

SELF ASSESSMENT EXERCISES IN INTENSIVE CARE MEDICINE

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Abstract

Intensive Care Medicine (ICM) is no longer the exclusive preserve of anaesthetists as both emergency medicine and general medicine trainees now also train in this increasingly important speciality. This edition of the JRAMC self assessment question series serves to cover some of the current 'hot topics' in ICM and enables readers with experience of ICM to test their knowledge as well as educating junior trainees in relevant subjects that they may be unfamiliar with. Similarly, the Focus On . . . series of papers elsewhere in this journal demonstrate the increasing importance of ICM in the deployed Field Hospital setting.

You are an ST3 trainee doing a three-month attachment to the Intensive Care Unit of a large DGH. It is the beginning of your first night shift and you have just completed the hand-over ward round from the outgoing ST3.

Question 1

A 68-year-old man was admitted 10 days previously with a severe exacerbation of his COPD. He now has a percutaneous tracheostomy *in situ*, no longer requires sedation and has been weaning from the ventilator well. His nurse asks you to review him as his oxygen saturations have begun to fall over the last minute. You also note that his blood pressure and urine output have been slowly tailing off over the last 4 hours.

- What immediate course of action should you take?
- What are the possible causes of his deterioration?
- Why is the patients urine output falling, and what should you do about it?
- When managing a ventilated patient what measures should you ensure are in place?
- What would be an acceptable PaO₂ and PaCO₂ in this patient?

Question 2

Just as you are about to eat your on-call evening meal the Emergency Department SpR calls to ask you to assist with a 61-year-old lady with a history of essential hypertension and angina who has arrived following an out of hospital VF cardiac arrest with restoration of a spontaneous cardiac output after 2 shocks administered by the paramedic team. 'Downtime' was only 5 minutes but the patient remains GCS 3. When you arrive a few minutes later CPR has recommenced. The ECG monitor trace is shown in Figure 1.

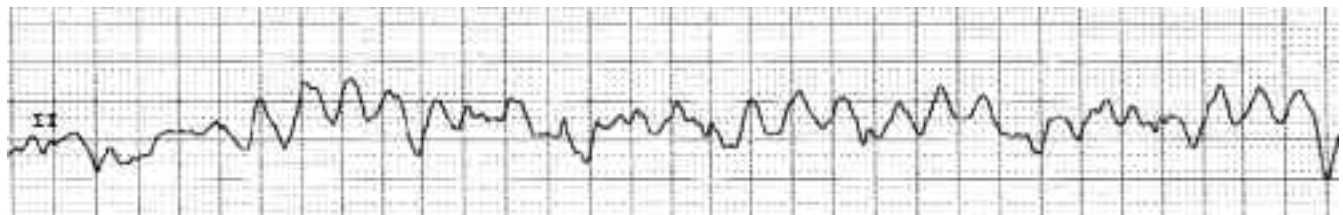


Figure 1. ECG trace.

- What is the diagnosis, and what is the immediate treatment?
- What pharmacological adjuncts are there to this treatment?
- After 3 further cycles of CPR and two further shocks there is a return of spontaneous circulation (ROSC). The patient remains GCS 3 and has no spontaneous respiratory effort. How should she be managed now?
- If after 5 minutes the patient becomes asystolic and a further 20 minutes later there remains no return spontaneous of circulation (ROSC), what would be a reasonable course of action? How would you approach this issue?

Question 3

While you are in the Emergency Department, a 24-year-old soldier, who lives in the accommodation block, arrives markedly hypotensive, tachycardic and obtunded with a GCS of 10/15 (E2, V3, M5). Her arterial blood gases (ABG) are shown in Figure 2. You also note a widespread rash over her torso and limbs, as illustrated in Figure 3.

- What is the most likely diagnosis?
- What should be your immediate course of action?
- After the insertion of a central line, her mixed venous blood gas shows a SvO₂ of 55%, what does this mean and how should it guide therapy?
- Once on ITU, what general measures should be taken for her care?
- Are any infection control measures needed and if so, what?

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RADIOMETER ABL 700 SERIES			
ABL725 Accident and Emergency			
PATIENT REPORT Syringe - S 195uL Sample # 32080			
Identification			
Patient ID			
Date of birth			
Patient Last Name			
Patient First Name			
Operator			
FO ₂ (l)	85.0 %		
Sample type	Arterial		
Blood Gas Values			
pH	7.151	[-]
pCO ₂	6.88 kPa	[-]
pO ₂	15.5 kPa	[-]
Oximetry Values			
ctHb	15.9 g/dL		
sO ₂	98.4 %	[-]
FO ₂ Hb	98.5 %	[-]
FCOHb	1.0 %	[-]
FHHb	1.8 %	[-]
FMetHb	0.9 %	[-]
pO ₂ (A-a) _e	34.41 kPa	[-]
Electrolyte Values			
cK ⁺	3.7 mmol/L	[-]
cNa ⁺	139 mmol/L	[-]
cCl ⁻	106 mmol/L	[-]
Metabolite Values			
cGlu	5.9 mmol/L	[-]
Acid Base Status			
cBase(Ecf) _e	-12.2 mmol/L		
cHCO ₃ ⁻ (P.st) _e	12.7 mmol/L		
Notes			
c	Calculated value(s)		
e	Estimated value(s)		

Figure 2. ABG analysis printout.



Figure 3. Rash on lower limbs.

Question 4

On your return to the ICU, one of the nurses calls you over to see an 18-year-old female patient who is being ventilated for an exacerbation of asthma. Her airway pressures are rising, tidal volumes falling and inspection of her blood gases reveals her PaCO₂ is rising.

- What difficulties may be experienced when ventilating an asthmatic patient?
- List the drugs which should be used in the management of this condition, and by which route should they be given?
- What sedative regime should be used?

Question 5

Having been to bed for a couple of hours, you rise early to examine the patients before the morning ward round. When you look at the patient in bed 8, you note his pupils are dilated and fixed. You know from the handover round from yesterday that he is a head injured patient from a road traffic collision 2 days ago, who suffered a diffuse axonal injury. The neurosurgical unit was full, so they had asked your consultant to keep him sedated and ventilated on your unit, for a rescan at 48 hours, with a view to waking him up. He had been stable for the preceding 48 hours.

- What is the most likely diagnosis, and what other clinical features may support this?
- What actions should be taken?
- Despite these measures, the pupils remain dilated and a repeat CT scan shows no lesion remedial to surgery or further medical intervention. During the ward round the team discuss brain stem death testing. What are the key elements of brain stem death testing?
- What current legislation is there regarding tissue donation?
- How can you maximise the chances that any donated organs remain viable?

Answers to Self Assessment Questions

Question 1

a) Your first priority should be to ensure the airway is clear, in this case concentrating on the tracheostomy position and patency. Newly inserted tracheostomies should have a reliable and well-established tract after around 1 week. However, care must always be taken to ensure that the tube lies correctly in the lumen of the trachea. The use of a soft bougie or flexible fiberoptic endoscope can aid safe repositioning of the tracheostomy tube. The maximum possible FiO₂ should be administered. Assess the breathing and circulation and manage with additional ventilatory pressure support, fluid boluses and/or vasopressors/inotropes. Check for signs of pneumothorax, which may include urgent chest radiography. If the oxygen saturations continue to fall, or the ventilator does not appear to be delivering adequate volumes, 100% oxygen should be delivered by hand ventilation [1].

b) The most likely cause of his acute deterioration is from either tracheostomy tube blockage (for example due to mucus plugging) or becoming dislodged. The respiratory compromise may be due to worsening gas transfer due to ventilator-associated pneumonia (VAP). His general deterioration is most likely to be due to an underlying SIRS or septic response to this. Patients on mechanical ventilators are at high risk of acquiring VAP, and many measures have been suggested to prevent it – few have shown much promise in larger studies. However recent NICE guidelines suggest that chlorhexidine mouth washes and nursing with a head up tilt should be used. Supraglottic suction and selective decontamination of the digestive tract may prove to be beneficial, but better studies are needed [2]. As this patient is systemically unwell, he should be started on a new course of antibiotics, if possible under guidance from the microbiologist.

c) The falling urine output is likely to represent a degree of renal impairment most likely due to hypovolaemia / hypotension. However, renal failure from disease (such as infection), or a post-renal cause such as bladder catheter blockage must also be ruled out.

The initial approach to falling urine output should be to fully assess the patient exploring for possible causes of hypotension such as signs of an infective focus or active haemorrhage. Hypotension should be treated with crystalloid fluid boluses and consideration of the use of blood products or artificial plasma expanders. In cases of sepsis patients may require vasopressors or inotropes to maintain an adequate blood pressure in order to improve renal

perfusion. Prolonged hypoperfusion may lead to acute renal injury and the subsequent need for renal support therapy. Initial blood tests should be taken to monitor renal function. Bladder catheter blockage may be investigated and managed by a 50ml sterile water catheter flush. There is no place for furosemide or 'renal (low) dose dopamine' in these patients [3,4].

d) Measures for managing ventilated patients include both equipment and personnel issues. Resources for urgent advanced airway management must be readily accessible and near to the patient, even during transfers. Vacuum/suction, high flow O₂ and an alternative method of ventilation (e.g. Bag/Valve/Mask (BVM)) must be immediately available. Aids to intubation such as gum elastic bougies and a range of laryngoscope blades and should also be carried. 'Rescue methods' of airway management, including supraglottic airway devices (e.g. LMAs) and surgical airways, should be available.

Continuous physiological monitoring should include SpO₂, invasive blood pressure, end-tidal CO₂ and ECG. Carried/available drugs should include standard ALS therapeutic agents, appropriate anaesthetic drugs and muscle relaxants, vasomotor agents (such as metaraminol), fluids and sufficient oxygen for the time required (if on transfer). Recent guidelines have mandated the use of end tidal CO₂ for all patients with artificial airways (including supraglottic devices) at all times [5].

Close supervision is required for all ventilated patients in order to detect and react to changes in clinical condition. 1:1 nursing is the usual standard of care on ICU wards. Intubated and ventilated patients should be managed by a minimum of two members of staff (usually an appropriately trained doctor and critical care nurse) during transfers or in locations other than the ICU, such as the resuscitation room or general wards. All attendant staff will need to be aware of the location and competent in the use of emergency equipment and medications.

e) A higher than normal PaCO₂ can usually be tolerated in view of the history, and bearing in mind the patients 'normal' level may be high due to pre-existing pulmonary disease. Most practitioners will accept any CO₂, so long as it does not cause an acidosis. This has been termed "permissive hypercapnia" and is relatively contraindicated in the presence of a coexisting brain injury. It is probably more important to limit the tidal volumes used to ventilate this patient to 6-8ml/kg, as this has been shown to be protective against the development of Adult Respiratory Distress Syndrome (ARDS), which still has a mortality of 40% [6]. Invariably this will need to be accompanied by a high respiratory rate to maintain minute volume – but even with this the resulting acidosis may be severe, some authorities now advocate a sodium bicarbonate infusion in these patients. The recently developed lung assist device (Novolung – Inspiration Healthcare), may prove to have a use for decarboxylation in this group of patients [7].

The primary objective is to maintain an adequate PaO₂ (>8 KPa). Cardiac output, haemoglobin level and SaO₂/SpO₂ are more relevant in terms of the delivery of oxygen to the tissues. High Peak End Expiratory Pressure (PEEP) (whether sustained or temporary such as during a recruitment manoeuvre) may be effective in improving oxygenation, as may positioning the patient prone. High inspiratory pressures should be minimised by utilising pressure limiting ventilator modes such as bilevel positive airway pressure (BIPAP). In the future, High Frequency Oscillation may be used to improve oxygenation.

Question 2

a) The rhythm is Ventricular Fibrillation (VF). The treatment is primarily defibrillation and cardiopulmonary resuscitation (CPR). Resuscitation Council (UK) Adult Advanced Life Support (ALS) algorithms [8] should be followed.

An immediate, single biphasic shock of 150-360J (according to manufacturer's recommendations) is required, and CPR should be

recommended immediately after the shock has been delivered. Adult CPR is carried out at 100/min with a ratio of 30 compressions to 2 ventilations. If the patient has been intubated chest compressions may continue uninterrupted at 100/min. The emphasis between shocks is to ensure that compressions are effective and to minimise breaks in them. Therefore the team leader should monitor the quality of CPR as the person doing chest compressions will get tired. If there are enough rescuers this person should change about every 2 min. Brief rhythm checks should take place every 2 minutes. If the patient remains in VF or pulseless VT further shocks should be delivered at 150-360J.

Reversible causes of the arrest should be considered, recognised and treated - the 4H's and 4T's (Table 1).

- | |
|---|
| <ul style="list-style-type: none"> • Hypoxia • Hypovolaemia • Hyperkalaemia, hypokalaemia, hypocalcaemia, acidaemia etc • Hypothermia • Tension pneumothorax • Tamponade • Toxic substances • Thromboembolism (pulmonary embolus/coronary thrombosis) |
|---|

Table 1. The "4H's and 4T's".

b) Based largely on experimental data, the use of adrenaline is still recommended as an adjunct to CPR (1mg intravenously, every 2-4 minutes), despite the lack of convincing human data. The alpha-adrenergic actions of adrenaline cause vasoconstriction, which increases myocardial and cerebral perfusion pressure during compressions.

In comparison with placebo and lidocaine, the use of amiodarone in shock-refractory VF improves survival to hospital admission. If VF/pulseless VT persists after three shocks, amiodarone 300 mg should be administered by bolus injection. A further dose of 150 mg may be given for recurrent or refractory VF/VT, followed by an infusion of 900 mg over 24 hours. If amiodarone is not available, lidocaine 1mg/kg may be used as an alternative, but should not be given if amiodarone has already been given. A total dose of 3mg/kg lidocaine should not be exceeded during the first hour.

Magnesium (8 mmol = 4 ml of a 50% solution) should be administered for refractory VF if there is any suspicion of hypomagnesaemia (e.g. patients on potassium-losing diuretics). Other indications are ventricular tachyarrhythmias in the presence of possible hypomagnesaemia, torsade de pointes or digoxin toxicity. Sodium bicarbonate therapy is not routinely indicated in cardiac arrest unless due to known tricyclic antidepressant (TCA) overdose or severe hyperkalaemia.

The likely cause for cardiac arrest in this case is a myocardial infarction or pulmonary embolus. Consideration may therefore be given to the use of thrombolysis, or percutaneous coronary intervention in suitable centres [8].

c) Mechanical ventilation should be maintained in order to achieve normocarbida and normal oxygen levels. Circulatory support may require judicious use of fluids, or diuretics/vasodilators in the case of left ventricular failure (LVF). Inotropes/vasopressors may be required to maintain an appropriate BP and urine output (bearing in mind the patients 'normal' BP). Balloon pump catheters may also be utilised post-arrest in order to improve cardiac output. Once stabilised, the patient should be transferred to the most appropriate critical care area for ongoing management.

Two randomised clinical trials have shown improved outcome in adults who remain comatose after initial resuscitation from out-of-hospital VF cardiac arrest and were cooled [9,10]. Unconscious adult patients with spontaneous

circulation after out-of-hospital VF cardiac arrest should be cooled to 32-34°C as soon as possible, for at least 12-24 h. Mild hypothermia may also benefit unconscious patients with spontaneous circulation after out-of-hospital cardiac arrest due to a non-shockable rhythm, or after cardiac arrest in hospital. External and/or internal cooling techniques may be used. An infusion of 30ml/kg cooled (4°C) 0.9% saline will decrease core temperature by 1.5°C. Intravascular cooling enables more precise control of core temperature than external methods, but it is unknown whether this improves outcome. Shivering should be avoided by ensuring adequate sedation and giving neuromuscular blocking drugs, as boluses. After 12-24hrs, the patient should be rewarmed slowly (0.25-0.5°C/hr) avoiding overshooting and subsequent hyperthermia. The optimum target temperature, rate of cooling, duration of hypothermia, and rate of rewarming have yet to be determined. The complications of mild therapeutic hypothermia include increased infection, cardiovascular instability, coagulopathy, hyperglycaemia, and electrolyte abnormalities such as hypophosphataemia and hypomagnesaemia [11].

d) Several factors will influence the decision of whether to stop the resuscitative effort. There is a risk of permanent neurological injury following prolonged resuscitation despite even the most effective CPR and ALS care. Survival to hospital discharge is known to be around 21% for all cases of VF arrest. When considering such decisions bear in mind the four key ethical principles of beneficence, non-maleficence, justice and autonomy.

It is generally accepted that asystole for more than 20 minutes in the absence of a reversible cause, and with all ALS measures in place, is grounds for abandoning the resuscitation attempt. It is also recommended that resuscitation should continue for as long as VF persists. Certain mitigating circumstances, such as hypothermia at the time of the cardiac arrest, may alter the risk of neurological injury and prognosis.

Family knowledge of the patients' prior wishes, especially the presence of an advance directive or current DNAR order, should be explored if this has not previously been done. The final decision to stop resuscitation should be made by the team leader although in consultation with the other team members. This decision should be based upon the available evidence of an underlying cause, prior medical information and the anticipated prognosis. It is recognised that this is ultimately based upon clinical judgement and, as such, should involve the most experienced/senior clinician present.

Once a decision has been made this must be clearly and effectively communicated to all relevant staff and any friends or relatives present at the time. Family members should be given the opportunity to be with their relative at the time of death if they wish to do so.

Question 3

a) This soldier is septic, and given the rash it is most likely to be meningococcal septicaemia.

b) As with any patient, the ABC must be assessed and immediate resuscitation started. Meningitis still has a high mortality, although it is becoming rarer as more people get vaccinated, and is therefore a medical emergency. Senior clinical staff should be involved early. The key to successful treatment is early and appropriate antibiotic therapy.

Most units have adopted the 'Surviving Sepsis Campaign' [12]. This details 5 steps that must be undertaken within 6 hours of the diagnosis of sepsis being made (Table 2).

c) This shows inadequate oxygen delivery to tissues, or may also represent raised uptake. As Table 2 shows this needs to be improved, and can be achieved by transfusion up to a haematocrit of 30% + inotropes to maximise cardiac output. Dobutamine has been suggested as the inotrope of choice in severe sepsis.

1. Measure serum lactate
2. Obtain blood cultures
3. Administer broad spectrum antibiotics
4. If hypotensive and / or lactate > 4 mmol/l
Give a fluid bolus of 20ml/kg crystalloid
Add in vasopressor therapy if MAP remains below 65 mmHg
5. If hypotension or high lactate persists
Achieve a CVP of > 8 mmHg
Achieve a central venous saturation > 70% (or mixed venous saturation > 65%)

Table 2. The Surviving Sepsis Campaign 6 hour Resuscitation bundle.

d) The Surviving Sepsis Campaign details a further 4 steps that must be taken within 24 hours of admission. These are presented in Table 3, and should be addressed on the ITU (if not earlier).

1. Low dose steroids according to local policy
2. Drotrecogin alpha (Xigris®) administered, again according to local policy
3. Glucose maintained at the lower limit of normal
4. Inspiratory pressures of less than 30 cmH₂O if mechanically ventilated

Table 3. Surviving Sepsis 24 hour Sepsis Management Bundle.

Steroids and early antibiotic therapy have a specific place in the treatment of meningitis, and are discussed in detail elsewhere in this edition as is the indication for lumbar puncture [13].

In addition to these measures, general holistic care of the critically ill patient should be started – this should include DVT prophylaxis, pressure area care, early enteral nutrition and 30 degree head up tilt [12].

e) Health workers are only at risk from direct droplet/secretion contact, so standard barrier nursing is adequate. However there is considerable risk of spread within the accommodation block that the soldier has been brought from, so early involvement of the public health authorities is required for contact tracing and advice on antibiotic prophylaxis. Meningitis remains a notifiable disease.

Question 4

a) An asthmatic patient can be very difficult to mechanically ventilate, and every effort should be made to treat the disease without intubation. The essential problem is an obstruction to expiration, therefore the expiratory phase needs to be prolonged otherwise gas trapping can occur leading to an increase in intra thoracic pressure (auto PEEP). The patient will then need ever increasing amounts of inspiratory pressure to maintain tidal volumes, increasing the risk of barotrauma and pneumothorax.

However, if the expiratory phase is prolonged there will need to be less time in inspiration, to allow a short enough respiratory cycle to maintain an adequate minute volume. In order to maintain an adequate tidal volume with a shortened inspiratory phase, higher inspiratory pressures will be needed. Therefore, a vicious circle can be set up of ever increasing airway pressures, PaCO₂ and eventually falling blood pressures from rising intra-thoracic pressure. The key to managing this is prevention by maximising bronchodilator therapy. Briefly disconnecting from the ventilator and gently compressing the chest can also reduce auto PEEP, and many other novel therapies are being trialled (e.g. Heliox, novolung).

Ultimately, accepting the hypercapnia and controlling the acidosis with a slow bicarbonate infusion maybe the only option.

b) The British Thoracic Society regularly publish guidelines for treating asthma [14], although much of the evidence base is drawn from experience with life threatening asthma in spontaneously breathing patients. It is not unreasonable to extrapolate these to the ventilated patient.

Beta agonist therapy must be maximised, there is no evidence that intravenous administration is any better than via nebuliser [15] and indeed it may be worse. Salbutamol should be given by continuous nebuliser (5-10 mg/hr) if at all possible; the intravenous route should only be used if continuous nebulisation is not practical.

There is good evidence that the early administration of steroids can reduce mortality [16]. In the Intensive Care setting hydrocortisone 100mg every 6 hours would be appropriate. There is no benefit in administering inhaled steroids in addition to this [17].

Ipratropium Bromide would appear to work synergistically with beta agonists, and so should be administered as a nebuliser (0.5 mg every 6 hours) alongside salbutamol.

Magnesium has been shown to be safe and effective as a single dose [18]. It is therefore useful to use to 'break the cycle' as a one-off dose. Its safety in repeated doses in asthma has been evaluated, although it has also been used in other conditions. It would not be unreasonable to expect it to continue to have a smooth muscle stabilisation effect, but the more that is given the more likely it is to effect skeletal muscle as well. One-off doses of 2g IV over 20 minutes are likely to be of benefit.

Aminophylline has not been shown in metanalysis as beneficial for mild-moderate asthma [19]. For this reason it is difficult to recommend its use, especially given its multiple side effects. However, one study has shown evidence for benefit in severe acute asthma unresponsive to multiple doses of β_2 agonists and steroids [20]. It should be only used with a senior doctor's involvement.

Routine use of antibiotics is not recommended as exacerbations are rarely due to bacterial infections [14].

c) One of the real problems with 'brittle' asthmatic patients on ventilators is that any degree of awareness may provoke bronchospasm again. Patients therefore need to be kept heavily sedated until the steroids have had a chance to calm down the inflammatory response. Although there is good evidence that a daily sedation hold for ITU patients decreases the duration of mechanical ventilation and the length of stay in the intensive care unit [21], great care should be taken in this group of patients before doing this.

Standard sedative regimes should be used. However, there may be benefit in using ketamine in resistant cases as it is a powerful bronchodilator as well as a sedative. Remifentanyl is an ultra short-acting opioid which has some theoretical advantages as it allows rapidly titratable levels of sedation, and makes patients remarkable tolerant of an endotracheal tube even at low doses.

Neuromuscular blockade is rarely required and can not be recommended in any but the most resistant of asthma cases. They probably do little for the bronchospasm, although may make ventilation easier as the skeletal muscles of the chest wall and abdomen relax. Their use is however associated with critical care polymyopathies, and greatly extended ICU stay.

Question 5

a) The most likely cause would be brainstem herniation ("coning") due to a critical, sustained rise in intracranial pressure (ICP). This leads to brainstem ischaemia as it is compressed into the spinal cord, so the body will attempt to maintain perfusion by increasing blood pressure. This will be detected by the baroreceptors which will cause a bradycardia – this is the Cushing Reflex. If the patient were not sedated it would be accompanied by a fall in GCS. Ultimately this process will result in Brain Stem Death; the marked symptom of which is apnoea.

It is important to rule out any other cause, as this patient has been on the unit for a while, it is unlikely any local trauma will be the cause of the pupil dilation, but some drugs may have this effect.

b) It is vital to restore blood flow to the brain stem, by maintaining cerebral perfusion pressure (CPP), this is given by the following equation:

$$CPP = MAP - ICP$$

As the most likely cause will be brain swelling leading to a raised ICP, measures to decrease this should be immediately started. If not already, the patient should be placed at a 30 degree head up tilt and neck collars removed if in place (if so, maintaining the neck in a neutral position using tapes and sand bags). Mannitol can be given to temporarily reduce ICP, and some units are now giving hypertonic saline. Equally, a brief period of hyperventilation can help. This reduces cerebral blood volume (by reducing PaCO₂) therefore decreasing ICP, but does so at the expense of cerebral blood flow so it can not be maintained as it has been associated with increased ischaemia.

It can also be seen from the above equation that it is vital to maintain a good blood pressure. In the absence of ICP monitoring (as is the case in most DGH's), a MAP of 90 mmHg is recommended. This usually requires the use of inotropic support - noradrenaline being the most commonly used.

As soon as the patient has been stabilised and all measures have been instigated, this patient will need a CT scan of his head to rule out an intracranial catastrophe, such as an acute haemorrhage.

c) Brain Stem Death Testing (BSDT) was formally adopted in the UK as a diagnosis of death, having been proposed by the Conference of the Royal Colleges in 1976. It is important to note that there still is no statutory definition of death in the UK, but BSDT has been subject to case law in the courts. Some other countries do not accept them, and this may have implications on military deployments. It is however important, as the diagnosis of death in the presence of a beating heart allows organ donation to occur (although some organs, e.g. kidneys, can be retrieved soon after the heart has stopped beating).

There are 3 prerequisites to testing:

1. There must be an agreed, identified pathology consistent with irredeemable brain damage.
2. The patient must be deeply unconscious with hypothermia, drugs (specifically depressant and neuromuscular blocking agents) and metabolic / endocrine causes of coma ruled out.
3. The patient must be apnoeic, requiring mechanical ventilation.

There are then 7 tests, which test the function of the cranial nerves in the brain stem, as detailed in Table 4. All 7 must show an absence of activity for the test to show conclusive absence of brain stem activity, which is the cornerstone of diagnosing death in this situation.

- Pupils must be fixed in diameter, and not react to light. (CN I & II)
- There must be no reaction when the cornea is lightly stimulated by touch (eg cotton wool) (CN V & VII)
- No eye movement should be seen when 50ml of ice cold water is instilled onto the tympanic membrane ie absent vestibulo – ocular reflex (CNIII & VIII)
- No motor response in a cranial nerve area to stimulation – usually supra ocular pressure is used. (CN V & VII)
- No gag should be present when the posterior pharyngeal wall is stimulated (CN IX)
- No cough reflex to a suction catheter being passed down the endotracheal tube (CN X)
- No respiratory efforts seen when disconnected from the ventilator. The aim is to let the PaCO₂ rise above 6.6kPa to ensure there is no reflex breathing. Oxygenation must be maintained at all times, and the CO₂ documented with arterial blood gasses.

Table 4. Brain Stem Death Tests.

The tests should be carried out by two doctors who have been fully registered with the GMC for at least 5 years, one of which should be a consultant. Both should be skilled in interpreting the tests, and neither be part of the transplant team. Two sets of tests need to be carried out, the second set to confirm the findings of the first, but there is no requirement to have a time interval between them. In practice it is normal to talk to the next of kin between the 2 sets of tests. The time of death is given as the time of the first set of tests [22].

d) In the UK, the law regarding removal of organs from people after their death is set out in the Human Tissue Act 2004 (covering England, Wales and Northern Ireland), and the Human Tissue (Scotland) Act 2006. Essentially it allows for the removal of organs for transplant, so long as the coroner has no objections. The issue of consent remains controversial as there is no legal need for relatives to consent, especially if evidence of the patient pre mortem wishes to donate can be found (e.g. the organ donation register). However, very few practitioners would allow organ donation to occur if living relatives object.

e) Standard care, maintaining normal haemodynamic and homeostatic parameters, will maximise the chances that organs can be used for transplantation. It should be anticipated that the brain dead patient will become hypotensive as a result of decreased sympathetic tone, cardiac dysfunction and diabetes insipidus (DI). Fluids, vasopressors and inotropes will be required and DDAVP given for DI. Lung injury should be minimised by limiting airway pressures and normothermia maintained (normal thermogenesis will be lost). The endocrine system should be supported with steroids, thyroid hormones and insulin as required.

It should be noted that continuing treatment in the brain stem dead patient to preserve organ function is considered completely ethical. It would not however be ethical (or indeed lawful) to initiate therapies (such as invasive ventilation), purely in order to preserve organs for donations. Any therapy instituted before BSDT must be purely for the benefit of the patient.

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