is maximal intrinsic efficacy – No, it is the equilibration dissociation constant. It is equal to the concentration of drug (D) at which 50% of the receptors are occupied. It doesn't reflect efficacy at all

GP07b [i] A partial agonist:

- A. Always antagonises a full agonist – No, not if the dose is low enough
- B: Can never be used to antagonise a full agonist – No, it is possible
- C: Has a dose response curve similar to that of a full agonist in the presence of a non-competitive antagonist. – Yes, this is correct (or more correctly – in the presence of a competitive irreversible antagonist...)
- D. ?

GP08 [fi] Placental transfer of drugs:

- A. Increases in late pregnancy – Probably the most correct option
- B. Increases late because of decreased albumin – Possibly correct for some drugs but not the whole picture
- C. Do not cross because > 600 daltons – No, but drugs with MW >500 have 'incomplete' transfer
- D. ?
- E. ?

Ref: Clin Pharmacokinet. 1995 Mar; 28(3): 235-69

GP09 [fh] Regarding pharmacokinetics:

- A. ?
- B. Half-life is inversely proportional to clearance - correct
- C. ?
- D. Half-life is proportional to steady-state - ?time to steady state
- E. B & D – Most correct based on the wording

GP10 [h] An ether bond:

- A. Formed from condensation of 2 alcohols – Yes, 2 ethanols -> diethyl-ether + H₂O
- B. Hydroxyl group on middle bond – No, R-O-R
- C. ?

GP11 [i] The NMDA receptor

- A. Ketamine is an agonist – No, it is a non-competitive antagonist
- B. Requires glycine as a modulating protein ("YES PROTEIN!") to have its effect - No
- C. Mg⁺² blocks the receptor – Yes, in the resting state
- D. Is not permeable to Calcium – No, it is a CALCIUM channel!

GP12 [i] Activated charcoal:

- A. Should be given with sorbitol – No, while it often is (to reduce the risk of constipation) it shouldn't ALWAYS be given with sorbitol (especially in children)

- B. Is not effective against theophylline – No, it IS effective in acute theophylline overdose
C. Should be given with ipecac – No, ipecac may make it difficult to give charcoal.
D. Should be given in a drug:charcoal ratio of 1:10 – No... no good evidence. The United States Pharmacopeia (USP DI, 1997) recommends the following oral dosage regimen.
- Children up to one year of age:1 g/kg
- Children 1 to 12 years of age:25 to 50 g
- Adolescents and adults:25 to 100 g
(A) could be the 'most correct' answer given this bunch of clowns...

GP13 [k] Therapeutic index:

- A. Easy to determine in humans – No - how many humans do the ethics committees allow you to kill? ☺
B. ?
C.
D.
E. Derived from LD50/ED50 – Yes, the ratio of the two In that order

GP14 [k] (A Basic drug with a pKa of 8.7)

- A. ?
B. ?
C. Will be predominantly ionised at plasma pH – Correct BH+ will predominate, and at a lower pH than pKa, more will be protonated/ionised

GP15 [k] Oxygen toxicity

- A. Causes convulsions at less than 100 kPa – No, that's only one atmosphere
B. Causes lipid peroxidation at less than 100 kPa – Yes (Nunn p502)...

GP16 [l] With regard to log/dose response curves:

- A. The response is fairly linear over the 20-80% range – This is probably the most correct, albeit confusing
B. The Dose is fairly linear over the 20-80% range – No, the LOG(dose) is though
C. The ED50 and slope are characteristic for each drug – No. For example, adding competitive antagonist will shift the curve to the right; adding a non-competitive (or competitive, irreversible) antagonist will change the slope for the same drug (Technically, this should be the EC50 not ED50 as referred to here – ED50 is from a DOSE/%RESPONDING curve not a LOG DOSE/RESPONSE curve... there is a difference)
D. ?
E. ?

GP17 - renumbered to another section.

GP18 [l] With regards to diffusion through a membrane:

- A. Directly proportional to thickness – No, inversely
B. Inversely proportional to thickness – Yes
C. Inversely proportional to Surface area – No, directly
D. Inversely proportional to concentration difference – No directly
E. ?

General Anaesthetics - Inhalational

IN01 [a] Which compound(s) is/are broken down in soda-lime?

- A. Nitrous oxide – Not sure, but contaminants (such as nitric oxide N2O) ...may cause problems...BOC gases state the the nitrous oxide cylinders contain AT LEAST 99.5% N2O
B. Halothane – Yes, and product is a volatile compound – a difluorovinyl compound which is nephrotoxic in rats – but not humans... (temp & humidity dependent)
C. Sevoflurane – Yes, unstable in soda lime but products apparently non-toxic in humans (Compound A) – temperature dependent – increase temp -> increase degradation
D. Desflurane – Yes, produces CO (related to the CHF2- moiety)
E. All of the above – YES... (therefore N2O by default MUST do something!!)

IN02 [a] Regarding nitrous oxide at 70%: - assuming the other 30% is oxygen!

- A. Synthesised from ? & N₂ at 273C – No, produced by heating ammonium nitrate to 240-270°C
- B. Decreases muscle blood flow by 30% - No
- C. Decreases cerebral autoregulation 24% - Possibly, provided there are no other inhalational agents (they tend to blunt N₂O's effect on increasing CBF and ICP)
- D. ?

IN02b [d] Nitrous Oxide:

- A. ?Increases/decreases CBF – Increases when used alone (effect blunted/abolished with other agents present)
- B. Is an effective oxidant – Yes, and will in fact support combustion. It will even enable combustion of agents that would not be flammable in air (don't forget that people use it in their cars to increase performance!)
- C. Is made by heating nitrogen and oxygen in an iron retort – No, heated ammonium nitrate
- D. Decreases pulmonary artery pressure in neonates – No, it actually increases pulmonary artery pressure by increasing PVR (thereby increasing the chance of persistent R->L shunt)

IN03 [abdfh] The following drugs are (potent) triggers for malignant hyperthermia EXCEPT:

- A. Decamethonium – Yes (triggers MH)
 - B. Suxamethonium – Yes (triggers MH)
 - C. Isoflurane – Yes (triggers MH)
 - D. Halothane – Yes (triggers MH)
 - E. Calcium - No – how is this possible?
 - F. Sevoflurane – Yes (triggers MH)
 - G. Tubocurarine – Yes (a few cases of MH reported) - interestingly it was originally used to treat MH (I wonder if it worked!?)
 - H. Nitrous oxide – Yes, apparently it is a WEAK trigger for MH in susceptible patients (but this is HIGHLY debateable)
- (Different options on different papers)

IN04 [a] IPPV with Isoflurane at 1 MAC results in:

- A. Depresses cardiovascular reflexes more than halothane – No, there is no reflex tachy with Halothane at all
- B. Causes decreased conduction velocity – No, in fact may increase conduction velocity via its indirect effect on transiently increasing SNS activity
- C. Maintains cerebral autoregulation – No, but effect is less than that of halothane, and it is responsive to changing pCO₂
- D. Equal respiratory depression to enflurane – No, it doesn't change rate whereas enflurane does
- E. Reduction in cardiac output by 25% - No, usually no change in cardiac output since reflex tachy continues despite decreased SV
- F. Increased vasodilatation – Yes, but does this really answer the question with the IPPV part??

IN05 [ae] The effect of increased cardiac output on Pa versus time for volatile agents is:

- A. No effect - No
- B. Decrease slope - Correct
- C. Decrease then increase slope – No
- D. Increase then decrease slope – No, initial slope UNCHANGED but the rest is DECREASED

IN06 [adk] Nitrous oxide:

- A. Supports combustion – Yes it does
- B. Is flammable - No
- C. Causes muscle rigidity – No, but it is a WEAK trigger for MH...apparently...
- D. In tissues is slower to reabsorb than oxygen – No, it diffuses out faster than oxygen
- E. Has a partition coefficient of 0.76 – No, for a start which coefficient are we talking about? (BG = 0.47, OG=1.2)
- F. All of the above
- G. Is formed by heating oxygen & nitrogen – No, ammonium nitrate to 240-270 degrees

- H. Induces methionine synthetase – No, inhibits it
- I. Oxidises the cobalt in vitamin B12 – Yes

IN06b [ef] Nitrous oxide:

- A. Has MW of 42 – No, (O=16, N=14) therefore MW=44
 - B. Critical temperature 32 C – No, 36.5 degrees C
 - C. Formed by using iron as a catalyst – Yes, this is how Priestly first produced it from NO – it's not how they make it now though...
 - D. Does not support combustion – No, it does support combustion
 - E. ?? has saturated vapour pressure of 24 kPa – No, it's a little higher than this... ☺
 - F. Produced using ammonium sulphate in an iron retort – No, ammonium nitrate
 - G. Boiling point 32C – No, boiling point is -89 degrees
 - H. ?? . . . ammonium nitrate . . . copper vessel ??
- (Multiple options as this represents 2 separate N2O questions on Mar98 paper)

IN07 [c] Desflurane

- A. Takes 5 minutes to reach equilibrium – No, longer than this >20-30 minutes
- B. Is fastest to approach equilibrium of any inhaled anaesthetic agent – No, Nitrous Oxide is faster despite the fact that Desflurane's Blood:Gas coefficient is lower at 0.45 compared to 0.47 for N2O
- C. Is a fluorinated diethyl ether – No, it is a ethyl-METHYL ether (fluorinated only)
- D. ?

IN08 [cd] Regarding sevoflurane:

- A. The vapour pressure is less than enflurane – Yes, SVP is less (the only agents with lower SVPs than Sevoflurane are METHOXYFLURANE & TRICHLOROETHYLENE)
- B. The vapour pressure is greater than Isoflurane – No, the SVP is less
- C. Cardiovascular side effects are similar to Isoflurane – Yes, main effect on SVR, no change in CO, some SNS stimulation
- D. Molecular weight less than Isoflurane – No, larger (it is the biggest)
- E. Boiling point greater than enflurane – Yes, 58 compared to 56 degrees for enflurane

IN08b [di] Sevoflurane:

- A. Is a methylethyl ether – No, it has a propyl-type arrangement
- B. Is odourless – No, has a slightly pungent/sweet odour
- C. Is stable in soda lime at 37 degrees – No, undergoes spontaneous degradation (temp dependent)
- D. Has a boiling point higher than enflurane – Yes, 58 compared to 56
- E. Has a molecular weight lower than desflurane – No, higher (168 compared to 200)

IN08c [fh] Sevoflurane:

- A. Molecular weight greater than enflurane – Yes, 184 compared to 200 (SEVOFLURANE IS THE BIGGEST)
- B. MAC less than enflurane – No, less potent therefore higher MAC
- C. Contains Cl & F – No, only Fluorine groups...
- D. SVP > enflurane - No

IN09 [cfj] Uptake of N2O when breathing 70%:

- A. More than one litre absorbed in the first minute - ?Correct option
- B. Equilibrium (?90%) is achieved in 3mins – No, usually 5-10 minutes
- C. Absorb 10 litres ?at time of ?90% equilibration / ?in first 3 mins
- D. At steady state, uptake is 200mls/min – No, at 'steady state' the uptake should technically be zero
- E. Produces surgical anaesthesia – No, this is obvious (It's MAC is 102% and that is only good for 50% of subjects...)

IN10 [cfgl] N2O causes the second gas effect because:

- A. It is relatively insoluble – Not really – it's because there is a LOT of it (ie. You need >70% to see the second gas effect)
- B. Reaches equilibrium faster than the more soluble second gas – No, this is true but doesn't account for this effect

- C. Larger volume – Well... a larger volume is absorbed, hence the effect but (D) probably 'more correct'
- D. Its high concentration – Yes, high concentration required

IN11 [d] Desflurane:

- A. Is non-irritant to the airways – No, it is quite irritating and pungent, hence not much use as an induction agent (can cause broncho/laryngospasm, coughing etc)
- B. Is more/less potent than Sevoflurane – Less potent (MAC of 6.0)
- C. Has a higher molecular weight than ?isoflurane/?enflurane – No, less (substitutes an F=19 for the Cl=35.5)
- D. Is a chlorinated methyl ethyl ether – No, only fluorinated

IN12 [dk] Effects of volatile agents include:

- A. Halothane increases hepatic artery and portal blood flow – No, decreases...
- B. Isoflurane causes hypotension by reducing cardiac output – No, effect mainly by decreasing SVR, almost no effect on CO
- C. ?
- D. ?

IN13 [dfhk] Problems with MAC:

- A. Large interspecies variability – No, but I thought it was only for human subjects...
- B. Affected by temperature and other factors - Yes
- C. Affected by obesity - No
- D. ?

IN13b [afil] MAC:

- A. Is decreased in the elderly – Yes, decreases with increased age
- B. Is unchanged throughout pregnancy - No, pregnancy decreases MAC
- C. Increases in hypothermia – No, decreases with decreased temperature
- D. ?Decreased/?increased with hyper/hypo-kalaemia – No, K doesn't affect MAC at all
- E. ?

Alt version (Jul 01) All the factors decrease MAC except:

- A. Pregnancy – Yes, decreases
- B. Hyperthermia – No, increases MAC
- C. Hypothermia – Yes, decreases
- D. Hypoxia – Yes, decreases
- E. ?

IN13c [gkl] MAC:

- A. Highest between ages 2 to 5 yrs – No, highest below 3 months of age
- B. Increases with pregnancy – No, decreases
- C. MAC BAR is concentration at which 95% do not move – No, it's to do with Adrenergic Response
- D. Is 0.2% halothane in 70% N₂O – Most correct, true value is 0.29% (MAC with 100% oxygen is 0.75)
- E. ?

Jul 01 version: With regards to MAC:

- A. The MAC of Halothane with 70%N₂O is 0.29 – Correct
- B. Concentration at which 95% of patients don't move after a surgical stimulus – No, this is 1.3 MAC
- C. MAC- BAR ?? – To do with adrenergic response
- D. Decreased by increased CO₂ – No, no change if PaCO₂ between 15-95mmHg
- E. ?

IN14 [eg] Systemic vascular resistance is LEAST changed with:

- A. Isoflurane – No, the decrease in BP is attributable to only the decrease in SVR
- B. Sevoflurane – No
- C. Desflurane – No
- D. Enflurane – No
- E. Halothane – Correct, minimal change in SVR

IN15 [efg] MAC awake during emergence when patient will respond to command:

- A. 0.1
- B. 0.2
- C. 0.3 – Not quite, see below
- D. 0.5 – Correct (see below)
- E. ?0.7 ?0.8

MAC-awake depends on the rate of 'expected awakening':

If the agent is ceased abruptly the MAC-awake will vary depending on the agent but it is roughly:

Halothane: 0.5

Isoflurane: 0.25

Desflurane & Sevoflurane: 0.33

It depends on the BRAIN -> BLOOD -> ALVEOLI gradient (it is only the alveoli we measure – the concentration at the 'effect site' will still be higher as it slowly leaves the brain)

The % at the brain will be the same (equivalent to 0.5 MAC)

If the agent is SLOWLY decreased then almost all agents have a MAC-awake of 0.5

IN16 [fh] Isoflurane & enflurane are:

- A. Structural isomers - Yes
- B. Enantiomers – No, these are stereo isomers that are mirror images of each other
- C. Diastereomers – No, these have more than 1 chiral centre and may differ in ALL properties (stereoisomers that are NOT mirror images)
- D. Optical isomers – No (?another name for enantiomers)
- E. Configurational isomers

IN17 [ab] Sevoflurane:

- A. Is broken down in the body to Compound A which has been shown to be toxic to rats – No, broken down by sodalime (KOH & NaOH but not CaOH)
- B. Has a blood:gas partition coefficient of 2.3 – No, 0.65
- C. Is an irritant causing coughing on induction – Not usually – this is mainly halothane
- D. Has a boiling point of 24 degrees centigrade – No, boiling point is 58.5 degrees
- E. Has Cl & F atoms in its structure – No, the only halide is fluorine
- F. None of the above - Correct

(Note: Compound A is a breakdown product produced in the CO₂ absorber; it is not produced by biotransformation)

IN18 [gi] With isoflurane anaesthesia, MAC awake is:

- A. 0.1% vol
- B. 0.3% vol
- C. 0.5% vol
- D. 0.5% vol - ?? 0.6% vol
- E. 1% vol

See IN15 comments... not really a straightforward question... If (D) was 0.6% I'd go for that (MAC isoflurane = 1.2%)...but it could also be (B) depending on how you interpret the question... I love how MCQs test one's knowledge...

IN19 [g] Isoflurane:

- A. Is a halogenated methyl ethyl ether – Correct (both F & Cl atoms)
- B. Higher boiling point than Sevoflurane – No, lower 48.5 compared to 58.5
- C. No odour – No, pungent odour
- D. Enantiomer of enflurane – No, enantiomers are mirror image stereoisomers (with 1 chiral centre)

IN20 [g] MAC of halothane with 70% N₂O is:

- A. 0.25% - Correct, approximately 0.29%
- B. 0.5%
- C. 0.75% - No, this is 1 MAC with 100% oxygen
- D. 1.0%

IN21 [g] All reduce MAC except:

- A. Aminopyridine – No - This drug is presumed to enhance the presynaptic release of acetylcholine by facilitating the entry of calcium ions into nerve endings (mainly at NMJ not muscarinic)
- B. ?

IN22 [f] N2O is NOT relatively contra-indicated with:

- A. Pneumothorax
- B. Ear surgery - depends on surgery type though... it can be used to inflate the middle ear by some ENT surgeons
- C. Postop nausea & vomiting
- D. Renal failure – Correct, there is no evidence of renal, hepatic or GIT toxicity

IN23 [h] Which of the following does NOT affect the speed of induction with a volatile agent?

- A. FRC – Yes, this does (decreases FRC means that induction is faster)
- B. Obesity – Yes, probably by the changes in FRC
- C. pCO₂ – No effect
- D. Cardiac output – Yes, this does
- E. ?

Alt version: Regarding the time constant for volatile anaesthetic uptake in the lungs

- A. Affected by agent concentration - Yes
- B. Affected by obesity - Yes
- C. Not affected by FRC – No, decreased FRC reduces the time constant
- D. Affected by restrictive lung disease - Yes

IN24 [i] 22g of Nitrous oxide at STP occupies a volume of:

- A. 3.6 L
- B. 11.2 L – Correct!
- C. 22 L (? or 22.4 L)
- D. 44.1 L

Here is the equation in case you're REALLY interested... ☺

$$\begin{aligned}V &= m^3 \\P &= 101.3kPa \\T &= 273K \\n &= 0.5mol \\R &= 8.31 J K^{-1} mol^{-1} \\V &= \frac{nRT}{P} = \frac{0.5 \times 8.31 \times 273}{101325} = 0.0112m^3 = 11.2l\end{aligned}$$

IN25 [j] Wash in (? washout) of volatile anaesthetics is reduced in neonates because:

- A. Reduced FRC – No, there FRC is proportionately the same as an adult
- B. Increased cardiac index – Yes, slows the rate of rise of FA/FI
- C. Decreased plasma protein levels? – No, nothing to do with this
- D. (Something about blood:gas partition coefficients being different in neonate) – No, obviously

Alt version which probably is the same question remembered differently:

The washout of inhalational anaesthetics

- A.. Increases with elimination by the liver - No
- B.. Related considerably with the duration of anaesthesia – Yes (tissue uptake & release - notably from animal studies)
- C. Increases in the neonates compared to an adult – No, decreased due to the increased CO

IN26 [l] With regard to compound A:

- A. Increased production in Baralyme compared to sodalime – Yes, (Baralyme™ contains Barium octahydrate, CaOH, KOH – Sodalime contains NaOH)
- B. More likely in children – No, it is related to the CO₂ absorber not the patient
- C. Sevofluranes metabolites cause hepatotoxicity – No, no evidence to support this (no change in LFTs either)
- D. Sevoflurane is METABOLISED to Compound A in the liver - No
- E. ?

IN27 [l] Concerning the effects of various volatile agents on cerebral blood flow under conditions of 1

MAC and normocarbida:

- A. Halothane produces greater increase than enflurane – Yes (see Fig 2.4 in Stoelting)
- B. Isoflurane produces greater increase than enflurane – No, other way around
- C. Any change produced depends upon cerebral metabolic rate – No independent of this
- D. Change in CBF is due to change in cardiac output – No, not at all
- E.

IN28 [I] Which of the following drugs is not associated with EEG epileptiform activity

- A. Propofol – No, there is no evidence of epileptiform activity (therefore the 'correct' answer)
- B. Enflurane – Yes, hence the need to 'discover' isoflurane
- C. ?
- D. ?
- E. ?

General Anaesthetics - Intravenous

IV01 [acd] Propofol:

- A. Has a pKa of 7 – No, pH is 7
- B. Has a pH of 11 – No, pKa is 11
- C. Causes hypotension due to myocardial depression – Direct myocardial effects at HIGH doses. Normally, hypotension mainly by reducing sympathetic vasoconstriction and indirect effects on the heart (blunting reflex tachy – may lead to bradys)
- D. Has 98% protein binding – Yes, highly protein bound
- E. ?

IV02 [adk] Thiopentone causes a decrease in BP by:

- A. Direct decrease in myocardial contractility – No, it lacks direct effects
- B. Fall in systemic vascular resistance – Possibly, by decreased SNS vasoconstriction
- C. Decrease in venous tone – Yes, ?the main reason, venodilatation and venous pooling does occur therefore decreased VR and decreased CO & BP. There is no blunting of reflex tachy unlike propofol
- D. ?

IV03 [abdg] Ketamine:

- A. Is a direct inotrope – No, in fact it is a direct NEGATIVE inotrope which is overshadowed by its central SNS stimulation
 - B. Causes bronchodilatation – Yes, by its muscarinic effects
 - C. Less likely to see emergence delirium (?psychotomimetic effects) in ?older/?younger females – More common in women, less in children
 - D. Reduces pharyngeal secretions – No, if anything it may increase secretions (muscarinic effects)
 - E. Leaves airway reflexes reliably intact – Trick question ('reliably'), it does leave them intact, but NOT reliably (Miller)
- (See IV17 for another Ketamine Q)

IV04 [ak] With regards the action of midazolam:

- A. Ring closure occurs immediately on injection – Yes, as soon as pH >4 then the ring closes and becomes highly lipid soluble
- B. ?
- C. ?

IV05 [dghk] Propofol depresses cardiac output predominantly by:

- A. Direct depression of myocardial contractility – No, it has DIRECT cardiac effects but only at high doses... therefore not 'predominantly'
- B. Decreased SVR – Yes, by decreased SNS vasoconstriction
- C. ?
- D. ?

IV06 [dk] Methohexitone:

- A. Has a molecular weight of 285 – No... it's actually 284.29

- B. Has a melting point of 158 degrees – No idea/who cares!!
- C. A 2.5% solution is isotonic – No... I don't think it even comes as a 2.5% solution, only 1%. From what I've read concentrations > 1% should not be used IV
- D. Is yellow – No, it's a white crystalline powder (in solution pH about 11)
- E. Has 4 isomers – Yes...

IV07 [e] Benzodiazepine binding site on GABA receptor is:

- A. Near Cl⁻ channel - No
- B. Inside the channel - No
- C. Outside the channel - No
- D. On the alpha subunit – Yes, the 2 alpha subunits

IV08 [e] The drug with the largest volume of distribution at steady state is:

- A. Propofol – 98% ppb, Vd=4L/kg
- B. Midazolam – Vd=1.5L/kg
- C. Etomidate – Vd = 3L/kg
- D. Thiopentone – 85% ppb, Vd=2.5L/kg
- E. Methohexitone – 70% ppb, Vd=2.2L/kg

IV09 [f] GABA:

- A. Is the principal inhibitory neurotransmitter in the spinal cord – No. While it is present in the spinal cord it is not the principal one... Glycine is...
 - B. Barbiturates decrease the dissociation time between GABA and its receptor – No, they increase the average channel opening time by the action of GABA on GABA_A receptors. In high doses they activate the channel directly.
 - C. ??A & B types?? – There are three types currently known (A & C – 5 subunit ion channels (Cl⁻), and B – G protein linked receptor (increases K⁺ efflux & decreases/blocks Ca²⁺ influx). A & B in CNS, C in retina exclusively
 - D. ?
- (see also IV18)

IV10 [a] Propofol is structurally related to:

- A. Althesin – No, a mixture of 2 steroids in Cremophor EL
- B. Etomidate - No
- C. Ketamine - No
- D. ?
- E. None of the above - Correct

IV11 [gi] Midazolam:

- A. Water soluble at physiological pH – No, lipid soluble at pH >4
- B. Undergoes oxidative metabolism – No, extensive HYDROXYLATION (CYP3A4) in liver
- C. More lipophilic than lorazepam – No, lorazepam is more lipophilic than midazolam & diazepam
- D. Causes hypotension – Yes, therefore care in hypovolaemia (similar effect as thiopentone)
- E. Has a pKa of 7.4 (or ? 8.1) – No, pKa is 6.15 and commercial preparation pH is 3.5
- F. Causes retrograde amnesia – No, anterograde amnesia

IV12 [f] Thiopentone:

- A. Is the sulphur analogue of phenobarbitone – No, sulphur analogue of pentobarbitol
- B. Has higher protein binding than its oxy analogue – Yes, protein binding parallels lipid solubility
- C. ? 6% sodium bicarbonate – No, there is 30mg of ANHYDROUS sodium CARBONATE in an ampoule
- D. Isotonic at 2.5% concentration – Probably not, given that you can reconstitute it with water or saline...

IV13 [f] Propofol clearance is significantly increased in:

- A. Elderly – No, definitely decreased
- B. Metabolic acidosis – No effect
- C. Pregnancy – Yes, due to general enzyme induction
- D. ? (See also IN13b)

IV14 [i] Thiopentone:

- A. 100% reabsorbed in renal tubule – No, depends on urinary pH and not 100% is reabsorbed – some isn't obviously...
- B. Does not cross the placenta in significant amounts due to high plasma protein binding – No, it does (high lipid solubility)
- C. ??accumulate in the foetus – No, this is rare, despite the fact that it crosses the placenta easily. The [foetus]<<[maternal]

IV15 [j] Thiopentone:

- A. ? Tachyphylaxis if multiple administration in short period – Nope...
- B. ??

IV16 [j] Propofol:

- A. 10% eliminated unchanged – No, <0.3% excreted unchanged (due to high lipid solubility)
- B. Undergoes oxidative metabolism – No, extensive liver metabolism – phase II reactions only (1 & 4 glucuronide & 4 sulfate derivatives) – glucuronidation & sulfate conjugation
- C. Clearance depends on hepatic bloodflow – Whilst it does, I'm not sure if this is *entirely* true
- D. No effect / chronic liver disease – Correct, no effect has been demonstrated
- E. ?

IV17 [k] Ketamine:

- A. Direct acting negative isotope (“*It did say this*”) – Well... it is a direct acting negative INOTROPE
- B. ?Indirectly acts on SNS peripherally – ??Correct, it is a CENTRALLY mediated stimulation on SNS
- C. Directly on the sympathetic ganglia – No, it has CENTRAL SNS stimulating effects
- D. ?
- E. ?

Alt version: Ketamine:

- A. Is a negative isotope (“*it was isotope and not inotrope*”) – Well... it is a direct acting negative INOTROPE
- B. ?
- C. Directly stimulates autonomic ganglia - – No, it has CENTRAL SNS stimulating effects
- D. Is a competitive antagonist at NMDA receptors – No, it is a NON-competitive (irreversible) antagonist
- E. Directly stimulates alpha and beta receptors? – No, it has CENTRAL SNS stimulating effects

Comments:

[1] Both independently submitted versions of this MCQ contained a comment that one of the options was ‘negative isotope’ - Was this intended or a mistake by the examiners?

[2] Using the information contained in these 2 submitted versions, we can attempt to reconstruct the whole question as below. However, the question still does not look right: for example 3 options say ‘directly’ and only one says ‘indirect’ & the other does not use either term, so by ‘frequency analysis’, this suggests that one of A, C or E is correct. The problem with this is the College has in recent times been going through their whole MCQ Bank trying to eliminate this type of “design problem” where you can guess or narrow in towards the answer by looking at the frequency of numbers or words in the different options.

- KB 26-May-01

Reconstructed IV17:

Ketamine:

- A. Direct acting negative isotope – Well... it is a direct acting negative INOTROPE
- B. ?Indirectly acts on sympathetic nervous system peripherally – Correct, it is a centrally mediated stimulation on SNS
- C. Directly on the sympathetic ganglia – No, it has CENTRAL SNS stimulating effects
- D. Is a competitive antagonist at NMDA receptors – No, it is a NON-competitive antagonist
- E. Directly stimulates alpha and beta receptors – No, it has CENTRAL SNS stimulating effects

IV18 [l] With regard to GABA receptors: (OR: Which of the following is INCORRECT about GABA neurotransmission:)

- A. GABA-A found all over the body – No, CNS only

- B. Is an excitatory transmitter in 20% of CNS synapses – No, it is an inhibitory transmitter in approx 20%
- C. GABA-B is predominately post-synaptic – No, both pre- and post-
- D. GABA receptor located in spinal cord, medulla and rest in Cortex. – No, there are some in the retina (C)
- E. Is metabolised by deamination – No, by transamination
- F. Is metabolised by transamination by ?GABA transaminase – Correct – to succinic semialdehyde -> succinate -> TCA
- G. Stimulated by benzodiazepines – No, they just facilitate the conductance of the channel to Cl when GABA binds
- H. Opposes action of glycine – No... not always (it has inhibitory actions, like GABA, in some locations) (Above is a composite of options from two GABA questions which were on the Jul 01 paper.)

IV19 [I] Propofol

- A. Causes decreased hepatic blood flow to influence its own clearance – Yes, it can, although clearance exceeds hepatic blood flow (lung uptake, etc)
- B. Relatively low clearance in Children – No, higher clearance
- C. Has a high rate of transfer from the peripheral to the central compartment on ceasing an infusion – Not really, this would imply it has a long context-sensitive half life... so no...
- D. Has clinically significant metabolites – No, inactive hydrophilic metabolites... which are not clinically significant (including the effect on urine colour....)
- E. Elimination half-life of 5 minutes – No, between 0.5-1.5 hours (context sensitive <40 minutes)

Local Anaesthetics

LA01 [acdg] Lignocaine has a pKa of 7.9 At pH 6.9, the percentage ionised is:

- A. 1% (or 5%)
- B. 10%
- C. 50%
- D. 90% - Correct (at pH of 5.9 it would be 99% ionised)
- E. 99%

$$\frac{[BH^+]}{[B]} = 10^{(pKa - pH)}$$

(Also remembered as: With a pKa of 7.9, what percent of lignocaine is ionised at intracellular pH?)

LA02 [a] Cocaine:

- A. Blocks reuptake of dopamine and noradrenaline – Yes, and also affects 5-HT reuptake
- B. Central effects are due to noradrenaline – No, also due to its effects on nerve conduction
- C. Crosses lipid soluble membranes because its pKa is 2.8 – No, pKa = 8.5
- D. Is not metabolised by plasma pseudocholinesterase – No, it is mainly but also by hepatic metabolism
- E. Rapidly absorbed by nasal mucosa – No, peak plasma concentrations not reached for 30-40 minutes

LA03 [a] Ropivacaine:

- A. Produces greater motor block than bupivacaine – No, less of a motor block apparently... although there is NO good evidence to suggest this (either way this is incorrect)
- B. Is prepared as the R enantiomer – No, the S-enantiomer
- C. Is less lipid soluble than lignocaine – No (see below)
- D. Has the same cardiotoxicity as lignocaine – No, it dissociates from the channels more slowly and it has a lower CC:CNS ratio

Relative to procaine (1) lignocaine has a lipid solubility of 150, whereas ropivacaine has a lipid solubility of 300; the pKa's are not the sole determinant of lipid solubility (ie. Comparing unionised fractions) but they do determine the % which is in the unionised form...

?None of the above?

LA03b [ci] Ropivacaine

- A. Is a pure R isomer – No, it is the S isomer
- B. Is an isomer of bupivacaine – No, it is a separate chemical structure
- C. Provides more motor block than bupivacaine – No, less motor block
- D. Has more toxicity than bupivacaine – No, less than bupivacaine (not than levobupivacaine though)

E. Has similar physico-chemical properties to bupivacaine – Yes, and pharmacokinetics

LA03c [ef] Ropivacaine differs from bupivacaine mainly by:

- A. More motor blockade than bupivacaine – No, less
- B. Mainly affecting A beta rather than A delta fibres – No, the other way around (muscle, pain)
- C. Lower cardiac toxicity than bupivacaine – Yes, in equi-effective doses - but not levo-bupivacaine
- D. ?
- E. None of the above

LA04 [ag] Bupivacaine:

- A. Is an aminoester local anaesthetic – No, amide local anaesthetic
- B. Is formed by substituting butyl for methyl on amino group of mepivacaine – No, the other way around - but a very badly worded question, or a trick question (it is the **butyl for methyl** bit I'm talking about...)
- C. ?Less/more toxic than tetracaine – More toxic, mainly due to higher doses required (less potent) and slower metabolism
- D. Adrenaline solution contains sodium metabisulphite – Yes, they do
- E. Equipotent to etidocaine in causing motor block – No, it preferentially causes motor block compared with bupivacaine. Its onset is faster than bupivacaine, but otherwise it is similar (including cardiotoxicity)

LA05 [d] With regard to molecular weight of local anaesthetics, which is the correct sequence?

- A. Cinchocaine > bupivacaine > lignocaine > prilocaine - Correct
- B. Bupivacaine > lignocaine > cinchocaine > prilocaine
- C. Bupivacaine > lignocaine > prilocaine > cinchocaine
- D. Prilocaine > bupivacaine > cinchocaine > lignocaine
- E. Lignocaine>bupivacaine>prilocaine>cinchocaine

(see also LA09, LA10)

Prilocaine = 220

Lignocaine = 234

Procaine = 236

Ropivacaine = 274 (ie Bupivacaine minus a CH₂)

Bupivacaine = 288

Cinchocaine = 343

Cinchocaine is DIBUCAINE (a quinolone local anaesthetic used for surface anaesthesia (over the counter) & assessment of PChE quality...)

LA06 [d] Lignocaine works by:

- A. Altering Na⁺ permeability – Yes, by interfering with voltage gated Na channels
- B. Altering membrane structure
- C. Reduced Ca⁺⁺ permeability
- D. Increased K⁺ permeability
- E. Ca⁺⁺ binding to tropomyosin

LA07 [d] Lignocaine:

- A. Has ?% uptake in lung – No, there is TISSUE BINDING but not uptake as such (as with the other amide-linked LAs...)
- B. Is 24% ionised at physiological pH – No, pKa is 7.85 which is higher than 7.4 – therefore >50% has to be ionised (in fact the answer is 73%)
- C. Reduces Na⁺ conductance (?) – Yes, probably the correct answer
- D. ?

LA08 [d] Lignocaine:

- A. Has active metabolites – Yes, both MEGX and GX have local anaesthetic properties
- B. Metabolism faster in females because of progesterone – No, nor does it affect toxicity (whilst progesterone also binds to alpha-1-acid glycoprotein it appears to bind to a different part of the molecule)
- C. Metabolism is independent of liver blood flow – No, it is as this is the main site of metabolism
- D. ?

LA09 [ei] Protein binding of local anaesthetics (in decreasing order):

- A. Procaine > bupivacaine > lignocaine > prilocaine
- B. Bupivacaine > lignocaine > prilocaine > procaine – **Correct!**
- C. Prilocaine > bupivacaine > lignocaine > prilocaine
- D. Lignocaine > bupivacaine > prilocaine > procaine
- E. Bupivacaine > lignocaine > procaine > prilocaine
- F. Bupivacaine>procaine>lignocaine>prilocaine

DURATION of action is related to ppb (more binding, longer duration)... either by plasma protein binding or actually binding to neuronal membrane proteins...

Procaine = 6%
Prilocaine = 55%
Lignocaine = 70%
Bupivacaine = 98%

LA10 [e] Local anaesthetics are metabolized in the following order:

- A. Bupivacaine>ropivacaine>lignocaine>prilocaine>procaine
- B to E. (The above in different orders)

Hepatic Extraction ratios

Lignocaine = 0.65
Bupivacaine = 0.4
Ropivacaine = 0.4

LA11 [e] Saxitoxin site on sodium channel is:

- A. Inside channel – **Yes, acts within the channel, blocking it. The guanidinium ion behaves like Na. It gains access to the channel from the outside though...**
- B. Outside channel
- C. On membrane outside
- D. ?

...just a badly remembered question of utmost importance to the daily use of local anaesthetics...

LA12 [f] The site of action of benzocaine is:

- A. Same site as saxitoxin – **No, saxitoxin blocks the channel from the outside; benzocaine from the inside**
- B. Inside Na⁺ channel /OR: At the channel mouth – **Yes, inside the channel**
- C. At axoplasmic end of Na⁺ channel – **Not only axoplasmic Na channels (they are elsewhere on the neurone...)... unless they mean 'inside the channel'?**
- D. At Ca⁺⁺ channel – **No**
- E. In the cell membrane – **No**

LA13 [f] EMLA cream contains:

- A. Soluble in water at >16 degrees C – **No, insoluble molten lipophilic liquid**
- B. 20% ionised at pH ??
- C. 80% ionised at pH ??..OR.. Base contains 80% local anaesthetic - **Correct**
- D. ?? amount of ionised drug
- E. All of the above

LA14 [g] What factor (?does not) influence the peak plasma levels after epidural injection of local anaesthetic?

- A. Vasoconstrictor
- B. Natural vasoconstrictor activity of the drug
- C. Hepatic clearance
- D. Renal clearance – **Correct**

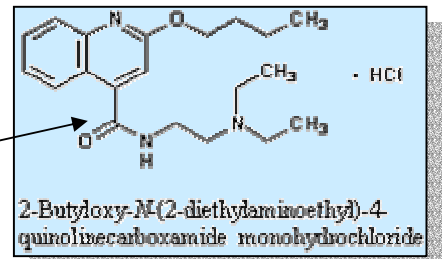
LA15 [g] Which ONE of the following is an amide?

- A. Tetracaine – **No, ester LA**
- B. Procainamide - **??? Is this a trick question???**
- C. Procaine – **No, ester LA**
- D. Prilocaine – **Yes, amide LA**

E. Cinchocaine – Yes, amide LA

LA15b [I] The following are all amides except:

- A. Bupivacaine
- B. Prilocaine
- C. Etidocaine
- D. Tetracaine – No, ester LA
- E. Dibucaine - This is a quinolone derivative but it is also an AMIDE...



LA16 [h] Lignocaine:

- A. Anti-arrhythmic effect - ??Na channel /open & inactivated state – Yes, rapidly binds to the open channel and rapidly dissociates
- B. Prolongs QRS
- C. ?
- D. ?

LA17 [hij] A solution of local anaesthetic contains 1:100,000 adrenaline. How much adrenaline has been added?

- A. 0.01%
- B. 0.1%
- C. 10 mcg/ml – Yes, 1:1000 is 1000mcg/ml or 1g/1000ml (so 1:100,000 is 1g/100,000ml)
- D. 100 mcg/ml
- E. 1000 mcg/ml

LA18 [i] Regarding the addition of adrenaline to a local anaesthetic administered epidurally, which of the following is NOT true?

- A. Significantly prolongs the duration of action of bupivacaine – No, there is no change. There is slower systemic absorption and lower peak plasma concentration though...
- B. Causes tissue acidosis at the site of injection – Yes, lactic acidosis
- C. Causes vasoconstriction - Yes

LA19 [jl] Regarding local anaesthetic plasma protein binding

- A. Is predominantly by albumin – No
- B. Is predominantly by alpha-1 acid glycoprotein – Yes...
- C. Is greater for tetracaine than for bupivacaine – No, bupivacaine 90-97% protein bound
- D. Neonates have a greater number of binding sites – No, neonates have less alpha-1 acid glycoprotein
- E. Plasma binding is directly proportional to local anaesthetic concentration – No... **DURATION OF ACTION**

(Comment: wording in option E was 'plasma binding' & not 'plasma protein binding')

LA20 [l] For a local anaesthetic agent at a given concentration:

- A. Effect is NOT dependent on resting membrane potential – No, it is
- B. Faster onset with increasing frequency of stimulation of nerve – Yes, this is frequency dependant blockade
- C. Unionised form blocks the surface receptor – No, it is the ionised form that blocks the receptor from within – the unionised form is purely to cross the plasma membrane
- D. Agent blocks the channel in the activated state – Yes, it blocks the channels when they're in the activated state...
- E. Faster onset with more negative resting membrane potential – No, the opposite is true

Muscle Relaxants & Antagonists

MR01 [ad] With regard to tetanic stimulation by a nerve stimulator:

- A. Used to determine residual curarisation – Yes
- B. Degree of fade is independent of stimulus duration – No
- C. Degree of fade is dependent on stimulus intensity – No
- D. Used to check depth of anaesthesia – No, used to check degree of muscle relaxation

MR02 [ak] Hyperkalaemia with suxamethonium is associated with:

- A. Abdominal infection – Yes (Miller)
- B. Parkinson's disease – No, nothing wrong with the NMJ (see Stoelting)
- C. Meningomyelocele – No, see Stoelting
- D. Cerebral palsy – No, see Stoelting
- E. Myotonic dystrophy - Yes

MR03 [abdeghi] Which of the following is NOT metabolised by plasma cholinesterase?

- A. Procaine – Yes, metabolised by butyrylcholinesterase (ie. PChE)
- B. Cocaine – Yes, metabolised by butyrylcholinesterase
- C. Dibucaine – No, metabolised in the liver but inhibits normal plasma cholinesterase
- D. Suxamethonium – Yes, metabolised by butyrylcholinesterase
- E. Esmolol – No, esterases in erythrocytes (possibly carbonic anhydrase), NOT plasma cholinesterase...
- F. Mivacurium – Yes, metabolised by butyrylcholinesterase

MR03b [ek] Which of the following is metabolised by plasma cholinesterase?

- A. Remifentanil – No, not plasma cholinesterase – non-specific plasma and tissue esterases
- B. Procaine - Yes
- C. Esmolol – No, erythrocyte esterase which is either the same as NMJ esterase or Carbonic Anhydrase depending on which study you believe...
- D. ?
- E. All of the above – No

Esmolol is NOT metabolised by PLASMA cholinesterase

MR03c [fi] Esterases metabolise all EXCEPT:

- A. Remifentanil
- B. Dibucaine – No, liver metabolism
- C. Pyridostigmine – No, hepatic and renal
- D. ?

MR04 [a] The action of nondepolarising neuromuscular blocking agents is PROLONGED by:

- A. Respiratory acidosis – Yes
- B. Increased temperature – No, decreased temperature
- C. Increased calcium – No, decreased calcium
- D. Increased potassium – No
- E. Decreased magnesium – No, increased magnesium

MR05 [a] Agents prolonging nondepolarising NMBA by desensitising the post-junctional membrane :

- A. Phenytoin – No, prejunctional effect
- B. Halothane – Yes, but not as much as the halogenated ethers
- C. Lignocaine
- D. Verapamil

MR06 [af] Which drugs (?competitively) inhibit acetylcholinesterase?

- A. Neostigmine
- B. Pyridostigmine
- C. Physostigmine
- D. Edrophonium
- E. All of the above – Yes, they all inhibit ACh-esterase, but if the question is COMPETITIVE/REVERSIBLE then edrophonium is probably the answer

MR06b [jk] The activity of plasma cholinesterase is decreased by the following drugs except:

- A. Neostigmine - Yes
- B. Organophosphates - Yes
- C. THA – Yes, tetrahydroaminoaridine (TACRINE) is a cholinesterase inhibitor (used in Alzheimer's)
- D. Maxalon – Yes, ?mechanism
- E. Cimetidine – Negligible effect

Is red cell esterase chemically the same as NMJ Acetylcholinesterase??
Plasma cholinesterase is butyrylcholinesterase

MR07 [cfhik] Regarding vecuronium

- A. It accumulates in renal failure – Yes, 30% excreted unchanged in renal failure
- B. Is a benzylisoquinolinium – No
- C. Is a bisquaternary amine – No, monoquaternary aminosteroid
- D. Is more lipid soluble than pancuronium – Yes, it is...
- E. Is predominantly renally excreted – No, 30% renally excreted

MR08 [dfg] In reversing neuromuscular blockade, which of the following combinations is best matched with respect to time of onset?

- A. Atropine & neostigmine
- B. Atropine & glycopyrrolate – This won't reverse neuromuscular blockade – although it has been known to be given in an effort to reverse someone! ☺
- C. Atropine & edrophonium – probably the best match
- D. Atropine & physostigmine
- E. Glycopyrrolate and edrophonium

(Comment: Option B is an unusual distractor for this question but it has been confirmed by a couple of people that this is the way it is on the paper)

MR09 [dfghj] Plasma cholinesterase:

- A. Metabolises dibucaine – No, dibucaine is metabolised in the liver. Its activity is inhibited by 80% with dibucaine
- B. Metabolises esmolol – No, this is red cell esterase
- C. Hydrolyses mivacurium at 80% the rate of suxamethonium – Yes, more like 88%
- D. Is unaffected by neostigmine – No, it is inhibited by neostigmine (hence the reason why neostigmine will prolong phase I block by suxamethonium)

MR09b [l] Suxamethonium

- A. Bigger molecule than vecuronium – No, smaller
- B. Needs to occupy 80% of nicotinic receptors to get effect – No, only enough to depolarise the membrane...
- C. Resistant to hydrolysis by acetylcholinesterase - Yes
- D. ?Is an antagonist at nicotinic receptors – No, agonist
- E. Increasing dose produces similar block – No, can get desensitisation / Phase II block

MR10 [df] With regard to the nerve stimulator in competitive blockade:

- A. Fade is dependent on stimulating frequency - Correct
- B. TOFC of four is a sign of adequate reversal – No, this doesn't take into account the amplitude or any fade
- C. ?
- D. ?

MR11 [d] Anticholinesterase agents:

- A. Carbamates duration of action is related to the time required for dissociation from the anionic site – No, duration of action related to time for the esteric site carbamylation to dissociate
- B. Carbamates act by acetylation of the esteratic site – No carbamylation at this site
- C. ?

(See also MR11b, MD28)

MR11b [jk] Carbamylation of acetylcholinesterase

- A. Ionic bonding at anionic site – This is true but not the answer to the question
- B. Ionic bonding at esteratic site – No, covalent bonding
- C. Covalent bonding at anionic site – No, ionic
- D. Covalent bonding at esteratic site - Correct
- E. None of above

(see also MR27 for similar Q)

MR12 [dgj] Mivacurium:

- A. Is metabolised at 80% the rate of suxamethonium – No, 88% (the cis-trans & trans-trans isomers)
- B. Takes 15 mins from ED95 dose to recovery of 95% twitch height
- C. Has an ED95 of 1.5 mg/kg – No, 80mcg/kg
- D. Trigger for malignant hyperthermia - No
- E. ? Duration of action is increased in renal failure – Yes, due to decreased plasma cholinesterase in renal failure

July 2000: Mivacurium:

- A. Twice the ED95 dose is 1.5mg/kg – No, 0.15mg/kg
- B. is metabolised at 80 to 90% the rate of suxamethonium - Correct
- C. After 2 x ED95 dose 95% return of twitch height after 15mins – No

MR12b [j] Mivacurium administered at a dose of 2 times the ED95 dose produces relaxation for:

- A. 10 mins
- B. 15 mins
- C. 20 mins
- D. 25 mins
- E. None of the above – Probably the most correct

While the twitch height returns to >25% of normal after 15-20 minutes there will still be relaxation for much longer than this...

MR13 [ehl] The Recovery Index 25% to 75% is 7 minutes for which drug?

- A. Vecuronium – No (10-15 minutes)
- B. Rocuronium – No (10-15 minutes)
- C. Mivacurium – Yes (6.5 minutes)
- D. Suxamethonium – No

Also recalled as: A muscle relaxant is administered at twice ED95 for a short dental case. Return of normal TOF ratio occurred at 7minutes. The muscle relaxant used was:

- A. Suxamethonium – No, all four twitches will be the same height always (except with Phase II blockade)... but this MUST be the correct answer... hmm
- B. Vecuronium
- C. Atracurium
- D. Rocuronium
- E. Mivacurium

The recovery index is the time taken to go from 25% of normal twitch height to 75% of normal twitch height...

MR14 [ej] Release of acetylcholine at the motor endplate:

- A. ?? gentamicin – No, Gentamicin certainly enhances the effects of NDMRs. It interferes with Ca on the AChR – this can be overcome by increasing extracellular calcium
- B. Botulinum toxin works by ?? – No, it blocks the release of Ach from the nerve terminals
- C. ?
- D. ?

July 2000 version: Release of acetylcholine at motor endplate:

- A. Hemicholinium directly interferes with release
- B. Only in response to action potential – No, normal miniature endplate potentials without AP
- C. Decreased by aminoglycosides / ?? prejunctional effect – Possible, they interfere with Ca interactions with the AChR and also may have presynaptic effects – can be overcome by increasing extracellular [Ca]
- D. Is Ca²⁺ dependent process – Yes, influx of Calcium required to allow vesicles to bind with membrane
- E. Always causes an action potential – No, especially with NMBs!! It causes a MEPP which if there are enough of them, an action potential...

MR15 [e] Gentamicin potentiates non-depolarising neuromuscular block by:

- A. Interfere with Ca⁺⁺ influx for exocytosis – Possibly but I have read studies which suggest it actually interferes with the binding of ACh with AChR – no good data

- B. ?
- C. ?
- D. ?

MR16 [fgil] Rocuronium:

- A. Monoquaternary at physiological pH – Yes... and every other pH!
- B. More lipid soluble than pancuronium – No, equally NON-lipid soluble!
- C. 30% metabolised (?deacetylated) in the liver – No, no metabolism in liver, excreted largely unchanged in the bile (60-70%)
- D. Rapid onset is due to its high potency – No, less potent (6x) therefore rapid onset
- E. Fastest onset is with 2 times ED95 dose – No, faster with 3x ED95
- F. Is bisquaternary – No, monoquaternary

MR17 [a] Plasma cholinesterase is inhibited 80% by 10^{-5} molar dibucaine (This is normal)

- A. In late pregnancy – Yes, the dibucaine number is a QUALITATIVE measure it does not measure the QUANTITY of plasma cholinesterase (which is in fact decreased in pregnancy)
- B. ?
- C. ?
- D. ?

MR18 [g] Which of the following do NOT prolong neuromuscular blockade?

- A. Volatile anaesthetics – Yes (prolongs)
- B. Antibiotics – Yes (prolong), some antibiotics (particularly aminoglycosides)
- C. Phenytoin – Yes, prolongs
- D. Beta-blockers – Yes, see Goodman & Gillman p203 (last paragraph)
- E. Hyperthermia – No, hypothermia does
(see also MR26)

MR19 [f] Malignant hyperthermia causes:

- A. Hypertension – No, ?mechanism
- B. Whole body rigidity - Yes
- C. Tachyphylaxis with a suxamethonium infusion – No...
- D. ?

MR20 [hl] Edrophonium:

- A. Longer half-life than neostigmine – Yes, 110 min compared to 80 min
- B. Onset slower than neostigmine – No, much more rapid (1-2 minutes compared to 7-11 minutes)
- C. ?Pyridostigmine
- D. Binds to anionic site of cholinesterase – Yes
- E. Relieves symptoms of myasthenia gravis - Yes
- F. ? Is reliable in reversing a Phase 2 block – No, probably not 'reliable' – depends on strength of block

MR20b [k] (“Edrophonium Q about elimination half times and metabolism”)

- A. ?
- B. ?

MR21 [h] . . ? . . with return of $\frac{3}{4}$ TOF ratio:

- A. ?
- B. ?
- C. ?
- D. ?
- E. ?Neostigmine may prolong the action of Mivacurium – Yes, by blocking the action of plasma cholinesterase

MR22 [hk] Atracurium:

- A. Has an active metabolite – No, laudanosine is a CNS stimulant but not at normal doses of atracurium
- B. Ester metabolism is a minor pathway of elimination – No, most is by ester hydrolysis (60%)

- C. Metabolism is by Hofmann elimination which is pH dependent ('Did not include temperature') – Yes, but does it need a temperature change?
- D. ?
- E. ?

MR23 [i] What muscle relaxant has an active metabolite with a half-life twice that of the parent compound?

- A. Rocuronium – No active metabolites
- B. Vecuronium – It does have active metabolites – apparently clearance is 3ml/kg/min cf 3-6ml/kg/min
- C. Pancuronium – No
- D. Atracurium or Cisatracurium – Laudanosine is not active at the NMJ – therefore by definition is NOT an active metabolite...
- E. None of the above

MR24 [i] Succinylcholine can cause:

- A. Bradycardia - Yes
- B. Histamine release – Yes, but not clinically important
- C. Tachycardia - Yes
- D. Hypertension - Yes, from stimulation of autonomic ganglia (as above in C)
- E. All of the above - Yes

MR25 [i] Neostigmine reversal of nondepolarising neuromuscular block

- A. Not affected by enflurane at 2 MAC – No, enflurane with enhance the block
- B. Varies depending on use of NDNMA by bolus or infusion – No... it depends on how many receptors are blocked and how much drug is left...
- C. Is/isn't affected by age – Isn't affected by age...
- D. ?

MR26 [i] Which of the following is associated with a decrease in duration or effect of nondepolarising neuromuscular blocking drugs:

- A. Volatile anaesthetic alkanes – No, increases the effect
- B. Volatile anaesthetic ethers – No, increases the effect (more than halothane)
- C. Aminoglycoside antibiotics – No, increases the effect
- D. Aminopyridine derivatives – Yes, see below...
- E. Local anaesthetic esters – No
- (see also MR18)

Alt version: Which of the following decreases the duration/depth of neuromuscular blockade?

- A. Enflurane at 2 MAC – No, increases
- B. Aminoglycosides – No, increases
- C. Bolus doses versus infusion – No, this shouldn't make much difference, just more convenient
- D. Aminopyridines – Yes, it intensifies the release of ACh and enhances the AChR sensitivity to it. Can be used to reverse block (including the effect of aminoglycosides)

MR26b [i] Neuromuscular blockade NOT prolonged by:

- A. Hyperthermia – Correct, it doesn't prolong neuromuscular blockade
- B. Gentamicin – No, prolongs
- C. Volatile agents – No, prolongs
- D. Hypothermia – No, prolongs
- E. ?

MR27 [jk] Neostigmine's mechanism of action:

- A. Binds covalently to esteric site on AChEsterase – Yes, after it has been de-esterified -> carbamylates
- B. Binds electrostatically to esteric site on AChEsterase – No, to the anionic site
- C. Binds to anionic site – Yes it does (the quaternary ammonium 'binds' here) – but only electrostatically, and not for long – once it is de-esterified it leaves
- D. Forms complex with AChEsterase with a shorter half-life than acetylcholine – No
- E. ("Some other long winded explanation requiring 30 seconds to read and impossible to remember.")

MR28 [j] With depolarising neuromuscular blocker:

A. Is competitively antagonised by NDMR

B. {"*Something about tetany & fade*"}

C. ?

D. ?

E. Shows post tetanic potentiation – No, doesn't show it – this is a feature of a Phase II Block

MR29 [j] Rocuronium administered in 2 times the ED95 dose:

A. Rapid onset, short duration

B. Rapid onset, Intermediate duration – Yes, but 3x ED95 is even better

C. Slow onset, intermediate duration

D. Slow onset, long duration

E. ("*some other combination.*")

MR30 [k] Anticholinesterase drugs

A. ?

B. ?

C. Used in treatment of Glaucoma – Yes, due to the muscarinic effects (ie miosis, making the drainage of aqueous easier)

D. ?

MR31 [k] Neostigmine:

A. Tertiary ammonium compound – No, quaternary ammonium (physostigmine is though – hence cross BBB, GIT etc)

B. ?

C. ?

MR32 [l] The dibucaine number for a normal person is:

A. 20

B. 40

C. 60

D. 80 – Yes, approximately

E. 100

MR33 [l] Muscle relaxants are less likely to cause anaphylaxis if:

A. Injected slowly – No, this isn't going to make a difference (unless it's an anaphylactoid reaction – but it would need to be very slow...!)

B. Suxamethonium is the most common cause – Possibly?

C. H1 and H2 blockers prevent anaphylaxis – No...

D. Always fatal – Not always... thank goodness

E. ?

MR34 [l] Laudanosine

A. ?

B. ?

C. ?

D. ?

E. ?

Major Analgesics/Opioids

OP01 [a] With regards to pethidine's physical properties:

A. It has an octanol coefficient of 10 – No, octanol partition coefficient is 38.8

B. It has a pKa of 8.4 – Yes, approx 8.5

C. ?

- D. ?
- E. ?

OP02 [a] Which factor does NOT predispose to bradycardia with fentanyl in doses of 50 mcg/kg?

- A. Calcium channel antagonist – No, this still will depress the baroreceptor response
- B. Beta-blocker – No, this still will further the baroreceptor response
- C. Benzodiazepines – Correct. This has no effect on the baroreceptor response
- D. ?
- E. Slow injection of drug – No, this still will depress the baroreceptor response

OP03 [aghi] Naloxone:

- A. Is not an antagonist of agonist-antagonist drugs – No, it is
- B. Is not an antagonist at μ & sigma receptors – No, it is an antagonist at ALL receptors (sigma receptors do not really exist...)
- C. Causes pulmonary oedema – Yes, it can - part of the increased SNS response (also tachycardia, hypertension & tachyarrhythmias)
- D. Can cause hypotension in experimental shock animal models – No, it has been used experimentally to improve outcome in hypovolaemic shock by increasing myocardial contractility
- E. May cause an abrupt increase in sympathetic tone – Yes, possibly due to pain following administration... not clear...

OP03b [c] Naloxone:

- A. Is effective at antagonising a full agonist but not a partial agonist - No
- B. Causes pulmonary oedema – Yes, part of the increased SNS response
- C. ?
- D. ?

OP04 [ah] {Diagram of numbered structure of morphine}

Which substitutions correct?

- A. N17 substitution gives antagonist activity – Yes, but does this also require –OH at C14?
- B. C6 methylation produces codeine – No, C3 methylation give codeine
- C. Glucuronidation occurs at C2 – No, usually at C3 & C6 (ie. Morphine-3-glucuronide & morphine-6-glucuronide)
- D. Diacetylation decreases lipid solubility – No, INCREASES it – diacetylmorphine is heroin, and this is the very reason why it works so quickly....

Also remembered as:

Morphine base structure with questions about substitutions

- A. C3 and C6 increase lipid solubility - ??? (well... diacetylmorphine (heroin) has substitutions there and it is VERY lipid soluble)
- B. Acetyl group on μ C3 gives heroine – No, needs both C3 & C6 diacetylation
- C. N- substitution gives antagonist – Possibly, but badly worded – does it not require C14 hydroxylation also?
- D. C5 glucuronidation site – No, occurs at C3 & C6 (ie. Morphine metabolites)
- E. C3 methyl gives codeine – Depends... Yes, PROVIDED that the –O– is still there (otherwise, no)

OP05 [afj] Pethidine in doses of 2 to 2.5 mg/kg causes all of the following EXCEPT:

- A. Bradycardia – No, it doesn't cause bradycardia due to atropine-like effects
- B. Decreased systemic vascular resistance – Yes, due to vasodilatation
- C. μ Normal arterial BP / μ decreased BP – Usually decreased
- D. Increased cardiac output – No, probably decreased

OP06 [a] Regarding the clearance of morphine:

- A. Affected by cirrhosis
- B. Affected by hepatic blood flow – Yes, metabolism close to hepatic blood flow (high hepatic ER)...
- C. Shows low hepatic extraction ratio
- D. ?
- E. ?

OP07 [dghj] Fentanyl:

- A. With pKa 8.4 is 90% ionised at physiological pH – Yes, 10:1 ratio of BH⁺:B
- B. Has an octanol coefficient of 10 – No, 813
- C. Is 1,000 times more potent than morphine – No, 100x (morphine – 0.1mg/kg ; fentanyl 1mcg/kg)
- D. Has first-pass lung uptake reduced to 20% by propranolol
- E. Has up to 50% uptake in the lung – No, 75% uptake (on first pass)
- F. Elimination half-life < 2 hour – No, it is longer than morphine, but larger V_d, hence short *duration*
- G. Carried on albumin mostly – No, not the MAIN protein for binding
- H. Carried on alpha-1 acid glycoprotein mostly – Correct, but albumin contributes a little
- I. Can cause hypertension with MAOI – Nope...

OP08 [d] An opioid which can not be used for TIVA:

- A. Morphine – Yes... (it IS in question OP11!)
- B. Pethidine – Yes
- C. Fentanyl – No, significant increase in context-sensitive half life when used for infusions >2 hours
- D. Sufentanil – Yes
- E. Alfentanil – Yes

OP09 [e] Nalbuphine:

- A. Works at mu receptor only – No, also at kappa & delta
- B. Has same side effects as pentazocine – Who knows...
- C. ?
- D. ?

OP10 [e] Pethidine

- A. 100mg is equal to 10mg morphine in effect – Correct, it is 10x less potent than morphine
- B. Increases heart rate – Not always, but often does
- C. No effect on cardiac output – No
- D. Is preferred to morphine for analgesia – No, but seems to be for obstetrics and biliary colic... despite there being NO good evidence for this at all!
- E. ?

OP10b [e] Pethidine produces:

- A. Miosis – Yes... but I've read that it often causes mydriasis (no reference unfortunately)
- B. More severe hypotension with comparable dose of morphine – No
- C. More biliary spasm than morphine – No, often less due to atropine-like effects but ?significant... probably not...
- D. ?

OP11 [e] TIVA with morphine causes the following EXCEPT:

- A. Mydriasis – No, usually miosis
- B. Muscle rigidity – Yes, possibly by blocking GABAergic inhibitory neurons – can make ventilation difficult
- C. Respiratory depression – Yes, it causes dose-dependent depression of ventilation
- D. ?

OP12 [ef] Codeine:

- A. Substitution at C6 position of morphine – No, C3 methylation
- B. 10% of codeine is metabolised to diacetyl morphine – No, 10% is metabolised to morphine. Large proportion is N-demethylated to nor-codeine (inactive)
- C. IM 100mg is equivalent to 10 mg morphine – Possibly, although Stoelting states that maximum analgesic dose is 60mg (but 10% of 100mg would liberate 10mg morphine from the codeine anyway...)
- D. Methyl substitution at the ?C5/?C6 position of morphine – No, C3 methylation
- E. Can be safely given IV because causes no histamine release – No, the exact OPPOSITE – it can't be given safely because of a large histamine release
- F. Has higher first pass effect than morphine – No, lower – hence it can be given orally

OP13 [f] Morphine metabolism:

- A. Principally metabolised to morphine-6-glucuronide – No, major metabolite is morphine-3-glucuronide (85%) which is inactive
- B. Metabolites have shorter half-life – No, longer elimination half-lives
- C. Found in extrahepatic sites – Yes, both liver and kidney glucuronidation (mainly liver though)
- D. Metabolites freely cross the blood-brain barrier – Yes, especially in high concentrations, but not as 'freely' as morphine...
- E. ?All have analgesic effect / ? Are 30% renally excreted – No, only morphine-6-glucuronide (10% metabolite) has analgesic effect
- F. In neonates, predominantly by sulphation – Possibly, sulphation is important compared to glucuronidation... but 'predominantly' ??
- G. In adults, mostly to morphine-3-glucuronide – Yes, 85%

OP14 [f] Buprenorphine:

- A. Effective orally – Yes, highly lipid soluble partial mu agonist and 30x as potent as morphine
- B. ?
- C. ?

OP15 [gi] Sufentanil:

- A. 30 times as potent as fentanyl – No, approx 10x as potent as fentanyl
- B. < 7% excreted unchanged in urine – No, 60% excreted in the urine
- C. Greater protein binding than fentanyl – Yes, fentanyl is only 60-80% protein bound
- D. Half-life of elimination between fentanyl & alfentanil – Yes, 2-3 hours (cf 3-4 & 1.5 hours)
- E. Predominantly bound by ?albumin/ ? alpha1-acid glycoprotein – Yes, 93% bound to alpha-1-acid-glycoprotein

OP16 [gj] Pethidine is the traditionally favoured opioid in obstetrics because:

- A. Norpethidine does not cross the placenta
- B. Does not undergo ion trapping
- C. Causes less neonatal depression
- D. It does not cross the placenta
- E. It is thought to cause less respiratory depression in the neonate. - Is this the correct answer... although the whole argument is bollocks...

OP17 [g] Pethidine:

- A. Better bioavailability than codeine – No, pethidine BA=50%, Codeine is 60%
- B. ?
- C. ?
- D. ?

OP18 [h] Pethidine:

- A. Norpethidine metabolite – Yes, 90% metabolised to this
- B. Pethidine 6-glucuronide – No, does this even exist?
- C. ?

OP19 [j] Alfentanil is more lipid soluble than fentanyl because:

- A. Has a pKa of 8.4 & is 90% unionized at physiological pH – No, the pKa is more like 6.4, but 90% IS INDEED unionised at physiological pH... ?incorrectly remembered question
- B. ?"n-Octanol coefficient is [some five digit number]." – Don't know what it's partition coefficient is...150
- C. ?
- D. ?

OP19b [l] Alfentanil works faster than fentanyl because:

- A. More lipid soluble – No, it is actually less potent than fentanyl (5-10x less potent)
- B. Higher concentration unionised at physiological pH – Correct, pKa is approx 6.4 – 90% unionised at physiological pH
- C. ?
- D. ?
- E. ?

OP20 [jk] Methadone:

- A. Phenanthrene derivative – No, this is the 'nucleus' of the morphine structure (the 4 rings)
- B. ?metabolism – Slow liver metabolism to inactive metabolites (elimination half-life – 35 hours)
- C. Peak plasma levels at 3 hours – Yes, peak plasma concentration (after oral) at about 4 hours
- D. Used in chronic cancer pain due to non addictive potential – No, that's not why it's used – more likely due to its long duration of action
- E. ?d & l isomers – No, it is not a racemic mixture

OP21 [k] Tramadol:

- A. Has beta blocking properties – No it doesn't
- B. Blocks noradrenaline reuptake – Yes, correct. It also blocks 5-HT reuptake and facilitates its release
- C. Has greater opioid activity than morphine (OR: As potent a mu agonist as morphine) – No, weak mu effects
- D. Is directly inhibited by yohimbine – No, yohimbine is an alpha-2-antagonist, thereby increasing the release of NA – this would potentiate the effects of tramadol on monoamine release...
- E. Only the +ve enantiomer is active – No, both isomers are 'active' but only one is for analgesia

OP22 [l] The most unlikely thing to occur with morphine administered in recovery is:

- A. Constipation
- B. Respiratory depression
- C. Sedation
- D. Nausea and vomiting
- E. Physical dependence – Yes, not likely at all...
- F. Pruritis

OP23 -Deleted

OP24 [l] Extrahepatic de-esterification of Remifentanyl

- A Occurs in RBC – Yes, possibly
 - B By Plasma Cholinesterase – No, it has shown to be independent of this
 - C NOT in incubated blood – No, it is still possible but may be slower
 - D Has (?mean) clearance less than 1L/min – No, clearance is a whopping 3L/min
 - E Has an active metabolite – Yes, but 300-1000x less potent...
- Alt options:
- C. Hydrolysis does not occur in vitro in incubated blood – No, it does, just not as rapidly - ?depends on temp
 - E. The drug is hydrolysed to an active metabolite which undergoes further hydrolysis – Yes, but 300-1000x less potent...
- (Q75 Jul01)

OP25 [l] The following are metabolites of morphine except:

- A. Morphine-6-glucuronide – Yes, active metabolite – 10% formed
- B. Morphine-3-glucuronide – Yes, this is an inactive metabolite – 85% of morphine metabolised to this
- C. Normorphone – Yes, approx 5%
- D. Codeine – Yes, a small amount may be formed
- E. Hydromorphone - No

OP26 [l] Fentanyl given at dose of 50-150 mcg/kg:

- A. Causes potent cardiac depression – No, it lacks direct myocardial depression & histamine release
- B. Does not cause muscle rigidity – No, it can potentially
- C. Has an elimination half-time of more than 3 hours – Yes, 3-4 hours
- D. Not enough to relieve the stress response to surgery – No, it may be enough to relieve this (but not completely abolish it)
- E. Preserve cardiac output – No, bradycardias not uncommon

Anticholinergics/Antimuscarinics

AC01 [defgh] Glycopyrrolate:

- A. Has mandelic acid rather than tropic acid – True, both atropine & scopolamine are derived from tropic acid, whereas glycopyrrolate is derived from mandelic acid
- B. Tertiary amine – No, it is a quaternary ammonium (therefore doesn't cross lipid barriers easily)
- C. ?
- D. ?

(Also: see MR08)

AC02 [fgj] Hyoscine:

- A. ?
- B. Quaternary ammonium compound – No, hyoscine (scopolamine) is a tertiary amine
- C. ?
- D. Causes mydriasis – Yes, can cause cycloplegia & mydriasis
- E. Causes confusion in the elderly – Yes, but it can occur at any age

AC03 [hi] Scopolamine d & l isomers:

- A. d is active – No, L-isomer is the active one
- B. Provided as racemic product – Yes, both atropine & scopolamine are racemic mixtures
- C. Doesn't cause central effects – No, they do (tertiary amine)
- D. ?

AC04 [j] Atropine:

- A. ?
- B. Increases anatomical & alveolar dead space – It does increase anatomical dead space but ALVEOLAR??
- C. ?
- D. ?

AC05 [l] Atropine & glycopyrrolate:

- A. Both are naturally occurring – No, glycopyrrolate is semisynthetic
- B. Cause confusion in the elderly – Yes, IV glycopyrrolate has been shown to cause an 'anticholinergic syndrome' in the elderly
- C. ?
- D. ?
- E. ?

Psychotherapeutic Drugs

PS01 [af] Benzodiazepines:

- A. Are all lipid soluble (OR: None are water-soluble) – No
- B. Are all renally excreted unchanged – No
- C. Causes retrograde amnesia – No, anterograde amnesia
- D. Lorazepam is more lipophilic than midazolam – No, less (midazolam and diazepam are similar)
- E. Block GABA receptors – No, bind to GABA receptors, increasing their affinity for GABA
- F. Have high therapeutic index - Correct

PS02 [cdh] Which is TRUE regarding monoamine oxidase inhibitors (MAOI)?

- A. Should/must be ceased for two weeks prior to general anaesthesia – No, just be mindful of interactions
- B. Cause hypotension and sedation in combination with pethidine – Yes & No... initially CNS stimulation & hypertension followed by hypotension and eventually coma, but no sedation
- C. Inhibit activity of indirect sympathomimetics – No, they augment their effects... dramatically
- D. Ingested tyramine causes hypertension due to indirect effects - Correct
- E. Includes doxepin and amitriptyline – No...

PS03 [dfj] Neuroleptic malignant syndrome:

- A. Occurs only with chronic use – No, can occur with a single dose and may be delayed
- B. 80% (60%) mortality – No (about 20-30%)
- C. ?Treated /? not treated with dantrolene – Yes, it can be treated with dantrolene

- D. Can be caused by acute withdrawal of L-Dopa therapy – Yes, it can...
- E. Is treated with bromocriptine – Yes, it can be...

PS04 [d] Inhibitors of monoamine oxidase A

- A. Allow tyramine to enter the circulation from the gut – Correct
- B. ?
- C. ?
- D. ?

PS05 [di] Benzodiazepines:

- A. Have no analgesic effect – Correct
- B. Have an antanalgesic effect – No
- C. Have an analgesic effect – No
- D. Have dose-related analgesic and antanalgesic effects – No

PS06 [fh] The benzodiazepine with the longest elimination half-life is:

- A. Diazepam – Yes, 20-50 hours
- B. Oxazepam – 5-15 hours
- C. Temazepam – 9-10 hours
- D. Midazolam – 2-6 hours
- E. Lorazepam – 10-20 hours
- F. Flunitrazepam – 5-15 hours

PS07 [f] Fluoxetine:

- A. Inhibits noradrenaline & adrenaline uptake - No
- B. Inhibits serotonin uptake – Yes, it's an SSRI
- C. ?
- D.

PS08 [gj] Flumazenil:

- A. Formulated in propylene glycol in commercial preparation
- B. Inverse agonist
- C. Is slowly metabolised making resedation unlikely – No
- D. Does not reliably reverse sedation and resp depression (in large agonist dose ?) – No, it does... and you can always give more...
- E. Is a partial agonist at mu opioid receptors - No...
Option D has also been remembered as:
- D. May significantly reverse evidence of sedation whilst hypoxia or hypercapnia persist – No, reverses all effects
- D. Reliably reverses the sedating effects of benzodiazepines but marked respiratory depression still can occur – Correct, especially if BZD elimination slower than flumazenil

PS09 [g] Diazepam:

- A. Half-life of 5 to 10 hours – No, much longer (20-40 hours)
- B. Metabolised to oxazepam & temazepam /?desmethyldiazepam – Yes, yes & yes...
- C. ?
- D. ?

PS10 [gh] Droperidol:

- A. Substituted phenothiazine – No, it's a butyrophenone
 - B. Reliably produces mental tranquillity – No, dissociative...
 - C. Does not act (directly) on CTZ – No, it does act directly here
 - D. Alpha-blockade with hypotension is not a problem with 2mg dose – No, could be a big problem in shocked patients...
 - E. Slows alpha rhythm on EEG – Yes...
- (Note: Mar 99 paper had 2 questions on droperidol)

PS11 [g] Monoamine oxidase inhibitors (MAOI):

- A. Moclobemide is a reversible inhibitor – Correct
- B. Interacts with tyramine to cause hypertension – No, it doesn't 'interact with' tyramine...
- C. Interacts with pethidine to cause hypothermia - No
- D. ?

PS12 [hk] Metabolites of diazepam, all EXCEPT:

- A. Temazepam – Yes
- B. Oxazepam – Yes
- C. Desmethyldiazepam – Yes
- D. Lorazepam – No...

[Comment: The main metabolic pathway for diazepam is diazepam -> desmethyldiazepam (active with long half-life) -> oxazepam (active) -> glucuronide conjugate (inactive, excreted). There is also a minor pathway diazepam -> temazepam (active, short half-life) -> glucuronide conjugate (inactive, excreted) Kerry 25-May-01]

PS13 [jj] With respect to action of midazolam:

- A. Acts on GABA-B receptors – No, GABA-A
- B. increases duration of opening of Cl⁻ channels – No, increases the affinity of the receptor for GABA
- C. ? competes with barbiturates for receptor site on GABA receptor – No, different location on receptor
- D. Metabolism is decreased by cimetidine – No, no effect (different enzyme) – but it does affect diazepam & desmethyldiazepam metabolism
- E. Decreases chloride conductance – No, increases (hence producing more IPSPs)
- F. Interacts with the B1 subunit of GABA – No, Benzodiazepine recognition sites are only found in GABAA receptor complexes containing $\gamma 2$ or $\gamma 3$ subunits along with α and β

PS14 [jj] Benzodiazepines - which statement is true ?

- A. ?
- B. Midazolam has ?active / ?inactive metabolites – Correct (if 'active')The primary metabolite of midazolam, 1- α -hydroxymidazolam, is at least as potent as midazolam with a 20% affinity for the benzodiazepine receptor. It has a half-life of approximately 1 hour.
- C. ?
- D. All depend on hepatic clearance – Correct?

PS15 [jj] Tricyclic antidepressants:

- A. Do not cause sedation
- B. Formed from modification of the phenothiazine ring – Correct?
- C. Avoid anti-cholinergic effects compared to other anti-depressants – No, some (amitryptiline) have marked anticholinergic effects
- D. Does not decrease reuptake of 5HT ?at 5HT3 R
- E. Decrease CNS amine levels

PS16 [jj] Diazepam 0.1 mg/kg given orally, the percent absorption is:

- A. 100% - It is almost 100% (but nothing is really 100% - so does that make (B) better?!)
- B. 94%
- C. ?
- D. ?

Cardiovascular Drugs

CD01 [aeg] Milrinone: A. Decreases pulmonary vascular resistance

- B. Increases systemic vascular resistance - Correct
- C. Is poorly absorbed when given orally – Partly, whilst it is only given IV, it used to be given orally and caused an increased mortality in heart failure...
- D. Chronic use causes thrombocytopenia – Virtually everything seems to do this! (it is a selective phosphodiesterase inhibitor)

Alt version: Milrinone causes:

- A. Chronic use causes thrombocytopenia

- B. Pulmonary vasoconstriction - ?correct
- C. Not effective orally – No, see above
- D. ?
- E. ?

CD01b [c] Milrinone:

- A. Cannot be given orally – Correct, only IV
- B. Is a phosphodiesterase III inhibitor that decreases cyclic AMP – Yes, and no – it increases cAMP
- C. Decreases peripheral vascular resistance
- D. Increases pulmonary vascular resistance

CD01c [i] Milrinone

- A. Is structurally related to thyroid hormone – No
- B. Is arrhythmogenic
- C. Has its effects via cAMP mediated increase in intracellular Ca²⁺ - Correct
- D. Increases myocardial oxygen consumption

CD02 [a] Sodium nitrite used in cyanide toxicity:

- A. Increases methaemoglobinaemia – Yes, this then mops up the CN ions
 - B. To produce increased hepatic sulphhydryl groups – No
 - C. Increases conversion to cyanocobalamin (?hydroxycobalamin) – No
 - D. Displaces cyanide from haemoglobin – No
 - E. Enhances oxidative phosphorylation – No
- (see also CD06, CD37)

CD03 [abfhik] Ephedrine:

- A. Is resistant to metabolism by MAO – Correct
- B. Is metabolised by COMT – No, it has no OH groups on the benzene ring
- C. Action is totally indirect – No, has indirect (uptake 1) and direct effects (due to OH on beta C)
- D. Acts via direct & indirect beta effect – Not entirely, it also has alpha 1 effects
- E. Action is purely alpha agonist – No, also has beta effects (mainly beta 1)

(Alternative versions) Ephedrine:

- A. Has direct alpha actions only - No
- B. Has direct beta actions only – No
- C. Has indirect (alpha) actions only – No
- D. ?
- E. Has both indirect & direct actions on alpha & beta receptors - Correct

Ephedrine:

- A. Alpha 1 and 2 and beta 1 & 2 & 3 – No, predominantly alpha-1 and beta-1 effects
- B. More alpha than beta – No, other way around
- C. “Indirect this and direct that”
- D. “Direct this and indirect that (etc)”

CD03b (Apr 2001 version) [k] Ephedrine:

- A. Increases skeletal muscle blood flow – No, predominantly beta-1 effects.... ?any beta-2
- B. Acts only by indirect effects – No, also has direct effects

Comments: The Apr 2001 paper contained 2 separate ephedrine questions - KB)

CD03c [l] (Jul 01 version) Ephedrine has:

- A. Direct agonist on alpha receptors
- B. Direct and indirect effects on alpha and beta receptors – Most correct answer
- C. Indirect actions on alpha receptors
- D. Direct actions on beta receptors
- E. Indirect actions on beta receptors

CD04 [af] The principal (?urinary) metabolite of adrenaline is:

- A. Normetanephrine
- B. Metanephrine
- C. 3,4-dihydroxy-mandelic acid
- D. 3-methoxy, 4-hydroxymandelic acid – Correct, also known as VMA (final step for adrenaline)
- E. 3-Methoxy 4-hydroxy phenylalanine

CD05 [adfgikl] Thiazide diuretics:

- A. Work mainly on PCT – No, active at the EARLY distal tubule
- B. Not effective if severely sodium depleted – No, it will still have an effect (just not AS effective)
- C. Action is independent of acid-base balance
- D. Increase GFR immediately – No, nothing is immediate!
- E. Decrease BP by decreasing contractility – No. No effect on heart directly – excessive use will result in hypovolaemia though...
- F. Cause hypoglycaemia – No, hyperglycaemia is a side effect (?mechanism)
- G. Interferes with kidney concentrating mechanisms – No, no effect on medullary gradients
- H. Causes hypocalcaemia – No, there is no net change in lumen potential (since Na & Cl remain) – therefore there is no change in Ca or Mg
- I. Used to treat hypercalcaemia – No, no effect
- J. Potentiate hyperglycaemia – Yes...?mechanism
- K. Are effective as antihypertensives by decreasing cardiac output – No...
(Multiple options remembered so possibly an amalgam of 2 questions)

MCQ-17 on July 2001 paper:

Thiazide Diuretics:-

- A. Increase calcium excretion in the urine. – No effect on Calcium
- B. Decreased efficacy in sodium depletion. – Correct. Less Na filtered then there is less available to the tubules anyway...
- C. Main site of action is the proximal tubule. – No, early distal tubule
- D. Cause equivalent amount of diuresis to frusemide – No, less effective
- E. ?

CD06 [a] Sodium nitroprusside in healthy patient:

- A. Decreases venous more than arterial resistance – No, the equal decrease in vascular tone MUST mean that arterial resistance is decreased to a greater extent than venous
- B. Has no effect on control of pulmonary vascular resistance – No, it does
- C. Decreases cerebral blood flow – No, increases
- D. Causes uterine relaxation – Correct
- E. Does not inhibit hypoxic pulmonary vasoconstriction – No, it does decrease HPV

CD07 [acdefhik] Which one of the following statements about clonidine is correct?

- A. Increase MAC requirements – No, decreases MAC values (by up to 50%)
- B. Cause transient hypertension with IV administration – Correct, however....
- C. With IV bolus causes hyper- then hypo-tension – Correct (but 'more correct' than (B))
- D. Causes hypotension immediately – No, not immediately
- E. Is not (?administered/absorbed) transdermally – No, it IS available as a patch
(see also CD12, CD36)

CD08 [cg] Regarding Digoxin:

- A. The aglycone portion causes the cardiac effects – No (see Stoelting)
- B. The glycone portion causes the cardiac effects – Yes, the glycone portion enables it to bind to myocardium increasing the local concentration
- C. ?
- D. ?

CD09 [ch] Digoxin:

- A. Decreases ventricular response due to vagal stimulation in AF – Yes, increases vagal tone

- B. Decreases myocardial oxygen consumption – No, increases
- C. Increases the R-T interval – No, decreases the QT interval
- D. Decreases AV conduction – Yes, hence its use for slowing rapid AF

CD10 [djk] Which of the following ECG changes would be most likely in digoxin toxicity:

- A. Increased PR interval - Yes
- B. Increased QT interval – No
- C. Peaked T waves – No, often flattening/inversion
- D. ST elevation – No, depression and downsloping (reverse tick)
- E. Ventricular extrasystoles - No

July 2000 version: Digoxin toxicity:

- A. Inverted T waves – No
- B. Prolonged PR interval – Yes, mainly
- C. Xanthopsia – Yes
- D. Prolonged PT interval – No

CD11 [df] Regarding digoxin overdose/toxicity:

- A. Serum level > 2.1 ng/ml is toxic – No, 0.5 – 2.5 is therapeutic. Above 3.0 is toxic
- B. Yellow vision – Yes
- C. Causes a long PR interval – Yes
- D. Causes xanthopsia – Yes
- E. Causes a long QT interval and bigeminy – No

CD12 [d] Clonidine:

- A. Elimination half-life of 3 hours (or 3 to 6 hrs) – No, 9 hours
- B. Excreted 50% unchanged in the urine (or 20% renally excreted) – Yes, according to Stoelting
- C. Oral bioavailability 50% - No, 70-95%
- D. Cannot be absorbed topically – No, it is available as a patch
- E. Is highly protein bound – No

CD13 -Deleted- same Q as CD05

CD14 [dfj] Adenosine:

- A. Slows conduction velocity and increases refractory period – No, slows conduction velocity but decreases refractory period
- B. Is metabolised in plasma – No, metabolised INSIDE red cells and vascular endothelium
- C. Decreases urate levels – No effect on urate levels
- D. Methylxanthines increase response - No, patients on methylxanthines are resistant to its effects (see also CD34)

CD15 [dh] Catecholamine substitution:

- A. Alpha carbon CH₃ substitution gives beta selectivity – No, it is the N substitution that determines this
- B. Beta-hydroxy substitution gives increased affinity – Possibly correct. The beta-OH substitution determines the POTENCY of the DIRECT actions
- C. D-dobutamine antagonist, L-dobutamine agonist – No, clinically used solutions are racemic mixtures. With respect to alpha effects (-) Dobutamine is a potent **partial** alpha agonist, whereas (+) Dobutamine lacks much alpha activity but has 7 times the beta agonist effects... (interestingly the AFFINITY for alpha receptors is identical though)
- D. ?

CD16 [abdf] Esmolol:

- A. Active at beta-1 & beta-2 receptors – Yes, only beta-1 selective at LOW doses
- B. Half-life < 2 minutes – No, elimination half life approx 9 minutes
- C. Has methanol as a metabolite – Yes
- D. Is metabolised by (?acetyl/?plasma) cholinesterase – No, by non-specific plasma esterases
- E. Is excreted unchanged in the urine – Yes, but only <1%
- F. Is a non-selective beta-1 receptor antagonist – Yes... and no...??

CD17 [dfghl] Osmotic diuretics (?Mannitol):

- A. Less sodium delivered to distal tubule – No, more but concentration is lower
 - B. Hypotonic medulla – No, isotonic with plasma
 - C. Increased sodium loss – Correct
 - D. Urine osmolality > plasma osmolality – No, they are the same
 - E. Increased sodium reabsorption / ?causes hyponatraemia – No, decreased sodium reabsorption by effectively diluting the sodium filtered
 - F. ?MW greater than 600 – No, the osmotic effect is dependant on particle NUMBER not just their size!
 - G. Washes out the medullary interstitial gradient - Correct
- (see also MD07)

MCQ-16 on July 2001 paper:

Osmotic diuretics:

- A. Include mannitol and the dextrans – No, mannitol and urea
- B. Wash out the medullary osmotic gradient – Correct
- C. Cause sodium retention – No, sodium loss
- D. ?
- E. Have a molecular weight >600 – No, the osmotic effect is dependant on particle NUMBER not size!

CD18 [d] Guanethidine:

- A. Causes sedation as a side effect – No, it doesn't cross the BBB (unlike reserpine)
- B. Postural hypotension occurs – Yes, commonly
- C. Decreases reuptake of catechols presynaptically – Yes, it displaces NA from vesicles presynaptically AND decreases their reuptake
- D. ?

CD18b [fl] (Q24 on Jul01 paper) Guanethidine:

- A. Acts primarily at/on? the CNS – No, peripherally on sympathetic nerves
- B. Produces anti-hypertensive effect primarily by presynaptically inhibiting release of noradrenaline – Yes, it displaces NA from vesicles presynaptically AND decreases their reuptake
- C. Highly lipid soluble – No, doesn't cross BBB
- D. Mental depression is a troublesome side effect – No, doesn't cross BBB
- E. Orthostatic hypotension is not a prominent side effect – No, it IS prominent

CD19 [dh] Labetalol:

- A. Alpha agonist & beta agonist – No
- B. Alpha agonist & beta antagonist – No
- C. Alpha antagonist & beta antagonist – Yes, selective competitive postsynaptic alpha-1 blockade AND beta blockade
- D. Is a more potent alpha blocker than phenoxybenzamine – No
- E. Alpha > beta effect – No

CD20 [efhik] Frusemide:

- A. 30% plasma protein binding – No, 98% plasma protein bound
- B. ??% absorption – oral bioavailability is about 60%
- C. Elimination half-life less than one hour – Yes, 30 minutes
- D. Promotes active secretion – No, it simply inhibits the Na/K/2Cl-cotransporter (secondary active transport)
- E. Affects the uricosuric effect of probenecid
- F. Effects not decreased until large decrease in GFR
- G. Causes a diuresis which is dependant on GFR over a wide range

Apr 2001 version: Frusemide

- A. Has 30% (?35%) protein binding – No, 98%
- B. Has an elimination half-life less than 1 hour – Yes, 30 minutes
- C. 90% excreted in bile – No, bile loss is more like 5% (80% is excreted unchanged in urine)
- D. Increases rate of secretion in the renal tubules – No, nothing to do with secretion

CD20b [j] Frusemide does NOT cause:

- A. Hyponatremia – Yes, it does
- B. Hypokalemia – Yes, it does
- C. Hypouricemia – No, it causes HYPURicaemia
- D. Hypomagnesemia – Yes, it does
- E. Hypocalcemia – Yes, it does

CD21 [ef] The antiarrhythmic effect of lignocaine:

- A. Because it increases the refractoriness of in cardiac muscle – No, minimal effect on normal cardiac muscle
- B. Therapeutic level 2-5ng/ml – No, 2-6 mcg/ml
- C. ?

CD22 [f] The effects of beta blockers – the following is not true

- A. Relax uterine muscle – True, relaxes the uterus (Sasada & Smith)
- B. Increased AV conduction – No, decreases AV conduction
- C. Decreased lipolysis - True
- D. Increased SVR – True (unopposed alpha)
- E. Mask hypoglycaemia – Yes, it may mask the effects of hypoglycaemia (or even cause it - ?debatable)

CD23 [abjk] Phentolamine:

- A. Is a selective alpha-1 antagonist – No, prazosin is selective
- B. Binds covalently to the alpha receptor – No, phenoxybenzamine does
- C. Causes bradycardia – No, reflex tachycardia due to alpha-2 block
- D. Is a selective alpha-2 antagonist – No, Yohimbine is
- E. Increases cardiac output – Yes

CD24 [ai] A non-selective beta-blocker with low extraction ratio, long half-life and ISA:

- A. Atenolol – No ISA (intrinsic sympathomimetic activity)
- B. Propranolol – No ISA
- C. Metoprolol – No, ISA
- D. Labetolol – Yes, weak ISA
- E. ?

CD25 -Deleted - same Q as CD05

CD26 [fg] Sotalol:

- A. Non-selective beta-blocker
- B. Contraindicated in long QT
- C. Does . ? . to ?K current
- D. Used in the treatment of torsades – No, it can CAUSE it
- E. Class II anti-arrhythmic drug – True

CD27 [g] Trimetaphan:

- A. Crosses the blood-blood barrier – No, a quaternary ammonium ganglion blocker
- B. Incompatible with thiopentone – Yes, it is a quaternary ammonium compound and the solution's pH is low...
- C. ?

CD28 [g] Diazoxide:

- A. Has diuretic activity – No, if anything can cause a decreased urine output
 - B. Opens ATP-dependent K channels – Yes, a direct and highly selective mitoK(ATP) channel opener
 - C. Not absorbed orally – No, it can be given orally
 - D. ?
- (a direct smooth muscle vasodilator and antihypoglycaemic agent – decreases insulin release from pancreas)

CD29 [ghjj] Phenylephrine:

- A. Metabolised by COMT – No, a synthetic NON-catecholamine
- B. Causes mydriasis – Correct, often used topically to produce pupillary dilatation
- C. Metabolised by MAO – Yes, hepatic MAO
- D. Effect lasts (?same time as/?longer than) noradrenaline – It is less potent but longer lasting the NA
- E. Acts by indirect method only – No, predominantly direct alpha-1 effects with minor indirect NA release

CD30 [f] Regarding hydrallazine:

- A. Fast acetylators have shorter half lives than slow acetylators – Yes (and lower bioavailability 30%)
- B. Acts via SNS mechanism – No, direct effect on vascular smooth muscle
- C. Slow acetylators decrease half-life – No, increase (and also have higher bioavailability – 50%)
- D. Has diuretic action – Not usually, either maintains or slightly increases renal blood flow
- E. Clearance > 50ml/kg/min
(see also CD32, CD35)

CD31 [g] Which ONE of the following beta-blockers is selective for beta-1 receptors?
(No other details)

CD32 [h] Which of the following statements about hydrallazine is (?true/false)?:

- A. Acts via alpha 1 receptors – No, mechanism is not clear (appears to interfere with Ca transport into vascular smooth muscle)
- B. ?
- C. ?
- D. ?
- E. Has a duration of action of 1-2 hours – No, 2-4 hours (if given IV – longer if oral)

CD33 [h] Concerning Dobutamine

- A. Levo has alpha 1 antagonist and beta agonist effects – No, the (-) isomer is a PARTIAL AGONIST at alpha-1
- B. Levo has partial alpha agonist effect and beta effects – Correct – partial alpha and full beta effects – see above
- C. Is a pure beta agonist – No, alpha effects too...
- D. ?

CD34 [ikl] Adenosine

- A. Causes AV block via action at A1 receptors
- B. Causes bronchoconstriction via A2 receptors
- C. Causes renal vasodilation
- D. Causes profound depression of the SA node
- E. Decreases AV transmission

(see also CD14)

CD35 [i] Mechanism of action of hydralazine

- A. Selective cerebral, coronary, renal vasodilator – No
- B. Alpha agonist – No, it has direct effects unrelated to alpha receptors
- C. None of the above – Correct... it's not selective and it is not an alpha agonist
- D. ?
(see also CD30, CD32)

CD36 [j] Clonidine:

- A. Causes hypertension and tachycardia – No, it is used to treat hypertension, but it can cause rebound hypertension if ceased abruptly or transient hypertension with IV use
- B. Causes bradycardia – Yes
- C. A single dose given orally is significantly less effective than an intravenous dose – No, effective orally
- D. Counteracts the hypertensive response in pheochromocytoma – No it is used as a test for pheos – ie. Should cause a drop in BP unless there is an active pheochromocytoma producing hypertension. It is a

CENTRALLY acting alpha-2 agonist

E. ? (see also CD07, CD12)

CD37 [j] The first sign of sodium nitroprusside toxicity is:

- A. Cyanide toxicity – Yes...
- B. Tachyphylaxis – No, this isn't a sign of toxicity
- C. Hypotension – No, this is the desired effect
- D. ? (see also CD02, CD06)

CD38 [k] Dexmedetomidine:

- A. Alpha-1 antagonist – No, alpha-2 agonist (like clonidine)
- B. ?
- C. Decrease in intraocular pressure - Correct
- D. Partial alpha2 agonist – No, full agonist
- E. Less selective than clonidine - No much more selective (1600:1 cf 200:1)

CD39 [l] Amiloride:

- A. Potassium sparing antidiuretic which blocks the aldosterone receptor – No, that's spironolactone...
Amiloride works in the absence of aldosterone
- B. Blocks luminal sodium channels in the collecting tubules – No, blocks Na resorption and K excretion in the distal tubule...
- C. Increases potassium excretion – No, it is a K sparing diuretic
- D. Is metabolised by the liver – No, it undergoes NO hepatic metabolism
- E. Has a short elimination half time – No, elimination half-time is 6-9 hours (what EXACTLY is short ...)

CD40 [i] With regard to sodium nitrite in CN toxicity:

- A. Causes MetHb – Yes, and this mops up the CN- ions
- B. Used to create more hydrocobalamin – No
- C. Used to displace CN from Hb – No
- D. Creates more sulfhydryl groups – No

CD41 [l] Methylxanthines:

- A. (Something about Ca^{++} currents) – No
- B. (Something about K^+ currents) - No
- C. Inhibit adenosine receptors – No
- D. Decrease plasma glucose level – No
- E. Cause diuresis by acting on renal tubules – Yes, but it is more complicated than just this...
- F. Physically addictive - Not by definition... although I struggle without a coffee

Endocrine Drugs

EN01 [ad] Chlorpropamide:

- A. Inhibits ADH secretion
- B. Has a short duration of action (? Half-life < 12 hrs)
- C. Increases glucose entry into cells – Correct, but indirectly due to its effects on increasing insulin output
- D. Is prolonged in renal failure - Correct

EN02 [dl] Sulphonylureas:

- A. High incidence of lactic acidosis
- B. Good in patients with depleted insulin stores
- C. Metformin & phenformin are examples
- D. Increased glucose utilisation in the peripheries - Correct
- E. Are related to sulphonamides

Jul 01 version: With regards to sulfonylureas:

- A. Work effectively if Insulin stores depleted
- B. Cause a lactic acidosis
- C. Tolbutamide, (something else), phenylformin are examples (? Spelling)
- D. Highly protein bound - Correct

E. ?

EN03 [l] Glipizide is:

- A. A biguanide
- B. Half life 4-6hrs
- C. Causes metabolic acidosis /lactic acidosis
- D. Not contraindicated in hepatic failure
- E. Highly bound to albumin - **Correct**
- F. Is ineffective in patients with low insulin stores – **Correct... possibly**

Miscellaneous Drugs

MD01 [ad] Oxytocin:

- A. Synthetised in posterior pituitary – **No, hypothalamus, secreted from posterior pituitary**
 - B. Poorly absorbed orally – **Correct, it is a peptide therefore there is no oral absorption**
 - C. Metabolised by oxytocinase in the liver – **Yes, it undergoes liver and kidney oxytocinase metabolism**
 - D. Bolus dose will increase central venous pressure – **No, decreases if anything at all**
 - E. Bolus dose will increase systemic vascular resistance – **No, decreases if anything**
- (see also EM15)

MD01b [gh] Oxytocin:

- A. Has diuretic effect – **No, slight ADH effect**
- B. Partially depolarises uterine muscle / ?effect on membrane threshold – **Probably most correct... ?real wording of question**
- C. Causes emesis – **No. Older preparations were contaminated by ergot derivatives – which cause N&V**
- D. Increases threshold of receptors for depolarisation – **No, acts directly on the receptors -> G-protein -> increases intracellular cAMP**
- E. Hypertension – **No, at high doses can cause hypotension by relaxing vascular smooth muscle**

MD01c [i] Oxytocin:

- A. Ringed octapeptide – **No linear nona-peptides with disulfide ring at one end**
- B. Effects on uterus antagonized by beta agonists – **No**
- C. ADH like effect – **Yes, only slight effect**
- D. ?

MD02 [acdfhi] Cisapride:

- A. Will increase gastric motility in the presence of atropine – **No, because despite the fact it acts on 5-HT₄ receptors (agonist) the final common step is Ach on muscarinic receptors...**
- B. Can be used to treat opioid induced gastric stasis – **Yes, apparently**
- C. Decreases/increases lower oesophageal sphincter tone (?due to atropine) – **Yes, it INCREASES it...**
- D. Decreases gastric pH – **No, no effect on gastric acid secretion**
- E. Increases gastric volume – **No, if anything it would decrease it**
- F. Blocks histamine receptors – **No, it is a 5-HT₄ receptor agonist**
- G. Agonist at D₂ receptors – **No, as above**

MD03 [adf] Regarding the plasma half-life of heparin:

- 7A. Clearance affected by warfarin – **No**
- B. Depends on site of injection – **Not really**
- C. Less for low MW heparins – **No**
- D. Depends on dose given - **Yes**

MD03b [d] Heparin:

- A. Has a half life dependent on dose – **Yes, 60 minutes for 100U/kg and 150 minutes for 400U/kg**
 - B. Inactivates factors XII, XI, X, IX – **No, it enhances the protease activity of antithrombin III on the ACTIVATED form of the factors listed here**
 - C. ?
 - D. ?
- (see also MD49)

MD04 [ahk] Paracetamol:

- A. Has an active metabolite – No, it has metabolites which cause liver toxicity or have been implicated in renal disease, but they're not 'active' in the true sense of the word
- B. Interferes with renal blood flow – No, it has weak peripheral anti PG synthesis effects (compared with central effects)
- C. Does NOT cause gastric irritation – Yes, this is correct
- D. Causes methaemoglobinaemia – No. Patients with defective metabolism of phenacetin -> paracetamol (eg glucose-6-phosphate deficiency) metabolise phenacetin to metabolites which may cause methHb
- E. Maximum adult dose 4g – No, over what time frame?

Apr 2001 version: Paracetamol:

- A. Frequently causes dyspepsia (?gastric irritation) – No, does not produce gastric irritation
- B. Acid-base abnormalities common with overdose
- C. Maximum dose 4 grams in adult – No, over what time frame, etc etc – badly worded
- D. ?
- E. ?

MD04b [fgj] Paracetamol:

- A. Is a powerful anti-inflammatory agent – No, it is a WEAK anti-inflammatory (weak peripheral effects). Main effects are analgesia and antipyrexia (centrally mediated)
- B. Should never be given in a dose > 20 mg/kg to children – No, the recommended dose is 30mg/kg (this all depends on dosing frequency which is not mentioned)
- C. Increased risk of hepatic necrosis in chronic alcoholics - ????
- D. Sulphate conjugation is major metabolic pathway - ????
- E. pKa 3.5 - ????
- F. ?Glutathione conjugation – No, *p*-aminophenol & *N*-acetyl-*p*-aminobenzoquinonimine, which accumulates in renal papillae, may have oxidised metabolites which bind to tissues and deplete store of glutathione -> leads to cell necrosis

Alt version remembered from Feb 2000:

Paracetamol:

- A. Has analgesic, antipyretic and anti-inflammatory effects – Yes, but no significant anti-inflammatory effects
- B. Is metabolised to BENZOQUINONIMINE which is inactivated by conjugation to glutathione – Not quite, see above
- C. Dose should not exceed 4000mg/day in an adult – True, although you probably could safely, this is all the recommendations etc etc
- D. Gastric irritation is common – No, none at all

MD04c [jj] Paracetamol:

- A. Minimum toxic dose 8-12G/day in an adult – it depends...
- B.-E. ?

MD05 [a] Aspirin:

- A. At low doses inhibits prostacyclin – No, it doesn't inhibit prostacyclin – it inhibits its synthesis
- B. Reversibly inhibits lipoxigenase - No
- C. Irreversibly inhibits cyclooxygenase - Yes
- D. Can cause asthmatic reactions - Yes

MD06 [cdhi] Serotonin (5-HT) is most common in:

- A. Platelets
- B. Enterochromaffin cells
- C. Cerebral cortex (?neurones) – Yes, as well as limbic system, spinal cord, cerebellum & hypothalamus
- D. Pineal gland
- E. GIT
- F. Mast cells

MD07 [cdfgi] Mannitol:

- A. Metabolised in the liver – No, undergoes no metabolism at all
 - B. Half-life is proportional to GFR – No, inversely proportional
 - C. Increases Na⁺ - No, increases Na loss
 - D. Excretion is dependent on GFR – Yes, completely filtered at glomerulus (no SECRETION though...)
 - E. Urine will be hyperosmolar compared to plasma
 - F. Absorbed orally – No, no oral absorption, hence its use for constipation
 - G. Isotonic – No, hypertonic usually...
 - H. Clearance dependent on GFR – Yes...
- (see also CD17)

MD08 [cdg] Gastric drugs: Which is true?

- A. Sucralfate is a mixture of sulphated sucrose and bismuth that sits in the ulcer – No, sulphated disaccharide and aluminium
- B. Gastrin & acetylcholine directly & indirectly inhibit H⁺ secretion – No, they both stimulate acid secretion
- C. Misoprostil decreases gastric acid and causes marked constipation – Yes, and No -> diarrhoea (Goodman & Gillman)
- D. Pirenzepine is less effective than H₂ blockers – Yes, (Peck & Williams)
- E. Omeprazole reversibly inhibits proton pump – No, irreversible inhibition

MD09 [ci] A decrease in renal function might be expected with:

- A. Gentamicin – Yes, accumulates in renal cortex & can produce ATN
- B. Cis-platin – Yes, well known dose limiting side effect
- C. Busulphan – Yes, from precipitation of urates
- D. Methotrexate – Yes, may be up to 10%
- E. All of the above - Yes

MD10 [c] Thrombocytopenia is a side-effect of which of the following:

- A. Busulphan - Yes
- B. Cis-platin - Yes
- C. Methotrexate - Yes
- D. All of the above – Yes... they're all bad...

MD11 [dfh] Theophylline levels increased with:

- A. Smoking – No, decreased. Elimination is 2x as fast
- B. Phenytoin – No, decreases levels
- C. Cimetidine – Yes, increases levels
- D. ?

MD12 -renumbered EN02

MD13 [di] When a beta agonist binds to protein G (linked receptor?):

- A. There is a fall in cAMP – No, an increase in cAMP by activation of adenylate cyclase
- B. The signal is amplified 108 times – Possibly, but how did they work this out??

(Comment: Several sources indicate that the wording on the paper in July 97 was as above but this doesn't make sense as a beta-agonist does not bind directly to the G protein but to a G-protein coupled receptor)

(Comment Mar 2000: This question has now been corrected to read: "When a ligand binds to a receptor linked to a G-protein: ")

(see also EM18 in Physiol MCQs)

MD14 [dk] Dantrolene:

- A. Is a benzyl-isoquinoline derivative – No, it is a direct acting neuromuscular blocker (not an -urium) – it is a HYDANTOIN derivative (like phenytoin is)
- B. Undergoes oxidative and reductive metabolism
- C. Inhibits sodium channel activation – No, acts on the ER to prevent Ca influx (?on the ryanodine receptor)
- D. Causes a marked reduction in contractility – in what? Skeletal – yes, Cardiac – no

- E. Not effective as prophylaxis because of poor oral bioavailability – No, it is used orally (It is used as prophylaxis for MH & spasticity)
- F. Acts via ryanodine receptor – Possibly – can't find exact data

Alt version: Dantrolene:

- A. Benzylisoquinolonium – No (it has nothing to do with atracurium and the like)
- B. Undergoes hepatic and renal metabolism – No, hepatic metabolism yes, but only renal elimination of metabolites and unchanged drug – no 'metabolism' renally
- C. Profound myocardial depression – No, different physiology
- D. Poor oral bioavailability – How do you define what is poor?? It is used orally, and the dose is approx 4x the IV dose – therefore BA is ~25%

MD15 [d] Omeprazole:

- A. Irreversibly inhibits the parietal cell – Yes, it irreversibly inhibits the proton pump (not specifically mentioned in question)
- B. Acts at apical membrane of parietal side – Yes, it acts within the canaliculi of the parietal cell (on the apical side)
- C. Acts at the basolateral membrane of the parietal – No, luminal

MD16 [e] Diclofenac:

- A. Plasma protein binding is% - 99.7% plasma protein binding
- B. Percent absorption . . % - Bioavailability is 50% - ?absorption data though...
- C. Mechanism of action via increase in endorphins – No, non-selective reversible COX inhibitor with variable effects on lipoygenase
- D. ?

MD17 [ek] Regarding phenytoin

- A. Acts via blockade of Na channels and via effect on K channels – No, it has no effect on K channels – it works by enhancing Na efflux and preventing Na influx in the motor cortex – some possible Ca effects also
- B. Weak base with pKa 8.3 – No, it is a weak ACID with pKa of 8.3 – if it were a weak base, it would be 90% ionised at physiological pH!!
- C. Has active metabolites – No, inactive metabolites
- D. ?
- E. ?

MD18 [egik] Which ONE of the following decrease gastric pH?

- A. Omeprazole – No, proton pump inhibitor will decrease acid secretion, therefore INCREASE pH
- B. Famotidine – No, H₂ receptor antagonist thereby reducing acid secretion
- C. Calcium salts – Yes, if talking about circulating Calcium ('antacids' containing Ca don't though?)
- D. Misoprostil – No, it is a prostaglandin analog which would decrease acid production
- E. PGE₂ – No, this acts on Gi linked receptors which decreases acid production

July 2000 version : Which ONE of the following decreases gastric acid secretion?:

- A. ?
- B. Misoprostil – Yes, a prostaglandin analog
- C. Cisapride – No, no effect on gastric acid secretion
- D. Na citrate – No effect, it is a buffer ONLY
- E. Metoclopramide – No, no effect

Apr 2001 version: Decrease gastric pH:

- A. Calcium salts – Yes, if talking about circulating blood-borne calcium
- B. H₂ antagonists (?ranitidine) – No, increases pH
- C. Omeprazole – No, increases pH
- D. Pirenzipine – No, increases pH (it is a relatively selective M₁ receptor antagonist – also has M₄ effects)
- E. PGE₂ – No, increases pH

MD19 [fgil] NSAIDs:

- A. Exhibit no selectivity for COX 1 & 2 – No, some appear to... but looks can be deceiving
- B. Exert renal effects other than effect on afferent arterioles - Correct
- C. Cause renal toxicity separate to inhibition of prostaglandins – No, not well worded...
- D. Aspirin & ketorolac irreversibly bind COX1 & 2 – No, aspirin is irreversible but ketorolac is reversible
- E. Directly cause gastrointestinal ulceration – No, indirectly

Alt version: NSAIDs:

- A. All inhibit COX 1 – No, not all
- B. Aspirin and ketorolac inhibit COX irreversibly – No, aspirin is irreversible but ketorolac is reversible
- C. They can cause renal toxicity by mechanisms other than alterations in renal blood flow by PG mediators. – Yes, it can be by direct toxic effects of metabolites (eg paracetamol)

MD20 [fg] Irreversible cardiomyopathy can be due to: (OR: Which of the following causes dose-dependent cardiac toxicity?)

- A. Vincristine – No
- B. Bleomycin – No
- C. Danorubicin – Yes
- D. Asparaginase – No
- E. Cyclophosphamide – Yes, rarely though...
- F. All of the above

MD21 [fh] Streptokinase:

- A. Acts on circulating plasmin – No, it binds to plasminogen and forms a complex which converts plasminogen -> Plasmin (fibrinolysin)
- B. Is antagonised by aminocaproic acid (EACA) – Yes, this drug inhibits plasminogen activators and has a small effect by inactivating plasmin
- C. ?
- D. ?
- E. ?

MD22 [gk] Gastric lavage:

- A. Not useful if more than one hour has elapsed – Not necessarily – the drug may itself delay gastric emptying
 - B. In children, use normal saline instead of water – Not necessarily, although over-zealous lavage has resulted in water intoxication. Normal saline has resulted in hypernatraemia
 - C. Contraindicated if poison corrosive – Correct, also hydrocarbons with high aspiration potential
 - D. Is performed in the right lateral position – No, left lateral and head down (20 degrees – tilt-table)
 - E. Should not be performed in the unconscious – No, not a problem if airway protected
- (Comment: The restriction in unconscious patients is they should be intubated for airway protection)

MD23 [gk] Long term prednisolone 20mg/day will result in:

- A. Increased lymphocyte count – No, lowers
- B. Increased capillary permeability – No effect
- C. Metabolic alkalosis – Yes, hypokalaemic metabolic alkalosis
- D. ??glucose – causes hyperglycaemia

MD24 [g] NSAIDs cause gastric side-effects by:

- A. Direct effects on mucosa – No, hence the reason for 'enteric coated' aspirin
- B. Indirect effects - Yes
- C. ?

MD25 [g] Phenylbutazone:

- A. Interferes with heparin metabolism - No
- B. Increases warfarin plasma concentration – Yes, by displacing it from protein binding sites
- C. Decreases warfarin plasma concentration – No, increases
- D. Reduces the elimination of warfarin – No, no effect on warfarin elimination (bile and urinary excretion of metabolites), despite the fact that it does decrease urine output (increased Na resorption)

July 2000 version: Phenylbutazone's effect on the coagulation system are due to: (NSAID used for gout)

- A. Binding to albumen, displacing warfarin – Yes, also displaces oral hypoglycaemics & thyroid hormones
- B. Inhibiting warfarin metabolism - No
- C. ? some interaction with aspirin – None that I'm aware of
- D. ? effect on platelets – No effect on platelets

MD26 [fh] With respect to prednisone:

- A. Prednisone is converted to active prednisolone in the gut – Not sure WHERE exactly it is converted, but this is the most correct (also considering the alternative wording below)
- B. Prednisone 5mg is equivalent to 100mg cortisol – No, equivalent to 20mg of cortisol
- C. Betamethasone has equivalent mineralocorticoid activity – No, it lacks mineralocorticoid activity
- D. Methylprednisolone ? – Slightly more potent (4mg of 5mg prednisolone)

Alternative version of options A & E:

- A. Prednisone is converted to prednisolone after absorption from the gut. – Yes, rapid conversion
- E. Betamethasone has adrenocorticoid and mineralocorticoid activity – No, lacks mineralocorticoid activity compared with prednisolone

MD27 [fh] Aspirin:

- A. Greatest absorption is from the stomach – No, small intestine mainly, especially if gastric pH increased
- B. Peak plasma level is achieved in 30 minutes
- C. Has cross-reactivity with all NSAIDs – Yes, whilst allergy is rare there is cross-reactivity with other NSAIDs
- D. Half-life 4 hours – No, aspirin's half-life is 15-20 minutes, salicylic acid's is 2-3 hours.

July 2000 version: Aspirin:

- A. Plasma half-life 4 hrs – No, see above
- B. Peak plasma concentration within 10mins of oral administration
- C. Requires conversion to salicylic acid for activity – No, while this does occur, it is not required for activity
- D. ? is more ?? than salicylic acid
- E. Better absorption if food in stomach – No, food delays absorption
- F. Cross reactive sensitivity with all NSAIDs – Yes, see above

MD28 [f] Organophosphates:

- A. Phosphorylate the esteratic site – Yes, all of them do this
- B. Phosphorylate the anionic site – No, only some of them do this (in addition to above)
- C. ?
- D. ?

(See also MR11, MR27)

MD29 [gi] Warfarin affects:

- A. Factor XIII – (fibrin stabilising factor), Not directly BUT XIII -> XIIIa by activation by Thrombin. If prothrombin is decreased by warfarin it technically DOES affect factor XIII to a degree...
- B. Protein S – Yes, and protein C.
- C. ?

MD30 [hi] Bleomycin

- A. Related to nitrogen mustard – No, it's a chemotherapeutic antibiotic
- B. Can cause agranulocytosis (or: frequently causes myelosuppression) – No, this is in contrast to most other chemotherapeutic agents
- C. Causes pulmonary toxicity in 90% of patients – No, in approximately 4% of patients
- D. Is an alkylating agent – No, an antibiotic which fragments DNA
- E. Causes pulmonary oxygen toxicity due to production of superoxide radicals – Yes, that's the current theory

MD31 [h] Which drug causes the most anaphylaxis?

- A. Suxamethonium – Yes... interesting considering it's just to ACh joined at the hip
- B. High potency non-depolarisers – While they do, the incidence is higher with sux (used more?)
- C. ?

D. ?

MD32 [h] Syrup of Ipecac

- A. Is not effective in phenothiazine overdose – (eg. Chlorpromazine, prochlorperazine, etc) – It is not recommended for use as emesis may result in seizures. Gastric lavage is preferred...
- B. Has peripheral irritant and direct CTZ action – Yes, both direct GIT irritant and CTZ action
- C. The syrup is more potent than the fluid – No, the extract (fluid) is much more potent (14x) and should be diluted...
- D. ?

MD33 [i] Regarding antiemetics which drug has anti-5HT₃, anti-H₁ and anti-D₂ actions:

- A. Ondansetron – No, only anti-5-HT₃ effects
- B. Scopalamine – No, antimuscarinic
- C. Domperidone – No, peripheral anti-dopamine effects only
- D. Droperidol – No, D₂, NA, 5-HT and GABA effects
- E. Prochlorperazine – No, central D₂ blocking and alpha blockade
- F. Chlorpromazine – Correct

Alternative versions:

* Which of the following anti-emetics have D₂, ACh, 5 HT-3 antagonist effects?

* Which drug is a D₂ antagonist, H₁ antagonist and 5HT₃ receptor antagonist?

MD34 [hi] With regard to nitric oxide

- A. It is anaesthetic at high concentration – No, nitrous oxide can be...
- B. May improve V:Q mismatch - Yes...
- C. Is a liquid in the cylinder, gas at room temperature – No... according to the BOC gases website, it has NO liquid phase!
- D. ?

MD35 [il] Ethanol

- A. About 35% excreted via the lungs – No, partition coefficient – blood:air = 2000:1
- B. Concentration falls at a fixed rate with respect to time – No, Log [ethanol] does though
- C. Only 60% is metabolised, the remainder being excreted in expired air – No, see above
- D. Is excreted at a rate independent of the plasma concentration – No, metabolised at a rate independent of plasma concentration (zero order kinetics – a fixed AMOUNT metabolised per unit time)
- E. Constant elimination independent of plasma concentration - Correct
- F. Elimination is not dependant upon amount absorbed from GIT – No, zero order kinetics

MD36 [i] Which drugs cause convulsant activity?

- A. Cocaine – Yes, CNS local anaesthetic toxicity
- B. Lithium – Yes (it has a narrow therapeutic range)
- C. Norpethidine – Yes
- D. Enflurane – Yes, hence the reason for the development of isoflurane
- E. All of the above - Yes

MD37 [i] Metoclopramide

- A. Increases gastric emptying faster with an oral dose than an IV dose – No, it takes 3 times as long to work as IMI and 30 times as long as IV dose (which takes 1 minute)
- B. Causes diarrhoea in children – Yes, it can do this (but also in adults)
- C. Is a dopamine agonist – No, dopamine antagonist (hence effect centrally on CTZ and prolactin release)
- D. ?

MD38 [ij] Physostigmine

- A. Causes (? excitatory activity / ?alerting response) on the EEG
- B. Doesn't cross the blood brain barrier – No, it does (it is a tertiary amine anticholinesterase drug)
- C. Doesn't cause sedation
- D. Only has its effects at nicotinic receptors – No, at all ACh receptors (muscarinic & nicotinic)
- E. Causes amnesia

F. Causes excitatory activity on the EEG

G: Is/isn't a quaternary ammonium that does/doesn't cross BBB – It isn't and it does

MD39 [j] Drugs filtered and secreted in the PCT include:

- A. Penicillin – Correct
- B. Probenecid – Correct (mainly secretion at the PCT though)
- C. Chlorothiazide
- D. ?

Also remembered as:

Which basic drug is secreted by the kidney for excretion?

- A. Procainamide
- B. Probenecid – Yes...
- C. Penicillin – Yes...
- D. Acetazolamide

MD40 [j] Which of the following is bacteriostatic only?

- A. Penicillin – No, bacterocidal
 - B. Gentamicin – No, bacterocidal
 - C. Vancomycin – No, bacterocidal
 - D. Trimetophan – Errr.... Trimethoprim is bacterostatic... Trimethaphan is a ganglion blocker with peripheral alpha-blocking effects at high doses
 - E. ?Cefoxitin /?cefuroxime – No, bacterocidal
- (see also MD40)

MD41 [j] With respect to serotonergic receptor action, which ONE of the following is true?

- A. Sumatriptan is a 5HT1 antagonist – No, agonist of 5HT1 receptors (vascular) causing cranial artery vasoconstriction – relieves MIGRAINES
- B. Ondansetron is a 5HT3 agonist – No, 5HT3 receptor ANTAGONIST
- C. ?Serotonin is a 5HT3 agonist - ????
- D. Metoclopramide is a 5HT4 agonist – Yes, it is an agonist here, and an antagonist at 5HT3 receptors
- E. ?

MD42 [j] Acetazolamide:

- A. ? secreted by the renal tubules
- B. ? diuresis
- C. ? develop tachyphylaxis

MD43 [j] Best antiemetic for motion sickness:

- A. Metoclopramide
- B. Ondansetron
- C. ?
- D. ?
- E. Hyoscine – Yes, this acts by inhibiting the vestibular inputs to the vomiting centre and the vomiting centre itself...

MD44 [j] Complications of salbutamol used in asthma treatment include the following EXCEPT:

- A. Tachycardia - True
- B. Decreased V/Q mismatch - True
- C. Tremors - True
- D. Pulmonary oedema - True
- E. Hyperkalaemia – No, if anything hypokalaemia is a problem with very high doses

MD45 [k] (Antibiotic sensitivities against certain bacteria)

- A. Penicillin and ...?
- B. Amoxycillin and ...staph +?
- C. Flucloxacillin and G +ve?
- D. ?cephalosporin and ...?

MD46 [k] Aspirin overdose

- A. Causes metabolic & respiratory acidosis - **Correct**
- B. Causes metabolic & respiratory alkalosis - **No**
- C. Causes metabolic alkalosis & respiratory acidosis - **No**
- D. Causes metabolic acidosis & respiratory alkalosis - **Correct**

This is a badly worded answer. Technically A & D are correct. Initially it causes stimulation of ventilation and therefore a respiratory alkalosis, followed by a metabolic and respiratory acidosis. The metabolic acidosis is more common in children than adults

MD47 [k] Atropine overdose in neonates

- A. Causes hyperpyrexia – **Yes, can cause atropine fever (due to blocking of sympathetic postganglionic muscarinic sweat receptors)**
- B. ??

MD49 [kl] Low molecular weight heparin

- A. Has better bioavailability – **No**
- B. Molecular weight 1/10 that of normal heparin – **Yes, almost -> approximately 4000-5000 daltons**
- C. More protein bound than heparin – **No, it has LESS protein binding than unfractionated heparin**
- D. ?
- E. ?

MD50 [kl] Desmopressin

- A Increases factor X
- B Increases factor V
- C Causes sustained severe hypertension – **No, DDAVP (1-deamino-8-D-Arginine-Vasopressin)**
- D Can be used to improve haemostasis in haemophilia – **No, in haemophilia factor VIII is required (endogenous factor VIII is inactive – that's the problem)**
- E Increases factor VIII activity - **Yes**

MD51 [l] An intravenous infusion of 8.4% sodium bicarbonate to a healthy adult may cause:

- A. Hypotonicity – **No, hypertonic**
- B. Intracellular Acidosis
- C. Ionized Hypercalcaemia – **No, alkalosis causes a decrease in the ionised calcium (hence the tendency of hyperventilation – increased Ca protein binding)**
- D. ?Respiratory Alkalosis
- E. Rebound Metabolic Acidosis

MD52 [l] Cyclo-oxygenase-1 (COX-1) isoenzyme:

- A. Is increased by inflammation – **No, it is the COX-2 iso-enzyme which is**
- B. Is ?predominant mode of action of indomethacin – **No, it's the most potent non-selective NSAID**
- C. Is increased by lipopolysaccharide – **No...**
- D. Is NOT involved in gastric mucosal protection – **No, of course it is...**
- E. Is increased by cytokines – **No, COX-2 is increased by cytokines, growth factors & tumor promoters**

MD53 [l] Caffeine

- A. Is a CNS depressant – **No stimulant**
- B. Causes cerebral vasoconstriction – **Yes, it is a cerebral vasoconstrictor...**
- C. Reduces the acidity of gastric fluid secretion (or: Not a gastric irritant) – **No, increases acidity and irritates stomach**
- D. Reduces plasma glucose level – **No (in overdose causes hyperglycaemia)**
- E. Is a potent diuretic – **No, it's not that potent compared to agents specifically for that use**
- F. Has been shown to be dependence producing – **No... although...**
- G. Does not show an improvement in psychomotor function – **No, it does... apparently... keep drinking that coffee...**

Statistics

ST01 [afh] Tests that use ranking of data:

- A. Can be applied to any distribution – Most likely the correct answer
- B. Include the chi square test – No, data doesn't need to be ranked
- C. Have greater power than non-ranking tests – Not usually
- D. ?Normal distribution – Not usually

ST02 [ai] Standard error of the mean:

- A. Is proportional to N – No, inversely proportional to the square root of n
- B. Is greater for sample than SD of population – There is no standard error of the mean for a SAMPLE, only a POPULATION
- C. Measures variance within a sample – No, within a population
- D. Measures dispersion around population mean – Yes, theoretically – the population mean can't be measured...

Alt version: Standard error of the mean:

- A. Measure of sample variability – No, meant to indicate population variability
- B. Measure of difference between sample & population mean – Yes
- C. SEM > SD – No, it is smaller (it is equal to the sample mean divided by the square root of the sample size)
- D. ?

ST03 [af] Use of chi-square test inaccurate with:

- A. 2x2 contingency table – No, only if the values are >5
- B. Expected value of any cell < 5 – No, only if it only has 2x2 table
- C. Observed value in any cell < 5 – No, only if it only has 2x2 table

ST04 [d] The mean in a very large sample:

- A. Numerically greater than the standard deviation – Not necessarily (it could be zero)
- B. Is always equal to the mode – No, not if they're not normally distributed
- C. Is more than the median - No, not necessarily (requires normal distribution)
- D. Represents a normal distribution – No, not necessarily
- E. Gets larger as the sample size increases – No, should just become more 'accurate'

ST05 [e] The standard normal distribution:

- A. Standard deviation is one – Yes
- B. Mean, median & mode are the same – Not quite true...
- C. Mean is one – No, zero
- D. Mode is one – No, zero

ST06 [ej] In a study for depth of epidural catheter insertion, the mean is 4.4 and the standard deviation is 0.3 Which ONE of the following is true?

- A. If a normal distribution, 68% of values would lie between 4.1 and 4.7cm – Yes, 68.2% (assuming adequate sample size)
- B. None was greater than 5.5 cm (or ?6.8cm) – No
- C. The least distance was...?? – No, this cannot be determined from this information
- D. 99% of the sample lies within 1.96 SD of the mean – No, 95% will lie within this range (assuming adequate sample size ie. >30)
- E. 500 patients had catheters at some length. – No!

ST07 [e] Simple linear regression:

{graph of straight line crossing y axis at +3}

- A. $y = 3 + 6x$
- B. $y = 3 + 0.6x$
- C. ?
- D. None of the above – Correct... it should NOT cross the axis

ST08 [fgl] Which one of the following statements regarding the standard deviation is true?

- A. Mean +/- one SD includes 50% of values – No, 68.2%

- B. Mean +/- one SD includes 66.7% of values – No, 68.2%
- C. Mean +/- two SDs include 99% - No, just over 95%
- D. Mean +/- three SDs include 99.73% - Correct
- E. Mean +/- 1.96 SD includes 99.73% - No, 95%

ST09 [f] Ordinal data:

- A. Assumes a normal distribution – Not usually
- B. ?
- C. ?

ST10 [i] Paired t-test

- A. Assumes the normal distribution – Not really 'assumes' but it requires normally distributed data
- B. Is a nonparametric test – No, is a parametric test

ST11 [i] In a clinical trial, a patient either vomits or not. What type of data is this?

- A. Ordinal – No, may be more than one option
- B. Nominal – Yes
- C. Rational – No, not a scale with a zero point
- D. Interval – No, not a scale without a zero point

ST12 [ijk] Odds ratio:

- A. Is prevalence vs. incidence
 - B. Gives an indication of ?? in exposed vs non-exposed patients
 - ??C. Formula is Number of positive outcomes/ Number of negative outcomes – No...
 - ??C. Formula is Number of positive outcomes/ Number of possible positive outcomes – No...
 - D. Gives the prediction of a disease outcome knowing the risk factors – Probably the most correct
 - E. Gives prediction of risk factors with a known disease outcome
- The Odds Ratio is the ratio of the odds of disease for the experimental group relative to the odds of disease in the control group

ST13 [j] With respect to 95% confidence intervals:

- A. Equals mean +/- 1.96 SE – No, confidence intervals don't ONLY reflect the mean – can be other parameters... plus the sample size needs to be >30
- B. Will contain the population mean 95% of the time – No, see above
- C. Tells variability of sample – No...
- D. Tells 5% chance of finding sample result – No...
- E. Assumes a normal distribution – Yes, confidence intervals ARE normally distributed and I think this is also a 'requirement' for their use (ie data needs to be normally distributed)...

ST14 [l] Student's t-test

- A. Used to compare 2 groups – Yes and no... usually used to compare the SAME group before & after intervention (ie. A paired t-test, as opposed to an independent group (unpaired) t-test)
- B. Used if groups have different variance – No, they are required to have identical variance
- C. For small size samples – Not necessarily
- D. ?
- E. ?

ST15 [l] All of the following tests EXCEPT one, can all be used to compare two dissimilar groups:

- A. Chi square – Yes – for NOMINAL data, The chi-square test is a statistical test which computes the probability that there is no significant difference between the expected frequency of an occurrence with the observed frequency of that occurrence
- B. Mann whitney U test – Yes, the non-parametric version of the Independent Group t-test
- C. Wilcoxon signed ranks sum test – Yes, the non-parametric version of the Student's Paired t-test
- D. Spearman rank order – No, used to compare the level of association of 2 variables (non-parametric equivalent of the Pearson's Correlation coefficient)
- E. Kruskal Wallis – Yes, the non-parametric version of the ANOVA (analysis of variance test / F test)

