



GENERAL FELLOWSHIP EXAMINATION

EXAM REPORT AUGUST / OCTOBER 2013

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

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

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

In all sections of the exam the candidate has to demonstrate performance consistent with that of a competent senior registrar / junior consultant, i.e. demonstrate 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	October 2013	May 2013	October 2012	May 2012	October 2011
Presenting for written (Including OTS)	53	27	43	41	55
Carrying a pass from a previous attempt	11	7	13	11	11
OTS Exempt	0	0	0	0	0
Total number presenting (written + carry + OTS)	64	34	55	52	66
Invited to orals (> 50% in written section)	28	18	29	26	33
Total number invited to oral section	39	25	42	37	43

Analysis of performance in individual sections	October 2013	May 2013	October 2012	May 2012	October 2011
Successful in the written section	28/53 53%	18/27 67%	29/43 67%	26/41 63%	45/55 81%
Successful in the Hot Case section	22/39 56%	9/25 36%	21/41 50%	15/37 40%	39/56 69%
Successful in both Hot Cases	10/39 26%	7/25 28%	10/41 24%	7/37 19%	22/56 39%
Successful in the Viva section	30/39 77%	15/25 60%	36/41 86%	22/37 59%	44/56 78%

Sectional pass rates	October 2013		May 2013		October 2012		May 2012	
	Pass rate	Highest individual mark	Pass rate	Highest individual mark	Pass rate	Highest individual mark	Pass rate	Highest individual mark
Hot Case 1	54%	80%	52%	75%	40%	100%	32%	80%
Hot Case 2	49%	90%	44%	90%	50%	93%	43%	77%
Viva 1	56%	70%	48%	75%	90%	90%	57%	97%
Viva 2	85%	90%	88%	81%	88%	75%	57%	75%
Viva 3	62%	90%	56%	75%	40%	80%	81%	91%
Viva 4	85%	95%	52%	76%	81%	93%	30%	60%
Viva 5	54%	80%	60%	90%	98%	95%	43%	80%
Radiology Viva	72%	100%	64%	83%	48%	90%	49%	90%
Communication Viva	49%	87%	44%	88%	67%	100%	84%	100%
Procedure Viva	79%	76%	20%	98%	71%	100%	68%	85%

Oral section pass rates	October 2013	May 2013	October 2012	May 2012	October 2011	May 2011
Candidates who scored >50% in written section and passed the overall exam	18/27 67%	11/18 61%	24/29 83%	19/26 73%	36/45 80%	17/22 77%
All candidates invited to oral section and passed the overall exam (written + carry + OTS)	28/39 72%	13/25 52%	31/42 74%	20/37 54%	43/56 76%	18/29 62%
Overall Pass Rate	28/64 44%	13/34 38%	31/56 55%	20/52 38%	43/66 65%	18/43 42%

EXAMINERS' COMMENTS

Written Paper

Fourteen of the thirty questions had an overall pass rate of less than 50%. Topics covered by questions with a pass rate of less than 40% related to data interpretation, the role of early goal directed therapy in septic shock, hypotension in pancreatitis, metabolic changes in stress and starvation and the management of iron poisoning.

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

It was noted that some answers relating to discussion or critical evaluation of a topic were not at the required level in terms of synthesis of information and depth of discussion. It also appears that candidates do not always read the questions carefully and thoroughly. Candidates are advised to include in their answer only information that is relevant to the question and to write legibly.

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 20/30 questions.

Hot Cases

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

- A tendency for candidates to have a formulaic approach to examination of the patient and a fixed routine, rather than a flexible approach that is appropriately adapted to the given situation. Candidates are discouraged from asking routine questions if they are not relevant to the case in point.

- Candidates taking either too long with the initial review of equipment, monitors, infusions, etc. before starting hands-on examination of the patient, or, at the other extreme, only performing a cursory review and missing items on the far side of the bed. Candidates are reminded that the time needed for this initial review will vary with the clinical case in question but should not unnecessarily delay examination of the patient. Candidates are strongly advised to make efficient use of the time available.
- Candidates taking an inappropriately long time on one part of the examination, leaving inadequate time for assessment of other areas. Candidates are reminded that they need to make a judgement of which aspects of the examination on which to focus and the key areas may be different for different clinical scenarios.
- Candidates not always matching the focus of the clinical examination and presentation of their findings to the clinical question posed at the start of the case.
- A tendency for some candidates to miss key clinical signs such as heart murmurs or focal neurological deficits.
- A tendency for candidates to make observations of clinical parameters without interpretation or synthesis, for example, repeating the numbers displayed on the monitor without comment on the presence/absence of haemodynamic instability, hypoxia etc.
- An inability for some candidates to confidently and appropriately answer the question, "What would *you* do?"

Candidates who performed well in the Hot Cases demonstrated the following:

- Respect and consideration for the patient.
- Competent and efficient examination technique.
- The seeking of information that was relevant to the case.
- Ability to interpret and synthesise their findings appropriately.
- Presentation of their conclusions in a systematic fashion, addressing the issue in question.
- Discussion of management issues in a mature fashion, displaying confident and competent decision-making.
- Overall performance at the expected level (competent Senior Registrar / Junior Consultant).

Candidates are advised that they should not sit the General Fellowship Examination until they can confidently examine patients, present the relevant clinical findings and discuss management issues at the appropriate level, *i.e. demonstrate that they are capable of safe, effective, independent practice as a competent Senior Registrar / Junior Consultant*. Candidates are also encouraged to practice examination of individual systems.

Vivas

As in past exams, this is the section in which candidates tend to perform well. Only one viva had an overall pass rate of less than 50% - Viva 8 (communication).

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 images from the SAQs have been removed.

PAPER 1

SAQ 1

A 76-year-old female is admitted to the ICU following elective aortic and mitral valve replacement. Transoesophageal echo assessment at the end of surgery showed an ejection fraction of 20%. Her preoperative creatinine was 340 $\mu\text{mol/L}$. Total bypass time was 240 minutes. On arrival in ICU the patient has the following indices;

Temperature	35°C
Atrial pacing (AAI)	80/min
Systemic blood pressure	85/55 mmHg
Pulmonary artery pressure	60/30 mm Hg
Cardiac index	1.5 litres.min.m ⁻²
Systemic vascular resistance	1700 dyn.sec.cm ⁻⁵
Pulmonary artery wedge pressure	10 mmHg
Central venous pressure	8 mmHg

The patient is currently on adrenaline 4 $\mu\text{g/min}$ by infusion.

- a) List the specific clinical and haemodynamic issues for this patient on admission to ICU.
- b) Outline your management of these issues.

Answer

a) The main clinical and haemodynamic issues identified are:

- Elderly female patient post double valve surgery.
- Pre-existing renal impairment.
- Long bypass time.
- Systemic hypotension (MAP 65 unlikely to be adequate for this patient).
- Low output state (CI, EF post bypass).
- Increased afterload / vascular impedance (SVR).
- Probable fluid responsiveness (PAWP, CVP).
- Moderate pulmonary hypertension.
- Low core temperature.

b) This patient is high risk (female, age, long bypass time, pre-existing renal impairment, low EF). Management consists of:

- Re-warming.
- Judicious fluid replacement as she re-warms.
- Improved volume state may augment CI but given poor EF unlikely to be sole intervention needed.
- Titration of adrenaline infusion, aiming for CI > 2.2
- Bedside echo to evaluate effect of fluid and increased adrenaline, exclude tamponade and check valve function (mitral regurgitation can increase PAP and decrease cardiac output).
- Consideration of other vasoactive agents (dobutamine, milrinone, levosimendan) or IABP insertion if persisting low output state.
- Assess adequacy of pacing and consider changing mode to A-V pacing (heart block common after AVR) and /or increasing rate to 90 bpm.
- Correct post-op coagulopathy and replace blood losses to maintain Hb > 80 G/L. Surgical review if significant blood loss via drains.
- Evaluation of any other cause of low output state e.g. tension pneumothorax, dynamic hyperinflation.
- Close monitoring of renal function and early institution of renal replacement therapy if oligo-anuric or rising creatinine.
- Consideration of inhaled nitric oxide to reduce pulmonary hypertension and RV afterload.

SAQ 2

Outline the advantages and disadvantages of the various techniques used in the diagnosis and monitoring of vasospasm secondary to aneurysmal subarachnoid haemorrhage.

Answer

Techniques that have proven or demonstrated potential in the diagnosis and monitoring of vasospasm include:

Clinical:

In the conscious patient, may be detected clinically by new focal neurology or a drop in GCS. Advantages: No additional costs and readily available, can be repeated easily, non-invasive (usually), has to be performed at the bedside. Major disadvantage is lack of specificity often necessitating CT/angiography. Also lacks sensitivity, vasospasm can occur without a clinical

correlate, early in the disease. Operator dependent.

Conventional 4 vessel DSA angiography:

- Remains the gold standard for diagnosis of vasospasm.
- May allow therapeutic intervention (angioplasty) at the time.

Disadvantages: invasive, risks of bleeding, embolism, radiation/contrast exposure and transport. Requires skilled interventional radiology, and therefore resource heavy. Risk of stroke (quoted about 1%, but probably a little lower) just from the angio, plus the dissections etc. that occur as well.

Detects vessel narrowing, not necessarily poor flow to distal tissue in all cases (either increased flow rate through narrow vessel or collateral supply. May lead to over treatment.

Transcranial Doppler (TCD):

- It is low risk, performed at the bedside, non-invasive and able to be repeated daily enabling trend analysis.
- Disadvantages:
 - The technique is however operator dependent and there is high inter-observer variability.
 - Debate exists regarding correlation of flow velocity and vasospasm and although high velocities (> 200cm/sec) are predictive, lower velocity may not be as good. - The technique may be more accurate when MCA velocity is indexed to the ipsilateral extracranial carotid artery (Lindegaard index, >3 strongly predictive).
 - Colour coded TCD may offer greater accuracy than plain TCD alone.

CTA/MRI:

May be combined with perfusion allowing characterisation of both vascular anatomy and associated perfusion abnormalities.

MR diffusion weighted imaging accurately identifies brain tissue at high risk of infarction; perfusion weighted imaging reveals asymmetries in regional perfusion.

Both methods show correlation with delayed ischaemic neurological deficit (DIND).

Disadvantages: Image clarity will be affected by clip/coil and contrast related issues need consideration. The overall diagnostic capability of this modality however remains unclear until further prospective studies are performed. Similar disadvantages as per angiography with respect to transport, radiation (for CT), contrast exposure, interpretation by experts.

SPECT/PET:

- Can be used to obtain a picture of brain perfusion and metabolism and have shown variable correlation with vasospasm as assessed by more conventional methods.

Disadvantages: They are resource heavy not easily available, radiation exposure, patient transport are issues.

EEG:

May provide prognostic information, focal areas of slowing correlate with angiographic

vasospasm and a decrease in alpha to delta ratio strongly correlates with ischaemia. Sensitivity and specificity for detecting vasospasm is high.

Disadvantage: Not readily available however and there may be issues with interpretation.

Tissue sensors:

The use of measures of tissue oxygenation using parenchymal sensors and microdialysis for monitoring biochemical indices of ischaemia are largely research tools.

SAQ 3

3.1

A 24-year-old female with a history of depression presents with seizures and decreased consciousness.

The following are her arterial blood gas analysis, taken on FiO₂ 0.3:

Parameter	Patient Value	Normal Adult Range
Barometric pressure	760 mmHg (100 kPa)	
pH	7.39	7.35 – 7.45
PCO ₂	40 mmHg (5.3 kPa)	35 – 45
PO ₂	110 mmHg (14.6 kPa)	
Bicarbonate	24 mmol/L	22 – 27
Base Excess	-0.4 mmol/L	-2 – +2
Sodium	136 mmol/L	135 – 145
Potassium	4 mmol/L	3.5 – 4.5
Chloride	118 mmol/L*	110 – 110
Glucose	4.2 mmol/L	3.0 – 7.8
Lactate	0.8 mmol/L	0.5 – 2.2

- a) What is the likely cause of her presentation?
- b) Give your reasoning.

Answer

- a) Lithium toxicity.
- b) Negative anion gap and history of depression.

3.2

A 64-year-old male has been an in-patient in your Intensive Care Unit for one week following a subarachnoid haemorrhage.

The following data were obtained from a CSF sample taken from the external ventricular drain:

Parameter	Patient Value	Normal Adult Range
Glucose	3.8 mmol/L	2.2 – 3.9
Protein	0.46 G/L	0.15 – 0.5
White Cell Count	$20 \times 10^6 / L^*$	< 5
Red Cell Count	$10\,000 \times 10^6 / L^*$	< 5

Interpret these results.

Answer

The WCC is elevated but the WCC:RCC ratio is normal (1:500) and represents normal findings after sub-arachnoid haemorrhage but does not exclude infection.

3.3

The following blood results were obtained from a 63-year-old female in the ICU. She has septic shock, coagulopathy and requires renal replacement therapy. Her condition has deteriorated in the last few hours:

Parameter	Patient Value	Normal Adult Range
Sodium	136 mmol/L	135 – 145
Potassium	4.3 mmol/L	3.2 – 4.5
Chloride	104 mmol/L	100 – 110
Bicarbonate	14 mmol/L*	22 – 27
Urea	15.0 mmol/L*	3.0 – 8.0
Creatinine	0.34 mmol/L*	0.07 – 0.12
Total Calcium	2.4 mmol/L	2.15 – 2.6
Ionised Calcium	0.9 mmol/L*	1.1 -1.3
Phosphate	1.3 mmol/L	0.7 – 1.4
Albumin	26 G/L*	33 – 47
Globulins	35 G/L	25 – 45
Total Bilirubin	35 micromol/L*	4 – 20
Conjugated Bilirubin	30 micromol/L*	1 – 4
γ -Glutamyl Transferase	120 U/L*	0 – 50
Alkaline Phosphatase	180 U/L*	40 – 110
Lactate Dehydrogenase	3800 U/L*	110 – 250
Aspartate Aminotransferase	210 U/L*	< 40
Alanine Aminotransferase	400 IU/L*	< 40

- What complication has occurred?
- Give the reasons for your answer.

Answer

- a) Citrate toxicity secondary to regional citrate anticoagulation for CRRT.
- b) Evidenced by:
- High anionic gap metabolic acidosis
 - Low ionised calcium
 - High total:ionised calcium ratio
 - Liver impairment

3.4

The following results were obtained from a 32-year-old male:

Parameter	Patient Value	Normal Adult Range
Plasma		
Sodium	138 mmol/L	135 – 145
Potassium	3.4 mmol/L	3.4 – 5.0
Chloride	118 mmol/L*	100 – 110
Bicarbonate	15 mmol/L*	22 – 27
Arterial Blood Gas		
FiO ₂	0.3	
pH	7.32*	7.35 – 7.45
PO ₂	125 mmHg (16.4 kPa)	
PCO ₂	30 mmHg (4 kPa)*	35 – 45 (4.6 – 6.0)
Base Excess	-10 mmol/L*	-2 – +2
Urine		
pH	5.0	4.6 – 8.0
Sodium	40 mmol/L	
Potassium	10 mmol/L	
Chloride	80 mmol/L	

- a) Describe the abnormalities on the blood investigations.
- b) What is the underlying mechanism for the primary abnormality?

Answer

- a)
- A-a gradient of 50.
 - Normal anion gap metabolic acidosis with appropriate respiratory compensation.
- b) Mechanism is bicarbonate loss from GI tract as urinary anion gap is negative.

SAQ 4

Describe the clinical signs and investigations available to predict poor neurological outcome in comatose survivors of cardiac arrest.

Include in your answer the factors that may confound the interpretation of these signs and investigations.

Answer

Observations and Investigations:

Clinical Signs:

- Absent brain stem reflexes.
- Myoclonic status epilepticus within the first 24 hours.
- (Generalised and repetitive myoclonus is strongly associated with poor outcome, with a reported false positive rate of 0%. Conversely, single seizures and sporadic myoclonus, do not accurately predict poor outcome.)
- Absence of pupillary responses – within days 1 to 3 after CPR.
- Absent corneal responses - within days 1 to 3 after CPR.
- Absent or extensor motor responses – after 3 days post CPR.

Electrophysiological:

EEG patterns of generalised suppression, burst suppression, or generalised periodic complexes are strongly associated with poor outcome, but the prognostic accuracy is not considered as high as SSEP.

Bilateral absence of N20 component of SSEP with median nerve stimulation within 1-3 days post CPR is strongly associated with poor outcome.

Biochemical:

Serum neuron-specific enolase levels $> 33\mu\text{g/L}$ at days 1-3 strongly associated with poor outcome.

(S100, CSF CKBB are not considered accurate enough for prognostication.)

Radiological:

Imaging may reveal catastrophic intracerebral cause for the arrest.

(Diffuse swelling on CT scan is common, but predictive power not known, role of MRI/PET also unclear.)

Confounding Factors:

Induced Hypothermia – majority of studies carried out before induced hypothermia widely used. Evidence that cooling may alter interpretation of these results, but to what extent remains unclear

Time of assessment: Period of at least 72 hours post CPR recommended. Unclear how hypothermia effects this.

CT scan done too early may not show changes

Sedatives / neuro- muscular blockers

Metabolic derangements

Presence of shock

Organ failure

Role of “self-fulfilling prophecy” in interpreting studies

SAQ 5

With reference to the reporting of clinical trials in the literature:

- a) What is a meta-analysis?
- b) What are the advantages of a meta-analysis over the interpretation of an individual study?
- c) List the features of a well-conducted meta-analysis.
- d) What is “publication bias” and how can this impact on the validity of a meta-analysis?

Answer

- a) A form of systematic review that uses statistical methods to combine the results from different studies
- b)
 - ↑ Statistical power by ↑ sample size.
 - Resolve uncertainty when studies disagree.
 - Improve estimates of effect size.
 - Inconsistency of results across studies can be quantified and analysed e.g. heterogeneity of studies, sampling error.
 - Presence of publication bias can be investigated.
 - Establish questions for future RCTs.
 - May provide information regarding generalisability of results.
- c)
 - Clearly defined research question.
 - Thorough search strategy that makes it unlikely that significant studies have been missed.
 - Reproducible and clear criteria for inclusion in the meta-analysis.
 - Adequate and reproducible assessment of the methodological quality of the included studies.
 - Use of appropriate statistical methods to assess for heterogeneity between studies and pooling of the results of studies when appropriate.
 - Utilisation of methods to ensure that the results of the meta-analysis are reproducible; e.g. two reviewers perform aspects of the study (the search, the application of the inclusion/exclusion criteria, the assessment of validity, the data extraction).
 - Assessment for the presence of publication bias/small study bias with report of the results of these analyses.
- d)
 - Publication bias is the publication or non-publication of studies depending on the direction and statistical significance of the results. A meta-analysis evaluating studies where there has been publication bias will be flawed, no matter how well conducted in other aspects.
 - Publication bias may also extend to bias of selection of studies for inclusion in a meta-analysis based on language, journal of publication, ease of access, field of research etc., (dissemination bias).

SAQ 6

Define delirium and describe your management approach to this problem in the ICU.

Answer

Definition:

The acute onset of a disturbance of consciousness with inattention, changes in cognition and/or perception, that fluctuates over time, occurs as a consequence of a general medical condition and is not better accounted for by a pre-existing, established or evolving dementia.

Management:

1. Early recognition of delirium.
2. Stabilise and ensure safety of the patient:
 - Attend to airway, breathing and circulation issues as required.
3. Consider and rule out any potentially life-threatening causes of delirium (comprehensive history and physical examination will provide clues):
 - Hypoxia
 - Hypoglycaemia
 - Intra-cerebral haemorrhage
 - Meningitis/Encephalitis
 - Poisoning
 - Wernicke's Encephalopathy
 - Infection: local (wound, anastomotic leaks etc.), UTI, lung etc.
 - Withdrawal from drugs
 - Hypertensive encephalopathy
 - Metabolic derangements – sodium, renal, liver.
4. Consider non-pharmacological strategies to control symptoms:
 - Application of hearing aids, spectacles and dentures if worn.
 - Continuous re-orientation of patient - verbal, visual with photographs etc.
 - Enlist help of family members and/or interpreters.
 - Ensure as close to normal as possible sleep/wake cycles in a quiet and calm environment.
 - Treat pain, constipation, urinary retention if present.
 - Check drug chart and beware/attend to poly-pharmacy and cessation of drugs associated with delirium.
 - Mobilise early.
 - Avoid physical restraints if safe to do so.
5. Obtain pharmacological control of symptoms if necessary:
 - Anti-psychotics are first line agents e.g. haloperidol, olanzepine, quetiapine.
 - Haloperidol 1-5mg iv q30 PRN. Dose can be doubled and repeated. Beware long QTc.
 - Benzodiazepines as first line agents for alcohol withdrawal only. Often used in combination with anti-psychotics in difficult to control symptoms in non-alcohol withdrawal delirium.
6. Treat cause if found:

- O₂, antibiotics, thiamine, glucose, electrolyte replacement, intra-venous fluids if dehydrated.

Investigations:

Directed by history, physical examination and differential diagnosis but include/consider;

Basic:

FBC, EUC, Glucose, LFT, ABG, CXR, ECG, Urinalysis + Dipstick, Septic screen, Urinary Drug Screen.

Advanced:

CT Brain, LP, TTE.

SAQ 7

7.1

A 65-year-old male has been brought into the Emergency Department after being found unconscious at home. He has a heart rate of 87 beats/min, a blood pressure of 96/59 mmHg, and temperature of 31.2°C.

Below is his biochemical profile and arterial blood gas analysis on a Hudson mask delivering 6 L/min oxygen:

Arterial Blood Gas:		
Parameter	Patient Value	Normal Adult Range
pH	7.07*	7.35 – 7.45
PaO ₂	59 mmHg (7.8 kPa)*	
PaCO ₂	25 mmHg (3.3 kPa)*	35 – 45 (4.6 – 6.0)
Bicarbonate	7 mmol/L*	22 – 26
Base Excess	-22 mmol/L*	-2 – +2
Lactate	0.8 mmol/L	< 2.0
Venous Biochemistry:		
Parameter	Patient Value	Normal Adult Range
Sodium	133 mmol/L*	135 – 150 mmol/L
Potassium	6.2 mmol/L*	3.4 – 5.0
Chloride	94 mmol/L*	100 – 110
Urea	25.9 mmol/L*	3.0 – 8.0
Creatinine	271 µmol/L*	50 – 120
Total Bilirubin	13 µmol/L	< 20
Albumin	42 G/L	35 – 50
Alanine Aminotransferase	360 U/L*	< 35
Aspartate Aminotransferase	612 U/L*	< 40
γ-Glutamyl Transferase	52 U/L*	< 40
Alkaline Phosphatase	123 U/L	35 – 135
Creatine Kinase	335 U/L*	30 – 140
Calcium (corrected)	2.65 mmol/L*	2.15 – 2.60
Magnesium	1.52 mmol/L*	0.7 – 1.10
Phosphate	3.91 mmol/L*	0.8 – 1.50
Glucose	10.5 mmol/L*	3.0 – 5.4
Ketones	6.6 mmol/L*	< 0.5

- Describe the acid-base abnormalities seen in the arterial blood gas analysis.
- List three possible causes of the ketosis.
- What is the most likely cause? Give your reasoning.

Answer

- High anion gap metabolic acidosis (ketones and other unmeasured anion).
Respiratory acidosis / inadequate respiratory compensation.
- Alcoholic ketosis.
Diabetic (euglycaemic) ketoacidosis.
Starvation ketosis.
- Alcoholic ketosis.
Combination of severe AG acidosis with high level of ketones (too high for starvation ketosis) and abnormal liver enzymes (less likely with DKA).

7.2

A 50-year-old Scottish male tourist presents with a three-day history of nausea, vomiting, general lethargy and dizziness. He had similar symptoms one year previously while on holiday in Cyprus and has had multiple presentations to his GP since then with general lethargy and weight loss.

The results of his investigations are as follows:

Parameter	Patient Value	Normal Adult Range
FiO ₂	0,3	
pH	7.29*	7.35 – 7.45
pCO ₂	22 mmHg (2.9 kPa)	35 – 45 (4.6 – 6.0)
PO ₂	108 mmHg	
SaO ₂	99%	
Bicarbonate	11 mmol/L*	22 – 26
Base Excess	-14 mmol/L*	-2 – +2
Lactate	0.8 mmol/L	< 2.0
Sodium	116 mmol/L*	135 – 150
Potassium	4.7 mmol/L	3.4 – 5.0
Chloride	89 mmol/L*	100 – 110
Urea	1.3 mmol/L*	3.0 – 8.0
Creatinine	40 µmol/L*	50 – 120
Glucose	4.8 mmol/L	3.0 – 5.4
Albumin	39 G/L	35 – 50
Calcium (corrected)	2.08 mmol/L*	2.15 – 2.64

- What is the likely diagnosis?
- What investigation would you order to confirm your diagnosis?

Answer

- Hypoadrenalism or Addisonian crisis.

- b) Random cortisol.

7.3

A 61-year-old male, due to have a colonoscopy as an out-patient, is brought into the Emergency Department on the day of the procedure having been found collapsed at home, unresponsive with increased tone in his limbs.

Parameter	Patient Value	Normal Range
Urea	3.6 mmol/L	2.1 – 7.1
Creatinine	50 micromol/L*	53 – 97
Sodium	100 mmol/L*	136 – 146
Potassium	2.9 mmol/L*	3.5 – 5.1
Chloride	62 mmol/L*	98 – 107
Bicarbonate	35 mmol/L*	22 – 32
Glucose	5.0 mmol/L	3.0 – 6.0

- a) What is the likely cause of the biochemical disturbance?
b) Briefly list the steps in your immediate management.

Answer

- a) Water intoxication secondary to bowel prep.
- b)
- Airway control and treat seizures as indicated.
 - Correct hypovolaemia.
 - Check serum osmolality (expected to be low).
 - Hypertonic saline to increase $[Na^+]$ by approx. 0.5 mmol/L/hour to achieve safe level to limit seizures (> 118 mmol/L) – balance between gradual increase in sodium and achieving safe level to limit seizures.
 - Correct hypokalaemia.
 - Fluid restriction.
 - Cease any medications that predispose to hyponatraemia (anti-depressants, thiazide diuretics, PPIs, ACEIs).
 - CT brain to assess for cerebral oedema.

SAQ 8

A two-year-old boy is suspected of ingesting iron tablets.

- a) List the clinical features, and the underlying pathophysiology, of iron poisoning.
b) Briefly outline your management of this child.

Answer

a)

Clinical Feature	Mechanism
Nausea, vomiting, diarrhoea	Direct corrosive effect on GIT
Abdominal pain	Direct corrosive effect on GIT Gut ischaemia
Shock	Disruption of cellular metabolism Fluid losses from GIT 3 rd space losses and vasodilatation
Anion gap metabolic acidosis	Disruption of cellular metabolism
Acute liver failure Jaundice, coma, low BSL, coagulopathy	Disruption of cellular metabolism Shock and hypovolaemia Hepatic necrosis
Renal failure Oliguria	Disruption of cellular metabolism Shock and hypovolaemia

b) Management consists of:

- Resuscitation as indicated with concurrent specific assessment and management of the toxidrome.
- Resuscitation:
 - ABCs
 - Priority is early restoration of circulating volume
 - Boluses of 10-20 ml/kg crystalloid and assess response
- Assessment for signs and symptoms indicative of iron toxicity.
- Risk assessment:
 - History of ingestion – type, quantity of tablets and time of ingestion
 - Iron preparations differ in the amount of elemental iron contained.
 - < 20 mg/kg elemental iron is asymptomatic
 - 20 – 60 mg/kg causes GI symptoms
 - > 60 mg/kg causes systemic toxicity
 - > 120 mg/kg is potentially lethal
- Children rarely ingest more than 60 mg/kg.
- Specific investigations
 - BSL
 - Serum iron level
 - ABG
 - AXR – useful in confirming ingestion
- Decontamination
 - Iron not absorbed to activated charcoal
 - Whole bowel irrigation indicated for confirmed ingestions > 60 mg/kg – difficult and potentially hazardous in 2-year-old
 - Surgical or endoscopic removal of tablets if lethal ingestion or WBI not feasible
- Antidotes
 - Desferrioxamine chelation therapy in cases of systemic toxicity (high serum

iron level or metabolic acidosis on ABG)

- Ongoing assessment of response to resuscitation and antidotes.
- Disposition
 - Asymptomatic at 6hr and negative AXR may be discharged home
 - Monitoring and treatment in paediatric centre (ward, HDU, ICU depending on severity)

SAQ 9

- a) List the clinical features of severe symptomatic **hypercalcaemia** and outline the treatment of this condition.
- b) List four common causes of ionised **hypocalcaemia** and for each give the underlying mechanism.

Answer

- a)
 - Clinical features:
 - CNS and PNS
 - Confusion
 - Coma
 - Hypotonia
 - Hyporeflexia
 - Paresis
 - Renal findings
 - Renal stones
 - Volume depletion
 - Renal failure
 - GI findings
 - Constipation and fecal impaction
 - Pancreatitis
 - Gastric ulcer
 - Cardiac findings
 - Arrhythmias
 - Hypotension
 - Shortened QT interval

Signs related to underlying malignancy:

Treatment:

Treatment includes reduction of hypercalcaemia and treatment of underlying cause.

Measures for reduction of hypercalcaemia- (listing of agents adequate, doses & mechanism not expected)

- Saline/frusemide diuresis- correction of dehydration with about 2L of fluid and 80mg Frusemide 2-4 hourly (only when volume has been adequately replaced) with replacement of urine losses with fluid & monitoring of potassium, calcium, phosphate and magnesium. Caution in cardiac or renal failure.

NB: Although use of frusemide is controversial/may not be beneficial, most current textbooks still include it.

- Bisphosphonates- Disodium etidronate or pamidronate
- Corticosteroids- especially if due to sarcoidosis or vit D toxicity
- Calcitonin
- Dialysis- Peritoneal or haemodialysis against calcium-free or low calcium concentration dialysate (citrate anticoagulation with CVVHDF)
- (Gallium nitrate)- nephrotoxic
- (Mithramycin)- contraindicated in renal or hepatic failure
- (Octreotide)

b)

Causes of ionised hypocalcaemia – any 4 individual causes:

- 1) Decreased PTH activity:
 - Hypoparathyroidism
 - Pseudohypoparathyroidism
 - Hypomagnesaemia
- 2) Vitamin D deficiency:
 - Malabsorption of vitamin D/calcium- steatorrhoea
 - Vitamin D deficient rickets, osteomalacia in adults
- 3) Excess calcium losses/binding:
 - Rhabdomyolysis
 - Pancreatitis
 - Critical illness (burns, sepsis, toxic shock, etc.)
 - Frusemide and saline diuresis
 - Hyperphosphataemia
 - Citrate toxicity (CVVHDF)
 - Alkalosis / Hyperventilation
 - Fluoride toxicity
 - Oxalate poisoning
 - Tumour Lysis
 - Drugs – biphosphonates, calcitonin, phenytoin
- 4) Unknown mechanism:
 - Hypermagnesaemia

SAQ 10

10.1

- a) What are the clinical features of Horner's syndrome?
- b) What additional features are associated with lateral medullary syndrome?

Answer

- a) Horner's syndrome:
 - Ptosis

- Miosis
- Anhidrosis
- Enophthalmos

b) Lateral Medullary Syndrome:

- Horner's Syndrome as well as;
- Nystagmus
- Ipsilateral V, IX and X cranial nerve lesions
- Ipsilateral cerebellar signs
- Contralateral pain and temperature (spinothalamic) loss over the trunk and limbs

10.2

A 75-year-old male is a patient in your ICU, day three following an elective oesophagectomy.

Examine the photograph provided.

- What complication has occurred?
- What is your management of this complication?

Answer

- Chylothorax secondary to thoracic duct damage.

NB: Both parts needed for whole mark.

- Conservative management with drainage, octreotide and TPN.
Surgical management with pleurodesis or thoracic duct ligation.

10.3

With reference to the image depicted below:

- What is the diagnosis?
- List three characteristic signs of your diagnosis shown in the image.
- List three abnormalities that may be seen on chest XRay in this condition.

Answer

- Rheumatoid arthritis.

b) Ulnar deviation:

- Z deformity of the thumb
- Boutonnières deformity (left little finger)
- Swan neck deformity (right middle and ring fingers)
- Swelling of metacarpo-phalangeal joints
- Wasting of small muscles of the hand

c) Pleural effusion:

- Pleural thickening.
- Nodules.

- Interstitial fibrosis.
- Bronchiectasis.
- Skeletal rheumatoid changes e.g. shoulder arthritis.
- Caplan's syndrome in patients with pneumoconiosis.

SAQ 11

A 28-year-old male has been involved in a high-speed motor vehicle crash and admitted to your hospital. His initial GCS at the scene was 5 (E2, V2, M1). He has been intubated and has a hard collar in place.

- Outline your approach to clearing the cervical spine in this man. Justify your answer.
- List the potential problems associated with the inability to clear the cervical spine at an early stage.

Answer

- The patient is sedated and so the cervical spine cannot be cleared clinically so will keep collar in place. Also check correct size and fitting.
 - Radiological clearance
 - Plain C-spine films are no longer suggested as routine part of trauma series but fractures on CXR and pelvic XR associated with increased risk of C-spine injury
 - High resolution 64 slice helical CT of the entire cervical spine and T1 with sagittal and coronal reconstructions
 - Review with radiologist
 - With technically adequate studies and experienced interpretation, the combination of multi-slice helical CT with reconstruction CT scanning provides a false negative rate of < 0.1%
 - Clear radiologically and if low risk for ligamentous injury and patient unlikely to be extubated in 24-48 hr., remove collar.

Or:

If no bony injury but need to exclude ligamentous injury, perform MRI.

There is no 100% accurate method to exclude C-spine injury and management is a balance of risk-benefit for that individual. In some cases clearing the C-spine early may not be possible and leaving the collar in situ is a balance between management of potentially "unstable" C-spine and the risk of complications from the collar.

- Prolonged immobilization is associated with significant morbidity
 - Decubitus ulceration (especially related to cervical collar)
 - Increased need for sedation
 - Delayed weaning from respiratory support
 - Delays in percutaneous tracheostomy
 - Central venous access difficulties
 - Enteral feeding intolerance due to supine positioning
 - Pulmonary aspiration due to supine positioning
 - DVT due to prolongation of immobility
 - Increased risk of cross-infection due to extra staff / equipment involved in position

SAQ 12

A 67-year-old female has presented acutely with a diagnosis of tetanus. She sustained a laceration one week earlier while gardening and has now developed generalised spasms and respiratory distress.

Outline your specific management of this patient including management of the anticipated complications of tetanus.

Answer

This case is consistent with a diagnosis of severe generalized tetanus.

Management comprises:

- Airway management with intubation and mechanical ventilation. Respiratory distress is most likely due to involvement of muscles of respiration and/or laryngospasm but pneumonia should be looked for and treated. Early tracheostomy may be indicated.

Neutralisation of unbound toxin

- Human tetanus immune globulin 3000-6000 units IM (some authorities advocate 500 units).

Source control and limitation of toxin production

- Debridement and cleaning of wound
- Appropriate antibiotics for 7-10 days – penicillin, 3rd generation cephalosporins (nb both GABA antagonists which may aggravate symptoms), metronidazole, erythromycin, doxycycline

Control of spasms

- Sedation with benzodiazepines +/- neuromuscular blockers. Intrathecal baclofen has been used. Avoid stimulation

Management of autonomic dysfunction

- Cause of death if respiratory failure avoided by intubation and ventilation
- Magnesium sulphate has been shown to be effective and labetalol (dual alpha and beta blocker) has also been used.
- Clonidine may be useful.

Initiation of full active tetanus immunization (with diphtheria and pertussis) given at site separate from TIG injection.

SAQ 13

13.1

A 57-year-old female has the following haematological and coagulation profile post admission to the ICU after a laparotomy for intra-abdominal sepsis with significant blood loss.

Parameter	Patient Value	Normal Adult Range
Haemoglobin	65 G/L*	115 – 165
White cell count	$2.77 \times 10^9/L^*$	3.5 – 11.0
Platelets	$14 \times 10^9/L$	150 – 400
Prothrombin Time	28.9 seconds*	12.0 – 15.0
International Normalised Ratio	2.7*	0.8 – 1.1
Activated Partial Thromboplastin Time	122.5 seconds*	25.0 – 37.0
Fibrinogen	1.1 G/L*	2.2 – 4.3

- List two likely causes of the coagulation abnormalities.
- State how you would correct the coagulopathy and give your reasoning.

Answer

- Haemodilution with inadequate replacement of blood and clotting factors DIC.
- Ensure patient is normothermic and correct acidosis
 - Platelets to increase platelet count
 - FFP to replace factors II, V, VII, IX, X, and XI.
 - Cryoprecipitate to replace factor VIII, and fibrinogen if FFP does not reverse INR.
 - Consider tranexamic acid and/or Activated Factor 7
 - Exclude on-going surgical haemorrhage

13.2

A 44-year-old male presents with dyspnoea and is diagnosed as having multiple pulmonary emboli on a computerised tomography pulmonary angiogram (CTPA). He is commenced on 1000 units of heparin per hour IVI after a 5000 unit intravenous bolus. During the night his heparin infusion has steadily increased to 1500 units per hour.

These blood results are from the following morning:

Parameter	Patient Value	Normal Adult Range
Prothrombin Time	12 seconds	12 – 16
Activated Partial Thromboplastin Time	38.3 seconds*	25.0 – 37.0
Fibrinogen	3.8 G/L	2.2 – 4.3
D-Dimer	$> 20.0 \mu g/ml^*$	< 0.5

- Give two reasons for the relatively low APTT despite heparin therapy.
- List four causes for an increased predisposition to venous thromboembolic disease.

Answer

a)

- ATIII deficiency
- Increased heparin clearance
- Increased heparin binding proteins
- Technical problems such as drug preparation error, disconnected IV line, pump problem, extravasated IV cannula

b)

- Protein C def Protein S def
- AT III def
- Malignancy
- Factor V Leiden Lupus anticoagulant
- Immobility
- Smoking
- Cardiac failure
- Local venous obstruction
- Surgery
- Trauma
- Obesity

13.3

A 52-year-old female was admitted the previous night with an altered level of consciousness that improved rapidly with administration of glucose.

She is referred to ICU the next day with confusion, ataxia and a worsening level of consciousness. Her CT head is normal.

The blood sugar level in the morning is 8 mmol/L on a 5% Dextrose infusion at 80 mL/hr. Her full blood count from the previous night is available as follows:

Parameter	Patient Value	Normal Adult Range
Haemoglobin	88 G/L*	130-180
White Cell Count	7.4 x 10 ⁹ /L	4.5 – 11
Platelets	88 x 10 ⁹ /L*	150 – 400
Mean Cell Volume	110 fL*	80 – 98
Mean Cell Haemoglobin	30 Pg	27 – 33
Mean Cell HAemoglobin Concentration	320 G/L	310 – 360
Prothrombin Time	12 seconds	12 – 18
Activated Partial Thromboplastin Time	36 seconds	32 – 38

- a) What is the likely cause of her confusional state?
- b) What specific treatment would you prescribe for this?

Answer

- a) Wernicke's encephalopathy.
- b) Thiamine 100 mg IV daily.

SAQ 14

Critically evaluate the use of selective decontamination of the digestive tract (SDD) in the ICU.

Answer

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 orabase and enteral antibiotics (eg polymixin B, tobramycin and amphotericin)
- Parenteral antibiotic eg 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 (eg Staph aureus, E coli and C albicans) and opportunistic, aerobic Gram-negative bacilli (eg 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 groups studied include general ICU, burns, gastrointestinal surgery and transplant 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

SAQ 15

15.1

For each of the microbes listed below, list the most appropriate antibiotic(s) for treatment of infection resulting from these organisms:

- Candida glabrata
- Clostridium perfringens
- Listeria monocytogenes
- Neisseria meningitides
- Multi-resistant Acinetobacter
- Nocardia
- Penicillin-intermediate pneumococcus
- Vancomycin-resistant enterococcus

Answer

Organism	Agent
Candida glabrata	Voriconazole or caspafungin or Amphotericin B
Clostridium perfringens	Penicillin or Meropenem or Metronidazole
Listeria monocytogenes	Penicillin or Ampicillin
Neisseria meningitides	Ceftriaxone or Penicillin (high dose)
Multi-resistant Acinetobacter	Amikacin. Polymixins
Nocardia	Sulphonamides
Penicillin-intermediate pneumococcus	Ceftriaxone or Vancomycin
Vancomycin-resistant enterococcus	Linezolid or Daptomycin

15.2

Briefly outline the dosing adjustment and the monitoring necessary for each of the following drug groups in patients with established septic shock and moderate to severe renal dysfunction (without dialysis):

- a) Aminoglycosides
- b) Fluoroquinolones
- c) Beta-Lactams
- d) Carbapenems
- e) Glycopeptides

Answer

- a) Aminoglycosides
 - High initial dose and monitor trough concentrations. Extend interval. May be necessary to decrease dose and monitor with MIC data
- b) Fluoroquinolones
 - Reduce frequency but maintain dose. Monitor QT interval
- c) Beta Lactams
 - Can reduce dose OR frequency Monitoring unnecessary
- d) Carbapenems
 - As for Beta Lactams
- e) Glycopeptides
 - High dosing on day one dose adjustments according to Cmin and dependent on degree of renal dysfunction

SAQ 16

Critically evaluate the role of Early Goal Directed Therapy (EGDT) in septic shock.

Answer

EGDT definition:

Within 6 hours of presentation to the Emergency Department intensive monitoring of specific circulatory parameters with the aggressive management of these parameters to specified targets:

Parameters

1. CVP \geq 8-12 mmHg
2. MAP 65 – 90 mmHg
3. Urine output \geq 0.5 ml/kg/hr
4. Mixed venous oxygen saturation \geq 65% / ScvO₂ \geq 70%
5. Haematocrit \geq 30%

Interventions

1. Reduce work of breathing by early use of mechanical ventilation

2. Fluid resuscitation
3. Use of vasoactive medication
4. Transfusion

Rationale:

The principle of applying EGDT for septic shock is based on the observations that:

- Early treatment for Myocardial Infarction, Acute Ischaemic Stroke and
- Trauma improves patient outcomes.
- Patients presenting to ED with sepsis have measurable O₂ deficit
- Evidenced by high lactate and high ScvO₂.
- For septic shock the hypothesis is that early optimization of the compromised
- Septic circulation may reduce mortality.

Evidence:

The evidence for the intervention is based on an American, single-centre RCT (Rivers 2001) and a recent Chinese multicenter study supporting EGDT - Surviving Sepsis Guidelines: Grade 1C (inconsistent results, well done observational studies/control RCTs) recommendation

Limitations of Rivers study include the following:

Study population limited to ED presentations and did not include ward patients

Single centre

Non-blinded

Control group had an above-average mortality rate

Unclear which interventions are most important – whole EGDT protocol or one single component

Target parameters are restrictive

Use of ScvO₂ and pressure monitoring has not been tested in the target population

Transfusion target to improve DO₂ contradicts restrictive transfusion practice and may be associated with increased mortality in the critically ill

Results of ANZ ARISE and related international studies (ProCESS and ProMISE)

Awaited

Adverse effects:

Protocols for implementing EGDT usually result in more fluid being administered, more use of vasoactive medication and more use of blood transfusion.

Therefore potential adverse effects relate to:

1. Fluid overload
2. Arrhythmias
3. Adverse effects of blood transfusion

Proscriptive targets may not suit all (eg higher MAP needed for elderly patients, lower MAP and Hct targets for young, fit patients).

Statement of Candidate's Own Practice:

Summary statement including any reasonable strategy.

SAQ 17

List the possible reasons why a patient with septic shock from infected pancreatitis may have ongoing hypotension despite intravenous fluid therapy, antibiotics and escalating inotrope requirement.

Answer

Primary problem not fixed:

Untreated focus of infection/ inadequate primary source control e.g. pancreatic abscess, infected pseudocyst.

New septic site e.g. central line/ hospital acquired pneumonia / cholecystitis, urinary tract.

Systematic approach i.e. Hypovolaemic / obstructive / cardiogenic / distributive +/- endocrine etc.

- Hypovolaemia or hidden bleeding
- E.g. From surgical site/ peptic ulcer, “third space” losses (e.g. ascites from peritonitis)
- Undiagnosed or new “obstructive shock”:
- Tension pneumothorax / Pericardial effusion / gas trapping (auto PEEP) / pleural effusions / pulmonary emboli
- Severe Intra abdominal hypertension
- Dysrhythmia e.g. SVT, junctional rhythm etc.
- New myocardial ischaemia
- New/ undiagnosed cardiac valve pathology
- Severe adrenal / pituitary / thyroid dysfunction.
- Drug reaction / anaphylaxis
- Electrolyte abnormalities such as hypophosphataemia and hypocalcaemia (the latter particularly with pancreatitis)

Technical:

CVL fallen out or not in a central vein / no pressors in the infusion bag

Measurement error – e.g. arterial line not zeroed/under or over damped, transducer height, wrong NIBP cuff size etc.

Miscellaneous:

Radial / central arterial monitoring discrepancy with severe vasoconstriction

Upper limb vascular disease (radial arterial line) or obstruction (e.g. dissection or aorto-occlusive disease: femoral arterial line)

Anti hypertensive drugs taken as part of patients usual medications

SAQ 18

18.1

A 62-year-old female is brought into hospital with suspected organophosphate poisoning.

- a) List six acute clinical features associated with this condition.
- b) List the antidotes indicated in this condition and the rationale for their use.

The following data are taken from this patient:

Parameter	Patient Value	Normal Adult Range
Cholinesterase	0.3 KU/L*	3.4 – 9.0
Cholinesterase mixing	33%*	100%

c) What does the result of the mixing test indicate?

Answer

a)

- Diarrhoea
- Urination
- Miosis
- Bronchospasm
- Bronchorrhoea
- Emesis
- Lachrymation
- Salivation
- Fasciculations
- Tremor
- Weakness
- Respiratory muscle weakness
- Bradycardia (tachycardia may be present)
- Hypotension
- Agitation
- Coma
- Seizures

b)

- Atropine to control clinical features of cholinergic excess – anti-muscarinic. Large doses may be required
- Pralidoxime to reactivate acetyl choline esterase – only effective before irreversible binding or “ageing” takes place

c)

- This mixing test is suggestive of free organophosphate present in the blood OR inadequate dose of pralidoxime.

18.2

A 19-year-old male with a history of substance abuse presents to the Emergency Department with respiratory distress.

Parameter	Patient Value	Normal Adult Range
FiO ₂	0.4	
pH	6.94*	7.35 – 7.45
PO ₂	140 mmHg (18.4 kPa)	
PCO ₂	17* mmHg (2.2 kPa)	35 – 45 (4.6 – 6.0)
HCO ₃	4* mmol/L	22 – 27
Base Excess	-28 mmol/L*	-2.0 – +2.0
Sodium	127* mmol/L	135 – 145
Chloride	113* mmol/L	95 – 105
Potassium	3.9 mmol/L	3.5 – 5.0
Urine pH	7.2	4.6 – 8.0

- a) Describe the acid-base disturbance.
- b) What is the likely cause of the acid-base disturbance?

Answer

- a) Normal anion gap severe metabolic acidosis with incomplete compensation.
- b) Renal tubular acidosis Type 1 distal secondary to chronic toluene abuse.

18.3

The following blood results are from a 78-year-old female with Type 2 diabetes and chronic renal failure presenting with breathlessness. Her GP has been treating her with flucloxacillin for cellulitis of her lower limbs.

Parameter	Patient Value	Normal Adult Range
Urea	15.3 mmol/L*	3 – 8
Creatinine	309 µmol/L*	45 – 90
Sodium	139 mmol/L	134 – 146
Potassium	4.4 mmol/L	3.4 – 5.0
Chloride	115 mmol/L*	100 – 110
Glucose	12.1 mmol/L*	3.0 – 5.4
pH	7.11*	7.35 – 7.45
PCO ₂	13 mmHg (1.7 kPa)*	35 – 45 (4.6 – 6.0)
Bicarbonate	4 mmol/L*	22 – 27
Base Excess	-24 mmol/L*	-2 – +2
Lactate	0.6 mmol/L	< 2.0
Measured osmolality	309 mOsm/L*	280 – 300

- a) Describe the acid-base abnormalities in the above results.
- b) List three possible causes for this biochemical disturbance.

Answer

- a) Severe compensated metabolic acidosis with a raised anion gap (! 20), normal osmolar gap and " gap 0.4 (" gap suggests mixed AG and NAG MA or renal failure)
- b) Possible causes
 - DKA
 - Renal failure
 - Pyroglutamic acidosis

SAQ 19

Outline how you would plan the ICU response to an influenza epidemic, including in your answer how you would increase resources.

Answer

Activate ICU/Hospital pandemic plan, if available.

Liaison / pandemic planning with other departments within the hospital, ambulance services, ICUs of other hospitals and state department of health.

Surveillance & early detection of influenza patients.

Increase ICU bed capacity.

Increase ICU healthcare staffing levels.

Anticipated need for ICU equipment – identify where additional equipment can be resourced (ED, OR etc.)

Infection control measures to reduce the spread to other patients and ICU staff.

Provision of antiviral prophylaxis / virus vaccine (if becomes available) for the staff.

Establish real-time communication link with laboratory and healthcare administration.

Increased ICU bed capacity:

- Opening additional beds in existing non-commissioned physical critical care bed spaces.
- Defer elective surgery requiring post-operative ICU/HDU care.
- Progressively convert HDU beds to Intensive Care
- Identify potential additional capacity for ICU ventilated beds in alternative clinical areas such as recovery, CCU, peri-operative units and respiratory units.
- Discharge of suitable patients to other ward areas (with appropriate upgrade in medical/nursing support)
- Maximise the use of non-ventilatory strategies in care of ICU patients freeing up devices and equipment for patients for whom mechanical ventilation is essential
- Facilitate end-of-life discussions and decisions in those appropriate ICU patients assessed as not reaching a meaningful recovery
- Increase threshold for referral of patients for ICU from other hospitals
- Consider using available private hospital ICU capacity.

Increased staffing:

- Increase nursing staff shift length (e.g. 8 to 12 hour shifts)
- Expansion of nursing capacity by increasing casual, agency or bank staff support
- Cancellation of leave for medical and nursing staff
- Provision of anti-viral prophylaxis and virus vaccine (if becomes available) to staff to reduce staff absenteeism due to sickness
- Train staff from other non-ICU monitored areas to provide intensive care
- Secondment of additional medical staff from elective duties (e.g. anaesthesia)
- Change in nurse:patient ratio to provide intensive care
- Allocation of pregnant / immuno-compromised staff to "non-flu" patients
- Train staff in the use of PPE

SAQ 20

A 53-year-old male presents following a motor vehicle accident. He complains of severe abdominal pain but has no chest or long bone injuries.

He has previously had a mechanical mitral valve replacement. His medications include warfarin.

The following image is a slice from his CT body scan.

- a) List the abnormalities on the CT scan image.
- b) Outline the advantages and disadvantages of CT scanning in the assessment of blunt abdominal trauma.
- c) Outline your immediate management of his coagulation state.

Answer

- a)
- Ruptured liver
 - Free intraperitoneal fluid (blood) +++
- b) Advantages: non-invasive; ability to exclude retroperitoneal injuries; ability to grade solid organ injury; shows where the intra-abdominal blood is coming from; may reveal associated pelvic and spinal injuries; ability to detect clinically unsuspected injuries

Limitations: radiation dose; the need to give intravenous contrast; relatively poor sensitivity for hollow viscus, mesenteric, retroperitoneal, and diaphragmatic injuries; difficulty getting an unstable patient to the CT scanner.

- c)
- Competing interests of life-threatening haemorrhage and need for anticoagulation (MVR) and in this instance haemorrhage is greater risk
Cease warfarin therapy and give:
Vitamin K 5 – 10 mg IV (recommended in Australian guidelines but controversial as may cause resistance when warfarin needs re-starting. Balance of risks and lower dose may be preferable)
AND
Prothrombin complex concentrate (Prothrombinex-VF) 50 IU/kg
AND
Fresh frozen plasma 150 – 300 ml
OR If PCC not available
FFP 15 ml/kg
Tranexamic acid as soon as possible
Other blood products (packed cells, platelets, cryoprecipitate) as indicated
Titrate therapy against measurement of coagulopathy (APTT, PT, fibrinogen, platelets)
TEG if available
Prevent / correct hypothermia, acidosis, hypocalcaemia

SAQ 21

21.1

The following image is a snapshot of ventilator graphics for an 80kg patient in the ICU intubated and mechanically ventilated.

- a) Describe the abnormalities displayed in this image.
- b) What changes (if any) would you make to the ventilator settings?
- c) Give three possible causes for the appearance of the pattern demonstrated by the pressure time curve.

Answer

- a) Resistance to inspiratory flow - peak inspiratory pressure approx. 40 cmH₂O and plateau pressure less than 20 – high peak to plateau pressure
Prolonged expiration with low peak flow indicative of expiratory resistance

Expiratory flow not returned to baseline at end of expiration indicating auto-PEEP / gas trapping / dynamic hyperinflation

b)

- Reduce rate
- Reduce T insp
- Reduce VT
- (Check intrinsic PEEP)

c)

- Kinked tubing / blocked filter
- Kinked / blocked ETT
- Asthma

21.2

A 54-year-old female with scleroderma and worsening dyspnoea on exertion presents with the following respiratory function tests:

Test	Actual	Predicted
FEV1	1.96 litres	2.66 litres
FVC	2.52 litres	3.11 litres
FEV1/FVC	78%	85%
PEF	7.50 L/sec	6.47 L/sec
FRC	2.18 litres	2.77 litres
RV	1.08 litres	1.84 litres
TLC	3.64 litres	5.17 litres
DLco	10.4 ml/min/mmHg	24.7 ml/min/mmHg
KCO (DICO/VA)	2.85 ml/min/mmHg	4.77 1/min/mmHg

- a) Describe and explain the results of the respiratory function tests.
- b) Suggest a possible cause.

Answer

a)

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

b)

- Pulmonary fibrosis.

SAQ 22

Critically evaluate the role of vasopressin in septic shock.

Answer

Introductory statement:

Vasopressors have a role in septic shock to offset hypotension caused by vasoplegia. Vasopressin is an endogenous neuroendocrine peptide that acts on multiple receptors with multiple effects including potent vasoconstriction

Rationale for use of vasopressin in septic shock:

Low levels of vasopressin have been demonstrated in patients with septic shock (compared to cardiogenic shock).
Infusion of vasopressin reduces the need for other vasoactive medication and increases both urine output and creatinine clearance.
In septic shock, exogenous vasopressin appears to act preferentially on V1 receptors on the smooth muscle of the vasculature rather than the renal V2 receptors.
Patients with septic shock may be relatively catecholamine resistant and not respond well to nor-adrenaline and potentially respond better to vasoactive agents acting at different receptors.

Evidence:

The largest study to date is an International RCT (VASST)
Patients: Septic shock on low dose nor-adrenaline infusion
Intervention: Vasopressin vs. Comparator: Noradrenaline (Other doses of open-label vasopressors given according to clinical indication)
Outcome: No effect on the primary outcome of 28-day mortality. Subgroup of those with less severe shock appeared to benefit.

Potential adverse effects:

Ischaemia: Cardiac/Gastrointestinal/Cutaneous
Reduction in cardiac output
Liver function abnormalities
Platelet dysfunction

Opinion:

1. Vasopressin may benefit patients when it is started early, but the subgroup benefit may be a chance finding and needs to be interpreted with caution.
2. Higher doses of vasopressin (fixed dose infusion used in the studies) may be needed for patients with more severe shock.
3. Either way there is a clear need to monitor closely for adverse effects if vasopressin is used for septic shock.

SAQ 23

A 37-year-old previously healthy man was admitted to your ICU five days ago after a motor vehicle crash with chest and abdominal injuries. He is currently intubated and ventilated, with FIO₂ 1.0 and PEEP 10 cmH₂O. He is deeply sedated and on noradrenaline and adrenaline infusions at 10mcg/min each. He has become oliguric.

His blood biochemistry, haematology and arterial blood gases are as follows:

Venous Biochemistry		
Test	Value	Normal Adult Range
Sodium	138 mmol/L	135 – 145
Potassium	7.1 mmol/L*	3.5 – 4.5
Chloride	104 mmol/L	95 – 105
Urea	27 mmol/L*	2.9 – 8.2
Creatinine	260 µmol/L*	70 – 120

Haematology		
Test	Value	Normal Adult Range
Haemoglobin	120 G/L*	135 -180
White Blood Cells	12.8 x 10 ⁹ /L*	4.0 -11.0
Platelets	42 x 10 ⁹ /L*	140 - 400

Arterial Blood Gases		
Test	Value	Normal Adult Range
pH	7.01*	7.35 – 7.45
PCO ₂	45 mmHg (6 kPa)	35 – 45 (4.6 – 6.0)
PO ₂	70 mm Hg (9.3 kPa)*	
Bicarbonate	11 mmol/L*	22 - 26
Base Excess	-19 mmol/L*	-2.0 – + 2.0
Glucose	7.5 mmol/L*	4 – 6
Lactate	13 mmol/L*	< 2.0

- Summarise the findings of the blood tests.
- List the likely causes of the raised lactate.
- Briefly outline your management priorities for this man.

Answer

- High anion gap metabolic acidosis Note AG 33 - NOT adequately explained just by a lactate of 13 mmol. Delta ratio approx. 2
 - Inadequate or inappropriate respiratory compensation
 - Hypoxaemia (P/F ratio 70)
 - Acute renal failure
 - Hyperkalaemia,
 - Thrombocytopenia
 - Anaemia
 - Leukocytosis
 - Mild hyperglycaemia (? Stress-induced)
- Sepsis with shock
 - Ongoing hypovolaemia
 - Hypoperfusion eg septic cardiomyopathy; abdominal compartment syndrome
 - Possible gut ischemia
 - Perhaps adrenaline (also seen with other catecholamines – unpredictable)

- d) Management Priorities should encompass both immediate resuscitation and investigation for the cause of the abnormalities.

Respiratory:

Clinical examination and CXR looking for cause of hypoxia – consider lung contusion, haemothorax/pneumothorax with shock, ARDS secondary to other process. Institute ARDS ventilation strategy if appropriate.

Cardiovascular:

Clinical examination and further investigations to determine cause of inotrope and vasopressor requirement. Consider ECHO. Fluid resuscitation if hypovolaemia suggested by examination /ECHO findings.
Cease adrenaline if possible.

Renal

Emergency management of hyperkalaemia – calcium, bicarbonate, dextrose, insulin, followed by institution of renal replacement therapy.

Examination and investigation for cause of deterioration:

Abdominal examination and measurement of intrabdominal pressures

Examination for potential sources of infection, including GI, lines, ventilator acquired pneumonia, wounds, urine. Consider empirical antibiotic treatment if thought to be septic aetiology.

Serum lipase, troponin, CK, blood cultures.

Examination to exclude limb compartments, rhabdomyolysis.

Imaging as suggested by results of examination – may require abdominal/thoracic CT scan, renal USS if anuric, angiography/endoscopy if evidence of ongoing bleeding.

SAQ 24

Discuss the role of physiotherapists in the management of patients in the ICU.

Answer

Physiotherapists are part of the multidisciplinary team providing care to patients in the ICU.

Physiotherapists perform an assessment that includes the respiratory, cardiovascular, neurological, and musculoskeletal systems to formulate treatment plans.

The traditional focus of treatment has been the respiratory management of both intubated and spontaneously breathing patients however emerging evidence of the longstanding physical impairment suffered by survivors of intensive care has resulted in physiotherapists re-evaluating treatment priorities to include exercise rehabilitation as a part of standard clinical practice.

The goals of respiratory physiotherapy management are to promote secretion clearance, and to maintain or recruit lung volume, in both the intubated and spontaneously breathing patient. In the intubated patient, physiotherapists commonly employ manual and ventilator hyperinflation and positioning as treatment techniques whilst in the spontaneously breathing patients there is an emphasis on mobilisation.

Physiotherapists have a role in maintaining joint and muscle function in those who are at risk of contractures, for example in neurological injuries and patients with prolonged paralysis. A trend toward an emphasis on exercise rehabilitation over respiratory management is

increasingly evident as survivors of a prolonged ICU stay can suffer deconditioning, muscle atrophy, and weakness that may impact upon quality of life.

Additional roles include the fitting of cervical collars, spinal braces, slings etc. in trauma patients, setting up TENS machines and patient education (exercise, rehab etc.)

SAQ 25

With reference to thyroid function:

- a) Briefly outline the thyroid function/hormone profile expected in the sick euthyroid syndrome or non-thyroidal illness syndrome (NTIS).
- b) For each of the following drugs, list its effect(s) on thyroid function.
 - i. Amiodarone
 - ii. Propranolol
 - iii. Glucocorticoids
 - iv. Opiates
- c) Briefly outline your pharmacological approach to the treatment of thyrotoxic crises. Include in your answer the rationale for each drug used.

Answer

- a)
 - Low serum total T3 is most commonly observed-mean values are 40% of normal.
 - Free T3 is also reduced but less so.
 - Reverse T3 (rT3) is increased. Low T3 is caused by a reduced peripheral conversion of T4 to T3 secondary to inhibition of type 1 5'-deiodinase.
 - Serum T4 and TSH may transiently rise then return to normal.
 - On recovery T3 and rT3 return to normal.
- b)
 - i. Amiodarone
Inhibition of peripheral conversion T4 to T3
 - ii. Propranolol
Inhibition of peripheral conversion T4 to T3
 - iii. Glucocorticoids
Inhibition of peripheral conversion T4 to T3
Suppression of TSH secretion
 - iv. Opiates
Suppression of TSH secretion
- c) A sequential, multidrug approach is vital and the order of therapy is important. Three pathways need consideration-halting synthesis, preventing release of stored hormone and blockade of peripheral effects including blocking conversion of T4 to T3 as well as control of adrenergic symptoms.

Halting synthesis:

First line therapy with Thionamides- thiouracils (Propylthiouracil or PTU) and or imidazoles (methimazole and carbimazole) may be used. Both block thyroperoxidase coupling of idotyrosine residues in formation of T4 and T3. PTU (not imidazoles) will also block peripheral conversion of T4 to T3.

Both given gastrically/PO/retention enema.

Halting release:

Thionamides block synthesis only but not secretion of preformed glandular stores of hormone. Separate treatment is needed to inhibit proteolysis of colloid and continuing release of T3 and 4. Inorganic iodine therapy either with orally administered Lugol solution or potassium iodide should be used. **Iodine should only be used 30 -60 minutes AFTER administration of Thionamides since hormone synthesis may be stimulated.** Alternatives include Li Carbonate and some of the older radiographic contrast agents.

Blocking peripheral action:

B blockade is essential to control peripheral actions of thyroid hormone.

Propranolol is commonly used either gastrically or IV. A drop in T3 levels may be seen with its use (decreases T3-T4 conversion). Glucocorticoids have a role in that they also block conversion of T4 to T3 and may treat any relative adrenal or vasomotor insufficiency that occurs.

SAQ 26

26.1

The following results are from the arterial blood gas analysis of a 46-year-old male ventilated in ICU for three weeks with severe community-acquired pneumonia and ARDS:

Parameter	Patient Value	Normal Adult Range
FiO ₂	0.6	
pH	7.5*	7.35 – 7.45
PO ₂	79.0 mmHg (10.5 kPa)	
PCO ₂	45.0 mmHg (6.0 kPa)	35 – 45 (4.6 – 6.0)
Bicarbonate	36 mmol/L*	22 – 27
Base Excess	12 mmol/L*	-2.0 – +2.0
Sodium	138 mmol/L	135 – 145
Potassium	5.0 mmol/L	3.5 – 5.0
Chloride	97 mmol/L	95 – 105

- Describe the abnormalities.
- Give one likely cause.

Answer

- Metabolic alkalosis (PCO₂ appropriate using $0.7 \times [\text{HCO}_3] + 20 \pm 5$) A-a DO₂ = 295 (P/F 130 “moderate” ARDS)
- Resolution of primary respiratory acidosis with delayed correction of metabolic compensation
Diuretic therapy

26.2

A 75-year-old female insulin-dependent diabetic presents to the Emergency Department semi-comatose. She has been unwell for several days and has a past medical history of left ventricular failure treated with digoxin and a thiazide diuretic.

The following data are from arterial blood gas analysis on admission:

Parameter	Patient Value	Normal Adult Range
FiO ₂	0.4	
pH	7.40	7.35 – 7.45
PO ₂	82.0 mmHg (10.8 kPa)	
PCO ₂	32.0 mmHg (4.2 kPa)*	35 – 45 (4.6 – 6.0)
Bicarbonate	19 mmol/L*	22 – 27
Potassium	2.7 mmol/L*	3.5 – 5.0
Glucose	67 mmol/L*	3.0 – 7.8
Anion Gap	34 mmol/L*	7 – 17

Interpret the acid-base disturbance and give your reasoning.

Answer

- High AG implies severe metabolic acidosis
- Δ Ratio > 3 indicates pre-existing metabolic alkalosis
- PCO₂ slightly lower than expected for compensation ($1.5 \times [\text{HCO}_3] + 8$)
- Implying mild respiratory alkalosis
- History suggests DKA and K⁺ depletion secondary to diuretic use
- Severe compensated metabolic acidosis 2o DKA with mild respiratory
- Alkalosis and pre-existing metabolic alkalosis

26.3

A 59-year-old male with a past medical history of hypertension and dyslipidaemia presents with sore muscles, jaundice and oliguria.

The following data are taken from venous blood investigations on his admission:

Parameter	Patient Value	Normal Adult Range
Urea	25.5 mmol/L*	3.0 – 8.0
Creatinine	523 μ mol/L*	45 – 90
Sodium	138 mmol/L	134 – 145
Potassium	6.2 mmol/L*	3.4 – 5.0
Chloride	105 mmol/L	98 – 108
Bicarbonate	16 mmol/L*	22 – 32
Calcium (corrected)	2.07 mmol/L*	2.20 – 2.55
Phosphate	1.6 mmol/L*	0.8 – 1.5
Creatine Kinase	60110 U/L*	< 200
Bilirubin	108 μ mol/L*	< 20
Alkaline Phosphatase	221 U/L*	35 – 135
Alamine Aminotransferase	1073 U/L*	< 40
γ -Glutamyl Transferase	437 U/L*	< 60
Albumin	29 G/L*	35 – 50

a) What is the diagnosis?

b) Give four likely causes in this patient.

Answer

a) Rhabdomyolysis.

b)

- Muscle ishaemia / compartment syndrome secondary to peripheral vascular disease
- Infection
- Drugs / toxins e.g. statins, alcohol
- Inflammatory myopathies
- Endocrine disorders

26.4

A 68-year-old Type 2 diabetic with a history of alcohol abuse is admitted with abdominal pain and the following results:

Parameter	Patient Value	Normal Adult Range
pH	6.87*	7.35 - 7.45
PaCO ₂	8 mmHg (1.1 kPa)*	35 - 45 (4.7-6.0 kPa)
PaO ₂	149 mmHg (20 kPa)	
Bicarbonate	1.4 mmol/L*	22 – 26
Lactate	16 mmol/L*	< 2
Sodium	142 mmol/L	134 – 145
Potassium	4.7 mmol/L	3.5 – 5.1
Chloride	107 mmol/L*	95 – 105
Urea	14 mmol/L*	3.4 – 8.9
Creatinine	170 µmol/L*	60 – 110
Aspartate Aminotransferase	60 U/L*	< 40
Alanine Aminotransferase	70 U/L*	< 40
Lactate Dehydrogenase	1400 U/L*	50 - 150
Total bilirubin	20 µmol/L	< 20
Glucose	6.5 mmol/L*	3.0 – 5.4
Serum osmolality	314 mOsm/kg*	275 – 295

a) Give three likely diagnoses.

b) List two additional investigations that you would perform based on the above information.

Answer

a)

- Ischaemic bowel
- Metformin induced lactic acidosis
- Cardiogenic shock
- Thiamine deficiency
- Pancreatitis
- OR Any reasonable diagnosis

b) Two of the following investigations:

- Diagnostic laparoscopy or laparotomy
- CT abdomen

- Troponin
- Red cell transketolase,
- Lipase

SAQ 27

- a) Outline the metabolic changes seen in:
 - i. Starvation
 - ii. Stressed state
- b) List the consequences of underfeeding in the critically ill.

Answer

a) Starvation:

Overall an adaptive hypometabolism whereby fat is used as the primary energy fuel and protein is relatively spared. (*Essential point*)

Increase in lipolysis and ketosis with marginal increase in catabolism, glycogenolysis or gluconeogenesis.

Mobilization of protein, glucose and lipids is passive as a result of decrease in insulin levels.

After 24-48hrs gluconeogenesis does increase from peripherally released amino acids and glycerol (from lipolysis) -supplies glucose dependant tissues e.g. brain, immune system and renal medulla.

Beyond 48hrs ketosis occurs and FFAs are used for energy, which minimizes the need for amino acids and so preserves muscle.

Decrease energy expenditure with stable albumin initially.

Urine urea low if adequate protein and energy stores.

Stress:

Endogenous 'stress' mediators such as cortisol, catecholamines, GH, glucagon and cytokines are increased and contribute to the pattern of metabolism and mobilisation of the fuel required

Catabolism, glycogenolysis, and gluconeogenesis increased.

Lipolysis with no increase in ketosis.

Mobilisation of protein is an active process.

Energy expenditure is active.

Albumin levels drop precipitously. (negative acute phase reactant)

Gluconeogenesis decoupled from hormone control so can increase blood glucose levels

Urine urea increases (>10g/day)

b)

- Impaired immune function
- Increased incidence of infection
- Weakness and fatigue
- Decreased ventilatory drive
- Prolonged mechanical ventilation
- Poor wound healing
- Muscle breakdown
- Depression and apathy
- Prolonged ICU and hospital stay

SAQ 28

A 20-year-old primigravida presents at 37 weeks gestation with jaundice, headache, blurred vision and hypertension (140/90 mmHg). The antenatal period was otherwise unremarkable. She is febrile, drowsy, pale, icteric and has pedal oedema. The uterus is palpated as for a full term pregnancy with a normal CTG trace. Examination is otherwise normal.

The following are her early blood results:

Parameter	Patient Value	Normal Adult Range
Hb	80 G/L*	115 – 160
Platelets	$52 \times 10^9/L^*$	140 – 400
International Normalised Ratio	1.8*	0.9 – 1.3
Activated Partial Thromboplastin Time	55 seconds*	25 – 38
Lactate Dehydrogenase	654 U/L*	110 – 250
Fibrinogen	1.0 G/L*	1.5 – 4.0
Total Bilirubin	51 micromol/L*	< 20
Urea	30 mmol/L*	3 – 8
Creatinine	298 micromol/L*	70 – 120
Potassium	5.1 mmol/L*	3.2 – 4.5

- a) List four likely diagnoses for this clinical presentation.
- b) For each of your differential diagnoses:
 - i. List the important management interventions.
 - ii. List one additional diagnostic test.

Answer

- a)
 - Pre-eclampsia
 - HELLP Syndrome
 - Sepsis with DIC
 - HUS-TTP
 - Acute fatty liver of pregnancy
- b)
 - Pre-eclampsia
 - i. Deliver baby
 - ii. Control BP
 - Hydralazine, beta blockers
 - I SNP/GTN if intravenous agent required.
 - iii. Prevention of seizures
 - iv. Magnesium sulphate

Urinalysis – protein, WBCs, RBCs, casts
Evidence of infection or proteinuria (pre-eclampsia)
Renal US

- HELLP Syndrome
 - i. Deliver baby

- ii. Regular monitoring of platelet count and liver function
- iii. Supportive measures whilst observing in HDU for dangerous complications – hepatic haemorrhage/rupture, progressive renal failure, pulmonary oedema.

Peripheral blood film smear

Reticulocyte count, haptoglobins, conjugated/unconjugated bilirubin

Haemolysis screen

Full liver function tests

Sepsis with DIC

- i. Timely delivery of baby in consultation with obstetrician.
- ii. Early broad-spectrum antibiotics.
- iii. Cardiovascular support – adequate volume resuscitation and establish MAP > 65mmHg.

Blood, sputum, urine and vaginal swab for MC&S

Septic screen

HUS-TTP

- i. Deliver the baby.
- ii. Fresh frozen plasma
- iii. Therapeutic plasma exchange
- iv. Corticosteroid therapy
- v. Monoclonal antibody therapy – Rituximab

Evidence of haemolysis or MAHA

Reticulocyte count, haptoglobins, conjugated/unconjugated bilirubin

Haemolysis screen

ADAMTS 13

Acute fatty liver of pregnancy

- i. Timely delivery of baby once mother stabilised
- ii. Correction of DIC
- iii. Supportive therapy
- iv. Monitoring and treatment of complications post delivery e.g. pancreatitis
- v. Consideration for liver transplantation in with irreversible severe liver failure despite delivery and aggressive supportive care

Full set liver function tests

BSL

Liver US

Evidence of haemolysis or MAHA

Reticulocyte count, haptoglobins, conjugated/unconjugated bilirubin

Haemolysis screen

SAQ 29

A patient in the Intensive Care Unit develops complete heart block with hypotension and has a temporary transvenous pacing wire inserted.

- a) Define the pacing threshold and describe how you would test and set it.
- b) Describe how you would check the pacing sensitivity.
- c) What is the purpose of setting the pacing sensitivity?

The bedside nurse informs you that the output has been increased markedly over the course of his shift to maintain capture.

d) What reversible factors might cause this problem?

Answer

a)

- This is the minimum amount of current (in mA) required to initiate depolarization of the paced chamber.
- Set rate paced rate 10 above HR (to ensure patient's rhythm is over ridden minimizing risk of R on T) and set to full demand mode (i.e. high sensitivity- low mV) and output current of 5mA.
- Decrease output until 1:1 capture is lost.
- Slowly increase output till 1:1 capture is regained. This is the pacing (stimulation) threshold.
- The final setting is usually double the pacing threshold.

b)

- This is only checked when the patient has an intrinsic rhythm which affords some cardiovascular stability.
- Set the pacemaker rate 10 below patients intrinsic rate
- Set the output to a very low value e.g., 0.1mA
- Setting the pacemaker to asynchronous mode by turning the sensitivity to its lowest value (highest mV setting) AFTER setting the output current to its lowest value (e.g. 0.1mA) so as to not capture but trigger the pacing indicator.
- The sensitivity is gradually increased (lower mV) until the pacemaker senses the patients intrinsic HR and the pacing indicator no longer illuminates but the sensing indicator does.
- This is the sensing threshold.
- The final setting is usually half this determined value.

c)

- This tests the ability of the pacemaker to sense the patient's intrinsic cardiac activity when one is present so that the pacemaker does not deliver an inappropriate stimulus in competition with the patient intrinsic rate when it is functioning in demand mode.
- Prevents R on T phenomena

d)

- Poor wire placement, movement of the wire
- Acid Base abnormalities,
- Hypo or Hyperkalaemia,
- Hyperglycaemia,
- Drugs e.g. B blockers, calcium antagonists.

SAQ 30

30.1

List six design features of a standard endotracheal tube which improve its safety

Answer

- Clear non-toxic plastic
- Single use
- Radio-opaque line so visible on CXR
- High volume low pressure cuff with pilot tube
- Murphy's eye
- Bevelled tip to assist insertion
- Centimetre markings to assess depth of insertion
- Black line to guide insertion to appropriate depth
- Standard 15mm connector
- Size labelling on pilot balloon

30.2

List the important pieces of information that may be obtained from an arterial waveform tracing.

Answer

- Systolic, diastolic, mean and pulse pressures
- Heart rate and rhythm
- Effect of dysrhythmias on cardiac output / perfusion
- ECG lead disconnect / problem
- Continuous cardiac output using pulse contour analysis
- Specific diagnostic waveform morphologies eg slow rising pulse in AS, pulsus paradoxus in tamponade, dynamic hyperinflation
- Systolic pressure variation, pulse pressure variation may be useful in predicting fluid responsiveness

30.3

List the ultrasound features of a pneumothorax.

Answer

- Loss of comet tails and "marching ants" appearance
- Ribs and pleura move together
- "Lung point" – motionless horizontal lines are replaced by normal lung appearance moving from non-dependent to dependent region and also seen with inspiration and the probe held stationary.
- Loss of "waves on the beach" appearance in M-mode

HOT CASES

Liverpool Hospital

59-year-old female, day 5 in ICU, post in-hospital PEA arrest of unknown cause with a background of insulin-dependent diabetes and chronic renal failure. Clinical findings included a pansystolic murmur, peripheral oedema, left pleural effusion, hepatomegaly and Charcot's joints. Candidates were told that she had been admitted to hospital for insertion of a permacath for dialysis and subsequently had a PEA arrest in the ward. Candidates were asked to examine her and establish likely causes for the arrest. Other discussion points included interpretation of investigations and indications for dialysis.

24-year-old male day 27 in ICU with a background of Lennox-Gustaut syndrome and a recent diagnosis of acute promyelocytic leukaemia, admitted with neutropaenic sepsis and bilateral pulmonary infiltrates. He had failed weaning and had a recent deterioration with worsening hypoxia. Clinical findings included gross fluid overload, bilateral pleural effusions with left collapse/consolidation and pericardial effusion. Candidates were asked to assess him and formulate a management plan. Discussion points included weaning strategies, principles of CRRT and management of sepsis, including possible sources.

70-year-old female day 2 post laparotomy for repair of obstructed ventral hernia and division of adhesions with a background of severe COPD. Clinical findings included kyphosis, wheeze and bronchial breathing, atrial fibrillation, right ventricular impulse, obstructed pattern on ETCO₂ trace and presence of intra-abdominal pressure monitor. Candidates were asked to examine her with a view to planning further management. Other discussion points included interpretation of investigations, management of AF and COPD, ventilation weaning strategies, timing of tracheostomy, intra-abdominal hypertension and nutritional support.

75-year-old female with MSSA mitral valve endocarditis admitted to ICU 5 days earlier following a MET call for a sudden fall in conscious state to GCS 4 and intubation in the ward. Clinical findings included signs of left lower lobe consolidation, pansystolic murmur at the apex and marked oedema. Candidates were told that she presented with fever, delirium and hypotension and were asked to examine her with a view to establishing the diagnosis. Discussion points included the differential diagnosis, interpretation of investigations, treatment of endocarditis and drug dosing with CRRT.

55-year-old female, day 3 ICU, following SAH secondary to aneurysmal bleed. Clinical findings included an intubated, awake, responsive patient with a dense left hemiplegia and the presence of an EVD, arterial puncture site at the groin and a nimodipine infusion. Candidates were told that she had presented post collapse 3 days earlier and were asked to examine her neurological system. Discussion points included interpretation of the CT brain and ECG, the differential diagnosis for this patient and the management of SAH including complications and prognosis.

47-year-old female, almost 3 months in ICU with severe ARDS and multi-organ failure secondary to influenza A pneumonia and subsequent complications including anuric renal failure, pancreatitis with pseudocyst and critical illness weakness. Clinical findings included morbid obesity, generalized weakness, bibasal crackles, significant peripheral oedema and the presence of a tracheostomy, dialysis catheter and abdominal drain. Candidates were told that she had presented with pneumonia 80 days previously and were asked to examine her with a view to determining why weaning had taken so long and how they would proceed from this point. Other points of discussion included interpretation of imaging, abnormal blood results and the management of pancreatitis.

17-year-old male admitted overnight with multi-trauma from a motor vehicle crash. Clinical findings included an intubated but responsive patient with a laparotomy wound, painful hip/pelvis, seat belt marks and a dressing over his left knee. Candidates were told that he had been admitted overnight following a motor vehicle crash with GCS 13, hypotension, tachycardia and a positive FAST and underwent a splenectomy, and were asked to perform a tertiary survey. Discussion points included cervical spine clearance and management of the post-splenectomy patient.

Nepean Hospital

67-year-old male, re-admitted to ICU 26 days earlier following a MET call for reduced level of consciousness. He was admitted initially post decompressive craniotomy for a spontaneous SDH and his first ICU stay had been complicated by refractory intracranial hypertension, failed extubation, non-convulsive status epilepticus and DVTs. Clinical findings included right hemiparesis and the presence of a right-sided craniectomy and a tracheostomy. Candidates were asked to examine him and assess whether he was ready for discharge to the ward. Further discussion points included interpretation of CT and MRI brain, the discrepancy between the anatomical site of the SDH and clinical findings, management of tracheostomy, DVT prevention and management and prognosis.

43-year-old male, day 5 in ICU for an aneurysmal SAH and subsequent clipping of two aneurysms. Clinical findings included left hemiplegia, oliguria and the presence of bilateral EVDs and a nimodipine infusion. Candidates were asked to identify why he was slow to wake. Discussion points included interpretation of CT brain and CSF microscopy, causes of decreased level of consciousness and management of vasospasm.

69-year-old female with a history of recurrent falls, two weeks in ICU following a fall resulting in left-sided rib fractures, pneumothorax and surgical emphysema. Clinical findings included cachexia, reduced breath sounds left base and the presence of a tracheostomy. Candidates were told that she had presented with respiratory failure following a fall and were asked to examine her and assess why she was failing to wean. Discussion points included interpretation of investigations and management of weaning.

62-year-old female, with a long-term tracheostomy for airway patency following CVA, admitted to ICU two weeks earlier with hypoxic respiratory failure. Clinical signs included morbid obesity, reduced breath sounds bibasally, left pleural effusion, atrial fibrillation and right hemiparesis. Candidates were asked to assess her with respect to her failure to wean. Other points for discussion included interpretation of CXR, management of AF, CCF and infection, nutritional support and the plan should weaning be unsuccessful.

72-year-old man, day 2 in ICU, following presentation with ARDS on the background of a recent admission with H. influenza pneumonia. Clinical findings included high ventilatory requirements, bilateral crackles and wheezes and signs of left upper lobe consolidation on auscultation. Candidates were asked to examine him and give a differential diagnosis. Additional points for discussion included interpretation of CXR, ventilatory management, choice of antibiotic and diagnostic criteria for ARDS.

Westmead Hospital

39-year-old male, day 3 ICU, following presentation with uncontrolled hypertension and subsequent right parietal bleed, and pulmonary oedema (now resolved), on a background of obesity, type 2 diabetes and chronic renal failure. Clinical findings included altered conscious state with no localising signs, reduced breath sounds at both bases, elevated CVP, pericardial rub and a gallop rhythm, generalised anasarca and the presence of a right

femoral venous vas cath. Candidates were told that he had presented 3 days earlier with altered sensorium and shortness of breath on the background of diabetic nephropathy and were asked to examine him and to identify the reason for his presentation. Discussion points included interpretation of imaging and investigations, contributing factors for his presentation and reasons for the elevated CVP.

70-year-old female, one month in ICU, with failure to wean following elective surgery for MVR and tricuspid valve annuloplasty. Clinical findings included ventricular pacing with cannon a waves, parasternal heave, systolic murmur at the left sternal edge and mid-diastolic murmur at the apex, tender hepatomegaly and the presence of a tracheostomy and a midline sternotomy scar. Candidates were told that she had difficulty weaning from ventilatory support following TV annuloplasty and MVR 4 weeks previously and were asked to examine her focussing on the cardio-respiratory system. Discussion points included reasons for failure to wean post MVR, mechanism of cannon a waves and management issues specific to the case.

20-year-old male, scheduled for a left hemicolectomy, with a background including congenital myopathy with severe neuromuscular weakness and restrictive lung disease, ventilator dependence and permanent tracheostomy and frequent hospital admissions for recurrent chest infections. Clinical findings included kyphoscoliosis, wasting and weakness of all muscles with preserved sensation, signs of restrictive lung disease and the presence of a tracheostomy and a PEG. Candidates were asked to identify the key issues for his peri-operative management. Other discussion points included interpretation of investigations and imaging and aspects of his chronic co-morbidities.

81-year-old male admitted 17 days earlier with SAH secondary to ruptured right PCOM aneurysm with bradycardia and AICD malfunction and new onset fever. Clinical findings included signs of biventricular failure, left upper limb weakness and the presence of craniotomy wound, EVD and permanent pacemaker/AICD. Candidates were told he had presented with a headache and altered conscious state and now had a fever and were asked to examine him and determine a management plan. Discussion points included interpretation of imaging and investigations, the differential diagnosis and causes of fever.

59-year-old male, day 5 ICU, following laparotomy for ischaemic bowel with decompensated liver cirrhosis. Clinical findings included reduced GCS, jaundice and the presence of an open abdomen with tension sutures and 2 drains and a heparin infusion. Candidates were asked to examine him, identify the issues and formulate a management plan. Other discussion points included interpretation of imaging, weaning plan, management of coagulation status and nutritional support.

33-year-old male, day 17 ICU, admitted with haemorrhagic shock from multiple stab wounds, including liver laceration and perforated bowel. Clinical findings included decreased bibasal breath sounds, moderate oedema, polyuria, open abdomen with VAC dressing and the presence of a tracheostomy. Candidates were asked to examine him, identify the current issues and formulate a management plan. Other discussion points included management of weaning and nutritional support.

51-year-old female, day 2 ICU, with large retroperitoneal bleed and resolved haemorrhagic shock related to dual anticoagulation for right lower leg DVT and arterial occlusion. Clinical findings included bronchial breath sounds at the left base, ischaemic right leg and abdominal bruising. Candidates were asked to assess her suitability for extubation. Discussion points included interpretation of imaging and management of her coagulation status.

49-year-old male, day 2 ICU, with mesenteric and retroperitoneal bleed following emergency coronary angiogram and anti-platelet and anticoagulation therapy for inferior STEMI. Clinical findings included a distended tender abdomen and the presence of a right groin arterial puncture site. Candidates were told he had been given multiple anti-platelet agents for ACS and had developed respiratory failure and been intubated and was now oliguric with elevated creatinine. Candidates were asked to determine the reasons for his deterioration. Discussion points included interpretation of the ECG and CT abdomen, cause for his abdominal distension, cause for his renal failure, management of his coagulation status and suitability for extubation.

VIVAS

Viva 1

You are called into the resuscitation room in your Emergency Department to assess a trauma patient who has been involved in a high-speed car crash. She appears morbidly obese with an estimated weight of 170kg.

How does the pattern of traumatic injury differ in obese patients?

Viva 2

A 62-year-old male is admitted to the ICU post-operatively having undergone a transthoracic oesophagectomy for squamous cell carcinoma of the oesophagus. The patient is extubated post-operatively but requires reintubation due to respiratory failure.

What are the likely causes in this patient?

Viva 3

You are referred a 35-year-old female by the Emergency Department, who presented with a reduced level of consciousness. She has been intubated and ventilated and is haemodynamically stable.

Her CT brain is normal, and she is apyrexial.

She has no known past medical history.

Investigations on admission are shown in TABLE 1*

Interpret these biochemical findings.

**Table 1 not included in the report.*

Viva 4

A 59-year-old male, accepted for admission to the ICU from the Emergency Department (ED) following a severe traumatic brain injury, and now sedated, intubated and ventilated, is escorted to ICU by an ED Consultant.

What are the critical features of the handover that you would want to know?

Viva 5

A 58-year-old woman has been brought to the Emergency Department (ED) having been found with a reduced level of consciousness. Witnesses describe possible seizures. She has been unwell for a number of weeks with complaints of dysuria and frequency and progressive lethargy.

Her current medications are atorvastatin and metoprolol. She has been intubated in the ED because of her level of consciousness, and is easy to ventilate and haemodynamically stable. Non-contrast brain CT is normal.

What are the likely causes for this patient's presentation?

Viva 6 (Equipment viva)

A 53-year-old male returns from theatre on high dose inotropic support following Aortic Valve Replacement and Coronary Artery Bypass Graft x 4. He had a long pump time of 252 minutes and pre and post-op left ventricular function are very poor with global hypokinesis.

There are no surgical issues. His cardiac index is 1.6 L/min/m².

His urine output for the last hour is 2 mL and his serum HCO₃⁻ is 15 mmol/L with a lactate of 8 mmol/L.

He is ventilated in a mandatory mode with FiO₂ = 0.85 & PEEP 15cmH₂O.

What options are available to improve this man's tissue perfusion?

Viva 7 (Radiology viva)

Consisted of 3 CXR images and 3 CT scans for interpretation.

Viva 8 (Communication viva)

The Senior Registrar has just performed a percutaneous tracheostomy, on a 17-year-old female, Ashleigh, recovering from meningococcal sepsis.

As the supervising consultant you were unavoidably called away to a separate emergency. The procedure was undertaken with due diligence, but has been complicated by a posterior tracheal tear (acute bilateral pneumothoraces and massive subcutaneous emphysema).

You as the consultant need to inform the guardian (an ex nurse) of this unfortunate complication, but have also been made aware that the consent for the procedure was obtained by the junior registrar and was rushed, lacked detail and was incomplete.

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