



## Faculty of Intensive Care

### REPORT OF GENERAL FELLOWSHIP EXAMINATION

JULY/AUGUST 2000

This report is prepared to provide candidates, 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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Twenty-six candidates presented for this examination. Thirteen were successful.

#### ORAL SECTIONS

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

There were thirteen stations with three rest stations (two of these were after the interactive stations). Most candidates passed this section. A systematic approach to the types of investigations examined was more likely to maximise the candidates' score. The stations included:

1. *Chest X-rays* including bibasal atelectasis, tension pneumoperitoneum, pericardial effusion, PAC in aberrant SVC and pneumonia. A list of abnormal findings and further investigations was often requested.
2. *ECGs* including torsades de pointes, pulmonary embolism, VT and VF.
3. *Biochemistry* including blood profiles of hepatitis, TTP and metabolic acidosis.
4. *Procedure station* involved practical management of cardiorespiratory arrest secondary to upper airway obstruction.
5. *Rest Station*
6. *General radiology* including a CT of a thoracic aortic aneurysm, degenerative cervical spine in a patient after MVA, and a normal CT head in a patient with severe headache. Abnormal findings and further investigations were requested.
7. *Paediatric investigations* including gastric dilatation after MVA, widened mediastinum after horse kick, pleural effusion and pneumothorax, and subdural haematoma on CT associated with limb fractures in a drowsy infant.
8. *Arterial Blood Gases* including simple and mixed disorders involving metabolic alkalosis, respiratory alkalosis and respiratory acidosis.
9. *Rest Station*

10. *Chest X-rays* including hyperinflated lungs, pulmonary oedema, pericardial effusion and ruptured thoracic aorta.
11. *Miscellaneous Equipment* including an underwater seal drainage system, pads for pacing/defibrillation, activated charcoal with sorbitol, and a heat & moisture exchanger. Roles, advantages and disadvantages were requested.
12. *Communication with an actor*. This involved explanation to the mother of a young girl who has suffered a severe head injury and has just been found to have absent brainstem reflexes.
13. *Rest Station*

### **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:

- Management of hypoxic brain injury after heroin OD.
- Management of a 32 week pregnant woman with staph. Pneumonia and infective endocarditis.
- Renal failure in a post-partum patient with known scleroderma.
- Ventilatory management of severe asthma.
- ICU management of a patient with severe pancreatitis and its complications.
- Management of shock after major trauma.

### **The Clinical Section**

The Clinical Section was conducted at the Royal Melbourne Hospital.

Only ten out of twenty-six candidates passed this section. Candidates should listen carefully to the introduction given by the examiners and direct their examination accordingly. Patients were usually presented as problem solving exercises.

Cases encountered included patients with:

#### **COLD CASES**

- Myasthenia gravis
- Aortic Valve Disease
- Mitral Regurgitation
- Multiple sclerosis
- Peripheral neuropathy
- Polycystic kidneys

#### **HOT CASES**

- Multiple trauma
- Prolonged respiratory failure
- Multisystem organ failure
- Bacterial endocarditis
- Cushing's syndrome and weaning difficulty
- End-stage renal failure & cardiac surgery

## **WRITTEN SECTIONS**

It is imperative to answer the specific question asked. A structured, orderly response considering all aspects of management is required.

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.

## **SHORT ANSWER QUESTIONS**

### ***1. List the clinical effects of severe accidental hypothermia.***

Definition: "severe" (usually mild 32-35, moderate 28-32, and severe < 28C). Accidental implies spontaneous decrease in core temperature, usually in a cold environment (more common in elderly, neonates, unconscious, exhausted, hypothyroid etc). Mortality significant. Signs are modified by associated injuries, medications, extremes of age, etc.

Temperature control lost (become poikilothermic, cooling to ambient temperature)

Cardiac :       arrhythmias (eg. bradycardia, AF and VF)  
                  decreased mean blood pressure, contractility, cardiac output

Respiratory:   decreased respiratory rate, respiratory acidosis

CNS:           variable effects on mentation and motor function; impaired judgement, disorientation  
                  hyporeflexia

Haematology: coagulopathy, platelet dysfunction

Gastrointestinal: pancreatitis

Renal: polyuria, dehydration, ARF

### ***2. List the mechanisms of central venous catheter infection, and list the measures you undertake to prevent this infection.***

Usual source of infection is the insertion site, hub, infusate or haematogenous spread from distant site. Major mechanisms causing CVC infection are therefore:

#### **(1) Contamination during insertion. Prevented by:**

- Sterile precautions during insertion (gloves, gown, mask, appropriate cutaneous antiseptic)

#### **(2) Contamination of insertion site after insertion:**

- Use of subclavian site for insertion (rather than jugular or femoral)
- Adequately fix catheter to prevent movement
- Use appropriate combination of dressings and observation of site (change dressing if soiled)
- Avoid prolonged connection of solutions prone to contamination (lipid, propofol)
- Remove catheter as soon as need for it diminishes

#### **(3) Subsequent contamination due to breaking of sterile connections (multi-flows, 3-way taps):**

- Limit number of lumens, decrease breaks in system, clean injection ports before accessing system (eg. Alcohol), use alternate route for blood transfusion
- Use of anti-microbial impregnated catheters

#### **(4) Subsequent contamination from systemic infection elsewhere:**

- Aggressive treatment of other infections, remove catheters as soon as possible
- Use of anti-microbial impregnated catheters

**3. List the indications for and contraindications to the use of non-invasive ventilation in acute respiratory failure.**

Non-invasive ventilation usually encompasses face mask (full or nasal) CPAP with or without additional ventilatory support.

Indications can be based on physiology or on specific aetiological cause:

**1. Desire to provide increased respiratory support without the need for endotracheal intubation.**

The respiratory support may take the form of:

- increased FIO<sub>2</sub> (closed circuit)
- increased End Expiratory Pressure, or
- increased inspiratory pressure (as continuous positive airway pressure or additional inspiratory support in the form of pressure or volume assisted breaths)

**2. Desire to delay or prevent the complications and morbidity associated with mechanical ventilatory support via an endotracheal tube.**

The specific types of respiratory failure that may benefit from Non-Invasive Ventilation should be listed (ideally with some indication of the level of evidence in the published literature to support the approach).

Conditions that may benefit include:

- Hypercapnic respiratory failure:
  - acute exacerbation of COPD, post-extubation acute respiratory failure, respiratory failure in patients with cystic fibrosis, patients awaiting lung-transplantation, patients who are not candidates for intubation (eg. DNR?, terminal illness).
- Hypoxaemic respiratory failure
  - cardiogenic pulmonary oedema, postoperative respiratory failure, post-traumatic respiratory failure, respiratory failure in AIDS, patients who are not candidates for intubation.

A list of contraindications should include those situations that make the potential disadvantages or complications of non-invasive ventilation worse. Differentiation into absolute and relative is arbitrary.

- lack of experience in technique (technical & mechanical problems), intubation required for other reasons including airway protection and sputum clearance, uncooperative patients (confused, comatose, reluctant), full stomach (risks of aspiration), local trauma (nose/face), fractured base of skull, oesophageal surgery (risks of gastric/oesophageal insufflation)

**4. What useful information can be gained from respiratory pressure-volume loops in the management of the ICU patient?**

The usefulness of the information depends on the awareness of the limitations of the technique.

PV loops require either steady state (super-syringe technique) or quasi-steady state techniques (slow constant flow) to minimise effects of flow characteristics on pressure. The use of non-constant flow requires mathematical/computerised correction of the curve.

Traditionally curves have been performed from zero PEEP, and are dependent on the recent ventilatory history.

Assumptions have been made that the change in slope at the lower end of the inspiratory curve (lower inflection point) reflect recruitment of all/most/some of the collapsed and recruitable alveoli.

These assumptions are being questioned. This point/zone of inflection has been proposed to be used as a way of choosing a level of PEEP to allow recruitment or prevent de-recruitment. Despite some published literature seeming to support this approach, many limitations have been raised (eg. recent ventilatory history, variability due to underlying lung disease (primary versus secondary) presence of decreased compliance of abdominal or chest wall, greater importance of expiratory component of curve, etc.).

The Upper Inflection Point has been proposed as a way of detecting overdistension of the lung. Many published studies have suggested that this is an oversimplification, as many areas of lung may already be overdistended (eg. CT studies) before the UIP is reached. Setting the ventilator to prevent "overdistension" is possible but may not be clinically relevant.

Inflection points/zones on the descending part of PV curve may eventually become useful to titrate levels of PEEP to prevent de-recruitment.

The shape of the dynamic PV curve (and its deviation from expected) may allow some degree of estimation of the magnitude of the patient's (as opposed to mechanical) work of breathing, or the presence of airway obstruction.

#### **5. Outline the indications, contraindications, side effects and details of administration for benzyl penicillin, ciprofloxacin and cotrimoxazole.**

##### ***Benzyl penicillin:***

Indications: treatment of infection with presumed susceptible organisms (eg. Strep pyogenes, clostridium [Gram positive bacilli, Gram positive and Gram negative cocci and spirochaetes]). Prophylaxis of bacterial endocarditis and orthopaedic trauma or surgery. Prophylaxis after splenectomy.

Contraindications: previous adverse reactions (including allergies) to penicillin; infection with organism not susceptible; high dose with renal dysfunction (CNS toxicity).

Side effects: include sensitivity reactions, superinfection (including pseudomembranous colitis), GI upset, fitting, fever, interactions with other drugs, physical incompatibilities.

Administration: IV or IM. Reconstituted powder. Adult dose 1.2 to 24g over 24 hrs in 4 – 6 doses.

##### ***Ciprofloxacin:***

Indications: treatment of infection with presumed susceptible organisms (Gram positive cocci and Gram negative bacilli including adjuvant or sole use for resistant organisms [eg. MRSA, pseudomonas, legionella]).

Contraindications: previous adverse reactions (including allergies) to quinolones (including nalidixic acid), pregnancy, children (<18 years).

Side effects: include sensitivity reactions, superinfection (including pseudomembranous colitis), tendon rupture (esp. with steroids), GI upset, CNS stimulation, interactions with other drugs (especially via inhibition of P450 system), haematologic/liver enzyme effects.

Administration: IV - Adult dose 200-300 mg over 60 minutes, every 12 hours. Oral – Adult dose 250-750 mg every 12 hours.

##### ***Cotrimoxazole:* (sulphamethoxazole + trimethoprim)**

Indications: Seldom used as first line therapy in ICU but may be used for treatment of infection with susceptible organisms (Gram positive cocci and Gram negative bacilli including adjuvant or sole use for resistant organisms [eg. pseudomonas, xanthomonas, pneumocystis]), and pneumocystis pneumonia (HIV, bone marrow transplant).

Contraindications: allergy to sulphonamides or trimethoprim (including allergies), blood dyscrasias, marked renal or hepatic impairment, megaloblastic bone marrow.

Side effects: include sensitivity reactions (eg. photosensitivity), superinfection (including pseudomembranous colitis), GI upset, CNS effects, interactions with other drugs, haematologic effects.

Administration: IV - Adult dose 75-100 mg/kg per day of SMX (15-20 mg/kg/day TMP) in 3 or 4 doses. Oral (tablets or syrup) – Adult dose 800mg/160mg (DS) every 12 hours, (? daily for prophylaxis).

**6. How would you determine the aetiology of severe hypercalcaemia? List the treatments appropriate for each aetiology.**

Major causes of hypercalcaemia are:

Increased calcium release from bone

- erosion of bone (malignant neoplasms eg. lung, breast, haematologic (include multiple myeloma), head & neck, renal, prostate)
- release of calcium from bone with immobilization
- humoral stimulation of calcium release (mainly PTH but also other hormones)

Increased calcium intake

- calcium supplements, milk alkali syndrome

Both of these are augmented by the presence of renal impairment.

Aetiology determined by combination of history, examination and investigations.

*History and Examination*

- clinical features relate to symptoms due to hypercalcaemia (protean) and those due underlying cause (specific or general of malignancy, immobilization, diet and medications)

*Investigations*

- to confirm malignancy or bony involvement (Xrays of chest, spine etc.)
- to assess bone turnover (alkaline phosphatase, urinary hydroxyproline)
- to assess level of PTH

Treatment is dependent on underlying aetiology, but general measures are aimed at minimising calcium entry into and maximising exit from the circulation :

1. Increased calcium excretion

- volume resuscitation to restore intravascular volume and tissue perfusion (usually normal saline, also inhibits calcium reabsorption in renal tubule)
- frusemide (increase calcium filtration and decreased reabsorption). Aim ? 200-300 ml/hr.

2. Reducing calcium release

- biphosphonates (eg. etidronate) are absorbed to hydroxyapatite crystals and inhibit bone resorption and formation and inhibit osteoclast activity. Administered intravenously; onset of action 24-48 hours.
- calcitonin is less effective. Inhibits osteoclast activity and increases calcium excretion. Parenteral administration but faster onset of action (6-24 hours).
- plicamycin, gallium also used. Inorganic phosphate may be effective (multiple mechanisms) but risks calcium precipitation
- glucocorticoids useful in some scenarios (excess intake or production of Vit D; haematologic malignancies [tumouricidal effects])

3. Others

- correction of other electrolyte abnormalities (eg. K, Mg)
- removal of offending drugs (eg. thiazides, Vitamins A & D, calcium)
- restriction of calcium intake
- mobilisation (to reduce calcium release from bone)

## 7. *Outline your ICU management of an ICU patient with ventricular tachycardia.*

Pulseless VT: managed as per cardiac arrest protocol (immediate unsynchronised defibrillation [up to 3 sequential shocks if necessary], followed by CPR, intubation/IV/oxygen, consider antiarrhythmics [lignocaine, amiodarone, potassium and magnesium], administer adrenaline 1 mg every 3 minutes, exclude reversible causes [5Hs and 5Ts]).

VT with a pulse: if deteriorates or unstable haemodynamically manage as for pulseless VT. If stable administer oxygen/obtain IV access and rapidly exclude reversible factors (including wire/PA catheter in RV, hypokalaemia, hypomagnesaemia, others as indicated by a systematic review to exclude other reversible causes. Drug therapy according to scenario but useful drugs include lignocaine (for ischaemia/post cardiac surgery: 1-1.5 mg/kg IV then infusion), procainamide (50 mg/min to max of 17 mg/kg), sotalol (1 mg/kg) or amiodarone (5 mg/kg over 20 minutes).

## 8. *Compare and contrast the pharmacodynamics of dopamine and dobutamine.*

Pharmacodynamics imply what the drug does to the body. Consideration should be given to mechanism of action, effects on various organs, relationship of dose to effect, indications for use, and type of adverse effects. These drugs are essential parts of the intensivist's armamentarium, and a good level of understanding should have been displayed.

### ***Dopamine:***

- Immediate precursor of noradrenaline, and also serves as a neurotransmitter in central and peripheral nervous systems.
- Doses of 0.5 to 2 mcg/kg/min (via stimulation of DA-1 and DA-2 receptors) increase renal blood flow, urine flow and sodium excretion (inhibit sodium resorption in proximal tubules)
- Haemodynamic effects are due to noradrenaline release (up to 50%) and direct stimulation of alpha, beta and noradrenergic receptors. Can lose effect with time (due to depletion of noradrenaline stores in periphery and heart).
- Doses of 2-5 mcg/kg/min increase cardiac contractility and cardiac output with minimal change in heart rate/BP/SVR. Increasing dose up to 10 mcg/kg/min increase CO/HR and BP.
- Doses above 10 mcg/kg/min result in increasing alpha adrenergic mediated vasoconstriction.
- Can increase intrapulmonary shunt (increase CO), but pulmonary vasoconstriction can occur.
- Dopamine also stimulates receptors in the zona glomerulosa of the adrenal cortex to decrease aldosterone secretion.
- A selective increase in renal and splanchnic blood flow occurs, and low doses have been thought to prevent the vasoconstrictive effects of other agents. The clinical significance of these effects are controversial (?harm to GIT via shunting from mucosa).
- Dopamine inhibits TSH and prolactin release as well as other potential negative effects on anterior pituitary function.
- Other side effects include nausea/emesis, tachyarrhythmias (particularly AF), anginal pain, profound vasoconstriction (including if local extravasation [treat with phentolamine]), and impairment of hypoxic ventilatory drive.
- Used to increase cardiac output and as a mild vasopressor (cardiogenic or septic shock), and as a diuretic (no evidence to support renal protective role).

### ***Dobutamine:***

- Racemic mixture of + and - isomers. + isomer stimulates both beta-adrenergic receptors. - isomer is potent selective alpha-1-adrenergic agonist. No indirect stimulation of receptors. Metabolite (3-O-methyldobutamine) is potent inhibitor of alpha receptors. Net effect is balance between various receptor effects.

- At commonly used dose ranges (2-15 mcg/kg/min) increases contractility, with little effect on HR at doses < 10 mcg/kg/min. Usually little effect on SVR and PVR as balance between alpha-1 and beta-2 effects (CVP and PAWP usually decrease). Some tolerance with time but less than with dopamine.
- Enhances urine output by increasing cardiac output. No other significant metabolic or endocrine effects.
- Side effects include dysrhythmias (less than dopamine), tachycardia, headaches, anxiety, tremors, changes in BP.
- Used to increase cardiac output without need to effect peripheral resistance (cardiogenic or septic shock) or desire to have metabolic/endocrine effects of dopamine. Also used to assess for myocardial ischaemia (stress test).

9. *List the advantages and disadvantages of three commonly used techniques for percutaneous tracheostomy.*

Commonly used techniques include: Ciaglia, Griggs (portex), a combination of these, and the Translaryngeal approaches.

***Ciaglia:***

Advantages: initial technique, widely used, well known, well documented complication rate (low), gradual dilatation, able to insert any type of tracheostomy tube

Disadvantages: need experienced operator and airway operator, endotracheal tube positioning may damage vocal cords or lose PEEP/minute ventilation/protection of airway, takes minutes to dilate and spray of blood stained respiratory gases with each inspiration unless hole completely covered each time, damage to posterior wall of trachea with initial perforation and subsequent dilatations (? minimised by use of bronchoscope)

***Griggs (Portex):***

Advantages: less steps in technique, faster dilatation (may be used in emergency), able to insert any type of tracheostomy tube

Disadvantages: need experienced operator and airway operator, needs sterilization of forceps if previously used, endotracheal tube positioning may damage vocal cords or lose PEEP/minute ventilation/protection of airway, more abrupt dilatation (may cause more tracheal damage), spray of blood stained respiratory gases with each inspiration unless hole completely covered, damage to posterior wall of trachea with initial perforation and subsequent dilatations (? minimised by use of bronchoscope), may want to insert different tracheostomy tube (wasting tube in pack)

***Translaryngeal:***

Advantages: well documented complication rate (very low, especially bleeding), has been safely used with marked coagulopathy, initial tracheal puncture under vision from inside trachea, avoids damage to posterior wall of trachea, allows ventilation (separate tube) throughout procedure, can be done as one person technique

Disadvantages: less widely known technique out of Europe, more fiddly technique, need experienced operator, requires light source and scope (rigid or flexible), ventilation may be difficult with small endotracheal tube, pulling through the tracheostomy tube may damage vocal cords, only able to insert one type of tracheostomy tube (not tube with inner cannula), need to use different technique to change type of tube

10. *List your indications for the use of corticosteroids in the management of refractory shock.*

Candidates should demonstrate a good understanding of the background and current interest in this area. The use of corticosteroids during refractory shock is undergoing a resurgence of interest following the publication of a number of interesting papers. A large RCT assessing the use of high doses of steroids (eg. dexamethasone [Shock Pack 120mg] or methylprednisolone [2g]) in septic



shock was associated with no overall benefit, and indeed a higher mortality in the subgroup with renal impairment. More recently, interest has developed in the use of more physiologic doses of corticosteroids (eg 120 to 240 mg of hydrocortisone) to treat a presumed inadequate intrinsic cortisol response in the presence of refractory shock. The aetiology of the cortisol deficiency could be due to adrenal, pituitary or hypothalamic.

Typical indications for physiologic doses (bolus or infusion) could include:

- known previous steroid dependence,
- recent steroid administration (eg. course for > 1 week in last 6 months),
- shock associated with coagulopathy (adrenal or pituitary haemorrhage more likely),
- prolonged inotrope/vasopressor dependency (> a few days),
- use of very high doses of inotropes/vasopressors to maintain acceptable goals.

#### ***11. Discuss the mechanism, clinical symptoms and management of upper respiratory tract injuries due to burns.***

Upper respiratory tract burns can be life-threatening unless appropriately recognised and treated.

Severity of inhalational injury has been related to various factors: heat of inhaled gases, composition of gases (presence of particles, steam and toxic products), duration of exposure, and pre-injury state.

Most of the upper respiratory tract injury is due to the thermal insult (augmented by duration of exposure).

Initial symptoms may relate to associated injuries (facial burns), early oedema (intra-oral, pharyngeal, supraglottic/glottic/subglottic) with respiratory distress secondary to airway obstruction and increased work of breathing (tachypnoea, indrawing of soft tissues, tracheal tug), and patient may be coughing or spitting carbonaceous material (signs are those of upper airway burn).

Management includes that of associated systemic effects such as burns to body (hypovolaemic shock etc), and inhalation of toxins (carbon monoxide, cyanide etc.).

Management of the airway includes appropriate positioning of patient (eg. sitting up), close monitoring, and early definitive management of airway patency. Oedema worsens over the first few hours (persists for days) and may rapidly cause airway obstruction in untreated patients. Elective intubation should be considered early. A safe technique which took into account the potential for full stomach and difficult intubation was expected to be detailed.

#### ***12. What is the role of cardioselective betablockers in the management of severe heart failure in ICU?***

Cardioselective betablockers (eg. atenolol, metoprolol & practolol) have less effect on the beta-2 receptors (less vasoconstriction and less bronchoconstriction). No specific benefits of any subgroup of beta-blockers has been confirmed in the management of severe heart failure.

The candidates should be able to discuss the complex role of betablockers in heart failure. The role of betablockers in heart failure management is complex enough outside of the ICU. This has been clarified further in the last few years by publication of articles confirming the benefit of addition of betablockers to the conventional heart failure regimen (ACE inhibitor + diuretics) in the outpatient setting (with decreased symptoms, slowing progression, improving LV function and even improving survival). These benefits have been demonstrated initially with carvedilol (non-selective betablocker) and more recently with metoprolol (cardioselective betablocker). Titration of the medication needs to be slow and judicious. It is unknown whether similar benefits can be obtained in the ICU setting, especially given the beneficial effects of short-term administration of inotropic agents in dilated cardiomyopathy.

Betablockers may have some benefit in the setting of tachycardia (increased resting sympathetic tone), but many patients may experience worsening of symptoms. Success of treatment of

arrhythmias with betablockade depends upon the magnitude of the coincident decrease in contractility (other agents may be preferred eg. amiodarone). Prevention of sudden death (malignant ventricular arrhythmias) may be achieved.

Treatment of myocardial ischaemia with betablockers may have beneficial effects (oxygen requirements, improved relaxation, decreased arrhythmias).

Betablockade may be beneficial in the setting of hypertrophic cardiomyopathy (with diastolic dysfunction), by decreasing myocardial oxygen consumption, decreasing ischaemia and improving relaxation (lusitropy).

### ***13. Outline the role of monitoring in the management of upper airway obstruction.***

Monitoring should be considered as either part of routine examination (clinical monitoring) or requiring additional equipment or investigations. Assumed in this case that the presence of an endotracheal tube or tracheostomy would prevent an upper airway obstruction.

Clinical: essential part of monitoring. Clinical criteria more likely to lead to decision to intervene. Consider importance of assessment of level of consciousness, extent of obstruction (soft tissue indrawing – supraclavicular, tracheal tug, intercostal muscles), and ability to cope with increased work of breathing (tachycardia, tachypnoea, sweating).

Equipment: pulse oximetry (limited information, better with lower FIO<sub>2</sub>), ECG (rhythm, ischaemia), capnograph (respiratory rate, pattern of expiratory flow), invasive pressures (IA/CVP/PAWP – may help assess extent of intrathoracic pressure change and therefore work of breathing).

Investigations: arterial blood gases (direction of change may assist in decision to intervene eg. CO<sub>2</sub>/pH).

### ***14. What are the implications for ICU practice of the increasing incidence of antibiotic resistance?***

More focus on techniques for prevention and control:

- hand washing practice, placement of hand basins, use of gloves, use of protective clothing
- isolation techniques (standard precautions, transmission based precautions [airborne, droplet, contact])
- increasing need for surveillance

More focus on judicious use of antibiotics (individual versus community needs)

- appropriate initial cover, tailor quickly according to results of investigations, stop as soon as no longer needed
- reserve/restrict use of some antibiotics (eg. vancomycin) with use of alternatives whenever possible

Need to alter antibiotic prescribing habits:

- initial antibiotic regimen may not cover infecting organism
- community acquired organisms have more resistance eg. penicillin no longer always useful against streptococcus or haemophilus
- third generation cephalosporins lose value after first week in hospital
- may have to use uncommonly used antibiotics (intravenous cotrimoxazole)
- broader antibiotic use may increase superinfection (C difficile, fungi, etc.)
- antibiotics used for prophylaxis may need to change accordingly (? vancomycin for cardiac surgery)

Increased cost:

- newer antibiotics or combinations used to treat some resistant infections
- additional susceptibility testing required
- invasive lines impregnated (antibacterial) or changed more frequently
- ? prolonged ICU and hospital stay of nosocomial infections

Have no available antibiotics to treat infections with some resistant organisms.

**15. Comment briefly on the statement: "Isotonic saline is an inappropriate fluid to use in the management of the patient with diabetic ketoacidosis".**

As with any drug/fluid there are problems associated with the use of normal saline as the sole fluid to resuscitate the extracellular fluid deficit of DKA. Diabetic ketoacidosis is associated with a number of metabolic disturbances, but most of the acute clinical problems are due to a lack of insulin (hyperglycaemia, ketone body formation) and resultant osmotic diuresis (severe volume depletion [loss of water and sodium], total body electrolyte depletion [eg. K, Mg, PO<sub>4</sub>], lactic acidosis, renal insufficiency). The major contributors to the initial metabolic acidosis are the presence of ketone bodies (increased anion gap), lactic acidosis (increased anion gap), and hyperchloraemia (normal anion gap). The first two of these will be adequately treated by intravascular volume expansion and administration of exogenous insulin. Administration of isotonic saline (0.9% sodium chloride) may result in delayed correction of bicarbonate (ie. persistence of metabolic acidosis), now due predominantly to hyperchloraemia (normal anion gap).

Delayed correction of bicarbonate:

- may increase the time that the patient will need to be monitored closely (potentially confusing assessment patient response to treatment)
- increases the minute ventilation (and work of breathing) required to maintain steady state (lower CO<sub>2</sub> for a given pH)
- increases the temptation to administer exogenous bicarbonate (with associated risks of hypokalaemia, hypophosphataemia, hypernatraemia etc.)

Alternative crystalloid solutions are available (eg. hartmanns/ringers lactate/plasmalyte/hypotonic saline) and should be considered early in the fluid resuscitation of these patients. Choice of fluid should be based on the response of the patient to therapy (ie. ongoing, repeated assessment of Na [corrected for glucose], K, HCO<sub>3</sub> and Chloride).

## 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. "intubate" does not explain the technique chosen or the potential problems of difficult intubation. An organised/systematic approach is expected.

### QUESTION 1

*A 44 year old man, with morbid obesity (175 cm tall and 210 kg) presents to the Emergency Department with respiratory failure. He is obtunded with an arterial blood gas (ABG) showing pH 7.25, Pa CO<sub>2</sub> 82 mmHg and PaO<sub>2</sub> 53 mm Hg. CXR reveals cardiomegaly and clear lung fields.*

**(a) Describe your management of this problem in the first 24 hours.**

Management includes history and examination, investigations (appropriate and interpreted) and ongoing therapy (including triage, monitoring, pharmacology and non-pharmacological interventions).

**ABG** information given confirms hypoxic and hypercapnic respiratory failure (on some unspecified level of supplemental oxygen), with an acute on chronic respiratory acidosis (with a compensatory metabolic alkalosis [calculated  $\text{HCO}_3$  of 36]).

**Cardiomegaly** could be due to an AP portable, semi-erect film, but the cardiac enlargement should not be discounted (? cardiomyopathy, ?? pericardial effusion). Obtundation should not be assumed to be due to the hypercapnia.

The **goal** of overall management should be to ensure safety of the patient (attention to ABCs), support the patient (posture: upright or on side, consider non-invasive ventilatory support), and identify and treat any specific reversible causes.

**History and examination** should suggest/exclude many diagnoses including: ischaemic heart disease, cardiac failure (left and right sided), chronic obstructive lung disease, venous thromboembolism, respiratory tract infection, CNS disorder (stroke/haemorrhage), diabetes (and DKA/Hyperosmolar Hyperoncotic Non-ketotic Coma) --or other endocrine problem (eg. hypothyroidism) and the potential for drug related problems (prescribed or over the counter eg. codeine).

Simple investigations should be ordered and reviewed to assist above differential diagnosis and assist treatment (eg. blood glucose, electrolytes, full blood examination, ECG) and to confirm suspicions.

Treatment should be directed at clinical suspicions (appropriate antibiotics [drugs and doses], heparin or equivalent [prevention or treatment], bronchodilators [including steroids] etc.).

Discussion of attempts to prevent intubation should be provided (difficulty with intubation, and risks of ventilation higher). Non-invasive ventilation is covered in short answers, but general principles should be mentioned.

*It is day 4. He is intubated and ventilated, and no precipitant was found for his respiratory failure. CXR reveals an obscured left hemi-diaphragm and new infiltrates behind the heart.*

**(b) Outline your management.**

Ongoing management now relies on reversal of factors resulting in initial requirement for ventilation (predominantly fatigue by exclusion), removal of factors keeping him ventilator dependent, and consideration of techniques to prevent and treat left lower lobe collapse consolidation.

Reversal of initial fatigue will occur with adequate provision of rest (including sleep, and minimization of imposed work of breathing [eg. an adequate sized ETT (probably at least 8 mm), the use of the smallest amount of work to trigger the ventilator (eg. flow triggering), and the use of adequate amounts of ventilatory support (eg. pressure support, or similar mode)]. The patient will need to be awake as much as possible during the day (using appropriate sedation regimen if necessary overnight).

Left lower lobe collapse of some form may be minimized by the use of higher levels of PEEP, appropriate posturing (including semi-prone and prone), and the at least intermittent use of adequate tidal volumes (eg. sigh or IMV breath). The possibility of nosocomial pneumonia and other differential diagnoses (eg. pulmonary emboli) needs to be entertained, and excluded if appropriate.

*It is day 7 and he has had a tracheostomy performed. He is on SIMV 12 breaths by 800 ml tidal volume, with an FIO<sub>2</sub> of 0.6 and his ABG is now pH 7.49, PaCO<sub>2</sub> 49 mmHg and PaO<sub>2</sub> 159.*

**(c) Describe your strategy for weaning.**

The principles of weaning are no different in this patient. He now has a reasonable PaO<sub>2</sub> on 60%, and has an appropriate PaCO<sub>2</sub> for his degree of metabolic alkalosis. General factors preventing weaning need to be excluded (cardiovascular function, metabolic state, nutrition, endocrine function [eg. thyroid], adequate sleep). Carbon dioxide production could be minimised by the use of lower CHO feeds if this is a clinical problem. Metabolic alkalosis could be reduced by the use of acetazolamide (with the risk of increasing ventilatory work required to maintain a given pH).

Ventilatory strategies should be similar to (b) above, with more detailed plan including: attention to posture (sitting, lying on side), day/night cycling, minimising work of breathing during rest (triggering work, work to overcome resistance and compliance of lung and that imposed by chest wall and abdomen), and some periods of increased work (to assess ability to breath with no or minimal ventilatory support).

Rest periods may require high levels of PEEP and pressure support (or equivalent) to counter imposed work. Pressures delivered may overestimate actual trans-pulmonary pressures.

Depending on the difficulty of intubation, the patient will need to be kept for a period of time in the Intensive Care Unit after demonstrating ability to breath overnight for himself (eg. 48 hours) or may stay until the team are happy for him to have the tracheostomy tube removed.

Ongoing supportive care is required throughout to prevent Intensive Care Unit related problems (eg. pressure care, DVT prophylaxis, supportive psychological care, prevention of device related infections etc.).

## QUESTION 2:

*List the determinants of cardiac output in the ventilated Intensive Care patient.*

The determinants of cardiac output are many and varied. A good understanding is required. The standard four factors usually considered to control cardiac output are: heart rate (and rhythm), myocardial contractility, preload and afterload. Many interactions between these factors may be present at one time. (Also could think of in terms of Cardiac Output = Heart Rate \* Stroke Volume). Heart rate : loss of atrial kick (AF, nodal rhythms etc.), tachycardias, bradycardias, ectopic beats all may significantly decrease stroke volume and cardiac output.

Myocardial contractility : generally consider:

neurally mediated (sympathetic activity [increases], parasympathetic activity [decreases]),  
hormonally mediated (adrenal medulla [adrenaline, noradrenaline], adrenal cortex [corticosteroids increase], thyroid hormone (increase), insulin increase], other [growth hormone, glucagon, endothelins increase; circulation myocardial depressants including some cytokines, nitric oxide etc.])

oxygen (hypoxia: moderate stimulates, severe depresses), carbon dioxide (direct: lower increases, higher decreases)

drugs (beta-agonists/blockers, calcium agonists/blockers, inodilators, etc.)

electrolytes (especially calcium, magnesium, phosphate)

Preload:

decreased preload with normal left ventricular compliance: absolute hypovolaemia, relative hypovolaemia (venodilatation; increased resistance to venous return: including increased intrathoracic pressure with IPPV/PEEP; right heart dysfunction including AMI, pulmonary hypertension eg. pulmonary emboli)

decreased preload with decreased LV compliance: LV hypertrophy, ischaemia, beta-agonists

**Afterload:**

changes in intrathoracic pressure, sympathetic tone (inhibition/paralysis etc), vasodilatation (eg. direct effects of drugs, anaphylaxis etc).

***Describe the role of cardiac output measurement in Intensive Care, including indications, and how it may change therapy.***

The role of cardiac output measurement depends largely on local practice. Units will vary in both aggressiveness of determination of cardiac output (PA catheter, echocardiography etc.), and in the way that the information is used (targeting particular goals, having monitoring protocols). Much of our haemodynamic and respiratory management can be done without regular assessment of cardiac output. The **levels of evidence** to support roles, indications and changes in therapy should be provided.

Traditional use of cardiac output is to facilitate differentiation of types of shock (high, normal or low output states) and to titrate specific therapies either targeted to alter cardiac output (inotropes, vasodilators) or to minimise deteriorations (eg. with the use of vasoconstrictors, or high airway pressures). The use of supranormal goals in general ICU patients may actually worsen outcome (though some subsets may benefit: eg. ? perioperative high risk, ??trauma). The harm of interventions should be mentioned.

If cardiac output is deemed to be inadequate (absolute or with confirmatory variables), then therapies to increase cardiac output include: fluids, inotropes, vasodilators and pacing.

***Critically analyse two commonly used techniques for the measurement of cardiac output.***

**Commonly used\*** techniques to measure cardiac output are few. Many techniques can be used, these include: intermittent thermodilution (inject cold fluid\*), "continuous" thermodilution (heated\*), use of doppler (echocardiographic probes\*: transthoracic, trans-oesophageal, suprasternal, oesophageal), transthoracic bioimpedance\*, and calculated using the Fick equation\* (intermittent mixed venous oxygen, continuous SvO<sub>2</sub> catheter), and variations on arterial waveform analysis.

Appropriate critical analysis will include balances between advantages and disadvantages (including invasiveness, insertion, limitations, misinterpretation, other data obtained etc.), and detail of accuracy (bias and precision) as well as indicator of reproducibility (eg. coefficient of variation).

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Chairman  
Court of Examiners  
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