SECOND PART EXAMINATION

EXAM REPORT AUGUST / OCTOBER 2017

This report is prepared to provide candidates, tutors and Supervisors of Training with information regarding the assessment of candidates’ performance in the CICM Second Part Examination. Answers provided are not necessarily model answers but a guide as to what was expected and for use as an educational resource. Trainees should discuss the report with their tutors so that they may prepare appropriately for future examinations. Trainees should not rely solely on writing practice answers to previous exam questions for exam preparation, and first establish a strong knowledge base from learning at the bedside and studying relevant texts, journals and on-line sources.

The exam comprises a written section and an oral section. The written exam consists of two 2.5hr papers of 15 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 five previous exams is provided.

In all sections of the exam the candidate has to demonstrate performance consistent with that of a junior consultant, i.e. demonstrate he/she has the ability for safe, effective, independent practice as an Intensivist. Candidates who are not at this level are encouraged to defer their attempt at the exam.

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<tbody>
<tr>
<td>Presenting for written (Including OTS)</td>
<td>49</td>
<td>40</td>
<td>49</td>
<td>41</td>
<td>52</td>
<td>35</td>
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<td>Carrying a pass from a previous attempt</td>
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<td>9</td>
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<td>21</td>
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<td>OTS Exempt</td>
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<td>0</td>
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<tr>
<td>Total number presenting (written + carry + OTS)</td>
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<td>49</td>
<td>63</td>
<td>55</td>
<td>64</td>
<td>56</td>
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<tr>
<td>Invited to orals (&gt;50% in written section)</td>
<td>39</td>
<td>24</td>
<td>34</td>
<td>27</td>
<td>35</td>
<td>27</td>
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<tr>
<td>Total number invited to oral section</td>
<td>47</td>
<td>33</td>
<td>48</td>
<td>41</td>
<td>47</td>
<td>48</td>
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</table>
### Analysis of Performance in Individual Sections

<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td><strong>Successful in the written section</strong></td>
<td>39/49 80%</td>
<td>24/40 60%</td>
<td>34/49 69%</td>
<td>27/41 66%</td>
<td>35/52 67%</td>
<td>27/35 77%</td>
</tr>
<tr>
<td><strong>Successful in the Hot Case section</strong></td>
<td>33/47 70%</td>
<td>15/33 45%</td>
<td>33/48 69%</td>
<td>18/41 44%</td>
<td>26/47 55%</td>
<td>32/48 67%</td>
</tr>
<tr>
<td><strong>Successful in both Hot Cases</strong></td>
<td>18/47 38%</td>
<td>11/33 33%</td>
<td>24/48 50%</td>
<td>7/41 17%</td>
<td>13/47 28%</td>
<td>17/48 35%</td>
</tr>
<tr>
<td><strong>Successful in the Viva section</strong></td>
<td>36/47 77%</td>
<td>24/33 73%</td>
<td>38/48 79%</td>
<td>18/41 44%</td>
<td>31/47 66%</td>
<td>40/48 83%</td>
</tr>
</tbody>
</table>

### Sectional Pass Rates

<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td><strong>Pass rate</strong></td>
<td><em>Highest individual mark</em></td>
<td><em>Pass rate</em></td>
<td><em>Highest individual mark</em></td>
<td><em>Pass rate</em></td>
<td><em>Highest individual mark</em></td>
<td><em>Pass rate</em></td>
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<tr>
<td>Hot Case 1</td>
<td>60% 100%</td>
<td>42% 90%</td>
<td>65% 93%</td>
<td>37% 80%</td>
<td>45% 80%</td>
<td>60% 80%</td>
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<tr>
<td>Hot Case 2</td>
<td>62% 98%</td>
<td>55% 95%</td>
<td>65% 90%</td>
<td>46% 90%</td>
<td>62% 85%</td>
<td>56% 88%</td>
</tr>
<tr>
<td>Viva 1</td>
<td>64% 90%</td>
<td>73% 85%</td>
<td>65% 88%</td>
<td>71% 92%</td>
<td>53% 93%</td>
<td>83% 90%</td>
</tr>
<tr>
<td>Viva 2</td>
<td>30% 68%</td>
<td>73% 90%</td>
<td>67% 85%</td>
<td>32% 70%</td>
<td>45% 88%</td>
<td>96% 95%</td>
</tr>
<tr>
<td>Viva 3</td>
<td>51% 83%</td>
<td>55% 71%</td>
<td>77% 95%</td>
<td>66% 90%</td>
<td>77% 85%</td>
<td>79% 100%</td>
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<td>Viva 4</td>
<td>62% 83%</td>
<td>73% 93%</td>
<td>46% 90%</td>
<td>51% 80%</td>
<td>79% 78%</td>
<td>52% 88%</td>
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<td>Viva 5</td>
<td>79% 100%</td>
<td>70% 77%</td>
<td>44% 95%</td>
<td>76% 85%</td>
<td>66% 85%</td>
<td>92% 90%</td>
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<td>Procedure Viva</td>
<td>45% 78%</td>
<td>73% 90%</td>
<td>79% 100%</td>
<td>66% 85%</td>
<td>40% 90%</td>
<td>46% 81%</td>
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<tr>
<td>Radiology Viva</td>
<td>66% 95%</td>
<td>73% 94%</td>
<td>100% 92%</td>
<td>41% 89%</td>
<td>40% 95%</td>
<td>84% 90%</td>
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<tr>
<td>Communication Viva</td>
<td>91% 100%</td>
<td>52% 95%</td>
<td>60% 95%</td>
<td>10% 85%</td>
<td>47% 78%</td>
<td>65% 100%</td>
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### Oral Section Pass Rates

<table>
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<tr>
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<tbody>
<tr>
<td>Candidates who scored &gt;50% in written section and passed the overall exam</td>
<td>30/39</td>
<td>17/24</td>
<td>25/34</td>
<td>15/27</td>
<td>27/35</td>
<td>19/27</td>
</tr>
<tr>
<td></td>
<td>77%</td>
<td>71%</td>
<td>74%</td>
<td>56%</td>
<td>77%</td>
<td>70%</td>
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<tr>
<td>All candidates invited to oral section and passed the overall exam (written + carry + OTS)</td>
<td>37/47</td>
<td>21/33</td>
<td>39/48</td>
<td>18/41</td>
<td>32/47</td>
<td>37/48</td>
</tr>
<tr>
<td></td>
<td>79%</td>
<td>64%</td>
<td>81%</td>
<td>44%</td>
<td>68%</td>
<td>77%</td>
</tr>
<tr>
<td>Overall Pass Rate</td>
<td>37/57</td>
<td>21/49</td>
<td>39/63</td>
<td>18/55</td>
<td>32/64</td>
<td>37/56</td>
</tr>
<tr>
<td></td>
<td>65%</td>
<td>43%</td>
<td>62%</td>
<td>33%</td>
<td>50%</td>
<td>66%</td>
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### EXAMINERS’ COMMENTS

**Written Paper**

A high percentage of candidates passed the written section in this sitting. Only five of the thirty questions had an overall pass rate of less than 50%.

As in previous exams, candidates who failed questions did so for one or more of the following reasons:

- Insufficient knowledge of the topic in question
- Insufficient detail and/or depth of the answer
- Poorly structured answer
- Inadequate reference to supportive evidence where relevant
- Failure to answer the question as asked
- Omission of all or part of the question

Candidates that failed questions most often gave insufficiently detailed answers that were not at the level expected of a junior consultant. Candidates often gave generic “proforma” answers that did not deal with the specific issues in the question.

Candidates are advised to read the questions carefully and thoroughly and ensure they answer the question as asked and address all parts of each question. **Candidates are reminded to make sure their writing is legible and to avoid using non-standard abbreviations.** Candidates are also reminded that professional conduct is assessed throughout the exam process and that inappropriate comments written on the answer paper are not acceptable.

Candidates who failed the written section passed an average of 12/30 questions compared with candidates scoring >50% and gaining an invitation to the oral section, passing an average of 22/30 questions.
SECOND PART WRITTEN EXAMINATION

(A) Write your answers in the blue book 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 (E.g. Table form)

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

Discuss: Explain the underlying key principles. Where appropriate, this may include controversies and/or pros and cons

NOTE

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

Please note that in this report all images from the SAQs have been removed.

Question 1

You have received a phone call from a junior colleague at a remote location. A previously well 32-year-old male has presented with nausea and hypotension following a confirmed bite on his leg from a brown snake. A retrieval team will arrive in approximately three hours; until then your colleague is the only medical officer available.

a) Outline the telephone advice you would give them. Include guidance on what complications they might expect to arise and how to manage them. (80% marks)

Several days after arrival in your Intensive Care Unit (ICU) the patient develops oliguric renal failure.

b) List the possible causes. (20% marks)
ANSWER TEMPLATE

a)
- Ensure patient is in an appropriate monitored area
- Give face mask oxygen, obtain iv access. Fluid resuscitation if hypotensive.
- Apply pressure bandage over the bite site and aim to cover entire leg. Splint limb and keep immobile.
- Patient has features of systemic envenomation and should therefore receive appropriate antivenom, one vial is adequate dose. No requirement for premedication with adrenaline or steroids.
- Ideally take baseline blood tests, including coagulation studies U&E, FBE, CK, LFTs.
- Given the circumstances it would be reasonable to either release the pressure bandage after antivenom administration or keep it in place until the patient has been retrieved (Note to examiners – some mention of what to do with the PB expected, although either option acceptable)
- Discussion with National Poisons Information Centre

Complications include:
- Anaphylaxis to antivenom – manage by stopping infusion, airway management as indicated and fluid resuscitation. May require adrenaline – use with caution due to concern of raised blood pressure and potential coagulopathy.
- Coagulopathy: - likely very high INR, undetectable fibrinogen
- If no active bleeding does not require specific management other than antivenom. If severe or life-threatening bleeding, reasonable to give FFP after antivenom.
- May develop severe hypotension or cardiac arrest. Manage according to basic ALS principles
- Neurotoxicity and cardiotoxicity rare and mild with brown snake envenomation

b)
- Potential causes of renal failure.
- Thrombotic microangiopathy secondary to consumptive coagulopathy
- Rhabdomyolysis
- ATN secondary to prolonged hypotension/arrest.
- Secondary sepsis
- Transfusion mismatch

Examiners Comments:

Many candidates ignored the setting of a remote location completely, and gave a management plan that was applicable to a tertiary centre (e.g., TEG and ROTEM; “intubate” without reference to the skill of the junior doctor, etc.).

Some candidates appeared unaware of even the most basic aspects of snake bite management e.g., pressure immobilization, VDK, monovalent versus polyvalent etc.

Many candidates used an ABCDE template which prioritized airway and breathing above the first-aid of snake bite; also, it resulted in not covering the coagulopathy aspects well enough.

The answer for the renal failure again seemed templated (pre-renal, renal, post-renal) and lacked context - there were very few references to the snake bite and antivenom as possible causes of renal failure.

<table>
<thead>
<tr>
<th>Maximum Score</th>
<th>8.4</th>
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<tr>
<td>Percentage Passed</td>
<td>36.7%</td>
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Question 2

A 37-year-old male has been admitted to your ICU following an explosion in his garage. He has suffered a mixture of partial and deep burns estimated at 35% total body surface area, and has been intubated in the Emergency Department. After one hour of resuscitation in your unit he remains hypotensive with a blood pressure of 80/50 mmHg.

List the potential causes and outline how you would diagnose and manage them.

ANSWER TEMPLATE

1. Spurious
   a. Damped or poorly functioning, zeroed, arterial line
   b. Inappropriate sized cuff
      i. Check line, cuff size
      ii. Measure second site, alternative modality

2. Hypovolemia
   a. Review volumes of administered fluids to date
   b. Confirm size and depth of burn
   c. Check calculations for fluid resuscitation are correct
   d. Rising haematocrit, ECHO findings
      i. Increase fluid resuscitation rate

3. Bleeding from occult/missed injury
   a. Review/repeat trauma imaging
      i. Blood product resuscitation, correction of coagulopathy
      ii. Operative/Interventional radiology interventions to treat cause

4. Sepsis
   a. Too early for burn sepsis – possible intraabdominal or thoracic blast injury
      i. Broad spectrum antibiotics and source control

5. Distributive
   a. High cervical spine injury
      i. Review imaging, vasopressors
   b. Anaphylaxis to drugs
      i. Review history, examine for rash/bronchospasm, adrenaline
   c. Cyanide toxicity
      i. Mixed venous oxygen, empirical antidote administration

6. Cardiogenic
   a. Takustubo, underlying cardiac disease, blast injury, myocardial toxins
      i. ECHO, ECG, Inotropic support

7. Obstructive
   a. Tension pneumothorax
      i. CXR, drainage
   b. Abdominal compartment syndrome
      i. Bladder pressure, escharotomies, laparotomy/laparostomy
   c. Tamponade
      i. Echo and pericardiocentesis
Examiners comments:

Most candidates were not able to amalgamate the three crucial aspects of this patient i.e., trauma in a burns patient in the setting of a closed area explosion.

Many focused solely on the burns with little reference to the trauma.

Many used a generic ABCD template without applying it to the patient.

Many answer structures were haphazard with an initial list of the causes followed by the management, with the result that the management for a number of the differentials were missed.

The best answers used a table or bulleted list approach taking about causes as well as management.

<table>
<thead>
<tr>
<th>Maximum Score</th>
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<td>Percentage Passed</td>
<td>44.9%</td>
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Question 3

A 76-year-old male returns to the ICU following a right sided thoracotomy and right upper lobectomy. He is extubated but has a large air leak from the intercostal catheter.

a) Describe your assessment and specific management of the air leak. (50% marks)

b) The patient desaturates and requires re-intubation. Describe your management of his ventilation. (50% marks)

ANSWER TEMPLATE

a) Describe your assessment and specific management of the air-leak.

- History: discuss with the surgeon and anaesthetist the intraoperative course and any air-leak at the end of the operation
- Assess his respiratory state-respiratory rate and pattern, saturations and oxygen. Support this as indicated but with awareness that re-intubation and PPV will likely worsen the air-leak
- Examine the patient-are the drains connected correctly, are they on suction, what is the suction set at, is the suction entraining air through the chest wall?

Investigations: ABG and urgent CXR

Management:

- Assess the degree of right lung expansion on the postoperative CXR
- Consider removing or reducing the suction on the drain if lung expanded
- If lung not expanded check icc patency and insert another icc if needed
- Discuss with the surgeon any operative interventions

b) The patient desaturates and requires re-intubation. Describe your ventilatory management.

- High ventilation pressures will worsen any air leak so low to no PEEP, low peak airway pressures and toleration of hypercarbia
- Spontaneous patient triggered ventilator modes with pressure support may reduce the air-leak compared with mandatory modes
- Consider:
- Potential need for lung isolation/bronchial blocker
- Oscillation
- ECMO

Examiners comments:

Many candidates answered part A as if the patient was already intubated, having not read the stem carefully. Very few referred to suction on the drain, or principles in managing broncho pleural fistula. There was little emphasis on additional drains and importance of try to reinflate lung.

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<td>Percentage Passed</td>
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Question 4

4.1

The following results are from a 35-year-old female with fever, shortness of breath and known renal calculi.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patient Value</th>
<th>Adult Normal Range</th>
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</thead>
<tbody>
<tr>
<td>FiO₂</td>
<td>0.21</td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.30*</td>
<td>7.35 – 7.45</td>
</tr>
<tr>
<td>pCO₂</td>
<td>25.0 mmHg (3.3 kPa)*</td>
<td>35.0 – 45.0 (4.6 – 6.0)</td>
</tr>
<tr>
<td>pO₂</td>
<td>117 mmHg (15.6 kPa)</td>
<td></td>
</tr>
<tr>
<td>SpO₂</td>
<td>98%</td>
<td></td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>12.0 mmol/L*</td>
<td>22.0 – 26.0</td>
</tr>
<tr>
<td>Base Excess</td>
<td>-15.0 mmol/L*</td>
<td>-2.0 – +2.0</td>
</tr>
<tr>
<td>Lactate</td>
<td>1.7 mmol/L*</td>
<td>0.5 – 1.6</td>
</tr>
<tr>
<td>Sodium</td>
<td>135 mmol/L</td>
<td>135 – 145</td>
</tr>
<tr>
<td>Potassium</td>
<td>4.1 mmol</td>
<td>3.5 – 5.0</td>
</tr>
<tr>
<td>Chloride</td>
<td>105 mmol/L</td>
<td>95 – 105</td>
</tr>
<tr>
<td>Glucose</td>
<td>5.8 mmol/L</td>
<td>3.5 – 6.0</td>
</tr>
<tr>
<td>Creatinine</td>
<td>324 μmol/L*</td>
<td>45 – 90</td>
</tr>
<tr>
<td>Urea</td>
<td>29.0 mmol/L*</td>
<td>3.0 – 8.0</td>
</tr>
<tr>
<td>Albumin</td>
<td>42 g/L</td>
<td>35 – 50</td>
</tr>
</tbody>
</table>

a) Describe the acid base abnormalities. (30% marks)

b) Suggest one likely aetiology. (20% marks)

**ANSWER TEMPLATE**

a) Describe the acid base abnormalities.

  Metabolic acidosis
  Anion gap elevated (18)
  Delta ratio 0.5 – so coexisting normal AG acidosis

b) Suggest one likely aetiology.

Renal tubular acidosis (type 1) secondary to renal stones for NAGMA and urosepsis for HAGMA
Guidance – any plausible answer that addresses all the acid-base abnormalities

4.2

A 69-year-old male has been intubated and ventilated in the Emergency Department for worsening respiratory distress and abdominal pain. He was diagnosed with oesophageal cancer 3 months ago and has received chemotherapy followed by an oesophageal stent. He has non-insulin dependent diabetes.

The following blood results were obtained:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patient Value</th>
<th>Adult Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>FiO₂</td>
<td>0.5</td>
<td>7.35 – 7.45</td>
</tr>
<tr>
<td>pH</td>
<td>7.13*</td>
<td>7.35 – 7.45</td>
</tr>
<tr>
<td>pO₂</td>
<td>253 mmHg (33 kPa)</td>
<td>35.0 – 45.0 (4.6 – 6.0)</td>
</tr>
<tr>
<td>pCO₂</td>
<td>25.0 mmHg (3.3 kPa)*</td>
<td>35.0 – 45.0 (4.6 – 6.0)</td>
</tr>
<tr>
<td>SpO₂</td>
<td>99%</td>
<td></td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>8.0 mmol/L*</td>
<td>22.0 – 26.0</td>
</tr>
<tr>
<td>Base Excess</td>
<td>-19.0 mmol/L*</td>
<td>-2.0 – +2.0</td>
</tr>
<tr>
<td>Lactate</td>
<td>10.0 mmol/L*</td>
<td>0.5 – 1.6</td>
</tr>
<tr>
<td>Sodium</td>
<td>136 mmol/L</td>
<td>135 – 145</td>
</tr>
<tr>
<td>Potassium</td>
<td>4.6 mmol/L</td>
<td>3.5 – 5.0</td>
</tr>
<tr>
<td>Chloride</td>
<td>103 mmol/L</td>
<td>95 – 105</td>
</tr>
<tr>
<td>Glucose</td>
<td>15.5 mmol/L*</td>
<td>3.5 – 6.0</td>
</tr>
<tr>
<td>Urea</td>
<td>54.0 mmol/L*</td>
<td>3.0 – 8.0</td>
</tr>
<tr>
<td>Creatinine</td>
<td>644 μmol/L*</td>
<td>45 – 90</td>
</tr>
<tr>
<td>Albumin</td>
<td>20 g/L*</td>
<td>35 – 50</td>
</tr>
<tr>
<td>Ionised calcium</td>
<td>1.15 mmol/L</td>
<td>1.10 – 1.35</td>
</tr>
</tbody>
</table>

Interpret the data provided and give likely causes for the abnormalities in this patient. (50% marks)

**ANSWER TEMPLATE**

Interpret the data provided and give likely causes for the abnormalities in this patient

Increased Aa gradient – aspiration, ARDS, fluid overload – any plausible cause
High anion gap metabolic acidosis, elevated lactate – sepsis in immunosuppressed individual, consider oesophageal perforation, cardiac failure, metformin toxicity
Respiratory acidosis – primary lung pathology, inadequate ventilator settings for degree of acidosis
Delta ratio 1.1 (taking into account albumin)
Renal impairment- sepsis, dehydration,
Hyperglycaemia, low albumin – diabetes, stress response, malnutrition.

**Guidance to examiners: answers which are more specific to the known patient problems score more – e.g. oesophageal perforation, metformin toxicity**

<table>
<thead>
<tr>
<th>Maximum Score</th>
<th>9.5</th>
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<tbody>
<tr>
<td>Percentage Passed</td>
<td>81.6%</td>
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</tbody>
</table>
Question 5

With respect to the use of hypertonic saline (HTS) in the critically ill, list the indications, mechanisms of action and outline the supporting evidence as well as the potential adverse effects.

ANSWER TEMPLATE

The main indications for the use of hypertonic saline in the critically ill are:

- Osmotherapy to manage intracranial hypertension
- Correction of (symptomatic) hyponatraemia.
- Fluid resuscitation in hypovolaemic shock (uncommon)
- Burns resuscitation
- Has been used in tricyclic poisoning
- Mucolytic in nebulized form – e.g. for cystic fibrosis, induced sputum sample, used in bronchiolitis with positive trial evidence

The range of concentrations of HTS used clinically varies from 1.8 – 30%.

Needs to be given via central venous access

Mechanisms of action
- Marked osmotic shift of fluid from the intracellular to the interstitial and intravascular space.
- Reverses the increase in endothelial cell volume in shock and ischaemia, limiting capillary leak.
- Plasma viscosity is reduced by increased water content improving blood flow.
- Hypertonicity has a direct relaxant effect on vascular smooth muscle. End result is increased capillary blood flow. This may help counteract vasospasm in SAH.
- HTS induced increase in intravascular volume leads to an autoregulatory reduction in intracerebral blood volume.
- Increased cardiac output – increased preload, reduced PVR and SVR and reduced myocyte oedema.
- Immuno-modulatory effects and reduction of intestinal apoptosis in haemorrhagic shock

Potential adverse effects

- Hypernatraemia
- Acute hyperosmolar state
  - Osmotic demyelination syndrome
  - Acute heart failure
  - Pulmonary oedema

- Hyperchloraemic acidosis
- Hyperosmolar renal failure
- Dilutional coagulopathy

Theoretical risks
- Increased rate of blood loss secondary to rapid volume expansion
- Reverse osmosis phenomenon in disrupted blood brain barrier with worsening cerebral oedema
- Severely dehydrated risk of worsening cellular dehydration
- Acute cerebral dehydration potentially result in shearing on bridging vessels and SAH

Most of these risks are theoretical or can be avoided by careful use in patients with hyponatraemia and monitoring
Evidence supporting use of HTS

Fluid resuscitation
Studies evaluating HTS in resuscitation in various shock states have shown benefit in outcomes including blood pressure, fluid balance and mortality. Comparison is difficult as different concentrations of HTS used, different case mix and other methodological issues. Concerns about potential of HTS to increase bleeding have not been proven. Overall HTS seems effective in increasing blood pressure in haemorrhagic shock. Its use in other forms of shock is not so well supported.

Osmotherapy to control ICH
Studies have evaluated HTS in traumatic brain injury and subarachnoid haemorrhage and used as either 7.5% boluses or 3% continuous infusions and HTS appears to be effective in reducing ICP. No evidence to suggest a better neurological outcome or survival benefit.

Summary of clinical use

*Note: This template was long and complex and candidates were not expected to cover all the points in order to pass.*

<table>
<thead>
<tr>
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<th>6.7</th>
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<tbody>
<tr>
<td>Percentage Passed</td>
<td>32.7%</td>
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</tbody>
</table>

**Question 6**

With respect to a patient presenting with clinical features suggestive of tetanus:

a) List six other potential differential diagnoses other than tetanus that you would consider. (30% marks)

b) How would you confirm the diagnosis of tetanus? (30% marks)

c) Excluding general supportive measures (e.g. airway management), describe the specific management of tetanus. (40% marks)

**ANSWER TEMPLATE**

a) The differential includes:
- Strychnine poisoning
- Drug induced dystonia
- Dental/local infections
- Stiff person syndrome
- Hypocalcaemia
- Malignant Hyperthermia
- Stimulant Use
- Serotonin syndrome
- Seizure disorder
- Psychiatric disorders

b) The diagnosis is a clinical one.
- Appropriate History
- Vaccinations status
• Tetanus prone wound
• Appropriate clinical features
• C. tetani is cultured from the wound in only 1/3 of cases. There are no specific lab tests to confirm the diagnosis

c) Passive immunisation.
Human antitetanus immunoglobulin (HIG) has now largely replaced antitetanus serum (ATS) of horse origin as it is less antigenic. It is recommended that HIG be administered to unimmunised patients or those where their immunisation status is unknown if they present with contaminated wounds.

Eradication of the organism.
• Wound care. The infected site should be cleaned and all necrotic tissue should be debrided.
• Antibiotics. As tetanus spores are destroyed by antibiotics they should be administered.
• Recommendations include Metronidazole, penicillin and erythromycin.

Management of spasms
• Intrathecal Baclofen
• Magnesium
• Diazepam
• Muscle relaxants with mechanical ventilation

Management of autonomic dysfunction
• Sedation
• Alpha and beta blockers

Note: Mention of Human Antitetanus Immunoglobulin was considered essential to score a passing mark

<table>
<thead>
<tr>
<th>Maximum Score</th>
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<tbody>
<tr>
<td>Percentage Passed</td>
<td>59.2%</td>
</tr>
</tbody>
</table>

Question 7

In the setting of haemodynamic collapse secondary to drug overdose, give the pharmacological antidote/s for each of the agents listed below. For each antidote cited, give the rationale/mechanism of action.

a) Digoxin.

b) Tricyclic anti-depressants.

c) Beta blockers.

d) Lignocaine.

ANSWER TEMPLATE

Detail in template more than required for full marks:
<table>
<thead>
<tr>
<th><strong>Digoxin</strong></th>
<th><strong>Digoxin Fab Fragments (Digibind)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Digibind has a much higher affinity (high affinity (10^9)–(10^{10}) L/mol) for digoxin than the (\text{Na}^+/\text{K}^+) ATPase digoxin receptor site</td>
<td></td>
</tr>
<tr>
<td>• Binds to digoxin in the extracellular spaces preventing digoxin binding to the (\text{Na}^+/\text{K}^+) ATPase</td>
<td></td>
</tr>
<tr>
<td>• Creates a concentration gradient that extracts digoxin from the intracellular space</td>
<td></td>
</tr>
<tr>
<td>• Bound digoxin is then renally eliminated with digibind</td>
<td></td>
</tr>
<tr>
<td>• If potential for cardiac arrest due to digoxin – antidote of choice</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>TCA</strong></th>
<th><strong>Sodium bicarbonate</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Alkalising solution – leading to increased pH.</td>
<td></td>
</tr>
<tr>
<td>• Favours the neutral or non-ionised form of TCA making it less available to bind to sodium channels.</td>
<td></td>
</tr>
<tr>
<td>• Cardiac muscle more inotrope responsive</td>
<td></td>
</tr>
<tr>
<td><strong>Sodium load</strong></td>
<td></td>
</tr>
<tr>
<td>• Increased extracellular Na concentration increasing the electrochemical gradient across cardiac cell membranes, potentially attenuating the TCA-induced blockade of rapid sodium channels</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Beta Blockers</strong></th>
<th><strong>Glucagon</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Activates adenylate cyclase in cardiac muscle cells at a site independent from B-adrenergic agents, causing increase in cAMP leading to increased intracellular calcium augmenting contractility.</td>
<td></td>
</tr>
<tr>
<td>• Large doses required and tachyphylaxis occurs</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>High Dose insulin +/- glucose therapy</strong></th>
<th><strong>Several theories of effect:</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Insulin release from B-islet cells is impaired following overdose (especially Ca blocker)</td>
<td></td>
</tr>
<tr>
<td>• Overdose appears to disrupt fatty acid metabolism and create relative insulin resistance in myocardium.</td>
<td></td>
</tr>
<tr>
<td>• State of CHO dependence in stressed myocardium and insulin resistance can be overcome with high dose insulin therapy</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Atropine</strong></th>
<th><strong>Anti-cholinergic agent</strong></th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th><strong>Lignocaine</strong></th>
<th><strong>Lipid emulsion therapy</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Has been used in poisonings involving other lipophilic medications</td>
<td></td>
</tr>
<tr>
<td>• Thought to act as a lipid “sink”: increasing plasma concentration of lipid – shift of lipophilic medications from tissue to plasma.</td>
<td></td>
</tr>
<tr>
<td>• Also providing myocardium with an energy source. Case reports of effect in b blockers and ca channel blockers</td>
<td></td>
</tr>
<tr>
<td>• Used as an adjunct to other therapies.</td>
<td></td>
</tr>
</tbody>
</table>

| **Maximum Score** | **8.7** |
| **Percentage Passed** | **75.5%** |
Question 8

Outline how the pathophysiological changes associated with morbid obesity may impact on the management of critically ill obese patients.

ANSWER TEMPLATE

Anatomical problems:
- Weight
  - Too heavy for certain investigations e.g. CT
  - Difficulty in establishing non-invasive monitoring such as ECG and NIBP
  - Too large for many ICU beds leading to discomfort
  - Difficult to mobilise and move around between beds-chair
  - Difficult to deliver cares due to inability to access areas required, and requiring multiple staff to do so

- Excess adipose tissue
  - Difficult venous and arterial access: establishing, securing, and suitable equipment
    - Increased risk of complications of vascular access
  - Difficult epidural access
  - Difficult to clinically examine
  - Invasive procedures e.g. chest drains more difficult.

- Metabolic and endocrine
  - Altered body composition leading to different catabolism in the critically ill, and different nutritional requirements
  - Altered response to endogenous hormones in the critically ill
  - Altered inflammatory response “Chronic inflammatory state” making interpretation of biomarkers of infection difficult
  - Increased incidence of crystal arthropathy which is often difficult to diagnose because of size
  - Insulin resistance, hyperglycaemia

Respiratory:
- Prone to atelectasis due to abdominal mass
- More difficult to ventilate due to higher airway pressures with obesity acting like a restrictive lung deficit
- Increased incidence of central and peripheral sleep apnea increasing the difficulty of ventilator weaning and extubation

Cardiovascular
- Hypertension making BP responses more variable to sedation and catacholamines, and increasing risk of CVA's
- Coronary artery disease leading to potential episodes of myocardial ischaemia and arrhythmias
- Peripheral vascular disease leading to skins changes and ulceration, with greater propensity for pressure sores and difficulties with skin integrity
- VTE

Airway
- Difficult bag-mask ventilation due to excess facial tissue
- Difficult laryngoscopy
Musculoskeletal

- Greater incidence of arthritis and bony pain
- PRESSSURE AREAS
- Increased incidence of soft tissue infections

Pharmacological

- Pharmaceutics
  - Actual weight different from ideal body weight making drug dose calculation more difficult

- Pharmacokinetics
  - Altered absorption of drugs through the sc / topical route due to altered blood flow
  - Altered distribution of drugs due to altered plasma proteins and fat solubility / distribution
  - Altered metabolism of drugs due to impaired hepatic function
  - Altered excretion of drugs due to impaired renal function

- Pharmacodynamics
  - Larger doses of medications often needed increasing potential for exaggerated side-effects and toxicity

Emotional and environment

- Increase incidence of depression and mood disorders affecting interaction in the critically ill
- Prejudice and stigma of staff towards the difficulty of looking after morbidly obese patients

Maximum Score 7.8
Percentage Passed 77.6%

Question 9

9.1

A 74-year-old female has been admitted to your ICU with urosepsis. She is previously well with no previous hospital admissions. She was commenced on prophylactic subcutaneous heparin on day one of her admission and the following blood results were obtained:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Adult Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin</td>
<td>10.1 g/L*</td>
<td>9.8 g/L*</td>
<td>9.6 g/L*</td>
<td>120.0 – 160.0</td>
</tr>
<tr>
<td>White Cell Count</td>
<td>18.4 x 10⁹/L*</td>
<td>14.2 x 10⁹/L*</td>
<td>10.5 x 10⁹/L</td>
<td>4.0 – 11.0</td>
</tr>
<tr>
<td>Platelet count</td>
<td>120 x 10⁹/L*</td>
<td>101 x 10⁹/L*</td>
<td>88 x 10⁹/L*</td>
<td>150 – 350</td>
</tr>
</tbody>
</table>

On day three, one of your trainees performs a “HITTS screen” which is reported as positive. The patient has remained clinically stable.

Describe your approach to this situation and give a rationale. (70% marks)
ANSWER TEMPLATE

Probability of HITTS is low due to:
Timing of onset is too fast with no history of previous exposure
The platelet fall is not greater than 50%
There is no associated thrombosis or skin necrosis
There is a likely alternative cause – sepsis
The HITTS ELISA test has is not very specific and may give false positives

Therefore:
Reasonable to stop heparin in short term (although not mandatory)
No requirement for commencing alternatives
Could repeat test in short term
More accurate test (SRA- serotonin release test) not likely to be immediately available but will guide future management

9.2

A 45-year-old male was admitted with life threatening shock after being involved in a motor vehicle accident, requiring emergency surgery with large volume blood loss. Post-operatively following return to ICU, he was noted to become hypotensive, febrile and oozy from various drip and operative sites. Red urine was noted.

The following blood results were obtained:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patient Value</th>
<th>Adult Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin</td>
<td>87 g/L*</td>
<td>120 – 160</td>
</tr>
<tr>
<td>White Cell Count</td>
<td>18.9 x 10⁹/L*</td>
<td>4.0 – 11.0</td>
</tr>
<tr>
<td>Platelet count</td>
<td>132 x 10⁹/L*</td>
<td>150 – 350</td>
</tr>
<tr>
<td>Sodium</td>
<td>138 mmol/L</td>
<td>135 – 145</td>
</tr>
<tr>
<td>Potassium</td>
<td>4.3 mmol/L</td>
<td>3.5 – 5.0</td>
</tr>
<tr>
<td>Chloride</td>
<td>102 mmol/L</td>
<td>95 – 105</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>20.0 mmol/L*</td>
<td>22.0 – 26.0</td>
</tr>
<tr>
<td>Glucose</td>
<td>5.3 mmol/L</td>
<td>3.5 – 6.0</td>
</tr>
<tr>
<td>Urea</td>
<td>15.2 mmol/L*</td>
<td>3.0 – 8.0</td>
</tr>
<tr>
<td>Creatinine</td>
<td>80 µmol/L</td>
<td>45 – 90</td>
</tr>
<tr>
<td>Creatinine Kinase</td>
<td>2000 U/L*</td>
<td>55 – 170</td>
</tr>
<tr>
<td>Urine Myoglobin:</td>
<td>trace</td>
<td></td>
</tr>
<tr>
<td>Urine Haemoglobin:</td>
<td>++</td>
<td></td>
</tr>
</tbody>
</table>

Based on his clinical history and the lab report, what is the likely cause of his post-operative deterioration? How will you confirm your diagnosis? (30% marks)
**ANSWER TEMPLATE**

Mismatched transfusion.
Check patient’s and donor groups and re check cross match.

<table>
<thead>
<tr>
<th>Maximum Score</th>
<th>9.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percentage Passed</td>
<td>59.2%</td>
</tr>
</tbody>
</table>

**Question 10**

You have been asked to review a 53-year-old female with known alcoholic liver disease. She has had a progressive fall in her conscious level over the last 24 hours and the medical team are concerned she is developing hepatic encephalopathy (HE).

a) List four alternative diagnoses to HE that you would consider in this circumstance. (10% marks)

b) List six clinical signs that would be suggestive of HE. (30% marks)

c) Discuss the specific management of severe HE in this setting. (60% marks)

**ANSWER TEMPLATE**

a.
- Drug or alcohol effects
- Seizure disorder
- Traumatic injury
- Septic encephalopathy
- Hypoglycaemia and other electrolyte disorders
- Uraemia

b.
- Asterixis
- Hypertonia
- Hyperreflexia
- Clonus
- Hippus
- Bradykinesia
- Nystagmus

c.
- Basic principles of management include:

**Excluding other causes of altered mentation**

HE is a diagnosis of exclusion (part [a] of question 10). Serum ammonia levels should not be used as a diagnostic tool or to monitor response to treatment.

**Identifying and treating precipitating cause**
- Increased nitrogen load (GI bleed, excess protein intake, infection)
- Decreased toxin clearance (hypovolaemia, renal failure, constipation, port-systemic shunt, medication non-compliance, acute on chronic liver failure)
- Altered neurotransmission (sedating medications, alcohol, hypoxia, hypoglycaemia)
Reducing nitrogen load in the gut/Ammonia formation

First Line-

- Non-absorbable disaccharidases- Lactulose is metabolized by bacteria in the colon to acetic and lactic acid, which reduces colonic pH, decreases survival of urease producing bacteria in the gut, and facilitates conversion of ammonia (NH3) to ammonium (NH4+), which is less readily absorbed by the gut. The cathartic effect of these agents also increases faecal nitrogen waste.
- Antibiotics- Rifaximin is a minimally absorbed oral antibiotic with broad spectrum activity against gram-positive and gram-negative aerobic and anaerobic bacteria. Oral neomycin and metronidazole have been used to treat hepatic encephalopathy in the past but due to concerns of toxicity and side effects, rifaximin is now the preferred antibiotic.

Second line- (all not required to score full marks)

- Probiotics- As gut bacteria play a central role in producing ammonia it has been theorized that altering gut flora using probiotics may be beneficial in HE.
- Polyethylene Glycol- Commonly used, safe and highly effective laxative that has recently been proposed as a possible agent for HE
- Flumazenil- benzodiazepine antagonist at GABA receptors. Can result in clinical improvement but no mortality benefit
- Ammonia scavengers- increase ammonia clearance and reduce systemic concentrations by providing an alternative pathway for renal ammonia clearance.
- L-ornithine l-aspartate (LOLA)- increases glutamine synthase and urea excretion. Similar clinical improvement when compared to lactulose.
- Zinc- Low zinc is associated with impairment of urea cycle enzymes leading to elevated ammonia levels.
- Porta-systemic shunts- Medically refractory HE should raise suspicion of a spontaneous splenorenal shunt and patients who have undergone TIPS should be considered for shunt reversal if severe HE persists

Supportive care

Management of cerebral oedema- Lactulose or rifaximin can be beneficial for the treatment of gradual-onset encephalopathy in patients with prior cirrhosis, but additional, aggressive treatment of brain edema with osmotic diuretics is required in new, fulminant forms to prevent secondary, permanent brain-stem damage and to sustain patients through liver transplantation.

Nutrition- plays a key role in managing HE and preventing recurrence. Optimal daily energy intake should be 35 to 40 kcal/kg ideal body weight with daily protein intake of 1.2 to 1.5 g/kg ideal body weight. Multivitamin should be considered with the addition of specific treatments for clinically apparent vitamin deficiencies.

Assessing the need for long term therapy and liver transplant evaluation

Liver transplant evaluation should be considered in appropriate candidates once a diagnosis of overt hepatic encephalopathy is made

<table>
<thead>
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<th>Maximum Score</th>
<th>7.0</th>
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<tbody>
<tr>
<td>Percentage Passed</td>
<td>26.5%</td>
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</table>
Question 11

11.1

In relation to describing the mode of mechanical ventilation, define the following terms:

a) Flow triggered.

b) Pressure limited.

c) Time cycled. (30% marks)

ANSWER TEMPLATE

a) Flow Triggered:
   • The signal to start inspiration is a change in flow, i.e. a change in flow results in opening of the inspiratory valve

b) Pressure Limited:
   • Pressure is factor that limits the way gas flows into the lung during inspiration. The pressure within the lung cannot exceed the set limit.

c) Time Cycled:
   • Time is the signal that stops inspiration i.e. the inspiratory valve closes and the expiratory valve opens OR Inspiration switches to expiration once the set inspiratory time elapses.

11.2

List four potential causes for auto-triggering during pressure support ventilation. (40% marks)

ANSWER TEMPLATE

• Trigger set too low (too sensitive)
• Cardiac impulse
• ETT leak/circuit leak
• Chest drain
• Condensation in circuit

11.3

The waveform below (Figure 1) is from a ventilator with the following settings:

*Volume control SIMV, PS 10, PEEP 5.*

*(Image removed from report.)*

The bedside nurse informs you that the patient appears to be “struggling against the ventilator”.

a) Given the appearance of the waveform, what is the likeliest cause of the patient’s distress? (10% marks)
The following waveform (Figure 2) is from a ventilator with the following settings:

*Volume control SIMV, PS 10, PEEP 5.*

*(Image removed from report.)*

The bedside nurse informs you that the airway pressures have increased.

b) Given the appearance of the waveform, what is the likeliest cause of the increased airway pressure and how would you treat it? (10% marks)

**ANSWER TEMPLATE**

(i) Patients effort to initiate a breath is not recognised by the ventilator

(ii) Double triggering - Two breaths occur in less than half mean inspiratory time. Occurs when patient demand outlasts set inspiratory time. Treat by increasing tidal volume, increasing inspiratory time, increasing sedation or paralysis

(iii) Flow starvation. Patients inspiratory demand is greater than that delivered by ventilator resulting in scooped out appearance of pressure waveform.

<table>
<thead>
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<th>Maximum Score</th>
<th>8.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percentage Passed</td>
<td>71.4%</td>
</tr>
</tbody>
</table>

**Question 12**

You have been asked to assess a 76-year-old male, scheduled to have a very large incisional hernia repaired electively. He has a history of obstructive sleep apnoea (OSA) requiring nocturnal continuous positive airway pressure (CPAP). A recent echocardiogram, done to evaluate pedal oedema and newly diagnosed elevated creatinine, has revealed an ejection fraction of 55%, a right ventricular systolic pressure (RVSP) of 90 mmHg, and moderate tricuspid regurgitation (TR). He has a history of recurrent deep venous thrombosis (DVT) and pulmonary embolism (PE) due to a deficiency of Factor V Leiden and is currently on Rivaroxaban.

a) List the specific risks associated with his co-morbidities in the perioperative period. (40% marks)

b) Outline strategies that could be used to minimize these risks in the perioperative period. (60% marks)

**ANSWER TEMPLATE**

a) OSA:
   - Acute respiratory failure,
   - Cardiac arrhythmias,
   - Cardiac ischaemia including cardiac arrest
   - Risk of exacerbation by opiates may make analgesic management difficult

Pulm hypertension:
   - Acute right heart failure during perioperative period related to hypoxia and pain, Severe hypoxaemia,
   - Cardiac dysrhythmias,
Renal failure

Renal dysfunction:
- Fluid overload,
- Electrolyte and acid base imbalance,
- Altered drug metabolism

DVTs, PE, Factor V Leiden deficiency:
- High risk of thrombotic events esp PE with exacerbation of RHF.

Anticoagulation:
- Bleeding risk,
- Thrombotic risk if ceased and not adequately covered.

b) Preop:
- Consider need for surgery,
- Consider timing of surgery
  - Surgery should be done when co-morbidities are optimised
  - Organisational factors: Surgery should be conducted when ICU support available, experienced surgical and anaesthetic team available, early on list (so not cancelled), in hospital with appropriate support (ICU and usual physicians)
- May be relevant to discuss goals of therapy with patient, surgical team and pre-existing physicians
- Optimise cardiac status especially with respect to pulmonary hypertension, need for pulmonary vasodilators. Cardiology input would be valuable
- Optimise respiratory status especially with respect to nocturnal NIV; settings, interface
- Plan anticoagulation over perioperative period.
- OK to mention IVC filter if recent PE. But not necessary
- Optimise renal function; may need renal consult and exclusion of reversible causes
- Liase with surgeon, anaesthetist, treating physicians regarding management plan

Intra-operative
- Use of regional techniques / local blocks to reduce requirement for systemic analgesia
- Appropriate monitoring and lines

Post-operative
If intubated:
- Early extubation and early mobilisation to avoid atelectasis if possible
- Consider role of extubation to NIV
- Continue usual nocturnal NIV
- Consider opiate sparing analgesic regimen, consider regional techniques (will need to consider anticoagulation issues)
- Avoid NSAIDS due to renal dysfunction
- Avoid fluid overload
  - Maintain vascular tone with low dose noradrenaline
  - Consider vasopressin if high dose pressor required due to less effect on pulmonary vasculature
- Restart anticoagulation as soon as safe, in keeping with pre-operative plan
- Consider surveillance duplex monitoring if > 24-48hours off anticoagulation
- Avoid nephrotoxic medications

Organise follow-up with usual physicians on discharge to the ward.
Question 13

Critically evaluate the timing of elective tracheostomy in ICU patients.

**ANSWER TEMPLATE**

**Introduction**
Tracheostomy is performed in critically ill adults requiring prolonged invasive ventilation as a strategy to reduce respiratory tract injury, improve patient comfort and/or to facilitate weaning. Timing of tracheostomy has been a subject of debate and may be considered as “early” at <10 days or “late” >10 days although these definitions may vary.

**Rationale**
There has been debate as to whether “early” trache may confer advantages of reduced morbidity and mortality. Disadvantages of tracheostomy include airway trauma, bleeding and death and this may be increased by doing an “early” tracheostomy in patients who may otherwise die or be extubated before 10 days. Early tracheostomy is a consideration in patients with neurological issues (brain injury, GBS, CVA etc.) and shorten time on ventilator.

**Evidence**
Many studies and meta-analyses of variable quality have evaluated this issue. Methodological issues include differences in “early” and “late” timing, prediction of which patients will require “long-term” ventilation, exclusion/inclusion of specific patient groups and diagnosis of end-points such as VAP. Cochrane Review 2012 considered 4 studies (latest 2010) to meet inclusion criteria. Conclusions were that quality of evidence to date was poor and results conflicting. Recent RCT TracMan Study from UK 2013 – tracheostomy at 1-4 days v >10 days invasive ventilation. Early tracheostomy associated with shorter duration of sedation but increased number of procedures and associated complications with no beneficial effect on overall mortality not ICU/hospital LOS. Studies have evaluated patients with respiratory failure and not those intubated for neurological injury.

**Own Practice**
Any reasonable approach acceptable.

**Summary**
Lack of evidence to support early v late trache overall. Selected patients e.g. neurotrauma, GBS, stroke may benefit from early. Probably best decided on case by case basis. Involves invasive procedure with attendant risks and complications and needs appropriate expertise.
Question 14

With respect to gram positive infections in the ICU:

a) What type of infections are commonly caused by Coagulase-negative Staphylococci (CoNS)?
   (10% marks)

b) What are the differences in clinical presentation between infections with CoNS and those with *staphylococcus aureus*?
   (40% marks)

c) What blood culture findings would suggest true bacteraemia rather than contamination with CoNS?
   (40% marks)

d) What empirical antimicrobial therapy is preferred for suspected CoNS infections?
   (10% marks)

**ANSWER TEMPLATE**

a) What infections are commonly caused by Coagulase-negative Staphylococcal Infections?
   • Coagulase-negative Staphylococci (CoNS) commonly cause prosthetic device infections, such as:
     o Prosthetic heart valves,
     o Prosthetic joints
     o Vascular grafts
     o Intra-vascular devices
     o CNS shunts

b) Compared to Staphylococcus Aureus infections, what different clinical presentation is expected from infection with coagulase-negative staphylococci?
   • CoNS are less virulent than staphylococcus aureus, and hence:
     o Signs of localised infection are subtle
     o Rate of disease progression is slow
     o Systemic findings are limited.
     o Fever may be absent
     o Acute phase reactants, may be normal or slightly elevated.
     o Abscess formation commoner with S. Aureus
     o Patients with S Aureus usually sicker

c) What blood culture findings (in addition to clinical suspicion) would suggest true bacteraemia rather than contamination with CoNS?
   • Multiple isolations of the same strain from separate cultures
   • Growth of the strain within 48 hours
   • Bacterial growth in both aerobic and anaerobic bottles.

d) Empirical antimicrobial therapy
   • CoNS are usually resistant to Methicillin; hence, Vancomycin is the preferred empirical therapy.

<table>
<thead>
<tr>
<th>Maximum Score</th>
<th>8.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percentage Passed</td>
<td>49.0%</td>
</tr>
</tbody>
</table>
Question 15

*(Images removed from report.)*

*Please note: The following ECG has been recorded at 25 mm/sec and gain setting of 10 mm/mV.*

A 69-year-old male presents with a fractured neck of femur following a syncopal episode. He is now well and has an ECG (Figure 1 shown on page 14) prior to his surgical procedure.

a) What does the ECG show?  

b) What complication is likely to have led to his fall, and how would you manage it?

The ECG (Figure 2 shown on page 15) is of a haemodialysis patient presenting with pulmonary oedema.

c) What test will you do to confirm the likely underlying diagnosis?

d) What is your immediate management for this condition?

The ECG (Figure 3 shown on page 16) is from a 35-year-old male who presents with paroxysmal tachycardia.

e) What condition is demonstrated? Describe the characteristic features.

f) What would be the possible pharmacological options if his tachycardia were to recur?

**ANSWER TEMPLATE**

a) Tri-fascicular block

b)  
- **Cause** – Complete heart block
- **Management** –
  - Correct electrolyte and endocrine abnormalities (e.g. K+, thyroid function tests)
  - Consider influence of drug therapies such as digoxin, calcium channel antagonists
  - Investigate for ischaemic heart disease
  - Referral to cardiology unit for further evaluation (?permanent pacemaker)

c) Potassium level

d) Counteract cardiotoxic effects of hyperkalaemia
- Calcium chloride
- Sodium bicarbonate
Shift potassium into the cells
- Dextrose and insulin
- Beta agonists
Remove potassium (and water)
- Urgent haemodialysis

e) Wolf-Parkinson-White syndrome
- short PR interval, less than 3 small squares (120 ms)
- slurred upstroke to the QRS indicating pre-excitation (delta wave)
- broad QRS
- secondary ST and T wave changes

f) IV procainamide or amiodarone is preferred, but any class Ia, class Ic, or class III antiarrhythmic can be used

Maximum Score | 10
---|---
Percentage Passed | 98.0%

Question 16
With respect to the management of patients presenting with acute pancreatitis, briefly discuss the following issues:

a) The optimal timing and method of delivery of nutrition. (40% marks)

b) The role of antimicrobials. (40% marks)

c) The role of endoscopic retrograde cholangio-pancreatography (ERCP). (20% marks)

ANSWER TEMPLATE

(a) Method of delivery of nutrition
- Mild pancreatitis – oral diet if tolerated. Commence at admission or within 24 hours.
  - No superiority of enteral over oral in this group (NEJM 2014)
- If unable to tolerate oral intake
  - Enteral preferred to TPN
    - Cochrane 2010 – reduced mortality and other end-points (including infective, MOF)
    - Jejunal not shown to be superior to gastric feeding. Limited evidence (2 small meta-analyses).
      - Gastric feeding succeeds in delivering nutritional targets in 90%
      - No evidence of benefit in delaying feeding awaiting jejunal tube placement – especially in light of apparent benefit of early feeding.
- Commence enteral feeds within 48 hours of admission, TPN >5 days

(b) Use of antimicrobials
- Prophylactic antibiotics not recommended
  - Not indicated for peripancreatic fluid collections or necrosis without clinical (or radiological) evidence of sepsis
  - Number of meta-analyses – no improvement in mortality, rates of infected necrosis
- If clinical suspicion of infected necrosis or peripancreatic collection – FNA with culture (high sensitivity)
- Antibiotics if positive FNA result OR unstable and sepsis suspected while awaiting further investigation
- If used – choose appropriate antibiotic(s) with GP and Gn cover. Consider antifungal agents.
- Treatment of other infective complications – e.g. hospital-acquired pneumonia, line-related, urinary tract.

(c) Role of ERCP
- Not routinely indicated
- May be cause
- Should be performed early (24-48 hrs.) in acute gallstone pancreatitis associated with persistent biliary obstruction or cholangitis
  - May not be tolerated / safe in critically unwell patient – consider percutaneous drainage as alternative

<table>
<thead>
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</table>

**Question 17**

List the strategies available for the control of postpartum haemorrhage and give the advantages and disadvantages of each.

**ANSWER TEMPLATE**

<table>
<thead>
<tr>
<th>Tx</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Physical – vigorous bi-manual massage</strong></td>
<td>Immediate use, no specific equipment.</td>
<td>Only works in uterine atony</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Worsens traumatic injury</td>
</tr>
<tr>
<td><strong>Pharmacological</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oxytocin (first line)</td>
<td>Simple, rapid action</td>
<td>Hypotension and tachycardia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Risk on the CVS unstable pt with no haemorrhage control</td>
</tr>
<tr>
<td>Ergometrine (second line)</td>
<td>Simple, rapid action</td>
<td>Hypertension, N&amp;V</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vasospasm of the arteries in overdose-&gt; gangrene, angina, ischaemia</td>
</tr>
<tr>
<td>Prostaglandin (third line) Misoprostol PR</td>
<td>Simple</td>
<td>B/Constriction, flushing</td>
</tr>
<tr>
<td>Carboprost, IMI or intrauterine</td>
<td></td>
<td>Asthma is Contra Ind</td>
</tr>
<tr>
<td></td>
<td></td>
<td>May inc pulmonary shunting and maternal hypoxia</td>
</tr>
<tr>
<td><strong>Surgical</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Manual removal of placenta/retained products</td>
<td>Removes bleeding cause</td>
<td>Needs GA in theatre</td>
</tr>
<tr>
<td>Surgical repair Soft tissue trauma/artery ligation</td>
<td>Definitive Tx</td>
<td>Needs GA.</td>
</tr>
<tr>
<td>Bakri Balloon +/- BT Cath</td>
<td>Immediate control</td>
<td>Infection risk</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Not definitive Tx</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Can mask ongoing bleeding.</td>
</tr>
<tr>
<td>Hysterecomy</td>
<td>Definitive Tx</td>
<td>fertility</td>
</tr>
<tr>
<td><strong>Radiological</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Selective embolisation of pelvic vessels</td>
<td>May be definitive</td>
<td>Only available in tertiary centres</td>
</tr>
<tr>
<td>Balloon tamponade bilateral femoral arteries as a temporizing measure</td>
<td>Can avoid hysterectomy</td>
<td>Not suitable in catastrophic haemorrhage</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Temporising measure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Risk of ischeamia to pelvic organs</td>
</tr>
<tr>
<td><strong>REBOA</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Maintain normal physiological milieu</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avoid acidosis</td>
<td>Not strictly a</td>
<td>May not be effective alone</td>
</tr>
<tr>
<td>Hypothermia</td>
<td>control option but will not allow normal haemostasis to occur if absent</td>
<td>Correction of coagulopathy may require product transfusion with attendant possible complications</td>
</tr>
<tr>
<td>---------------------</td>
<td>---------------------------------------------------------------------------</td>
<td>-----------------------------------------------------------------------------------------------</td>
</tr>
</tbody>
</table>

*Note: Candidates who referenced the recently published WOMAN trial (Lancet, May 2017) were given credit.*

<table>
<thead>
<tr>
<th>Maximum Score</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Percentage Passed</td>
<td>40.8%</td>
</tr>
</tbody>
</table>

**Question 18**

You are supervising a registrar who suffers a needle stick injury during the insertion of a central line in a patient with a history of intravenous drug use.

Outline your approach to this problem.

**ANSWER TEMPLATE**

**Immediate Response:**
- Stop the procedure
- Ensure patient is safe
- Takeover / delegate patient management as required

**Further response:**
- Wash the registrar’s wound immediately with soap and water
- Express any blood from the wound
- Initiate injury-reporting system used in the workplace
- Patient may need to be consented and then tested for HIV, hepatitis B, Hepatitis C
- Refer registrar to designated treatment facility: Emergency Department / Infectious Disease / Physician / Immunology as per hospital protocol
- With consent, registrar to be tested immediately and confidentially for HIV, hepatitis B and C
- Document the exposure in detail for your own record and for the employer
- If the patient is HIV positive, post exposure prophylaxis needs to be started within two hours of the exposure.
- For possible Hepatitis C exposure, no treatment is recommended but advice must be obtained from Infectious Disease Specialist
- If the source patient tests positive for HIV, hepatitis B, hepatitis C, get post-exposure prophylaxis in accordance with CDC guidelines and as per recommendations from Infectious Disease Specialist or another expert.
- Registrar to have follow up with post exposure testing
- Advise re: taking precautions (including safe sex) to prevent exposing others until follow up testing is complete.
- If exposed to blood borne pathogen, he/she should not donate blood for six months until cleared
- Counselling: While definitive testing is essential, counsel the registrar that the risk factors for infection are: deep injury, visible blood on devices, and needle placement in a vein or artery, lower risk with solid suture needle. Related to procedure: Review of registrar’s technique, equipment used, unit policy for procedural training, assessment of competency, etc.
Question 19

a) With respect to contrast-induced nephropathy (CIN), list six risk factors for its development. 
(30% marks)

b) Outline the strategies that have been used for prevention of CIN. 
(70% marks)

ANSWER TEMPLATE

a) Risk factors for CIN: (any 6)
   1. Age > 75 years
   2. Pre-existing kidney disease (creatinine > 120 umol/L)
   3. Diabetes Mellitus
   4. Congestive Heart Failure
   5. Liver Cirrhosis
   6. Nephrotic Syndrome
   7. Peripheral Vascular Disease
   8. Dehydration or prior diuretic use, especially frusemide
   9. Multiple Myeloma
   10. Use of 1st generation hyperosmolal ionic contrast agents
   11. High dose of IV contrast
   12. Treatment with nephrotoxic agents, such as NSAIDs, aminoglycosides, amphotericin & cyclosporine A

b) • Use nonionic low-osmolal agents/avoid high osmolal agents
   • Use lower doses of contrast and avoid repetitive, closely spaced studies (e.g. <48 hours apart).
   • Avoid volume depletion and NSAIDs
   • For patients at high risk, in the absence of contraindications to volume expansion, intravenous fluids prior to and continued for several hours after contrast administration. While no placebo-controlled studies have proven a benefit of prophylactic intravenous fluid in these risk groups, indirect data support its use.
   • Either isotonic bicarbonate or isotonic saline may be used, preference for isotonic saline since less expensive and no risk of compounding errors. There are no commercially available isotonic sodium bicarbonate solutions available. Rate e.g. 1 mL/kg/hour for 6 to 12 hours preprocedure, intraprocedure, and for 6 to 12 hours post procedure.
   • For at-risk patients, acetylcysteine may be administered the day before and the day of the procedure, based upon its potential for benefit, low toxicity and cost. Although data are conflicting, acetylcysteine may be warranted based on some studies showing a benefit. If acetylcysteine is administered, a preferred dose is 1200 mg orally twice daily rather than 600 mg twice daily the day before and the day of the procedure.
   • (No role for mannitol or other diuretics prophylactically. However, diuretics may be used to treat volume overload if present
   • Prophylactic hemofiltration or hemodialysis after contrast exposure to prevent contrast nephropathy is not recommended
   • Avoidance of contrast, alternative imaging modalities.
Question 20

20.1

A 25-year-old female with a 5-day history of anorexia, nausea and vomiting presents to hospital after a convulsion and is transferred immediately to your ICU. She is G3P2 and 30/40 gestation.

The following blood results are obtained:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patient Value</th>
<th>Adult Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>FiO₂</td>
<td>0.28</td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.54*</td>
<td>7.35 – 7.45</td>
</tr>
<tr>
<td>pO₂</td>
<td>87 mmHg (11.6 kPa)</td>
<td></td>
</tr>
<tr>
<td>pCO₂</td>
<td>33.0 mmHg (4.4 kPa)*</td>
<td>35.0 – 45.0 (4.6 – 6.0)</td>
</tr>
<tr>
<td>SpO₂</td>
<td>94%</td>
<td></td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>28.0 mmol/L*</td>
<td>22.0 – 26.0</td>
</tr>
<tr>
<td>Base Excess</td>
<td>4.5 mmol/L*</td>
<td>-2.0 – +2.0</td>
</tr>
<tr>
<td>Sodium</td>
<td>127 mmol/L*</td>
<td>135 – 145</td>
</tr>
<tr>
<td>Potassium</td>
<td>2.3 mmol/L*</td>
<td>3.5 – 5.0</td>
</tr>
<tr>
<td>Chloride</td>
<td>84 mmol/L*</td>
<td>95 – 105</td>
</tr>
<tr>
<td>Glucose</td>
<td>4.8 mmol/L</td>
<td>3.5 – 6.0</td>
</tr>
<tr>
<td>Creatinine</td>
<td>354 μmol/L*</td>
<td>45 – 90</td>
</tr>
<tr>
<td>Urea</td>
<td>29.0 mmol/L*</td>
<td>3.0 – 8.0</td>
</tr>
<tr>
<td>Haemoglobin</td>
<td>177 g/L*</td>
<td>120 – 160</td>
</tr>
<tr>
<td>White Cell Count</td>
<td>25.4 x 10⁹/L*</td>
<td>4.0 – 11.0</td>
</tr>
<tr>
<td>Platelet count</td>
<td>29 x 10⁹/L*</td>
<td>150 – 350</td>
</tr>
<tr>
<td>Prothrombin time</td>
<td>15.0 sec</td>
<td>12.0 – 16.5</td>
</tr>
<tr>
<td>INR</td>
<td>1.1</td>
<td>0.9 – 1.3</td>
</tr>
<tr>
<td>APTT</td>
<td>28.0 sec</td>
<td>27.0 – 38.5</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>5.7 g/L*</td>
<td>2.0 – 4.0</td>
</tr>
<tr>
<td>D-Dimer</td>
<td>16.8 mg/L*</td>
<td>&lt; 0.5</td>
</tr>
</tbody>
</table>

Describe the important metabolic abnormalities and give one explanation for each. (40% marks)

**ANSWER TEMPLATE**

Describe the important abnormalities and give one explanation for each?

- Raised Aa gradient (aspiration, pneumonia, any plausible)
- Metabolic alkalosis – dehydration, vomiting
- Raised anion gap – sepsis, seizures, renal failure
- Respiratory alkalosis – pain, anxiety, post ictal
- Hypokalaemia, hyponatraemia – dehydration
- AKI – sepsis, TTP, dehydration, eclampsia
- Haemoconcentration – dehydration
- Leucocytosis – sepsis
- Thrombocytopenia – sepsis, TTP, HELLP
- Elevated fibrinogen, D-Dimer – sepsis
A 58-year-old female presents following an intentional overdose. Her arterial blood gases are presented below:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patient Value</th>
<th>Adult Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>FiO₂</td>
<td>0.21</td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.36</td>
<td>7.35 – 7.45</td>
</tr>
<tr>
<td>pCO₂</td>
<td>16.0 mmHg (2.13 kPa)*</td>
<td>35.0 – 45.0 (4.60 – 6.00)</td>
</tr>
<tr>
<td>pO₂</td>
<td>111 mmHg (14.8 kPa)</td>
<td></td>
</tr>
<tr>
<td>SpO₂</td>
<td>97%</td>
<td></td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>9.0 mmol/L*</td>
<td>22.0 – 26.0</td>
</tr>
<tr>
<td>Base Excess</td>
<td>-15.0 mmol/L*</td>
<td>-2.0 – +2.0</td>
</tr>
<tr>
<td>Lactate</td>
<td>25.0 mmol/L*</td>
<td>0.5 – 1.6</td>
</tr>
<tr>
<td>Sodium</td>
<td>150 mmol/L*</td>
<td>135 – 145</td>
</tr>
<tr>
<td>Potassium</td>
<td>4.5 mmol/L</td>
<td>3.5 – 5.0</td>
</tr>
<tr>
<td>Chloride</td>
<td>117 mmol/L*</td>
<td>95 – 105</td>
</tr>
<tr>
<td>Glucose</td>
<td>4.0 mmol/L</td>
<td>3.5 – 6.0</td>
</tr>
</tbody>
</table>

a) Describe the acid base abnormalities. (40% marks)

Her lactate as measured on ABG is 25 mm/L, but the result on a blood sample taken at the same time and measured in the laboratory is only 5 mmol/L.

b) What is the most likely diagnosis? Explain the mechanism of the differences in measured lactates. (20% marks)

**ANSWER TEMPLATE**

a)  
- Metabolic acidosis and respiratory alkalosis
- Elevated anion gap
- Delta AG/Delta HCO₃⁻ 0.8 evidence of non-anion gap acidosis
- Elevated lactate

b)  
- Ethylene glycol toxicity
- Less commonly reported = paracetamol and isoniazid
- Ethylene glycol itself is not involved in lactate production.
- EG metabolites (glycolic and glyoxylic acid) react with the analytical reagent L-lactate oxidase used in lactate electrodes which equip many blood gas analyses due to similar structures with lactic acid. Serum lactate measured using a different technique so “lactate gap” develops

*Specific details of the assays not required.*

Maximum Score 7.8  
Percentage Passed 65.3%
Question 21

After 14 days in ICU with a diagnosis of community-acquired pneumonia, a patient’s signs and symptoms have not improved despite antimicrobial therapy.

a) List the factors that might be responsible for the slow resolution. (60% marks)

b) Outline your assessment to identify the cause of the slow resolution. (40% marks)

ANSWER TEMPLATE

a)
Factors contributing to non-resolution or delayed resolution of pneumonia
1. Host factors:
2. Agent (organism factors)
3. Extent of disease
4. Due to Complication of Pneumonia
5. Incorrect Diagnosis: Diseases mimicking pneumonia

Host factors:
- Age > 60
- H/o Smoking
- Comorbidities: COPD, CCF, DM, CRF, alcoholism
- Malnutrition
- Immunosuppressed host
- Underlying lung disease

Agent or Organism factors
- Resistant organism: especially in patients treated with beta lactams in the recent past, hospitalised in last 3 months, pneumonia in the last 1 year.
- Nosocomial pneumonia: MRSA in a hospitalised patient, with indwelling IV catheters, dialysis patients etc. Pseudomonas aerogenosa infection,
- Unusual pathogen: TB, atypical mycobacterium, nocardia, actinomyceces, Pneumocystis jiroveci,
- Fungal: Aspergillus, Cryptococcus, and Histoplasma etc

Extent of disease
- Bilateral multi-lobar pneumonia
- Associated with bacteraemia

As a result of Complications of pneumonia.
- Empyema
- Abscess
- Metastatic infection such as infective endocarditis
- ARDS/fibrotic lung disease

Diseases mimicking pneumonia
- Malignancy
- Systemic vasculitis
- Collagen vascular disorder
- Pulmonary oedema, CCF, heart failure with preserved EF, mitral regurgitation
- Drug induced pneumonitis
- Radiation pneumonitis
- Hypersensitivity Pneumonitis.
b) Assessment will involve history, examination and investigations to delineate which of the causes from the above list may be contributing.

**History:**
- Detailed history of travel, pets, occupation, medication, addiction and family history
- Past medical history; e.g. radiation for lymphoma or breast cancer, systemic disease e.g., RA
- Allergies
- Medications

**Examination:**
- Looking for signs of complication and signs suggestive of other systemic illness such as collagen vascular disorders.
- Assess for other sources of sepsis e.g. abscess, infectious endocarditis, catheter-related

**Investigation:**
Will depend upon the findings of the history and examination. Specific respiratory investigations may include:
- Repeat Tracheal aspirates- Send for fungal and cultures for unusual organisms
- Bronchoscopic aspirates both for infectious causes and cytology
- US guided pleural tap if fluid present
- CT Chest: High resolution chest CT to detect parenchymal abnormalities, including emphysema, airspace disease, interstitial disease, and nodules  
  - Chest CT also detects sequestered foci of infection, such as lung abscess and empyema, and helps direct biopsy procedures.
- Thoracoscopic or open Lung biopsy: If bronchoscopy is non diagnostic and failure to improve and large specimens are required then open lung biopsy can be resorted to.

**Other investigations may include:**
- Echocardiography
- Vasculitis screen
- EPG, IEPG, immunology screen, HIV serology

<table>
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**Question 22**
Critically evaluate the use of selective decontamination of the digestive tract (SDD) in the ICU.

**ANSWER TEMPLATE**

**Introductory statement**
SDD is a prophylactic strategy to prevent or minimise the incidence of nosocomial infection from endogenous organisms and to prevent or minimise cross-infection by the application of non-absorbable oral and enteric antibiotics and parenteral antibiotics. Classically SDD has four components:
- Administration of orobase and enteral antibiotics (e.g. polymixin B, tobramycin and amphotericin)
- Parenteral antibiotic e.g. cefotaxime
- Good hygiene to prevent cross-contamination
Microbiological surveillance of throat swabs and faecal samples. Variations exist:

- Oropharyngeal eradication only (SOD)
- Enteral only
- Oral and enteral only

Different antibiotics OR any reasonable and adequate introduction

**Rationale**

Nosocomial infections cause significant morbidity and mortality in the ICU. These infections arise from a limited number of potentially pathogenic micro-organisms (PPM) carried by healthy individuals (e.g. Staph aureus, E coli and C albicans) and opportunistic, aerobic Gram-negative bacilli (e.g. Klebsiella, Pseudomonas Acinetobacter) that colonise individuals when critically ill. The goal of SDD is to prevent or eradicate, if already present, at the start of ICU admission, the carriage of PPMs from the oropharynx and GI tract, leaving the indigenous flora, which protect against overgrowth with resistant bacteria, largely undisturbed.

**Arguments against:**
- SDD might lead to increased antibiotic resistance of colonising bacteria
- There is already a significant overuse of antimicrobial therapy

**Evidence**
- Over 60 RCTs with >15,000 patients (mostly in Europe) show benefits in terms of:
  - mortality (NNT ~18)
  - overall infection
  - lower airway infections
  - blood stream infections
  - oropharyngeal carriage
  - rectal carriage
  - MODS
  - ICU length of stay
  - Patient patients.
- The evidence does not suggest an increase in MROs
- However the number of trials with good scientific methods are few
- In the trials that suggested benefit, there was baseline variance in patient demographics and overall care
- The trials that suggest benefit have been conducted in areas with a low prevalence of multi-resistant organisms (northern Europe).
- There is a suggestion that selective oral decontamination is equally as effective as SDD, so the iv cephalosporins are not required. Await the results of the international multi-centre RCT SuDDICU

**Summary statement and Personal approach** - Any reasonable statement of candidate’s own approach, for example
- Risk benefits
- Adoption by communities vs. units
- Protocols driven by local flora and practice vs. world evidence
- Not widely used in intensive care practice in ANZ
- The need for a definitive trial, especially in the ANZ community

<table>
<thead>
<tr>
<th>Maximum Score</th>
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<tbody>
<tr>
<td>Percentage Passed</td>
<td>83.7%</td>
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</table>
Question 23

A 42-year-old miner has been transferred to the unit after an industrial accident in a remote location. He was entrapped in a fire underground.

He arrives 30 hours after injury, weighs 70 kg and is 1.75 m tall.

He is intubated and ventilated. His admission chest x-ray shows widespread bilateral pulmonary infiltrates.

His initial arterial blood gas shows the following:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patient Value</th>
<th>Adult Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>FiO₂</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>6.93*</td>
<td>7.35 – 7.45</td>
</tr>
<tr>
<td>pO₂</td>
<td>55 mmHg (7.3 kPa)</td>
<td></td>
</tr>
<tr>
<td>pCO₂</td>
<td>78.0 mmHg (10.4 kPa)*</td>
<td>35.0 – 45.0 (4.6 – 6.0)</td>
</tr>
<tr>
<td>SpO₂</td>
<td>87%</td>
<td></td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>16.0 mmol/L*</td>
<td>22.0 – 26.0</td>
</tr>
<tr>
<td>Base Excess</td>
<td>-14.0 mmol/L*</td>
<td>-2.0 – +2.0</td>
</tr>
</tbody>
</table>

His current ventilator settings are as follows:

- FiO₂ 1.0
- Respiratory rate 12 breaths/min
- Tidal volume 650 ml
- Peak Inspiratory Pressure 38 cmH₂O
- PEEP 5 cmH₂O

a) List six possible causes for his hypoxaemia. (20% marks)

b) Outline your management strategies for the treatment of his hypoxaemia. (80% marks)

**ANSWER TEMPLATE**

a)  
- Aspiration pneumonitis
- Blast Injury
- Smoke inhalation
- Misplaced ETT
- Fluid overload
- Pulmonary oedema
- Restrictive defect from circumferential burn

b) **General ventilator strategies based on ARDS net criteria**  
   - Check adequate placement of ETT (no R endobronchial intubation)
   - ARDS net ventilation Vt 6mls/kg = 420mls
   - best PEEP.
   - Use of recruitment maneuvers with derecruitment to assess best PEEP
   - CPAP 40/40 or step wise recruitment maneuvers
   - Use of flow loops
   - Aim Plateau <30cm H₂O avoid baro trauma
   - Increase I:E ratio towards 1:1 and increase rate if tolerated
Check for autoPEEP and use of broncho dilators
Treat reversible causes like PTX
C- rule out cardiomyopathy and improve V:Q match as cardiac function can be depressed in the severe inflammatory state.

**General adjunctive measures**
- Physio. Suctioning. Consider bronchoscopy
- Sedation, heavy sedation will be required. In advanced Hypoxia may require paralysis.
- Treat factors increasing metabolic demand (removal of eschar, treatment of sepsis, high risk of pneumonia)
- Optimize Hb and oxygen carrying capacity.
- If patient has been over resuscitated may require diuresis

**General Rescue therapies**
- Prone positioning- may not be practical in a burns patient
- Alternative ventilation strategies - prolonged
- ECMO in severe cases
- Nitric oxide or inhaled prostacyclin

**Burns specific measures**

**Bronchodilators**
- B2 agonists such as Adrenaline or Salbutamol
  - Adrenaline reduces blood flow to injured/obstructed airways improving V:Q mismatch

**Muscarinic receptor antagonists** – reduction of cytokines, reduction of mucus secretions
- NAC
  - Inhaled fibrinolytics for reduction of fibrin casts in volume and plugging.

**Bronchial Toileting.**
- bronchoscopy for cast removal and prevention and treatment of mechanical obstruction and plugging
  - ***rule out toxidromes, cyanide, CO, may need or antidote.****
- Ensure chest expansion not impeded by eschar
- May require escharotomies for free chest movement.

<table>
<thead>
<tr>
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<tr>
<td>Percentage Passed</td>
<td>65.3%</td>
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**Question 24**

With respect to the management of patients with aneurysmal sub arachnoid haemorrhage (aSAH), briefly discuss the role of the following:

- Nimodipine.
- Hypertensive / hypervolaemic / haemodilution, (HHH) therapy.
- Magnesium.
- Interventional radiology.
Nimodipine:
Level I evidence of improved neurological outcome. Calcium antagonist. Likely prevents neuronal damage by preventing influx of Ca more than by antagonising Sm muscle contraction and directly reducing incidence of vasospasm. May lead to hypotension.
Meta-analyses suggest oral efficacious
IV expensive; needs co-infusion
Recommended for all patients with aneurysmal SAH

Triple H therapy:
Haemodilution – no good evidence that works in isolation. Theoretically improved rheology => better perfusion
Hypervolaemia – no evidence that hypervolaemia is beneficial and fluid overload associated with worse outcomes. Hypovolaemia should be avoided as may exacerbate vasospasm. Volume loading often given to patients with clinical vasospasm to ensure euvolaemia
Hypertensive therapy: Unsecured aneurysm a relative CI to HT therapy. Demonstrated to improve cerebral blood flow. Not useful for prevention of vasospasm, but commonly used to treat cerebral ischaemia in the presence of vasospasm. May be titrated to clinical response. NA favoured agent. Sometimes high doses of pressor agents required to augment MAP. Risk of stress cardiomyopathy. Unless titrated to clear neurological signs the optimal MAP goals are unclear. Balance of risks vs benefits.

Interventional Radiology
Intra-arterial vasodilators: e.g. verapamil / papaverine / nicardipine. Clear angiographic benefit / used routinely for the treatment of vasospasm. Lacking high-level data on outcome benefit. Other angiographic interventions such as ballooning / stents are also utilised to good angiographic effect. Not routinely available in all centres. Caries the risks associated with angiography (transport, anaesthesia, contrast use, vascular injury, stroke)

Magnesium:
Several trials of MgSO4 – these have failed to show benefit. Not routinely indicated for the prevention of vasospasm although low magnesium may be associated with its development.

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<tr>
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<td>71.4%</td>
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Question 25

A 34-year-old male, who was previously well, has been admitted to your ICU with vomiting, malaise and oliguria. He has a new diagnosis of diffuse large B cell lymphoma and received his first round of chemotherapy four days ago.

The following blood results were obtained:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patient Value</th>
<th>Adult Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium</td>
<td>138 mmol/L</td>
<td>135 – 145</td>
</tr>
<tr>
<td>Potassium</td>
<td>6.2 mmol/L*</td>
<td>3.5 – 5.0</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>17.0 mmol/L*</td>
<td>22.0 – 26.0</td>
</tr>
<tr>
<td>Urea</td>
<td>21.0 mmol/L*</td>
<td>3.0 – 8.0</td>
</tr>
<tr>
<td>Creatinine</td>
<td>398 μmol/L*</td>
<td>45 – 90</td>
</tr>
<tr>
<td>Glucose</td>
<td>5.3 mmol/L</td>
<td>3.5 – 6.0</td>
</tr>
<tr>
<td>Magnesium</td>
<td>1.10 mmol/L*</td>
<td>0.75 – 0.95</td>
</tr>
<tr>
<td>Calcium corrected</td>
<td>1.78 mmol/L*</td>
<td>2.12 – 2.62</td>
</tr>
<tr>
<td>Phosphate</td>
<td>3.9 mmol/L*</td>
<td>0.8 – 1.5</td>
</tr>
<tr>
<td>Urate</td>
<td>1.10 mmol/L*</td>
<td>0.20 – 0.42</td>
</tr>
</tbody>
</table>

a) List a differential diagnosis for the kidney injury. (20% marks)

b) Outline your assessment and management of this patient. (80% marks)

ANSWER TEMPLATE

a)
It is likely the patient has tumour lysis syndrome (10/20).

Other differentials of acute kidney injury should be considered turn into list

- pre-renal (e.g. hypovolaemia, reduced perfusion)
- renal (e.g. glomerulonephritis, interstitial nephritis, vasculitis, ATN, acute cortical necrosis, drugs, pyelonephritis)
- post-renal (e.g. obstruction for calculi, tumour, clot)
- Chronic kidney injury which would put the patient at greater risk of tumour lysis is possible, but not likely if he is previously well.
- Sepsis is possible although it would usually occur at a later stage

(Marking Guide: 1 Mark for TLS; 0.25 marks for each reasonable differential)

b)
Resuscitation as needed to ensure adequate oxygenation and tissue perfusion.

Exclude differential diagnoses (history, examination and investigation including renal ultrasound).

- ECG, and if ECG changes consistent with hyperkalaemia, consider early intervention with (1) CaCl2 or Ca gluconate for temporary ECG stabilisation (new broad QRS or arrhythmia)
- (2) shift K intracellularly with either B2 agonist therapy (e.g. ventolin neb), insulin dextrose, or NaHCO3 if indicated

Specific management of tumour lysis syndrome

- IV fluids to encourage a diuresis
- Once well hydrated diuretics could be considered to encourage a diuresis (frusemide is most widely used, acetazolamide may alkalise the urine which will increase the solubility of uric acid but reduce the solubility of CaPO4 and hence is less widely used)
Rasburicase (to break down uric acid)
  - Urine alkalinisation not recommended if rasburicase has been given
  - If rasburicase not available allopurinol maybe given (however, this is not as effective and will not breakdown uric acid, it merely reduces further uric acid formation)

Haemodialysis and/or filtration is generally instituted for standard indications, although it maybe instituted earlier in tumour lysis syndrome or if the patient remains oliguric.

Treat hypocalaemia only if symptomatic (cramps, paraesthesia) as excessive Ca replacement may precipitate CaPO4.

Hyperphosphataemia is most efficiently treated with haemodialysis and/or filtration, insulin dextrose and oral phosphate binders have a limited role.

Seek and treat sepsis.

Consider excluding renal obstruction with US.

Routine supportive care
Optimise oxygenation (if fluid overloaded maybe need supplemental oxygen)
Optimise perfusion and blood pressure
Withhold nephrotoxic medications drugs (NSAIDS, ACE-I, ARBs)
Consider dose modification of renally excreted drugs
DVT prophylaxis
Single room/ neutropenic precautions

<table>
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<td>71.4</td>
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Question 26
Discuss strategies to limit antimicrobial resistance (AMR) in the ICU.

**ANSWER TEMPLATE**

Factors driving antimicrobial resistance (AMR) include inappropriate use of antibiotics, inadequate monitoring and surveillance, poor infection control practices and failing antibiotic pipeline.

Strategies to limit AMR include:
1. **Antimicrobial Stewardship**
   - Appropriate antimicrobial prescribing (right indication, right drug(s), right dose, right dosing regime, right duration)
   - Liaison with microbiology / infectious diseases team
   - Knowledge of local antibiograms
   - Streamlining to narrow spectrum drugs / oral agents when appropriate
   - Education of staff
   - Computer-assisted prescribing
   - Prescribing protocols
   - Cycling of antibiotics (uncertain benefit)
   - Antimicrobial prescribing committee

2. **Infection control**
   - Hand hygiene
   - Barrier precautions
Environmental cleaning
Isolation / cohorting of patients
Surveillance / screening / monitoring
Appropriate staff:patient ratios
Limit indwelling devices / appropriate asepsis for insertion etc
Care bundles to reduce VAP, reduce time on ventilator, early enteral feeding etc

3. Other
Vaccination programs
Adequate source control e.g. surgical drainage of abscesses
Future directions include:
More rapid and accurate diagnosis of sepsis
Advances in genomics
Immunomodulating agents
Use of bacteriophages
Use of antibiotics in agriculture and animal husbandry
New drug development
Synergistic combinations of antibiotics and drugs with no antimicrobial effect (eg minocycline and loperamide enhances action against staph aureus)

<table>
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<td>Percentage Passed</td>
<td>57.1%</td>
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</table>

Question 27

A previously well 28-year-old male is brought to the Emergency Department following an accident in the garden. He was on a ladder pruning a tree when he touched an overhead power line and was electrocuted. He was thrown to the ground, unconscious and had bystander CPR. Paramedics arrived after 10 minutes, and intubated and ventilated the patient who had return of spontaneous circulation and a Glasgow Coma Scale of 5 at the scene.

a) List the major issues that you would consider in the initial management of this patient. (40% marks)

b) After four days, he develops anuric acute kidney injury (AKI). Describe how you will assess the factors contributing to the AKI. (60% marks)

ANSWER TEMPLATE

a)
The potential issues the that need to be considered in this patient include

- Electrocution
- Trauma from the fall
- Hypoxic-ischaemic brain injury
- Aspiration

1. Electrocution
- Myocardial damage/Unstable rhythm
- External burns
- Rhabdomyolysis/ internal tissue burn / compartment syndrome.
- Electrolyte abnormalities e.g. hyperkalaemia
- Traumatic injuries as below
- Hypovolaemia due to fluid extravasation
• Neurological damage – central and peripheral, including autonomic neuropathy

2. Hypoxic-Ischaemic Brain injury

3. Trauma from the fall
   • Head and or spine injury
   • Blood loss
   • Abdominal injury
   • Rib fractures
   • Long bone/ pelvic injury

4. Aspiration
   • Pneumonitis
   • Foreign body aspiration

b) The assessment of factors contributing to AKI in this setting

Pre-renal causes
   • Most likely
     o Ongoing/ new hypovolaemia
     o Low cardiac output secondary to myocardial injury
     o Renal artery/vein injury from trauma
   • Assess volume status
   • History, examination, monitoring, investigations
   • Check Hb
   • *(echocardiography
   • Urinary fractional sodium excretion

Renal causes
   • Most likely
     o Rhabdomyolysis
     o Other nephrotoxin
     o Abdominal compartment syndrome
     o Drug reaction -> interstitial nephritis
   • Examination for ongoing compartment syndrome, check CK
   • Assess medications and cease any nephrotoxins (NSAIDS, gentamicin, vancomycin)
   • Examination of abdomen, measure intra-abdominal compartment pressure, consider renal ultrasound with duplex if retroperitoneal haematoma
   • Urinary microscopy to look for casts, assess medications for potential causes (penicillins, cephalosporins, pantoprazole)

Post renal causes
   • Most likely IDC obstructed, or clot in renal pelvis, pelvis causing ureteric obstruction
   • Ensure IDC not blocked
     o Flush catheter, bladder ultrasound
     o Renal ultrasound to exclude obstruction

<table>
<thead>
<tr>
<th>Maximum Score</th>
<th>8.2</th>
</tr>
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<tbody>
<tr>
<td>Percentage Passed</td>
<td>67.3%</td>
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</table>
Question 28

A colleague directs your attention to a recently published randomised trial on a therapeutic intervention.

Outline the features of the trial that would lead you to change your practice.

**ANSWER TEMPLATE**

Points to consider in the answer would be:

1. Does the population studied correspond with the population the candidate expects to treat?
2. Were the inclusion/exclusion criteria appropriate?
3. Was the trial methodology appropriate – was there adequate blinding and randomisation?
4. Was the primary outcome a clinically relevant or a surrogate endpoint?
5. Was the length of follow up adequate?
6. Was the trial sufficiently powered to detect a clinically relevant effect?
7. Were the groups studied equivalent at baseline?
8. Is the statistical analysis appropriate – was there an intention to treat analysis, have differences between groups at baseline been adjusted for? Are there multiple sub group analyses, and if so were they specified *a priori*?
9. Is this a single centre study or multi centre?
10. Were the results *clinically* significant rather than just statistically significant?
11. Is the primary hypothesis biologically plausible with pre-existing supporting evidence?
12. Are the findings supported by other evidence – have these results been replicated?
13. Would there be logistical and/or financial implications in practice change?
14. Are there important adverse effects of the treatment?

<table>
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<tr>
<th>Maximum Score</th>
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<td>Percentage Passed</td>
<td>65.3%</td>
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</table>

Question 29

Compare and contrast heparin induced thrombotic thrombocytopaenia syndrome (HITTS) and thrombotic thrombocytopaenic purpura (TTP) with respect to their pathogenesis, clinical features, relevant laboratory findings, and treatment.

**ANSWER TEMPLATE**

<table>
<thead>
<tr>
<th>Condition</th>
<th>TTP</th>
<th>HITTS</th>
</tr>
</thead>
</table>
| Aetiology 1 mark each | 1. Reduction in ADAMTS 13 level  
   i. Hereditary causes  
   ii. Acquired causes e.g. Sepsis, surgery, pancreatitis, pregnancy | Autoantibodies to platelet factor 4 (PF4) complexed with heparin |
| Clinical Features 1 mark each | Small vessel thrombosis-  
   Microangiopathic haemolytic anaemia with multisystem involvement but renal and CNS dominate- | Occurs within 4-10 days of heparin administration- arterial and venous thrombosis, thrombocytopenia |
Laboratory 2 marks each

| Laboratory | Low platelets, intravascular haemolysis with evidence of micrangiopathic changes to RBC morphology. LDH, Haptoglobin, COOMBS test, ADAMTS 13 deficiency | Ant PF4 antibody, Functional Assays- (serotonin release, Heparin induced platelet activation) |

Treatment 1 mark each

| Treatment | Plasmapharesis is the mainstay of treatment for Acquired TTP, FFP for hereditary TTP | Cessation heparin Non heparin based anticoagulation (Lepuridin etc) |

Maximum Score 9.0
Percentage Passed 73.5%

Question 30

a) List the ECG criteria that are helpful in distinguishing ventricular tachycardia (VT) from supraventricular tachycardia (SVT) with aberrant conduction. For each listed criterion, indicate which diagnosis it makes more likely. (30% marks)

b) List the specific management strategies that may be used to treat torsades de pointes. (30% marks)

c) List the important differences in managing a cardiac arrest in a post-operative cardiac surgical patient in ICU as compared to a non-cardiac surgical patient. (40% marks)

**Answer Template**

**a)**
- Capture beats: VT
- Fusion beats: VT
- Concordance in chest leads (or absence of RS complex): VT
- Typical RBBB or LBBB morphology: SVT
- R to S interval >100ms: VT

*(Note: there are some more specific criteria from diagnostic algorithms – if correct these should receive credit.)*

**b)**
- Correction of electrolyte abnormalities or hypothermia
- Magnesium
- Isoprenaline
- Phenytoin
- Sodium Bicarbonate
- Lignocaine
- Electrical cardioversion
- Atrial overdrive pacing
- Cessation of provoking drugs

**c)**
- Immediate VF or pacing (if indicated) before external cardiac massage – can delay ECM up to one minute to administer shock/pace
- No need for pulse check – observe monitored waveforms/ECG sufficient for diagnosis
- Avoid adrenaline/vasopressin bolus
• Cease all infusions until reviewed
• If IABP in situ set to pressure trigger
• If PEA and paced, turn off pacemaker to exclude underlying VF
• Plan for emergency re-sternotomy, ideally within five minutes.

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<tr>
<th>Maximum Score</th>
<th>7.0</th>
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<tr>
<td>Percentage Passed</td>
<td>49.0%</td>
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EXAMINERS’ COMMENTS

Hot Cases

The Hot Cases run for twenty minutes with an additional two minutes at the start of each case for the candidate to be given both a verbal and a written introduction to the case in question. This is to give candidates more opportunity to take in the relevant information and to plan a focussed approach to examination of the patient.

The following comments are a guide to the expected standard for performance in the Hot Cases:

- Candidates should demonstrate professional behaviour, treating the patient with consideration and respect.
- Candidates should address and answer the question asked of them in the introduction to the Hot Case.
- Candidates should interpret and synthesise information as opposed to just describing the clinical findings.
- Candidates need to seek information relevant to the clinical case in question.
- Candidates should be able to provide a sensible differential diagnosis and appropriate management plan. A definitive diagnosis is not always expected and in some cases may yet to be determined.
- Candidates should not rely on a template answer or key phrases but answer questions in the context of the clinical case in question.
- Candidates must be able to describe, with justification, their own practice for specific management issues.

Candidates who performed well in the Hot Cases, as in previous exams, were able to demonstrate the following:

- A professional approach showing respect and consideration for the patient.
- Competent, efficient and structured examination technique and also able to appropriately adapt the examination to suit the clinical case in question.
- Seeking of information relevant to the case.
- Appropriate interpretation and synthesis of their findings.
- Presentation of their conclusions in a concise and systematic fashion, addressing the issue in question.
- Listing of a differential diagnosis that is relevant to the clinical case in question.
- Appropriate interpretation of relevant investigations.
- Discussion of management issues in a mature fashion, displaying confident and competent decision-making.
- An appreciation of the complexities and key issues of the case.
- Overall performance at the expected level (Junior Consultant).

Candidates who did not perform at the acceptable standard did so for reasons including the following:

- Missing or misinterpreting key clinical signs on examination.
- Failure to perform a focussed examination relevant to the case in question.
- Incomplete or poor technique for examination of a system.
- Poor synthesis of findings with limited differential diagnosis, sometimes compounded by missed key clinical signs on examination.
- Poor interpretation of imaging and data.
- Failure to grasp the key issues relevant to the case in question and a lack of insight into the problems.
• Inability to construct an appropriate management plan for the case in question.
• Hesitancy and/or uncertainty in stating a management plan.
• The need for significant prompting during the discussion with knowledge gaps.
• Limited time for discussion as a consequence of taking too long to present the clinical findings or to interpret basic data.
• Inability to convey the impression that he/she could safely take charge of the unit.

It is apparent that some candidates are very nervous and this affects their exam performance. Candidates badly affected by nerves may benefit from sessions with a performance psychologist, drama coach, public speaking coach or similar.

Candidates are advised that they should not sit the Second Part Examination until they can confidently examine patients, present the relevant clinical findings, synthesise all the information and discuss management issues at the appropriate level, i.e. demonstrate that they are capable of safe, effective, independent practice at the level of a Junior Consultant. Candidates should practise Hot Cases from the commencement of their exam preparation. To this end, candidates are encouraged to do the following in their daily clinical practice as preparation for the Hot Cases:

• Seek the opportunity to take charge of the unit and be responsible for management decisions.
• Practise examination of individual systems.
• Treat every case to be assessed at work as a Hot Case, i.e. pose a relevant question (e.g. ‘Why is this patient not progressing?’ ‘What is the cause of the new fever?’ ‘Is this patient ready for extubation?’), perform a focussed exam and then present your findings to a colleague.

Vivas

The overall pass rate for the vivas was 77%, compared with 80% for the written paper and 70% for the Hot Cases. Two vivas had a pass rate less than 50%, those related to nutrition and pacing. Candidates who failed a viva mostly did so because of knowledge gaps, poorly structured answers and inability to give the rationale for their responses. As in the discussion for the Hot Cases, candidates should not rely solely on generic statements, key phrases and template answers, and, instead, tailor their responses to the specifics of the question and be able to justify and expand their response. Candidates are encouraged to practise viva technique and to discuss patient management, including the rationale for their decisions, with senior colleagues. As with the Hot Cases, candidates who are very nervous or have a poor technique may benefit from training with a performance coach.
SECOND PART ORAL EXAMINATION

CLINICALS “HOT CASES”

A 50-year-old male. Day 16 ICU. Presented with throat pain and shortness of breath. Required an incision and drainage of a retropharyngeal abscess and a surgical tracheostomy. Candidates were asked to examine him and formulate a weaning plan for the next week. Discussion points included interpretation of CT findings, and the role of hyperbaric therapy.

A 21-year-old female. Day 5 ICU. Presented the Emergency Department after a hanging injury. Candidates were asked to examine her, and outline the current clinical issues, including an assessment for extubation.

A 33-year-old female. Day 55 ICU. Acute respiratory failure post-partum. Delivered 35/40 live healthy baby. Initially intubated and ventilated for five weeks, then deteriorated and placed on ECMO sixteen days ago. Candidates were asked to examine her, identify issues requiring ICU support, and develop a management plan for the next week. Areas of discussion included the differential diagnosis of her respiratory failure and issues surrounding anticoagulation.

A 78-year-old male. Day 6 ICU. Admitted following a motorcycle accident. Candidates were asked to examine him with a view to establishing his injuries, current issues, and to determine a management strategy. Areas of discussion included plan for removal of intercostal catheters and the role of tracheostomy.

A 70-year-old male. Day 5 ICU. Admitted following a boating accident in which he suffered a head injury. Candidates were asked to examine him to identify injuries, complications and formulate a management plan. Areas of discussion included the management of cardiac arrhythmias and the potential for extubation.

A 40-year-old male. Day 8 ICU. Fall from 4m with resulting head injury. Initial GCS of 4 and fixed dilated pupils. Candidates were asked to examine him, establish the nature of his injuries, prognosis and comment on his subsequent management. Areas of discussion included the role of decompressive craniectomy and issues of prognostication in traumatic brain injury.

A 47-year-old male. Day 3 ICU. Found by the roadside with a GCS of 8. Mechanism of injury unknown. Candidates were asked to assess him with a view to determining why he still needed intensive care. Areas of discussion included prognostication in traumatic brain injury, the role of intracranial pressure monitoring, and the management of cervical spine injuries.

A 46-year-old female. Day 10 ICU. High speed motor vehicle accident. Candidates were asked to examine her with a view to determining her clinical status and to provide a management plan. Areas for discussion included clinical signs of cervical spine injury, and the management of planned withdrawal of care.

A 54-year-old female. Day 3 ICU. Admitted after bilateral lung transplantation. Candidates were asked to assess her suitability for extubation. Discussion points included post-operative complications and methods for optimising graft function.

A 71-year-old male. Day 4 ICU. Admitted following out of hospital cardiac arrest. Candidates were asked to assess his suitability for extubation. Discussion points included prognostication and the role of targeted temperature management.

A 66-year-old male. Day 12 ICU. Type I respiratory failure. Lung transplant six months previously for idiopathic pulmonary fibrosis. Candidates were asked to examine him and outline a respiratory
weaning plan. Discussion points included differential diagnosis, complications of immunosuppression and management of muscle weakness.

A 49-year-old female, presenting for an elective coronary angiogram that was complicated by a LAD dissection and subsequent cardiogenic shock, requiring urgent coronary artery bypass grafting. Now at day 15 post admission she has developed a new fever of 39°C. Candidates were asked to assess her with regards to the cause of her fever.

A 69-year-old male, originally been admitted 4 weeks prior to a peripheral hospital with a perforated duodenal ulcer, and was transferred to the current institution 6 days ago with a suspected perforated oesophagus. He had been extubated one day ago. He had a background history of excess alcohol consumption and hypertension. Candidates were asked to assess him to determine his major management priorities and barriers to discharge to the ward.

A 56-year-old male now day 3 in the ICU following an emergency craniotomy. He had a background history of hepatitis C. He had received no sedation for 48 hours but remained obtunded. Candidates were asked to assess him with regards to his prognosis.

A 58-year-old male with a background history of end stage renal failure due to reflux nephropathy, kairoscerosis, chronic osteomyelitis of the left foot and MRSA colonisation, had been intubated 9 days prior following a MET call in the renal dialysis unit. Candidates were asked to evaluate him for possible extubation.

A 48-year-old female with end stage renal failure secondary to reflux nephropathy who had been admitted also following a MET call for hypotension and stridor. Candidates were asked to assess her cardiovascular system and suggest a management plan.

A 77-year-old male admitted to the ICU 22 days prior. He was admitted to the hospital for a parastomal hernia repair, and developed post-operative hospital acquired pneumonia. His sedation had been ceased 10 days prior, but he remained unconscious. Candidates were asked to examine him with regards to his failure to wake, determine the cause/s and suggest an initial management plan.

A 32-year-old female with no significant background medical problems, who had been admitted to the ICU following a respiratory arrest on the ward. She remained mechanically ventilated after 33 days, and the candidates were asked to assess the cause for her prolonged requirement for mechanical ventilation.

A 64- year-old male who sustained a major traumatic injury 4 days previously from a 3-metre fall with prolonged extrication was presented. He was in shock at the scene with a body temperature of 30.1°C and a Glasgow Coma Score of 13. The patient remained sedated and ventilated. Candidates were asked to perform a primary survey and in addition to identify other relevant issues and discuss a management plan for the subsequent 24 hours. The discussion included interpretation of a pelvic CT-scan and pelvic injuries, causes for the acute kidney injury that the patient sustained and the approach to a significantly elevated troponin in this context.

The patient presented was an 84-year-old male who had been in ICU for 2 days following a fall from a balcony at a rehabilitation facility. The patient had a decreased level of consciousness at the scene, was haemodynamically unstable and received a massive transfusion. He had a background history of a myocardial infarction, pulmonary embolism on warfarin and essential thrombocytosis for which he was receiving hydroxyurea. The patient had an intracranial pressure monitor in place. Candidates were directed to examine the patient to identify the key issues and discuss a management plan for the next 24 hours. The discussion included interpretation of trauma imaging, causes of shock in a trauma patient and intracranial pressure monitoring.
A 73-year-old male of non-English speaking background was presented who had been in ICU for 24 days following a tissue mitral valve replacement and coronary artery grafts x3. He had a failed extubation 10 days previously and had a tracheostomy in place. He had type II diabetes and an above knee amputation. Candidates were asked to assess the patient and formulate a weaning plan. Investigations that were discussed included the chest X-ray, ECG and routine blood tests. The discussion focused on the weaning plan, management of fluid overload and causes of macrocytosis in this patient.

A 63-year-old male who had spent 108 days in ICU was presented. He suffered multi-trauma from a motorbike accident, which resulted in bilateral flail chest, splenic laceration and pelvic fractures. The patient also required a bowel resection and ileostomy formation for ischaemic bowel. The patient had a tracheostomy, which was inserted 3 weeks into his admission and also had positive blood cultures for methicillin resistant staphylococcus epidermidis. Candidates were asked to assess the patient and make a plan to wean him from the ventilator. The patient was cachectic, weak and areflexic. He had a high-output from his stoma and had a rash over the anterior abdominal wall. The discussion focused on the weaning plan, potential causes for the rash and management of methicillin resistant staphylococcus epidermidis.

A 60-year-old male who had an intracranial event 7 days ago was presented. The neurological recovery was poor. Candidates were told that they had to meet with the patient’s family later that day to discuss prognosis and were instructed to examine the patient to enable them to have this discussion. Investigations discussed included a CT-brain, which showed an extensive subarachnoid haemorrhage, blood tests and chest x-ray. The patient had an old fistula site and an incision from renal transplant surgery. Discussion included complications of subarachnoid haemorrhage and factors affecting prognostication in the patient.

A 65-year-old female who had suffered a massive haemorrhagic stroke was presented. Candidates were told that staff were concerned that she had developed brain death and asked to examine the patient to determine whether she was brain dead. The discussion included pre-requisites for brain death testing and management of the brain dead organ donor.

A 66-year-old female who was admitted 4 days previously for abdominal pain and hypotension was presented. She had a history of chronic obstructive airways disease and was a smoker. Candidates were told that she had ongoing abdominal pain and were asked to examine her and discuss a plan for further management. The patient had an open abdomen. The discussion included management of nutrition, the role of immunonutrition and plans for communication with the surgeons and the patient’s family.

A 59-year-old male admitted 17 days previously with a rash over his abdomen and legs with profound hypotension was presented. He had a history of hypertension, epilepsy and excess alcohol use. He had an elevated temperature, a fluctuating level of consciousness and was slow to progress. He had grown streptococcus pyogenes in his blood cultures. Candidates were asked to examine him and discuss causes for this slow progress and how to manage him. The discussion included the complications of group A streptococcal infections, management of hospital acquired pneumonia and delirium.

A 37-year-old male with a retropharyngeal abscess, delirium and mild airway compromise. This case was clinically rich with signs of systemic sepsis; stridor; delirium, and clinical signs of consolidation and effusion at the right lung base. CT scans confirmed the respiratory signs; with additional air in the soft tissues. There were significant management issues for discussion including the assessment and management of delirium; airway management and antibiotic choices. Most candidates performed to a satisfactory or high standard.

A 48-year-old male, day 66 ICU. This gentleman with hereditary haemorrhagic telangiectasia was initially admitted with massive upper gastrointestinal bleeding. Despite initial multiple laparotomies
and endoscopic procedures for ongoing bleeding, there was continued slow gastrointestinal blood loss requiring transfusion each 3 to 4 days. In addition, there were multiple long-term problems including a weakness syndrome; ventilator dependence and weaning; tracheostomy management; chronic liver disease with ascites and portal hypertension; intermittent nosocomial infection and depression.

A 45-year-old male who presented with headache due to cerebral toxoplasmosis as an AIDS defining illness. The patient remained intubated; and EVD was in-situ and was awake and cooperative. There were multiple abnormal neurologic signs including bilateral nystagmus, hypertonia and hyperreflexia and extensor plantar responses. Candidates were asked to assess readiness for extubation.

A 55-year-old female day 10 following a subarachnoid haemorrhage due to a ruptured R MCA aneurism. A craniectomy and bone flap were present; an EVD in situ and neurologic outcome was poor; with an un-sedated GCS of 6 and focal neurology.

A 55-year-old female day 1 post CABG. Comorbidities of adult polycystic kidney disease with end stage renal failure on peritoneal dialysis. A functioning AV fistula was present. There was haemodynamic stability, the patient remained sedated. Discussion points included provision and mode of renal replacement therapy.

A 45-year-old male, day 16 ICU presented with a spontaneous T6-L5 epidural haematoma and a posterior SAH. This was clearly an unusual presentation. Initially there were symptoms of facial pain and weakness which resolved, with a normal initial CT scan. Subsequently there was an acute flaccid paraparesis and repeat imaging revealed the epidural haematoma and an additional intracranial sub arachnoid haemorrhage. An AVM was postulated but not found. Clinically, a tracheostomy was in-situ with a fluctuating conscious state, and an areflexic paralysis of both legs. There were multiple issues for discussion including aetiology, weaning and prognosis.

VIVAS

Viva 1

A 65-year-old male has just been admitted to your ICU following an out of hospital cardiac arrest.

He is currently receiving 6 mcg/min of adrenaline with a blood pressure of 100/70 mmHg, and has been anuric for the last two hours. He has a Glasgow Coma Score of 6. His current temperature is 37.6°C.

What factors would you take into consideration in managing this patient’s temperature?

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(This viva focussed on issues relating to temperature control and therapeutic hypothermia.)

Viva 2

You have been asked to assess a 34-year-old female who has presented with progressive lethargy, malaise and confusion.

She has a background of previous morbid obesity and underwent bariatric surgery in the last 6 months with subsequent extensive weight loss.
She has been intubated for lowered conscious state in the emergency department.

On examination she is dehydrated, afebrile with a blood pressure 120/80 mmHg, pulse 118/min, SpO₂ 99% on FiO₂ of 0.3.

Prior to intubation she was noted to have bilateral nystagmus.

What is the differential diagnosis?

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(This viva focussed on nutritional and metabolic issues.)

Viva 3

You are asked to review a 64-year-old male who had an emergency abdominal aortic aneurysm repair last night. His sedation has been ceased in anticipation of extubation, but the bedside nurse is concerned that he appears to be in a lot of pain.

His relevant co-morbidities include ischaemic heart disease, type 2 diabetes mellitus and chronic back pain for which he has been on long term opioid treatment.

Outline how you would assess the situation.

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(This viva focussed on issues relating to assessment and management of pain in critically ill patients.)

Viva 4

You are taking over the care of a 26-year-old male who remains ventilated one week after a motor vehicle accident in which he sustained severe intraabdominal and head injuries as well as multiple fractures. He underwent an immediate trauma laparotomy and required a splenectomy and repair of a bowel perforation.

He has had persistent fevers since admission, and has received a course of Piperacillin and Tazobactam. He is currently receiving low dose noradrenaline, and parenteral nutrition via a non-tunelled central venous catheter.

He has a history of intravenous drug use.

Give a differential diagnosis for the fever in this patient and outline your management.

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(This viva focussed on issues relating to fungal sepsis.)
Viva 5

A 75-year-old previously fit and well female was admitted to the ICU with hypoxaemic respiratory failure, due to community acquired pneumonia.

She developed multi-organ failure over the first 3 days, and required extensive support. Her cardiorespiratory status gradually improved but she now remains ventilator dependent at day 10.

What might be the specific barriers to her weaning from the ventilator, and how might they be managed?

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(This viva focussed on issues relating to prolonged weaning from mechanical ventilation.)

Viva 6 – Procedure Station

You are asked to review a 68-year-old male. He is two days post aortic valve replacement for aortic stenosis with good left ventricular function.

His recovery has been uneventful other than a junctional bradycardia of 40 bpm that has required VVI pacing at 80 bpm via an epicardial right ventricular wire placed at the time of surgery.

His nurse asks for your help as his blood pressure has been falling. He is on nasal prong oxygen, and remains conscious.

His bedside monitor looks like this:

(Image removed from report.)

How would you approach this problem?

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(This viva focussed on issues relating to managing temporary pacing.)

Viva 7 – Radiology Station

(The radiology station comprised two plain films, three CT scans and one MRI.)

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Viva 8 – Communication Station

You are the ICU Consultant managing Paul, a 22-year-old male who was admitted to your ICU yesterday with septic shock and multi-organ failure secondary to severe community acquired pneumonia. He had been well until the 24 hours prior to his admission.
Currently he is on 100 ug/min of noradrenaline, vasopressin at 0.04 units/min, hydrocortisone 200 mg/d, is invasively ventilated, on CVVHDF, and on a 50% dextrose infusion. He is receiving blood products for bleeding from multiple sites. He is on appropriate antibiotics. His vasopressor doses and lactate (currently 13) are rapidly rising.

The consensus of your ICU specialist colleagues is that he will be unsupportable and die within the next 12 hours, and there are no further therapeutic options.

His relatives have just arrived and are waiting to speak to you.

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