REPORT OF GENERAL FELLOWSHIP EXAMINATION

MARCH/MAY 2002

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 future examinations.

Twelve candidates presented for this examination. Nine were successful.

ORAL SECTIONS

Objectives Structured Clinical Examination (OSCE) Section

There were twelve stations with two rest stations (both after the interactive stations). All 12 candidates passed this section. A systematic approach to the types of investigations examined was more likely to maximise the candidate's score.

Station

1. *Chest X-rays* including pneumothorax after chest trauma, Intra-Aortic Balloon Pump, and upper lobe consolidation. A list of abnormal findings was often requested.

2. *Other X-rays* including CTs and X-rays demonstrating pneumocephalus after head trauma, aortic dissection, lung cavitation and consolidation, and ruptured oesophagus. A list of abnormal findings was often requested.

3. *ECGs* demonstrating a malfunctioning pacemaker, hyperkalaemia, acute myocardial infarction, P pulmonale and bi-fascicular block.

4. *Biochemistry.* Examples included cholestasis, raised anion gap metabolic acidosis, hypochloraemic metabolic alkalosis and adrenal insufficiency.

5. *Procedure station.* Involving chest drainage for a tension pneumothorax.

6. *Rest*

7. *Paediatrics.* ECGs demonstrating atrial flutter/fibrillation, heart block, bundle branch block, prolonged QT syndrome and WPW syndrome.

8. *Arterial Blood Gases* including respiratory acidosis, mixed respiratory and metabolic acidoses, mixed respiratory and metabolic alkaloses, mixed metabolic acidoses.

9. *Chest X-rays* including lobar collapse, chest drains, cardiac failure, interstitial lung disease, and malpositioned devices and tubes.

10. *Miscellaneous Equipment* including examples of types of double lumen tubes, laryngoscopes, pulmonary artery catheters, cricothyrotomy kits and airway exchange catheters.

11. *Communication with an actor* involved discussion of ongoing management of a patient recovering from critical illness, and the potential role of alternative approaches.

12. *Rest*.

Cross Table Viva Section

There were six structured Vivas of ten minutes each. There were two minutes provided to read a scenario outside each viva room. 11 out of 12 candidates passed this section. Candidates should be prepared to provide a reasonable strategy for management of conditions that they may not be familiar with.

The topics included:

- Management of withdrawal of therapy.
- Management of 3 month old child with bronchiolitis.
- Management of post-operative renal failure.
- Management of severe respiratory failure due to Varicella pneumonitis.
- Diagnosis and management of acute pancreatitis.
- Initial and ongoing management of injuries due to major trauma.

The Clinical Section

The Clinical Section was conducted at the Royal Brisbane Hospital.

Only seven out of twelve candidates passed this section. Candidates should listen carefully to the introduction given by the examiners and direct their examination accordingly. Patients were presented as problem solving exercises (including an assessment of patients with difficulty in weaning, worsening neurological state, and worsening renal function). Exposing the patients should be limited to those areas that are necessary for that component of the examination, and in keeping with the modesty requirements of the patients.

Cases encountered included patients with:

COLD CASES
- Splenomegaly
- Pulmonary fibrosis
- Chronic lymphocytic leukaemia
- Pleural effusion
- Scleroderma

HOT CASES
- Worsening neurological state
- Failure to wean
- Potential airway difficulties
- Fever and renal impairment
- Cardiac failure

WRITTEN SECTIONS

Short Answer Questions

It is imperative that candidates 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.

1. *Please list the possible aetiology, features of the presentation, and outline your principles of management of rhabdomyolysis.*

Aetiology: consider trauma and muscle compression (including immobility), exertional rhabdomyolysis (eg. heat stroke, grand mal seizures), drugs and toxins (via coma/immobility, agitation/hyperthermia, myotoxins eg. HMG-CoA reductase inhibitors, myonecrosis secondary to non-depolarising neuromuscular blockers), infections, inflammatory myopathies (eg. polymyositis), electrolyte abnormalities (esp. hypokalaemia and hypophosphataemia), hyperthermia (eg. malignant hyperpyrexia and neuroleptic malignant syndrome), metabolic myopathies.

Presentation: Consider history of exertion, fitting, drug exposure (including illicit), immobility, family history, and previous episodes. Patient may complain of painful or weak muscles, and pigmented urine. Investigations reveal markedly elevated muscle enzymes (especially CK), acute oliguric renal failure, and electrolyte abnormalities (hyperkalaemia, hyperphosphataemia, hypocalcaemia, hyperuricaemia and metabolic acidosis).

Principles of management: consider general supportive care, adequate fluid resuscitation, forced alkaline diuresis (including mannitol), specific treatment of underlying cause (eg. dantrolene, phosphate replacement, cooling, removal of precipitants, treatment of infection, fasciotomies etc), and correction of electrolyte abnormalities.

2. *Critically evaluate the role of induced hypothermia in the management of critically ill patients in Intensive Care.*

Rapidly expanding area, answer needs covering of various areas.

Evidence to support use: comatose survivors after cardiac arrest had improved neurological survival (recent PRCT NEJM X 2); controversial/equivocal for severe head injuries (GCS 3-8), certainly demonstrated to decrease ICP; early evidence to support use in stroke and perhaps myocardial infarction; anecdotal evidence to support cooling to at least normothermia (eg. management of malignant hyperpyrexia); experimental for ARDS; use as adjuvant to minimise cerebral insult (prophylaxis) in the operating theatre during cardiac surgery (deep hypothermic circulatory arrest) and some neurosurgical procedures.

Technique: need to define temperature (eg. 32-33 degrees C), method to cool (blankets, surface cooling, intravenous device), and duration of therapy (eg. 12-24 hours or days).

Potential problems: immune suppression (increased infections), risks bleeding, vasoconstriction, shivering (necessitating neuromuscular paralysis and adverse effects of immobility).

3. *Outline your plan of management for a rapidly deteriorating patient with severe airflow obstruction who is a known difficult intubation.*

Initial management should ensure assessment and management of airway, breathing and circulation, as well as level of consciousness. Must be prepared for difficult intubation (essential equipment should be listed, checked and ready; adequate skilled assistance should be present; backup plans are essential). Specific plan should be elucidated with relation to reason for difficult intubation (eg. limited mouth opening, versus high anterior larynx etc.). Main difficulty is that bag-valve-mask ventilation or laryngeal mask ventilation may be impossible. The use of facemask CPAP may provide some time if not contraindicated by deteriorating neurologic state. Bronchoscopic or blind nasal intubation may be reasonable if operator adequately skilled in techniques. Paralysis may otherwise be essential. Early resort to surgical airway may be appropriate if problems develop.

4. *Outline your peri-operative management of a patient with ischaemic heart disease having an elective right hemicolectomy.*

Recent evidence based guidelines published by ACC/AHA. Management in concert with cardiologists and surgeons. Ideal management often limited by resources.

Pre-operative assessment to determine patient risk. Based predominantly on exercise tolerance (eg. MET [metabolic] equivalents), history (or not) of recent myocardial infarction, and stability of symptoms. Standard preoperative investigations would include creatinine, urea and electrolytes, full blood examination, ECG and CXR. More detailed cardiovascular investigations may be required (eg. exercise test, echocardiography and angiography). Coronary arterial revascularisation (angiography/stenting/surgery) may be indicated before elective surgery (balance with risk of delaying surgery if for malignancy). Left ventricular function should be optimised. Cardiopulmonary exercise testing may enhance risk stratification. Peri-operative beta-blockade would probably have already been commenced but is reasonable unless contraindicated (started days before surgery and targeting resting heart rate 50-60).

Intra-operative management (this operation = intermediate cardiac risk) may be aided by invasive monitoring (eg. intra-arterial, pulmonary arterial lines and/or trans-oesophageal echocardiography). ST monitoring is reasonable, as are intravenous nitrates. Prevention of hypertension/tachycardia and hypotension are expected. Epidural analgesia dependent on anaesthetic preference.

Post-operative phase continues intra-operative stability, and optimises pain relief. Monitoring of ECG and CKMB/troponin to determine extent of ischaemic risk (determined by 24 to 48 hours). Early reinstitution of beta-blockade (intravenous if necessary) and heparin/LMW heparin. Severe cardiac failure may require inotropic support and/or longer period of invasive monitoring and observation (eg. 48 hours).

5. *Outline the role of decontamination of the digestive tract in the management of patients who present with a drug overdose.*

Balance between potential severity of poisoning, time from ingestion and risk to the patient of interventions considered. Most overdoses do not develop significant toxicity but reasonable to use technique with low morbidity and reasonable efficacy in all except clearly non-toxic ingestions (eg. single dose activated charcoal [1g/kg]). Induced emesis with ipecac induces risks without evidence of decreased absorption. Gastric lavage is associated with reasonable decrease in absorption if performed early (e.g. < 1 hour), though it is associated with increased risks (including visceral

injury and aspiration); it may have additional benefit if combined with activated charcoal. Repeat doses of charcoal are usually not of additional benefit except perhaps where a large amount of toxic substance adsorbed by charcoal was ingested (especially slow release preparations). Whole bowel irrigation (using polyethylene glycol e.g. golytely) may have specific benefit with slow release preparations or agents that are poorly absorbed by activated charcoal. Rarely endoscopy or surgical removal is indicated.

6. *Compare and contrast percutaneous and surgical tracheostomy.*

Surgical tracheostomy is the time-honoured approach. Best operating conditions (coping with complexities of anatomy), best control of bleeding and airway. Requires operating time and staff, and transport to operating theatre. Lower incidence of peri-operative complications. Higher incidence of tracheal stenosis, postoperative bleeding and stomal infection.

Percutaneous tracheostomy refers to a number of different techniques. In particular the gradual dilatation [Ciaglia], forceps dilation [Griggs], Rhino and translaryngeal techniques. Most comparative data is for the Ciaglia technique. Blind external technique (which can be bronchoscopy assisted to improve visualization/placement) which seems to be significantly operator dependent. Some neck anatomy problems provide relative contraindications. Permits smaller incision, but lesser exposure and not usually performed with diathermy available. Only require intensive care staff, though airway maintenance is probably more critical, with respiratory acidosis and loss of airway more likely. No delays due to theatre requirements, no transport required, and takes less time to perform. Higher incidence of anterior tracheal wall injury and posterior wall perforation. Lower incidence of postoperative haemorrhage, infection and tracheal stenosis.

7. *List the causes of hyperglycaemia in the intensive care patient population, and outline your management of hyperglycaemia.*

Causes: consider diabetes mellitus (previously known or not known, type I or II, on diet, oral agents, insulin or combination), secondary causes of diabetes (e.g. pancreatitis, haemochromatosis, Cushing's syndrome, acromegaly), insulin resistance (e.g. sepsis, systemic inflammatory response/stress response [including multiple trauma], beta-agonists [endogenous or exogenous], exogenous corticosteroids), carbohydrate load (e.g. feeding enteral/parenteral, peritoneal dialysis).

Management: consider control of factors worsening response to insulin (sepsis, drugs, stress response), control glucose within acceptable range (minimise metabolic and immune effects), recommence oral agents or use insulin (dependent on severity). Principle of glucose control in diabetics include always some insulin, administer some glucose, measure glucose frequently, expect sudden changes, and avoid hypoglycaemia. Recent studies suggest tight glucose control using insulin infusions if necessary may dramatically reduce mortality after myocardial infarction (in diabetic patients: DIGAMI), and in the surgical intensive care (Van den Berghe et al).

8. *Outline the aetiology, clinical manifestations and treatment of phrenic nerve palsy after cardiac surgery.*

Aetiology: potential contributing factors include difficult dissection, internal mammary artery dissection, excessive retraction (sternum/pericardium), use of topical cooling (eg. slush) and haematoma from internal jugular venipuncture.

Clinical manifestations: Can be unilateral (esp. left) or less common bilateral. Usually manifest by respiratory difficulties. Patient may not be able to be weaned from mechanical ventilation, or may have significant post-operative requirements for respiratory support. Persistent collapse and/or pneumonia may develop. Clinical examination may reveal decreased movement on affected side, decreased breath sounds (± signs of collapse/consolidation), significant dullness to percussion, with absence of normal tidal percussion. Radiological investigations confirm elevated hemidiaphragm (&/or collapse/consolidation), which moves paradoxically on sniff test (fluoroscopy).

Treatment: usually expectant for underlying lesion. Supportive care plus specific treatment of complications (eg. aggressive physiotherapy, non-invasive ventilatory support).

9. *Critically evaluate the use of cisapride, metoclopramide and erythromycin for gastric emptying in Intensive Care patients.*

Cisapride: selectively enhances physiologic release of acetylcholine at level of myenteric plexus. Part of effect via activation of serotonin (5-HT4) receptors. Enhances oesophageal peristaltic activity, gastric emptying, intestinal propulsive activity and colonic transit. Extensively metabolised via cytochrome P450 3A4 enzymes. Highly protein bound. Only administered orally. Significant adverse effects and interactions, especially prolonged QT interval (and arrhythmias) in particular when administered in patients at risk of arrhythmias or when administered concurrently with drugs that prolong QT or drugs that inhibit P450 3A4 enzymes (e.g. azole antifungals, macrolide antibiotics, and protease inhibitors). Problems with limited availability, restrictions on prescribing, large number of documented interactions.

Metoclopramide: mode of action unclear (? via selective dopamine-2 receptor antagonist effects); sensitises tissues to the action of acetylcholine (motility effects abolished by anticholinergic drugs and narcotic analgesics). Increases tone and amplitude of gastric contractions, relaxes pyloric sphincter and increases peristalsis of duodenum and jejunum. Administered orally, IV or IM. Conjugated by liver and renally excreted (reduced clearance with renal failure). Minimal protein binding. Dopamine agonist activity responsible for adverse effects (e.g. sedation, dystonic/extrapyramidal reactions).

Erythromycin: macrolide antibiotic that seems to stimulate motilin receptors, and enhances motilin release from enterochromaffin cells of duodenum. Enhanced contractile effects on gastric antrum and duodenum. Administered orally or intravenously (probably IV more effective). Highly protein bound. Substantial hepatic metabolism. Prolonged QT and arrhythmias reported, as have hepatic dysfunction, overgrowth of non-susceptible organisms and colitis (Cl. difficile). Elevated levels of many other drugs (as a result of inhibition of metabolism) can lead to toxicity (e.g. theophylline, HMG-CoA reductase inhibitors, anti-epileptics, digoxin, warfarin etc).

10. *Outline the aetiology, clinical manifestations and possible preventative measures for nosocomial infections in Intensive Care.*

Aetiology: dependent on cause. Combination of overgrowth of endogenous flora, immune suppression, impairment of natural defences (eg. endotracheol tube, invasive catheters), and cross contamination with pathogens. Commonest are pneumonia (thought related to aspiration of organisms colonising oro-pharyngeal and gastric contents, decreased gastric pH, exposure to water borne organisms), surgical wound infections (contamination at time of surgery), sinusitis (tubes, immobility, nasal congestion), line related sepsis (entry site contamination, blood borne contamination, contamination of intravenous lines).

Clinical manifestations: apart from systemic manifestations of sepsis (leukocytosis/leukopaenia, fever, etc) are dependent on cause. Pneumonia (purulent secretions, impaired oxygenation, radiological infiltrates etc.), sinusitis (purulent discharge), line related (local inflammation). Diagnostic techniques are specific to cause.

Possible prevention: consider general infection control measures (including surveillance, continuous quality improvement, avoid un-necessary immunosuppression [steroids, glucose control], avoid un-necessary antibiotics). Decrease cross infection (standard precautions [esp. hand washing] and transmission based precautions; cleaning/disinfection and sterilization of equipment; avoiding reuse of single use items). Pneumonia (eg. semi-recumbent position, aspiration above cuff, possibly selective decontamination, infrequent changing of circuit). Line related (eg. sterile insertion, antibiotic/antiseptic impregnated lines, perhaps tunneling, surveillance, change according to protocol).

11. *Outline the causes, consequences and management of intrinsic PEEP.*

Causes: consider increased expiratory resistance (prolonged expiratory time constant: eg. bronchospasm, narrow/kinked ETT, inspissated secretions, exhalation valves/HME/filters), increased minute ventilation (inadequate expiratory time).

Consequences: consider increased intra-thoracic volume (with increased pressures for a given Vt and risks of barotrauma), increased intra-thoracic pressure (decreasing venous return, and increasing inspiratory work to trigger the ventilator).

Management: consider treatment of reversible factors (bronchospasm, secretions, expiratory devices), prolongation of expiratory time (decrease respiratory rate, increase inspiratory flow) or decrease tidal volumes, application of exogenous PEEP (to 50 – 85% of accurately measured intrinsic PEEP) to decrease inspiratory triggering work and improve distribution of inspired gas.

12. Compare and contrast the methods of delivery of beta-2 agonists in intubated patients.

Consideration should be given to pharmacodynamics (dose requirements, side effect profile, effectiveness), as well as cost and other interrelated effects. Methods of delivery include intravenous, sub-cutaneous, via metered dose inhaler and via nebuliser.

Intravenous: excellent systemic delivery assured, but to areas that are perfused. Systemic effects maximal so side effects are more pronounced.

Sub-cutaneous: easy to administer, but less predictable effects as delayed peak effect and lower bioavailability. Systemic side effects still prominent.

Metered dose inhaler: easy to administer via adapter; many multiples of non-intubated dose are required [eg. 10 puffs per treatment]; does not require breaking of ventilatory circuit; very low bioavailability, optimal via inline spacer (but adds cost, breaks circuit at least once, may become reservoir for infection); minimal systemic side effects.

Nebuliser: can be given continuously; maximises local delivery while minimising systemic absorption; easy to administer but requires specific equipment; requires break in circuit for each treatment; variable interaction with ventilator [some cannot compensate for flow].

13. *Outline the pathophysiology, complications and treatment of hyper-osmolar non-ketotic coma.*

Pathophysiology: insulin deficiency (and/or resistance) impairs peripheral glucose utilisation in skeletal muscle, increases fat and muscle breakdown and promotes hepatic gluconeogenesis; glucagon excess also promotes hepatic gluconeogenesis. Other stressors may precipitate (e.g. infection, myocardial infarction, and surgery), partially by increasing cortisol and catecholamine release; omission of normal treatment may also be responsible. Osmotic diuresis results in significant fluid depletion (e.g. 8 to 10 litres), with associated deficits of potassium and phosphate (despite variable plasma levels).

Complications: CNS depression/coma, hypovolaemia, hyperosmolality, metabolic acidosis, potassium and phosphate depletion, and thromboembolism. Cerebral oedema if glucose lowering or fluid shifts too rapid.

Treatment: of underlying precipitants (sepsis, myocardial infarction), replace fluid deficit (± invasive monitoring) without rapidly dropping osmolality, insulin therapy (eg. infusion), careful monitoring and replacement of electrolytes (esp. potassium, phosphate), prevention of pulmonary thromboembolism.

14. *Outline the diagnostic features, complications and treatment of patients with Wolf-Parkinson-White syndrome.*

Diagnosis: history of arrhythmias (palpitations, dizzy, lightheaded), sudden death. ECG in sinus rhythm demonstrates short PR interval and delta waves. Electrophysiological evidence of AV conduction through AV bypass tract (bundle of Kent).

Complications: recurrent arrhythmias (narrow QRS complex orthodromic AV re-entrant tachycardia, wide QRS antidromic AV re-entrant tachycardia, atrial fibrillation (broad complex and may be very fast [> 200/min]), ventricular fibrillation), sudden death.

Treatment: (1) acute treatment of arrhythmias: a) Narrow SVT (as per SVT). b) Wide QRS SVT (procainamide; avoid adenosine, verapamil, digoxin and beta-blockers; treat as if VT). c) Atrial fibrilation: (Appropriate agents include procainamide, amiodarone and flecainide. Avoid agents which might slow AV conduction but not decrease conduction through bypass tract: i.e. adenosine, verapamil, digoxin and beta-blockers). (2) investigation via electrophysiologic evaluation: usually curative ablation of accessory pathway, or no treatment if asymptomatic, or occasionally prophylactic medications.

15. *Outline the indications for high frequency oscillation in Intensive Care, and the mechanism of gas exchange when using high frequency oscillation.*

Indications: usually as part of experimental therapy or as part of a controlled clinical trial. Potential rescue therapy for ARDS in Intensive Care Units who have suitable equipment available and are experienced in its use, where “open lung” ventilation strategy (adequate recruitment and avoidance of overdistension) is desirable. In Paediatric Intensive Care as rescue therapy for severe respiratory failure.

Mechanism of gas exchange: normal bulk flow is much less important, as the tidal volumes used are much smaller than anatomical deadspace; gas delivery into the system (as bias flow) will still

provide some gas exchange. Other potential mechanisms described (many of which may work simultaneously) include Taylor dispersion (dispersion of molecules beyond the bulk flow front), augmented diffusion (gas mixing within alveolar units), coaxial flow patterns (net flow one way through centre of airway, other direction via periphery) and Pendelluft mixing (between lung units, mixing of gas due to impedance differences).

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. The examiners apportioned marks according to difficulty and required time within each question.

An organised/systematic approach is expected.

Question 1

You are called to see a 65 year old male tourist who has been admitted to your emergency department after being hit by a car while attempting to cross a busy street. He is unconscious and has obvious chest and limb injuries.

(a) Please outline your initial management of this patient.

Organized approach is essential. ATLS/EMST approach should be used. Most emergency departments that receive trauma do so with facilities that support a trauma team concept. Initial management should be undertaken as part of the trauma team, with roles usually well delineated.

Initial management requires simultaneous primary survey, resuscitation and assessment of history, followed by a secondary survey then definitive care.

Primary survey involves assessment of adequacy of airway, breathing and circulation (with interventions at each point whenever identified), followed by assessment of neurological state (pupils, level of consciousness, localising signs) and adequate exposure to assess major injuries. Indications for endotracheal intubation should be clearly described (GCS < 9, hypoxia/respiratory distress etc.). Initial ventilatory management should be detailed (respiratory rate, tidal volume, blood gas goals etc). Fluid administration and goals of resuscitation should be discussed. Relevant history should be obtained from ambulance officers, family, witnesses etc. In particular details about the mechanism of injury and patient's previous medical condition, medications and allergies etc.

Secondary survey involves a detailed head to toe examination to assess extent of injuries (including flanks, back and rectal examination), as well as a detailed neurological assessment.

Definitive care involves planning for surgery, other specialist involvement and transfer as appropriate.

(b) Please discuss the timing and nature of any investigations which you would perform.

Urgent early investigations include urea and electrolytes, full blood examination and blood group and cross match (done when initial venous access is obtained). It is reasonable to also perform arterial blood gas analysis and a coagulation profile at this time.

During the resuscitation phase before the secondary survey, it is reasonable to get a lateral cervical spine, supine chest X-ray, and pelvis X-ray, as long as this can be done without moving the patient to a separate area. Some specific abdominal assessment should be made as the patient is unconscious (DPL, FAST or CT scan), earlier if haemodynamically unstable. A urinary catheter (unless contraindicated) should be inserted at this time to monitor urine output, and an ECG should be obtained (± echocardiography or CVP monitoring if unsure of cardiovascular status).

More specific X-rays of suspected or high risk areas (eg. full cervical spine series, chest CT and head CT, limb and thoracic and lumbar spine X-rays) should be done when patient is haemodynamically stable and ideally before transfer to ICU or theatre (unless required urgently). Definitive exclusion of thoracic aortic injury (trans-oesophageal echocardiography or CT angiography) should be performed if clinically indicated when haemodynamically stable.

Repeat assessment of blood gases, Hb and coagulation may be needed early.

Intra-cranial pressure monitoring may be required depending on clinical status or CT appearance (in this 65 year old man). This is not usually urgent, but may facilitate titration of modalities to control ICP and CPP. It may be inserted in ICU or pre-operatively if prolonged time in the operating theatre is anticipated.

(c) Please discuss your plan for his definitive care (including fixation of long bone fractures etc.).

This patient has major trauma with head, limb and chest injuries, and should be managed in a centre that is experienced in trauma care. If this hospital is not able to provide sufficient services then early communication with a receiving hospital is essential, and plans made for expedient transfer.

Specific neurosurgery may be necessary if intracranial haemorrhage is detected and should be performed within the first few hours. Thoracic surgery is rarely required (eg. dependent on amount of bleeding from intercostal tubes), but surgery will be required for long bone fractures. Compound fractures should be dealt with early (hours), as should injuries with vascular compromise. Other operations are less urgent and the role of early fixation of fractures is controversial. In the absence of significant respiratory compromise it is probably reasonable to progress to early fixation. If instead there is concern about respiratory status then external fixation rather than internal fixation may be preferable on the first day, followed by more specific management a few days later.

Question 2

The mortality in patients with ARDS has only shown a gradual decline over the last two decades.

(a) What specific modalities of treatment have contributed to the improvement in mortality?

Many promising specific therapies have failed to demonstrate improved mortality when studied in more detail (eg. prone positioning, nitric oxide, exogenous surfactant, liquid ventilation, ECMO, infusion of PGE1, ketoconazole and N-acetylcysteine). Most of these therapies are not routinely used, and are therefore unlikely to contribute to improved outcomes.

Specific therapies that have recently been demonstrated to potentially improve survival include the use of low tidal volume ventilation (ARDSnet, NEJM), in addition to recruitment and higher levels of PEEP (Amato, NEJM) and the use of steroids in late ARDS (Meduri JAMA). These techniques are also relatively recent in acceptance/still being evaluated, and are not yet in widespread use.

A combination of factors not individually shown to be beneficial regarding mortality is more likely to have resulted in benefit. Most patients die a non-respiratory death, so general "supportive care" is more likely to have made an impact on survival. Specific approaches to be considered include:

- Prevention: of pulmonary thromboembolism, gastrointestinal bleeding, nosocomial infections (pneumonia, line-related sepsis), complications of sedation and paralysis (better understanding of pharmacology).
- Early diagnosis and treatment: of nosocomial infections, deficiencies in pituitary-adrenal access.
- Early nutritional support: especially with regard to early enteral feeding.
- Additional ventilatory strategies: including better synchrony of patient with ventilator, allowing lower levels of sedation/paralysis; semi-recumbent positioning etc.

(b) Discuss why the observed decline in mortality has not been greater in magnitude?

A number of factors need to be considered, in particular the large amount of background noise making accurate assessment of improvements near impossible. Indeed, the studies that have actually shown benefit may not be extrapolatable to the majority of the ARDS population seen in Intensive Care.

The mortality of ARDS is not usually due to respiratory disease per se, but instead to multiple organ dysfunction. This in turn is due to a multiplicity of factors (including the underlying disease process that resulted in ARDS [eg. pancreatitis, sepsis, burns], inflammatory response due to ARDS, nosocomial infections. No single specific therapy is likely to prevent the cascade of events that result in inflammation. Insufficient studies have been performed to consistently demonstrate one technique has benefits, let alone which combinations of therapies may be useful.

ARDS is also the end result of a large number of predisposing insults. The outcomes vary dramatically between subgroups (eg. trauma versus pneumonia). More specific classification or stratification may allow more accurate comparisons.

As a result of better general supportive care, patients that would not previously been considered salvageable could now be going on to develop ARDS, and are more likely to have an adverse outcome. It is probably impossible to accurately compare outcomes now with decades ago, given the inability to control for the many factors that influence outcome.

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