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ANAESTHESIA

Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

Perioperative Medicine

2008A9: What are the hazards of laser surgery for a vocal cord papilloma, and how can they be minimised?

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1. LASER – light amplification by stimulated emission of radiation. Monochromatic, non-divergent, focused onto small area.
2. Hazards:
    a. Pollution: plume containing vapour and cellular debris causing headache, pulmonary illness and theoretical cross contamination
    b. Inadvertent energy transfer:
        i. Blood vessels: misdirection of laser
        ii. Patient and staff: eyes and skin
        iii. Combustible products: fire hazard
    c. Gas embolism: coolant gas in Nd:YAG probes
    d. Hypoxia:
        i. Inadequate ventilation: FiO_2 limitation, jet ventilation
        ii. Smoke, debris
3. Risk minimisation:
    a. Systems:
        i. Accreditation and dedicated LASER officer
        ii. Staff training for LASER surgery, fire training
        iii. Communication with teams and theatre staff
        iv. Guidelines: equipment, personnel
    b. Preparation:
        i. Signs on covered windows
        ii. Equipment: LASER proof glasses for staff, cover reflective surfaces
        iii. Ventilated theatre with plume suction
        iv. Extinguisher available
    c. Technique of shared airway:
        i. Jet ventilation vs. spontaneous breathing room air
        ii. Avoid combustible preparations
        iii. Minimise FiO_2
        iv. Resistant airway devices (laser tape, laser flex, bivona fome cuff)
        v. Fill cuff with saline
        vi. Wet drapes around patient with wet gauze on eyes
    d. Surgical
        i. Skilled operator
        ii. Minimum laser power, time and density

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2008A-10: A patient with biventricular failure and an automated implanted cardiac defibrillator presents for elective surgery. Describe how the presence of the device influences your perioperative management.

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1. Biventricular pacing with an AICD is usually indicated in the context of severe LV failure to improve cardiac output by optimising ventricular synchrony (cardiac resynchronisation therapy).

   Indications: asynchrony (atrioventricular, inter and intraventricular)

2. Pre-operative:
   a. Cardiac history, examination and investigations:
      i. Degree of heart failure, underlying rhythm, current medications
      ii. Urgency/indication of surgery
      iii. Recent ECHO, EUC, CXR
   b. Consultation
      i. Cardiologist
      ii. Pacemaker technician with check in last 6 months
      iii. Type, model, mode, response to magnet, defibrillator, rate response
      iv. Storage info: arrhythmia, defibrillator discharge
3. Intra-operative:
   a. General:
      i. Have magnet available
      ii. Have external defibrillation / pacing pads
      iii. Case during day hours
   b. Monitoring:
      i. arterial line +/- invasive central monitoring
      ii. 5 lead ECG in pacemaker mode
   c. Pacemaker function:
      i. Deactivate anti-tachycardia, defibrillation
      ii. Do not switch to asynchronous as the device is therapeutic
      iii. Do not deactivate rate modulation
   d. Surgical:
      i. Avoid diathermy
      ii. If needed: bipolar better than monopolar
      iii. If monopolar: place pad away from chest site, perpendicular direction
4. Post-operative:
   a. HDU for monitoring
   b. Switch on anti-tachycardia
   c. Recheck pacemaker
   d. Post-op analgesia

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2008A12: Outline the issues in preoperative assessment specific to a patient with acromegaly presenting for transsphenoidal hypophysectomy.

Acromegaly is an endocrine syndrome resulting from excess GH secretion by a pituitary adenoma. Its important perioperative manifestations are due to:

a. Endocrine effects – hyper or hyposecretion of anterior pituitary hormones
b. Mass effect – macroadenoma compression of surrounding structures

Assessment

1. Consultation/Notes:
    a. Recent evaluation by endocrinologist and neurosurgeon
    b. Macro vs. microadenoma
    c. Previous surgery or radiotherapy
    d. Medical therapy:
        i. Octreotide: N+V, impaired glucose tolerance, bradycardia, long QT
        ii. Bromocriptine: postural hypotension
        iii. Steroid maintanence
2. History:
    a. Cardiovascular: association with cardiomyopathy, exercise tolerance, symptoms of heart failure, HTN,
    b. Respiratory: obstructive symptoms (OSA), positional stridor
    c. Neurological: symptoms of raised ICP, early morning headache, postural (worse supine), N+V, visual field defect (bitemporal hemianopia); nerve palsies
    d. Endocrine:
        i. increased bone, hand, jaw size
        ii. Cushing's, thyroid symptoms
        iii. Diabetes: perioperative tight BSL control
3. Examination:
    a. Patient size, weight and height – do theatre equipment accommodate
    b. Detailed airway assessment: macroglossia, macrognathia, OA TMJ, difficult BVM
    c. IV access issues (thick skin)
    d. Endocrine: Cushing's features, Hyper/hypothyroid
    e. Mass effect: detailed neurological exam
4. Investigations:
    a. ECG, CXR
    b. MRI/CT review: likelihood of ↑ICP, surgical difficulty
    c. Bloods:
        i. FBC, EUC
        ii. Endocrine: TFTs, ACTH/Cortisol, FSH/LH, GH, Prolactin
    d. Cardiac: ECHO, stress test
5. Consent:
    a. Lumbar drain
    b. Invasive monitoring
6. Post-Op:
    a. ICU/HDU bed: neurological monitoring

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2008B5: List and evaluate strategies to prevent perioperative thromboembolism for a patient having a radical prostatectomy.

VTE is a major cause of peri-operative morbidity, accounting for 10% of inpatient hospital deaths. Additional issues in this situation are: tendency to bleed intra-operatively with radical prostatectomy, and impaired post-operative mobility.

Measure	Advantage	Disadvantage
General		
RA vs. GA	↓ incidence of DVT in orthopaedic surgery (↓venous stasis, module inflammatory response), analgesia	No evidence for ↓PE, dense block limits mobility → ↑ DVT risk, risks of neuraxial block, still require GA
Surgical technique	Clean, fast operation with minimal trauma → ↓ blood loss, pain	
Non-drug	Can be sole prophylaxis in low risk	
Fluid	Pre, intra and post-op, Reduces risk of DVT (↓viscosity)	
TEDS	Non-invasive improve venous flow, and effective (50% reduction), ↓ incidence 75% combined with heparin	Expensive, use with pharmacological agents for ↑ effect, requires good fit, cannot use in PVD / CCF, skin breakdown, discomfort
Calf compressors		
Early mobilisation	Physiotherapy, good analgesia, treat PONV, early discharge	
Drug	Gold standard ↓ VTE risk by 60-70% alone	
Heparin SC		Bleeding, HITS
Clexane SC	More effective, predictable effect, no monitoring needed	Non-reversible, ↓ dose renal failure
Warfarin		Takes time to establish INR

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2008B7: Outline the implications of severe spastic cerebral palsy for anaesthesia for major orthopaedic surgery in a child.

Cerebral palsy is clinical entity of motor, sensory +/- cognitive impairment of varying degrees caused by congenital or acquired insults. Classified by extremity involved (mono/di/hemi/quadriplegia) and characteristics of dysfunction (spastic, ataxix, hypotonic, dystonic). There is a high frequeny of epilepsy.

Implications

System	Implication	Solution
General	Paediatric age – consent needed with parents Cognitive impairment – difficulties with explaining cannulation, PCA use Association with other clinical syndromes Drug dosing – lower weight than expected	Parental input early May require mild anxiolysis High index of suspicion on pre-admission clinic Weigh child
Respiratory	Aspiration risk – oesophageal dysmotility, lower LOS tone, impaired gastric emptying Poor Dentition Restrictive lung disease - ↓FRC, VALI Weak cough – prone to infections	Perform RSI, difficult airway trolley with paediatric sizing, start antacid prophylaxis Care with ventilation pressures and volumes Chest physio
Neurologic	Epilepsy – peri-operative seizures Meds may induce hepatic enzymes	Continue prophylaxis peri-op
Musculoskeletal	Thin skin, large joints, flexion deformities – prone to pressure injury, neuropathy, pressure ulcer Kyphoscoliosis – difficult epidural / spinal Prone to hypothermia Difficult IV access	Pressure area care – padding Continue anti-spasmodics Warming blanket, fluids, pre-post op EMLA cream
Surgical	Long operation time – blood loss, hypothermia, SPICA application requires patient movement down table – dislodgement ETT, IVC	G+H, early warming Care and depth of anaesthesia
Post-operative	Painful procedures May not be able to use PCA Sensitive to opiates Difficult to assess pain	Lumbar epidural placed under GA vs. nurse initiated prn vs. opioid infusion HDU bed Parents can help interpret pain

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2008B15: What symptoms and signs suggest the presence of sleep apnoea? How does the presence of sleep apnoea alter your anaesthetic plan?

Sleep Apnoea is a syndrome of disordered breathing and apnoea. Cessation of airflow at the mouth > 10sec, >5 episodes/hour

1. Symptoms:
    a. Respiratory:
        i. Night-time snoring
        ii. enuresis
    b. CVS:
        i. Pulmonary hypertension: SOBOE
        ii. RHF: oedema, plethora, cyanosis
    c. CNS:
        i. Daytime somnolence, reduced performance
        ii. Children – Irritability, hyperactivity
    d. Medications:
        i. Use of alcohol, opioids
2. Signs:
    a. General:
        i. Obesity
        ii. Congenital disease – Down's syndrome
    b. Respiratory
        i. Apnoeic episodes > 30 sec
        ii. Changes to airway anatomy:
            1. large neck circumference
            2. tonsillar, adenoid enlargement
            3. craniofacial abnormalities
            4. narrow maxilla, mandible
            5. macroglossia
    c. CVS:
        i. HTN
        ii. ↑JVP, RV heave, TR
3. Anaesthetic plan: high risk of airway obstruction
    a. Pre-op:
        i. Assess risk: STOPBANG questionnaire
        ii. Early consultation with specialist pre-op for optimisation
        iii. Investigations:
            1. Sleep studies
            2. ABG
            3. ECG, CXR
            4. ECHO – pulmonary HTN, RV failure
        iv. Plan:
            1. Alcohol, smoking cessation, weight loss
            2. Post-op HDU
            3. Bring CPAP machine
    b. Intra-op:
        i. Regional preferable to GA
        ii. Difficult IV access
        iii. Avoid long-acting sedation – midazolam, morphine

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iv. Airway:
    1. Difficult BVM, LMA, intubation
    2. Rapid desaturation
    3. Pre-oxygenate head up
    4. ↑ incidence of GORD
v. Maintenance:
    1. short acting drugs with small VD (desflurane, remifentanil)
    2. minimise opiates and relaxants
vi. Ventilation - PEEP
vii. Extubate upright and awake, fully reversed

c. Post-op:
    i. Short-acting opioids
    ii. HDU bed with monitoring
    iii. Beware of REM patterns – highest risk day 2-3 due to rebound ↑ REM sleep
    iv. Use CPAP machine
    v. Consider overnight stay if day case

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2009A5: Discuss issues relevant to the perioperative management of a 70-year old patient with a 10 year history of Parkinson's disease undergoing a Total Knee Replacement. He is currently taking levodopa/carbidopa 5 times a day.

Parkinson's is a neurodegenerative syndrome due to imbalance of dopamine and acetylcholine, characterised by: bradykinesia, resting tremor and rigidity.

1. Pre-operative considerations:
    a. Recent specialist consultation:
        i. Optimisation
        ii. Medication compliance
    b. Past anaesthetic history
    c. History and examination:
        i. Symptoms: extent of bradykinesia, rigidity,
        ii. Extra-Parkinson's syndrome:
            1. frontal dementia
            2. autonomic effects: postural hypotension, gastric stasis, urinary retention, sialorrhoea
        iii. Bulbar symptoms: dysphagia
        iv. Contractures: difficult IV access, regional
    d. Consent:
        i. complications, need for ventilation, rehabilitation issues
        ii. difficulties due to dementia, depression
    e. Advice:
        i. Continue medications as usual and bring to hospital
2. Intra-operative:
    a. Monitoring: consider arterial line, nerve stimulator, IDC
    b. Regional preferable to GA – monitor neurology, avoid sedation (if not demented)
    c. Induction: beware aspiration risk
    d. Drug interactions:
        i. Beware NMS – withdrawal of levodopa
        ii. Avoid relaxants, anticholinesterases and anticholinergics (precipitate symptoms)
        iii. Avoid anti-dopaminergic drugs: maxalon, droperidol. Use domperidone.
        iv. MAO-B inhibitor (selegiline) with pethidine → hypertension
        v. Opioids ↑ muscle rigidity
3. Post-operative:
    a. Continue levodopa in recovery. No IV formulations therefor may need to administer through NG tube
    b. Specialist consultation in ward – neurologist, rehabilitation
    c. Falls risk
    d. Multi-disciplinary: dietician (nutrition), OT, rehab team (home support)

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2009A9: Evaluate the usefulness of initiating beta-blocker therapy to prevent myocardial ischaemia in a 65-year-old for surgery for peripheral vascular disease in four days' time.

Issues:

1. If the patient is already on a beta-blocker, this should be continued
2. The POISE trial represents the largest trial on this subject. It shows that high dose peri-operative metoprolol is associated with a significant:
    a. ↓myocardial ischaemic events
    b. ↑stroke, hypotension and mortality
3. The timing of commencement is controversial. Some studies (POISE) start the day of surgery, others commence weeks prior.
4. Maximum benefit could be obtained by titrating to heart rate (<60bpm).

This patient:

1. Femoral-popliteal bypass represents high risk surgery for cardiovascular events (>5%)
2. Assess cardiac risk:
    a. major active cardiac conditions: recent MI within 12 weeks, unstable angina, decompensated CCF, uncorrected CHD, major valvular disease (especially AS), unstable arrhythmias
    b. major cardiac risk factors: stable IHD, CCF, DM, CVA, CRI
    c. minor risk factors: age > 70, untreated HTN, rhythm not SR, controlled AF,
    d. Assess level of function: < 4 METS → high risk
    e. CABG/revascularisation in last 5 years, symptom free → ↓risk
3. Contraindications to beta-blockers: asthma/COPD, decompensated heart failure, heart block / bradyarrhythmias, Ca^{2+} blocker, severe PVD
4. Indications:
    a. ischaemic heart disease, HTN, angina, tachyarrhythmia.
    b. Inducible ischaemia on pre-operative testing

Conclusion: decision to commence beta-blocker is not clear cut and necessitates balances assessment of cardiac risk, indications and contraindications. Without further information I would not start a beta-blocker as there is insufficient time to titrate dose, and uncertain risk/benefits.

Addition: Evidence from trials -

1. MAVS 2006 (metoprolol after vascular surgery) – vascular surgery, no difference in mortality or cardiac events, but ↑ bradycardia and hypotension requiring treatment in treatment group.
2. POBBLE 2005 – vascular surgery, no difference in mortality or cardiac events, but ↑ bradycardia and hypotension requiring treatment in treatment group.
3. POISE 2008 - > 8000 patients multicentre RCT. ↓ cardiovascular events (HR 0.83), but increased risk death (HR 1.3), stroke (HR 2.2). For every 1000 patient treated in POISE: 15MIs prevented, 7 AF prevented, 3 CABG post-op avoided, 8 more deaths, 5 more strokes and 53 episodes significant hypotension. Issues: used large dose metoprolol (100mg daily) vs. titrated dose.

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2009B2: List the advantages and disadvantages of tight perioperative glycaemic control on a diabetic patient who is on insulin. How would you manage the glycaemic control of such a patient having a minor procedure under general anaesthesia?

Glucose homeostasis is important for: aerobic metabolism, immune function (wound healing, infection control), fluid and electrolyte homeostasis. Tight glycaemic control can be defined as BSL 4.5-6 mmol/L in the perioperative period. This can be done with:

- Sliding scale
- Insulin infusion +/- dextrose

Advantages	**Disadvantages**
Evidence suggests: Improved outcome in ICU post CABG	Evidence suggests: ↑ mortality in ICU (NICE-SUGAR), ↑ hypoglycaemic episodes – neurological impairment
Improved wound healing	↑ cost, investigations
Reduced infection rates	May require HDU/ICU
Acute hyperglycymia: HONK, ketosis, diuresis, electrolyte disturbance, catabolic state	
Motivation of patient to continue good outpatient management	

Management aims: BSL 5-10mmol/L

- Avoid significant hyperglycaemia
- Avoid hypoglycaemia

1. Pre-op:
    a. Pre-admission clinic:
        i. Assessment of baseline control: HbA1C, usual readings (diary)
        ii. Assessment of complications: neuropathy, nephropathy, cardiac
            1. Assessment of medications: Hypoglycaemic agents, Insulin
        iii. Specialist review if poor control
    b. Plan: 1st on morning list
    c. Instructions:
        i. Fast 6 hours solids, 2 hours CF
        ii. With-hold metformin for 24 hours prior, other PO hypoglycaemics for 12 hours prior
        iii. Give ½ usual basal insulin in the morning, full dose in evening
        iv. Check BSL mane, if <4 have apple juice, come to hospital early
        v. Bring meds to hospital
2. Intra-op:
    a. If regional / awake, and good pre-op control, do not need further monitoring
    b. If GA, consider one-off BSL intra-op
    c. Dextrose-free fluids unless hypoglycaemia

Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

   d. Maintain normovolaemia, normothermia
3. Post-op
   a. BSL check hourly in recovery, chart sliding scale
   b. Continue usual insulin once eating
   c. If prolonged fasting, give IV dextrose 5% 100mLs/hour, + insulin titrated
   d. Discharge if stable
   e. If erratic BSLs, consider overnight admission

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2009B9: What are the indications for prophylaxis against perioperative bacterial endocarditis? Justify your choice of antibiotics.

Indications for prophylaxis – as per new ACC/AHA guidelines. This has narrowed the indications resulting in fewer instances of prophylaxis. Best risk reduction is good dental hygiene and main risk is poor hygiene.

1. Patient factors:
    a. Prosthetic valve or other foreign intracardiac material
    b. Previous infective endocarditis
    c. Congenital heart disease:
        i. Cyanotic – Uncorrected, or palliated
        ii. Acyanotic - Corrected < 6 months, or residual defects at site/adjacent to prosthesis
    d. Cardiac transplant with valvuloplasty
    e. Rheumatic heart disease + Indigenous
2. Surgical factors
    a. Airway contamination: Dental surgery, Nasal intubation, Rigid bronchoscopy, Airway trauma, Adenotonsillar surgery
    b. Urinary tract: instrumentation in the presence of UTI

Choice of antibiotics

Factors:

1. Require coverage of oral flora (step viridans, staph epidermidis, enterococcus)
2. Inexpensive
3. Easy to administer, take: PO take 30min prior, IV give within 30min incision

1. Airway:
    a. Amoxicillin 2g (50mg/kg) PO or Ampicillin 2g (50mg/kg) IV
    b. Penicillin allergy:
        i. Clindamycin PO 600mg (20mg/kg) OR cephalexin PO 2g (50mg/kg)
        ii. Clindamycin IV 600mg OR cephazolin 1g
2. Urinary tract:
    a. Add Gentamicin IV 120 mg (1.5mg/kg)
    b. Add Vancomycin IV 1g (20mg/kg) over 1-2 hours if penicillin allergic

Changes from previous guidelines (no longer included): bacteraemia of daily activities are common, very large NNT, risk of antibiotic administration outweighs benefits

1. Patient factors: HOCM, MVP, previous successful CHD repair
2. Surgical factors: diagnostic procedures of GI and respiratory tract, genitourinary procedures

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2009B14: A patient has smoked 20 cigarettes a day for over 25 years. What are the expected physiological changes that would occur in the first three months following cessation of smoking, including a time frame for these changes? What are the clinical benefits, with regard to anaesthesia, of smoking cessation in this patient?

Physiological changes of smoking cessation

Change	**Cessation Time frame**
Carbon monoxide – $T_{1/2}$ 4 hours, impairs O_2 delivery by binding to Hb 200x affinity, shifting HbO_2 DC left, and inhibiting cytochrome oxidase, negative inotrope, arrhythmogenic	$T_{1/2}$ = 4 hours → 12 hours
Airway reactivity - ↑ risk of bronchospasm, laryngospasm	
Nicotine – vasoconstriction, sympathomimetic on heart. NRT still recommended perioperatively, even in CHD.	$T_{1/2}$ = 30min → 2 hours
Ciliary function and hypersecretion: ↑ secretion for 2 weeks post cessation ↓ to normal levels by 6-8 weeks ↓ clearance of secretion for 2 months post-cessation (↑ risk pneumonia)	Starts 2-3 days ↑ secretion 2 weeks ↓ normal 6-8 weeks
Hypercoagulability – polycythaemia, ↑ fibrinogen	2 weeks
Immune function – impaired phagocytic function	6 months
Respiratory function tests – improve due to ↓ small airway reactivity	4-6weeks
Metabolism – induction of enzymes → ↑ analgesic requirements	6-8 weeks

General: full benefit takes 6 months cessation, but benefits begin immediately. Despite ↑ risk pulmonary complication if 2-8 weeks of cessation, long term benefit outweighs risk.

Clinical Benefits of Smoking cessation

1. Airway: ↓ laryngospasm, bronchospasm, cough
2. Anaesthesia: MAC and opioid requirements, return to normal hepatic metabolism,
3. Cardiac: ↓ perioperative cardiac risk
4. Respiratory: Improved respiratory V/Q matching, improved O_2 carrying capacity, ↓ mucous secretion, ↓ atelectasis, chest infection → ↓ risk of desaturation, hypoxia
5. Post-op:
    a. Improved analgesia efficacy
    b. ↓risk infection, improved wound healing
    c. ↓ ICU admissions, hospital stay and cost

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

20010A2/2005A10: List the hazards to the patient associated with the prone position under general anaesthesia. How can these be minimised?

Problems	**Minimisation**
Positioning no monitoring, line/airway disconnection, ABC instability.	Disconnect lines for transfer to avoid entanglement Pre-oxygenate, and ensure adequate MAC, haemodynamic stability prior to disconnection. Team leader to co-ordinate, airway/neck support. Reconnection in order of importance: circuit, pulse ox, IV access
Airway: Lack of access Change in position during positioning / OT → fall out, endobronchial, oedema Risk of damage to neck / spine	Intubate, secure with tape. Recheck clinically with every position change In line traction with position change
Respiratory: Splinting if abdomen, thorax not free to move (↓FRC, ↑ ventilation pressures) Otherwise respiratory function improves in prone (↑FRC, ↓V/Q mismatch)	Support thorax from clavicle to iliac crests with table support (parallel chest/thorax rolls)
Cardiovascular: IV access IVC compression → ↓VR, ↑ lower limb, epidural pressures risk DVT Epidural venous congestion → ↑ bleeding spinal Risk of gas embolism Resuscitation: CPR impossible in prone	Insert extra IVC, arterial line ensure all are working prior to and after positioning, keep visible if possible. Calf compressors MAP > 70mmHg using fluids, vasopressors Cross match, cell saver Avoid Hypovolaemia (↓ venous pressure)
Neurological – ↑ ICP as head just below heart Cerebral ischaemia – compression of carotid / IJV Ischaemic optic neuropathy due to ↓ MAP, ↑ oedema	Support weight of head with bony prominences and malar areas with special mask. Avoid neck rotation. Face mask padding, eye lubricant and pads.
Pressure areas – Face, pelvis, breasts, ankles, knees, genitalia dependent areas → oedema → ↑ risk Upper limb – brachial plexus, ulnar nerve Lower limb – LFCN, sciatic Vascular – femoral vessel compression	Padding on ulnar nerve, breasts, groin Limit shoulder abduction, elbow flexion < 90 Arms in neutral position Ensure padding does not extend below iliac crest to compress femoral vessels
GI / Renal - ↑ intra-abdominal pressures → prone to ischaemia, ↓GFR	Keep abdomen free as above Vigilance of urine output
Equipment: requires special table, + servicing Staff: risk of injuries	Co-ordinator, at least 4 people required (head, legs, 2 for tors)

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2010A3: A 20-year old female with a BMI of 48 presents for an elective diagnostic laparoscopy for endometriosis. She has no other medical conditions. Describe the potential problems associated with anaesthetising this patient.

Morbid Obesity

1. Pre-op:
    a. Co-morbid disease requiring optimisation – diabetes, obstructive sleep apnoea
    b. Difficult IV access
    c. Equipment –
        i. bed, railings
        ii. monitors – NIBP, pulse ox
        iii. patient transfer
2. Intra-op:
    a. Induction:
        i. Airway:
            1. ↑ neck circumference → difficult laryngoscopy
            2. rapid desaturation
            3. ↑ risk aspiration (↑ gastric volume, ↓pH)
        ii. Drug dosing difficulty:
            1. Lipid soluble → based TBW
            2. Water soluble → based IBW
    b. Maintenance:
        i. Breathing: restrictive lung disease → ↓FRC, compliance, ↑VQ mismatch
        ii. ↑ ventilator pressures, can't do under LMA → need intubation, barotrauma risk
        iii. Pressure area risk
    c. Emergence:
        i. High risk of airway obstruction
        ii. Slow emergency with sevoflurane
3. Post-op:
    a. High risk of respiratory complications
    b. Sequestration of GA / opioids in fat → high risk of post-op airway obstruction
    c. ↑risk DVT

Laparoscopy and pneumoperitoneum

1. Induction
    a. Risk of venous air embolism
2. Maintenance: ↑ duration due to difficulty, ↑ likelihood of conversion to open, higher pressures required, Trendelenburg compounds this
    a. CVS: ↓↓ VR → ↓BP, CO; prone to ischaemia of brain, heart. Compounded by PEEP
    b. Resp: ↑WOB, airway pressures,
    c. CNS: ↑$ETCO_2$ → ↑ICP
    d. Renal: ↓UO, GFR
3. Post-op:
    a. ↑ PONV
    b. ↑ Pain - ↑ duration of surgery

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2010A6: A 40-year old man with HOCM presents for elective laparoscopic cholecystectomy. Describe the principles of intraoperative haemodynamic management for this patient. How would you manage hypotension post induction of general anaesthesia in this patient?

HOCM is a familial autosomal dominant myocardial condition characterised by LV hypertrophy and dynamic LVOT obstruction during systole. Cardinal features are asymmetrical hypertrophy of the IV septum, as well as systolic anterior motion of the anterior mitral valve causing systolic obstruction +/- MR.

Principles of haemodynamic management

1. Based on pre-operative assessment of severity of condition:
    a. History and examination: exercise tolerance,
    b. Investigations:
        i. ECHO/TOE: LVH, diastolic dysfunction, SAM (systolic anterior motion of anterior mitral valve)
        ii. Recent cardiology review
        iii. ECG
    c. Current treatment:
        i. Drugs
        ii. AICD
2. Risks of HOCM:
    a. Dynamic outflow obstruction – hypertrophy, and SAM mitral valve exacerbated by ↑ contractility, ↓ pre/afterload
    b. Diastolic dysfunction – LVH, stiffness, tachycardia, and AF
    c. Myocardial ischaemia – LVH, ↑ wall tension,
    d. Arrhythmias
3. Physiological principles: Adequate preload, afterload, Low-normal heart rate (60-80), Low contractility:
    a. Avoid sympathetic stimulation
    b. Beta-blockers
    c. Immediate cardioversion of tachyarrhythmias
4. Laparoscopy: causes ↓ preload, ↑ sympathetic stimulation with insufflation and reverse Trendelenburg
    a. Limit inflation pressures 155mmHg and gradually increase
    b. Slow changes of position, and limit head up
5. Endocarditis prophylaxis no longer routinely necessary

Hypotension post induction – potential emergency requiring simultaneous diagnosis and treatment

1. Recheck airway, breathing → 100% O_2
2. Exclude obvious reversible cause: may need on table ECHO (TTE/TOE) to differentiate between HOCM, ischaemia, hypovolaemia
    a. Obstructive: pneumothorax, air embolism
    b. Distributive: anaphylaxis
    c. Cardiac: ischaemia, arrhythmia
    d. HOCM: worsening LVOT obstruction due to ↓ preload, ↓afterload
3. If severe, call for help, inform surgeon

Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

   a. ↑ preload – fluids, vasopressors
   b. Release pneumoperitoneum, normalise position
4. Treat cause:
   a. Induction agent → vasopressor
   b. Arrhythmia → immediate cardioversion
   c. Anaphylaxis → adrenaline
   d. HOCM → fluids, vasopressors, beta-blockade (if ↑HR), NO INOTROPES, minimise SNS stimulation (analgesia)

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2010B3: A 45-year old man with a longstanding history of alcoholism is booked for upper gastrointestinal endoscopy and banding of oesophageal varicies following an episode of haematemesis. How is the severity of this patient's liver disease assessed? How do these findings influence your evaluation of this patients perioperative risk?

Assessment of Liver Disease

History, Examination, Investigations to review:

1. Hepatic:
    a. Portal hypertension: haemorrhoids, caput medusa, previous varicies, ascites, splenomegaly, fluid status (need for drainage, TIPS)
    b. Co-morbid disease: viral hepatitis, HIV
2. Cardiovascular: consider TTE
    a. Hyperdynamic circulation – ↓SVR, hypotension
    b. Cardiomyopathy
    c. Ischaemic heart disease
3. Respiratory:
    a. Pleural effusions, splinting from ascites, pulmonary hypertension
    b. Porto-pulmonary anastomosis
4. Renal:
    a. Salt, water retention
    b. Hepatorenal syndrome – poor prognosis
5. Haematological:
    a. Anaemia
    b. Thrombocytopenia
    c. coagulopathy
6. CNS:
    a. Wernekie's encephalopathy, Korsakoff psychosis
    b. Hepatic encephalopathy (Grade 0 – asymptomatic, 1 – drowsy orientated, 2 – disorientated, 3 – stupor, rousable, 4 – coma)

Evaluations of perioperative risk

1. Patient risk assessed as above. This can be quantified using specific scores: Child-Pugh Score

Marker	**1**	**2**	**3**
Albumin	>35	28-35	<28
Bilirubin	<25	25-40	>40
INR / PT	<1.7	1.7-2.3	>2.3
Ascites	none	mild	Severe
Encephalopathy	none	Grade I/II	Grade III/IV

Grade	**Points**	**1 year survival**	**2 year survival**	**Periop mortality**
A	5-6	100%	85%	< 5%

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BSc(Med) MBBS(Hons) FANZCA

B	7-9	80%	57%	5-50%
C	10-15	45%	35%	>50%

2. Surgical risk
    a. Blood loss – amount, duration, ongoing or controlled
    b. Method - banding, glue, injection, conversion to open
3. Anaesthetic
    a. Pre-op complications: renal failure, cardiac ischaemia
    b. Optimisation: transfusion, fluid resuscitation,
4. Risk of complications –
    a. Encephalopathy
    b. Renal failure
    c. Cardiac complications
    d. Haemorrhage
    e. Sepsis

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2010B7: Describe the common classification code for permanent pacemakers. Outline the principles involved in the perioperative management of patients with a permanent pacemaker.

Pacemaker Classification

Letter	Name	Description
I	Pace	V – ventricular, A – atrial, D – dual, O – none
II	Sense	As above
III	Action	T – trigger, I – inhibit, D- dual, O – none
IV	Programmability	R – rate response, O – none, P – simple, M – multi, C - communicating
V	Anti-tachycardia	S – shock, P – pace, D – dual, O – none

Principles of perioperative management

Overall aims are to:

- Maintain cardiac output
- Prevent device malfunction: inhibition, triggering, asynchronous pacing, damage, inappropriate shock

1. Pre-operative: notes, history, examination and referral
    a. Pacemaker:
        i. Indication – bradycardia, tachycardia, heart failure,
        ii. Type – as above, brand name
        iii. Last service – battery, setting, recent activity
        iv. Underlying rhythm
        v. Malfunction – shocks,
        vi. Response to magnet
    b. Patient:
        i. Past anaesthetic history
        ii. Detailed cardiac history
    c. Surgical: operation type, duration, need for diathermy/MRI
    d. Investigations:
        i. ECG, CXR (check position)
        ii. Bloods – EUC, CMP
    e. Referral:
        i. Pacemaker technician, clinical
        ii. Cardiologist
    f. Pacemaker action:
        i. Turn off AICD – diathermy detected as VF → shock
        ii. Turn off rate modulation if site near sensor
        iii. Turn off anti-tachycardia function (if AICD function)
        iv. Consider reprogramming to asynchronous – AOO, VOO, DOO
2. Intra-operative:
    a. Equipment:
        i. Magnet on standby – generally inhibits sensing → asynchronous pacing (if no AICD) OR inhibits anti-tachycardia if AICD, indicated if PM dependent.
        ii. External pacing pads ready or positioned away from PM box
    b. Monitoring:
        i. ECG – turn on pacemaker mode (can see pacing spikes)

   ii. Consider arterial line – quick feedback of cardiac output
c. Surgical:
   i. Minimise diathermy: bipolar preferable, pads axis perpendicular to PPM. Diathermy may inhibit, reset or damage PM output

3. Post-operative:
   a. Refer back to clinic and reactivate anti-tachycardia, rate modulation and AICD.

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2010B11: A 78-year old female presents for fixation of a displaced femoral fracture. She has longstanding mitral regurgitation and is known to have a mean pulmonary artery pressure of 60 mmHg. She reports orthopnoea but is not short of breath at rest. What are the issues of concern in your preoperative assessment? How would you manage pulmonary vascular resistance perioperatively?

Preoperative Assessment issues

1. Patient:
    a. Cardiovascular: MR with severe pulmonary hypertension (mean PAP > 50mmHg) is an active cardiac condition requiring optimisation. There is a risk of worsening pulmonary HTN, R heart failure, cardiogenic shock and death.
        i. Assess for other cardiac disease (failure, valvular, ischaemic, myopathy), functional capacity.
        ii. Severity markers:
            1. BP, JVP, displaced apex, loud P2, RV heave, hepatic congestion, peripheral oedema, ascites
        iii. Assess if cardiac reason for fall (syncope)
        iv. Consult cardiologist
        v. ECG (AF, RA strain), ECHO required
    b. Trauma:
        i. exclude other complications – concealed haemorrhage, head injury, other broken bones, long lie
        ii. Requires – FBC (blood loss), GH, EUC, CMP, consider troponin
        iii. Consider volume resuscitation and regional pain relief
2. Anaesthetic:
    a. Timing of surgery – consult with orthopaedics and cardiology, prefer to optimise prior to surgery
    b. Consent and risk discussion
    c. Position: patient orthopnoeic supine, so this may preclude regional depending on severity
3. Surgical:
    a. No immediate surgical urgency → can wait until appropriate investigations done
    b. Risk of fat embolism increases with delay of surgery

Management of pulmonary vascular resistance

Overall Aims:

1. Minimise pulmonary vascular resistance, maintain SVR
2. Minimise effects of MR: promote forward flow of blood as regurgitant jet worsens pulmonary HTN

1. Pre-op:
    a. Consider agents to reduce PAP if an acute issue: nitric oxide, Ca^{2+} blockers, bosentan, IV prostacyclin
    b. Optimise pain relief, anxiety, avoid hypoxia, hypotension
2. Intra-op:

a. Preparation: resuscitation equipment available, nitric oxide
b. Monitoring: arterial line consider PICCO, large bore IVC, IDC, 5 lead ECG, consider CVC with PAC or TOE
c. MR: aim full (adequate fluid to fill LV), normal fast HR (80-100), avoid ↑SVR (keep BP close to baseline), keep in SR (AF requires immediate shock)
d. Pulmonary vascular resistance:
    i. Avoid factors which ↑ PVR: hypoxia, hypercapnoea, acidosis, hypothermia, pain
    ii. Ventilation:
        1. preferable to keep spontaneously breathing → use regional technique or LMA
        2. if ventilation necessary, no PEEP, minimise inspiratory pressure, fast rate, low TV
    iii. Minimise extent of MR as above
    iv. Pharmacotherapy:
        1. Avoid N_2O, ketamine, adrenalin
        2. Inhaled nitric oxide 5-20ppm, Prostacyclin
        3. Isoflurane the ideal volatile
e. Systemic vascular resistance: methods to ↓PVR may also ↓SVR causing hypotension
    i. Balance need to ↑SVR for coronary perfusion vs. need to ↓ SVR for mitral regurgitation.
    ii. Careful use of vasopressors eg. noradrenaline

3. Post-op:
    a. HDU/ICU post op
    b. Regional analgesia

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2010B13: Outline the principles of an initial management plan for diabetic ketoacidosis, having regard to the physiological derangements involved.

Diabetic ketoacidosis is a medical emergency due to insufficiency of insulin characterised by hyperglycaemia, dehydration, hyperkalaemia and ketoacidosis.

Principles of Initial management Plan

1. General principles:
    a. Consult endocrinologist as this is an endocrine emergency
    b. Transfer to HDU
    c. Monitoring:
        i. ECG
        ii. Bloods: ABG q2 hourly initially then q4 hourly when BSL < 15 or acidosis resolved
        iii. IDC for urine output
        iv. Arterial line especially if haemodynamically compromised
        v. Large bore IVC
    d. Treat underlying cause: infection, ischaemia, fasting, dehydration, non-compliance
    e. Complications:
        i. High risk infection – consider perioperative antibiotic prophylaxis
        ii. CV collapse, arrhythmia, coma
        iii. Respiratory collapse (fluid overload)
2. Hyperglycaemia
    a. Assess: : insulin deficit → hyperglycaemia → fatty oxidation → ketosis
    b. Aim: normalise BSL gradually, aim to ↓1mmol/hour down to 6-10mmol/L
    c. Method:
        i. Insulin infusion starting 0.1IU/kg
        ii. Titrate according to local protocol
        iii. When BSL < 15mmol/L, add 5% dextrose to fluid infusion
3. Hypovolaemia:
    a. Assess: severity of dehydration due to osmotic diuresis from glucose
        i. Mild <5%: normotensive, ↑HR, ↑RR, normal behaviour, CR normal
        ii. Mod 5-7%: ↑↑HR, ↓BP, ↑↑RR, agitated → drowsy, CR > 2 sec
        iii. Severe >7%: hypotensive, comatose, CR > 4sec
    b. Aim: replace fluid deficit over 8 hours
    c. Method:
        i. Resuscitate with normal saline.
        ii. Calculate deficit, give 20mls/kg bolus, then the rest over 24 hours, adding maintenance
        iii. Add maintenance (using 4/2/1mh/kg/hour rule)
        iv. Aim UO > 0.5mg/kg/hour
4. Electrolytes: likely overall Na and K osmotic loss through kidneys, but readings may be high-normal due excess water loss
    a. Assess:
        i. Usually ↑ K (>5mmol/L) due to dehydration, acidosis
        ii. Pseudohyponatraemia: Corrected Na = Na + 0.4(BSL)
    b. Aim: to normalise K and to avoid hypokalaemia from diuresis, vomiting, insulin, ↑aldosterone, correction of acidosis
    c. Method:
        i. If K > 5.5mmol/L, no initial replacement required

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   ii. If K < 5.5mmol/L, give 20mmol/L KCl in addition to replacement fluids
   iii. ECG monitoring
5. Acidosis:
   a. Assess – ABG → may be combination lactic, ketoacidosis
   b. Aim: normalise pH
   c. Method: no sodium bicarbonate required unless severe acidosis pH < 7.0. No evidence for this.

Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2010B15: Identify the key features, what pattern of disorder is demonstrated by these tests? What are the possible causes? What are the implications of general anaesthesia for an adult patient with Curve B presenting for a knee arthroscopy? Describe the abnormalities on this capnograph. What is your differential diagnosis? How would you identify the likely cause in the intraoperative setting?

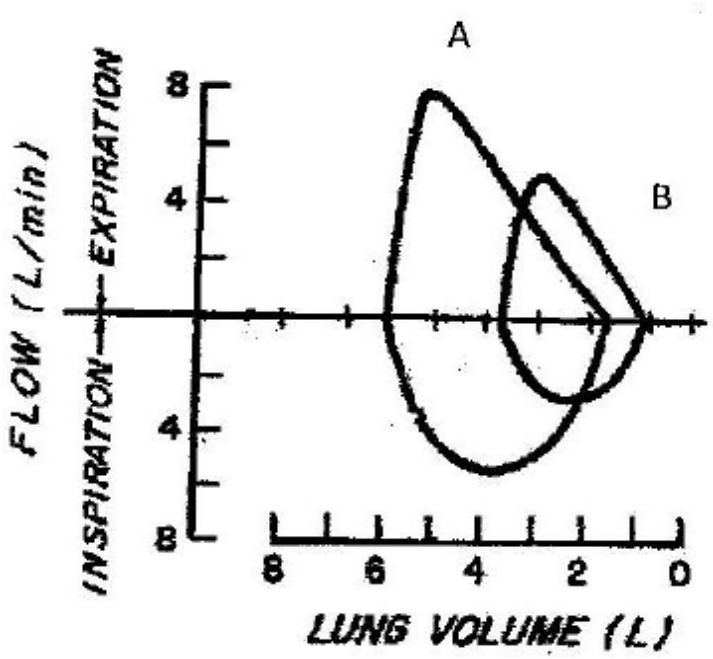
		% predicted	Lower limit of normal
Forced vital capacity – FVC	1.74 litres	60%	2.41 litres
Forced expiratory volume in 1 sec – FEV_1	1.47 litres	70%	1.82 litres
FEV_1/FVC ratio	84.5%		68.2%
Forced expiratory time - FET	9.1 secs		
Residual volume - RV	0.85 litres	39%	1.5 litres
Total lung capacity - TLC	2.81 litres	54%	4.22 litres
Diffusing capacity - DLCO	8.75 ml/min/mmHg	39%	14.9 ml/min/mmHg

Key Feature and Disorder Pattern

1. Moderate-severe restrictive lung disease with impaired diffusing capacity

Causes

1. Restrictive chest wall – obesity, scoliosis, spinal cord injury, neuromuscular disease, abdominal splinting
2. Restrictive lung parenchyma – fibrosis (drugs, dust, autoimmune, infection), CF, interstitial lung disease
3. Iatrogenic – pneumonectomy, lobectomy



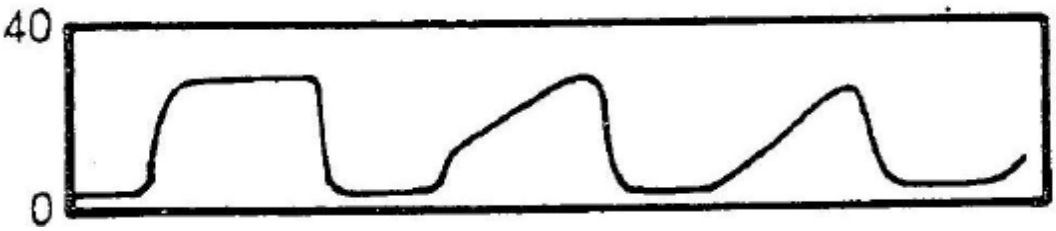
Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

Implications for GA - Severe restrictive lung disease

1. Pre-op:
    a. Recognition of severity – identify cause, and complications (RHF, Pulmonary HTN)
    b. Optimisation with referral to specialist
    c. Consider discussion of regional technique (spinal, femoral/sciatic block)
    d. Consider booking overnight stay if GA
2. Intro-op:
    a. Regional may be preferable
    b. Induction – rapid desaturation on apnoea (small FRC), and slow gas induction
    c. Maintenance –

        Supine position, and respiratory effects of GA may make spontaneous breathing unsuitable → ↓FRC, ↑VQ mismatch

        i. Application of CPAP during spontaneous breathing will improve lung compliance (more efficient position of pressure-volume curve)
        ii. Ventilation with fast RR (12-15), low TV (5-6mls/kg), relative high I:E ratio. Avoid high inspiratory pressure → risk barotrauma, pneumothorax.
3. Post-op
    a. Close monitoring of saturations, RR
    b. Regional anaesthesia, minimise opioid pain relief



Describe abnormalities

1. Steep expiratory upstroke
2. Progressive drop of expiratory CO_2 level

Differential diagnosis

1. Equipment: mechanical obstruction
    a. Patient: Bronchospasm, exacerbation COPD, aspiration, displaced ETT, anaphylaxis

How to identify cause intraoperatively

1. Disconnect from ventilator and hand bag with 100% FiO_2
2. Check circuit from ETT → filter → expiratory limb → valve, CO_2 absorber → APL valve. Remove clots, fluid, kinks
3. Auscultate chest
4. Assess response to bronchodilator

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2011A2: A patient with known idiopathic pulmonary fibrosis (fibrosing alveolitis) presents for an open right hemicolectomy. What are the respiratory issues facing this patient in regard to their general anaesthetic? Explain your intraoperative ventilation strategy.

Idiopathic pulmonary fibrosis is a heterogeneous disease resulting in fibrosis of lung parenchyma with resultant restrictive lung defect and diffusion impairment.

Respiratory issues for GA

1. Patient: full workup at pre-admission clinic
    a. Pulmonary fibrosis: requires optimisation with specialist
        i. Physiology: RFTs, diffusion studies
            1. ↓ FRC, ↓TLC/FVC (>50% severe)
            2. Perfusion → diffusion limitation (DLCA < 60% severe, VQ mismatch
            3. Restrictive lung disease: ↓ lung compliance, ↑ WOB, small TVs
        i. Disease severity – need for O_2, exercise tolerance
        ii. Treatment – steroids, immunosuppressants → immunocompromise
    b. Co-existing disease:
        i. Pulmonary hypertension – ECHO
        ii. Superimposed infection, metastasis
2. Surgical: impact of hemicolectomy
    a. Invasive operation with associated pain and fluid shifts → difficult to extubate
    b. Consider thoracic epidural
3. Anaesthesia:
    a. GA / muscle relaxation: further ↓ FRC, lung volumes, VQ matching and lung compliance
    b. Ventilation:
        i. risk of baro/volu/atelectrauma, pneumothorax
        ii. may worsen pulmonary hypertension
        iii. CPAP post-op will improve compliance
    c. Pain:
        i. Will hinder clearance of chest secretions
        ii. Systemic treatment (opioids) risk respiratory depression

Intraoperative ventilation strategy

1. Principles:
    a. Maintain oxygenation
    b. Avoid volutrauma, barotrauma, atelectrauma, pneumothorax
    c. Minimise worsening of pulmonary pressures
2. Strategy:
    a. Intubation to allow controlled ventilation and protect airway
    b. Pressure control: limit Max pressure 30cm H_2O, low TV (5-7mls/kg), high RR (12-15), low PEEP (5cm H_2O), longer I:E ratio (1:1)
    c. FiO_2 titrated to SaO_2 > 92%, high O_2 can worsen fibrosis
    d. Permissive hypercapnoea

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2011A10: What factors contribute to acute kidney injury in the perioperative period? Outline the efficacy of perioperative strategies to reduce acute kidney injury.

Acute kidney injury is defined by RIFLE criteria: 3 types (risk, injury, failure) and 2 outcome measures (loss, ESRF)

- Risk: serum creatinine ↑ x1.5, GFR ↓ > 25%, UO < 0.5mls/kg/hour for > 6 hours
- Injury: serum creatinine ↑ x2, GFR ↓ > 50%, UO < 0.5mls/kg/hour for > 12 hours
- Failure: serum creatinine ↑x3, GFR ↓ > 75%, UO < 0.3mls/kg/hour for > 24 hours
- Loss: persistent failure at 4 weeks
- ESRF: persistent failure at 3 months

Factors contributing to AKI

1. Pre-renal:
    a. ↓ systemic perfusion: general anaesthetics contribute to ↓ GFR, spinal above T4 ↓ SNS to kidney
        i. Hypotension – shock of any cause
        ii. Hypovolaemia – blood, fluid loss
        iii. Hypoxia
        iv. Anaemia
    b. ↓ renal perfusion: Cross clamping, pneumoperitoneum, ↑ intra-abdominal pressures,
    c. Risks: age, diabetes, HTN
2. Renal:
    a. Ischaemia due to prolonged insult
    b. ATN: ischaemia, rhabdomyolysis, drugs
    c. Interstitial nephritis: PPIs, aspirin
    d. Nephrotoxins: drugs (aminoglycosides, ACE, diuretics, NSAIDs), contrast
    e. Pre-existing conditions: infection, stones
3. Obstructive
    a. Blocked catheter
    b. Surgical related – tumour, ureteric
    c. Anaesthesia urinary retention (spinal, opioids)

Efficacy of perioperative Strategies

1. Pre-operative
    a. History and examination for risk: pre-existing renal impairment, fluid status (IV fluids, UO)
    b. Optimisation of:
        i. Fluid status
        ii. Cessation of nephrotoxins
2. Intra-operative:
    a. Monitoring: IDC for urine output, consider arterial line + CVP if large fluid shifts
    b. Goal-directed: intravascular volume repletion is the best proven method of renal protection
        i. Maintain MAP > 65mmHg, CVP 8-12mmHg
        ii. Maintain normovolaemia
        iii. Aim UO > 0.5mls/kg/hour
    c. Drugs to avoid: aminoglycosides, NSAIDs, IV contrast

Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

   d. No evidence: mannitol, dobutamine, furosemide, ANP, Ca blockers
   e. Surgical: minimise clamp time, pneumoperitoneum, place shunt
3. Post-operative:
   a. HDU for monitoring of electrolytes, fluid balance, urine output

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

Describe the abnormalities on this ECG (Long QT). What are implications of this abnormality for anaesthesia?

Long QT Syndrome

1. QT interval – beginning of Q wave to end of T wave, Normal < 430 ms female, <440ms male, QTc = QT/√RR, prolonged repolarisation
2. Causes:
    - Congenital – 1:3000
    - Acquired:
        1. Sympathetic stimulation – anxiety, pain, exercise

Implications for Anaesthesia

- Major cause of sudden cardiac death in young population
- The patient is at risk of spontaneous VF, Torsades de Points

1. Pre-operative:
    a. Assessment of risk:
        i. Previous episodes of arrhythmias
        ii. Family history of sudden death
    b. Requires cardiology referral
    c. Optimisation:
        i. commence or continue beta-blocker
        ii. Avoid ↓Mg, K, Ca
        iii. Cease causative drugs: TCAs, methadone, cisapride
2. Intra-operative:
    a. Monitoring: consider arterial line
    b. Avoid triggers to increasing QT interval:
        i. Volatile anaesthetics (use TIVA)
        ii. Specific drugs: suxamethonium, ketamine, butyrophenones, (droperidol), antibiotics (erythromycin), type Ia/III anti-arrhythmics, glycoppyrollate
        iii. Positive pressure ventilation (spontaneous breathe if possible)
        iv. Hypothermia
        v. Hypercarbia
        vi. Pain
        vii. Electrolyte abnormalities
    c. Anticipate treatment of arrhythmias:
        i. High risk patients should have external defibrillator pads attached
        ii. Magnesium loading
3. Post-operative: HDU monitoring

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2011A15: How would you identify a patient with autonomic neuropathy associated with diabetes? What are the anaesthetic implications from a cardiovascular perspective?

Identification of autonomic neuropathy

1. History:
    a. Diabetes – risk factors
        i. Long standing type 1 diabetes
        ii. Poor control (HbA1c)
            1. other microvascular disease: nephropathy (renal impairment), retinopathy, neuropathy (paraesthesia)
    b. Symptoms: multisystem disease, frequently silent
        i. CNS - Postural syncope, dizziness,
        ii. GI – bloating, N+V, reflux, early satiety
        iii. Metabolic - Poor exercise, heat tolerance, impotence
        iv. Endocrine – impaired response to hypoglycaemia
2. Examination:
    a. Postural hypotension
    b. Poor heart rate variability – should ↑ > 15 with deep inspiration, valsalva ratio >1.2
3. Investigations: Bloods – HbA1c

Anaesthetic Implications

1. Pre-op
    a. Glycaemic control important, aim BSL 6-10
2. Intra-op:
    a. Monitoring: arterial line, 5 lead ECG
    b. Induction: high risk of aspiration → RSI with tube
    c. Maintenance:
        i. Cardiovascular: tight control of volume and BP required
            1. ↑ risk of myocardial events, ↑mortality from events
            2. Poor cardiac function
            3. Impaired reflex responses to fluid shifts
            4. Labile BP and HR responses
        ii. Normothermia (bear hugger, fluid warmer)
3. Post-op:
    a. Increased risk of peri-operative cardiac event
    b. May require HDU monitoring (silent infarct)

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2011B9: You are called to anaesthetise a 70 year old man with a perforated bowel for laparotomy, three days after colonoscopy. Outline the measures you will take to reduce the likelihood of this patient developing acute lung injury.

Acute Lung injury – diffuse heterogeneous condition characterised by hypoxaemia, non-cardiogenic pulmonary oedema, ↓ lung compliance, and capillary leakage . Diagnosed by:

1. CXR – bilateral pulmonary infiltrates
2. PCWP < 18mmHg
3. PaO_2:FiO_2 < 300mmHg (ALI), or <200mmHg (ARDS)

Cause	Risk reduction
Anaesthetic: Blood product transfusion – FFP > platelets > packed cells	Minimise transfusion. Strict transfusion protocol. Use alternatives – cell saver. Maintain normothermia, normovolaemia.
Ventilator: Barotrauma Volutrauma Atelectrauma O_2 toxicity	Ensure correct ETT placement, aim to minimise intubation time → extubate on table if analgesia, warm. Limit ventilator pressures to < 30cmH_2O, volumes < 8mLs/kg, use ↑RR if necessary, PCV better than VCV, increase inspiratory time Avoid atelectasis – recruitment, PEEP 5cmH_2O, treat pain post-op (thoracic epidural, PCA) Titrate O_2 requirements to SaO_2 > 95%, avoid prolonged ↑↑FiO_2
Medical: Sepsis Fluid Overload Lung infection, aspiration Chronic conditions – COPD, asthma	 Treat aggressively with antibiotics, continue perioperatively Restrictive use, avoid fluid overload. Monitor with arterial line, central line, use vasopressors. RSI to avoid aspiration pneumonia, premedicate with Na Citrate, ranitidine Optimise conditions pre-op: nebulised medicines

Risk Reduction of ALI

1. Pre-operative:
    a. Recognise risk factors as above
    b. If time allows, fast for 6 hours but balance with risk of worsening sepsis, atelectasis
    c. Suction NGT prior to induction
    d. Administer Na Citrate, ranitidine
    e. Consider thoracic epidural
2. Intra-operative:
    a. RSI with CP
    b. Protective ventilation strategy
    c. Volume, blood product strategy
    d. Normothermia, Normoglycaemia
    e. Antibiotic treatment or prophylaxis
3. Post-operative:
    a. Aim to extubate awake (thoracic epidural)

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

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BSc(Med) MBBS(Hons) FANZCA

ADDITIONAL NOTES

Treatment

1. Keep PaO_2 > 60mmHg while minimising O_2 or ventilator trauma.
    a. Titrate to ↓FiO_2
    b. Pressure control ventilation: TV < 8mLs/kg (minimum 4mLs/kg), pressures < 30cmH_2O, increase inspiratory time (↓PIP), PEEP 5-10cmH_2O
2. Unclear:
    a. Prone positioning
    b. Steroids
    c. Nitric oxide

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2011B13: A 50-year old man presents with confusion and the following electrolyte profile: Na 155, K 4, HCO3 15, Cr 120, Hb 200. What are the possible causes of this abnormality? How can they be distinguished?

Abnormalities

1. Hypernatramia
2. Metabolic acidosis (low bicarbonate)
3. Polycythemic
4. Renal impairment

Differential Diagnosis

1. Hypernatraemia classified by volume states:
    a. Hypervolaemic (Na > water excess): iatrogenic hypertonic saline infusion
    b. Euvolaemic (TBW deficit, normal Na): mild dehydration, diarrhoea, vomiting
    c. Hypovolaemic (water > Na deficit): dehydration, refusal to drink, GI loss (vomiting, diarrhoea), Renal loss (Diabetes insipidus, DM, diuretics).

Distinguishing

1. History:
    a. Background – polycythemia vera, renal impairment, diabetes mellitus/insipidus
    b. Presenting complaint
        i. Dehydration, exercise, not drinking,
        ii. Fluid intake, thirst response, urine output
        iii. CNS pathology: tumour
    c. Medications:
        i. Laxatives, bowel preparation
        ii. Diuretics
2. Examination:
    a. Observations
    b. Volume status assessment: HR, BP, JVP, skin turgor, CNS confusion, RR, capillary refill
    c. Focal CNS examination
3. Investigations:
    a. Blood gas: acidosis differential
        i. Anion gap – lactic acidosis, uraemia
        ii. Non-anion gap – diarrhoea, diuretics
    b. EUC:
        i. Low phosphate, magnesium suggests bowel loss
    c. Urine osmolality and Na
    d. Desmopressin response
4. Management: address underlying cause
    a. Hypervolaemic: diuretics + 5% dextrose
    b. Euvolaemic:5% dextrose
    c. Hypovolaemic:0.9%Na Cl, followed by 5% dextrose
    d. DI: desmopressin + 0.9% Na Cl

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2011B15: Explain your approach to thromboprophylaxis in the patient undergoing total knee replacement.

DVT/PE is a major cause of peri-operative morbidity and mortality, accounting for 10% of all in hospital deaths. 40-80% of high risk patients will develop peri-operative DVT.

Approach

Pre-operative

1. Risk stratification:
    a. Identify risk factors: optimise and treat these pre-operatively
        i. Low flow: immobility, CCF, obesity
        ii. Hypercoagulable: malignancy, coagulation disorders (ATIII/PC/PS deficiency, factor V Leiden), pregnancy, OCP, polycythemia,
        iii. Endothelial: surgery, PVD, smoking, previous VTE, cholesterol, sepsis
    b. Identify risks of bleeding which may interfere with prophylaxis
    c. Surgical risk: joint replacements, pelvic, abdominal
2. Perioperative management of patients on warfarin:
    a. High risk: cease warfarin 5 days prior to surgery, commence clexane 1.5mg/kg daily, admit to hospital day prior for heparin infusion aim APTT 60-90, ceased 4 hours prior to surgery with APTT just prior.
    b. Intermediate risk: cease warfarin 5 days prior, commence clexane 1.5mg/kg daily when INR <2, take last dose 24 hours prior to surgery.
3. Specific instructions:
    a. Education re: importance of mobility
    b. Fluid hydration prior to surgery

Intra-operative

1. General measures:
    a. Fluid hydration keep euvolaemic and well hydrated
    b. Regional techniques ↓ compared to GA
    c. Consider femoral nerve block for post-operative analgesia
2. Mechanical:
    a. Use of calf compressors and TEDs: can only use on non-operative leg, limited in CCF, PVD
3. Chemical: ↓ rate by 67%
    a. Administer prophylaxis during/after surgery (in discussion with surgeons, timed with neuraxial block):
        i. Heparin 5000U SC BD
        ii. Clexane 40mg SC daily

Post-operative

1. Adequate analgesia to encourage early mobilisation
2. Fluid hydration
3. Continue prophylaxis clexane 40mg daily, or heparin 5000U bd
4. Some centres use rivaroxiban or fondaparinaux for 1 month post-op
5. Recommence treatment warfarin

Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

ADDED NOTES - Guidelines

1. Patient risk factors:
    a. Previous DVT or PE
    b. Hypercoagulation disorder: factor V Leiden, anti-phospholipid syndrome, PC/PS deficiency, ATIII deficiency
    c. Active Malignancy
    d. Pregnancy
    e. OCP
2. Surgical risk:
    a. Minor surgery
    b. Major surgery: lasting >45min
3. Types of prophylaxis
    a. Mechanical:
        i. TEDS
        ii. Calf-compressors
    b. Chemical:
        i. Prophylaxis:
            1. Heparin SC 5000 units BD
            2. Clexane SC 40mg daily
        ii. Treatment:
            1. IV heparin infusion
            2. Clexane 1.5mg/kg daily or 1mg/kg bd
            3. Fondaparinaux 2.5mg SC bd
            4. Warfarin (long term) INR 2-3

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2012A2: A 65-year-old man is on your list for an arthroscopic acromioplasty that is to be performed in the beach chair position. List the complications associated with this position. Describe how the risk of these complications can be minimised.

Beach chair position: patient sitting with hips flexed 40-50 degrees. Used for shoulder surgery.

Problem	**Risk Minimisation**
General: Unfamiliar position	Educate team re: position Experienced personnel in room Use of special beach chair
Airway: lack of access during case risk of dislodgement	Intubate and tape securely Make curtains to allow visualisation of airway during case Detach circuit during transfer
Circulation: ↓ Cerebral perfusion as head is above heart → risk of watershed stoke to CNS, eyes, cervical SC Risk of venous air embolism ↓ VR → ↓ BP, DVT risk Lack of IV access Risk of dislodgement Lack of access to chest in case of arrest	Monitoring – arterial line Keep transducer at level of head, pulse oximetry at ear, aim MAP at brain > 65mmHg. Generous fluid resuscitation with vasopressor use Avoid neck ties Clinical awareness of problem, surgeon to minimise exposure of capsule to air. Consider CVC insertion in RA if high risk. DVT prophylaxis Multiple sites of IV access prior to transfer. ECH monitoring on back with padding
Transfer to beach chair table: Risk of dislodgement of lines, ETT Injury during transfer	Multiple assistants needed for transfer, clear communication Detach lines prior to transfer Specific people in charge of line and airway monitoring
Pressure care: damage to Face - eyes Ulnar nerves Brachial plexus	Clear communication between anaesthetists and surgeons Special face mask with eyes lubricated, padded, taped Special adjustable head rest – neck in neutral position Ulnar padding with arm rest Minimise traction to neck Secure thorax and hips with straps Regular checking during case

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2012A3: A 60-year old man is admitted to the HDU following laparotomy for relief of a large bowel obstruction. He has a urinary catheter in situ. Three hours later he remains oliguric. Define oliguria. What are the potential causes of oliguria in this patient? How would you differentiate between these causes?

Oliguria – reduced urine output < 0.5mls/kg/hour OR < 500mls/24 hours

Potential Causes

1. Pre-renal:
    a. ↓ renal BF – hypotension, Hypovolaemia (vomiting, diarrhoea),haemorrhage, shock, hypoxaemia, low cardiac output (CCF)
    b. Abdominal compartment syndrome
    c. ADH response to stress
    d. Surgical – damage to renal vessels
2. Renal:
    a. Acute tubular necrosis from renal ischaemia
    b. Contrast nephropathy
    c. Drugs – gentamicin, NSAIDs
    d. Surgical – renal damage
    e. Pre-existing impairment: nephrectomy, polycystic, pyelonephritis
3. Post-renal:
    a. Ureteric obstruction, damage
    b. Bladder outlet obstruction
    c. Catheter kinked or blocked

Differentiating

1. History:
    a. Renal disease: previous renal impairment
        i. Hypertension, diabetes
        ii. Intrinsic: polycystic kidney, nephrectomy
    b. Perioperative fluid:
        i. Intake – PO, IV
        ii. Urine output: pre-operative, intraoperative
    c. Drugs: look for any nephrotoxins including contrast
    d. Nature of illness: prolonged fluid loss through vomiting, diarrhoea
2. Examination:
    a. Volume status: hypovolaemic → pre renal failure. HR, RR, CR, thirst, mucous membranes
    b. Catheter: kinked, blocked, blood
3. Investigations:
    a. FBC, EUC – creatinine, urea, electrolytes, ↓Hb (bleeding)
    b. Bladder scan will show obstruction
    c. Urine osmolality:
        i. High: pre-renal
        ii. Low: renal → failure to concentrate urine
    d. Pyelogram: look for obstruction
    e. TOE: renal blood flow, cardiac function
    f. Urologist consult → cystoscopy

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ANAESTHESIA

Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2012A8: A 35 year old female is booked for thyroidectomy. Her blood results are as follows. TSH 0.1 (0.3-3), Total T4 20 (4-11), Free T4 4 (0.7-1.8), Free T3 120 (60 – 175). Interpret the thyroid function tests. Justify when you would proceed to thyroidectomy in this patient. What is the management of an intraoperative thyrotoxic crisis?

Interpretation

The patient is thyrotoxic with ↓TSH (negative feedback inhibition), ↑T_4

Proceeding with Surgery

1. Proceed with surgery when $T_{3/4}$ parameters and symptoms normalised by non-surgical means → euthyroidism. Uncontrolled thyrotoxicosis risks: ↑ cardiac risk, thyroid storm, goitre + airway difficulty.
    a. Consult Endocrinologist
    b. Methods: usually takes 6-8 weeks due to thyroid hormone stores in colloid
        i. Carbimazole (inhibit thyroid peroxidase) or propylthiouracil (inhibit ionidase)
        ii. Radiation
        iii. Iodine therapy
2. Factors which would expedite surgery
    a. Failure of medical treatment
    b. Enlarging mass with airway obstruction
    c. High possibility of malignancy

Thyrotoxic Crisis: medical emergency, 20-30% mortality

1. Recognise signs: usually occurs 6-24 hours post-op, but may occur intra-op with thyroid handling
    a. CVS – tachycardia, hypertension, cardiovascular collapse, arrhythmias, CCF
    b. Resp - ↑ $ETCO_2$
    c. Metabolic – fever, sweating, N+V
    d. Neuromuscular – rigidity
    e. Differential: serotonin syndrome, NMS, MH, phaeochromocytoma, carcinoid, awareness, light anaesthesia
2. Call for help, resuscitation trolley.
3. Inform surgeons to stop handling thyroid.
4. Check ABCs: ETT position, 100% O_2, fluids
5. Specific therapy:
    a. NG PTU
    b. NG lugol's iodine 5-10 drops q6h OR IV Na iodide 500mg tds
    c. Rehydration with saline + glucose (thyroid hormone depletes glucose)
    d. IV hydrocortisone 100mg → for adrenal insufficiency, ↓ T_4 → T_3 conversion
6. Manage complications:
    a. Cardiovascular: IV esmolol 0.5mg/kg + infusion 5mg/kg/hour. Aim HR < 80
    b. Active cooling + paracetamol
7. Transfer to ICU ventilated

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2012A10: An adult patient from the ICU with severe ARDS requires a laparotomy for an acute abdomen. What are the features of ARDS? Explain your perioperative ventilation strategy.

ARDS – adult respiratory distress syndrome

1. Heterogenous lung disorder characterised by hypoxaemia, poor lung compliance, non-cardiogenic pulmonary oedema, and capillary leakage.
2. Diagnosis:
    a. Bilateral infiltrates on CXR
    b. Absence of cardiac cause, PCWP < 18mmHg
    c. PaO_2:FiO_2 < 200 mmHg
3. Causes: sepsis, burns, transfusion products, pancreatitis, pneumonia, drugs, ventilator
4. Prognosis:
    a. Complete recovery possible
    b. Pulmonary fibrosis, death

Perioperative Ventilation Strategy: oxygenation, prevention of further injury, treatment of cause

Strategy	**Explanation**
Limit FiO_2, titrating to PaO_2 > 60mmHg	Excess O_2 worsens ARDS by O_2 toxicity and free-radical formation. Baseline PaO_2 required to maintain organ perfusion
Pressure control ventilation Peak pressures < 30cmH_2O Longer inspiratory time	PCV assists with more heterogenous ventilation of alveoli with different time constants Avoid barotrauma and volutrauma → damage to alveoli. Longer inspiratory time limits peak inspiratory pressure, more time to alveoli to expand.
Tidal volumes 4-8mls/kg IBW	Avoids volutrauma. Minimum TV required for appropriate ventilation
Permissive hypercapnoea	Can ↑RR to compensate for ↓TV But may be inadequate for CO_2 blow-off Risks of hypercapnoea < risks of ventilator lung injury Keep pH > 7.2
PEEP 5-10cmH_2O Recruitment manoeuvres	Limit atelectrauma PEEP limits inspiratory pressures
Drugs: Nitric oxide Neb prostaglandin (iloprost)	No evidence of survival benefit
Fluids	Restrictive use may benefit

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2012A13: Describe the risk factors for perioperative stroke. Describe how you would minimise the risk in a high-risk patient having major orthopaedic surgery.

Stroke: neurological syndrome resulting from infarction of brain tissue. This is either ischaemic (85%) or haemorrhagic (15%).

Risk factors:

1. Patient:
    a. Male, age > 70
    b. Previous CVA/TIA, recent CVA 20x risk
    c. Vascular risk factors: DM, renal impairment, smoking, HTN, chol
    d. Cardiac risk: AF, PFO
2. Surgical:
    a. Type: Neurosurgery, carotid surgery, cardiac bypass surgery
    b. Emergency surgery
    c. Long duration with large fluid shifts
3. Anaesthetic:
    a. Impaired perfusion – hypoxaemia, hypotension, anaemia, ↓ cardiac output
    b. Position – beachchair, prone
    c. Coagulopathy, hypertension → ICH

Minimising Risk

1. Pre-op
    a. Identify risk factors, optimise:
        i. Cardiac disease, PFO, requires ECHO – normalise BP
        ii. DM – normalise BSL
    b. Postpone if recent MI, CVA (for at least 6 weeks)
    c. Management of anti-coagulants, anti-platelets (discuss with specialist + surgeon)
        i. Continue aspirin if permissible
        ii. High risk patients on warfarin → clexane bridge
    d. Instructions: smoking cessation
2. Intra-op:
    a. Monitoring:
        i. consider arterial line, 5 lead ECG, pulse oximetry on face
        ii. detection of cerebral perfusion: jugular venous oximetry, Transcranial doppler
    b. Maintenance:
        i. Haemodynamic stability – keep MAP within 20% baseline
        ii. Keep Hb > 80
        iii. Normothermia, Normoglycaemia
        iv. Normocapnoea
        v. Avoid air in lines
    c. Vigilance during risk periods:
        i. Cementing – pre-oxygenate
3. Post-op:
    a. Maintain normovolaemia, O_2 and Hb
    b. Neurological assessment with early specialist referral if new focal deficits
    c. HDU if at risk

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2012B2: *A 75-year-old man presents for right hemicolectomy for an obstructing lesion of the ascending colon that has failed to settle with conservative management. He had a drug-eluting stent placed eight months ago, and is currently on clopidogrel and aspirin. Discuss and justify your plan for perioperative management of his antiplatelet therapy?*

Principles of management

1. Patients with drug eluting stents should be on dual antiplatelet therapy for at least 12 months to prevent in-stent thrombosis. The perioperative period is associated with rebound hypercoagulability which further increases thrombosis risk. 25% risk of thrombosis if ceased.
2. The administration of anti-platelet therapy significantly increases the risk of bleeding.
3. There is risk in delaying surgery
4. Communication is vital between the anaesthetist, surgeon and cardiologist.

Management Plan

1. Pre-operative: requires risk assessment of bleeding vs. thrombosis
    a. Cardiac assessment:
        i. Reason for stent insertion, vascular distribution (proximal LAD high risk, distal vessel low risk)
        ii. Medication – antiplatelet agents, dose and last taken, compliance
        iii. Letters – cardiac assessment, recent ECHO, angiogram
        iv. Symptoms – exercise tolerance, new angina, SOBOE
    b. Surgical presentation:
        i. Level of obstruction, likely surgical difficulty, risk of bleeding (likely low).
        ii. Urgency – can this surgery be delayed if transfer required?
    c. Other co-morbid disease:
        i. Bleeding disorders
        ii. Clotting disorders, AF, DVT/PE, renal or liver disease
    d. Examination:
        i. Volume status, HR, BP
    e. Investigations:
        i. ECG – look for evidence of ischaemia
        ii. Bloods – optimise Hb, coags
        iii. Cross match blood, Platelets
        iv. Consider platelet mapping, TEG
    f. Decision-making
        i. Consider short-acting bridging therapy:
            1. tirofiban -
            2. abciximab -
        ii. Consider availability and proximity to cath lab; and/or cardiothoracic revascularisation surgery → transfer if appropriate.
2. Intra-operative:
    a. Monitoring:
        i. 5 lead ECG
        ii. Arterial line
    b. Bleeding:
        i. Early intervention with packed cells, platelets
        ii. Normothermia, calcium, acid-base
3. Post-operative:

Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

a. HDU / CCU for monitoring of ischaemia
b. Monitoring of blood loss
c. Recommencement of clopidogrel post-op with loading

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2012B7: In regard to total parenteral nutrition: What are the indications? (30%). What are the complications? (70%)

Total parenteral nutrition is the intravenous administration of nutrition to supplement and/or replace enteral feeding.

Indications: enteral feeding NOT meeting metabolic demands of patient

1. GI Malabsorption syndromes:
    a. Surgical – short gut syndrome
    b. Sepsis, burns, peritonitis
    c. Prolonged diarrhoea
    d. IBD – Crohn's, UC
2. Enteral blockage:
    a. GI surgery: resection, fistula, obstruction
3. Severe malnutrition
4. Improved wound healing in ICU
5. ↓ GCS for prolonged period (NG feeding usually preferred)

Complications:

1. Formula:
    a. Inadequate infusion: malnutrition, hypoglycaemia
    b. Excessive infusion:
        i. Hyperglycaemia
        ii. Hyperlipidaemia: fatty liver, cholecystitis
        iii. Fluid overload
    c. Electrolyte abnormalities, acidaemia
    d. Vitamin, trace element deficiencies
    e. Immunosuppression
2. Intravenous line:
    a. Infection
    b. Misplacement: arterial puncture, pneumothorax
    c. Pain, discomfort
3. Re-feeding:
    a. Refeeding syndrome: ↓K, ↓Mg, ↓PO_4

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2012B4: What is the natural history of aortic stenosis? (30%) What are the key echocardiographic features in haemodynamically significant aortic stenosis? (70%)

Aortic stenosis – valvular pathology resulting in obstruction to outflow during ventricular systole at the aortic valve.

Natural History

1. The natural history varies according to pathology and co-morbid disease. Factors Accelerating deterioration:
    a. Aetiology: calcific degeneration
    b. Cardiac ischaemia
2. History of symptoms: significant if SOBOE, syncope or angina. 50% survival –
    a. Angina – 50% at 5 years
    b. Syncope – 50% at 3 years
    c. SOB – 50% at 2 years
3. General pathophysiology
    a. Valvular pathology results in outflow obstruction (valve area ↓ 0.1cm^2/year
    b. Increased ventricular pressures required for adequate ejection → LVH
    c. Increased cardiac demand
    d. Cardiac ischaemia
    e. Terminal event – arrhythmia, MI

ECHO features

1. Aortic stenosis grading:
    a. Mild: area < 2cm^2, mean gradient <25mmHg
    b. Moderate: area 0.8-1.2: mean gradient > 25
    c. Severe: area 0.6-0.8, mean gradient >40
    d. Critical: area <0.6, gradient >50
2. Haemodynamically significant:
    a. Quantitative:
        i. Pressure gradient = 4 x velocity^2
        ii. ↓LVEF
        iii. Diastolic failure: atrial kick 40%, ↑LVEDP, ↓LVEDV
    b. Qualitative:
        i. Restriction of aortic valve leaflets, thickening, calcification
        ii. LVH
        iii. LA dilatation
        iv. Pulmonary HTN and RV failure

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2012B9: A developmentally delayed, uncooperative adult requires a magnet resonance imaging scan for investigation of deteriorating control of seizures. What issues do you foresee in terms of providing general anaesthesia in the MRI suite for this patient?

Issues for providing general anaesthesia

1. Pre-operative: should be seen prior to day of procedure
    a. Consent – likely unable to give informed consent
        i. Need next of kin or guardian
    b. Medical comorbidities – developmental delay associated with other medical conditions: epilepsy, congenital syndromes, cardiac disease.
        i. Epilepsy requires optimisation, ensuring medications are taken on the day
    c. MRI suite:
        i. Transfer equipment – anaesthetic machine, ventilator, drug trolley, airway and resuscitation trolley
        ii. Skilled team – anaesthetic assistant, anaesthetist, ORA
        iii. Long IV lines and tubing, with large visible monitors
        iv. Patient – investigate for contraindications → PPM, AICD, metal coils, metallic tattoos, cochlear implants
    d. Organisation: need to book staff and MRI suite and allow extra time (often case takes one session)
2. Intra-operative:
    a. Induction – often unable to get IV access due to lack of compliance.
        i. Gas induction followed by IV access and intubation
    b. Transfer:
        i. Care with transfer to MRI
        ii. Remote access to patient: need to intubate, long lines, visible monitor
    c. Maintenance: avoidance of seizures
        i. Maintain normothermia, normocapnoea, Normoglycaemia, adequate depth of anaesthesia
3. Post-operative:
    a. Transfer to recovery (this may be a long distance)
    b. High chance of emergence delirium
    c. Skilled staff to recover
    d. Ensure discharge criteria are fulfilled if day patient

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Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

2012B13: Discuss the key areas of concern in your preoperative assessment of a patient for excision of a large tonsillar mass.

Preoperative assessment

1. Issues:
    a. Shared airway surgery
    b. Risk of airway obstruction perioperative, especially induction
    c. Post-operative care
2. History:
    a. Mass: thorough history of evolution, treatment
        i. Local obstruction: positional dyspnoea, stridor, facial plethora
        ii. Systemic: infective (fevers, sweats), neoplastic (fevers, weight loss)
        iii. Urgency of treatment: pre-operative treatment to reduce size (chemo, radiotherapy)
    b. Other medical history: especially cardiorespiratory co-morbidities
    c. Recent anaesthetics
    d. Plan will vary depending on age (child unable to undergo awake airway procedures)
3. Examination:
    a. Direct visualisation or nasendoscopy
    b. Local obstruction: Pemberton's, stridor, tachypnoea
    c. Airway assessment:
        i. anterior column – mouth opening, jaw protrusion, MP score
        ii. posterior column – neck extension, sniffing position
4. Investigations:
    a. Imaging of neck: local invasion, metastasis, airway calibre
    b. CXR
    c. Bloods – with G+H
5. Preparation:
    a. Liaison, form plan with surgeon – possibility of tracheosomty, surgical airway
    b. Consent: awake Fibreoptic plan requires patient compliance
    c. Book ICU/HDU bed