The Faculty of Intensive Care Medicine

NEW EXAMPLE EXAM QUESTIONS

The FFICM Court of Examiners has released these additional example questions from the FFICM Final Examination question bank.

1 Structured Oral Examination (SOE) questions
2 A Objective Structured Clinical Examination (OSCE) station

Please note that questions no longer used in the exam will not be adjusted following removal from the question bank. Answers displayed on the website may not be current.

It is useful to read these in conjunction with the Examination Guide available in the FFICM Examination webpages.

Structured Oral Examination (SOE) questions

In the structured oral exam the examiner is instructed to ask the questions written in bold. Examiners are given examples of the topics that may be covered in the answer.

SOE: ACUTE KIDNEY INJURY

What do you understand by the term Acute Kidney Injury (AKI)?

- 2012 the Kidney Disease; Improving Global Outcomes (KDIGO) organisation: have updated the RIFLE(risk,injury, failure, loss of kidney function and end-stage kidney disease) and AKIN classification.
- AKI is defined as any of the following:-Creatinine > 26.5 micromoles/L within 48 hours or rise of 50% within 7 days, or urine output < 0.5ml/kg/hr for 6 hours.
- AKI is divided into 3 stages: Serum creatinine or Urine output:
    - ○ Stage 1 1.5 – 1.9 times baseline, or > 26.5, <0.5 ml/kg for 6-12 hours ○ Stage 2 2.0-2.9 times baseline <0.5 ml/kg/hr for >12 hours ○ Stage 3 3.0 times baseline/creatinine > 354, <0.5 ml/kg/hr for > 24 hours or on RRT.

What are the limitations with the definitions?

- AKI may develop pre-admission and baseline creatinine may be unknown
- No account of different aetiologies of AKI
- Urine output is modified by prior diuretic administration and low urine output is not necessarily a marker of AKI (e.g. post op)
- Creatinine level is influenced by non renal factors such as low muscle mass, muscle injury and drugs (ciproxin, co-trimoxazole). High bilirubin interferes: creatinine underestimated.

How does AKI influence the outcome of the critically ill patient?

- Increases mortality and length of stay in ICU: organ failures
- Around 50% of patients needing renal replacement
- Increased risk of nosocomial infection and difficulty weaning from ventilation
- Greater than 30% end with CKD and some require long term dialysis
- High cost of treating AKI

What are the common causes of AKI?

- PRERENAL: shock, sepsis, intravascular depletion, raised IAP
- RENAL: Toxins (drugs, contrast, myoglobin), glomerulonephritis, interstitial nephritis (drugs)
- POSTRENAL: stones, tumours, external compression, surgical ligation

How would you distinguish the underlying cause?

- Clinical assessment history, examination and fluid and drug chart review.
- Urine dip test for blood, protein, nitrites.
- Urine and blood cultures
- Urine microscopy: blood, wbc, casts, crystals
- Urine Na interpret with caution.
- Imaging U/S, within 24 hours to exclude outflow obstruction and assess renal size.

Specific investigations:

- FBC, Film: HUS/TTP, malaria, interstitial nephritis eosinophilia
- Autoimmune markers ANA, ANCA, AGBM, complement (SLE, endocarditis)
- Creatinine kinase myoglobinuria (rhabdomyolysis), Protein electrophoresis (myeloma)urate and LDH (tumour lysis)
- Intraabdominal pressure for compartment syndrome

What measures can prevent AKI?

- Fluid resuscitation with crystalloid not colloids (starches, gelatins associated with renal injury).
- Vasopressors to maintain MAP in vasomotor shock (premorbid BP as target)
- Avoid nephrotoxic drugs (NSAIDS, aminoglycosides) and diuretics (only use to treat fluid overload)
- Dopamine, atrial naturetic peptide not recommended
- Contrast induced AKI: Ensure normovolaemic and maintain IV fluids for 12 hours postprocedure. Isotonic bicarbonate may be beneficial. N Acetyl Cysteine controversial
- Rhabdomyolysis: saline/isotonic bicarbonate. Maintain urine output > 100 mls/hr, pH > 6.5
- Abdominal compartment syndrome: >20-30 cm H_2O consider need for decompression

SOE: ANAPHYLAXIS

When should you suspect an anaphylactic reaction?

- A diagnosis of anaphylactic reaction is likely if a patient who is *exposed to a trigger* (allergen) develops a *sudden illness* (usually within minutes of exposure), especially a known trigger
- Anaphylaxis is likely when ***all*** of the following 3 criteria are met:
- Sudden onset and rapid progression of symptoms
- Life-threatening Airway and/or Breathing and/or Circulation problems
- Skin and/or mucosal changes (flushing, urticaria, angioedema)
- Skin or mucosal changes alone are not a sign of an anaphylactic reaction
- Skin and mucosal changes can be subtle or absent in up to 20% of reactions
- There can also be gastrointestinal symptoms (e.g. vomiting, abdominal pain, incontinence)

What are the potential mechanisms of anaphylaxis?

- Release of histamine, tryptase, leucotrienes, prostaglandins, platelet activating factor, other mediators
- Interaction between an allergen & allergen specific IgE bound to IgE receptors on mast cells & basophils
- IgE independent immune mechanisms
- Direct degranulation of mast cells
- Idiopathic

How would you treat an acute episode of anaphylaxis?

- *Immediate treatment*
- Recognition that they are seriously unwell and early call for help. ABCDE approach
- Adrenaline therapy if indicated: Give IM adult dose 500ug or IV titrate 50ug
- *Patient positioning*
- Patients with Airway and Breathing problems may prefer to sit up to help breathing easier
- Pregnant patients should lie on their left side to prevent caval compression • *Remove the trigger if possible*
- Stop any suspected trigger (e.g., stop intravenous infusion of a gelatin solution or antibiotic)
- Remove the stinger after a bee sting, early removal more important than the method of removal
- *Cardiorespiratory arrest following an anaphylactic reaction*
- Start cardiopulmonary resuscitation (CPR) immediately and follow current guidelines. Give adrenaline
- *After stabilisation consider:*
- Chlorphenamine 10 mg IV (adult dose). Hydrocortisone 200 mg IV (adult dose)

What additional investigations and follow-up measures should be considered?

- FBC, U/E, ABG, clotting, sugar, 12-lead ECG, CXR.

Mast cell Tryptase concentration

- Timing of samples important as Tryptase concentrations in the blood may not increase significantly until 30 minutes after the onset of symptoms, and peak 1-2 hours after onset.
- The half-life of tryptase is short (approx 2 hours), concentrations may be back to normal in 6-8 hours.
- 3 timed samples: i) Initial sample as soon as feasible ii) 2nd sample at 1-2 hours after start of symptoms, and iii) 3rd sample either at 24 hours or in convalescence

5 Follow-up measures

- Patients with a good response to initial treatment may suffer early recurrence of symptoms
- Should be kept under observation for up to 24 hours. This caution is particularly applicable to:
    - Severe reactions with slow onset caused by idiopathic anaphylaxis,
    - Reactions in individuals with severe asthma,
    - Reactions with the possibility of continuing absorption of allergen, ○ Patients with a previous history of biphasic reactions,
- Record keeping: Comprehensive note keeping with timing of drugs and events and physiology
- Reporting of reaction: Report to MHRA using yellow card. Discuss fatal anaphylactic reactions with coroner
- Specialist referral: All patients presenting with anaphylaxis refer to an allergy clinic to identify the cause.
- Auto-injector for patients at increased risk of idiopathic anaphylaxis, or continued high risk eg bee stings food
- Patient education: Patients need to be able to recognise the early symptoms , advised to carry their adrenaline auto-injector, and they and family/school, trained in using the autoinjector. Wear bracelet (e.g., Medic Alert)

SOE ECLAMPSIA

How is eclampsia defined?

- Eclampsia is defined as the development of convulsions and/or unexplained coma during pregnancy or postpartum in patients with signs and symptoms of preeclampsia.
- Pre-eclampsia: hypertension(diastolic>90mmHg or rise of >15mmHg from baseline, proteinuria, oedema.
- Involves release of vasoconstrictor factors with damage to endothelium including kidneys and liver.

How may pre-eclampsia present and progress?

- Headache, blurred vision, papilloedema, clonus, altered mental status, and epigastric or right upper quadrant pain may occur. Sometimes oedema, hypertension and proteinuria may be minimal or even absent
- Haemolysis, Elevated Liver enzymes and Low Platelet count (HELLP). Liver damage/rupture/haematoma may occur. Pulmonary oedema in 6%.
- Retinal detatchment.
- Cerebral oedema.
- DIC.

When does eclampsia typically occur?

- 91% of all cases of Eclampsia occur at or beyond 28 weeks gestation but it can can occur up to 4 weeks post partum (Late post partum eclampsia is defined as eclampsia that occurs more than 48 hours but less than 4 weeks after delivery)

Describe the management of eclampsia

- NICE guidance available 'Hypertension in pregnancy'
- Objectives are to stop seizures, stabilise mother and prevent foetal damage
- ABC approach –patient on left side to prevent pulmonary aspiration and relieve IVC obstruction by uterus
- Treat seizures with intravenous Magnesium Sulphate (Loading dose 4g given intravenously over 5 minutes followed by infusion of 1g /hour for 24 hours. If recurrent seizures then further 2-4 g given over 5 minutes)
- Keep blood pressure < 150/80 - 100 mmHg. Agents to consider are Labetalol (iv or oral); Hydralazine (iv) or Nifedipine (oral). Avoid ACE inhibitors, Atenolol, Angiotensin receptor blocking drugs and diuretics.
- Fluids: caution. Balance risks of precipitating left ventricular failure, pulmonary oedema with cerebral perfusion and uteroplacental blood flow.
- Foetal monitoring by cardiotocograph (CTG). Consider steroids for foetus. Deliver baby
- Consider other causes such as: cerebrovascular accidents, brain tumours, thrombotic thrombocytopenic purpura,hypoglycaemia, hyponatraemia and other seizure disorders, thromboembolic disease or amniotic fluid embolism

What are the complications of magnesium sulphate infusion and how should it be monitored?

- Complications: respiratory depression, hypotension, arrhythmias, slurred speech, drowsiness, double vision, flushing, nausea and vomiting.
- Monitoring should include: Glasgow Coma Score, respiratory rate, arterial line, urine output • ECG monitoring
- Check patella reflex every 2-4hours: if depressed then stop infusion.
- Serum Magnesium levels should be monitored particularly if there is renal dysfunction • (Stop infusion if RR < 10 breaths/minute)

SOE NON-INVASIVE VENTILATION

How can non-invasive ventilation be delivered?

- Using dedicated non-invasive ventilators.
- Using NIV modes on conventional ICU ventilators.
- Delivered via nasal masks (either covering the nose, or nasal cushions placed into the nostrils), full-face masks which may cover the nose or mouth or may literally cover the whole face.
- Helmets: similar to but stiffer than CPAP helmets
- Non-invasive ventilation can also be delivered via a cuirasse or iron lung

In what situations may non-invasive ventilation be beneficial?

- NIV reduces the need for tracheal intubation and invasive ventilation in acute exacerbations of COPD, cardiogenic pulmonary oedema and ventilatory failure in the immunocompromised patient.
- Intervention to avoid of re-intubation in patients who have recently been extubated after a period of mechanical ventilation.
- Elective weaning strategy in patients who are difficult to wean from invasive ventilation.
- Prophylactic use to reduce post-operative atelectasis.
- Palliation: breathlessness and symptoms of CO2 retention in patients with MND with a max inspect mouth pressure worse than 40cmH2O and without severe bulbar involvement or MND.

Where is NIV contraindicated?

- Impaired level of consciousness: inability to protect airway. May be used if due to high CO_2 which may recover.
- Severe confusion,
- Copious secretions,
- Patients with facial injuries or burns.
- High risk vomiting/bowel obstruction.
- Unstable head and neck: may lose airway if head topples to one side. Try soft collar in this situation.
- Upper GI surgery: controversial.

What settings are effective?

- Normally initially set in terms of IPAP or EPAP at pressures of 10 and 5 respectively.
- Can be increased to 20 IPAP, above which point leakage is common
- Adjustment of face mask straps to achieve a better seal may involve slackening straps to avoid distortion

When is NIV more likely to fail?

- Hypoxia.
- Unilateral white out from consolidation.
- Persistent metabolic acidosis.
- Poor mask fit: may cause facial ulceration and high delivery pressures.
- Failure to improve @ 4 hours: plan B, review options for invasive ventilation.

An Objective Structured Clinical Examination (OSCE) station

Each station is marked out of 20. In the exam neither the examiner nor the candidate are aware of the weighting of each part of the station.

METABOLIC ABNORMALITIES PRESENTING TO THE EMERGENCY DEPARTMENT

1 **Considering the history, blood test results and 12-lead ECG labelled 'Patient A'. What is the most significant metabolic abnormality?**
Hyperkalaemia 2

2 **Suggest a likely cause for the metabolic abnormality.**
Rhabdomyolysis 2
If the candidate suggests neuroleptic malignant syndrome, state that the patient is normothermicand proceed with the subsequent questions

3 **Suggest a differential diagnosis for the depressed level of consciousness.** Drug overdose / Alcohol intoxication / Intracerebral haemorrhage / Trauma
Two from this list of answers required 1

4 **What investigation is diagnostic of rhabdomyolysis?**
Creatine kinase 2

5 **Look at the history, arterial blood gas and blood test data labelled 'Patient B'. What is the mostsignificant metabolic abnormality?**
Metabolic acidosis 1

6 **Suggest likely causes.**
Methanol 1
Ethylene glycol poisoning 1
Methanol may also be called methyl alcohol or wood alcohol but 'methanol' is the required answer. You may prompt by asking "Do you know another name for... ?"

7 **List treatment options for methanol poisoning.**
Fluid resuscitation 1
Sodium bicarbonate 1
Ethanol 1 Fomepizole 1
Haemodialysis 1
CANDIDATE GUIDANCE

You will review history and initial investigations for two patients in the Emergency Department.

Artefacts (on pages below)

- Patient A history including biochemistry data
- Patient A ECG
- Patient B history including blood gas data and biochemistry data

Patient A

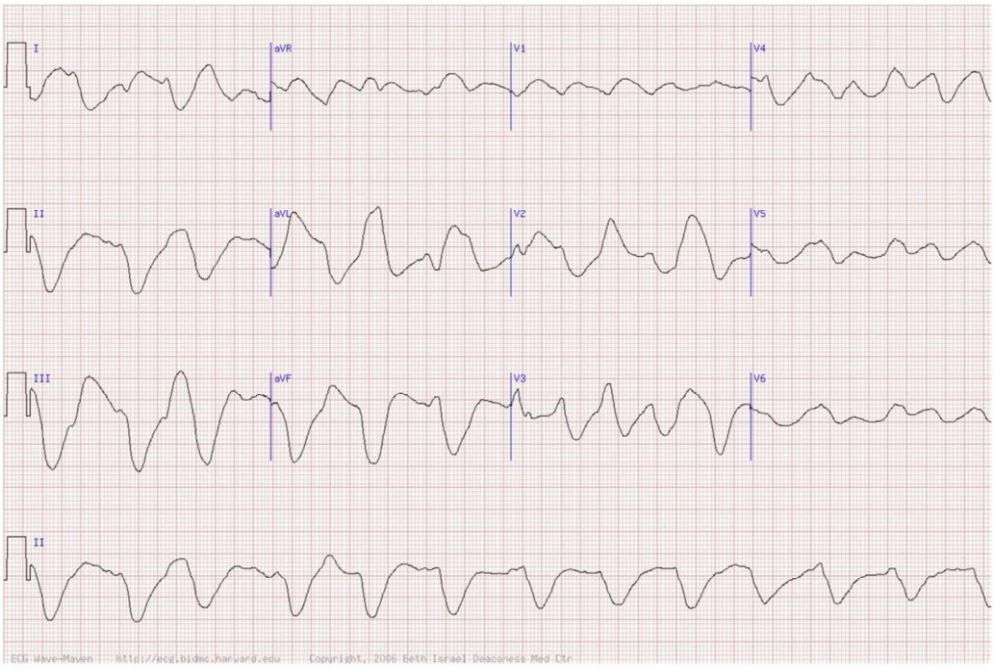
A 19 year old was found unconscious at home. He has been brought to the Emergency Department by paramedics.

Initial bloods on admission to the Emergency Department show

	Measured	Reference
Urea	11.7 mmol/l	3.6-7.1 mmol/l
Sodium (Na)	129 mmol/l	136-145 mmol/l
Potassium (K)	9.4 mmol/l	3.5-5.0 mmol/l
Bicarbonate (HCO_3)	14.6 mmol/l	21-28 mmol/l
Creatinine	163 µmol/l	<133 µmol/l
Glucose	8.7 mmol/l	4.2-6.4 mmol/l
Calcium (Ca^{2+})	1.87 mmol/l (corrected)	2.2-2.6 mmol/l
Albumin	27 g/l	35-55 g/l
Phosphate (PO_4^{2-})	2.7 mmol/l	1.0-1.4 $mmol/l^{-1}$

A 12-lead ECG is available

Patient A



Patient B

A 69 year old man is admitted to the Emergency Department with breathlessness and confusion.

A clear
B RR 32 symmetrical, big volumes
C cool peripheries HR 124 in AF BP 94/67 mmHg
D GCS E3 V4 M6 PERL 3mm No focal neurological deficit

His initial arterial blood gas data on 15 $l.min^{-1}$ oxygen are as follows

	Measured	Reference
Hydrogen ion (H^+)	178 $nmol/l^{-1}$	36-44nmol/l
pH	6.75	7.34-7.44
$PaCO_2$	2.4 kPa	4.7-5.9 kPa
PaO_2	39.6 kPa	11-13 kPa
Bicarbonate (HCO_3)	5.3 mmol. l^{-1}	21-28 mmol/l
Base excess	-23.7 mmol. l^{-1}	-2 -+2 mEq/l
Glucose	9.3 mmol. l^{-1}	4.2-6.4 mmol/l
Urea	12.9 mmol. l^{-1}	3.6-7.1 mmol/l
Sodium (Na)	137 mmol. l^{-1}	136-145 mmol/l
Potassium (K)	5.7 mmol. l^{-1}	3.5-5.0 mmol/l
Creatinine	211 umol. l^{-1}	<133 µmol/l
Lactate	3.4 mmol. l^{-1}	0.6-1.7 mmol/l