EXAM REPORT MAY 2011

This report is prepared to provide candidates, tutors and Supervisors of Training with information regarding the assessment of candidates’ performance in the General Fellowship Examination. Answers provided are not necessarily model answers but guides as to what was expected. Candidates should discuss the report with their tutors so that they may prepare appropriately for future examinations.

The exam comprises a written section and an oral section. The written exam consists of two 2.5 hour papers of 15 ten-minute short answer questions each. Candidates are required to score at least 50% in the written section to be eligible to sit the oral section. The oral exam consists of eight interactive vivas and two separate clinical “hot cases”.

The tables below provide an overall statistical analysis as well as information regarding performance in the individual sections. A comparison with data from the three previous exams is provided.

Please note all images from the SAQs have been removed.

General Fellowship Examination Report: March / May 2011 – Compiled by Prof. Bala Venkatesh and Dr Mary Pinder.
**Number of candidates presenting**

<table>
<thead>
<tr>
<th></th>
<th>May 2011</th>
<th>October 2010</th>
<th>May 2010</th>
<th>October 2009</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presenting for written (Including OTS)</td>
<td>36</td>
<td>48</td>
<td>43</td>
<td>50</td>
</tr>
<tr>
<td>Carrying a pass from a previous attempt</td>
<td>7</td>
<td>8</td>
<td>7</td>
<td>14</td>
</tr>
<tr>
<td>OTS Exempt</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Total number presenting (written + carry + OTS)</td>
<td>43</td>
<td>58</td>
<td>52</td>
<td>68</td>
</tr>
</tbody>
</table>

**Number of candidates presenting to the orals**

<table>
<thead>
<tr>
<th></th>
<th>May 2011</th>
<th>October 2010</th>
<th>May 2010</th>
<th>October 2009</th>
</tr>
</thead>
<tbody>
<tr>
<td>Invited to orals (&gt;50% in written section)**</td>
<td>22</td>
<td>33</td>
<td>30</td>
<td>36</td>
</tr>
<tr>
<td>Carrying a pass from a previous attempt</td>
<td>7</td>
<td>8</td>
<td>7</td>
<td>14</td>
</tr>
<tr>
<td>OTS Exempt</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Total number invited to oral section</td>
<td>29</td>
<td>43</td>
<td>39</td>
<td>54</td>
</tr>
</tbody>
</table>

**Please note - One candidate who passed the written section in May 2011 elected to postpone their attempt at the oral section until October 2011.**

**Overall pass rates**

<table>
<thead>
<tr>
<th></th>
<th>May 2011</th>
<th>October 2010</th>
<th>May 2010</th>
<th>October 2009</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall Pass Rate</td>
<td>18/43</td>
<td>35/58</td>
<td>27/52</td>
<td>46/68</td>
</tr>
<tr>
<td></td>
<td>42%</td>
<td>60%</td>
<td>52%</td>
<td>68%</td>
</tr>
<tr>
<td>Pass Rates amongst those who scored &gt;50% in written section</td>
<td>17/22</td>
<td>27/33</td>
<td>24/30</td>
<td>31/36</td>
</tr>
<tr>
<td></td>
<td>77%</td>
<td>82%</td>
<td>80%</td>
<td>86%</td>
</tr>
<tr>
<td>Pass Rates amongst those invited to oral section (written + carry + OTS)</td>
<td>18/29</td>
<td>35/43</td>
<td>27/39</td>
<td>46/54</td>
</tr>
<tr>
<td></td>
<td>62%</td>
<td>81%</td>
<td>69%</td>
<td>85%</td>
</tr>
</tbody>
</table>
## Analysis of performance in individual sections

<table>
<thead>
<tr>
<th></th>
<th>May 2011</th>
<th>October 2010</th>
<th>May 2010</th>
<th>October 2009</th>
</tr>
</thead>
<tbody>
<tr>
<td>Successful in the written section</td>
<td>23/36</td>
<td>33/48</td>
<td>30/43</td>
<td>36/50</td>
</tr>
<tr>
<td></td>
<td>64%</td>
<td>69%</td>
<td>70%</td>
<td>72%</td>
</tr>
<tr>
<td>Successful in the Clinical section</td>
<td>15/29</td>
<td>32/43</td>
<td>20/39</td>
<td>34/54</td>
</tr>
<tr>
<td></td>
<td>52%</td>
<td>74%</td>
<td>51%</td>
<td>59%</td>
</tr>
<tr>
<td>Successful in the Viva section</td>
<td>20/29</td>
<td>40/43</td>
<td>32/39</td>
<td>47/54</td>
</tr>
<tr>
<td></td>
<td>69%</td>
<td>93%</td>
<td>82%</td>
<td>87%</td>
</tr>
</tbody>
</table>

### Sectional pass rates - Clinicals

<table>
<thead>
<tr>
<th></th>
<th>May 2011</th>
<th>October 2010</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pass rate</td>
<td>Highest individual mark</td>
</tr>
<tr>
<td>Hot Case 1</td>
<td>48%</td>
<td>83%</td>
</tr>
<tr>
<td>Hot Case 2</td>
<td>45%</td>
<td>86%</td>
</tr>
<tr>
<td>Successful in both Hot Cases</td>
<td>6/29 (21%)</td>
<td>22/43 (51%)</td>
</tr>
</tbody>
</table>
EXAMINERS’ COMMENTS

Written Paper

• The pass rate was less than 50% for 12 of the 30 questions. Questions with a particularly low pass rate (≤ 33%) included the SAQs relating to drug absorption and clearance, infection control policy, management of PEA cardiac arrest with tamponade, and statistics.

• OSCE style questions, usually carrying a high pass rate, relating to haematology, lung function tests and ECGs were also answered poorly.

• As in previous exams, there was a lack of specificity and precision. It appears that candidates do not always read the question carefully.

• Candidates scoring >50% and gaining an invitation to the oral section passed an average of 20 questions compared with those failing the written section who passed an average of 10 questions.

Hot Cases

The overall pass rate was similar to previous exams. Concerns expressed by the examiners included:

• Poor clinical examination of patients with missed / misinterpreted clinical signs such as heart murmurs, respiratory signs and neurological signs.

• Lack of an individual approach tailored to the case in question.

• Poor use of the time available for clinical examination, with candidates spending either too much time on one system to the exclusion of others, or not spending sufficient time examining a key system.

• Demonstration of theoretical knowledge but inability to apply this knowledge or use diagnostic reasoning.

• Failure to “join the dots” eg missing the diagnosis of infective endocarditis in a patient with staphylococcal bacteraemia and subsequent AVR.

• Poor interpretation of investigations, in particular ECGs, and imaging.

• Poor discussion of management issues, sometimes overly hesitant and sometimes making decisions that would have resulted in a critical adverse event for the patient.

Vivas

The pass rate in the vivas for this exam was considerably lower than that of previous years. Of particular concern was poor performance in the procedure station requiring a demonstration of basic life support skills.
GENERAL FELLOWSHIP WRITTEN EXAMINATION

(A) Write your answers in the blue books provided.

(B) Start each answer on a **new page** and indicate the **question number**. It is not necessary to rewrite the question in your answer book.

(C) You should aim to answer each question in **ten** minutes.

(D) The questions are worth **equal** marks.

(E) Record your **candidate number** and each **question number** on the cover of each book and hand in all books.

GLOSSARY OF TERMS

**Critically evaluate:** Evaluate the evidence available to support the hypothesis.

**Outline:** Provide a summary of the important points.

**List:** Provide a list.

**Compare and contrast:** Provide a description of similarities and differences (Eg. Table form).

**Management:** Generic term that implies overall plan. Where appropriate, may include diagnosis as well as treatment.

**NOTE**

Where laboratory values are provided, abnormal values are marked with an asterisk (*).

Please note all images from the SAQs have been removed.
1. Outline the pathophysiological changes associated with end-stage kidney disease (dialysis dependent) that may impact on the management of critically ill patients.

Renal:
- Low/no urine output

Metabolic and Endocrine:
- Associated
  - Hyperkalaemia
  - Abnormal Ca++
  - Hyperphosphataemia

Need for dialysis determines fluid prescribing, feeding and any protein restriction

Cardiovascular:
- Hypertension very common
- Atherosclerosis common
- Pericarditis common

Respiratory:
- Prone to pulmonary oedema

Neurological:
- Dialysis disequilibrium
- Polyneuropathy and myopathy

Skin:
- Fragile skin

Haematological:
- Anaemia
- Platelet dysfunction

Gastrointestinal:
- Impaired gastrointestinal motility
- Increased risk of bleeding related to gastric ulceration

Immunological:
- Increased risk of infection

Pharmacological:
- Altered clearance of medications that have predominant renal excretion

Vascular access:
- Fistulas used for dialysis may complicate CVC and arterial access

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>33%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>7.0</td>
</tr>
</tbody>
</table>

2. Answer the following questions about transjugular intrahepatic portosystemic shunts (TIPS):

a) What is a TIPS procedure and why is it used in patients with portal hypertension?

The hepatic vein is accessed via the internal jugular vein and IVC. A needle is then passed to connect the hepatic vein with the large portal vein near the centre of the liver, the needle tract dilated and a stent inserted to maintain the tract and form the shunt between the higher pressure portal vein and the lower pressure hepatic vein. This reduces portal hypertension.
b) What are 2 recognised indications for this procedure?

   a) Variceal bleeding that has failed endoscopic and pharmacological treatment.
   b) Refractory ascites

c) Excluding mortality list 5 COMMON complications of TIPS procedure

   • thrombosis
   • occlusion of the stent
   • capsular puncture
   • bleeding
   • encephalopathy
   • stent migration

d) Describe one classification system used in assessing severity of chronic liver disease and outline its utility.

Either: Childs-Pugh score
Classified A,B or C by a composite of Total bilirubin, albumin, INR, ascites and hepatic encephalopathy. Originally used for prognostication for surgery – also used for prognostication in chronic liver disease and prediction of likelihood of complications of cirrhosis

Or: MELD score severity scoring system for assessing severity of chronic liver that uses the serum bilirubin, creatinine and INR. Initially developed to predict three month survival in patients post TIPS. Now used for prognosis of liver disease and prioritizing liver transplant recipients

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>86%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>8.0</td>
</tr>
</tbody>
</table>

3. A 60 year old man presents with a history of vomiting followed by the sudden onset of chest pain a few hours ago. On examination he has surgical emphysema over his neck and chest and evidence of a left pleural effusion.

Discuss your management of this patient.

History and examination strongly suggestive of spontaneous oesophageal rupture

Initial management
Check ABCs and resuscitate if necessary
More extensive history and examination to look for other causes of surgical emphysema (eg CVC, barotrauma, pneumothorax), chest pain (eg pneumothorax, pulmonary embolus, musculoskeletal) and pleural effusion.
Look for signs of sepsis, shock and hypoxia.
Admit to highly monitored area in view of high risk of rapid clinical deterioration.
NBM

Investigations
Contrast CT abdo chest and neck / gastrograffin swallow (avoid barium)
CXR: pleural effusion, ± pneumomediastinum, ± pneumothorax
Pleural fluid: presence of food particles, pH<6 and high amylase concentration indicative of oesophageal rupture but amylase may be high in pancreatitis.
Culture of pleural fluid
Blood culture
oesophagoscopy
Definitive treatment
Broad spectrum antibiotics including anaerobic cover plus antifungals
Pleural drainage
Early (within 24 hr) thoracotomy and repair or endoscopic placement of stent depending on whether patient shows signs of sepsis. Surgery preferred for septic patients, conservative if contained perforation.

Pass rate 58%
Highest mark 8.25

4. You are asked to help resuscitate a 75 year old man who has just arrived in the emergency department. He has a blood pressure of 80/45 mmHg, HR 140 /min, and a temperature of 38.5°C after 2 litres of normal saline resuscitation. The only history available is of significant cardiac disease.

Outline your approach to the management of his haemodynamic profile.

Consider mixed aetiology for shock
- Cardiogenic (cardiac history, severe sepsis, rhythm)
- Distributive shock (sepsis)
- Obstructive shock (PE, tamponade) – less likely but will probably get mentioned. Maybe give less marks for this than the other causes

Establishing relative contribution of each to the hypotension
- Clinical Signs
  - Distributive; warm and dilated (if adequate filling), temperature, potential source sepsis
  - Cardiogenic
    - LVF; tachycardia, bibasal crepitations, gallop
    - RVF; JVP, hepatomegaly, oedema
  - Escalating monitoring
    - Minimal: ECG, NIBP, SpO2
    - ABP, CVP progressing to Central Venous O2 Sat / TTE / PICCO / PAC as indicated
  - Laboratory Investigations directed at cause
    - Lactate, Troponin, ECG, CXR, Sepsis screen, UA
  - Collateral history

Interventions
- Optimise preload
  - Cardiogenic
  - Optimize preload (low from redistribution
  - Optimize contractility
  - Rhythm; rate control / normalization (cardioversion?)
  - Inotropic support
    - Dobutamine / Milrone / Levosimenden / Adrenaline / Nor Ad (increases coronary art perfusion pressure) Caution with inodilators while still hypotensive
  - IABP
  - CPAP
  - Reversible / Specific factors
• Exclude / treat ischaemia (heparin / angio, revascularisation etc.)
• Distributive
• Optimize preload
• Vasopressor support
• Noradrenaline
• Adjuncts: Vasopressin / Steroid (infusion or bolus)
• Mixed pathology issues
• Risk of Noradrenaline alone is an increased afterload with worsening cardiogenic shock / peripheral perfusion

• Start with inotrope and then add vasopressor; dobutamine / norad combination
• Adrenaline may a safer choice (inotrope + vasoconstriction)

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>58.3%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>8.0</td>
</tr>
</tbody>
</table>

5.

a) Outline the effect of critical illness on enteral drug absorption

• Multiple factors may alter gastrointestinal mucosal absorption including mucosal oedema, disordered gastrointestinal motility and disordered mucosal blood flow
• Gastric emptying / gut motility affected by drugs (opioids, Anticholinergics, antacids, inotropes), enteral nutrition, brain or spinal injury, diabetes
• Incomplete oral medication disintegration or dissolution
• Changes in pH

b) List the reasons for altered drug clearance in the critically ill.

Liver function
Reduced clearance
With hepatic dysfunction present in more than half the critically ill patients, drug clearance may be reduced because of:
  a. Lower hepatic blood flow
  b. Decreased hepatocellular enzyme activity
  c. Lower bile flow
  d. Administration of other drugs competing for enzymes
Increased clearance
Hepatic enzyme induction by certain drugs may increase clearance of others

Renal function
Reduced clearance
Compromised kidney function may be secondary to reduced perfusion, intrinsic damage secondary to ischaemia or drug toxicity and immunologic injury
A decrease in GFR would increase the half-life of medications that are renally cleared and may result in drug or metabolite accumulation
Increased clearance
Increased cardiac output in early sepsis increases GFR and increased drug clearance
Burns, use of diuretics and hypertonic saline also result in increased GFR and potentially increase clearance
Protein binding changes
Three major proteins affecting drug protein binding – albumin, alpha 1 acid glycoprotein and lipoproteins

Reduced clearance
Some proteins (eg alpha 1-acid glycoprotein binding morphine) are increased in critically ill resulting in reduced clearance

Increased clearance
Albumin is reduced so there will be a higher concentration of free drug for drugs normally bound to albumin resulting in increased clearance

Protein binding affected by other factors including accumulation of endogenous binding inhibitors, qualitative changes on binding sites, competition for binding by other substances, pH changes

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>31%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>6.25</td>
</tr>
</tbody>
</table>

6.1. This blood gas report was taken from a lady hospitalised for recurrent urinary tract infections. She was transferred to the ICU because of nosocomial pneumonia.

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>FiO₂</td>
<td>0.3</td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.53</td>
<td>7.35 – 7.45</td>
</tr>
<tr>
<td>pCO₂*</td>
<td>31 mmHg (4 kPa)</td>
<td>35 – 45 (4.6 – 5.9)</td>
</tr>
<tr>
<td>pO₂</td>
<td>83.7 mmHg (11 kPa)</td>
<td>80 – 110 (10.5 – 14.5)</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>25 mmol/L</td>
<td>24 – 32</td>
</tr>
<tr>
<td>Standard Base Excess*</td>
<td>3.3 mmol/L</td>
<td>-2.0 – +2.0</td>
</tr>
</tbody>
</table>

a) Comment on the acid-base status.

Mixed respiratory and metabolic alkalosis

b) List 2 likely causes of the acid-base derangement in this patient.

Respiratory alkalosis from the hyperventilation due to the pneumonia
Metabolic alkalosis from vomiting or diuretic use.

6.2. A 40 year old 70 kg male has gram negative sepsis and has developed bilateral pulmonary infiltrates. The following are data from blood gas analysis.

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>FiO₂</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>pH*</td>
<td>7.31</td>
<td>7.35 – 7.45</td>
</tr>
<tr>
<td>pCO₂*</td>
<td>31 mmHg (4 kPa)</td>
<td>35 – 45 (4.6 – 5.9)</td>
</tr>
<tr>
<td>pO₂</td>
<td>110 mmHg (14.5 kPa)</td>
<td>80 – 110 (10.5 – 14.5)</td>
</tr>
<tr>
<td>Bicarbonate*</td>
<td>15.1 mmol/L</td>
<td>24 – 32</td>
</tr>
<tr>
<td>Standard Base Excess*</td>
<td>-10.0 mmol/L</td>
<td>-2.0 – +2.0</td>
</tr>
</tbody>
</table>

a) Could this blood gas be consistent with the definition of acute respiratory distress syndrome (ARDS)? Give your reasoning.

No. The P/F ratio is 220. By definition, the problem would be acute lung injury rather that ARDS at this stage.
b) What dose of sodium bicarbonate (in mmol) would be required to reverse
the metabolic acidosis? Show your calculation method.

Dose sodium bicarbonate = Wt (kg) x 0.3 x -SBE = 70 x 0.3 x 10 = 210 mmol

6.3. Following laparotomy for haemoperitoneum, a patient is transferred to ICU.
Blood biochemistry and arterial blood gas analysis on admission to ICU are
as follows:

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium*</td>
<td>147 mmol/L</td>
<td>135 – 145</td>
</tr>
<tr>
<td>Potassium</td>
<td>3.6 mmol/L</td>
<td>3.2 – 4.5</td>
</tr>
<tr>
<td>Chloride*</td>
<td>124 mmol/L</td>
<td>100 – 110</td>
</tr>
<tr>
<td>Haemoglobin*</td>
<td>106 G/L</td>
<td>115 – 155</td>
</tr>
<tr>
<td>pH</td>
<td>7.32</td>
<td>7.35 – 7.45</td>
</tr>
<tr>
<td>pCO₂*</td>
<td>32.4 mmHg (4.3 kPa)</td>
<td>35 – 45 (4.6 – 5.9)</td>
</tr>
<tr>
<td>pO₂*</td>
<td>63 mmHg (8.4 kPa)</td>
<td>80 – 110 (10.5 – 14.5)</td>
</tr>
<tr>
<td>Bicarbonate*</td>
<td>16.0 mmol/L</td>
<td>24 – 32</td>
</tr>
<tr>
<td>Standard Base Excess*</td>
<td>-9.0 mmol/L</td>
<td>-2.0 – +2.0</td>
</tr>
</tbody>
</table>

a) Describe the acid-base status.
Normal anion gap metabolic acidosis with appropriate respiratory compensation

b) What is the likely cause of this disturbance?
Resuscitation with large volume saline infusion.

c) What is the underlying biochemical mechanism?
ECF dilution by fluid with strong ion difference of zero

6.4. A 33 year old female has gram negative bacteraemia and septic shock. The
following are data from blood gas analysis.

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barometric pressure</td>
<td>760 mmHg (100 kPa)</td>
<td></td>
</tr>
<tr>
<td>FiO₂</td>
<td>0.3</td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.43</td>
<td>7.35 – 7.45</td>
</tr>
<tr>
<td>pCO₂*</td>
<td>23 mmHg (3.1 kPa)</td>
<td>35 – 45 (4.6 – 5.9)</td>
</tr>
<tr>
<td>pO₂*</td>
<td>107 mmHg (14.3 kPa)</td>
<td>80 – 110 (10.5 – 14.5)</td>
</tr>
<tr>
<td>Bicarbonate*</td>
<td>15 mmol/L</td>
<td>24 – 32</td>
</tr>
<tr>
<td>Standard Base Excess*</td>
<td>-8.6 mmol/L</td>
<td>-2.0 – +2.0</td>
</tr>
<tr>
<td>Lactate*</td>
<td>23.0 mmol/L</td>
<td>0.2 – 2.5</td>
</tr>
<tr>
<td>Sodium*</td>
<td>147 mmol/L</td>
<td>137 – 145</td>
</tr>
<tr>
<td>Potassium*</td>
<td>6.7 mmol/L</td>
<td>3.2 – 4.5</td>
</tr>
<tr>
<td>Chloride*</td>
<td>95 mmol/L</td>
<td>100 – 110</td>
</tr>
</tbody>
</table>

a) List the acid-base abnormalities.
Lactic acidosis
Anion gap elevation (37 mEq/L)
Metabolic alkalosis
Respiratory alkalosis

Pass rate 86.1%
Highest mark 9.25
7. As director of ICU, the general manager of your hospital asks you to review your current infection control policy following an increase of 200% in the number of newly acquired MRSA infections during an ICU admission in the past 2 months.

Outline your approach to this request.

1. Obtain relevant details of the increase in infection rate- is it a real increase i.e. is there an increase in the rate of MRSA per 100 or 1000 admissions or have the no of admission gone up significantly too, clinical relevance of finding i.e. is the 200% related to case mix changes, no of patients, demographics and type of patients, duration of ICU stay, details of ICU stay/ procedures/ antibiotic usage, genetic of MRSA- community acquired/ hospital acquired, is MRSA same or similar to the prevalent strain or is it a new strain

2. Review current infection control policy- when it was written, people involved in writing it, MRO rate at the time of writing policy.

3. Get expert help- infectious disease specialists, infection control specialist either from your hospital or from elsewhere.

4. For a review team with yourself, 1 or 2 other intensivists form your ICU, ICU nursing mangers and 1 or 2 nurses and infectious diseases/ infection control experts.

5. Review cases, review previous policy, review experience in peer hospitals in your vicinity and in your state if available, perform a literature review of the evidence base on this topic.

6. Document all above in the form of a report with key findings and recommendations-key finding should include- clinical relevance of findings, cohort of patients affected, current rate of MRSA in your hospitals compared to peer hospitals, postulated causes for the current increase, possible causes why current infection control policy may not have been effective, antibiotic usage and their impact on the increase in MRSA. Recommendations include- infection control i.e. hand washing, vector control if relevant, rigorous cleaning of bed spaces and areas of clinical use. Antibiotic use review, consider antibiotic stewardship, employing appropriate staff for infection control e.g. cleaners, infection control nurse, regular infectious diseases consultant rounds. May need to prepare business case if any new staff or equipment will be needed. Also regular review of rates and distribution of these to all staff should be considered.

7. Discuss report with staff in your ICU for comments or any suggestions.

8. Submit report to general manager

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>25%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>7.25</td>
</tr>
</tbody>
</table>
8.1. A 40 year old previously well male presents with a ruptured appendix and associated peritonitis (Day 0). He returns to theatre 3 days later with ischaemic colitis and requires a right hemicolectomy. At laparotomy, he is noted to have extensive thrombosis in his superior mesenteric vein and portal vein. Attempts to anticoagulate him postoperatively (day 5 onwards) with intravenous heparin have been unsuccessful.

His post op haematology results are as follows:

<table>
<thead>
<tr>
<th></th>
<th>Day 0</th>
<th>Day 1</th>
<th>Day 3</th>
<th>Day 5</th>
<th>Day 7</th>
<th>Day 9</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>INR</td>
<td>1.2</td>
<td>1.7</td>
<td>1.8</td>
<td>1.6</td>
<td></td>
<td></td>
<td>0.8 – 1.3 seconds</td>
</tr>
<tr>
<td>APTT</td>
<td>36</td>
<td>38</td>
<td>36</td>
<td>28*</td>
<td>31*</td>
<td>37*</td>
<td>24 – 35 seconds</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>5.8</td>
<td>1.8</td>
<td>36</td>
<td>28*</td>
<td>31*</td>
<td>37*</td>
<td>2.0 – 5.0 g/L</td>
</tr>
<tr>
<td>INR mix</td>
<td>1.9</td>
<td>1.4</td>
<td>1.7</td>
<td></td>
<td></td>
<td></td>
<td>0.8 – 1.3 seconds</td>
</tr>
<tr>
<td>APTT mix</td>
<td>32.5</td>
<td>30</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>30 – 40 seconds</td>
</tr>
<tr>
<td>D dimer</td>
<td>&gt;4.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&lt; 0.5 mg/L</td>
</tr>
</tbody>
</table>

* On I.V. heparin
APTT therapeutic range for I.V. heparin therapy: 60 – 90 seconds

Additional tests performed on Day 7
A. Tests of hypercoagulability (plasma)
Antithrombin (functional) 20% (Reference: 80 – 120%)

B. Factor assays (plasma)
Factor VIII 4.10 IU/ml (Reference: 0.5 – 1.5)

C. Anti-Factor Xa assay (plasma)
Anti-Factor Xa 0 IU/ml (Reference for IV heparin therapy: 0.3 – 0.7)

a) What are the possible factors preventing therapeutic anticoagulation in this patient?

- Disseminated intravascular coagulation
- High clot burden
- Antithrombin III deficiency
- High Factor VIII levels

b) List 2 strategies to effect anticoagulation with intravenous heparin.

- Change to low molecular heparin, instead of unfractionated heparin
- Give cryoprecipitate and/or fresh frozen plasma (if there is confirmed ATIII deficiency)
- Give antithrombin III concentrate
8.2. A 28 year old man presented with a persistent epistaxis to the emergency department.

The coagulation profile was as follows:

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>INR</td>
<td>1.2</td>
<td>0.8 – 1.2</td>
</tr>
<tr>
<td>APTT</td>
<td>50 seconds</td>
<td>25 – 39</td>
</tr>
<tr>
<td>Platelets</td>
<td>250 X 10⁹/L</td>
<td>150 – 350</td>
</tr>
<tr>
<td>Bleeding time*</td>
<td>16 minutes</td>
<td>2 – 8</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>3 g/L</td>
<td>1.5 – 4</td>
</tr>
<tr>
<td>FDPs</td>
<td>&lt; 10 mg/L</td>
<td>0 – 10</td>
</tr>
<tr>
<td>Thrombin clotting time</td>
<td>15 seconds</td>
<td>12 – 17</td>
</tr>
</tbody>
</table>

a) **What is the most likely diagnosis?**
Von Willebrand’s disease

b) **What would you confirm your diagnosis**
- History – easy bruising, mucosal bleeding
- Family history
- Plasma vWF levels
- Factor VIII levels/activity

8.3. A 50 year old female presents with a right deep vein thrombosis and haemoptysis.

These blood results are from her admission:

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>PT</td>
<td>12 seconds</td>
<td>12 – 14</td>
</tr>
<tr>
<td>APTT*</td>
<td>69 seconds</td>
<td>34 – 38</td>
</tr>
<tr>
<td>Thrombin time</td>
<td>16 seconds</td>
<td>14 – 18</td>
</tr>
<tr>
<td>APTT mixing test</td>
<td>60 seconds</td>
<td></td>
</tr>
</tbody>
</table>

a) **What is the APTT mixing test and what is its significance in this patient?**

It involves mixing patient’s plasma with normal pooled platelet free plasma. If it normalized then the elevated APTT is due to factor deficiency. Partial correction suggests an inhibitor.

These results suggest antiphospholipid syndrome in this patient

Pass rate 42%
Highest mark 8.0
9.1. A 52 year old female with systemic lupus erythematosus (SLE) and worsening dyspnoea on exertion presents with the following respiratory function tests:

<table>
<thead>
<tr>
<th>Test</th>
<th>Actual</th>
<th>Predicted</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV₁</td>
<td>1.96 litres</td>
<td>2.66 litres</td>
</tr>
<tr>
<td>FVC</td>
<td>2.52 litres</td>
<td>3.11 litres</td>
</tr>
<tr>
<td>FEV₁/FVC</td>
<td>78%</td>
<td>85%</td>
</tr>
<tr>
<td>PEF</td>
<td>7.50 L/sec</td>
<td>6.47 L/sec</td>
</tr>
<tr>
<td>FRC</td>
<td>2.18 litres</td>
<td>2.77 litres</td>
</tr>
<tr>
<td>RV</td>
<td>1.08 litres</td>
<td>1.84 litres</td>
</tr>
<tr>
<td>TLC</td>
<td>3.64 litres</td>
<td>5.17 litres</td>
</tr>
<tr>
<td>DLco</td>
<td>10.4 ml/min/mmHg</td>
<td>24.7 ml/min/mmHg</td>
</tr>
<tr>
<td>KCO (DICO/VA)</td>
<td>2.85 ml/min/mmHg</td>
<td>4.77 ml/min/mmHg</td>
</tr>
</tbody>
</table>

a. Describe and explain the results of the respiratory function tests. Suggest 1 possible diagnosis.

- Moderate restrictive defect
- High peak expiratory flow; due to fibrotic lung stretching airways open on full inspiration
- Small residual volume; due to cellular infiltration / fibrosis resulting in reduced lung compliance
- Reduced DLco (impaired gas transfer) due to both;
  A) Reduced lung expansion (restriction) and
  B) Damage to the lung parenchyma

Diagnosis: Pulmonary fibrosis.
9.2. You are asked to review a 44 year old male known epileptic following a prolonged generalised tonic-clonic convulsion. He is intubated and ventilated. Arterial blood gas analysis is as follows:

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>FiO₂</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>pH*</td>
<td>7.15</td>
<td>7.35 – 7.45</td>
</tr>
<tr>
<td>pCO₂</td>
<td>35 mmHg (4.6 kPa)</td>
<td>35 – 45 (4.6 – 6)</td>
</tr>
<tr>
<td>pO₂*</td>
<td>105 mmHg (14 kPa)</td>
<td>75 – 98 (10 – 13)</td>
</tr>
<tr>
<td>HCO₃*</td>
<td>10.3 mmol/l</td>
<td>22 – 26</td>
</tr>
</tbody>
</table>

a) List the abnormalities on the blood gas and give the most likely cause of each abnormality.

- Metabolic acidosis – lactic acidosis secondary to prolonged seizures
- Respiratory acidosis (or inadequate compensation) – central hypoventilation or inadequate mechanical ventilation
- Increased A-a gradient - aspiration pneumonia
10. Compare and contrast the use of continuous veno-venous haemodialysis (CVVHD), intermittent haemodialysis (IHD) and slow continuous ultrafiltration (SCUF) in the intensive care patient.

<table>
<thead>
<tr>
<th></th>
<th>CVVHD</th>
<th>SCUF</th>
<th>IHD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vascular access</strong></td>
<td>Good vascular access required via double lumen catheter in central vein</td>
<td>Good vascular access required via double lumen catheter in central vein</td>
<td>Double lumen catheter or A-V fistula Higher flows than with CVVHD or SCUF</td>
</tr>
<tr>
<td><strong>Anticoagulation</strong></td>
<td>Continuous anticoagulation generally required</td>
<td>Continuous anticoagulation generally required</td>
<td>Intermittent anticoagulation only while on dialysis</td>
</tr>
<tr>
<td><strong>Fluid shifts</strong></td>
<td>Slow fluid shift</td>
<td>Slow fluid shift</td>
<td>Rapid fluid shift</td>
</tr>
<tr>
<td></td>
<td>Least fluid removed per hour</td>
<td>More fluid removed than CVVHD but less than IHD</td>
<td>Greatest fluid removal possible over time</td>
</tr>
<tr>
<td><strong>Electrolyte shifts</strong></td>
<td>Slow electrolyte shift all sized molecules removed</td>
<td>Small molecules removed much less than CVVHD and IHD</td>
<td>Rapid electrolyte shift all sized molecules removed but less than CRRT</td>
</tr>
<tr>
<td><strong>Cerebral dysequilibrium</strong></td>
<td>Disequilibrium uncommon</td>
<td>No disequilibrium</td>
<td>Disequilibrium syndrome more likely, but still not common</td>
</tr>
<tr>
<td><strong>Mode of solute clearance and efficiency</strong></td>
<td>Ultrafiltration Convection, diffusion, adsorption</td>
<td>Ultrafiltration only not intended for solute clearance</td>
<td>Ultrafiltration, diffusion, convection, less adsorption Most efficient</td>
</tr>
<tr>
<td><strong>Haemodynamic stability</strong></td>
<td>Significantly reduced haemodynamic instability</td>
<td>Minimal haemodynamic instability</td>
<td>Higher incidence of Haemodynamic instability</td>
</tr>
<tr>
<td><strong>Practical considerations</strong></td>
<td>Needs expertise and equipment</td>
<td>Needs expertise and equipment</td>
<td>Expertise more widespread</td>
</tr>
<tr>
<td><strong>Cost</strong></td>
<td>Most costly</td>
<td>Less cost than CVVHD</td>
<td>Cheapest</td>
</tr>
</tbody>
</table>

Pass rate 67%
Highest mark 9.0
11. The organisms below were isolated and demonstrated antimicrobial sensitivities as listed.

Enterococcus faecalis

<table>
<thead>
<tr>
<th>Drug</th>
<th>Sensitivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ampicillin</td>
<td>R</td>
</tr>
<tr>
<td>Chloramphenicol</td>
<td>R</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>R</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>R</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>R</td>
</tr>
<tr>
<td>Nitrofurantoin</td>
<td>R</td>
</tr>
<tr>
<td>Cotrimoxazole</td>
<td>R</td>
</tr>
<tr>
<td>Teicoplanin</td>
<td>R</td>
</tr>
<tr>
<td>Vancomycin</td>
<td>R</td>
</tr>
</tbody>
</table>

Klebsiella pneumoniae

<table>
<thead>
<tr>
<th>Drug</th>
<th>MIC (µg/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cefpodoxime</td>
<td>≥ 2</td>
</tr>
<tr>
<td>Ceftazidime</td>
<td>≥ 2</td>
</tr>
<tr>
<td>Aztreonam</td>
<td>≥ 2</td>
</tr>
<tr>
<td>Cefotaxime</td>
<td>≥ 2</td>
</tr>
<tr>
<td>Ceftriaxone</td>
<td>≥ 2</td>
</tr>
</tbody>
</table>

a) What is the significance of these results?

b) List 1 appropriate antimicrobial in each case.

Van A VRE
Linezolid, Daptomycin, (Tigecycline, Quinupristin-dalfopristin)

ESBL
Carbapenem (imipenem, meropenem, and perhaps ertapenem), Colistin, Aminoglycosides, Ciprofloxacin

c) List the strategies available to reduce the development of these organisms in ICUs.

Strategies to improve the efficacy and utilization of antimicrobial therapy
- Antibiotic evaluation committees
- Protocols and guidelines to promote appropriate antimicrobial utilization
- Hospital formulary restrictions of broad-spectrum agents
- Substitution of narrow-spectrum antibiotics (such as first generation cephalosporins and aminoglycosides)
- Mandatory consultations with infectious diseases specialists
- Antibiotic cycling by regularly cycling different antimicrobial classes
Infection control measures
- Handwashing compliance: Alcohol-based hand wash is more effective than traditional soap and water in cleansing hands of bacteria
- Barrier precautions with gloves and gowns
- Isolation
- Surveillance for multidrug-resistant bacteria for the early identification and control
- Monitoring and disseminating the incidence and prevalence of isolation of multidrug-resistant bacteria
- Limiting LOS and invasive devices (idc / ett/ vascular)

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Pass rate</td>
<td>61%</td>
</tr>
<tr>
<td>Highest mark</td>
<td>8.25</td>
</tr>
</tbody>
</table>

12. List the major biochemical abnormalities that are usually associated with the following conditions:

a) Adrenal insufficiency.
b) Refeeding syndrome.
c) Tumour lysis syndrome.
d) Ethylene glycol toxicity.

<table>
<thead>
<tr>
<th>Adrenal Insufficiency</th>
<th>Hyponatraemia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hyperkalaemia</td>
</tr>
<tr>
<td></td>
<td>Non anion gap acidosis</td>
</tr>
<tr>
<td></td>
<td>Hypoglycaemia</td>
</tr>
<tr>
<td></td>
<td>Hypercalcaemia</td>
</tr>
<tr>
<td>Refeeding Syndrome</td>
<td>Hypophosphataemia</td>
</tr>
<tr>
<td></td>
<td>Hypokalaemia</td>
</tr>
<tr>
<td></td>
<td>Hypomagnesaemia</td>
</tr>
<tr>
<td></td>
<td>Hyperglycaemia</td>
</tr>
<tr>
<td>Tumour Lysis Syndrome</td>
<td>Hyperphosphataemia</td>
</tr>
<tr>
<td></td>
<td>Hyperkalaemia</td>
</tr>
<tr>
<td></td>
<td>Hypocalcaemia</td>
</tr>
<tr>
<td></td>
<td>Hyperuricaemia</td>
</tr>
<tr>
<td></td>
<td>Metabolic acidosis</td>
</tr>
<tr>
<td>Ethylene Glycol Toxicity</td>
<td>High anion gap acidosis</td>
</tr>
<tr>
<td></td>
<td>High osmolar gap</td>
</tr>
<tr>
<td></td>
<td>Hypocalcaemia</td>
</tr>
</tbody>
</table>

| Pass rate | 100%     |
| Highest mark | 9.0    |
An Anaesthetist from a provincial hospital has a 20 year old man with suspected fat embolism syndrome following an isolated femoral fracture that was repaired earlier that day. The patient has become increasingly hypoxic and difficult to ventilate, but transfer to a metropolitan centre has been delayed for 12 hours due to bad weather.

His arterial blood gases on SIMV mode of ventilation are as follows: \( \text{FiO}_2 \, 1.0, \) pH 7.21, \( \text{PaO}_2 \, 65 \text{ mmHg (8.6 kPa)}, \) \( \text{PaCO}_2 \, 72 \text{ mmHg (9.3 kPa)}, \) \( \text{HCO}_3 \, 28 \text{ mmol/L}. \) He has a four quadrant infiltrate on his chest X-Ray.

Outline the advice that you would give to help your colleague manage this patient’s ventilation.

General
- Confirm Diagnosis
  - ARDS criteria: CXR, PF ratio, Etiology, no overload
    - exclude other etiologies - where is the ETT (not RMB), no pneumothorax, aspiration etc.
    - What ventilator is he using, are you familiar with it’s modes (such as pressure control, volume control)
    - Ventilatory strategy –pressure and volume limitation to minimise barotrauma
    - PEEP increments to effect, ensuring Plateau Pressure < 30 cm H2O
    - Heavy sedation and paralysis to minimize O2 consumption and CO2 generation to GCS 3 and no spontaneous ventilation
    - Targets for ventilation SpO2 > 90-95 and PO2 > 60
    - permissive hypercapnia as long as pH > 7.1
    - prone position probably not appropriate (if staff not experienced)
    - recruitment manoeuvres

Fluids
- CVP only to ~PEEP+2 as maximum
- consider frusemide if CVP PEEP +5
- use inotrope to maintain MAP > 60 - suggest noradrenaline
- Transfuse only for Hb approaching 7
  - Reassure him and make yourself available for advice 24/7

(Mention of NO, liquid ventilation, surfactant, TGI – no role in this setting)

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>64%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>9.75</td>
</tr>
</tbody>
</table>
14. Outline the value of the following in determining prognosis for neurological recovery in an adult patient admitted to ICU, after successful cardiovascular resuscitation from an out-of-hospital cardiac arrest:

a) Peri-arrest data
b) Clinical examination
c) Neuro-imaging
d) Neurophysiology
e) Biomarkers

a. Peri-arrest data:

Initial rhythm, bystander CPR, time to ROSC intuitively helpful and commonly considered, but have not been shown to correlate with individual outcome. Co-morbidities and pre-arrest performance status may determine overall survival.

b. Clinical Examination:

Unreliable and of no predictive value before 24 hours, clinical assessment at ≥ 72 hours conventional
- Appropriate pre-conditions: absence of sedation/relaxants, adequate CVS resuscitation, normothermia, corrected biochemistry etc.
- All data pertains to studies before the common use of therapeutic hypothermia, and the effect of this intervention unknown. May need longer than 72 hours to obtain reliable data from CNS examination in patients treated with induced hypothermia
- GCS < 4, absent corneal response, absent pupillary response to light indicative of poor prognosis
- Myoclonus not sufficiently predictive to be reliable in isolation but myoclonic status epilepticus is a poor prognostic feature

c. Neuro-imaging:

- CT may be performed early to exclude a CNS cause of arrest
- CT signs of poor prognosis include qualitative assessment, and quantitative assessment of white matter Houndsfield unit ratio. Optimum timing not clear
- MRI demonstration of diffuse cortical lesions or sub-cortical lesions is associated with poor outcome

d. Neurophysiology:

- No neurophysiology study reliably predicts outcome at < 24 hours
- EEG findings of: diffuse suppression to < 20 mV, burst suppression, generalised seizures, diffuse periodic complexes indicate poor prognosis
- EEG shown to have increased false positive prediction for poor outcome after induced hypothermia
- SSEP: bilaterally absent cortical responses to median nerve stimulation seems highly accurate (0% False Positive Rate), not studied after induced hypothermia

e. Biomarkers:

- Neurone specific enolase (NSE) most studied, some studies show 0% FPR for poor outcome, but cut-off levels vary, studies small

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>44%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>9.5</td>
</tr>
</tbody>
</table>
15. You are working as an ICU specialist in a small regional hospital. You are called to give urgent assistance with a 65 year old male who has presented to the Emergency Department with increasing shortness of breath, one week after discharge from a metropolitan hospital following apparently uncomplicated cardiac surgery. Post intubation he has rapidly deteriorated and is now unresponsive with no recordable blood pressure. The cardiac monitor shows sinus tachycardia.

a) How will you respond to this crisis?

- Confirm cardiac arrest
- Good BLS i.e.:
  - Check ETT position (pull back to 22cm), listen to chest and confirm ETCO2 trace(10)
  - Check adequate CPR: correct position (lower half of sternum), correct rate/depth and technique (depress 4-5cm at 100/min and asynchronous ventilation with respiratory rate 8-10)
- Call for additional help
- Confirm IV access
- Continue CPR for 2 min
- Adrenaline

b) You suspect cardiac tamponade. Describe how you would perform blind pericardiocentesis.

- Some asepsis
- Identify landmarks: Left paraxiphoid (traditional) Left parasternal (4th intercostal space left parasternal)
- For a left paraxiphoid approach 45° to the abdominal wall, head for the left shoulder, aspirate as you go
- Could connect a V lead to the base of the needle and watch ECG to look for a change in the QRS morphology, or ST elevation if the needle contacts the myocardium
- Aspirate fluid/blood
- Consider placing a catheter/pigtail
- Blood stained pericardial fluid will not clot whereas intraventricular blood will

c) What clinical signs might have indicated pericardial tamponade as the cause prior to the arrest?

- Distended neck veins
- Muffled heart sounds
- Hypotension
- Tachycardia
- Pulsus paradoxus
- Absent apex beat
- ECG findings – low voltage complexes and electrical alternans

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>25%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>8.1</td>
</tr>
</tbody>
</table>
16. A 72 year old male with known triple vessel coronary disease has returned to the ICU following an uncomplicated coronary artery bypass grafting (CABG). An intraoperative transoesophageal echo showed good left ventricular systolic function and no significant valvular dysfunction. Mediastinal drains have been inserted and there is minimal blood loss. Thirty minutes after return to the ICU his BP falls to 70/40 mmHg. Give the 4 most likely causes for this.

- Artifactual due to inaccurate monitoring system
- Hypovolaemia
- Vasodilatation associated with re-warming
- Vasodilatation associated with drugs (anti-hypertensives, vasodilators, sedation
- Anaphylaxis
- Pneumothorax
- Cardiac tamponade

Outline your immediate management

- Check monitoring system
- Fluid bolus
- Stop vasodilators
- Consider use of a short acting vasopressor such as metaraminol
- If not responding to fluid and short acting vasopressors consider a catecholamine infusion such as noradrenaline
- Cease any potential allergens

The patient’s BP stabilises and 2 hours later you are called to review him since he has developed significant shivering. List 4 possible treatment options

- Increase sedation / analgesia
- Consider paralysis
- Increase external warming
- Consider pethidine

On day 1, the patient is extubated and stable. You are shown the following ECG:

What does it show?

- Diffuse concave ST elevation suggestive of pericarditis

What treatment is required?

- If the patient is asymptomatic, then no treatment is required

1 hour later, quite unexpectedly, the patient has a VF cardiac arrest. What are your principles of management?

- Basic ALS. Immediate defibrillation, ensure airway and ventilate with 100% oxygen
- Contact the cardiothoracic team immediately and prepare for possible sternotomy
- Internal cardiac massage and internal defibrillation if re-sternotomy on the ICU

| Pass rate | 67% |
| Highest mark | 9.0 |
17. Critically evaluate the use of sodium bicarbonate therapy in Diabetic Ketoacidosis

- Definition of DKA and its pathophysiological consequences
- The possible rationale for the use of sodium bicarbonate
  - Severe acidaemia (generally pH < 7.10 although no hard data)
  - Severe hyperkalemia
  - Bicarbonate loss from Renal or GI tract
- The possible problems of giving sodium bicarbonate
  - Worsening of intracellular acidaemia
  - Hypokalaemia & Hypernatraemia
  - Large bolus of hypertonic solution
- No evidence for the use of HCO₃⁻ to treat acidaemia, or improve cardiac contractility. In fact many different texts have different values for the cut off pH which requires treatment, suggesting no real consensus.
- The correction of the acidaemia is achieved by correcting the underlying pathophysiology with fluids and insulin
- Some evidence for the use of HCO₃⁻ in hyperkalaemia, as a temporising measure, assuming underlying renal function is maintained
- Theoretical potential for giving HCO₃⁻ with renal wasting of HCO₃⁻ or GI loss if delta ratio is <1 (usual for DKA)
- Evidence suggesting that HCO₃⁻ is associated with worse outcome, however this in paediatrics, in patients who presented sicker (lower PaCO₂ and higher urea on presentation). However this does not assume causality and paediatric patients can compensate for longer.
- Despite the lack of evidence it would appear that most intensivists have a personal cut-off pH at which they consider giving HCO₃⁻

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>22%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>7.25</td>
</tr>
</tbody>
</table>
18.

1. The blood results of a 75 year old who presents with lethargy, confusion and weight loss are shown below:

<table>
<thead>
<tr>
<th></th>
<th>Patient value</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium</td>
<td>141 mmol/L</td>
<td>135 – 145</td>
</tr>
<tr>
<td>Potassium</td>
<td>3.8 mmol/L</td>
<td>3.5 – 5.0</td>
</tr>
<tr>
<td>Chloride</td>
<td>100 mmol/L</td>
<td>97 – 109</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>29 mmol/L</td>
<td>24 – 32</td>
</tr>
<tr>
<td>Urea</td>
<td>11.5 mmol/L</td>
<td>3.0 – 8.0</td>
</tr>
<tr>
<td>Creatinine</td>
<td>150 µmol/L</td>
<td>70 – 110</td>
</tr>
<tr>
<td>Calcium</td>
<td>4.69 mmol/L</td>
<td>2.10 – 2.60</td>
</tr>
<tr>
<td>Phosphate</td>
<td>0.4 mmol/L</td>
<td>0.8 – 1.5</td>
</tr>
<tr>
<td>Albumin</td>
<td>44 G/L</td>
<td>38 – 48</td>
</tr>
</tbody>
</table>

a) What is the likely diagnosis?
   Underlying malignancy

b) What other biochemistry would you request and why?
   PTH to exclude primary hyperPTH – very high calcium indicates malignancy but patients with malignancy have higher incidence of hyperPTH than general population so both conditions can co-exist

c) Briefly explain the pathogenesis of the biochemical abnormalities.
   Hypercalcaemia:
   Malignancy:
      PTH rp (Parathyroid related peptide)
      Ectopic PTH,
      Bone lysis
   Increased PTH:
      Leading to increased osteoclast activity leads to hypercalcaemia by bone reabsorption. It also acts at renal tubule to reabsorb Ca++ and increases conversion 25-OHD to 1,25 (OH)2D increases intestinal absorption of Ca++

   Elevated urea and creatinine:
      Secondary to hypovolaemia

d) Outline your management of this patient.

   • Fluid replacement with NS
   • Biphosphonates
   • Calcitonin
   • Steroids act by decreasing calcitriol
   • Dialysis
   • Treat underlying malignancy
   • Parathyroidectomy if raised PTH
   • (Diuretics no longer recommended)
2. A 35-year-old female with a history of poorly controlled hypertension presents with paraesthesia and weakness. Her blood results are shown below:

<table>
<thead>
<tr>
<th></th>
<th>Patient value</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium</td>
<td>145 mmol/L</td>
<td>135 – 145</td>
</tr>
<tr>
<td>Potassium</td>
<td>1.8 mmol/L</td>
<td>3.5 – 5.0</td>
</tr>
<tr>
<td>Chloride</td>
<td>85 mmol/L</td>
<td>97 – 109</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>40 mmol/L</td>
<td>24 – 32</td>
</tr>
<tr>
<td>Urea</td>
<td>3.4 mmol/L</td>
<td>3.0 – 8.0</td>
</tr>
<tr>
<td>Creatinine</td>
<td>80 µmol/L</td>
<td>70 – 110</td>
</tr>
</tbody>
</table>

Arterial blood gases

<table>
<thead>
<tr>
<th></th>
<th>Patient value</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.56</td>
<td>7.35 – 7.45</td>
</tr>
<tr>
<td>pO₂</td>
<td>85 mmHg (11.3 kPa)</td>
<td>80 – 110 mmHg (10.5 – 14.5 kPa)</td>
</tr>
<tr>
<td>pCO₂</td>
<td>46 mmHg (6.1 kPa)</td>
<td>35 – 45 mmHg (4.6 – 5.9 kPa)</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>40 mmol/L</td>
<td>23 – 33</td>
</tr>
</tbody>
</table>

a) Interpret these results

Metabolic alkalosis with partial respiratory compensation and severe hypokalaemia

b) List 2 likely diagnoses

Primary Hypertension most likely secondary to an aldosterone producing adenoma (Conn’s syndrome – 50-60%) or adrenal hyperplasia (40-50%)
Licorice ingestion
Liddle’s syndrome
Excessive diuretic use

c) Give 2 drugs used to treat this condition

Aldosterone antagonist (spironolactone or eplerenone)
Amiloride

d) List 3 other potential causes of these biochemical abnormalities

1/ laxative abuse
2/vomiting
3/diarrhoea
3/cushings and ACTH tumors
5/ primary metabolic alkalosis
19. With regard to the EEG:

a) List three indications for the use of the EEG in a critically ill patient

- Detection of non convulsive seizures and characterization of spells in patients with altered mental status with: (A history of epilepsy, Fluctuating level of consciousness, Acute brain injury, Recent convulsive status epilepticus, Stereotyped activity such as paroxysmal movements, nystagmus, twitching, jerking, hippus, autonomic variability)
- Monitoring of ongoing therapy: Induced coma for elevated intracranial pressure or refractory status epilepticus, Assessing level of sedation
- Prognosis: Following cardiac arrest, acute brain injury

b) What are the clinical implications of non-convulsive status epilepticus (NCSE) in the critically ill patient?

An underdiagnosed entity.

In several studies, the presence of NCSE and delay to diagnosis and treatment were each associated with significantly more frequent mortality. Periodic epileptiform discharges (PED) have also been associated with a significant increase in death or severe disability at hospital discharge in particular in neurologic disease/injury. NCSE may also occur in those without primary brain injury e.g. sepsis and conveys the same prognosis. Aggressive treatment as for convulsive status epilepticus is recommended.

c) List two EEG patterns that may be seen after hypoxic brain injury thought to be associated with a poor prognosis.

Note: none of these patterns are specific for death or poor outcome and must be regarded along with clinical assessment.

- Generalised suppression/isoelectric
- Generalised burst suppression especially if accompanied by epileptiform activity
- Epileptiform and generalised periodic discharges, especially myoclonus
- Alpha pattern coma

| Pass rate  | 56% |
| Highest mark | 8.0 |
“Damage control resuscitation” as applied to the management of the major trauma patient integrates permissive hypotension, haemostatic resuscitation and damage control surgery.

Outline the key principles of each of these three strategies, including the rationale.

a. Permissive hypotension

1. Keep SBP low enough to avoid exsanguination but high enough to maintain perfusion.
2. Relates to disruption of an unstable clot by higher pressures and worsening of bleeding.

b. Haemostatic resuscitation

i. Correct hypothermia

1. Decreases platelet responsiveness.
2. Increases platelet sequestration in liver and spleen
3. Reduces Factor function eg Factors XI and XII
4. Alters fibrinolysis

ii. Correct acidosis

1. pH strongly effects activity of Factors V, VIIa and X.
2. Acidosis inhibits thrombin generation
3. Cardiovascular effects of acidosis (pH <7.2) – decreased contractility and CO, vasodilatation and hypotension, bradycardia and increased dysrhythmias.

iii. Treat coagulopathy early and aggressively

1. Many coagulopathic changes occur early after trauma, therefore need to correct early.
2. Use much higher FFP to PRBC ratios (1:1/2:3) than previously used. Is associated with improved survival.
3. Higher platelet to PRBC transfusion ratios also becoming more popular but evidence is less clear.
4. Cryoprecipitate provides an additional option for Factor replacement for a lower volume of fluid.
5. rFVIIa has been used in trauma, but off label and anecdotally.

iv. The use of blood products instead of isotonic crystalloid fluid aiming for limited volume replacement

1. Large volume crystalloids can lead to dilutional coagulopathy and exacerbate bleeding.
2. Crystalloids have no O2 carrying capacity and do little to correct the anaerobic metabolism and O2 debt associated with shock.
3. Need less volume of blood product therefore likely to be less tissue and organ (eg lung, small intestine mucosa) oedema and failure (eg pulmonary oedema, abdominal compartment syndrome)
4. Hypertonic saline is another option (proven restored microvascular flow, decreased tissue oedema, attenuated inflammatory response).
c. Damage control surgery

1. Management of the metabolic derangement of ongoing bleeding supersedes the need for definitive surgery
2. Abbreviated operations that control haemorrhage and contain spillage from the alimentary and urogenital tracts.
3. Rapid transfer to ICU for correction of acidosis, coagulopathy and hypothermia
4. Definitive operation is deferred.
5. These operations tend to have a high complication rate
6. Survival is given preference over morbidity.

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>47%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>7.0</td>
</tr>
</tbody>
</table>

21. A 40 year old man with a history of ankylosing spondylitis and known difficulty with intubation on previous elective surgery is admitted to your ICU for hypoxic respiratory failure. A decision to perform a semi-elective, awake fiberoptic intubation in the ICU has been made.

Describe how you will prepare for this procedure.

(a) Preparation of patient

- Consent/explanation of procedure
- Obtain history of previous airway difficulty, technique used, complications, etc. (from patient, letter from anaesthetist). H/o allergies - esp. to local anaesthetics. Fasting status. Other co-morbidities, eg. coagulopathy.
- Clinical assessment of airway itself, mouth opening, nasal cavity/septum, range of neck movement, mental status including ability to understand and cooperate with proposed procedure, degree of hypoxia and ability to pre-oxygenate.

(b) Preparation of environment/personnel

- Appropriate lighting with ability to dim.
- Monitoring - ECG, pulse oximetry, arterial line, capnography set up.
- Adequate and working IV access
- Establish comfortable and adequate patient position, pillows, etc.
- Request help and ensure availability as appropriate - eg. Anaesthetist
- Ensure presence of adequate skilled assistants. Inform them in detail of steps of procedure and assign roles, as appropriate. (eg. observation of patient, administration of sedatives, optimisation of patient position, injection of LA, etc)
- Discuss a plan B, if technique were to fail.
- Keep resuscitation trolley easily available and ensure difficult airway equipment available.
(c) Preparation of equipment

- Check oxygen source and suction
- Check equipment for bronchoscopy- Intubating bronchoscope, light source, lubricant, suction for bronchoscope, (oxygen can be applied alternately through same port using 3-way tap) and injection port for local anaesthetic. Apply defogging solution, if available.
- Airway equipment- range of oral and nasal armoured tubes of appropriate size, oral intubating airways, soft nasopharyngeal airways, appropriate size laryngeal mask airway. Depending on choice of oral or nasal intubation, check, lubricate and load chosen tube onto bronchoscope.
- Equipment required for plan B.

(d) Preparation of drugs

- Systemic-
  - Antisialagogue- eg. glycopyrrolate
  - Consider proton pump inhibitor.
  - Midazolam/Fentanyl as appropriate (small doses as patient should be able to cooperate)

- Local anaesthetics- Very important in order to achieve success. Ensure not to exceed recommended doses and allow adequate time to act.
  - Nasal cavity and nasopharynx- 10% lignocaine spray with phenylephrine spray or cotton tipped pledgets soaked in 4% cocaine or nebuliser filled with 5ml of 4% lignocaine.
  - Oral cavity and oropharynx- 10% lignocaine spray or 2% lignocaine viscous gargles.

- Extra local anaesthetic may be required to spray during advancement of bronchoscope.

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>33%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>6.25</td>
</tr>
</tbody>
</table>

22. A 45 year old man was admitted to the intensive care unit after sustaining 40% BSA burns in a house fire. He was transported initially to a local hospital where initial resuscitation was commenced including mechanical ventilation for suspected inhalational injury. On arrival in your ICU an arterial blood gas was taken which is shown below:

<table>
<thead>
<tr>
<th></th>
<th>Patient value</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.14</td>
<td></td>
</tr>
<tr>
<td>pCO₂</td>
<td>34 mmHg</td>
<td>(4.5 kPa)</td>
</tr>
<tr>
<td>pO₂</td>
<td>195 mmHg</td>
<td></td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>8 mmol/L</td>
<td>24 – 32</td>
</tr>
<tr>
<td>Standard Base Excess</td>
<td>-16.1 mmol/L</td>
<td>-2.0 – +2.0</td>
</tr>
<tr>
<td>Chloride</td>
<td>120 mmol/L</td>
<td>98 – 108</td>
</tr>
<tr>
<td>Sodium</td>
<td>145 mmol/L</td>
<td>133 – 145</td>
</tr>
<tr>
<td>Potassium</td>
<td>4.8 mmol/L</td>
<td>3.2 – 4.5</td>
</tr>
<tr>
<td>Haemoglobin</td>
<td>180 g/L</td>
<td>115 – 160</td>
</tr>
<tr>
<td>Arterial Lactate</td>
<td>3.8 mmol/L</td>
<td>&lt; 1.5</td>
</tr>
</tbody>
</table>
a) List four potential contributing causes of the metabolic derangement

- Shock/Underesuscitation/hypovolaemia (elevated Hb and Lactate)
- Normal (0.9%) Saline fluid resuscitation
- Carbon monoxide poisoning
- Cyanide toxicity from smoke inhalation (elevated anion gap acidosis)
- Other missed injuries e.g. abdominal trauma, bleeding etc leading to hypoperfusion/shock
- Potential concurrent ingestions e.g. methanol, ethylene glycol

b) How would you classify the acid base derangement and explain your reasoning?

- Mixed metabolic acidosis

(Note: CO2 is also high for pH but less relevant because patient on IPPV)
Delta ratio indicates a greater fall in [HCO3-] than expected given increase in AG. This can be explained by a mixed metabolic acidosis, i.e. a combined high anion gap and normal anion gap acidosis.

c) The serum albumin is 18g/L. Outline how would this affect the anion gap.

- The plasma proteins are the major source of unmeasured anions. Hypoalbuminemia may mask an increased concentration of gap anions by lowering the value of the anion gap. Adjustment of the anion gap can be made by the application of correction factors (see Figge et al, CCM 1998).

d) Whilst on your ward round the RMO asks your opinion on the Stewart approach to acid base physiology. List the 3 independent variables that comprise this approach

- Strong ion difference
- Partial CO2 tension
- Total concentration of weak acid (ATOT)

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>83.3%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>10</td>
</tr>
</tbody>
</table>

23. A 35 year old female is 39 weeks pregnant. Her pregnancy has been complicated by hypertension and proteinuria. Her blood pressure is 160/120 mm Hg. You are called to the labour ward when she suffers a generalised (“grand mal”) convulsion.

Outline your overall plan of management.

**Initial management**
- ABC – ensure patent airway, oxygen via reservoir mask or bag-valve-mask assembly and support ventilation as needed
- Left lateral tilt
- Terminate the seizure
- Diazepam 5-10mg or Mg 4g IV up to 8 g
- Monitors / investigations
Management of Hypertension
Hydralazine
Labetalol
(Other agents are acceptable – late in pregnancy – increasing trend to use “mainstream” agents)

Treatment of convulsions
MgSO4 bolus followed by maintenance MgSO4
(Shown to be more effective than phenytoin or diazepam in preventing recurrent seizures)
Addition of Benzodiazepine / Barbiturate if recurrent seizures despite MgSO4

Planning for delivery
Brief period of resuscitation once seizures controlled.

Post partum management
Continue anti-convulsants until patient improves (diuresis, fall in BP).

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>94%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>10</td>
</tr>
</tbody>
</table>

24. A 38-year-old man with type 1 diabetes mellitus presents with two days of severe thigh pain. You are called to see him because of hypotension. On examination he is drowsy, BP 80/60 mmHg, HR 140/min and temperature of 40.2°C. There is gross swelling on the medial aspect of his right thigh with clear cellulitis and visible central necrosis.

Describe the management priorities in the first 24 hours and briefly justify your responses.

1. Resuscitation
   • High flow oxygen,
   • Support BP with fluids +/- vasopressors
   • Measure & fix BSL

2. Antibiotics
   • The presentation is that of necrotising fasciitis. T1DM a significant risk factor. Group A streptococcus (type 2) or polymicrobial aerobic and anaerobic organisms (type1) are both possible. Initial cover should be broad and include an extended spectrum beta-lactam or meropenem, and clindamycin. Clindamycin suppresses toxin formation from GAS, has other favourable in-vitro effects (facilitating phagocytosis).
   • Further survey: extent of cellulitis, perineal involvement.

3. Surgical Referral and post-operative management
   • Requires urgent debridement, with removal of dead/infected tissue back to bleeding tissue
   • Takes priority over other therapies including hyperbaric O2
   • Expectation of major blood loss and massive transfusion
   • Likely to be highly unstable post-operatively with major support requirement
   • Routine ICU care of patient with severe sepsis

4. Specific Therapies
   Intravenous Immunoglobulin
   • In vitro neutralisation of streptococcal super-antigens and clostridial toxins
   • Streptococcal toxic shock syndrome (with or without nec. fasc.) listed as “emerging” indication for IVIG by ARCBS, and available for use
5. **Hyperbaric O₂**
- Observational studies only
- Conflicting results with both reduction and increases in mortality seen cf. observational controls
- Possible reduction in need for debridement
- Usually bd to tds dives of 90 min at 3 atm.
- Severe organ failure may limit logistics

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>83%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>10</td>
</tr>
</tbody>
</table>

25.

1.

a) **What is this skin rash?**
- Erythema multiforme

b) **Give three causes**
- Herpes simplex infection
- Mycoplasma pneumonia
- Streptococcal infections
- Drugs
- SLE
- Leukaemia

c) **Apart from the skin, what other tissues are involved?**
- Mucous membranes, especially oral mucosa

2. **Examine the photograph shown below (Horner’s syndrome)**

a) **List three likely anatomical sites of lesions that can result in these eye signs**

Any three anatomical sites of the lesion:
- Hemispheric lesion (hemispherectomy, massive hemispheric CVA, thalamic CVA)
- Brainstem lesion (brainstem infarct, multiple sclerosis, brainstem tumour or encephalitis)
- Central cord lesion (syringomyelia, glioma, ependymoma, traumatic)
- T1 root lesion (Pancoast tumour, cervical rib, brachial plexus avulsion, aortic or subclavian aneurysm)
- Sympathetic chain (laryngeal, pharyngeal, thyroid or parathyroid surgery, carotid artery lesion, malignancy at base of skull)

b) **Give two associated clinical features that would help determine the site of the lesion?**

Any two of:
- distribution of loss of sweating
- distribution of loss of pain and temperature sensation
- motor deficit
- signs of central cord syndrome
wasting of small muscles of hand and clubbing
- cervical LN s
- Signs of head / neck surgery/trauma
- Subclavian artery bruit

3. A patient who underwent cardiac catheterization yesterday has this appearance of her foot.

a) What is the appearance due to?
- Cholesterol emboli or atheromatous emboli
- (accept ischaemia secondary to damaged / occluded femoral artery)

b) Give two causes of a metabolic acidosis in this patient related to this event
- acute renal failure
- mesenteric ischaemia
- limb ischaemia

4. The illustrations below illustrate a test being carried out in an unconscious patient. (Oculocephalic reflex) The patient’s head is being moved from side to side.

a) Name the neurological structures involved in the reflex being tested
- VIII nerve and vestibular nucleus
- III and VI nerves and nuclei
- Median longitudinal fasciculus

b) What does the result shown in the illustrations suggest about the cause of unconsciousness?
The cause is unlikely to be due to an anatomical lesion affecting the reticular activating system (Brain stem function is still present)

Pass rate 58%
Highest mark 8.37

26.

1. A 70 year old man was admitted to the emergency department with shortness of breath and hypotension. He was discharged a week ago from hospital after having undergone an uneventful coronary artery bypass grafting procedure.

a) What is the major abnormality on the ECG and what is the likely diagnosis?
Electrical alternans, Pericardial tamponade

b) What investigation is required to confirm your diagnosis?
Echocardiography.
2. Examine the trace from a patient with a pacemaker in the DDD mode. What problem is seen with this pacemaker?

Failure to sense the atrium

3. A 60 year old diabetic man was admitted following a syncopal episode to the emergency department. His GCS is 8. You have been called to assess him with a view to taking him to ICU.

a) Comment on the ECG

Normal ECG with sinus rhythm

b) List 5 further investigations you will perform

BSL, troponin, CT head, Head and neck vascular studies, Holter monitoring, Echo, EEG.

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>44%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>8.25</td>
</tr>
</tbody>
</table>

27.

a) List the possible causes of stridor at rest in a previously well 3 year old child

- viral croup
- epiglottitis
- inhaled foreign body
- severe bilateral tonsillitis, meeting in the midline (e.g. infectious mononucleosis)
- tonsillar abscess
- retropharyngeal infection/abscess
- spasmodic (recurrent allergic) croup
- allergic reaction/angio-oedema
- bacterial tracheitis
- intra-thoracic obstruction vascular rings (less likely in prev. well), peri-tracheal tumours
- diphtheria
- other congenital causes (laryngomalacia, tracheomalacia, tracheal webs etc) unlikely in this setting, no marks for these responses

b) What features elicited on history, examination and imaging would help in refining the diagnosis

1. History:
   - past history including neonatal problems, previous intubation
   - vaccination especially HiB
   - prodrome, URTI symptoms
   - choking episodes (FB)
   - febrile symptoms
   - cough (implies epiglottitis unlikely)
2. Examination
• (minimise disturbance to child, examine in parent's lap)
• toxicity & fever
• swallowing / drooling
• petechial rash in HiB sepsis
• inspect the throat (without instrumentation and if child cooperative), looking for tonsillar hyperplasia, uvula swelling, FB

3. Radiology:
• very limited utility, may be unsafe to transfer
• possibly if radio-opaque FB suspected
• lateral soft tissue neck of no/little value

c) What are the indications for intubation in this situation?
• Complete or imminent airway obstruction
• Worsening airway obstruction despite appropriate therapy (eg steroids + nebulised adrenaline in croup)
• Dangerous reduction in conscious state
• Uncorrectable hypoxaemia

d) List the key management issues in securing the airway
• Call for help
• Choice of anaesthetic technique - inhalational versus intravenous
• Failed intubation drill

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>72%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>8.8</td>
</tr>
</tbody>
</table>

28.

1. With regards to the device pictured below:

a) Identify the lumens / lines labelled A, B, C, D, E

A right atrial lumen
B thermistor
C mixed venous oximeter
D pulmonary artery lumen
E balloon inflation/deflation

b) List the parameters that can be directly measured using this device.

• Right atrial pressure
• Right ventricular systolic and diastolic pressure
• Pulmonary artery systolic and diastolic pressure
• Pulmonary artery occlusion pressure
• Mixed venous saturations
• Core temperature
2. With regards to the endotracheal tube pictured below, what is the purpose of the lumen labelled A

Suction port with aperture above the cuff enables continuous suction of subglottic secretions which and thus may limit nosocomial pneumonia occurring as a consequence of aspiration.

3.

a) What does the cylinder pictured above contain?

b) What parameters are monitored during administration of the cylinder’s contents?

a) Nitric oxide 800ppm and Nitrogen
b) \( \text{PO}_2 \) pulmonary artery pressure, methaemoglobin and nitrogen dioxide

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>50%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>7.45</td>
</tr>
</tbody>
</table>

29.

With reference to a randomized controlled trial, briefly describe the terms “blinding” and “allocation concealment”.

- Blinding and allocation concealment are methods used to reduce bias in clinical trials.
- Blinding: a process by which trial participants and their relatives, care-givers, data collectors and those adjudicating outcomes are unaware of which treatment is being given to the individual participants.
  - Prevents clinicians from consciously or subconsciously treating patients differently based on treatment allocation
  - Prevents data collectors from introducing bias when there is a subjective assessment to be made for eg “pain score”
  - Prevents outcome assessors from introducing bias when there is a subjective outcome assessment to be made for eg Glasgow outcome score.

- Traditionally, blinded RCTs have been classified as "single-blind," "double-blind," or "triple-blind"; The 2010 CONSORT Statement specifies that authors and editors should not use the terms "single-blind," "double-blind," and "triple-blind"; instead, reports of blinded RCT should discuss "If done, who was blinded after assignment to interventions (for example, participants, care providers, those assessing outcomes) and how.

Allocation concealment is an important component of the randomization process and refers to the concealment of the allocation of the randomization sequence from both the investigators and the patient. Poor allocation concealment may potential exaggerate treatment effects.
Methods used for allocation concealment include sealed envelope technique, telephone or web based randomization.
Allocation concealment effectively ensures that the treatment to be allocated is not known before the patient is entered into the study. Blinding ensures that the patient / physician is blinded to the treatment allocation after enrollment into the study.

<table>
<thead>
<tr>
<th>Pass rate</th>
<th>28%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest mark</td>
<td>7.6</td>
</tr>
</tbody>
</table>

30. 

(A) List 2 causes for this end-tidal CO2 pattern.

- Kinked/partially occluded tube
- Foreign body in airway
- Obstruction of expiratory limb of breathing circuit
- Bronchospasm

(B) List 2 causes for this end-tidal CO2 pattern

- Decreased respiratory rate
- Decreased tidal volume
- Increasing metabolic rate (fever, shivering, etc)

(C) Draw and label a pressure-time ventilator waveform showing-

(i) normal followed by
(ii) increased airway resistance.
(D) Draw and label a pressure-volume loop (volume targeted ventilation) showing-

(i) normal lung compliance
(ii) decreased lung compliance
(iii) increased lung compliance.

(E) Draw and label the following for a patient with a large cuff leak-

(i) Flow-time waveform
(ii) Volume-time waveform

Inspiratory flow does not return to zero. Expiratory flow looks normal, but area of expiratory portion less than area of inspiratory portion.

Volume wave does not return to zero at the end of expiration but instead resets at the start of the next ventilator breath.

Pass rate 56%
Highest mark 8.5
HOT CASES

Westmead Hospital

- 44-year-old male, intubated and ventilated in ICU for 16 days, post elective Ivor-Lewis oesophagectomy for distal oesophageal cancer complicated by anastamotic breakdown. Current issues included persistent fevers and poor nutritional state.

- 63-year-old female admitted with respiratory failure and septic shock secondary to severe community acquired pneumonia. Co-morbidities include diabetes, NHL in remission, COPD with prolonged steroid therapy and psoriatic arthropathy. ICU course complicated by ischaemic hepatitis and SVT. Slow to wean due to haemodynamic instability, respiratory failure and obesity.

- 56-year-old male post CABG x 5 with ischaemic cardiomyopathy and acute on chronic renal failure, now dialysis dependent.

- 67-year-old male, one day post AVR for aortic valve endocarditis following MSSA bacteraemia also resulting in cerebral emboli and multiple infarcts; splenic abscess requiring splenectomy; and septic arthritis. Physical signs included embolic phenomena, signs of lateral medullary syndrome, swollen left knee, laparotomy scar and signs of recent cardiac surgery.

- 58-year-old male one week post VF arrest secondary to blocked LAD with some neurological recovery but recent deterioration with acute pulmonary oedema. Other issues included acute kidney injury.

- 64-year-old male with background of CLL and recent chemotherapy, who presented with severe septic shock.

- 35-year-old male with out of hospital VF arrest admitted to ICU with cardiogenic shock following emergency coronary angiography and failed PTCA. Current issues included hypoxia, cardiogenic shock, sepsis, acute kidney injury and uncertain neurological recovery.

- 77-year-old female admitted to ICU from the ward with decreased level of consciousness and respiratory distress requiring intubation. In hospital for one month following fall at home and subsequent problems with swallowing, confusion and delirium. Uncertain underlying diagnosis.

- 29-year-old lady with severe hypoxaemic respiratory failure secondary to aspiration following an elective laparoscopic cholecystectomy.

- 49-year-old male with ongoing sepsis and multi-organ failure due to left lower lobe community-acquired pneumonia. Complications include left sided empyema and septic right knee.

- 39-year-old female, with background of atypical dermatomyositis and immunosuppression, readmitted to ICU with worsening respiratory failure and new onset sepsis. Recent discharge from ICU following PJP for which she required ECMO and prolonged mechanical ventilation. Current issues include neuromuscular weakness and prolonged hospital stay.

- 44-year-old female post resection of left lobe liver for hepatocellular carcinoma with background of partially corrected congenital heart disease.
- 43-year-old female with intra-abdominal sepsis and multi-organ failure, including acute on chronic renal failure. Co-morbidities include COPD, T2DM and severe chronic pain with multiple analgesic use.

- 31-year-old female admitted to ICU with SAH secondary to ACOM aneurysm, treated by coiling. Background history included coarctation of the aorta repaired in 1994 and congenital bicuspid aortic valve.

**Liverpool Hospital**

- Male with neutropenic sepsis, likely catheter related, 2 weeks after stem cell transplant for mantle cell lymphoma. Clinical findings include hyperdynamic shock, systolic murmur, ventilated on minimal oxygen.

- Female with grade IV SAH from PCOM aneurysm treated by coiling and complicated by temporal infarct and recurrent hydrocephalus. Current status unresponsive with right hemispheric infarct, some absent brain stem reflexes and likely to progress to brain death.

- Male polytrauma, pedestrian versus car, unstable cervical and thoracic spine fractures, splenectomy and open book pelvic fractures with external fixation.

- Male polytrauma secondary to MVA, fractured ribs, splenic rupture, long bone injuries and open book pelvic fracture awaiting fixation.

- Right basal ganglia bleed as a complication of warfarin therapy with ARDS and difficulty weaning.

- Female with traumatic SAH and cerebral contusion. History of alcohol abuse. Re-intubated for agitation and confusion, currently has aspiration pneumonia.

- Female with right MCA SAH awaiting coiling. Intubated on minimal support, left hemiparesis, agitated.

- Male polytrauma secondary to MVA, car versus tree, bilateral SAH, facial fractures, chest injuries, fractured thoracic and lumbar vertebrae. Failed intubation secondary to facial injuries requiring urgent tracheostomy.
VIVAS

Viva 1

A 27-year-old male presents with a severe head injury (GCS 4 at the scene), sustained in a high-speed motor vehicle collision. His initial CT scan in the Emergency Department shows a 2cm x 3cm x 2.5cm frontal haemorrhagic contusion and diffuse oedema. He is taken directly to the operating theatre where an external ventricular drain (EVD) is inserted. The patient is settled into the ICU and his secondary survey does not reveal any other significant injuries. The initial ICP is 32 mmHg after the EVD is connected.

1. What is your initial plan of management for the intracranial pressure?

The rest of the viva focussed on the management of refractory ICP including a discussion of the DECRA trial results.

Pass rate: 83%
Highest mark: 80%

Viva 2

An 18-year-old male has been involved in a high-speed motor vehicle accident. His initial GCS at the scene was 5 (E2,V2,M1) and he was intubated without any drugs. He has been a primary retrieval from the crash scene to your hospital.

On arrival he is intubated and has a hard collar in place. There is poor chest wall movement on the left side. He has a dilated left pupil and is noted to be bleeding from the left ear and nose. He has an obviously fractured left femur and shortened and externally rotated left leg.

Vital signs:
- Heart rate 130/min sinus rhythm
- BP 85/50 mmHg
- SpO₂ 90% (FiO₂ 1.0) on Oxylog transport ventilator
- Temp 34.0°C

1. How would you initially manage this patient?

The rest of the viva focussed on the immediate and the subsequent management of the multi-trauma patient together with a discussion of cervical spine clearance as well as coagulopathy.

Pass rate: 79%
Highest mark: 85%
Viva 3

A 21-year-old with gunshot injuries to his head and abdomen has been in your ICU for three weeks. He has a GCS of 10 with a tracheostomy and is being weaned off mechanical ventilation. He develops a new onset fever of 39.0°C on day 22 in ICU. He has a sinus tachycardia at 120/min and has a BP of 100/70. A CT abdomen was performed and a representative cut of it is as seen.

1. Please describe the prominent findings on the CT image

The CT showed a pancreatitis with a possible abscess. The viva was a discussion on the management of patient with acute pancreatitis.

Pass rate: 69%
Highest mark: 70%

Viva 4

A 35-year-old woman, gravida 2, para 1, who is 30 weeks pregnant, has been admitted to your ICU with pre-eclampsia. Her blood pressure is 160/100 mmHg treated with hydralazine and metoprolol prior to her admission. IV vasodilators are being considered. She has moderate proteinuria, normal liver function and a platelet count of 120 \( \times \) 10^9. There is no evidence of foetal distress. Her significant past history included a Factor V Leiden mutation and a history of proximal vein thrombosis during her first pregnancy.

1. What pharmacological regime would you recommend for DVT prophylaxis and why?

The rest of the viva was a discussion on thromboprophylaxis in pregnancy including the results of the recent PROTECT study.

Pass rate: 69%
Highest mark: 79%
You are asked to admit a 48-year-old lady who received ablative chemotherapy and an allogeneic bone marrow transplant two weeks ago for acute myeloid leukaemia. She has become progressively more dyspnoeic in the ward. A chest XRay demonstrates a diffuse pulmonary infiltrate.

Initial observations:
GCS 14
Temp 38.4°C
Pulse rate 140/min
BP 90/40 mmHg
Resp rate 35/min
SpO₂ 88% on 10 L/min O₂

The full blood count report from yesterday is at the bedside.
Hb 68 G/L (135 – 180)
WCC 0.2 x 10⁹/L (4.0 – 11.0) No differential
Platelets 39 x 10⁹/L (150 – 400)

Comment – occasional tear drops, occasional elliptocytes, occasional lymphocyte and neutrophil seen
1. What is your differential diagnosis?

The viva focused on the management of neutropenic sepsis.

**Pass rate:** 55%
**Highest mark:** 70%

**Viva 6**

Radiology Station (4 chest X-rays, and 1 each of a CT head, chest and abdomen were shown)

**Pass rate:** 86%
**Highest mark:** 86%

**Viva 7 Communication Station**

Mr Soul is an 82-year-old man who, despite severe dementia, has been living at home with his elderly wife. He is fully dependent for activities of daily living and was assessed last year as requiring high-level nursing care. He was admitted to your hospital 2 days ago with drowsiness. A CT scan of his head shows a large acute subdural haematoma. The neurosurgeon has said she is prepared to operate if the family so wish.

Last night a MET call was instituted because the nurse was worried about his breathing. The night Registrar assessed his GCS as 8, admitted him to ICU and intubated him before discussing the case with you.

You have now completed your morning ward round and have been asked to meet with his son/daughter Nick. The bedside nurse tells you he/she has medical power of attorney and is upset and unsure how to proceed.

**Pass rate:** 52%
**Highest mark:** 100%
You have just reviewed a 40-year-old man, who was in a motor vehicle accident, in the Intensive Care Unit. He has been admitted for optimization of analgesia and non-invasive ventilation for several rib fractures.

You are about to enter a relatives' room to speak to his wife, Jennifer. Jennifer is alone in this room and has just finished speaking with one of the nursing staff. The nurse has left the room and told you that Jennifer, who is the third trimester of a normal pregnancy, was not in the car at the time of the accident.

When you enter the room, you find Jennifer lying on the floor.

The viva focused on practical and theoretical aspects of basic and advanced life support in the pregnant patient.

Pass rate: 28%
Highest mark: 90%