SELF ASSESSMENT SECTION

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Multiple Choice Questions

1. *Drugs that block beta-adrenergic receptors (i.e."beta-blockers") can produce:*
a. Bradycardia
b. Asthma
c. Cold hands
d. Angina
e. Lethargy

2. *The following are normal values for a 3 year old child:*
a. Systolic blood pressure of 85mmHg
b. Respiratory tidal volume of 160ml
c. Pulse rate of 60 beats per minute
d. Respiratory rate of 10 breaths per minute
e. Circulating blood volume of 1.1 litres

3. *In children:*
a. The larynx is at a lower level than in an adult
b. The epiglottis is relatively large and floppy when compared to that of an adult
c. The narrowest part of the airway is at the cords
d. The larynx is less susceptible to oedema
e. Laryngospasm is more likely

4. *Volatile anaesthetic agents:*
a. Recovery is faster with agents with a high solubility in blood
b. Gaseous induction is slowed in high cardiac output states
c. More volatile is needed following an increase in inspired oxygen
d. Volatile anaesthetics all produce good analgesia
e. 50% of patients will move on skin incision with 1MAC of volatile agent

5. *Gastric emptying is slowed:*
a. In trauma
b. By opioids
c. By alcohol
d. In the first trimester of pregnancy
e. With hypothermia

6. *The cerebral blood flow (CBF) of a normal person is increased:*
a. If the intracranial pressure is increased
b. With administration of ketamine
c. If the mean arterial blood pressure rises from 90 to 110mmHg
d. When the arterial CO_2 increases
e. When placed in the head-down position

7. *Sympathetic stimulation may result in:*
a. An increased heart rate
b. An increase in myocardial contractility
c. An increase in skeletal muscle blood flow
d. An increase in cardiac output
e. Arrhythmias during halothane anaesthesia

8. *The height of a spinal block can be affected by:*
a. Local anaesthetic dose
b. Height of patient
c. Local anaesthetic baricity
d. The interspace used for the spinal injection
e. The addition of adrenaline to the local anaesthetic

9. *Characteristic features of cardiac tamponade include:*
a. Raised JVP
b. Tachycardia
c. Tracheal deviation
d. Hypotension
e. A fall in JVP on inspiration

10. *Suxamethonium*
a. Is effective when mixed with thiopentone
b. Muscle relaxation always wears off quickly following a single dose
c. Muscle relaxation always wears off quickly following multiple doses
d. Post suxamethonium muscle pains are worst in young adults
e. Causes arrhythmias

Clinical Scenario 1

A 32 year old woman presents with a three day history of central abdominal pain of increasing severity. She had an appendicectomy when aged 17, but otherwise has been fit and well. She has a respiratory rate of 30, pulse rate of 135 and a blood pressure of 90 / 55, and she feels hot and sweaty. Her abdomen is rigid with guarding, and a laparotomy is planned. Her Hb is 15.2, white cell count 17,000 and platelets 353 $x10^9$. Na^+ 145mmol/L, K^+ 5.2mmol/L, Creatinine 190 micromol/L and urea 12mmolL.

Question - how would you assess and manage this patient prior to theatre?

The clinical signs suggest that this patient is shocked. Sepsis would be the most likely cause, unless she had taken a toxic substance. Her blood results are all consistent with severe fluid depletion and the raised creatinine and urea rising indicate developing acute renal failure. The raised WCC supports the diagnosis of sepsis.

One should look for signs of poor organ perfusion such as oliguria, confusion, cool peripheries or a metabolic acidosis on arterial blood gas analysis. Initial treatment should involve face mask oxygen, intravenous fluid resuscitation and appropriate analgesia. The patient should be catheterised and urine output accurately measured. If available blood should be taken for a coagulation screen and a chest Xray performed if the patient is hypoxic or has clinical signs on auscultation.

The patient will require several litres of intravenous fluid (0.9% saline or equivalent) prior to going to theatre. If observations do not improve with intravenous filling then a central venous line may help assess how much intravenous fluid is appropriate. Commence broad intravenous antibiotic cover.

Following rapid sequence induction of general anaesthesia, surgery reveals adhesions and a perforated small bowel, with widespread faecal contamination of the peritoneum. Her blood pressure has fallen to 70 / 35, with a pulse rate of 155.

Question - how do you treat this blood pressure?

Hypovolaemia and sepsis are the most likely causes and a CVP line should be inserted. Give further fluid if her central venous pressure is low, or only transiently rises with fluids. Other treatable causes of hypotension should be excluded (excessive anaesthetic, tension pneumothorax, anaphylaxis, cardiac arrhythmias, ischaemia or tamponade, pulmonary embolism, hypothermia, gross electrolyte, acid base or glucose abnormalities).

In sepsis, hypotension is often not treatable by fluid resuscitation alone and inotropic support should be commenced. Understanding the pathophysiology of septic shock is helpful when choosing an appropriate inotrope. In sepsis, the infecting organisms release toxins into the blood stream, and the patient produces mediators such as cytokines and complement, all of which have systemic effects (*see Update in Anaesthesia No 13*). The cardiovascular effects include depression of contractility, vasodilatation, maldistribution of blood flow and damage to the endothelium (the lining of blood vessels). This results in a decrease in systemic vascular resistance and organ dysfunction and may lead to refractory hypotension, multiple organ failure and death. The hypotension is mainly due to a low systemic vascular resistance, and in 90% of patients the cardiac output is initially raised. Noradrenaline and adrenaline are frequently used in this clinical setting.

A vasopressor is a drug that increases the smooth muscle contraction in the walls of arterioles, thereby increasing the systemic vascular resistance. Adrenaline and noradrenaline are examples of drugs that have vasopressor action. As these drugs have a half life of just a few minutes, they are best delivered by infusion.

Noradrenaline would be the best choice in this patient because of its potent alpha action. Mix 4mg noradrenaline in 40mls dextrose 5% (1:10,000) and start an infusion via the CVP line at 5 - 10mls/ hour. Adjust the rate according to the response of the blood pressure. Measures of organ perfusion such as urine output provide a more reliable end point for treatment than blood pressure alone.

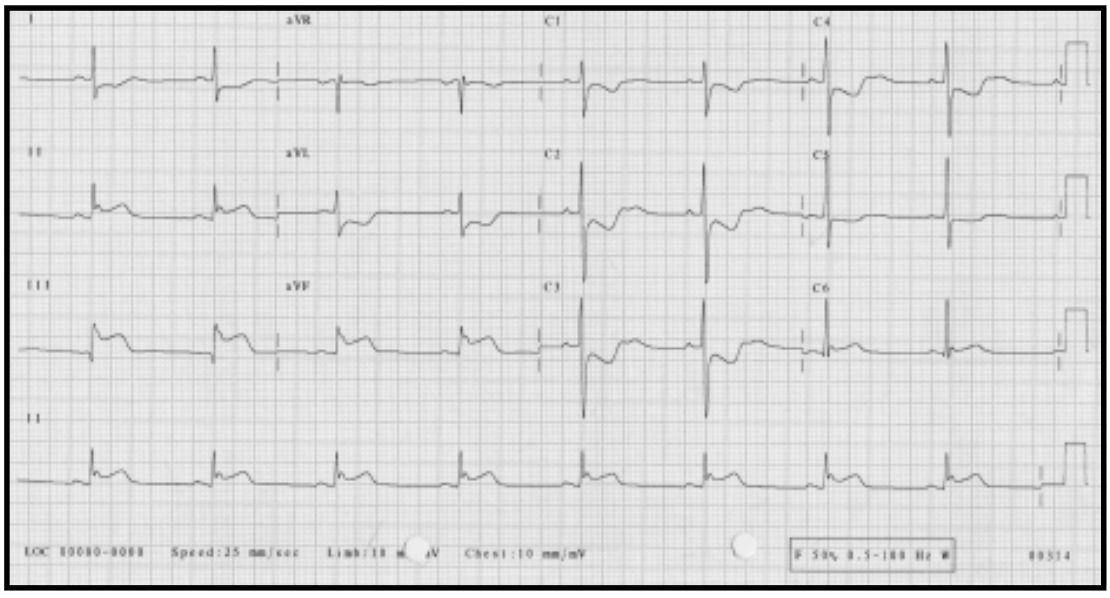
The patient will need postoperative care in an ICU or high dependency area to provide closed monitoring of oxygenation, blood pressure, fluid requirements, urine output, analgesia and conscious level. Blood count and electrolytes will need rechecked. Blood gases are useful, but often not available.

In these patients surgery is lifesaving, but patients should always receive some resuscitation before they are anaesthetised to prevent rapid decompensation following induction.

Clinical Scenario 2

A 69 year old man sustained a fractured femur, which was reduced and internally fixated under general anaesthetic. He has no past medical history of note, and takes no regular medication. Two days hours after the operation he complains of a dull, constant chest pain that goes down into his epigastrium. This has been present for the last few hours. A full blood count and serum electrolytes are taken, which are normal, and a chest x-ray and 12 lead ECG are organised.

Examine the following ECG:



Question - what is the diagnosis?

Acute inferior myocardial infarction. A diagnosis of MI requires 2

or 3 out of the following; a clear history, ECG changes or characteristic enzyme changes. The electrocardiogram (ECG) shows elevation of the ST segments in leads II, III and aVF. Causes of this finding include myocardial infarction, pericarditis and it is an occasional normal finding (chest leads V1 and V2, especially in Afro-carribean patients).

Elevation should be 2mm or more above the baseline (1mm in the limb leads) in 2 or more leads that are looking at similar parts of the heart. Leads II, III and aVF are the leads that look at the inferior surface of the heart, and changes in these leads points to an occlusion within the right coronary artery distribution. ST elevation occurs within hours of an infarct. Over the course of days, T-wave inversion occurs, and after several months have past, these changes slowly resolve.

Myocardial cell death produces characteristic changes in the levels of several serum enzymes, which can contribute to a diagnosis of myocardial infarction. Creatinine kinase (CK) levels of greater than twice their normal level and higher than 200 mmol/litre may indicate muscle cell death, but not specifically heart muscle. They rise within 12 hours of injury. If CK is raised (normal after surgery), measurement of the cardiac specific sub-type of this enzyme (CK MB) may be helpful. CK MB levels greater than 5% of the total CK level are usually specific for myocardial cell death.

Tropinin is a muscle protein that can give the most specific indication of myocardial damage. It rises to a peak level by 12 hours post infarct, and then falls over a few days. Other enzyme levels, such as aspartate transaminase and lactate dehydrogenase rise following a myocardial infarction. They are, however much less specific, and raised levels can follow damage to other tissues such as liver and skeletal muscle.

Question - describe the typical symptoms and signs of an MI?
Chest pain, which is classically central and 'heavy' or 'crushing' in nature, which may radiate to the jaw, neck, arms or epigastrium. If patient has pre-existing angina, it is frequently of a similar nature, but often more severe and lasts more than 30 minutes. The patient may have nausea or be vomiting. Symptom free or 'silent' infarcts are common, especially in diabetics or the elderly.

Patients undergoing an acute myocardial infarction often appear cold, pale and sweaty. Some patients become pyrexial. They may be hyper- or hypotensive and may have a sinus tachycardia or a dysrhythmia. Some patients will have signs of left ventricular failure, including tachypnoea and fine bibasal crackles on chest auscultation.

Question - how would you treat this patient?
Reassure, give high flow face mask oxygen. If possible move the patient to a coronary care unit or an intensive care unit and monitor the patient with a three lead ECG. An intravenous cannula should be sited and intravenous morphine and anti-emetic administered (if the patient is not currently receiving morphine, a slow intravenous bolus of 5-15mg is appropriate). The morphine acts as an analgesic and anxiolytic, and venodilates, reducing cardiac work.

The patient should be given 300mg of aspirin orally, and glyceryl trinitrate 0.5 mg sublingually. Thrombolysis should be considered, however in this patient's case the recent complex surgery was thought to present too great a risk of bleeding. Subcutaneous heparin (5000 units twice a day) should be given until patient is fully mobile (about day 5). The patient should be confined to bed for 24 hours, with continuous ECG monitoring, regular blood pressure measurement and daily 12-lead ECGs, serum cardiac enzyme and electrolyte assays. Smoking is a risk factor for ischaemic heart disease, and in the period following an acute myocardial infarction it can have particularly severe adverse effects, therefore it should be discouraged. The patient should be examined at least daily to check for complications.

Complications include:
- Arrythmias
- Heart failure/ Cardiogenic shock
- Hypertension
- Pericarditis (Dressler's syndrome)
- Deep vein thrombosis / Pulmonary embolism
- Ventricular aneurysm / Cardiac rupture
- Papillary muscle rupture, ventricular septal defects

ACE inhibitors, β blockers and statins are used in some hospitals, in some patients for long term prevention of further problems. Generally, ACE inhibitors are used in patients who have had an anterior myocardial infarct who show signs of left ventricular dysfunction. Some cardiologists use β blockers in the immediate treatment of myocardial infarction, however most patients in the UK will receive a first dose of β blockers at 12 - 24 hours, provided there is no heart block, bradycardia, hypotension or heart failure.

MULTIPLE CHOICE ANSWERS

1. TTTFT

Beta-blockers depress contractility and slow the heart. They were traditionally avoided in all forms of heart failure, but now, under the guidance of a cardiologist they may be useful in the treatment of some cases of heart failure. They may also precipitate asthma and should therefore be avoided in patients with a history of asthma or chronic obstructive pulmonary disease. Beta-blockers are avoided in patients with second or third degree heart block. Other side effects include fatigue, extremity coldness and glucose intolerance. Beta-blockers are a treatment for angina.

2. TFFFT

Normal ranges of physiological values in a 3 year old child include:

- Systolic blood pressure 80 - 100 mmHg
- Pulse rate 95 - 140
- Respiratory rate 25 - 30

Using the formula: Weight (in kg) = (Age +4) x 2

- Weight (approximately)14 kg

The tidal volume is 5 - 7 ml per kg for all ages, and the circulating blood volume is 75 - 80 ml per kg, which gives the following values:

- Tidal volume 70 - 98 ml
- Circulating blood volume 1050ml

3. FTFFT

The larynx of a child is situated at a higher level. The infant larynx is level with the third cervical vertebra compared to C6 in an adult. The epiglottis is U shaped and relatively long and the cricoid cartilage forms the narrowest part of the larynx prior to puberty. An inappropriately large tube may pass easily through the cords, and cause trauma at the level of the cricoid cartilage. The mucosa of a child is susceptible to oedema and this may result in airway obstruction after extubation. Children are prone to laryngospasm.

4. FTFFT

Inhalational agents that have a high degree of solubility in blood are absorbed in greater quantities during anaesthesia, and on termination of anaesthesia they are relatively slow at passing into the lung. A quantity of agent dissolved in a volume of blood will have a relatively low 'partial pressure' (when compared with agents with low solubility), and it is the 'partial pressure' that causes the agent to come out of solution in the lungs. The recovery from anaesthesia with an agent that is highly soluble in blood will therefore be slow.

In a high cardiac output state, the inhaled vapour is removed from the lungs and distributed around the body, preventing a sufficient 'partial pressure' from quickly building up. The 'partial pressure' build up in the lungs reflects the 'partial pressure' elsewhere in the body, including the brain. Induction is therefore slow.

Increasing oxygen does not affect the level of anaesthesia (unless there is a significant decrease in nitrous oxide). While trichloroethylene, methoxyflurane and ether produce good analgesia, other volatiles have little or no analgesic effects, and other analgesics are required to produce balanced anaesthesia during painful procedures. Anaesthetic effect is related to the partial pressure of the agent in the brain. At equilibrium (where the same partial pressure exists in blood and alveolar gas) the partial pressure in the brain bears a constant relationship to the partial pressure / concentration of the agent in the alveoli. The MAC or Minimum Alveolar Concentration is the concentration in the alveoli, at atmospheric pressure, that prevents movement in 50% of patients when exposed to a noxious stimulus such as skin incision. It is inversely proportional to the solubility of the drug in oil and is a measure of drug potency.

5. TTTFT

Stomach emptying can be slowed by many factors including trauma, anxiety, shock, hypothermia, intestinal obstruction, fatty foods, peritonitis, hypokalaemia, opioids, hyperglycaemia, anticholinergics, uraemia. Emptying may be slowed in late pregnancy

6. FTFTF

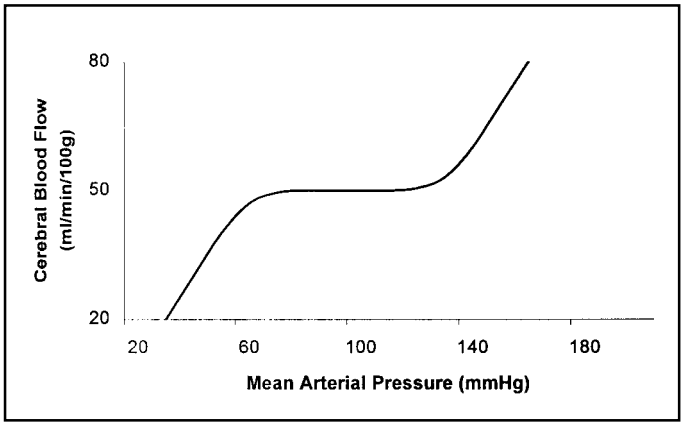
Cerebral blood flow (CBF) is related to the cerebral perfusion pressure.

CPP = MAP - (ICP + CVP)

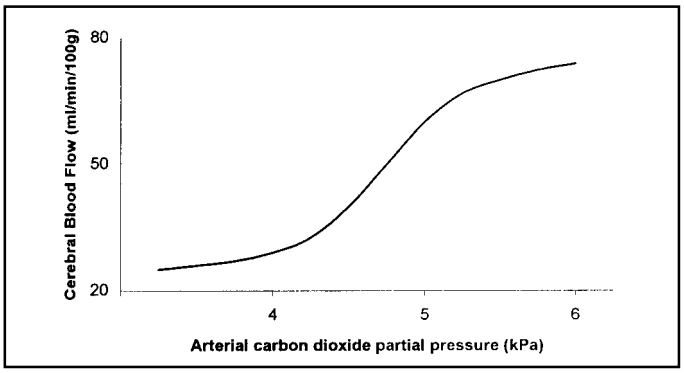
- ICP = Intracranial Pressure
- CVP = Cerebral Venous Pressure
- CPP = Cerebral Perfusion Pressure
- MAP = Mean Arterial Pressure

An increase in intracranial pressure therefore results in a reduction in cerebral blood flow. In a person with normal intracranial haemodynamics however, the blood flow is kept constant over a wide range of blood pressures due to "autoregulation". Loss of such autoregulation is common following head injury.

Cerebral Autoregulation



Cerebral Response to Blood Gases



CBF is increased by:
- Ketamine
- All volatile anaesthetics (Isoflurane has the least effect)

CBF is decreased by:
- Thiopentone
- Methohexitone
- Etomidate
- Propofol

ICP rises with an increase in intracranial contents. Increased CBF secondary to vasodilatation, leads to an increase in the volume of intracranial blood and may produce a rise in ICP. Anaesthetic agents that cause a marked increase in cerebral blood flow should be avoided in patients with raised ICP.

High CO_2 levels increase cerebral blood flow by vasodilatation, as do arterial oxygen partial pressures below 8kPa (60mmHg).

The veins draining the head have no valves preventing back flow of blood. A head down position therefore causes an increase in intracranial venous pressure. This effect causes little more than discomfort in patients with no intracranial pathology. However even a modest rise in ICP in patients with intracranial pathology may result in reduced CBF. A similar rise in cerebral venous pressure may result from tight neck collars or neck haematomas.

7. TTTTT

Sympathetic stimulation leads to a collection of effects called the "fright and flight" response. This includes a number of changes that include tachycardia, increased myocardial contractility and vasoconstriction in arterioles in the skin and gut. In contrast, vessels supplying blood to skeletal muscles dilate. The blood pressure rises in response to an increase in cardiac output and an increase in systemic vascular resistance.

Sympathetic stimulation can be produced by both the sympathetic nervous system and by circulating hormones from the adrenal medulla (adrenaline and noradrenaline). The sympathetic nervous system leaves the central nervous system from thoracic and lumbar spinal levels (T_1 - L_2, called the thoraco-lumbar outflow). When these nerves are blocked by spinal or epidural anaesthesia, the resulting vasodilatation can lead to a drop in blood pressure. If the sympathetic nerves that supply the heart (T_1 - T_4) are blocked, both heart rate and myocardial contractility will fall, and profound hypotension can result.

The parasympathetic nervous system produces opposite effects to the sympathetic nervous system, and acts to balance the sympathetic effects.

8. TTTFF

Factors affecting the height of a spinal anaesthetic block include:
- Local anaesthetic dose (i.e. volume multiplied by concentration) with respect to the size of the patient
- Local anaesthetic density (baricity)
- Patient posture
- Patient factors

Volume has a relatively minor effect on spread if dose is kept constant. The level chosen for spinal injection has little effect on the height of block achieved as the injection for spinal anaesthesia can only be carried out at L_2/L_3, L_3/L_4 or L_4/L_5. The spinal cord is present at and above L_1 and spinal cord damage may result from injections at or above this level, and the fused sacrum prevents injection below L_5. Needle size and the direction of the bevel have little effect on the level of block.

Many patient factors can affect the eventual height of block:
- Age - older patients require smaller doses
- Height - taller patients require a larger doses
- Weight - obese patients require smaller doses
- Pregnancy - patients require smaller doses
- Kyphosis / Scoliosis may affect the spread of local anaesthetic

Factors affecting duration of a spinal anaesthetic include:
- Dose of anaesthetic given
- Local anaesthetic agent chosen
- Addition of adrenaline

9. TTFTF

Cardiac tamponade results from a build up of blood or other fluid in the pericardium. If this interferes with normal filling of the heart ventricles, symptoms may include tachycardia, hypotension, a high jugular venous pulsation that rises on inspiration and muffled heart sounds. The volume of the pulse falls in inspiration - "pulsus paradoxus". If cardiac tamponade is recognised as the cause of shock it should be treated by immediate drainage.

10. FFFTT

All solutions of suxamethonium are destroyed by alkali, and should never be mixed with thiopentone. A small proportion of people take a long time to metabolise suxamethonium. Multiple doses also sometimes cause prolonged paralysis and in both these situations, patients may require ventilation for a few hours before muscle function returns. Suxamethonium frequently causes bradycardia, especially with a second dose in children. Some patients release high levels of potassium following suxamethonium (in particular, patients a few days following burns or spinal cord injuries, or severe muscle trauma), and this may result in serious life threatening arrhythmias or a cardiac arrest.