**Question 1**

Which anaesthetic drug is best described in the following scenerios..

This short acting muscle relaxant is eliminated by hydrolysing cholinesterases

A Succinylcholine

B Sugammadex

C Rocuronium

D Atracurium

Explanation A

This question appears an EMQ

Succinylcholine short duration of action is due to rapid hydrolysis by butyrylcholinesterase and pseudocholinesterase in the liver and plasma respectively

Extra EMQ type questions

A patient requires a rapidly acting non-depolarising muscle relaxant that is primarily metabolised by the liver= rocuronium

An anaesthetised patient has been given rocuronium, at the end of his procedure , reversal is managed with this modified cyclodextran=sugammedex

This muscle relaxant is appropriate in a patient with severe renal and hepatic impairment= atracurium

Atracurium is so extensively metabolised that its pharmacokinetic are independent of renal and hepatic function, and less than 10% is excreted unchanged by renal or biliary routes. Two separate processes are responsible for metabolism: ester hydrolysis and Hofmann elimination

**Question 2**

Your emergency department wants to buy Sugammadex. You present a CME on the important pharmacodynaics and pharmacokinetic of the drug. Which of the following is true?

A It is safe to use in patients with renal failure

B 4mg/kg is the dose required for immediate reversal of neuromuscular blocker

C It is effective in reversing both selective and non selective

D Muscle recovery with Sugammadex is faster than neostigmine

Explanation D

Sugammadex is a modified gamma cyclodextrin designed to selectively reverse the effects of the neuromuscular blockers rocuronium and vecuronium. There is evidence to suggest it is effective against pancuronium. It works by forming a complex with these drugs, reducing their availability to bind to nicotinic receptors in the neuromuscular junction

IVI administration, sugammadex has an elimination half-life of 2.2 hours. This is increased in elderly patients and decreased in children. M of the sugammadex dose is excreted unchanged in the urine, so its use in people with severe renal impairment is not recommended. Longer recovery times may be observed in older patients as well as people with cardiovascular disease, oedema or severe hepatic impairment.

If immediate reversal of rocuronium-induced blockade is required, the recommended dose is 16mg/kg of sugammadex three minutes after rocuronium administration. Currently there is no evidence to recommend sugammadex for the complete reversal of vecuronium.

4mg/kg is required for reversal of a deep block (post tetanic count 1 to 2). 2mg.kg is required for a shallow block (train of four =2). Sugammadex is more effective than neostigmine at reversing profound and shallow neuromuscular blockade

T1/2 is 2.2 hours. Following reversal of block there has been reports of recurrence of blockade. Close drug monitoring is required

**Question 3**

A patient requires ongoing muscle relaxation during a lengthy operation. His past medical includes renal and liver impairment form alcohol. Which of the non depolarising blocking drugs can be used?

A Vecuronium

B Rocuronium

C Pancuronium

D Atracurium

Explanation D

Atracurium is cleared form circulation via Hofamnn elimination-nonenzymatic and enzymatic hydrolysis of ester bonds. It is not dependent of organ function. Fever and alkalosis does increase metabolism of atracurium (Hofmann elimination increases)

Mivacurium metabolized via plasma cholinesterase and would be useful to use as well

Pancuronium, rocuronium and vecuronium rely on liver and/or kidney metabolism to clear the drug

**Question 4**

Which volatile anaesthetic is the least metabolized?

Your answer was not correct

A Isoflurane

B Sevoflurane

C Desflurane

D Halothane

Explanation C

The extent of hepatic metabolism, the order (most to least) of inhaled anaesthetic are methoxyflurane, halothane, enflurane, sevoflurane, isoflurane, desflurane, nitrous oxide. Nitrous oxide undergoes zero metabolism.

**Question 5**

Which of the following is an amide local anaesthetic agent?

A Cocaine

B Benzocaine

C Prilocaine

D Tetracaine

Explanation C

All the rest are Ester type local anaesthetics. Examples of Amide local anaesthetics are: lignociane, mepivacaine, bupivacaine, etidocaine and ropivacaine

Note- a nice way to remember it: local anaesthetics that are Esters :have just one '' i '' in their names eg procaine ,cocaine but Amides :have more than one '' i '' in their names lidocaine, bupivacaine.

**Question 6**

In pseudo (plasma) cholinesterase deficiency which of these two drugs will have a prolonged effect?

A Mivacurium and Esmolol

B Remifentanil and Esmolol

C Succinylcholine and Esmolol

D Succinylcholine and Procaine

Explanation D

Pseudocholinesterase deficiency will result in a prolonged effect of the following: succinylcholine, mivacurium, procaine, and cocaine. Iatrogenic causes of lower plasma pseudocholinesterase activity include medications such as the following:

Anticholinesterase inhibitors

Bambuterol

Chlorpromazine

Contraceptives

Cyclophosphamide

Echothiophate eye drops

Esmolol (esmolol is metabolised by esterases in the cytosol of red cells, not plasma or red cell membrane acetylcholinesterases)

Glucocorticoids

Hexafluorenium

Metoclopramide

Monoamine oxidase inhibitors

Pancuronium

Phenelzine

Tetrahydroaminacrine

**Question 7**

Prolonged duration of neuromuscular blockade is seen following a vecuronium infusion. Which of the following is NOT a possible cause?

A Severe burns

B Acidosis

C Long term steroid use

D Hypothermia

Explanation A

In rare cases, long-term use of neuromuscular blocking drugs to fascilitate mechanical ventilation in ICU settings may be associated with prolonged paralysis and/or skeletal muscle weakness. Patients may have received other drugs such as broad spectrum antibiotics, narcotics and steroids and may have severe diseases which lead to electrolyte imbalances, hypoxic episodes of varying duration, acid-base imbalance, hyperthermia and extreme debilitation any of which may enhance the actions of a neuromuscular blocking agent.

Hypothermia increases the duration of action and increases the time to recovery. Reduced clearance and rate of effect site equilibration may explain vecuronium's increased duration of action when core temperature is reduced.

**Severe burns and those with upper motor neuron disease are resistant to nondepolarising muscle relaxants.** This desensitization is probably caused by proliferation of extrajunctional receptors, which result in an increased dose requirement for the nondepolarising relaxant to block a sufficient number of receptors

**Question 8**

Which of these drugs can be used to treat central anticholinergic syndrome?

A Pyridostigmine

B Physostigmine

C Atropine

D Benztropine

Explanation B

Physositgmine is the only carbamate that is well absorbed form all sites (lungs, skin, eye, gut) and is distributed into the central nervous system.

EXTRA

Many of the drugs used in anesthesia and intensive care may cause blockade of the central cholinergic neurotransmission. Acetylcholine is of significance in modulation of the interaction among most other central transmitters. The clinical picture of the central cholinergic blockade, known as the central anticholinergic syndrome (CAS), is identical with the central symptoms of atropine intoxication. This behaviour consists of agitation including seizures, restlessness, hallucinations, disorientation or signs of depression such as stupor, coma and respiratory depression. Such disturbances may be induced by opiates, benzodiazepines, phenothiazines, butyrophenones, ketamine, etomidate, propofol, nitrous oxide, and halogenated inhalation anesthetics as well as by H2-blocking agents such as cimetidine. There is an individual predisposition for CAS--but unpredictable from laboratory findings or other signs

**Question 9**

Which of the following opioids have INACTIVE metabolites?

A Methadone

B Oxycodone

C Codeine

D Morphine

Explanation A

Morphine=hydromorphone.

Codeine= morphine

Oxycodone= oxymorphone.

Pethidine = norpethidine

Unlike codeine, morphine, hydromorphone, pethidine or oxycodone, methadone has no active metabolites and is therefore a good choice for patients at risk for toxicity from metabolite accumulation

**Question 10**

With the MAOI tranylcypromine, which drug will be least problematic?

A Ephedrine

B Propofol

C Phenylephrine

D Pethidine

Explanation B

Tranylcypromine is a MAOI, which will inhibit the catabolism of dietary amines-prevents breakdown of tyramine in the gut. When foods containing tyramine (cheese, tap beer, soy products and dried sausage) are ingested, the patient may develop a hypertensive crisis. The mechanism is poorly understood but is thought that tyramine displaces noradrenaline from the storage vesicles and enhance peripheral noradrenergic effects, including raising blood pressure dramatically. Similarly drugs with sympathommimetic properties may cause significant hypertension when combined with MAOIs. Over-the-counter preparations that contain pseudoephedrine and phenylpropanolamine are contraindicated in patients taking MAOIs. Pethidine is associated with serotonin syndrome when given with the MAOI drug group

**Question 11**

Which drug does NOT have antiemetic properties?

A Midazolam

B Ketamine

C Dexamethasone

D Ondansetron

Explanation B

Ketamine does not have antiemetic properties but is often used with propofol=Ketofol, as Propofol has antiemetic effects. The antiemetic effect of Propofol is stated in the current texts, but there is some controversy as to whether it really works as an antiemetic. See different articles reports below.

In a study reported in the clinical journal of anaesthetics it stated that many anaesthesiologists used propofol for its antiemetic effect. There is strong evidence for its antiemetic efficacy after anaesthesia maintained by a propofol infusion and also for its use in the post anaesthetic patient. However, there is little evidence to support its use purely at induction of anaesthesia or using it at the beginning or end of a case in an attempt to reduce postoperative nausea and vomiting. This is especially true in cases lasting longer than a few minutes

Other studies have reported: The group anaesthetised with propofol had significantly fewer emetic sequelae and the results suggest that propofol has a definite antiemetic action.

Online reports: Propofol is known to possess direct antiemetic effects. Propofol antiemetic use for induction and maintenance of anaesthesia has been shown to be associated with a lower incidence of postoperative nausea and vomiting (PONV) when compared to any other anaesthetic drug or technique.

**Question 12**

Which of the following inhaled gases are metabolised greater than 10%?

A Halothane

B Isoflurane

C Nitrous oxide

D Sevoflurane

Explanation A

Metabolism of inhaled anaesthetics:

Nitrous oxide 0%.

Sevoflurane 2-5%.

Isoflurane- <2%.

Halothane >40%.

Enflurane-8%.

**Question 13**

Which of the following is most likely to cause raised intracranial pressure?

A Propofol

B Diazepam

C Ketamine

D Thiopentone

Explanation C

Ketamine markedly increases cerebral blood flow, cerebral oxygen consumption and intracranial pressure. Inhaled anaesthetics decrease the metabolic rate of the brain but do increase cerebral blood flow. Thiopentone decreases cerebral metabolism, oxygen consumption and cerebral blood flow. Propofol reduces cerebral blood flow and cerebral metabolism. Benzodiazepines cerebral blood flow and ICP but to a smaller extent

Note: I have seen nitrous oxide as an option. Nitrous does increase cerebral blood flow and ICP

Extra: the latest review of ketamine in the prescribed TB states that ketamine is considered to be a vasodilator that increases cerebral blood flow as well as CMR02. Traditionally ketamine has not been used in patient with an already raised ICP. New evidence suggests that this undesired effect on cerebral blood flow may be blunted by the maintenance of normocapnia

Life in the fast lane literature review, states that there is no evidence that ketamine causes harm in traumatic brain injury and that Ketamine haemodynamic stability may actually be of benefit in TBI requiring rapid sequence induction

**Question 14**

The following cause an increase in intra-abdominal pressure?

A Suxamethonium

B Metoclopramide

C Neostigmine

D Morphine

Explanation A

In some patients especially muscular ones, the fasiculations associated with suxamethonium will cause a rise in intra gastric pressure from 5-40cmH20. The result of which may cause vomiting and aspiration. This effect is not seen with the non depolarising muscle relaxants

**Question 15**

Which of the following increase intra-ocular pressure?

A Suxamethonium

B Hypoventilation

C Halothane

D Ketamine

Explanation A

Intraocular pressure increases following administration of suxamethonium. It occurs 1 min after injection, is maximal at 2-4 min and starts to subside after 5min. The mechanism may involve contractions of tonic myofibrils or transient dilation of chorodial blood vessels. Despite this increase, the use of suxamethonium is not contraindicated unless the anterior chamber is to be opened.

Note: in the current textbook, there is no mention of Ketamine's effect on IOP. A web review states that ketamine does cause a small rise in IOP but not enough to be a concern. At dosages of 4 mg/kg or less, there are not clinically meaningful associations of ketamine with elevation of IOP.

**Question 16**

Thiopentone is a “short-lasting” barbiturate because?

A It is rapidly distributed throughout the body

B It is administrated by IV injection

C It is metabolised rapidly by brain and liver

D It is bound to the “sleep centre” in the brain

Explanation A

It is “short lasting” because of the rapid removal form brain tissue into the other highly vascularised tissues and is redistributed to muscle, fat and eventually all body tissues. It is only metabolised at a rate of 12-16% following a single dose. It facilitates the action of GABA and increase the opening of the Cl channels.

**Question 17**

Which of the following may be administered via the tracheal mucosa?

A Lignocaine

B Calcium chloride

C Theophylline

D Suxamethonium

Explanation A

Resuscitation drugs such as lignocaine, epinephrine, atropine, naloxone, and vasopressin (Mnemonic for resuscitation drugs that may be given down the ET tube is NAVEL) are absorbed via the trachea. Administration of resuscitation drugs into the trachea, however, results in lower blood concentrations than the same dose given intravascularly.

**Question 18**

Which of the following local anaesthetics shortens the action potential duration?

A Ropivacaine

B Lignocaine

C Bupivacaine

D Prilocaine

Explanation B

Class 1b antiarrhytmic drugs shorten the action potential. Lignocaine is a 1b antiarrhythmic drug. Other drugs include mexiletine. Class 1a lengthens the action potential and class 1c does not affect the action potential. Lignociane is the drug of choice for the termination of ventricular tachycardia and prevention of ventricular fibrillation after cardioversion in the ischaemic setting.

**Question 19**

The side effects of suxamethonium involve all of the following except:

A Urinary retention

B Tachycardia

C Muscle fasciculations

D Excess salivation

Explanation A

Suxamethonium is a rapid onset muscle relaxant which has a short duration. It is contraindicated with a family history of malignant hyperthermia, severe liver disease and hyperkalemia. Side effects include muscle pain, fasciculation and myoglobinaemia, tachycardia or bradycardia, hypertension and hypotension, bronchospasm, hyperkalaemia and hyperthermia. It may also cause excess salivation but not urinary retention.

It acts by mimicking acetylcholine at the neuromuscular junction. It cannot be reversed but is short acting.

**Question 20**

A patientt who is now day 4 stay in ICU with airway burns, requires intubation. Which of the following muscle relaxant drugs is CONTRAINDICATED?

A Vecuronium

B Gallamine

C Succinylcholine

D Rocuronium

Explanation C

Hyperkalaemia from tissue destruction may complicate management during acute resuscitation. Despite the risk of hyperkalaemia due to the burn, succinylcholine is ONLY contraindicated after the first 24hrs of the burn.

Normal muscle releases enough potassium during succinylcholine-induced depolarisation to raise the serum K by 0.5mmol/L. While this is insignificant in patients with normal serum potassium levels, a life threatening potassium elevation in patients with burn injuries, massive trauma, neurological disorders, severe sepsis, spinal cord injury, tetanus, closed head injury and denervation injuries is possible. Subsequent cardiac arrest may occur which can be quite refractive to conventional resuscitation requiring HCO3, insulin/glucose, dantrolene, (cation-exchange resin), calcium, bypass to reduce metabolic acidosis and hyperkalaemia. In denervation injuries, ACH receptor develops outside the neuromuscular junction (up-regulation). These extra-junctional receptors allow succinylcholine to effect wide spread depolarisation and excessive potassium release. Life threatening is not reliably prevented by a non depolariser type drug either. The risk of hyperkalaemia usually appears to peak in 7-10 days following injury, but the exact time of onset and the duration of the risk period vary

Note: cation exchange resins are usually not effective after a single dose and may produce serious side effects, especially in patients who are postoperative, patients with ileus or bowel obstruction or have transplanted organs. Bowel necrosis may occur. Due to these severe side effects, resins should only be used in patients who have life threatening hyperkalaemia, dialysis not readily available and other therapies to remove potassium have failed or are not possible

**Question 21**

Which of the following inhaled anaesthetics DOES NOT trigger malignant hyperthermia?

A Halothane

B Sevoflurane

C Nitrous oxide

D Ether

Explanation C

Malignant hyperthermia (MH) can be triggered by halogenated general anaesthetics including: ether, cyclopropane, halothane, methoxyflurane, enflurane, isoflurane, desflurane, sevoflurane. Nitrous gas does not trigger MH. Succinylcholine can also trigger MH

Advantages of N2O

Inert, non toxic

Minimal CVS effects

Low blood solubility

Rapid induction and recovery

Environmentally friendly

Non-explosive

Disadvantages

MAC (>100%)

High cost

No commercially available anaesthetic equipment