COLLEGE OF INTENSIVE CARE MEDICINE
OF AUSTRALIA AND NEW ZEALAND

SECOND PART EXAMINATION

WRITTEN EXAM REPORT

MARCH 2020

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 written exam consists of two 2.5 hour papers of 15 short answer questions each. The pass mark for the written section is derived by the Angoff method and for this sitting was set at 50%. The clinical section of the March sitting was postponed; this report provides details for only the written section.

The table below provides an overall statistical analysis in the written section. 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.

Overall Performance	March 2020	October 2019	May 2019	October 2018	May 2018	October 2017
Presenting for written (Including OTS)	50	57	44	67	49	49
Carrying a pass or exempted from a previous attempt	11	7	13	7	11	8
OTS Exempt	0	0	0	0	0	0
Total number presenting (written + carry + exempted + OTS)	61	64	57	74	60	57
Invited to orals (passed written section)	37	34	20	47	28	39
Total number invited to oral section	48	40	33	54	38	47

EXAMINERS' COMMENTS

Written Paper

The pass rate for the written section was 72%. Questions dealing with endovascular clot retrieval, pancreatic surgery, cardiopulmonary bypass and clinical signs were particularly poorly answered. A common comment from examiners was that candidates had resorted to generic answers without considering specific clinical issues in the question.

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 did poorly on 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 scored an average of 47% compared with those candidates who passed, whose average score was 57%.

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

With regards to rhabdomyolysis in the ICU patient:

a) List five causes. (20% marks)

b) What are the important features in the history and clinical examination, and what specific laboratory investigations would you request? (30% marks)

c) Outline the management. (50% marks)

ANSWER TEMPLATE

a)
Causes (2 marks; 0.5 marks per cause. No additional marks for multiple causes under same heading. Examples of medications/infections etc. required for marks.)

1. Trauma - Crush injury, electrocution, extensive burns, compartment syndrome
2. Exertional - Prolonged exertion, refractory seizures, severe agitation
3. Medications - corticosteroids, statins, antipsychotics, neuroleptics
4. Toxins - methanol, ethylene glycol, barbiturates, amphetamine, MDMA, CO poisoning, cyanide, snake, spider envenomation
5. Body temperature changes - Heat stroke, malignant hyperthermia, malignant neuroleptic syndrome, hypothermia
6. Infections - Influenza A and B, coxsackievirus, Epstein–Barr virus, primary HIV, legionella
7. Rarer causes – electrolyte disturbances (hypoPO4, K, Ca, Na), endocrine (HONK etc.)

b)
Review history to identify risk factors or causes. .History of pre-existing renal injury or conditions that might predispose to acute kidney injury or conditions that might predispose to it.
History of Trauma, seizures, immobility, drug exposure, muscle pain, dark coloured urine,
Clinical examination/assessment: Muscle compartment swelling, tenderness, weakness, fever, myoglobinuria, peripheral perfusion

Laboratory Tests
Elevated CK
Renal dysfunction Cr – elevated; urea:Cr ratio may be decreased
electrolyte abnormalities (hyperkalemia, hypocalcemia, hyperphosphataemia, hyperuricemia, lactic acidosis). Elevated AG – due to phosphates and organic acids released from muscle
Further investigations will depend on need to further identify underlying cause e.g. urine drug screen, blood alcohol, glucose, TFTs etc

c)
Management (5 marks)
Stop further skeletal muscle damage
Interventions will vary depending on cause; e.g. discontinuation of medications, control agitation, treat infection, correct metabolic abnormalities, cool or warm, surgery etc
Rapidly identify life or limb-threatening complications
If compartment syndrome needs orthopaedic consultation – monitor pressures +/- decompressive fasciotomy
Treat any significant electrolyte abnormalities, e.g. hyperkalaemia or hypocalcaemia
Prevent acute renal failure
Early and aggressive volume resuscitation with 0.9% N/saline. Dilutes nephrotoxins and promotes renal tubule flow. Urinary alkalization, forced mannitol diuresis and frusemide have been described; *(candidates were not given marks for mentioning these or penalised for omitting them)*
Renal replacement therapy: for usual traditional indications (hyperkalaemia, metabolic acidosis, volume overload and uraemia). Not advocated for myoglobin removal

Examiners Comments:

Candidates are reminded to read the question carefully; some answers included investigations such as CT scans and exploratory surgery, which are not "laboratory tests".

Well answered overall.

Maximum Score	7.5
Percentage Passed	78.0%

Question 2

a) Describe your initial ventilator settings for a patient just intubated for acute severe asthma. Explain the rationale for each of your choices. (50% marks)

b) Hypotension commonly occurs after intubation in an asthmatic. What are the potential aetiologies and what steps would you take to prevent and/or treat them? (50% marks)

ANSWER TEMPLATE

Either volume controlled or pressure controlled modes are acceptable.
Generally spontaneous modes are avoided early and when unstable, needs deep sedation +/- paralysis to facilitate non injurious mode of ventilation
FiO2 1.0 – newly intubated – titrate down asap as risk of O2 toxicity and Aa gradient not usually a problem
PEEP: – controversial – Conventional teaching advocates a PEEP 0 to minimise high Paw, but there will already be some dynamic hyperinflation with intrinsic PEEP – set PEEP in or around this. Acceptable to mention PEEP titration to pressure/volume curves, but not required.*(a discussion around what PEEP would be set with a reasonable justification was required for marks)*
VT 4-6ml per kg – limited by plateau pressure < 30 – note PIPs will be high and need to be tolerated, e.g. up to 50 cmH2O), ventilator alarms will need to be adjusted
RR / T insp. to be minimised to avoid dynamic hyper-inflation (or prolong exp. time)
Generally aim Pplat <30cm/H20 and PEEPi <10

Dehydration – unwell by days, inadequate PO intake and then positive intrathoracic pressure – decreasing preload further, minimised by IV fluids loading prior to intubation, and volume loading afterwards to treat.

Afterload reduction/obliteration of sympathetic stimulation – drugs (sedatives and bronchodilators), use vasoconstrictors (titrated metaraminol or noradrenaline), may alter induction drugs or doses used

Dynamic hyperinflation exacerbating the preload reduction. Prevention is with settings targeting lower RR, and shorter insp time, Pplat <30 cmH2O and PEEPi<10. Treat by disconnection of patient from the ventilator and transiently ceasing ventilation. Occasionally manual compression of chest required to aid expiration.

Tension pneumothorax. Prevention is by avoiding high tidal volumes/ mean airway pressures, and accepting high pCO2 if necessary. Paralysis to prevent coughing. Tension pneumothorax is treated with immediate decompression (e.g. with 14 G needle, then early intercostal chest drain).

*If dynamic hyperinflation as a cause of hypotension was not mentioned, a candidate could only score a maximum of 4/10)

Examiners Comments:

Generally, well answered other than the justification for PEEP use.

Maximum Score	7.8
Percentage Passed	92.0%

Question 3

With respect to advanced cardiac life support (ALS), outline the modifications to the standard adult ALS algorithm needed in the management of cardiac arrest in the following clinical situations. Give the rationale for the modifications where appropriate.

a) A 72-year-old female ventilated in ICU 4 hours post-cardiac surgery. (40% marks)

b) A 66-year-old male with accidental hypothermia and core temperature < 24ºC. (30% marks)

c) A 34-year-old 32/40 gestation pregnant female. (30% marks)

ANSWER TEMPLATE

a) 72-year-old female ventilated in ICU 4 hours post cardiac surgery

Team composition	• Requires Cardiac surgeon, operating theatre team and anaesthetist *(Call surgeon acceptable)*
Before-Ext cardiac massage	• Stop all intravenous drug infusions (to minimise drug errors & force new infusions to be made up) • VF/VT: Deliver 3 shocks if shockable rhythm • Asystole/Bradycardia: Connect epicardial pacing wires at 90Bpm DDD and maximum output or asynchronous • PEA and pacemaker connected, disconnect to exclude underlying VF
Resternotomy	• If no cardiac output in 1m. Will need bimanual cardiac massage and internal defibrillation post sternotomy. External cardiac massage no longer possible.
Adrenaline	• Judicious use aware of possibility of hypertension causing bleeding. Consider reduced dose or avoidance.
Amiodarone	• If 3 stacked shocks unsuccessful
Atropine	• 3mg if extreme bradycardia, asystole
IABP	• Set to pressure trigger if in situ as ECG trigger will not function

b) 66-year-old male with accidental hypothermia and core temperature < 24ºC

Checking for signs of life	Likely to need monitoring e.g. Echo rather than pulse or breathing check clinically. Monitor for up to 1minute
Prolonged CPR with stiff chest wall	May need mechanical chest compression devices and rotation of team members
Defibrillation	After initial 3 shocks as standard, delay till core temp >28-30C. Minimal shocking till rewarm
Drug dosing & intervals	With hold until temp >30C then double the usual interval between drug doses
Rewarming	Consider multiple strategies: space blanket, radiant heater, warm air blower, Warmed fluid irrigation of GIT and bladder, ECMO. Ensure rewarmed before declaring death
Vascular access	Use femoral route to avoid wires irritating heart and triggering VF/VT which may be shock resistant.

c) 34-year-old 32/40 gestation pregnant female

Team composition	Obstetrician, anaesthetist, paediatrician in event of needing resuscitative hysterotomy
Resuscitative hysterotomy	Needed if No ROSC in 4min
Manual displacement of uterus to left / left lateral tilt	To avoid IVC compression and decreased venous return
Higher hand position for chest compressions	Slightly higher on chest wall than for non-pregnant state
Early Intubation	Early as possible as higher risk of aspiration and diaphragmatic splinting by gravid uterus
Defibrillation pads	May need to be placed in bi-axillary position

Examiners Comments:

Nil.

Maximum Score	8.3
Percentage Passed	72.0%

Question 4

Briefly describe the indications, post-procedure management and procedural complications of endovascular clot retrieval (ECR) for acute stroke.

ANSWER TEMPLATE

Indications: **2 Marks**

- Ischaemic stroke with proven **large-vessel occlusion** on CT Angiogram (naming vessels not necessary) (1 mark)
- Onset of symptoms up to 24 hours if CT imaging is supportive. (0.5 marks)
- Functionally independent prior to stroke (0.5 marks)

Post-procedure management: **4 Marks**

- Admission to monitored environment (e.g. stroke unit / HDU), usually intubated for procedure, so may describe extubation plan
- Observe vascular access site for complications
- Neurological monitoring for haemorrhagic transformation or further ischaemic complications
- Blood Pressure Target: control as per agreed targets
- Aspirin, 24-hours after thrombolysis and exclusion of haemorrhagic complications
- Other neuroprotective measures (BSL, Na, position, temperature, CO2 etc.)
- Oral intake after speech pathology assessment in patients with clinical neurological deficit
- Mechanical DVT prophylaxis/stress ulcer prophylaxis.
- Longer term management dependent on extent of deficit

Complications: **4 Marks**

a) Direct device-related vascular injury:
   - vessel perforation
   - symptomatic intracranial haemorrhage
   - subarachnoid haemorrhage

- arterial dissection
- emboli to new territories
- vasospasm

b) Vascular access site complications:
- dissection
- pseudoaneurysm
- retro-peritoneal Haematoma
- infection

c) Contrast-related:
- Allergy
- Renal Injury

d) Any complications related to anaesthesia

Examiners Comments:

Many candidates provided only generic responses without specific detail.

Maximum Score	7.6
Percentage Passed	32.0%

Question 5

5.1

A 51-year-old male with a history of cirrhosis secondary to Hepatitis C is admitted for the first time with haematemesis. His gastroscopy is complicated by aspiration. He is admitted to ICU ventilated.

The following results were obtained:

Parameter	**Patient Value**	**Adult Normal Range**
FiO_2	0.4	
pH	7.16*	7.35 – 7.45
pO_2	109 mmHg (14.1 kPa)	
pCO_2	29.0 mmHg (3.87 kPa)*	35.0 – 45.0 (4.60 – 6.00)
SpO_2	95%	
Bicarbonate	10.0 mmol/L*	22.0 – 26.0
Base Excess	-17.0 mmol/L*	-2.0 – +2.0
Lactate	4.5 mmol/L*	0.5 – 1.6
Sodium	144 mmol/L	135 – 145
Potassium	4.4 mmol/L	3.5 – 5.0
Chloride	114 mmol/L*	95 – 105
Glucose	11.0 mmol/L*	3.5 – 6.0

Parameter	**Patient Value**	**Adult Normal Range**
Urea	17.0 mmol/L*	3.0 – 8.0
Creatinine	110 µmol/L*	45 – 90
Albumin	23 g/L*	35 – 50
Protein	41 g/L*	60 – 80

Total bilirubin	56 µmol/L*	< 26
Aspartate transferase	67 U/L*	< 35
Alanine transferase	101 U/L*	< 35
Alkaline phosphatase	78 U/L*	30 – 110
γ-Glutamyl transferase	36 U/L	< 40
Calcium (total)	2.13 mmol/L	2.12 – 2.62

a) Interpret these results, giving likely reasons for the abnormalities. (40% marks)

5.2

The patient becomes anuric and 6 hours after commencing continuous veno-veno haemodiafiltration (CVVHDF) with citrate anticoagulation has the biochemistry results shown below:

Parameter	**Patient Value**	**Adult Normal Range**
FiO_2	0.4	
pH	7.09*	7.35 – 7.45
pO_2	89 mmHg (11.9 kPa)	
pCO_2	31.0 mmHg (4.1 kPa)*	35.0 – 45.0 (4.6 – 6.0)
SpO_2	93%	
Bicarbonate	9.0 mmol/L*	22.0 – 26.0
Base Excess	-18.0 mmol/L*	-2.0 – +2.0
Lactate	2.1 mmol/L*	0.5 – 1.6
Sodium	142 mmol/L	135 – 145
Potassium	4.4 mmol/L	3.5 – 5.0
Chloride	107 mmol/L*	95 – 105
Glucose	8.0 mmol/L*	3.5 – 6.0
Ionised calcium	0.69 mmol/L*	1.10 – 1.35
Calcium corrected	3.70 mmol/L*	2.12 – 2.62

a) Give the likely cause of the biochemical abnormality with your reasoning. What adjustments may be made to the CVVHDF? (40% marks)

5.3

A 55-year-old patient is admitted with an exacerbation of chronic liver disease. Results of an ascitic tap and serum results are listed below:

ASCITIC FLUID	
Parameter	**Patient Value**
Appearance	Clear Yellow
pH	micro-clots present, no value obtained
Red Cell Count	0 erythrocytes/µL
White Cell Count	378 leukocytes/µL
Ascitic Fluid Protein	25 g/L
Ascitic Fluid Albumin	18 g/L

Ascitic Fluid Lactic Acid Dehydrogenase (LDH)	480 U/L
Gram Stain	no organisms seen

SERUM		
Parameter	**Patient Value**	**Adult Normal Range**
Serum Protein	32 g/L*	60 – 80
Serum Albumin	23 g/L*	35 – 50
Serum Lactic Acid Dehydrogenase (LDH)	320 U/L*	120 – 250

a) List four possible underlying diagnoses. (20% marks)

ANSWER TEMPLATE

5.1 40% marks

UEs: low HCO3 indicates metabolic acidosis, elevated Urea to Cr ratio likely related to GIT bleed (other causes are dehydration, excessive diuretics, high protein diet, steroids), elevated Cr likely due to kidney injury (likely pre-renal, renal causes possible (including hepatorenal syndrome), post-renal causes less likely).

ABG: There is an increased anion gap metabolic acidosis (20).
The delta ratio is 0.57 indicating a mixed high and a normal anion gap metabolic acidosis. High anion gap component likely secondary to shock from hypovolaemia, possibly sepsis from aspiration. Normal anion gap component may reflect saline resuscitation, renal impairment. There is partial respiratory compensation (expect the CO2 to be 23=1.5xHCO3 + 8), which is likely due to mechanical ventilation. There is an increased Aa gradient, presumably because of the aspiration. The elevated lactate may represent shock, liver impairment or treatment with catecholamines. Mildly elevated glucose presumably a stress response.

LFTS: Low albumin could indicate chronic synthetic liver disease or be due to acute sepsis/SIRS or related to volume expansion with non-albumin fluids. Elevated ALT related to hepatocellular injury most likely Hep C plus/minus hypoperfusion related to the haematemesis. Elevated bilirubin likely related to chronic cirrhosis (pre-hepatic causes are possible (including transfusion), and biliary obstruction is less likely as GGT/ALP not elevated).

5.2 40% marks

Citrate accumulation is likely. Features suggesting citrate toxicity are the high anion gap metabolic acidosis, history of liver disease, reduced ionized calcium and increased Ca gap (Ca total minus iCa). The dose of citrate should be reduced (e.g. by reducing the citrate-containing filtration replacement rate or increasing the dialysate rate (which will remove citrate, there are numerous protocols prescribing rate changes). Consider changing the CVVHDF circuit to one without citrate.

5.3 20% marks

Spontaneous bacterial peritonitis
Perforated viscus
Cirrhosis
Malignancy
TB

Examiners Comments:

Nil.

Maximum Score	9.3
Percentage Passed	94.0%

Question 6

Compare and contrast the clinical manifestations, aetiology, treatment and complications of posterior reversible encephalopathy syndrome (PRES) with herpes simplex virus (HSV) encephalitis.

ANSWER TEMPLATE

	PRES	**HSV encephalitis**
Clinical manifestations (4 marks)	The symptoms of PRES evolve rapidly over hours to days Hypertension is frequent but not invariable. The hypertensive crisis may precede the neurologic syndrome by 24 hours or longer. The clinical syndrome of PRES is characterized by - headaches - altered consciousness ranges from mild somnolence to coma - visual disturbances - seizures – Seizures are often the presenting manifestation Rarely patients can have symptoms referable to the upper cervical spinal cord (limb weakness, bladder dysfunction), along with one or more of the symptoms above.	Focal neurologic findings are usually acute - <1 week in duration - and include - altered mentation and level of consciousness - focal cranial nerve deficits - hemiparesis - dysphasia, aphasia - ataxia - focal seizures The majority of patients will have one of the above symptoms plus fever
Aetiology (1 mark)	- Hypertension - Immunosuppressive therapy e.g. cyclosporine - Renal disease - Autoimmune disorders - sepsis	HSV
Management (3 marks)	- reduction in BP, especially diastolic BP to 100/110 - Discontinuation of cytotoxic - Seizure therapy - Organ failure therapy - - in the peripartum setting treat as for eclampsia	IV acyclovir
Complications (2 marks)	- ischemia - Intracranial haemorrhage - death - Neurological deficit ranging from severe to mild	-Behavioural abnormalities -death - cognitive impairment -seizures

Examiners Comments:

Nil.

Maximum Score	8.5
Percentage Passed	72.0%

Question 7

7.1

A 73-year-old female presents to the Emergency Department with breathlessness, after a minor car accident two days earlier. Since the accident, she has had pain in her left knee and chest pain on breathing and coughing. She has previously had bilateral knee replacements.

She deteriorates over 3 hours in the Emergency Department and now looks unwell. Her vital signs are as follows:

Heart rate 105 beats/minute
Blood Pressure 80/45 mmHg
Temperature 38.2ºC

An arterial blood gas analysis is performed along with other blood tests as shown below:

Parameter	**Patient Value**	**Adult Normal Range**
FiO_2	0.5	
pH	7.18*	7.35 – 7.45
pO_2	68 mmHg (9.1 kPa)	
pCO_2	42.0 mmHg (5.6 kPa)	35.0 – 45.0 (4.6 – 6.0)
SpO_2	91%	
Bicarbonate	15.0 mmol/L*	22.0 – 26.0
Base Excess	-9.2 mmol/L*	-2.0 – +2.0
Lactate	3.4 mmol/L*	0.5 – 1.6
Sodium	135 mmol/L	135 – 145
Potassium	5.0 mmol/L	3.5 – 5.0
Chloride	105 mmol/L	95 – 105
Glucose	10.1 mmol/L*	3.5 – 6.0
Urea	12.0 mmol/L*	3.0 – 8.0
Creatinine	150 µmol/L*	45 – 90
Albumin	30 g/L*	35 – 50
Total bilirubin	36 µmol/L*	< 26
Aspartate transferase	405 U/L*	< 35
Alanine transferase	336 U/L*	< 35
Alkaline phosphatase	168 U/L*	30 – 110
γ-Glutamyl transferase	198 U/L*	< 40
Ionised calcium	1.04 mmol/L*	1.10 – 1.35
CRP	186 mg/L*	< 5
Haemoglobin	114 g/L*	120 – 160
White Cell Count	1.7×10^9/L*	4.0 – 11.0
Platelet count	87×10^9/L*	150 – 350

International normalised ratio (INR)	1.4*	0.9 – 1.3
Activated partial thromboplastin ratio (APTT)	41.0 sec*	27.0 – 38.5
Fibrinogen	4.0 g/L	2.0 – 4.0

a) Interpret the arterial blood gas analysis provided on page 7. (20% marks)

b) List six differential diagnoses for her presentation. (30% marks)

7.2

You are called to the Emergency Department to see a 56-year-old female with a diagnosis of acute severe asthma.

She has been given 4 sequential salbutamol nebulisers (5 mg dose), 200 mg IV hydrocortisone, and 500 mcg of subcutaneous adrenaline with no improvement.

When you arrive in the Emergency Department, she has some inspiratory stridor, and is only able to talk in single words. She is flushed, in sinus rhythm at 125 beats/minute, and has a blood pressure of 160/90 mmHg (no paradox). She is breathing room air with an O_2 saturation of 100%.

Auscultation reveals symmetrical breath sounds. There are no signs of heart failure.

An arterial blood gas analysis shows the following results:

Parameter	**Patient Value**	**Adult Normal Range**
FiO_2	0.21	
pH	7.56*	7.35 – 7.45
pO_2	117 mmHg (15.6 kPa)	
pCO_2	16.0 mmHg (2.1 kPa)*	35.0 – 45.0 (4.6 – 6.0)
SpO_2	100%	
Bicarbonate	14.0 mmol/L*	22.0 – 26.0
Base Excess	-8.7 mmol/L*	-2.0 – +2.0
Lactate	5.2 mmol/L*	0.5 – 1.6
Sodium	141 mmol/L	135 – 145
Potassium	3.5 mmol/L	3.5 – 5.0
Chloride	112 mmol/L*	95 – 105
Glucose	11.0 mmol/L*	3.5 – 6.0
Ionised calcium	1.21 mmol/L	1.10 – 1.35

a) Interpret the arterial blood gas provided above. (20% marks)

b) What are the disturbances in physiology contributing to her breathlessness? (30% marks)

ANSWER TEMPLATE

7.1

a) Metabolic acidosis. Anion gap is mildly elevated (15) with base excess of -9, and delta ratio of 0.3 suggesting both an elevated anion gap component and a normal anion gap component.
    a. Elevated anion gap component is accounted for by increased lactate
    b. Normal anion gap component (possibly due to renal impairment, sepsis)

   Lack of respiratory compensation for acidosis (or alternatively, a co-existing respiratory acidosis).
   A-a gradient of about 240, P:F ratio 136; implies severe hypoxia.

b) Differential diagnosis for presentation:
    a. Pulmonary embolism
    b. Fat embolism syndrome
    c. Chest trauma with haemothorax
    d. Pneumothorax
    e. Chest infection due to inadequate respiratory secretion clearance in setting of chest wall pain
    f. Septic arthritis at site of knee prosthesis with sepsis and ARDS
    g. Cardiac tamponade
    h. Pulmonary contusions
    i. Cardiac contusion

7.2

a) Interpret the arterial blood gas provided above
   normal A-a gradient of 13
   severe respiratory alkalosis
   Bicarbonate is lower than would be expected for an acute process, suggesting either a co-existing acidosis, or a chronic compensation.
   Elevated lactate, slightly elevated anion gap (15)

b) What are the disturbances in physiology contributing to her breathlessness?
   hyperventilation
   probable vocal cord dysfunction syndrome
   beta-agonist toxicity (lactaemia, increased Vco_2)

Examiners Comments:

Nil.

Maximum Score	9.3
Percentage Passed	92.0%

Question 8

a) List <u>six</u> possible causes of stridor at rest in a previously well 3-year-old child. (30% marks)

b) What features elicited on history, examination and imaging would help in refining the diagnosis? (40% marks)

c) What are the indications for intubation in this situation? (30% marks)

ANSWER TEMPLATE

a) List the possible causes of stridor at rest in a previously well 3 year old child
• viral croup
• epiglottitis
• inhaled foreign body
• severe bilateral tonsillitis, meeting in the midline (e.g.: infectious mononucleosis)
• tonsillar abscess
• retropharyngeal infection/abscess
• spasmodic (recurrent allergic) croup
• allergic reaction/angio-oedema
• bacterial tracheitis
• intra-thoracic obstruction vascular rings (less likely in prev. well), peri-tracheal tumours
• diphtheria
• other congenital causes (laryngomalacia, tracheomalacia, tracheal webs etc) unlikely in this setting, no marks for these responses

b) What features elicited on history, examination and imaging would help in refining the diagnosis
1. History:
• past history including neonatal problems, previous intubation
• vaccination especially HiB
• prodrome, URTI symptoms
• choking episodes (FB)
• febrile symptoms
• cough (implies epiglottitis unlikely)

2. Examination
• (minimise disturbance to child, examine in parent's lap)
• toxicity & fever
• swallowing / drooling
• petechial rash in HiB sepsis
• inspect the throat (without instrumentation and if child cooperative), looking for tonsillar hyperplasia, uvula swelling, FB

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

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

Nil.

Maximum Score	8.0
Percentage Passed	92.0%

Question 9

A normally well 19-year-old female (65 kg) is admitted to your ICU after she had an intentional ingestion of 50 tablets of (her mother's) verapamil 180 mg (sustained release). The ingestion was 4 hours ago.

On admission, she is conscious, feels lightheaded, and has a heart rate of 40 beats/minute and a blood pressure of 90/40 mmHg.

Describe your management. Include in your answer how she is likely to deteriorate, and what general and specific therapies you would employ as her condition worsens.

ANSWER TEMPLATE

Overarching Statement
This is a significant overdose of a non-dihydropyridine CCB, which would result in both vasodilatation and decreased inotropy/chronotropy. She already has symptomatic hypotension and bradycardia, which is likely to deteriorate and be prolonged due to the sustained release preparation ingested.

Immediate resuscitation –
- early central access & likely to require intubation early
- administration of IV crystalloid bolus for hypotension
- atropine/glycopyrrolate for bradycardia
- catecholamine support (adrenaline & noradrenaline); vasopressin

Gastrointestinal decontamination-
- single dose activated charcoal (despite ingestion 4 hours ago) 1g/kg up to 50g- if deteriorating LOC would need intubation and NG insertion.
- whole bowel irrigation- recommended as SR preparation.

Early contact with **Poisons Information Centre** (or equivalent) for advice.

Lipid "sink" therapy
- IV Lipid emulsion (20% intralipid). Described in the context of lipid soluble poisons, including verapamil.
    - (Bolus: 1.5ml/kg over 2 mins, Infusion: 1.5ml/kg.hr^{-1})

Specific therapies - Simultaneous rather than stepwise therapy in this case given severity of CCB poisoning.
- ***Calcium***- 10% Calcium chloride (10-20ml via CVC, followed by 0.25mmol/kg/hr, doses not expected). Monitor serum ionized calcium
- ***Glucagon***- useful as this patient is bradycardic (increases intracellular cAMP). 1-5mg IV push, repeat up to 15mg total. Hourly infusion based on bolus dose required to achieve response to bradycardia.
- ***High Insulin Euglycaemic Therapy (HIET)*** -Has positive inotropic effects which is required in this case, overcomes relative insulin resistance created by CCBs.
  Bolus- Insulin 1unit/kg IV with dextrose 25-50g, repeated to avoid hypoglycaemia, potassium supplements
  Infusion- Insulin 1 units/kg/hr IV; titrate upwards every 30 mins until hypotension corrected or maximum does of 10 units/kg/hr reached
  Dextrose- 0.5g/kg/hr; check every 30 mins and titrate to euglycaemia
  Potassium- ongoing supplementation
- Methylene blue if unresponsive vasoplegia (need assessment of cardiac output)
- Transvenous pacing for bradycardia

- Mechanical circulatory support- V-A ECMO- maintains organ perfusion and can maintain perfusion pressure.

Marks were allocated more for specific management strategies than general resuscitation. Drug doses were not required.
Mention of Lipid Sink therapy essential to score greater than 4 marks

Examiners Comments:

Nil.

Maximum Score	8.0
Percentage Passed	62.0%

Question 10

How would you reduce the red cell transfusion requirements in an actively bleeding multiple trauma patient?

ANSWER TEMPLATE

Early recognition and identification of location of bleeding (0.5)
Early haemorrhage control with basic haemostatic measures including: (1)
-Direct pressure
-Use of staples for soft tissue bleeding e.g. scalp bleeding
-Use of tourniquets in traumatic amputations
-Avoiding scene delays
Early definitive haemorrhage control with surgery or angiographic techniques (0.5)
Avoidance of excessive crystalloid infusion. (0.5)
“Permissive hypotension” is a fluid restriction strategy that limits dilutional coagulopathy, potentially limits clot dislodgement by maintaining a SBP 80-90mmHg.
Initial RCT single centre research (Bickel 1994 NEMJ) in penetrating torso injures showed mortality benefit in delayed fluid resuscitation. Further multi centre RCT research with blunt trauma confirmed the improved mortality in the permissive hypotension group.

The controversy exists in the presence of TBI (traumatic brain injury) and Spinal cord injury (SCI) and the avoidance of secondary brain injury. Brain trauma foundation guidelines aim for an SBP >90 or CPP > 60 to prevent this. Permissive hypotension is not suitable for these patients. There is no evidence for Hb level. The TRICC trial excluded these patients (1.5)

Avoid the lethal triad of hypothermia, acidosis, and coagulopathy.(0.5 mark each)
Ensure an ionised Ca2+ > 1 mmol/l. (0.5)
Maintaining fibrinogen > 1.5 g/L. (0.5)
Maintaining platelets > 100 x 10^9 /L. (0.5)
Recognition of the presence of medications causing coagulopathy or platelet dysfunction such as aspirin, clopidogrel, warfarin or a novel oral anticoagulant. In this instance the provision of platelets, FFP or prothrombin concentrate complexes may be appropriate. (1)

Point of care testing such as thromboelastography to facilitate rapid and targeted coagulopathy correction. (1)

The use of tranexamic acid < 3 hours (CRASH2). (0.5)
Appropriate cessation of the massive bleeding protocol. (0.5)

Examiners Comments:

Nil.

Maximum Score	8.0
Percentage Passed	44.0%

Question 11

Critically evaluate the role of proton pump inhibitors to prevent upper gastrointestinal bleeding in ICU patients.

ANSWER TEMPLATE

Rationale
Upper GI bleeding can occur due to stress ulceration in critically ill patients; risk of clinically significant bleeding estimated at around 1.5% in ventilated patients taking stress ulcer prophylaxis, historically up to 15% in those without prophylaxis.

Major risk factors for GI bleeding appear to be duration of mechanical ventilation and presence of a coagulopathy, also use of steroids, past history of peptic ulcer disease [Cook NEJM 1994]
Enteral nutrition may be preventative [Marik Crit Care Med 2010]

Many studies have shown a reduction in GI bleeding with the use of prophylaxis
PPIs are very effective at treating stress ulcer-related bleeding and are the most potent medications available to prevent GI bleeding in ICU patients [Barkun AN et al Gastroenterol 2012 Apr; 107(4)] Cook et al 2013, Int Care Med 2018).

Disadvantages
Side effects of use of PPI may include increase risk of VAP, C. Difficile infection, acute interstitial nephritis, and cost (included unintended long-term use). They may have an immunosuppressive effect.

Evidence
SUP-ICU [NEJM 2018] demonstrated that PPI use compared to placebo resulted in a reduced rate of clinically important GI bleeding (2.5% vs 4.2%], NNT = 59. No difference in mortality.
PEPTIC (JAMA 2020) demonstrated that in ventilated ICU patients, PPIs were more effective at reducing GI bleeding than H2RBs. No effect on mortality, ICU LOS or C Difficile infection rate. The study had a high crossover rate.
In a subset of cardiac surgical patients, the GI bleeding rate was very low, and mortality was increased with allocation to PPI group.

REVISE trial currently underway will provide an update on the beneficial effect of PPI compared to placebo for stress ulcer prophylaxis in ICU patients.

Overall, there is a clear need to define high-risk critically ill patient sub-group that is likely to benefit from stress ulcer prophylaxis, accounting for those that receive enteral nutrition. In light of PEPTIC, it is unlikely that PPIs offer a mortality benefit over H2RBs.

Summary**: (candidates should justify their own practice; there is not currently a clear "correct" answer for this and so this serves as an example only):**
In my practice, I would only use stress ulcer prophylaxis in ICU patients who are at high risk of GI bleeding (mechanically ventilated >48 hours and either: coagulopathy; shock/MODS/high illness severity; or high dose corticosteroids)
I would use H2RB as my standard prophylaxis medication

I would use PPI as treatment for any patients with signs of GI bleeding
I would assess the ongoing need for prophylaxis daily and cease when patient is no longer high risk, including when enteral nutrition is commenced.
I would routinely cease stress ulcer prophylaxis prior to discharge to the wards

Good answers contained the following points:
The rationale for using them.
Advantages (cheap, widely available)
Potential disadvantages
A summary of the evidence. The key points would be that they do appear to lower the incidence of GI bleeding, but do not seem to reduce mortality (and some suggestion that mortality might be increased in particular populations). A grasp of what the evidence suggests was sufficient, although detailed knowledge of recent studies was awarded marks.
A summary statement.

Examiners Comments:

Nil.

Maximum Score	8.5
Percentage Passed	78.0%

Question 12

A junior trainee in distress has asked to speak to you regarding a medical error they have made that has resulted in a life-threatening adverse outcome for the patient.

Outline the key points of the initial discussion with the trainee.

ANSWER TEMPLATE

The key points that the candidate needed to cover were:

1. Facilitating the initial critical incident debrief of the Registrar and allowing him/her to vent and tell his/her version of events

2. Ensuring there is ongoing psychological and emotional support for the Registrar
    a. Give him/her the option of standing down for the rest of the shift or providing support if he/she chooses to stay
    b. Arranging a mentor within the department (e.g. SOT)
    c. Ensuring there is back-up from friends/family at home
    d. Offering professional counselling

3. Providing
    a. Open disclosure with family advice on the medico-legal process that will ensue
    b. Need for comprehensive and accurate documentation in records and factual account for registrar's own records
    c. Early contact with medical defence organisation and hospital medico-legal advisors
    d. Reporting to coroner if/when the patient dies
    e. The event will be the subject of a Root Cause Analysis by the hospital

4. Counselling with regards to future career and training

5. Arrange follow-up meeting with mentor and departmental head for next day

Examiners Comments:

A common omission from candidates' answers was failing to discuss medico legal issues and root cause analysis.

Maximum Score	8.3
Percentage Passed	66.0%

Question 13

A 45-year-old male with a history of a renal transplant 3 years ago, currently on tacrolimus, mycophenolate and prednisolone is admitted to your ICU with pulmonary infiltrates, hypoxia and worsening renal function.

a) What are the potential infectious causes of the respiratory failure? Justify what empirical antimicrobial treatment you would commence. (50% marks)

b) Describe how you would manage his immunosuppressive therapy. (50% marks)

ANSWER TEMPLATE

a) *Infectious causes: Most likely to be bacterial (e.g. Strep pneumo);* however as is immunosuppressed patient consider and cover possible additional causative agents:
   - Bacteria most likely cause
     - Risk of bronchiectasis from mycophenolate → if this may be colonised with Pseudomonas aeruginosa needing cover
     - Early commencement of broad spectrum antibiotics with Pseudomonas coverage as per local sensitivity patterns (e.g. pip-taz, meropenem)
   - Viral pneumonia
     - Esp. *influenza/RSV/adenovirus* → oseltamivir
     - *CMV* pneumonia/pneumonitis – *less common* now with prophylaxis and at 3 years post-transplant; would not routinely investigate for unless other clinical suspicion or high risk (i.e. no prophylaxis immediately after transplant, prophylaxis recently stopped, donor +ve/recipient -ve). Treat with gancyclovir
   - Fungal – *Pneumocystis jirovecii* (*PJP)* very common if not on *prophylaxis* (trimethoprim-sulfamethozazole) –if clinical suspicion treats with Bactrim; otherwise ensure continue prophylaxis even if other cause identified

b) <u>Management of anti-rejection drugs & immunosuppression</u>
   - Tacrolimus – needs *levels monitored; many interactions* with other agents including anti-microbials →; if tacrolimus continued *dose will likely need to be reduced* in renal failure
   - Consider ceasing *all anti-rejection drugs* and managing with steroids alone during septic period
   - May require stress-dose steroids if septic irrespective of other anti-rejection drug management (i.e. risk of *adrenal insufficiency* on prednisone)
   - *Risk of neutropaenia* with mycophenolate + tacrolimus – if present may need to be reduced or ceased during acute infection + *G-CSF*

Examiners Comments:

Some candidates answered only part of the question. The relevance of the immunosuppression was not appreciated in some answers and a generic list of infectious causes was given.

Maximum Score	7.0
Percentage Passed	54.0%

Question 14

A tracheo-innominate artery fistula (TIF) is a rare but life-threatening complication of tracheostomy.

a) What are the contributing factors for TIF formation? (30% marks)

b) What are the clinical features that make you suspect a TIF and how would you confirm the diagnosis? (30% marks)

c) What is your management of a TIF? (40% marks)

ANSWER TEMPLATE

a) Contributing factors

- High pressure cuff (ideally < 20mmHg
- Low tracheostomy – below the 3rd or 4th tracheal ring
- Prolonged tracheostomy duration
- Neck/chest deformity
- Anomalous/high anatomic location of innominate artery
- Prolonged use of steroids/immunosuppression
- Localised infection

b) Clinical features

- Bloody secretions/haemoptysis/haemorrhagic shock
- Sentinel bleeding occurs in approximately 50% of patients, often pulsatile
- Time frame is usually > than 48 hours since tracheostomy insertion

Confirm diagnosis

- Bronchoscopy –> bleeding from right anterior wall at site of 6/7 tracheal rings
- Angio/CTA reveal blush from artery into trachea if appropriate to perform

c) Management of TIF (*combination of call for help, resuscitate and try and stop bleeding awarded marks*)

- Call for senior assistance including ENT/Cardiac surgeon as appropriate for institution
- Activate MTP/ensure blood products available
- Secure airway and compress artery with cuff
    - Bronchoscopy to position tracheostomy (if able to advance) or replacement with ETT are acceptable
    - Adjust the depth of the tracheostomy/ETT to put cuff pressure over the bleeding site
    - Hyperinflate the cuff
- If unable to compress artery with tube cuff, position ETT distal to bleeding to secure airway and provide digital pressure through the tracheostomy opening against the sternum to try and compress
- Endovascular stenting or surgical ligation as definitive management

Examiners Comments:

This was not well answered overall with a poor knowledge of this complication demonstrated.

Maximum Score	7.5
Percentage Passed	48.0%

Question 15

(Images removed from report.)

15.1

The ECG shown on page 13 (ECG 15.1) is from a 36-year-old male patient who presented with syncope.

a) Describe the abnormalities. (20% marks)

b) What is the likely diagnosis? (10% marks)

c) What is the treatment for this condition? (5% marks)

15.2

The ECG shown on page 14 (ECG 15.2) is from a 74-year-old female admitted for monitoring after facial surgery. There is no chest pain.

a) Describe the abnormalities. (25% marks)

b) What is the underlying diagnosis? (10% marks)

15.3

The ECG shown on page 15 (ECG 15.3) is from a 47-year-old female with breast cancer who presented with shortness of breath.

a) Describe the abnormalities. (20% marks)

b) What is the likely underlying diagnosis? (10% marks)

ANSWER TEMPLATE

15.1
a) Incomplete RBBB and ST elevation in anterior leads
b) Brugada syndrome
c) AICD

15.2
a) Deep TWI anterior leads
Left axis deviation
Moderate voltage criteria for LVH
ST abnormalities
Prolonged QTc

b) Critical LAD stenosis (Wellens syndrome)

15.3
a) Low voltage QRS complexes
Electrical alternans
Tachycardia

b) Pericardial effusion

Examiners Comments:

Most candidates were not familiar with ECG signs of critical LAD stenosis.

Maximum Score	8.8
Percentage Passed	82.0%

Question 16

A 65-year-old female, with a known medical history of rheumatoid arthritis, and a left mastectomy for breast cancer treated with radiotherapy 7 years ago, presents with respiratory failure requiring intubation and mechanical ventilation. Chest X-ray reveals a large left-sided pleural effusion, into which an intercostal catheter is placed.

With regard to this patient, discuss how examination of the pleural fluid may assist in identifying the cause of this effusion.

ANSWER TEMPLATE

Appearance (1.5 marks)
Clear, straw-coloured – more likely transudate (although still may be exudate)
Blood-stained – malignancy, pulmonary infarction
Yellow/green – rheumatoid
Pus – empyema
Turbid – inflammatory exudate

Transudate vs exudate (3 marks)
Clearly some overlap, but in general terms
Transudate – cardiac failure,
Exudate – malignancy, empyema, parapneumonic, , connective tissue disease (e.g. rheumatoid), pulmonary infarction, TB

Based on biochemical analysis
Different diagnostic criteria for distinguishing e.g. Light's Criteria Rule

Light's Criteria
Exudate if at least one of the following;
Pleural fluid protein / serum protein ratio > 0.5
Pleural fluid LDH / serum protein LDH ratio > 0.6
Pleural fluid LDH > 2/3 of the upper limit of the lab's normal serum LDH

Pleural fluid pH < 7.30 - exudate, with following differentials more likely;
Malignant effusion
Complicated parapneumonic effusion or empyema
Rheumatoid pleural disease
(SLE, TB)
pH < 7.2 is predictive of empyema, and is the best marker of a complicated parapneumonic effusion (1.5 marks)

Glucose – low concentration (< 3.3 mmol/L) or pleural fluid / serum glucose ratio < 0.5 not only supports exudate, but makes following differentials more likely;

Malignant effusion
Complicated parapneumonic effusion or empyema
Rheumatoid pleural disease (glucose can be particularly low) (1.5 marks)

Microscopy (1.5 marks)
Nucleated cell counts rarely diagnostic but may be supportive (e.g. >50,000/ml usually only complicated parapneumonic effusion/empyema)
Lymphocytosis – very high lymphocyte ratio (85-95% of total nucleated cells) suggests rheumatoid, TB,

Cytology (0.5 marks)
Malignant cells – overall sensitivity of only 60% in malignant effusions. Varies with type of malignancy.

Culture/Sensitivity (0.5 marks)
Empyema

To achieve high marks candidates needed to demonstrate an understanding of how individual results point to specific diagnoses, rather than simply listing test options.

Examiners Comments:

This question was not answered well. Those who provided a good diagnostic approach to the evaluation of pleural effusion in the clinical context scored higher compared to those who provided only a list of differential diagnoses. Many candidates did not know Light's criteria differentiating exudate from transudate.

Maximum Score	7.5
Percentage Passed	54.0%

Question 17

Discuss the role of resuscitative endovascular balloon occlusion of the aorta (REBOA) in resuscitation. Include in your answer: brief description, mechanism of action, potential indications, contraindications, and complications.

ANSWER TEMPLATE

Introduction/ Description
REBOA, by inflation of balloon at specific zones of the aorta to interrupt blood flow, haemorrhage below the level of the balloon can be controlled, while augmentation of the blood pressure cranial to the balloon. It allows temporary control of non-compressible intra-abdominal bleeding in order to proceed for definitive operation.
Has been used in many locations:
Intra-operatively, in the emergency department, interventional radiology and in the field.

Mechanism of action
Provides increase in afterload similar to a balloon pump. However, there is no deflation. Downstream stops haemorrhage by occlusion of vessel. Increases MAP during this time and consequently cerebral and myocardial perfusion.

Potential Indications of REBOA
A) Non-compressible torso haemorrhage from trauma (alternative for resuscitative thoracotomy for direct clamping of aorta)

B) Management of major exsanguination e.g.
-AAA rupture,
-Post-partum haemorrhage
- Abdominal or pelvic bleeding any cause i.e. elective surgical complication

C) CPR: Non-traumatic out of hospital/prehospital cardiac arrest or medical cardiac arrest use is on exploration

Contraindication:

A) Thoracic aortic injury and/or thoracic aortic diseases e.g. aneurysm
B) Inability to obtain femoral access/peripheral vascular disease
C) Penetrating thoracic trauma
D) Not a candidate for resuscitative thoracotomy

Complications

- Prolong occlusion of the aorta results in tissue ischemia- spinal, renal
- Aortic or iliac artery injury including rupture, dissection, perforation
- Arterial thrombosis
- Compromised lower limb perfusion and ischemia. Amputation may be required
- Metabolic complications including ischemic reperfusion injury, acute kidney injury, myocardial injury, lactic acidosis

Examiners Comments:

Nil.

Maximum Score	8.0
Percentage Passed	58.0%

Question 18

Using the headings of history, examination, biochemical findings, haematological findings, imaging and biopsy, compare and contrast the clinical features and investigation findings of Acute Fulminant Hepatic Failure with Decompensated Chronic Liver Disease.

ANSWER TEMPLATE

	Acute Fulminant	**Decompensated Chronic**
History	Acute, toxic ingestion (examples) Viral Infections, Ischaemia Post-surgery	Chronic Liver disease Encephalopathy GI Bleeding
Examination	Jaundice	Jaundice
	Coma	Coma
	Intracranial hypertension common	Intracranial hypertension rare
	No signs chronic liver disease	Signs chronic liver disease
	No ascites	Ascites/oedema
	No portal hypertension	Signs of Portal hypertension
Biochemistry	Hypoglycaemia common	Hypoglycaemia rare
	Hyponatraemia rare	Hyponatraemia common
	Severe acidosis	Mild acidosis
	High Bilirubin	High Bilirubin
	LFT’s severely deranged	LFT’s mildly deranged

	Renal failure common	Renal failure less common
Haematology	Platelets usually normal	Platelets usually low
	INR very high	INR mildly raised
	Fibrinogen low	Fibrinogen normal
Imaging		
Head	Cerebral oedema	No cerebral oedema
Abdomen	Normal – may show swollen liver or vascular cause of fulminant failure	Small fibrotic/cirrhotic liver, splenomegaly, varices.
Biopsy	Necrosis	Fibrosis

Credit was given to any additional correct answers under the relevant headings.

Examiners Comments:

Nil.

Maximum Score	7.9
Percentage Passed	64.0%

Question 19

You are asked to review a 46-year-old female on the surgical ward who has stridor 4 hours after a total thyroidectomy.

a) List six possible causes for this. (30% marks)

b) You determine intubation is necessary. Outline your approach to securing her airway and justify it. (70% marks)

ANSWER TEMPLATE

Causes

- airway obstruction secondary to haematoma,
- stridor secondary to hypocalcaemia (less likely so early)
- airway obstruction due to recurrent laryngeal nerve injury – unilateral versus bilateral,
- post extubation stridor – vocal cord edema
- airway collapse secondary to tracheomalecia if long term goitre
- Anaphlaxis/any other cause examiner is happy with,

Approach:

Immediate oxygenation measures:

Administer High flow Oxygen / Hudson mask with rebreath bag at 15L. Sitting up position better tolerated than lying flat

Can try Heliox – though to be effective will need 79% Helium, 21% Oxygen mix which may be inadequate if patient has poor gas exchange / pulmonary oedema

Declare Airway emergency – get help: Surgeon, Anaesthetist, Nursing staff

Decide on location for intubation: In ward, theatre or ICU – depending on distance to be moved and patient stability

Decide on and communicate plan of airway intervention including plan for failure: Plans A,B,C highlighting each approach

Ensure Team members are aware of roles & sequence of events and emergency responses

Check Equipment for familiarity and correct function

*Be clear about pros and cons of approaches for airway management (**Must justify one**)*

- IV bolus induction – Facilitates rapid airway access but loss of control of airway possible with need for rescue using front of neck access.
- Slow IV propofol infusion induction – maintains respiratory efforts, but familiarity with propofol kinetics and pumps needed.
- Slow Gas induction – maintains respiratory efforts, but an operating theatre and familiarity with anaesthetic machine needed.
- Awake intubation – maintains respiratory efforts, uses local anaesthesia, opiate, sedation avoiding paralysis – not possible in extremis, may hinder adequate view
- Awake Fibreoptic intubation – not appropriate in stridor where scope often obstructs airway inlet in stridorous awake patient.

Outline plan for failure: then front of neck access:

- Scalpel bougie technique (landmarks palpable)
- Midline incision, finger dissect, tracheal access (landmarks impalpable)

Examiners Comments:

Nil.

Maximum Score	8.2
Percentage Passed	76.0%

Question 20

An audit has revealed your ICU has an MRSA infection rate which exceeds national benchmarks. Outline the steps you would take to improve this situation.

ANSWER TEMPLATE

Investigation/planning-

Review the audit.
Breakdown of hand hygiene audit data by groups of staff.
Observe the daily habits of the unit staff and availability of hand washing stations
Liaise with ID department

Acknowledgement/ownership

Open disclosure within the unit of the problem. Where do the staff feel the problem lies?

Education

Local champions, train staff to preform hand hygiene audits, cleaning product education, hand hygiene education. Signage for staff. 5 moments of hand hygiene, visiting teams to the ICU.
Ensure regulations re clothing, jewellery etc are being followed
Encourage all staff patients and visitors to challenge staff that do not follow unit policy

Physical cleaning

Is the cleaning in the unit adequate, consider closure and re-opening after cleaning?

Unit design and procedures

Frequency of washing or disposal of linens, curtains
Isolation of high-risk patient, or cohorting
Availability and type of hand rub
Review nursing ratios and nursing procedures

Antibiotic Stewardship
Review antibiotic usage.
Assess community rate of MRSA

KPIs and Re assessment
Audit and review that changes are improving habits

Higher marks were allocated to answers which gave a structured approach consisting of
- Identifying the problem
  Instituting change measures, which must include hand washing
  Measuring the effect of the change.

Examiners Comments:

Generally answered well; generic answers without reference to the specific issue were marked poorly.

Maximum Score	7.7
Percentage Passed	78.0%

Question 21

21.1

A 75-year-old male with chronic lymphocytic leukaemia (CLL) presents with a 4-day history of fever, headache and neck stiffness.

On examination, his temperature is 39ºC, with a Glasgow Coma Score of E3M6V4 and a positive Kernig's sign. There are no focal neurological signs.

Cerebrospinal fluid results are shown below:

Parameter	**Patient Value**	**Adult Normal Range**
Opening Pressure	24 cmH_2O	12 – 25
Total protein	1.20 g/L*	0.15 – 0.45
Glucose	1.2 mmol/L*	3.3 – 4.4
White Cell Count	970 cells/µL*	< 5
Mononuclear cells	50 cells/µL*	< 3
Polymorphonuclear cells	920 cells/µL*	0
Gram-stain	Gram-positive cocci	

a) What is the most likely diagnosis? (10% marks)

b) What is the most appropriate anti-microbial therapy? (15% marks)

21.2

A 56-year-old female with rheumatoid arthritis on methotrexate is admitted to ICU with fever and acute confusional state following a brief diarrhoeal illness. On examination, her temperature is 38.6ºC with neck-stiffness and a Glasgow Coma Score of E3M5V4. Twenty-four hours later, her blood cultures grow gram-positive bacilli.

a) What is the most likely diagnosis? (10% marks)

b) What is the most appropriate anti-microbial therapy? (15% marks)

21.3

A 40-year old male with Human Immunodeficiency Virus (HIV) infection on anti-retroviral therapy presents with a two-week history of severe headache followed by fever, malaise, vomiting and confusion. A CT scan of his brain is reported as normal.

His blood CD4 count is reported as 77 cells/µL* (normal range: > 500).

Cerebrospinal fluid examination shows the following:

Parameter	**Patient Value**	**Adult Normal Range**
Opening Pressure	35 cmH_2O*	12 – 25
Total protein	0.90 g/L*	0.15 – 0.45
Glucose	2.1 mmol/L*	3.3 – 4.4
White Cell Count	48 cells/µL*	< 5
Mononuclear cells	42 cells/µL*	< 3
Polymorphonuclear cells	6 cells/µL*	0
Gram-stain	No organisms	

a) What is the most likely diagnosis? (15% marks)

b) What is the most appropriate anti-microbial therapy for this condition? (10% marks)

21.4

A 60-year-old patient with regular heavy alcohol intake is admitted to ICU in a stuporous state after a two-week history of difficulty in walking and repeated falls. On examination, his Glasgow Coma Score is E2M4V3, with bilateral nystagmus and limited outward movement of both eyes on turning his head. There is no neck stiffness, asterixis or focal neurological deficit. A CT scan of the brain shows generalised cerebral atrophy.

a) What is the diagnosis? (15% marks)

b) What is the specific treatment for this condition? (10% marks)

ANSWER TEMPLATE

(***drug doses are given in the template for completeness, but candidates were not expected to give them, and they did not attract marks***)

21.1

1. Streptococcal Pneumoniae (pneumococcal) meningitis
2. IV Benzyl Penicillin 2.4g 4-hrly or IV Ceftriaxone 2g 12-hrly
   Or
   IV Moxifloxacin 400mg daily or IV Vancomycin (penicillin or beta-lactam allergy), to target plasma trough levels of 15-20 mg/L

21.2

1. Listeria Monocytogenes meningitis
2. IV Benzyl Penicillin 2.4g 4-hrly or IV Bactrim 160/800mg 6-hrly (penicillin-allergy)

2.1.3

1. Cryptococcus Neoformans (cryptococcal) meningitis*
2. IV Amphotericin plus Flucytosine

* Although other causes infective meningitis with mononuclear preponderance (e.g. TB) are possible; headache on presentation, CD4 count < 100, CSF white cell count <50 makes cryptococcal meningitis the most likely diagnosis.

21.4 1. Wernicke's Encephalopathy
2. IV Thiamine in high doses (500mg thrice a day x 2 days; 250mg daily thereafter) followed by IV Glucose

Examiners Comments:

Nil.

Maximum Score	8.8
Percentage Passed	84.0%

Question 22

A 48-year-old patient with Guillain Barre Syndrome who has been hospitalised for 30 days was recently re-admitted to your ICU with septic shock. He required mechanical ventilation via his tracheostomy, vasopressor treatment, and is now recovering.

a) What factors in this patient contribute to an increased risk for nosocomial infections? (30% marks)

b) How would you reduce the risk of him acquiring another nosocomial infection while in the ICU? (70% marks)

ANSWER TEMPLATE

This question had several aspects to it that required structure to cover those elements. Candidates were expected to cover elements related to the specific patient care of the individual patient but also to cover general ICU aspects in regard to infection prevention management. Detailed descriptions were not required, as long as the general elements were covered with some relevant examples. Especially important points are underlined.

A)
Recognition that long stay patient in hospital who has a tracheostomy is a high risk patient for exposure to and/or colonisation with potential resistant flora and is therefore at risk for development of nosocomial infections (3 *marks)*

a. Increased risk if higher severity of illness, significant comorbidities ,diabetes, malnutrition or immunosuppressed (all critically unwell patients at risk)
b. Previous or ongoing antibiotic treatment, specifically when complex regimens and/or prolonged duration
c. Open wounds, pressure sores
d. Invasive devices – consider timely removal if not required or change when concern of colonisation

B)
1. Prevention of specific infections in the intensive care unit (3 *marks)*

a. Ventilator association pneumonia care bundle

i. Prevention colonisation oral cavity (oral hygiene,
ii. Prevention aspiration (Nursing 30-45 degrees, subglottic aspiration, cuff pressure maintenance)
iii. Minimize duration of ventilation (minimise sedation, early mobilisation)
iv. Endotracheal and circuit care (HME, avoid routine ventilator circuit change, suction when required for secretions).

b. CLABSI (central line associated blood stream infection) prevention bundle
   i. Insertion: Equipment (including PPE and catheter selection, dressing), preparation (including site selection) and sterile technique;
   ii. Care: daily check insertion sites for signs of inflammation, daily review need (and remove when not required), check for lumen patency, hand hygiene and swab hub when handling. Consider timely removal if not required or change when concern of colonisation/infection/inflammation
   iii. Documentation
   iv. Education (staff experience)

c. Infective diarrhea
   i. Vigilance/high index of suspicion for symptoms consistent with infective diarrhea, specifically for Clostridioides difficile
   ii. Contact precautions/isolation (including hand washing) when C difficile suspected/confirmed

d. Urinary tract infection
   i. consider timely removal of catheter if not required or change when concern of colonisation

   NG tube/sinusitis- consider PEG.

2. Environmental and personal aspects (*2 marks)*
   a. Hand hygiene
   b. Aseptic or sterile technique for procedures
   c. Personal protective equipment as per unit/hospital protocol, specifically in case of multi resistant organisms (ESBL, VRE, MRSA)
   d. Environmental hygiene (bench top cleaning, disposable versus non disposable curtains)
   e. Visitor education in regard to hand hygiene

3. Antimicrobial stewardship – (some mention of appropriate antibiotic choice and de-escalation) multidisciplinary approach to provide correct treatment to patients with infections, improve outcome and to reduce risk of resistance development (*2 marks)*
   a. Regular screening for colonisation with
      i. Surveillance cultures (tracheal aspirate, urine if catheterised)
      ii. Nasal/rectal swabs if concern for multi resistant organisms (VRE, MRSA)
   b. Knowledge of local microbiological data and resistance patterns
   c. Therapeutic guidelines on empiric antibiotic treatment
   d. De-escalation, change from parenteral to enteral antibiotics, avoid long duration when possible

Examiners Comments:

Nil.

Maximum Score	7.3
Percentage Passed	60.0%

Question 23

With respect to nutritional support in the critically ill:

a) Outline how you would assess the nutritional status of a patient with suspected malnutrition. (70% marks)

b) Outline the pathophysiology of severe re-feeding syndrome. (30% marks)

ANSWER TEMPLATE

a) Assessments of nutritional status:
This is notoriously unreliable as there are many conditions that can alter the non-specific markers of nutritional status.
A good history should include the circumstances of poor intake (duration, cause, etc.), a background of previous eating behaviours, and GIT symptoms (nausea, vomiting diarrhoea, weight loss)
i. Specifics in the examination, beyond the general examination and vital signs are:
- Anthropometric
- Weight, height and BMI calculation
- Arm circumference
- Triceps skin fold thickness

ii. Clinical:
- Hair: Hair loss or abnormal distribution (lanugo),
- Skin: Conjunctival pallor and skin pallor, xerosis (dry skin, A), spooning of nails (Iron), ecchymoses or petechiae (C or K), pressure ulcers, poor wound healing
- Mouth: Glossitis (Niacin, Folate, B12, B2, B6), bleeding or sores on the gums and oral mucosa (C), angular cheilosis or stomatitis (B2, B6), leucoplakia, poor dentition
- Neck: Thyromegaly
- Extremities: loss of muscle mass (arm circumference, bitemporal wasting), loss subcutaneous fat (triceps skin thickness), bone tenderness (Vit D)
- Neurologic: Peripheral neuropathy, reflexes, tetany, mental status, handgrip strength
Investigations to assess protein status for protein calorie malnutrition, must all be taken in context of other evidence of acute and chronic illness and will alter as part of acute phase response.
Serum albumin (longest half-life at 18 – 20d)
Serum transferrin (half-life of 8 – 9d), but also reflects iron status, and low transferrin should be considered an indicator of protein status only in the setting of normal serum iron.
Serum pre albumin (half-life at 2 – 3d) - responds quickly to the onset of malnutrition and rises rapidly with adequate protein intake, but altered in the acute phase response due to acute or chronic inflammation.
Other investigations:
- Anaemia with Fe levels, or B12 / Folate if macrocytic.
- Vitamin and trace elements
- Ca, PO4, Mg, Glucose, UEC are all non-specific
- Retinol binding protein

b) Pathophysiology of Re-feeding Syndrome

Reintroduction of glucose into diet after a considerable period of fasting
Insulin in response to glucose load moves the glucose into cells (with K and Mg)
The first step of glycolysis is the phosphorylation of glucose. This holds the glucose in cells. This leads to sudden and precipitous fall in phosphate that is the hallmark of refeeding syndrome
Severely reduced phosphate is available for ATP, cAMP
Failure of tissues with high energy requirement - heart, kidney, muscle (rhabdomyolysis), brain, respiratory (diaphragm)

Examiners Comments:

Nil.

Maximum Score	8.3
Percentage Passed	82.0%

Question 24

24.1

A previously well 24-year-old male presents with fevers, malaise and jaundice. Microbiological cultures are negative, and despite treatment with broad spectrum antibiotics he continues to deteriorate. The following results are obtained:

Parameter	**Patient Value**	**Adult Normal Range**
Sodium	129 mmol/L*	135 – 145
Potassium	5.1 mmol/L*	3.5 – 5.0
Chloride	105 mmol/L	95 – 105
Bicarbonate	14.0 mmol/L*	22.0 – 26.0
Urea	16.3 mmol/L*	3.0 – 8.0
Creatinine	659 µmol/L*	45 – 90
Glucose	7.0 mmol/L*	3.5 – 6.0
Magnesium	1.49 mmol/L*	0.75 – 0.95
Albumin	27 g/L*	35 – 50
Protein	45 g/L*	60 – 80
Total bilirubin	148 µmol/L*	< 26
Conjugated bilirubin	143 µmol/L	
Aspartate transferase	2250 U/L*	< 35
Alanine transferase	1218 U/L*	< 35
Alkaline phosphatase	43 U/L	30 – 110
γ-Glutamyl transferase	68 U/L*	< 40
Ionised calcium	0.97 mmol/L*	1.10 – 1.35
Calcium corrected	1.95 mmol/L*	2.12 – 2.62
Phosphate	1.11 mmol/L	0.80 – 1.50
Creatinine Kinase	500 U/L*	55 – 170
Iron Level	34 µmol/L	6 – 35
Ferritin	181,900 µg/L*	30 – 400
Transferrin	0.6 g/L*	2.0 – 3.6
Ammonia	78 µmol/L*	16 – 60

Parameter	**Patient Value**	**Adult Normal Range**
Haemoglobin	132 g/L	120 – 160
White Cell Count	5.2 x 10^9/L	4.0 – 11.0
Platelet count	24 x 10^9/L*	150 – 350

Parameter	Patient Value	Adult Normal Range
Prothrombin time	20.0 sec*	12.0 – 16.5
International normalised ratio (INR)	1.8*	0.9 – 1.3
Activated partial thromboplastin time (APTT)	77.0 sec*	27.0 – 38.5
Fibrinogen	0.7 g/L*	2.0 – 4.0
D-Dimer	66.0 mg/L*	< 0.5

a) Interpret the abnormalities. (30% marks)

b) What is the most likely diagnosis? (10% marks)

c) What are precipitants of this condition? (10% marks)

24.2

A 40-year-old patient with a background of alcohol abuse presents with a history of 8 days of diarrhoea and vomiting.

The following results are obtained:

Parameter	Patient Value	Adult Normal Range
Sodium	116 mmol/L*	137 – 146
Potassium	2.9 mmol/L*	3.5 – 5.0
Chloride	67 mmol/L*	95 – 110
Bicarbonate	14 mmol/L*	24 – 31
Urea	2.9 mmol/L*	3.0 – 8.5
Creatinine	46 µmol/L*	60 – 120
Glucose	6.8 mmol/L	3.0 – 7.8
Osmolality	254 mOsm/kg*	274 – 295
Phosphate	0.6 mmol/L*	0.7 – 1.4
Magnesium	0.7 mmol/L	0.7 – 1.05
Calcium corrected	2.3 mmol/L	2.1 – 2.6
Albumin	44 g/L	36 – 52
Bilirubin	13 µmol/L	0 – 18
Aspartate transferase	80 U/L*	0 – 30
Alanine transferase	67 U/L*	0 – 30
Alkaline phosphatase	148 U/L*	30 – 100
γ-Glutamyl transferase	480 U/L*	0 – 35

a) What is the acid-base disturbance in this patient? (20% marks)

b) What are the likely causes in this context? (30% marks)

ANSWER TEMPLATE

24.1

a)
Interpret the abnormalities.
Mild hyponatraemia and hyperkalaemia

AKI: Rise in creatinine out of proportion to rise in urea (ratio urea: creatinine < 100:1) – due to intrinsic renal dysfunction or associated severe liver injury
Reduced bicarbonate and normal anion gap metabolic acidosis
Hyperbilirubinaemia – mostly conjugated
Acute hepatocellular liver injury: ratio AST:ALT increased but not quite 2:1 indicative of alcoholic hepatitis (also not supported by only slightly elevated GGT) but suggestive other acute liver injury
Other synthetic liver dysfunction: INR 1.8; Ammonia mildly elevated
Mildly elevated CK - ? muscle injury
Severely elevated ferritin with normal iron levels.
Normal Hb/ Wcc
Markedly deranged coagulation: severe thrombocytopaenia, low fibrinogen, markedly elevated APTT and severely elevated D-Dimer – Suggestive of DIC type pathology

b)
What is the most likely diagnosis?
Given the severity of the elevated ferritin and multiorgan involvement, Haemphagocytic lymphohistioctyosis (haemophagocytic syndrome)

c)
What are precipitants of this condition?
Viral infections particularly EBV/CMV/HSV/VZC/Parvovirus
Malignancy especially lymphoma and leukaemia
Rheumatologic conditions

24.2

a)
Metabolic acidosis- Anion gap= 116-(67+14) = 35
Delta ratio= 23/10=2.3
HAGMA with metabolic alkalosis OR Increased SID

b)
Metabolic alkalosis – due to vomiting- Acid loss and contraction alkalosis.
HAGMA-
Lactic acidosis from hypovolaemia or bowel obstruction/sepsis,
Ketoacidosis from starvation/alcohol.

Examiners Comments:

Nil.

Maximum Score	9.3
Percentage Passed	82.0%

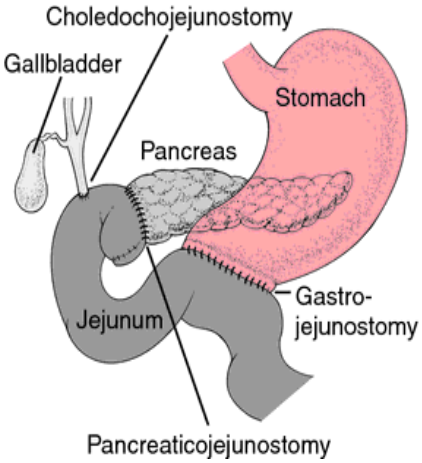
Question 25

a) With regards to open surgery for carcinoma of the head of the pancreas, list the anastomoses formed during a Whipples procedure. (30% marks)

b) List the complications of this procedure that are of relevance to its ICU management. (70% marks)

ANSWER TEMPLATE

a)
(A list of anastomoses was acceptable; the diagram is provided for clarity).



b)
List complications specific to this procedure of relevance to the Intensive Care management of patients who have had this procedure. (7 marks)

Specific early surgical complications (4 marks)
Primary haemorrhage
Pancreatic fistula
Pancreatitis
Bile leak
GI anastomotic failure
Portal or SMA thrombosis
Diabetes Mellitus
Gastric outlet obstruction

Late complications

Anastomotic stricture
Delayed gastric emptying
Pancreatic fistula
Gastric fistula
Malabsorption and electrolyte abnormalities secondary to the above

Examiners Comments:

There was poor knowledge of the anatomy of a procedure that is commonly encountered in intensive care.

Maximum Score	7.8
Percentage Passed	34.0%

Question 26

26.1

A randomised controlled trial examining a treatment for septic shock reports the following results:

"At 90 days after randomization, 27.9% patients who had been assigned to receive the treatment had died, as had 28.8% who had been assigned to receive placebo (odds ratio, 0.95; 95% confidence interval [CI], 0.82 to 1.10; P value = 0.50)."

a) Explain the meaning of the underlined terms. Interpret the result of the trial.
(40% marks)

26.2

A randomised controlled trial examining a treatment for lung injury reports the following results:

"The primary outcome was change in SOFA score over 96 hours. The mean SOFA score from baseline to 96 hours decreased from 9.8 to 6.8 in the treatment group (3 points) and from 10.3 to 6.8 in the placebo group (3.5 points) (difference, –0.10; 95% CI, −1.23 to 1.03; P = 0.86).

There were 30 prespecified secondary outcomes. Twenty-nine were not significantly different between the treatment and the placebo group. In exploratory analyses that did not adjust for multiple comparisons, day 28 mortality was 46.3% in the placebo group vs 29.8% in the treatment group (P = 0.03; between-group difference, 16.58% [95% CI, 2% to 31.1%])."

a) Interpret these results. (30% marks)

26.3

A prospective observational study examining the association between fluid therapy and outcome reports the following results:

"Crude 90-day mortality of patients who received colloids was higher than in patients treated exclusively with crystalloids; (25.5% vs. 15.4%, odds ratio (OR) 1.84, 95% confidence interval (CI) 1.56 to 2.18). After multiple logistic regression analysis, the adjusted OR was 0.923, 95% CI (0.87 to 1.19), p = 0.09."

a) Interpret these results. (30% marks)

ANSWER TEMPLATE

26.1

a)
Odds ratio: The odds of a patient in the treatment group dying within 90 days divided by the odds of patients in the placebo group dying within 90 days.

95% confidence interval: The range of values which is 95% certain to contain the population parameter of interest (in this case, Odds Ratio)

P Value: The probability of obtaining the observed, or more extreme results, assuming the null hypothesis is true. (3 marks)

The treatment tested does not reduce 90-day mortality compared to placebo (1 mark)

26.2
a)
The primary outcome does not demonstrate a significant difference between the two groups and so the overall result of the trial is negative. A secondary outcome of day 28-day mortality does show a significant difference in favour of the treatment – however as this is one of 30 secondary outcomes, with no adjustment for multiplicity of testing this is likely a false positive result and should be interpreted cautiously. (3 marks)

26.3
a)
There was a significantly higher mortality in patients who received colloids compared to those who received crystalloids. However, when other factors likely to influence mortality were taken into account by multiple logistic regression analysis, the difference was no longer statistically significant. The interpretation is that fluid choice is not significantly associated with 90-day mortality. (3 marks)

Examiners Comments:

Nil.

Maximum Score	9.8
Percentage Passed	78.0%

Question 27

Outline how the pathophysiological changes in septic shock affect the pharmacokinetics and pharmacodynamics of commonly used antimicrobials.

ANSWER TEMPLATE

The major changes in pharmacokinetic parameters of critically ill patients include alterations in volume of distribution (Vd) and clearance (Cl). Subsequently, these alterations affect the concentrations of antimicrobials in the body and the extent to which they are cleared.

The Vd is the volume in which the total amount of drug would have to be evenly distributed in to equal the same concentration as in the plasma. The toxins produced by various bacteria often lead to endothelial damage and result in increased capillary permeability. This leads to the phenomenon of “third spacing” where fluid shifts into the interstitial space from the intravascular space. These fluid shifts will increase the Vd of hydrophilic antimicrobials. Generally speaking, hydrophilic antimicrobials have a low Vd and therefore are greatly affected by these fluid shifts. Since lipophilic antimicrobials have a larger Vd, they typically distribute further into tissues and are less affected by these fluid shifts. Patients in the ICU often have hypotension as a result of septic shock, which requires the administration of fluid boluses. Additionally, heart failure and renal failure lead to more oedematous states where patients can retain large amounts of fluid. These situations also lead to increases in Vd of hydrophilic drugs.

Changes in protein binding can also have a substantial effect on the Vd, especially for drugs that are highly protein bound. Only unbound or free drug is microbiologically active. Hypoalbuminemia in critically ill patients can result in decreased binding of drugs and subsequently higher free concentrations of drugs. While free drug will distribute into tissues, critically ill patients often have

greater amounts of fluid in the interstitial space causing the antimicrobial concentrations in the tissues to remain low.

The administration of large volumes of fluid and use of vasopressors leads to a hypermetabolic state in which cardiac output and glomerular filtration rate are increased. The term often used to describe this enhanced elimination is augmented renal clearance. These physiological changes affect the clearance of drugs and can lead to sub-therapeutic levels of antimicrobials that are typically cleared by the kidneys. In contrast, decreased organ perfusion in the presence of end organ damage can lead to kidney and/or liver failure in which concentrations of these antimicrobials would be increased. Inadequate clearance or metabolism of these drugs would lead to accumulation and potential toxicity. Typically, equations such as Cockroft-Gault are used to estimate renal function; however, these are often not good predictors of renal function in critically ill patients due to the acute and rapid changes such patients often experience. Since many antimicrobials are dosed based on renal function it is even more challenging to ensure adequate doses are being administered. The most accurate way to calculate renal function is the use of 8- or 12-hour creatinine collections. In situations where renal replacement therapy is utilized, careful consideration of timing and supplemental dosing post-dialysis would be needed depending on the antimicrobial agent

Marks were awarded to answers which dealt with the following:

a. Effects of fluid shifts on volume of distribution
b. Effects on protein binding
c. Effects on organ function leading to an increase or decrease in clearance.

Examiners Comments:

Nil.

Maximum Score	6.8
Percentage Passed	52.0%

Question 28

a) What is meant by the term intermediate risk pulmonary embolism (PE) (submassive PE)? (30% marks)

b) Discuss the role of thrombolysis in patients presenting with intermediate risk PE. (70% marks)

ANSWER TEMPLATE

Latest definition according to European Society of Cardiology guideline [European Heart Journal (2020) 41, 543_603]

Intermediate Risk Pulmonary embolism can be either:
:PE without haemodynamic instability in a patient with evidence of RV dysfunction (dilatation on ECHO/CT, ECG changes, BNP) and myocardial necrosis (troponin)
or:
PE without haemodynamic instability in a patient who has one or more of the following features- age>80, cancer, Chronic heart failure, PR>110, SBP<100, SaO2< 90%. In addition, they may have either RV dysfunction or elevated cardiac troponin or none of these.

Intermediate Risk Pulmonary embolism is when PE presents without haemodynamic instability (SBP<90mmHg) but with evidence of RV dysfunction (dilatation on ECHO/CT, ECG changes, BNP) or myocardial necrosis (troponin).

Rationale for using thrombolysis (reperfusion treatment) is that it leads to faster improvement in pulmonary obstruction. However, the treatment decision needs to be balanced with the risk of life-threatening bleeding.

1) In High –Risk PE (Cardiac arrest/ Obstructive shock/persistent hypotension), thrombolysis is recommended as it reduces mortality.
2) Benefits less clear in Intermediate risk PE and thus a difficult clinical decision to make. Low mortality rate for Intermediate risk PE makes it difficult to justify the use of thrombolytic therapy in view of the risk of life-threatening bleed.
3) In Intermediate risk PE thrombolysis is hypothesised to improve functional outcomes (mainly dyspnoea) and new onset pulmonary hypertension, but there is lack of good quality evidence.

4) In Intermediate risk PE rescue thrombolysis is recommended only for patients who show signs of haemodynamic deterioration on anticoagulation therapy. All patients with intermediate risk PE should be observed in a monitored area for signs of deterioration.

5) Establishing multidisciplinary Pulmonary embolism management team may help in the decision-making process (low level evidence).

Examiners Comments:

Nil.

Maximum Score	7.7
Percentage Passed	72.0%

Question 29

In relation to cardiac surgery:

a) What are the complications of aortic cross clamping and cardiopulmonary bypass that may affect the post-operative ICU management? (80% marks)

b) What are the major risks from internal mammary artery grafting? (10% marks)

c) What are the major risks from radial artery grafting? (10% marks)

ANSWER TEMPLATE

a)
Respiratory complications:

- Left lower lobe collapse (poor re-inflation post bypass, phrenic nerve injury)
- Increased pulmonary vascular resistance (protamine)
- Acute lung injury (SIRS)
- Pulmonary oedema
- ?pneumothorax

Cardiovascular complications

- Myocardial stunning or infarction (inadequate myocardial protection)
- Coronary graft ischaemia (air embolism)
- Right ventricular dysfunction (pulmonary hypertension related to protamine)
- Hypoperfusion and end-organ ischaemia related to non-pulsatile flow and/or air/atheroma embolism from cross clamping

- Aortic dissection from cross clamping

Neurological complications
- Cerebrovascular events, watershed infarcts,
- neurocognitive dysfunction (low flow, thromboembolism)
- Phrenic nerve palsy (use of cold cardioplegia 'slush')

Renal complications
- Dysfunction related to ischaemia (non-pulsatile flow) and SIRS

Gastro-intestinal complications
- Splanchnic ischaemia (low flow, thromboembolism)
- Hepatitic dysfunction, acalculous / gangrenous cholecystitis, pancreatitis (hypoperfusion, SIRS)

Haematological complications
- Coagulopathy (effects of hypothermia and dilutional coagulopathy, residual heparinisation, activation of coagulation cascade during bypass)
- Anaemia (haemodilution, blood loss in the circuit)
- Platelet dysfunction (bypass circuit) Haemolysis (bypass circuit)
- Bleeding from aortic cannulation site

Metabolic complications
- Hypothermia (intra-operative cooling and delayed re-warming)
- Insulin resistance and hyperglycaemia (hypothermia)
- Electrolyte abnormalities (haemodilution, post-pump diuresis)

Immune-mediated complications
- Activation of coagulation cascade (blood contact with non-biological surfaces and blood-gas interface)
- SIRS (leucocyte and complement activation, cytokine release and expression of adhesion molecules stimulated by contact with bypass circuit)
- Allergic reactions to protamine

ALTERNATIVE TEMPLATE BASED ON PATHOPHYSIOLOGY

i. Effects related to blood contact with non-biologic surfaces and blood-gas interfaces
- Activation of coagulation cascade- consumptive coagulopathy, thromboembolic phenomena, haemolysis, rarely TTP.
- Systemic inflammatory response syndrome due to leucocyte and complement activation, cytokine release and expression of adhesion molecules- vasodilatory shock, fever, acute lung injury, liver dysfunction, multiorgan dysfunction.
- Platelet dysfunction

ii. Effects related to non-pulsatile flow
- Renal dysfunction
- Cerebrovascular events, watershed infarcts, neurocognitive dysfunction
- Splanchnic ischaemia

iii. Effects related to haemodilution
- Dilutional coagulopathy, anaemia.
- Electrolyte abnormalities

iv. Effects of hypothermia
- Coagulopathy
- Decreased tissue oxygen delivery
- Insulin resistance and hyperglycaemia

v. Effects of heparin and protamine
- Residual heparinisation leading to bleeding
- Increased pulmonary vascular resistance and right ventricular dysfunction from protamine, allergic reactions to protamine

vi. f) Effects related to aortic manipulation (cross-clamping and proximal grafts)
- Systemic embolisation with potential for neurologic, mesenteric and renal dysfunction.
- Aortic dissection from cannulation site
- Bleeding from bypass cannulation site
- Difficulty with myocardial protection resulting in postoperative myocardial dysfunction (especially right-sided) due to stunning or infarction

vii. g) Other
- Left phrenic nerve palsy (surgical injury, use of cold cardioplegia “slush”)
- Left lower lobe collapse (poor re-inflation post bypass, phrenic nerve injury)

b)
Artery spasm/kinking/thrombosis - resultant myocardial ischaemia/LVF
Increased risk of sternal devascularisation-> sternal non-union and infection especially with bilateral IMA grafts
Increased post-op bleeding with bilateral IMA harvesting
Aneurysm/pseudo-aneurysm of artery formation

c)
Spasm -> cardiac ischaemia
Arm complications-> haematoma/haemorrhage, infection, motor impairment (usually temporary), sensory impairment, pain, distal ischaemia (rare)

Examiners Comments:

Answers generally lacked structure and detail.

Maximum Score	6.5
Percentage Passed	22.0%

Question 30

a) List four causes and four clinical features of pseudo-bulbar palsy. (60% marks)

b) With regard to injury of the cervical spine, what are the key clinical findings that would differentiate a complete C3/4 injury from a complete C6/7 injury? (40% marks)

ANSWER TEMPLATE

a)

Causes: (any four) **(2 Marks)**
- Bilateral strokes (Internal Capsule Infarcts)
- Multiple sclerosis

- Progressive Supranuclear Palsy
- Parkinsons Disease
- Multisystem Atrophy
- Amyotrophic Lateral Sclerosis (motor neuron disease)
- High brainstem tumours
- Head Trauma

Clinical Features: (any four) **(4 Marks)**

- Facial expressions: absent (expressionless face)
- Speech: spastic dysarthria (husky, nasal voice)
- Difficulty in chewing
- Dysphagia, drooling, and nasal regurgitation
- Tongue : Spastic, pointed; Difficulty in tongue protrusion due to spasticity (No wasting/ fasciculations)
- Palatal movement: absent
- Gag reflex: brisk (exaggerated)
- Jaw jerk: exaggerated; clonic
- Emotional lability (pseudobulbar affect)

b)

What are the key clinical findings that would differentiate a complete C3/4 injury from a complete C6/7 injury? (4 marks)

	C3/4	C6/7
Motor function	○ 4 limb tetraplegia ○ Intact shoulder shrug (deltoids) ○ No power in UL or LL	○ Variable weakness of wrist flexion, elbow extension and hand function depending on the level ○ Preserved elbow flexion and shoulder girdle ○ Absent lower limb power
Reflexes	○ Absence of all reflexes in upper and lower limbs	○ Biceps intact ○ Brachioradialis may be present ○ Triceps and LL absent
Sensation	○ Sensory level at C4 (Runs just below clavicles but can extend to nipple line) ○ Absent sensation in UL and LL	○ Sensory level at C7 with preservation of sensation over forearm and radial aspect of hand (thumb side) ○ Loss or reduced sensation affecting middle finger, lateral aspect of hand and medial aspect of forearm
Respiratory	○ Phrenic nerve affected with weak diaphragm and reduced vital capacity	○ Phrenic nerve preserved, maybe mild or no reduction in VC depending on effect on other respiratory mechanics

Examiners Comments:

Answers generally lacked enough detail and were poorly structured.

Maximum Score	6.8
Percentage Passed	28.0%