# FFICM SOE Example Questions

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<tr>
<th>Question Number</th>
<th>ICM SOE Example 1</th>
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<tr>
<td>Question Title</td>
<td>Myasthenia Gravis</td>
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## 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.

### 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% of patients.
- The thymus is abnormal in 75% of patients with a thymoma in 10%.
- Associated autoimmune 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.

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<tr>
<th>5</th>
<th>What are the indications for ventilation?</th>
</tr>
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<tbody>
<tr>
<td>Objective</td>
<td>The candidates should be able to give a clear assessment of the patient</td>
</tr>
</tbody>
</table>
| 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) |
### Stem 1
**What factors do you consider when you chose an antibiotic?**

<table>
<thead>
<tr>
<th>Objective</th>
<th>The candidate should be able to give a clear explanation of the factors affecting antibiotic choice</th>
</tr>
</thead>
</table>
| 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 |

### Stem 2
**Why might antibiotic therapy fail?**

<table>
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<tr>
<th>Objective</th>
<th>The candidate should have a clear understanding of the reasons for antibiotic failure.</th>
</tr>
</thead>
</table>
| 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 |

### Stem 3
**How do Beta-lactams work?**

<table>
<thead>
<tr>
<th>Objective</th>
<th>The candidate should be able to explain the mechanism of action of common beta-lactams</th>
</tr>
</thead>
</table>
| Guidance for examiner | - Beta-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 beta-lactam antibiotics are due to their relative affinity for different PBPs. To bind to the PBPs, the beta-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 beta-lactams than gram-negative bacteria.  
- Penicillins poorly penetrate mammalian cells and are ineffective in the treatment of intracellular pathogens |
<table>
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<tr>
<th>4</th>
<th><strong>How does the presence of a toxin producing Group A streptococci, affect your choice of treatment?</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Objective</strong></td>
<td>The candidates should be able to give reasoning for more complex antibiotic choices</td>
</tr>
<tr>
<td><strong>Guidance for examiner</strong></td>
<td>• Use of clindamycin to reduce toxin production. 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.</td>
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<th>5</th>
<th><strong>Which antibiotic levels in common use are monitored routinely and why?</strong></th>
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<tr>
<td><strong>Objective</strong></td>
<td>The candidate should be able to explain which antibiotics need monitoring, how and why</td>
</tr>
<tr>
<td><strong>Guidance for examiner</strong></td>
<td>• 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</td>
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<tr>
<td>Question Number</td>
<td>ICM SOE Example 3</td>
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<td>Question Title</td>
<td>Heliox</td>
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**Candidate Guidance**  
You will be asked about the properties and use of Heliox

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<th>Stem</th>
<th>What is heliox?</th>
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<td><strong>Objective</strong></td>
<td>A simple knowledge of what Heliox is, is required</td>
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</table>
| **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|

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<th>Stem</th>
<th>What are the physical properties of heliox?</th>
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<td><strong>Objective</strong></td>
<td>Candidates should be able to explain the properties of heliox which make it useful clinically</td>
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</tbody>
</table>
| **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)|

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<th>How might these be clinically useful?</th>
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<tr>
<td><strong>Objective</strong></td>
<td>This question is designed to ask the candidate about how heliox may be beneficial</td>
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| **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. |

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<th>Stem</th>
<th>In which clinical conditions has the use of heliox been used?</th>
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<td><strong>Objective</strong></td>
<td>This question is aimed exploring the candidate’s knowledge of its clinical application</td>
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| **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

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<th>What are the problems associated with its use?</th>
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<td>Objective</td>
<td>The candidate should show understanding of its limitations and problems.</td>
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| 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. |
### Stem 1
**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 β MSH, γ lipotropin

### Stem 2
**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.

### Stem 3
**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

### Stem 4
**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

### Stem 5
**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 |