



# Faculty of Intensive Care

## REPORT OF GENERAL FELLOWSHIP EXAMINATION

MARCH/APRIL 2000

This report is prepared to provide candidate, tutors and their Supervisors of training with information about the way in which the Examiners assessed the performance of candidates in the Examination. Answers provided are not model answers but guides to what was expected. Candidates should discuss the report with their tutors so that they may prepare appropriately for the future examinations.

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Thirteen candidates presented for this examination. Nine were successful.

### ORAL SECTIONS

#### Objectives Structured Clinical Examination (OSCE) Section

There were eleven stations with two rest stations after the interactive stations. The stations and comments on performance included:

- Station 1.     *Chest X-rays*  
Generally well handled but common errors included falsely diagnosing pneumothorax in the presence of skin fold lines and contralateral lobar collapse.
2.     *Arterial Blood Gases*  
Included salicylate O/D, respiratory acidosis with compensation and severe metabolic alkalosis.
3.     *Paediatric investigations*  
Included blood profiles of DKA and TTP and CXR of endobronchial intubation.
4.     *CT scans*  
Generally poorly handled. Included pancreatitis, Type B aortic dissection and arocephaly associated with sinusitis and meningitis.
5.     *General X-ray*  
Included a CT of goitre and airway obstruction. X-rays of clostridial infection of foot and cervical fracture.
6.     *Paediatric Basic Life Support on a manikin*
7.     *Rest*

8. *ECGs*  
Included hyperkalaemia, prolonged QT Syndrome and Heart Block.
9. *Miscellaneous Equipment*  
Included questions on a tracheostomy kit, Evac tube and flask of Dextran 40.
10. *A series of investigations from a case of severe pneumonia* (CT, CXR, ECG)
11. *A series of investigations from a case of head injury* (biochem, colonised CSF, CT)
12. *Communication with an actor*  
This involved counselling a registrar who was failing to cope with the sudden death of a young patient.
13. *Rest*

### Cross Table Viva Section

There were 6 structured Vivas of ten minutes each. There were two minutes provided to read a scenario outside each viva room. The viva tables were generally well handled.

The topics were:

- Postoperative hyponatraemia and fitting
- Management of shock which turned out to be anaphylactic.
- Severe head injury.
- 16 year old with acute severe asthma.
- Transport of a critically ill patient from a rural GP setting.
- Burns with smoke inhalation and airway burn.

Common errors included an insistence on rapid sequence induction in the presence of a threatened airway and lack of an organised approach to a common problem (eg. shock, asthma). Clinical indications for intubation in asthma and side-effects of common ICU drugs were handled poorly.

### The Clinical Section

The Clinical Section was conducted at the Royal Adelaide Hospital.

Candidate performance in this section has improved over the last few years. It is a very important section and a common cause of difficulty passing the exam.

Cases encountered included patients with:

#### COLD CASES

- Motor neurone disease
- Aortic Stenosis
- Mitral Regurgitation
- Multiple sclerosis
- Pulmonary fibrosis
- Pleural effusion

#### HOT CASES

- Thoracic cord lesion
- LLL collapse
- Out of hospital arrest
- IVDU with respiratory failure
- Dermatomyositis and weaning difficulty
- Intermediate syndrome from organophosphate poisoning

## WRITTEN SECTIONS

This guide is meant to be an information resource and the views of a practising intensivist. It is not written under exam conditions and does not provide ideal answers. A sensible, senior registrar approach is sought.

Candidates still fail to answer the specific question asked.

## SHORT ANSWER QUESTIONS

1. *List briefly ways in which clinical illness may change the pharmacokinetics and pharmacodynamics of antibiotic therapy.*

There are many reasons for variable antibiotic pharmacology in ICU patients. Critical illness affects drug absorption, distribution and clearance via changes in fluid compartments, organ function and plasma protein concentrations.

The question specifically refers to the effects of critical illness, not genetic polymorphisms, drug interactions etc. The answer required a list. It should have been comprehensive but without too much detail.

### a) Changed pharmacokinetics

- i) Absorption – unpredictable oral bioavailability due to diarrhoea, ileus and potentially slowed IMI absorption due to impaired peripheral blood flow.
- ii) Distribution – volume of distribution commonly increased by increased total body water.  
Decreased protein binding may lead to shortened  $T_{1/2}$  and increased free drug eg. ceftriaxone.
- iii) Elimination: metabolism, biotransformation and excretion.  
Metabolism – may be slowed by acute hepatic impairment or reduced hepatic blood flow.  
Excretion – Nonrenal clearance (eg. hepatic) affected by biliary obstruction, renal clearance impaired by renal failure and variably restored by dialysis (eg. some detail on the effects on aminoglycoside dosing were expected).

- b) Pharmacodynamics refers to the effects of the drug. Effects on organ systems may be both toxic and therapeutic. There is obviously a close interplay with kinetics. The ways that critical illness influences the pharmacodynamics of antibiotics therefore may include:

- i) Antibacterial effect potentially reduced by increased  $V_D$ , impaired tissue blood flow etc or increased by failure to excrete.
- ii) Renal – more susceptible to renal failure because of impaired renal blood flow, dehydration (eg. aminoglycosides, amphotericin).
- iii) Cardiovascular – more susceptible to cardiovascular toxicity eg. bradycardia with vancomycin bolus.
- iv) CNS more susceptible to cerebral toxicity of high dose penicillins.

2. *You are intubating an hypoxic patient with a rapid sequence induction. You are unable to visualise the cords during laryngoscopy. What is your plan to manage this problem?*

This question required a safe management plan that would cover the possible contingencies. The candidate should have mentioned that proper planning and assessment is the key but details of preparation were not specifically asked for.

Thus one approach is:

- (a) If one is able to ventilate the patient:
- optimise laryngoscopy – extra pillow, McCoy blade, laryngeal manipulation etc.
  - consider alternatives to laryngoscopy – fiberoptic laryngoscopy
  - blind nasal, light wand
  - call for expert help
  - awaken patient
- (b) If one is unable to ventilate the patient:
- call for expert help
  - insert guedel airway and attempt ventilation with PEEP
  - insert LMA and attempt ventilation
  - if successful go to (a)
  - if unsuccessful attempt to establish a transtracheal airway
    - retrograde wire, cricothyrotomy, tracheostomy.

It was assumed that suction, SpO<sub>2</sub>, ECG, BP measurements were all preorganised.

References:

- (a) Difficult intubation: An Analysis of 2000 Incident Reports.  
Anaes Intens Care 1993; 21:601-607
- (b) Laryngeal mask airway and the ASA Difficult Airway.  
Anesthesiology 84: 3 ; 686-99.
- (c) The Unanticipated difficult airway with recommendations for management.  
Can. J Anaesthesia, 1998 45:7, 757-776.

3. *On a busy Saturday morning in your fully occupied 14 bed Intensive Care Unit a fire suddenly develops in the electrical switching box beside a central bed. What are the principles of handling this emergency?*

This question was aimed at testing fire drill awareness, a universal requirement. A suggested response is:

- (a) Rapidly remove all patients and staff from the immediate danger area. This means safely disconnecting lines, monitors and ventilators. Move the patients towards the exits and bag the ventilated patients.
- (b) Notify switchboard. Activate fire alarm, state location and nature of fire.
- (c) Shut all doors and windows. Turn off oxygen outlets.
- (d) Attempt to control and extinguish the fire with appropriate extinguishers and fire blankets, provided it is safe to do so.
- (e) If fire is uncontrolled, commence evacuation of the patients via the fire exits.

4. *A 36 week pregnant woman is involved in a car crash and suffers fractures to her left femur and tibia and left ribs 4-8. What are the cardiorespiratory changes in normal pregnancy? How do they effect her response to these injuries?*

This question had two parts. Lists would suffice. For example:

(a) Cardiorespiratory changes include:

- Increased blood, plasma and red cell volume from 6 weeks, maximal at 28 to 32 weeks
- Increased cardiac output (33% by 10 weeks, to 40-50% by 28 to 32 weeks)
- Decreased blood pressure due to low SVR
- Susceptibility to aorto-caval compression develops during second trimester (maximal at 36-38 weeks)
- Cardiac hypertrophy with increased wall thickness and chamber volume.
- Left axis deviation, horizontal heart
- Hb decreased due to relative haemodilution
- Upper respiratory tract oedema and capillary engorgement
- Increased minute volume, respiratory rate,  $VO_2$ , TV, RR. Chronic respiratory alkalosis
- Decreased FRC, RV, TLC, AWR

(b) The clinical effects on her response to the injury include:

- Larger blood volume allows blood loss to be relatively better tolerated
- Susceptible to supine hypotension
- Susceptible to basal atelectasis and hypoxia
- Difficult intubation
- High  $VO_2$  and low FRC means that hypoxia and hypercarbia develop rapidly with airway obstruction, apnoea etc

5. *List the theoretical advantages and disadvantages of coronary artery bypass grafting without cardiopulmonary bypass.*

Coronary artery bypass grafting off bypass is now widely performed. There are numerous techniques for getting access to the grafts and heart (eg. limited thoracotomy, video assisted).

(a) Theoretical advantages include:

- Avoidance of the effects of extracorporeal circulation including - coagulation/kalikrein activation, microembolus of air and platelet clumps to cerebral circulation
- Avoidance of aortic cannulation with attendant risks of arterial embolisation of air or atheroma
- Avoidance of atrial cannulation with attendant risk of atrial injury respectively
- Avoidance of the effects of cardioplegia including K load, fluid load, coronary air embolus
- Avoidance of risks of aortic cross clamping including atheroembolism, myocardial ischaemia
- Decreased costs (less equipment, less staff)

(b) Theoretical disadvantages include:

- Potential for myocardial ischaemia without the protection of cold cardioplegia during grafting.
- Unfavorable operating conditions with a beating heart increasing the risk of anastamotic bleeding, suboptimal revascularisation and myocardial ischaemia

[Information on graft patency, morbidity, mortality and quality of life from proper trials is not yet available]

Current status and future directions of minimally invasive cardiac surgery. *Current Opinion in Cardiology* 1999,14(5); 419-

Minimally invasive coronary bypass: a dissenting opinion. *Circulation* 98(6);495-7

**6. *List the potential complications associated with the management of a patient after intentional corrosive ingestion.***

Potential complications of intentional corrosive ingestion include:

- Acute: Oral, oesophageal, gastric burns of varying thickness
  - Laryngeal oedema and airway obstruction
  - Oesophageal, gastric perforation
  - Shock
  - Haemorrhage
  - Mediastinitis
  - Psychiatric problems
- Chronic/late:
  - Laryngopharyngo fibrosis with airway incompetence and chronic aspiration
  - Oesophageal fibrosis, stricture and stenosis
  - Psychosocial problems
  - Carcinoma

**7. *Nursing staff report that they are suctioning nasogastric feeds from the tracheostomy of a patient with cuffed tube in situ. How will you manage this problem?***

A practical problem. It may be addressed thus:

- (a) Sit the patient up if possible.
- (b) Determine if the patient is actually aspirating NG feed by mixing food dye or methylene blue with feeds and repeat ETT suctioning intermittently.
- (c) Check tracheal cuff pressures and absence of air leak, presence of seal. Ensure appropriate size tracheostomy in situ. Check tracheostomy tube position above carina and that cuff is at least 2 cm below the cords.
- (d) Check position of NG tube in stomach.
- (e) If all the above conditions are satisfactory and the patient still appears to be aspirating, the feeds will have to be ceased and investigations for a tracheo-oesophageal fistula may need to be instigated.

**8. *List your indications and contraindications for the use of the intraosseous needle. What are the risks associated with its use and how may they be minimised?***

Lists were acceptable.

(a) Indications:

- Venous access in collapsed, hypovolaemic or hypervolaemic child with no other venous access after several attempts
- Child up to 6 years of age
- Administration of drugs or fluids

(b) Contraindications:

- Age > 7years (relative contraindication, bone difficult to penetrate)
- Other access available
- No experience of technique

## (c) Risks:

- Osteomyelitis
- Compartment syndrome from fluid extravasation
- Bone marrow embolism

## (d) Minimising risks:

- Training and practice
- Sterile technique
- Establish conventional venous access ASAP and remove needle
- Limb observation

9. *Describe the principles of how the pulse oximeter determines "arterial oxygen saturation". List causes of the false reading of SpO<sub>2</sub>.*

## (a) The candidate should have been aware of the basic principles of pulse oximetry.

Pulse oximetry is based on the Beer-Lambert Law which states that, the concentration of an absorbing substance in solution can be determined from the intensity of light transmitted through the solution, given the intensity and wavelength of incident light, the transmission path length and the characteristic absorbency at a specific wavelength.

To arrive at oxygen saturation, the relative concentrations of reduced Hb and oxyhaemoglobin must be calculated. At wavelengths of 660nm and 940nm there is maximum separation of absorption. These wavelengths also penetrate tissue and LEDs emitting these wavelengths are readily available.

The pulse oximeter thus has two LEDs emitting light of these wavelengths through a vascular bed. A photodiode detector detects the intensity of transmitted light. It rejects the absorption from tissue and venous blood by sensing the pulsatile or AC components and rejecting the fixed or DC component.

Factory calibration is based on nomograms from young normals.

## (b) False readings may be caused by:

- Optical interference eg. abnormal haemoglobin, dye
- Signal artefact eg. fluorescent light
- False assumptions/calibration eg. inaccurate saturation's below 90%

Pulse Oximetry: principles and limitations. American J of Emerg Med 17,1;59-67.

10. *A 30 year old woman has been certified "brain dead". While awaiting organ donation she is hypotensive, polyuric and hypothermic. Outline your management.*

Efficient support of the potential organ donor is an integral part of IC practice. Since we know nothing of this patient's story a back to basics detailed approach to the patient should have included:

## (a) Check airway patency, tube position.

## (b) Ensure adequate ventilation:

- Examination, ABG, CXR ( to exclude pneumothorax/lung injury, hypoxia/hypercarbia)

## (c) Restore circulation with fluid challenge. Assess filling pressures and response to challenge.

- If diabetes insipidus is apparent (eg. urine output >300mls/hr, serum osmolality >300, urine osmolality <300 in the absence of diuretics) give 1ug of DDAVP IV or SC
- If restoration of fluid status does not restore BP and organ perfusion, commence vasoconstrictor infusion (aramine or noradrenaline)
- Moderate hypothermia (35°C) may be well tolerated and require no specific therapy

- Persistent hypotension in the presence of impaired pituitary function, as evidenced by DI. It may be an indication for intravenous corticosteroids and T3. There usually is no time for a random cortisol level
- Maintain fluid and electrolyte homeostasis eg. replacing urine output ml for ml

**11. What is the hepatorenal syndrome? List causes, mechanism, diagnosis and treatment.**

The hepatorenal syndrome is defined as profound oliguria and avid sodium retention in the setting of severe liver dysfunction.

**(a) Causes:**

Can occur in the setting of both acute and acute-on-chronic liver disease of almost any cause eg. hepatitis, gestational liver failure, cirrhosis.

**(b) Mechanism:**

The pathogenesis appears to be purely functional in that recovery of liver function or transplantation of the failing kidney leads to recovery of renal function. Mechanisms implicated include: hyperdynamic circulation with lowering of renal perfusion pressure, activation of the sympathetic nervous system and a combination of precapillary vasoconstriction and post capillary dilatation by vasoactive mediators leading to decreased glomerular ultrafiltration coefficient. Hypovolaemia and raised intra abdominal pressure from ascites may also be factors.

**(c) Diagnosis:**

Is based on history (deteriorating renal function in the presence of severe liver disease) and the combination of avid sodium retention ( $\text{UNa} < 30 \text{ mmol/l}$ ), oliguria, unremarkable urinalysis and sediment, absence of obstruction and exclusion of intravascular volume depletion.

**(d) Treatment:**

Classic teaching is that, in the absence of liver function recovery or liver transplantation, there is no treatment other than renal replacement therapy for established hepatorenal syndrome. Other measures to be considered or experimental are:

- Volume expansion and albumin infusion
- Paracentesis
- Relief of portal hypertension (TIPS, shunt)
- Vasopressin analogs (ornipressin), experimental
- Prostaglandin analogs – experimental

**12. Outline your postoperative management plan for a patient who has just returned from the operating theatre after undergoing bilateral thoracoscopic lung reduction for emphysema.**

Although not stated in the question, it was expected that the plan would only cover the immediate postoperative period. Success of the operation is dependent on patient selection, preparation, surgical skill and ICU care.

It should include:

- (a) Airway/Breathing** – the patient should be extubated as soon as possible to avoid the risks of barotrauma and nosocomial pneumonia. This is facilitated by a light general anaesthetic and thoracic epidural analgesia.
- (b) Circulation**– arterial line for BP monitoring and sampling. BP should be maintained with blood transfusion and low dose vasoconstrictor. Excessive amounts of crystalloid are avoided. Maintenance fluids (eg.  $1 \text{ ml/kg/hour}$  of  $5\% \text{ D} + \text{KCL} + \text{MgSO}_4$ ).
- (c) Analgesia** – thoracic epidural. If ineffective PCA & regular paracetamol.
- (d) Drains** underwater usually no suction.
- (e) Early mobilisation** into chair.
- (f) Antibiotics** as per preop sputum culture or 24 hours IV cephalothin.



- (g) Bronchodilators as indicated.
- (h) Investigations- CXR to check lung expansion.
- (i) ABG to check for hypercarbia.

Lung Volume Reduction surgery for Emphysema. Chest 110(1); 215-8  
 Anaesthesia for lung volume reduction surgery. Current Opinion in Anesthesiology  
 1998,11: 45-9

13. *A 35 year old man, recently returned from an African trek, is admitted with coma, severe hypoxia and dark urine. A thick film of blood shows malarial parasites. Outline your management over the first 48 hours.*

This is a medical emergency with a potentially high mortality due to plasmodium falciparum.

Initial management of acute severe malaria with these features should include:

- (a) Airway – the patient is unconscious so the airway will need to be secured.
- (b) Breathing – hypoventilation associated with the cerebral obtundation will necessitate IPPV to normalise PCO<sub>2</sub>. ARDS is common in this setting and will require titration of FiO<sub>2</sub>, PEEP and ventilatory mode (?PRVC,IRV etc).
- (c) Circulation – shock is not uncommon with severe malaria. Volume loading in clinical studies is usually counterproductive with associated worsening hypoxia. Inotropic support is usually indicated and renal failure may require CVVHD.
- (d) Diagnosis – secondary infection is uncommon, but other precipitants of deterioration should be excluded eg.. pneumonia.
- (e) Definitive therapy with antimalarials. Depending on known sensitivities from the area visited – quinine sulphate may be the treatment of choice (IV loading dose followed by eight hourly doses).
- (f) Invasive monitoring.
- (g) Metabolic support – hypoglycaemia is common.
- (h) Exchange transfusion – not medically justified.

14. *Discuss the pharmacology and place in the management of severe chronic heart failure of:*

- (a) *enalapril*
- (b) *spironolactone*
- (c) *digoxin*

A big question for ten minutes. Candidates were expected to only cover the surface. Some kinetics and dynamics and the role in CCF would be expected, eg:

- (a) Enalapril

Pharmacology: prodrug of angiotension II converting enzyme inhibitor enalaprilat. Long half-life, 11 hours. Well absorbed orally. No IV preparation available. Offset of action due to renal excretion.

Indications- hypertension, CCF, EF<35%

Ag II is now known to have numerous autocrine and paracrine effects. ACE inhibition produces far-reaching effects including-peripheral vasodilation, increased C.O., myocardial-remodeling etc.

Side effects include hypotension, renal failure in the setting of renovascular disease, hypotensive reaction to albumin infusion, hyperkalaemia, and cough.

Its role in severe chronic heart failure is well established in improving symptoms and exercise capacity, and improving survival due to myocardial and cerebral events.

## (b) Spironolactone

Pharmacology: competitive aldosterone inhibitor. Orally well absorbed. No IV preparation. Metabolised in the liver

Produces K-sparing diuresis via aldosterone blockade. Aldosterone most likely has other roles in blood vessels promoting fibroblast proliferation and dehydration.

Side effects include hyperkalaemia, dehydration and gynaecomastia.

Has been shown to produce increased survival in severe CCF (NHYA IV) when added to standard regimen of ACEI and loop diuretic. Recent placebo controlled trial stopped early because of marked benefit. Hyperkalaemia was not a problem.

## (c) Digoxin

Non-sympathetic inotrope. Blocks Na-K ATPase and indirectly increases intracellular Ca. Well absorbed orally. Renally excreted.

Produces:

- increased contractility
- increased myocardial automaticity
- decreased AV conduction.

Plasma concentration increased by amiodarone, verapamil.

Side effects include nausea, vomiting, visual disturbance AV bradycardias, tachycardias (flutter with block, VT, VF...).

O/D or toxicity may be treated with K, Mg, and antibodies.

Its role in CCF is well established in patients with AF to control heart rate, improve mortality, exercise tolerance and symptoms. In-patients with sinus rhythm and severe CCF unresponsive to other therapy, digoxin may produce improvement in symptoms but not mortality and hospital admission rate.

**15. A patient after coronary artery surgery develops severe haemoptysis after inflation of the pulmonary artery catheter balloon. A new infiltrate at the tip of the catheter is seen on chest X-Ray. Describe your immediate management.**

A rapid response in this setting of severe haemoptysis is expected but if there is time an angiogram or bronchoscopy may help to isolate the pulmonary vessel involved. Immediate management may be simple. For example:

- (a) Withdraw catheter 2-3cm and then refloat PA catheter with balloon inflated to occlude the pulmonary artery.
- (b) Insert double lumen ETT to secure the airway and attempt to isolate the affected lung. A single lumen tube advanced into the unaffected side may be a quicker and easier option.
- (c) Transfer to OR for immediate lobectomy if bleeding does not settle.

The application of PEEP has also been reported to stem the bleeding.

## LONG ANSWER QUESTIONS

The questions release information piecemeal and incompletely as in the clinical situation. Specific issues in the specific setting were expected to be addressed rather than broad generalities, eg. "secure airway" does not explain the problems of neck stabilisation or airway obstruction.

### QUESTION 1

*A 50 year old man is brought into the Emergency Department after acute flexion injury to the neck while surfing. He is unable to move both arms or legs and has a sensory level at C4-5. He is a heavy smoker with a history of chronic bronchitis.*

- (a) Outline your initial management.*
- (b) His breathing is laboured with a rate of 40 and with a paradoxical movement. What will you do?*
- (c) Two days later he develops fever, dirty sputum and basal CXR changes. What will you do?*
- (d) Five days later he is orally intubated and has a forced vital capacity of 200 mls. His unstable cervical injury has been managed by tongs and traction. What will you do to facilitate weaning?*
- (e) At 21 days he is ventilator dependent. He appears frustrated and angry. His wife believes that he wished to die and she requests withdrawal of therapy. What will you do?*

Candidates failed to understand the effects of a C4-5 lesion. There has obviously been a quick assessment of the patient so that we are told of paralysis and sensory level. Being a surfing injury, and not a high speed MVA, associated injuries may include hypoxia and near drowning.

- (a) The candidate should have had an appropriate hierarchy of priorities from this point. Textbook lists are inadequate. Actions should have been explained and related to the case. GCS and airway patency should be checked but if a sensory level could be accurately ascertained, the patient is possibly talking and maintaining an airway.*

Breathing will be of prime concern. A level at C4-5, perhaps complete, would produce loss of all intercostal and some diaphragmatic function. With his age and history of heavy smoking it is likely that intubation would be necessary. A clinical assessment of respiration and breathing pattern should be clearly elucidated, not just listed.

A safe technique for intubation should be detailed if the decision is to proceed (eg. blind nasal, 'rapid sequence' or fibre-optic bronchoscopic with in-line traction).

Blood pressure support: bradycardia and relative hypotension are expected. If organ perfusion is adequate, no action is necessary. An associated head injury will necessitate the use of inoconstrictor to maintain CPP or blood loss (eg. from ruptured spleen) will require volume resuscitation.

The candidate should then cover:

- **Diagnosis:**
  - history (recent and past)
  - complete assessment of neurological function
  - survey for other injuries
  - investigations: ( 3 view x-ray),
  - CT or MRI (why, pros and cons),
  - CXR

- Treatment:

- steroids: the NASCIS II study showed motor and sensory improvement with methylprednisolone 30mg/kg bolus and 5.4mg/kg infusion over 23hrs. Criticised widely and not used by all but the evidence of benefit is accumulating.
- surgical Vs medical treatment and early Vs late are undecided issues. Most surgeons would decompress a patient with incomplete lesion and significant canal narrowing
- NG tube - ileus
- IDC - urinary retention leads to bladder problems long term
- Temperature maintenance
- DVT prophylaxis
- Pressure area prevention

2. Paradoxical movement in this setting suggests paralysed intercostals and residual diaphragm function. This produces at least 30% loss of FVC and will mean a poor cough in a supine patient. If he is struggling to breathe, he has no hope of effectively coughing. If the candidate had intubated the patient in (a) that was OK. The waverers should put the tube in and explain their technique in detail. There is limited place for non-invasive ventilation in this setting.
3. A bread and butter ICU problem, ?nosocomial infection, but it has occurred early and may indicate aspiration pneumonia or exacerbation of his bronchitis leading to pneumonia. Is this infection? Other causes, a pulmonary infarct and sepsis elsewhere, should be considered.

The nosocomial pneumonia should be handled by tracheal aspirate, culture, antibiotics and physiotherapy. There is no strong evidence to support the routine use of covered brush, BAL techniques at this stage but bronchoscopy may aid sputum clearance if physiotherapy is ineffective. The likely pathogens and hence choice of antibiotics should be listed. H.flu should be included considering his history.

The next problem is to consider how to prevent further episodes of infection:

- vigorous physiotherapy with assisted coughing
- early tracheostomy and surgical stabilisation should be considered
- can the patient be mobilised into a Philadelphia collar (if he has a stable spine and complete lesion this may be possible)?
- can he be sat up?
- is there a place for an Evac tube or Pitt tube to aspirate secretions from above the cuff?

- (d) A VC of 200ml if correctly measured suggests weaning such a patient will be a major problem. The question does not explain whether the cervical injury is complete or incomplete and stable or unstable. This is central to this management. If the lesion is complete at C4-5 in this mature man with some degree of chronic lung disease, he is going to be very difficult to wean from tracheostomy and will require some degree of mechanical support.

If the lesion is incomplete then with time some recovery may be expected and the aim in the short term would be to maintain spontaneous respiration, prevent nosocomial infection etc.

As in (c), considering surgical stabilisation and tracheostomy are essential. Percutaneous forceps technique of tracheostomy with the neck stabilised in the neutral position is not contraindicated.

General weaning principles should also have been applied (eg. optimise nutrition, treat sepsis, optimise breathing circuit and ventilator, treat abdo distension and lung pathology).

- (e) This ethical and communication question requires resort to basic principles. This is an open scenario because clinical details helping with prognostication are not revealed. The candidate should not conclude that the prognosis is either hopeless or optimistic for functional recovery at this stage or that the wife has his interests at heart or that she knows his wishes.

Suggested steps in handling this problem include:

- (i) Information gathering. As much clinical and radiological information as possible about prognosis and further function should be accumulated. Information should also be gathered about his social setting, his lifestyle and previously expressed wishes, his family's resources to provide therapy at home.
- (ii) Communication. Over a period of time in family conferences this information should be clearly explained. Time should be set aside to sit down with all the close relatives in a quiet environment. Efforts should be made to involve the patient via communication aids (eg. Passy Muir valve, Pitt tube, Lip reading).
- (iii) Specific issues. Causes for the patients agitation should be sought. Depression treated. Financial issues should be addressed with the social worker.
- (iv) Actions then should be based on :
  - respect for human life.
  - respect for human dignity.
  - respect for individual autonomy.
  - respect for social justice.
  - assessment of the benefit and harm of continued therapy.

A competent patient is entitled to withdraw consent to treatment but it is early in his course. The responsibility of the doctor is to ensure that the patient is competent, is fully informed and the treating team has a consensus. This will take time.

**WHEN LIFE SUPPORT IS QUESTIONED EARLY IN THE CARE OF PATIENTS WITH CERVICAL-LEVEL QUADRIPLÉGIA NEJM 1993;328;7;P506-9**

## QUESTION 2

*A 58 year old man is brought in by ambulance moribund with barely palpable pulse and a sinus tachycardia.*

- (a) Outline your management in the first fifteen minutes.*
- (b) His condition improves with therapy. When his wife arrives she tells of his recent hip replacement complicated by a bleeding duodenal ulcer. What are the likely diagnoses? How will you establish the definite diagnosis and why?*
- (c) A large pulmonary embolus is confirmed. What management will you institute?*
- (d) He suddenly collapses. He is pulseless and unconscious with a persistent sinus tachycardia on ECG. What will you do?*
- (e) After successful resuscitation you consider the insertion of a caval filter. List the pros and cons of its use and explain the technique of insertion.*

A case of near electromechanical dissociation in a 58 year old man. This could be caused by hypovolaemic shock, anaphylaxis or cardiogenic shock etc. Therefore the candidate should have started with a comprehensive approach and be directed to specific problems.

(a) An outline is requested but it should contain some rationale, eg:

- A/B if the patient is breathing and talking apply a 100% oxygen mask. If not, bag with face mask and high-flow O<sub>2</sub>. LOC may improve rapidly with BP restoration, but if not, intubation and ventilation will be necessary.
- Quickly assess the patient's volume status (JVP visible?, veins engorged). Establish best venous access possible (peripheral IV, external jugular, femoral). If the patient appears hypovolaemic, commence bolus of fluid. Continue fluid boluses until filling pressures appear adequate as judged by rise in JVP, CVP (or PAOP) or occurrence of worsening respiratory distress (? pulmonary oedema).
- If the patient is not hypovolaemic on arrival or remains hypotensive despite achieving adequate filling pressures give 1mg increments of amine and commence an inoconstrictor infusion.
- As soon as possible insert an intra-arterial cannula. It is possible that the central BP is adequate.

Meanwhile, a primary survey should be undertaken to determine the cause and a detailed history sought. Life-threatening injuries are excluded. The patient is quickly examined from head to foot for signs of anaphylaxis (erythema, wheals etc), cardiac failure or tamponade (venous congestion, raised JVP). Tension pneumothorax or sepsis (hot, flushed, local signs) etc.

Investigations and initial treatments should be guided by the history and signs eg. intercostal catheter, pericardio-centesis.

- (b) This provides some help, but is not definitive. Possible causes still include pulmonary embolus, hypovolaemia from bleeding DU, myocardial infarction etc. A progression from simple/quick investigations to more complex but specific/diagnostic investigations should be outlined. There should have been a sense of appropriate priorities.

If there are signs of GIT bleed with hypovolaemia, then fluid resuscitation, NG tube insertion, endoscopy, FBC and coag screen will be indicated along with anti-ulcer therapy and perhaps surgery.

If there are signs of acute myocardial infarction, in the setting of recent DU, angiogram and angioplasty would be a preferable course perhaps after urgent echo.

If there are signs of massive pulmonary embolus (right heart failure), and initial tests are supportive (right heart strain on ECG, oligemic lung on CXR, distended RV on echo), a spiral CT would be indicated with commencement of IV heparin.

- (c) At last a diagnosis. Management at this stage would depend on his clinical state and include supportive measures (O<sub>2</sub>, inotropes, ulcer prophylaxis) and specific (heparin, IVC filter). Dobutamine (lesser forms of haemodynamic disturbance) and noradrenaline (severe RV failure) are the only inotropic agents supported by evidence in this setting. Adrenaline may be the only agent immediately available on the arrest trolley though.
- NB. Excessive fluid may distend the RV and worsen the situation.

- (i) If resuscitated adequately and haemodynamically stable, IV heparin would be the mainstay. Venous duplex scans may help to gauge further thrombus load. The history of DU and a large mobile DVT may be an indication for insertion of a caval filter but lysis appears contraindicated if the history is confirmed.
- (ii) If the patient is haemodynamically unstable and on large doses of vasopressor (preferably noradrenaline at this stage), embolectomy on bypass is indicated. Fibrinolysis is contraindicated by recent surgery and DU.

- (d) Ideally, intubate and ventilate with 100% O<sub>2</sub>, turn up the noradrenaline, repeat fluid bolus, commence CPR if indicated and transfer immediately to the OT for surgery. In the absence of cardiac surgery facilities, one is left with a Trendelenberg operation by a general surgeon or continued medical therapy or risk lysis.

**(e) The caval filter:**

The caval filter is effective in preventing pulmonary embolism from the lower limbs, but is not without problems.

- PROS:** Prevents PE and is particularly indicated in patients with:
- Contraindications to anticoagulation (HITTS, haemorrhage)
  - Recurrent embolism despite anticoagulation
  - Free floating IVC thrombus
  - Immediately after embolectomy when heparin is contraindicated

- CONS:**
- Requires expertise and equipment
  - IVC obstruction and long term venous stasis of the lower limbs may result
  - Technical problems:
    - misplacement obstructing renal veins
    - caval perforation
    - fracture end embolisation
  - In one study of patients with DVT the two year mortality was not reduced and venous stasis of limbs was common

**Technique:** Commonly the filters are placed by interventional radiologists using sterile technique under I.I. control. There are various types including removable filters. The umbrella type is outdated. Greenfield and the birdnest filter are still used in many parts of the world. If there is extensive clot in the iliofemoral veins, upper extremity access (IJ or SV or axillary) is indicated via a Seldinger technique. The delivery system is advanced through an introducer after localisation of renal veins by contrast venography and the filter is placed below the renal veins.

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