The Faculty of Intensive Care Medicine

Updated Feb 2022

FFICM SOE Example Questions

Question Number	**ICM SOE Example 1**
Question Title	**Myasthenia Gravis**

Candidate Guidance	This is a question about myasthenia gravis (MG)

The information provided for each stem does not constitute a model answer, it is an aide memoire and guidance for the examiner.

Stem		

1	**What is Myasthenia Gravis?**	
Objective	*This is designed to determine the candidate's knowledge of the disorder and its cause*	
Guidance for examiner	• Myasthenia Gravis is an auto-immune disorder due to auto-antibodies against the acetylcholine receptors in skeletal muscle that causes weakness. • There are two main forms: generalised and ocular (in which the weakness is limited to the eyelid and extraocular muscles). • The autoantibodies to acetyl choline receptors can be detected in 90% patients • The thymus is abnormal in 75% of patients with a thymoma in 10%. • Associated auto immune conditions occur in 5-10% including rheumatoid, pernicious anaemia and thyroid disease.	

2	**How do myasthenic patients present?**	
Objective	*This is a general question about presentation*	
Guidance for examiner	• Patients present with fatiguable muscle weakness. • They may present with diplopia, slurred speech or difficulty swallowing. • The ocular muscles are most commonly affected followed by bulbar weakness and then trunk and limb muscles. • They may present with a myasthenic crisis or occasionally a cholinergic crisis.	

3	**How is it investigated?**	
Objective	*The candidates should be able to describe how to diagnose MG and assess its severity and progression.*	
Guidance for examiner	• Blood tests including anti acetyl choline receptor auto-antibodies • Electrophysiology (EMG fatiguability) • CT or MRI of thorax (for thymus) • Edrophonium test • Occasionally muscle biopsy is required • Spirometry in crisis or sniff pressures	

4	**How is a myasthenic crisis treated?**	
Objective	*The candidates need to explain how they would manage a patient needing intensive care*	
Guidance for examiner	• Symptom control: Initially with anticholinesterase drugs: pyridostigmine 15-30mg 4-6hourly. Titrated against response.	

	• Patients may need respiratory support, intubation, ventilation, tracheostomy • Disease modifying: Immunosuppression: prednisone 40-60mg/day (2-3 weeks to work, but may worsen symptoms), azathioprine (many months) is mainstay • Immunoglobulins are effective in some patients. • Plasmapheresis may be used for rapid symptom control and for a poorly controlled patient who needs to go to theatre for another reason. • Thymectomy for those with thymoma and may be considered in resistant cases who do not have a thymoma.

5	**What are the indications for ventilation?**	
Objective	*The candidates should be able to give a clear assessment of the patient*	
Guidance for examiner	• Patients may develop respiratory failure due to respiratory muscle weakness and bulbar dysfunction. • Decreasing forced vital capacity (< 15-20 ml/kg) or low sniff/MIP • Failure to clear secretions • Hypercapnia, decreased conscious level • Pneumonia (either as precipitant or as consequence)	

Question Number	ICM SOE Example 2
Question Title	Microbiology

Candidate Guidance	This is a question about the principles of antimicrobial therapy

The information provided for each stem does not constitute a model answer, it is an aide memoire and guidance for the examiner.

Stem		

1	**What factors do you consider when you chose an antibiotic?**	
Objective	*The candidate should be able to give a clear explanation of the factors affecting antibiotic choice*	
Guidance for examiner	• The site or likely source • Community or hospital acquired • The diagnosis or likely organism • Patient allergies • Which antibiotics they have already had • Known local resistance patterns • Hospital prescribing policy	

2	**Why might antibiotic therapy fail?**	
Objective	*The candidate should have a clear understanding of the reasons for antibiotic failure.*	
Guidance for examiner	• Wrong drug, • Inadequate dose/Missed dose • Wrong frequency • Too late • Wrong route • Failure of penetration of the source • Emergence of resistant organisms • Lack of source control • If the patient is immunosuppressed	

3	**How do Beta-lactams work?**	
Objective	*The candidate should be able to explain the mechanism of action of common beta-lactams*	
Guidance for examiner	• β-Lactam antibiotics act on enzymes called *penicillin-binding proteins (PBPs)* responsible for building the bacterial cell wall. • They are active only against rapidly multiplying organisms. • Penicillin within the cell wall interferes with production of cell wall peptidoglycans and results in cell lysis in a hypo-osmotic or iso-osmotic environment. • There may be anywhere from 2 to 8 PBPs in a bacterium. • Differences in the spectrum and activity of β-lactam antibiotics are due to their relative affinity for different PBPs. To bind to the PBPs, the β-lactam antibiotic must first diffuse through the bacterial cell wall. Gram-negative organisms have an additional lipopolysaccharide layer that decreases antibiotic penetration. Therefore gram-positive bacteria are usually more susceptible to the action of β-lactams than gram-negative bacteria. • Penicillins poorly penetrate mammalian cells and are ineffective in the treatment of intracellular pathogens	

4	**How does the presence of a toxin producing Group A streptococci, affect your choice of treatment?**	
Objective	*The candidates should be able to give reasoning for more complex antibiotic choices*	
Guidance for examiner	• Use of clindamycin to reduce toxin production. T • This is combined with other antibiotics to reduce the development of clindamycin resistance. • Consider adjuvant therapy such as immunoglobulins • Debridement is likely to be necessary if the organism produces necrotising fasciitis.	

5	**Which antibiotic levels in common use are monitored routinely and why?**	
Objective	The candidate should be able to explain which antibiotics need monitoring, how and why	
Guidance for examiner	• Gentamicin: efficacy and ototoxicity and nephrotoxicity • Vancomycin: ototoxicity and nephrotoxicity. • Needs to maintain an effective level in the blood for a period of time to have its bacteriocidal effect on bacterial cell walls. • Easy to get high levels with organ failure especially renal failure • These are drugs with a low volume of distribution and a narrow therapeutic index • Vancomycin levels may be even more difficult to judge when patient on CVVH	

Question Number	ICM SOE Example 3
Question Title	Heliox

Candidate Guidance	You will be asked about the properties and use of Heliox

The information provided for each stem does not constitute a model answer, it is an aide memoire and guidance for the examiner.

Stem		

1	What is heliox?	
Objective	*A simple knowledge of what Heliox is, is required*	
Guidance for examiner	• Heliox (HeO2) is an oxygen – helium mixture, usually supplied as a 20:80 or 30:70 percentage mixture • Helium is an inert gas with a very low atomic weight and density	

2	What are the physical properties of heliox?	
Objective	*Candidates should be able to explain the properties of heliox which make it useful clinically*	
Guidance for examiner	• Heliox in an 80% mixture with oxygen is three times less dense than room air. • Reduced density leads to reduced resistance to gas flow in areas of turbulent flow. • As an inert gas helium is highly unlikely to cause any adverse reactions or events (none have been reported so far with its use)	

3	How might these be clinically useful?	
Objective	*This question is designed to ask the candidate about how heliox may be beneficial*	
Guidance for examiner	• As the diameter of patients' airways changes so does the prominent type of airflow – can be calculated using Reynold's number (larger airways have turbulent flow, smaller diameter airways have laminar flow) • As larger airways have predominantly turbulent flow, diseases of large airways may increase the resistance to turbulent flow and therefore increase the work of breathing. • The use of low-density gas like heliox to reduce resistance to flow may be clinically useful and potentially reduce the resistance to breathing that is seen with such diseases. • Therefore, traditionally used in upper airways obstruction or narrowing.	

4	In which clinical conditions has the use of heliox been used?	
Objective	*This question is aimed exploring the candidate's knowledge of its clinical application*	
Guidance for examiner	• Asthma– studies have demonstrated that during severe asthma / status asthmaticus heliox improves dyspnoea and PEFR and reduces muscle work and pulsus-paradoxus; also reduces peak AWP and CO2 in ventilated asthmatic patients; no evidence that it prevents intubation or affects mortality • COPD– reduces resistance to airflow during both inspiration and expiration; due to improved expiratory flow can have beneficial effects upon lung emptying and reducing dynamic hyperinflation and intrinsic PEEP; however, in patients with an acute exacerbation of COPD requiring NIV, heliox conferred no advantage in terms of mortality, intubation rates or ICU length of stay compared to oxygen air mixtures	

	• Bronchiolitis – found to have no beneficial clinical effects when used in ventilated children with bronchiolitis • Upper airway obstruction small case series have suggested clinical improvement in symptoms in patients with acute upper airway obstruction (laryngeal obstruction, croup, stridor, tracheobronchitis, mediastinal and laryngeal tumours) • Post extubation stridor– heliox has been demonstrated to provide better relief from the distress of post extubation stridor than oxygen air mixtures; no evidence that its use averts respiratory failure or extubation failure • Nebulisation – bench studies have suggested that heliox may facilitate greater progression of nebulized particles into distal airways than oxygen air mixtures

5	**What are the problems associated with its use?**	
Objective	*The candidate should show understanding of its limitations and problems.*	
Guidance for examiner	• "On / off" effect – heliox does not cure causes of increased airway resistance reduces resistance to airflow whilst a definitive treatment is sought • Cost – much more expensive than either oxygen or air; no studies have assessed the effect of heliox on length of ICU stay or time spent invasively ventilated in order to establish any potential offsetting of cost • Ventilator function – the different physical properties of heliox compared to oxygen air mixtures have led to problems with pneumotachometer function within ICU ventilators, which need calibrating for heliox – may lead to inaccuracy in volume measurement and underestimation of delivered tidal volumes. • Hot wire anemometers are more effective in measuring flows • Efficacy and limitations – at mixtures of less than 60% helium, heliox loses its favourable physical properties and has no benefits over oxygen air mixtures; this significantly limits its use in hypoxic patients requiring FiO2 > 0.4.	

Question Number	ICM SOE Example 4
Question Title	Endocrine question

Candidate Guidance	You will be asked to discuss hormones affected by critical illness

The information provided for each stem does not constitute a model answer, it is an aide memoire and guidance for the examiner.

Stem		

1	**Which hormones are secreted from the pituitary gland?**	
Objective	*This question tests the candidate's basic knowledge of the pituitary gland*	
Guidance for examiner	• Anterior lobe - TSH, ACTH, GH, FSH, LH, prolactin, gamma-melanocyte stimulating hormone, β lipotropin • Posterior lobe - vasopressin, oxytocin • Intermediate lobe – α and βMSH, γ-lipotropin	

2	**What happens to thyroid function in critical illness?**	
Objective	*Candidates should show a clear understanding of the difficulties in interpreting thyroid function tests in critically ill patients*	
Guidance for examiner	• Acute illness - rapid decline in T3 and rise in (inactive) reverse T3 (rT3). Brief rise in TSH/T4, but nocturnal rise in TSH absent. • Chronic illness - decreased T4 and T3 diminished pulsatile TSH, reduced TRH. T4 is converted peripherally to rT3 (inactive) rather than T3 because of altered enzyme action. • This low T3 syndrome is also called sick euthyroid syndrome. • Thyroid function tests are therefore unreliable in the critically ill.	

3	**What hormones are secreted from the adrenal gland?**	
Objective	*This is a basic question about the function of the adrenal gland*	
Guidance for examiner	• Catecholamines - adrenaline, noradrenaline, dopamine - controlled by sympathetic discharge as part of stress response • Glucocorticoids - cortisol, corticosterone • Androgens - dehydroepiandrosterone, androstenedione • Mineralocorticoids – aldosterone	

4	**How is the hypothalamic-pituitary-adrenal-axis affected by critical illness?**	
Objective	*The candidate should be able to describe the changes to the HPAA and its implications*	
Guidance for examiner	• Early stages - CRH and ACTH are increased, leading to a rise in cortisol, diurnal release is lost. • Chronic stages - Cortisol levels remain high, but ACTH decreases. Cortisol levels only return to normal in recovery phase. • Cortisol binding globulin levels decrease so the proportion of free cortisol is much higher	

5	**Describe the effects of vasopressin therapy**	
Objective	*Candidates should be able to describe what vasopressin is, why it might be used and how it works.*	

Guidance for examiner	• Increases retention of water through the collecting ducts in the kidney nephron via V2 receptors. This leads to increased insertion of 'aquaporins' – which transport solute free water back into the blood. • Increases vascular resistance via V1 receptors • Central nervous system: Involved in aggression, temperature regulation and memory amongst other things. • Catecholamine sparing • Used in Refractory shock – VASST study