Question 1

Short Title
You are phoned by the pre-assessment nurse

Section
Clinical - short case

Topic
Cardiac/Thoracic

Author
Mike Tremlett / Jonathan Purday

Opening question
A pre-assessment nurse asks you to see a 36 year old male listed for a nasal polypectomy. He is active, playing squash twice a week, and running regularly. He says that he was found to have a heart murmur in his teens but this was not followed up. Examination reveals an ejection systolic murmur at the left sternal edge.

How would you proceed?

Supporting information
ECG – CTH 5

Guidance to examiners
To pass (score 2) the candidate must reach HCM in a differential diagnosis, be aware of the resulting problems (especially LVOT obstruction) and principles of anaesthetic management.

Question
How would you proceed?

• Areas to consider:
  o Cause of murmur (innocent vs pathological – clinical features of each)
  o Possible pathological causes (congenital, calcified bicuspid valves, endocarditis [unlikely as patient is well])
  o Would you continue with op as pt is fit and well?

What further investigations would you ask for if any?

• Blood pressure = Normal (106/60)
• ECG (non invasive, cheap, easy to obtain)
• Transthoracic Echocardiogram

Here is his ECG. Is it normal?

• No. Very Abnormal. Shows Sinus Rhythm, evidence of left ventricular hypertrophy with T wave inversion in lateral leads. Normal PR and QTc. No pre-excitation. (LVH = S in V1 + R in V6 >35mm)
• Causes LVH = Hypertension, Aortic Stenosis, Athletic Heart, Hypertrophic Cardiomyopathy.

Tell them echocardiogram shows Concentric left ventricular hypertrophy. Probable Diagnosis = Hypertrophic Cardiomyopathy (HCM).
**Short Answer – Example Questions**

**What is HCM?**
- Autosomal Dominant condition
- Variable clinical spectrum – most asymptomatic. Very common: 1 in 500 of population
- Key problems:
  - All have hypertrophy of left ventricle (maximal LV wall thickness >15mm).
- May in addition have / develop:
  - Left ventricular Outflow Tract Obstruction (not present in 30% so term HOCM no longer used) =
  - Sudden cardiac death
  - Sudden cardiac death
  - Sudden cardiac death
  - Abnormal position mitral valve apparatus means leaflets of mitral valve = dragged into outflow tract obstructing flow from left ventricle. Causes:
    - Heart failure
    - Mitral regurgitation (eject, obstruct, leak)
  - Atrial fibrillation (atrial kick important as have poorly relaxing ventricle)
  - Myocardial ischaemia

**In patients with significant HCM, what principles would underlie general anaesthetic management?**
- CVS: No big case series, from first principles:
  - Avoid hypotensive events (worsen LVOT obstruction, and upset myocardial oxygen supply / demand balance). Maintain SVR
  - Avoid tachycardia – Myocardial oxygen supply / demand balance + time for ventricular filling with stiff ventricle, maintain SR, treat dysrhythmias aggressively (β- blockers useful)
  - Myocardial depression generally desirable (increased inotropy worsens LOT obstruction. Avoid inotropes if possible) ? CO monitoring
  - Consider arterial line, use TOE for cases with severe disease
  - Maintain good hydration and good venous return.
  - Blunt sympathetic responses, early treatment of hypotension with vasoconstrictors. Inhalational agents are recommended particularly Sevoflorane.
  - Regional anaesthesia is relatively contra-indicated but not relevant here

**How would you anaesthetise this patient for this operation?**
- Operation requires general anaesthetic
- Cardiology assessment/advice
- Discussion of airway management. Tube vs LMA
- Thorough cardiac assessment of rhythm / evidence of heart failure
- Avoid tachycardia/maintain SVR - many choices...

(Filler)
1) Intraoperative management of sudden onset Atrial fibrillation on table.
2) Cardiac arrest in HCM is a special circumstance. The use of inotropic agents is contraindicated if the arrest is thought to be due to LVOT obstruction likely to increase obstruction. α-Agonists, i.v. fluids, and rapid correction of arrhythmias are more appropriate.

Ref: http://ceaccp.oxfordjournals.org/content/9/6/189.full
Opening question
You anaesthetised a 34 year old woman for hysterectomy under general anaesthesia 6 weeks ago. The General Practitioner contacts you because the lady has seen him complaining that she was awake for part of the operation. What would you do?

Supporting information
Nil

Guidance to examiners
Fundamental topic for all anaesthetists.
To pass candidate must know incidence of awareness, must demonstrate how would manage patient compassionately and be able to categorise causes of awareness.

Question
• What would you do?
See patient promptly with senior colleague
• Obtain full history (timing of awareness, presence of pain, specific events + any specific phrases recalled)
• Review your anaesthetic record (FiO2, MAC inhalational agent used, ? raised pulse, BP, etc)
• Check not one of a series in same theatre / Critical incident form.

Define Awareness:
• Unexpected and explicit recall by patients of events that occurred during a GA.

Incidence of Awareness
• Multiple studies show incidence 1-2 per 1000 GAs (AIM 2004 US)
• Provisional NAP5 results suggests 1:15,000. Why the difference (actively looked for in AIM, passive reporting in NAP5)
• Majority = auditory recall of events (30% experience significant pain), intubation frequently felt
• 30% go on to have nightmares, anxiety, flashbacks or post traumatic stress
• Use of isolated forearm technique suggests even higher

Any groups at increased risk?
• Patient characteristics:
Patients with reduced cardiac reserve, difficult intubation, history of long term benzodiazepine, cocaine or alcohol usage
• Surgical groups
Short Answer – Example Questions

LSCS under GA, Cardiac Surgery, Trauma, Muscle relaxant usage.  
• Commonest at start and end of the procedure

Causes of Awareness?  
• Patient specific variability in anaesthetic drug requirements  
• Low levels of agents used deliberately because of low physiologic reserve (cardiac failure, hypovolaemia)  
• Physiological signs of awareness masked by other factors (eg: Beta blockade, pacemakers, NM blockade)  
• Equipment failure or misuse (Syringe drivers, vaporisers etc)  
• Human error

Describe your management of a patient who has experienced confirmed awareness?  
• Full and frank apology. Discuss how may be avoided with future operations.  
• Full documentation of meeting  
• Offer Counselling. Phone Defence organisation.

How may you pick up that patient may be aware intraoperatively?  
No monitor at present accurately measures depth of anaesthesia  
• Movement if unparalysed  
• Cardiovascular signs and lacrimation  
• Vapour concentration monitoring

Should we be routinely using monitors measuring electrical activity of the Brain (eg: BIS) during general anaesthesia?  
• No, although it should be considered when using TIVA  
• Discuss BAware Study (difference found between BIS use and not), RCOA advice, ASA Practice Advisory (2006) if time.  
• B-Unaware Study (2008) - BIS compared with ET volatile measurement, no difference in awareness
Question 3

Short Title
Tetanus and tracheostomies

Section
Clinical - Short case

Topic
Intensive care medicine

Author
Dr Mike Tremlett Autumn 2010

Opening question
A 64 year old farm labourer presents with a 48 hour history of increasing difficulty in swallowing, neck pains and stiffness, and episodes of severe spasms of his head and neck muscles. You asked to see him for possible ICU care.
What is the likely diagnosis?

Supporting information
Nil

Guidance to examiners
Whilst this is an uncommon disease in the developed world, it remains a major killer in the developing world and this author believes management remains relevant. Historically it has featured in the exam for more than 20 years, although not recently. Pass if aware of the clinical triad and some understanding of treatment particularly of the autonomic instability. Do not pass if cannot spot / treat tracheostomy obstruction.

Question
What is the likely diagnosis?
• Multiple possible but Tetanus must be considered.
• Differential diagnosis to include orofacial infection, dystonic drug reaction (prochlorperazine), hypocalcaemia, strychnine poisoning, hysteria.
The diagnosis of Tetanus was strengthened by further history of no vaccination for more than 40 years and puncture wound on arm.

What is tetanus?
• Caused by Gram positive rod (Clostridium tetani).
• Clinical features = triad of rigidity, muscle spasms and autonomic instability
• Autonomic instability = episodes of severe hypertension and tachycardia alternating with profound hypotension and bradycardia.
• Untreated death = due to acute respiratory failure (restrictive lung disease / aspiration / laryngeal spasm. In ventilated pts death now due to autonomic effects.

What is the underlying pathophysiology (briefly)?
• Bacteria = ubiquitous. May exist as spores in soil.
• Bacteria produces exotoxin (tetanospsamin) which is taken up by neurons, passes by retrograde transfer to cell bodies, causing prevention of release of neurotransmitters.
• Toxin has predominant effect on inhibitory (GABA) interneurons. Alpha motor neurons = first to be affected by loss of inhibition hence motor spasms as early clinical feature.
Dis-inhibited autonomic discharge leads to surges in sympathetic outflow and raised plasma catecholamine levels.

Management?
- Neutralise unbound toxin – Human Tetanus immune globulin
- Remove source of infection – Penicillin widely used but metronidazole probably better choice
- Control rigidity and spasms – Sedation with benzodiazepine. IPPV often needed required to correct respiratory failure or life threatening airway obstruction
- Control of autonomic dysfunction – Magnesium widely used as blocks catecholamine release from nerves and adrenal medulla, and reduces receptor responsive to catecholamines. Also is useful vasodilator and blocks neuromuscular junction (reduced spasms)
- (use of magnesium does not reduce the need for ventilator support)
- Severe cases require an ITU stay of 3-5 weeks commonly

This patient required a prolonged period of ventilation and a tracheostomy was performed and patient made a good recovery. He was weaned off ventilatory support, discharged to an HDU with the tracheostomy in place. 36 hrs post discharge you are called urgently as the patient has become unresponsive, hypoxic and is gasping for breath. His tracheostomy was performed 5 weeks previously. What would you do?
- ABC approach
- Attempt ventilation via tracheostomy
- If ventilation ineffective take tracheostomy tube out and put fresh tracheostomy tube in and try again.
  (Good tract should be present at 5 weeks.) Possibly use suction catheter as Seldinger technique
Fillers:
Management of tracheostomy tube displacement at 2 days post formation (tract not established)
What are the indications for tracheostomy?

Ref:
“Tychon the soldier was hit by an arrow in his back......He sounded like someone gnashing his teeth in a fury of rage. He was arched back in opisthotonos, his jaws locked together against his will. A friend forced some wine between his teeth, but Tychon could not swallow, and the liquid was expelled in spurts from his nostrils. The following day the iatros arrived. He whispers he said there was little he could do, with the exception of applying soothing plasters.” Hippocrates c425BC
Short Answer – Example Questions

**Question 4**

**Short Title**
Collapse on labour ward/amniotic fluid embolism

**Section**
Clinical - short case

**Opening question**
You are called to resuscitate a 45 year old, multiparous woman who has collapsed during labour. The midwife says that she had been breathless prior to this.

**Guidance to examiners**
Pass if they mention at least 5 possible causes of collapse and know how to manage a suspected case of AFE

**Question**
What might have caused this collapse?

**CVS**
- Haemorrhage/abruption
- Regional anaesthetic sympathetic block
- Cardiac disease (congenital, acquired)
- Embolism (amniotic, air, PE)
- Arrhythmias/vasovagal

**CNS**
- Convulsions (eclampsia, epilepsy, local anaesthetic toxicity)
- Intracranial lesion (stroke, tumour)

**Pharmacological**
- Local anaesthetics (toxicity, total spinal)
- Opioids (systemic and spinal)
- Misc – cocaine/drug/alcohol abuse

**Endocrine**
- Hypoglycaemia

**Others**
- Anaphylaxis, acute asthma, pneumothorax, aspiration, sepsis

**What are your main resuscitation concerns?**
- Labour ward staff less familiar with emergency equipment, invasive monitoring, drugs and possibly CPR drills
- Need to consider mother and foetus in case of cardiac arrest
- Presence of foetus makes CPR difficult
- Aorto-caval compression
- Risk of aspiration

**You suspect amniotic fluid embolism, what are the symptoms and signs of AFE?**
Clinical diagnosis characterised by acute hypotension or cardiac arrest, acute hypoxia or coagulopathy in the absence of any other potential explanation for symptoms/ signs observed.

**What is the incidence and risk factors for AFE?**
- 1.9 - 7.7 / 100,000 pregnancies. Maternal mortality between 11-43 %, neonatal survival 70-80%
Short Answer – Example Questions

- Older women, multiparous, forceful labour, medical induction/ tetanic uterine contractions, placenta accreta chorioamniotis, microsomia, IUFD, meconium stained liquor

What is the pathophysiology of AFE?
- Entry of amniotic fluid and fetal cells into the maternal circulation, triggering an inflammatory response similar to anaphylaxis with activation of complement cascade.
- Phase 1 – pulmonary artery vasospasm, pulmonary hypertension, hypoxia, myocardial damage, ALI.
- Phase 2 massive haemorrhage, uterine atony and DIC (may not be distinct phases)

What are symptoms and signs of AFE?
- Breathlessness/cough, tachypnoea, hypoxia
- Tachycardia, hypotension
- Confusion, agitation, headache, seizures (50%)
- Cardiac arrest
- Pulmonary oedema/ bronchospasm/ ALI
- Fetal distress/ bradycardia (<110bpm for 10 mins)
- Uterine atony
- DIC

What are the classical ECG changes?
- Tachycardia, R ventricular strain (S1 Q3 T3)

How will you manage her?
- Early multidisciplinary /consultant involvement
- Supportive, full monitoring/early invasive monitoring a-line/CVP, Serial ABGs, ICU when possible
- Continuously monitor fetus, facilitate early delivery , consider perimortem C/S in mothers unresponsive to resus (within 5 mins)
- Maintain uterine tone. Uterotonics / uterine artery embolisation in refractory cases
- Careful fluid balance, pulmonary oedema common, inotropic support, CO monitoring
- Correct DIC if present, involve haematology ?activated VIIa,

Fillers

Pulmonary embolism in pregnancy
Incidence between 1.4-4.2 %

What are the investigations for diagnosis of suspected PE in pregnant patient
Anticoagulants to be started and continued until PE excluded
ECG/ Chest x-ray
Compression duplex ultrasound if signs of DVT present, V/Q scans or CTPA ( computerised tomography pulmonary angiogram) if no signs if DVT
Abnormal Chest x-ray with clinical suspicion of PE perform CTPA. Patients to be advised that compared to CTPA, V/Q scan has a slightly increased risk of childhood cancer but lower risk of maternal breast cancer

Ref: UKOSS annual report 2015; Amniotic Fluid Embolism CEACCP 2007, vol7 (5), 153-156
RCOG guideline April 2015. theromboembolic disease in pregnancy and the puerperium
**Question 5**

**Short Title**
Grommets in a child with a cold

**Section**
Clinical - Short case

**Topic**
Paediatric

**Author**
Mike Tremlett

**Opening question**
You are asked to assess a 3 year-old child for planned bilateral myringotomies and grommets as a day case. The child has a runny nose. How would you approach this?

**Supporting information**
Nil

**Guidance to examiners**
Candidates to pass should have an understanding of day case criteria, have a rational approach to a child with an URTI and simple anaesthetic plan.

**Question**

**Areas to consider:**
- Appropriate as day case?
  - Factors may include:
    - Age – over 60 weeks post conceptual age (depends on unit)
    - Medical conditions (exclusions may include IDDM, sickle cell disease, poorly controlled asthma)
    - Social:
      - multiple siblings + single carer with no support
      - no mobile phone / landline
      - transport issues
      - distance from hospital (ideally not greater than 60 mins away, depends on circumstances and surgery)
    - Surgical:
      - not if operation requires continued post-op care or pain management
- Significance of runny nose?
  - History will differentiate allergic rhinitis from URTI
  - Severity of illness (Temp, colour discharge etc)
- Why is this operation being done?
  - Indication = reduced hearing due to glue ear usually (underlying Eustachian tube dysfunction)
Would you proceed if the child has a cold (temp 37.6°C, mucoid discharge 2 days, no chest signs)?

- Yes! Look for sensible discussion of reasons for and against
  - **For:**
    - Parents / child planned for it (time off work etc)
    - Untreated glue ear + cold commonly lead to acute middle ear infection
    - Complications of anaesthesia = minor and all easily treatable (laryngospasm, coughing, breath holding etc)
    - Duration of cold shortened by operative treatment
  - **Against:**
    - Definite increased risks of respiratory complications of GA with a URTI (especially high if intubating) (Viral myocarditis = v rare)

- **Conclusion:**
  - This author believes you should get on and do it following discussion with parents. Child as presented is not toxic and anaesthetic care does not need ET tube.

How would you anaesthetise this child for the operation?

- **Pre-op:**
  - Oral analgesia (paracetamol), topical cream. ?veins, midazolam premed if clingy anxious, tearful etc.
- **Anaesthesia:**
  - IV or gaseous induction, spont respiration (N2O, O2, Iso)
  - Raised incidence emergence delirium with Sevoflorane maintenance
  - Opioids not necessary
- **Post-op:**
  - Simple analgesia. Discharge criteria

(Filler) Management of laryngospasm at end of operation.
Question 6

Short Title
Neurological deficit after stenting of the thoracic aorta

Section
Clinical - short case

Topic
Vascular

Author
Simon Howell

Opening question
You are called to see a 62 year old male on the High Dependancy Unit. He underwent elective stenting of a thoracic aortic aneurysm (Thoracic EndoVascular Aortic Repair TEVAR) 6 hours ago. The procedure was performed under combined spinal epidural anaesthesia. The nursing staff are concerned that, although the epidural infusion was discontinued two hours ago, the patient cannot move his legs and is showing no evidence of recovery of his sensory or motor block. Why may he still be unable to move his legs?

Supporting information
Nil

Guidance to examiners
Candidates should be aware of the differential diagnosis and management of protracted neurological deficit in this setting. Detailed knowledge of the management of a spinal drain is not required and information on this is provided to assist the examiners.

Question
Why may he still be unable to move his legs?
• protracted local anaesthetic neurological blockade,
• epidural haematoma,
• spinal cord ischaemia due to:
  o hypotension during procedure
  o occlusion of spinal cord blood supply by the stent (intercostals vessels).
• embolic neurological injury.

How would you tell between these possible causes?
History / notes review / nursing observations:
• Establish time course of neurological deficit.
  o Was there recovery followed by deficit or no recovery from the initial block?
  o What is the temporal relationship to LA administration?
• Were there technical difficulties with the CSE?
• Have there been intra-operative or postoperative episodes of cardiovascular instability
• Is there evidence of a coagulation disorder? What anticoagulant and anti-platelet agents were given and when?
Examination:
• Confirm the presence and degree of the neurological deficit
Short Answer – Example Questions

What further investigation may be necessary?
• If there is any suspicion of epidural haematoma urgent MRI must be undertaken

How would you manage this patient?
Unless there is ongoing recovery indicating block regression you should.
• Ensure that the MAP is close to normal for the patient.
• Organise urgent MRI to exclude neuroaxial haematoma. If present will need urgent neurosurgical referral.
• Correct any coagulation deficit (after discussion).
• If there is no evidence of haematoma or ischaemia compromised spinal cord blood flow is possible.
Consider placing a spinal drain
  o Spinal cord perfusion is determined by MAP and CSP pressure. Placing a drain enables reduction of CSF pressure. (Detail below = exceptional candidates)
  o [Coagulation must be normal. Place drain below L1/2, Nurse flat, run at CSPressure of 10cm H2O.]
  o MAP must be maintained at or close to normal level.
  o After 24 hours the drain is clamped/closed and may be removed if there is no return of neurological deficit.

(Filler)

What are the problems associated with anaesthesia for planned placement of endovascular stents for Abdominal Aortic Aneurysms?
• Substantial co morbidities [coronary artery disease [50-70%], previous MIs [up to 50%], hypertension, renal impairment [10%], diabetes [10%].
• Elderly population
• Up to 4 hour procedure
• Chance of failure and needing to proceed to open procedure
• Undertaken in radiology suite (not theatre often)

(Filler) Choice of anaesthesia?
• Local anaesthesia + sedation vs Regional anaesthesia vs general anaesthesia
• Advantages disadvantages of local anaesthesia?