

X-Ray Interpretation – Ricky Raine (Groote Schuur Hospital/University of Cape Town)

Extensive collection of XR examples demonstrated.

- Why? What? When?
- Technical considerations – identification, position (side, rotation, angle), penetration, inspiration, associated devices
- Systematic review – bones, soft tissues, vascular structures, heart, Lung fields
- Cherniak 1983 – excellent diagram of segmental radiographic anatomy
- Silhouette sign very useful for suggesting opacification is in the lower (rather than middle or lingual) lobes
- Always check: airways, oesophagus, behind heart, costophrenic angles, mediastinum, hilar regions, apical areas
- Clinician's questions:
 - Why sudden onset of distress?
 - Why a fall in PaO₂?
 - Changes in airway pressure?
 - Source of fever?
 - Changes caused by intubation?
 - Changes caused by extubation?
 - Where is the device??
- ETT – 4-5cm from carina (At aortic knuckle). Cuff < width of trachea
- CVC – within thorax, outside RA, no pneumo- or hydrothorax
- NGT – in stomach or post-pyloric
- ICD – tip in area to be drained, side holes in pleural cavity.

Patient Evaluation – The ECG – Pieter le Roux (Tygerberg Hospital/University of Stellenbosch)

- What is the place of the ECG in modern practice?
 - Immediate medical or anaesthetic management
 - Prediction of perioperative complications
 - A 'baseline' for postoperative interpretation (but this is controversial; see Ashton J Am Geriatr 1991)
- ECG – the basics
 - Electric flux of ions; conduction system etc. all well understood (thanks Dr Einthoven!)
 - Good source – James M "Reading an ECG in 10 easy steps"
 - ECG guide on the iPhone
- Who needs routine ECGs?

- AHA/ACC Guidelines (2007) for perioperative cardiovascular evaluation in non-cardiac surgery
- Based on surgical risk, presence of co-existing diseases and clinical risk factors (5: IHD/abnormal Q waves, cardiac failure, cerebrovascular disease, diabetes mellitus, renal insufficiency (Creat>200).
- See guidelines for classes and levels of evidence:
 - Class 1 – should do ECG. Vascular surgery plus one or more risk factor
 - Class 2a – reasonable to do ECG: vascular surgery without risk factors
 - Class 2b – may be considered: risk factors, intermediate risk surgery
 - Class 3 – not indicated: no risk factors, low or intermediate risk surgery
- Noordzij 2006 – ECG changes signify increased risk of cardiovascular death (-3 vs 1.8%). ECG improves predictive value of cardiovascular indices. Prognostic value highest in high/intermediate risk surgery.
- Age alone is not really a valid indicator for getting an ECG; ASA status is more useful.
- How common are abnormal findings on routine ECGs?
 - Distressingly common! Incidence of abnormality is very proportional to ASA grade (10% of ASA1, 40% of ASA2, 60-80% ASA4-5). About 5% of routine ECGs have “Significantly abnormal” findings (Munro 1997)
- What are the most common new abnormalities?
 - T-wave abnormalities (53%)
 - Q waves (46%)
 - ST-segment abnormalities (38%)
 - LVH 2-19% depending on the criterion used
- Do unexpected ECG findings predict intraoperative problems?
 - ST-segment depression increases cardiovascular death (OR 4.5)
 - Tachycardia (OR 4.5)
 - 0-2% of routine ECGs lead to a change in management (Munro 1997)
- Selected unexpected findings:
 - Arrhythmia or conduction delay – any rhythm other than sinus increases risk. Controlled AF with a ventricular risk in the absence of other complications does not need further assessment. Ventricular rate of >100 requires slowing. Conduction delays managed according to current criteria for temporary pacing.
 - Silent infarct – 30% of all MIs are silent. ECG criteria are very specific but not very sensitive.
 - Known previous infarct – prolonged QRS is a warning sign; OR=4. Avoid operation for 6 months if possible; 3 months probably sufficient
 - LVH – many criteria; voltage-based seem to work well. Asymptomatic LVH increases risk of infarct by 4 times. HOCM is a specific form of LVH
 - T-wave changes – very difficult to quantify. Many causes
 - Brugada syndrome – ideally needs investigation; give patient the option
- Summary of role in 2012:
 - Expect lots of abnormal findings
 - Base selection on ASA grade rather than age
 - Beware abnormal rate, rhythm, previous IHD and chamber enlargement
 - Investigate and treat rhythms requiring pacing and any acute ischaemia

Evaluation of Cardiac Function for the Busy Clinician – Justiaan Swanevelder (soon to be UCT/GSH Anaesthesia Head of Department)

- Most anaesthetists are busy anaesthetists...and most have a fair understanding of cardiac physiology. Everything we do affects the cardiovascular system
- Many risk scoring systems exist; ASA status is for classification, not stratification
- History – dyspnoea, orthopnoea, PND, “asthma”, diabetics
- Examination – heart sounds, murmurs, bruits, cardiac failure, “wheeze”
- Think in terms of “cardiopulmonary interaction” rather than “cardiac function”
- Renal impairment and diabetes are the two greatest risk factors for a negative outcome.
- Medications galore!
- Echocardiography is the new gold standard
- Serum biomarkers useful; markers alone (eg. BNP) are not enough to stratify
- CPEX great; 6 minute walk test has been validated as an alternative
- “PAC may not be dead and buried, but it is certainly mortally wounded...”
- Cardiac output monitors are useful but have yet to come of age
- Point-of-care ultrasonography is the way of the future
- 3D quantitative ultrasound is now able to give very accurate volumes and ejection fractions

Pathology of renal dysfunction during severe inflammation/sepsis – Andre Coetzee – University of Stellenbosch/Tygerberg Hospital

- Patient scenario given with peritonitis, severe sepsis, renal injury
- Sepsis induced renal injury
 - Haemodynamic – hypo vs hyperperfusion
 - Non-haemodynamic – inflammatory cytokines
- Remember intra-renal distribution on blood flow – medulla gets far less flow than cortex (1.9 vs 4.2 ml/kg/min), and that sodium absorption is the major determinant of renal oxygen consumption (O₂ is required for Na absorption, in a direct relationship).
- Furosemide may be effective in shutting down sodium absorption and increasing medullary pO₂
- Often added insults:
 - Contrasted scans
 - NSAIDs
 - ??Dialysis – results in episodes of hypotension – conservative types of dialysis may be better - *what about outcomes data which suggest this doesn't make a difference?*
- Vasopressors cause renal vasoconstriction, but do improve perfusion pressure. Renal vasculature during sepsis does not seem to respond to vasoconstrictors (animal studies), so the perfusion pressure improves but flow does not suffer. Dopamine does NOT offer this advantage.

- Endothelins – cause vasoconstriction, but trigger vasodilation in other areas of the kidney. Blocking endothelins improve outcome in chronic ischaemia, but role in sepsis is not yet established.
- Nitrous oxide – iNOS increases initially but then becomes down-regulated. Blocking NO in the septic patient would worsen afferent and efferent vasoconstriction.
- TNF and interleukins – all cause decreased RBF and GFR. Blockade improve survival in animal models, but have not been tested in humans. Monoclonal antibodies are effective in septic animal models.
- Endotoxin – decreases GFR; well-defined risk of renal dysfunction/injury.
- RRT – CVVHD does not decrease ILs or TNF. Plasma exchange may improve survival.
- Ventilation – less apoptosis in the kidneys and gut in animal models where ventilation was protective.

Remifentanyl and extubation – Sean Bennet – Hull, UK

- Concepts – post-operative pain and controlled extubation
- 10 years ago: great new drug – stop it and the patient wakes up with a bang! Give the patient morphine at the end and they don't wake up. Haemodynamic instability at the beginning and the end. First talk of increased opiate requirements/tolerance.
- Rapid extubation in post-op patients where analgesia is not a problem
- Stress of surgery has a great impact on the awakening times.
- Post-operative analgesia – remi 0.02-0.05 mcg/kg/min by infusion. Awake ventilated, pain free. If in pain, give morphine 2-3mg and change to PSV; aim to extubate soon (within 1 hour of arrival in PAHCU/ICU).
- Use of remi/propofol sedation in ICU for patients who will likely need extubation soon. Good for painful procedures in ventilated patients.
- Conclusions:
 - Don't overdose remi (0.1-0.2 mcg/kg/min is sufficient)
 - Reduce slowly and try to have an awake patient with a low dose remi running
 - Bridge with fentanyl
 - Create a gap between surgery and extubation to have extubation controlled in a comfortable environment.

Regionals and the Difficult Airway – Eric Hodgson – Addington Hospital & Nelson Mandela School of Medicine, KZN, SA

- Identification of patients with difficult airways – ASA 11 point evaluation
- Mask ventilation – BONES
- Rescue – RODS
- Cric – Palpate
- 3 strikes = awake intubation
- Perc trache can be done with only skin infiltration

- Awake intubation – positioning
 - C-spine injury – neutral with inline stabilisation
 - Tumour/trauma – max comfort position
 - Others – sniff position
- Drugs – antacid & glycopyrrulate
 - Glyc reduces secretions which increases view and LA penetration (less dilution)
 - Non-particulate antacid
 - NO hypnotic (amnesia vs death!)
 - Dexmedetomidine is ideal (1mcg/kg bolus over 15 minutes via infusion pump, or 5-10mcg boluses every 3-5min)
 - Remi- or alfentanil (10/100mcg boluses before noxious events)
- Best way to prepare the nasal cavity is to pack the nose with local anaesthetic. Cocaine is still the drug of choice. Soak ribbon gauze in 1-2ml of 100mg/ml solution. Oxymetazoline and lignocaine is an alternative option.
- Pharyngeal anaesthesia – glossopharyngeal nerve block. Use a large needle (18G IV needle) so you can see blood very easily
- Laryngeal anaesthesia – superior laryngeal nerve block. Transcutaneous better.
- Intratracheal injection – transcricoid; 2% lignocaine; one breath in, one breath out.
- Nebulisation is a good method of local anaesthesia. 4% lignocaine best. Absorption is extensive, so stay below one third of maximum toxic dose for nebs (other 2 thirds in blocks or topical spray)
- Gargling is good – test first with water. Gargle 3ml 4% lignocaine; coughing indicates that the patient is aspirating local (which is a good thing)
- MADgic guide is a useful tool.
- When spraying local, allow 15-30 seconds between each spray and advancing to maintain analgesia and patient confidence.
- Size 6 ETT or Parker FlexTip
- Mail iti20187@mweb.co.za for presentation/notes.

General Anaesthesia for Caesarean Section: Best Practice – Warwick Ngan Kee – Chinese University of Hong Kong

- Not black and white – shades of grey!
- Decrease in maternal deaths often associated with the move away from GA for caesarean section.
- UK recommendation: Keep use of GA <5% for elective C/S and <15% for emergency C/S
- Rapid sequence spinal – limited asepsis, no-touch
- Risk from GA has decreased dramatically over time; the OR for death of GA vs RA's confidence interval now spans 1
- Evidence that GA causes neonatal depression is rather thin – dates back to Virginia Apgar in 1959! Deficit of good RCTs. Most quoted trial was observational, dating back to 1986.
- New biochemical evidence of neonatal acid-base status suggests that spinals cause more foetal acidosis.

- Difficult balance between maternal unconsciousness and maternal uterine/foetal depression
- Glycopyrrulate should be used as an antisynergist in preference to atropine due to its quaternary structure, as it does not cross the placenta
- Propofol has been shown to cause decrease in 1 minute Apgar scores in comparison to thiopentone; the clinical relevance is questionable.
- Etomidate is useful for patients with cardiac disease
- Ketamine is very useful for patients with haemorrhage. It also has an advantage for post-operative pain.
- Alfentanil before intubation reduces the haemodynamic changes and catecholamine release, but does cause significantly decreased Apgar scores.
- Remifentanil has a similar effect, but about 70% does cross the placenta. This causes a non-significant increase in time to spontaneous breathing; advising the paediatrician is important.
- Roc is as good as sux when used in conjunction with propofol. Ketamine plus roc is also excellent.
- The laryngeal mask may be as safe as intubation
- More than 30% oxygen (or maintaining a normal SpO2) is unlikely to be useful.
- Conclusions:
 - GA is probably still more risky, but the gap has closed dramatically
 - Propofol or ketamine are good options
 - Roc is fine, but with propofol or ketamine
 - Remi is good if you need an opiate
 - Use clinical judgement when deciding how much O2 to give.

Perioperative Management of the Morbidly Obese Patient undergoing Surgery – Errol Lobo – University of California, San Francisco

- Obese patients merit special consideration
- Limited respiratory reserves with decreased FRC, which decreases below closing volume
- Increased O2 consumption and CO2 production
- Increased blood volume, higher incidence of hypertension and IHD
- Increased CO, increased LVEDP with LVH
- Impaired systolic function
- Eventual hypertensive AND dilated cardiomyopathy
- 40% of obese patients who present for surgery have OSA
- OSA patients have roughly twice the complication rate and twice the hospital stay.
- STOP-bang method: Snore loudly, often feel Tired, Often fall asleep during the day, suffer elevated blood Pressure. BANG: BMI, Age, Neck circumference, Gender
- NB difference between OSA and Obesity Hyperventilation Syndrome
- Beware of the frequent high usage of OTC analgesics (esp NSAIDS) in this patient group due to high incidence of arthritis and back/knee pain
- Airway
 - Revolutionised by the introduction of video laryngoscopes

- Neck circumference of >54cm is the best single measure to indicate difficult intubation in obesity; when combined with MP3-4 it is even more sensitive (and suggests that awake FO intubation is indicated)
- Supine position for surgery is very poorly tolerated.
- Large amounts of opiates are required where the “fat compartment” is large – this can compound post-operative respiratory depression
- Neuraxial anaesthesia is a good alternative, but requires longer needles, and possibly a paramedian approach (especially for thoracic epidurals).
- Pre-emptive anaesthesia is useful; ketamine, other NMDA antagonists, and alpha-2 agonists (eg dexmedetomidine) are excellent. IV paracetamol, NSAIDs and even gabapentin are used too.
- Get patients to bring their CPAP machine if they have one – it improves pre-operative sleep and post-operative recovery!

Depth of Anaesthesia & Awareness – Robert Sneyd, Peninsula Medical School, Plymouth, UK

- Anaesthesia is a continuum with no units
- Depth of anaesthesia is a balance between chemically induced loss of consciousness and surgical stimulation
- Subjective assessment of DoA: Increasing BP&HR, sweating and lacrimation, movement (if not given a neuromuscular block), pupil dilation.
- Awareness
 - Conscious awareness = explicit and implicit awareness
 - Subconscious awareness = response to stimulation without cognitive function
 - Brice questionnaire – most important question is the “do you remember anything in between”
- Standard risk of awareness = 0.15% (1% in high-risk)
- Definitely damaging to patients
- How many cases of awareness are serious?
- DoA monitors:
 - Are not all equal (EEG vs AEP etc)
 - Linearised (100-0 with a fairly straight dose-response curve)
 - Monotonic (number keeps going down as you give more anaesthesia)
 - Techno-nonsense – BIS-box is a commercial secret
- Tightening up care (using ET gas monitor) show fairly equal results
- No change in home readiness with BIS, but slightly altered other factors (time to eyes open, tube out, etc.) – a few minutes each.
- Time at low BIS (<45) was a risk factor for mortality in several studies, but this was not found in other studies. Beware the “triple low” – low BIS, low MAC, low MAP.
- BIS does not predict movement, but it does predict awareness

Dose response evaluation and drug interactions of spinal and epidural drugs: concepts and implications – Warwick Ngan Kee – Chinese University of Hong Kong

- Why bother? Understanding the literature to allow application to practice, teaching purposes, or personal nerdy satisfaction.
- Example – differences between the effects of bupivacaine and ropivacaine
- Most of the science regarding dose-response curves can be traced back to insecticide research... outcomes are binary (dead or alive). In medical drug use, this requires an artificial threshold to be determined (success/failure). This is the concept of ED50
- Up-Down Sequential Analysis also uses a dichotomous outcome, deriving an ED50 in a very efficient manner, but it does not generate any information about other areas of the curve.
- Non-linear regression uses the full range of responses (non-dichotomous)
- New concept in dose-response analysis – Response-Dose analysis: “Multi-dose three-dimensional probability” curves
- Mixing and adding drugs:
 - Advantages (decreased side effects, increased efficacy, etc) and disadvantages (toxicity, interactions, etc)
 - Remember the isobolograms... compare additivity to synergism
 - Additivity is usually the effect of two drugs working at the same site, where synergism is the effect of two sites creating similar effects
 - Examples of different drugs give (opiates, ketamine, neostigmine, clonidine, etc)
- Key points:
 - Fundamental to understand the principles to understand research
 - Differences in potency explain many of the observed differences in local anaesthetics
 - Additivity is the rule rather than synergism

IV Fluids in Paediatrics – Isabeau Walker, Great Ormond Street Trust, London, UK

- Isotonicity is described in terms of sodium concentration (iso=131mol/L)
- The problem of fluids in children
 - Acute hyponatraemia!
 - Good paper = Arieff et al BMJ 1992 302:1218-22
 - Common theme in these kids – hypotonic fluid administration
 - Stress response (ADH drive)
 - Kids are unable to compensate (higher ratio of brain to skull size)
 - Difficult to recognise – malaise, lethargy, etc
 - Prevention – don’t use hypotonic fluids in children at risk or in those with plasma sodium <135 mmol/L
- Holliday and Seager formula
 - Maintenance (water requirement from formula, electrolytes from normal daily dietary intake) + replacement (isotonic)
 - 4/2/1 rule
 - Except....the stress response from NON-OSMOTIC stimuli

- ADH response is unfortunately quite unpredictable
- Use the formula as a guide to initiate therapy, and then clinical markers to adjust
- What about glucose?
 - Hypoglycaemia causes neurological damage and worse PICU outcomes
 - Hyperglycaemia causes osmotic diuresis and also worse outcomes
 - Prolonged starvation may be associated with hypoglycaemia (esp small kids)
 - Stress response worse with hypoglycaemia
 - 5% dextrose in RL caused hyperglycaemia during study
 - Most healthy children do NOT require routine intra-op dextrose
 - At-risk kids (malnourished, very young, or high requirements) should get a solution of 1-2.5% in isotonic solution (eg RL with 1% dex)
 - Age cut-off is about 6 years
- Restricted fluid regimens DO NOT protect against hyponatraemia but do increase risk of dehydration
- Post-operative maintenance – use isotonic fluid with 5% dextrose
- So, why do we still have a problem?
 - Difficult to change practice
 - Lack of local policies
 - Hypotonic fluids still often used intraoperatively
- How do we manage fluid requirements?
 - Isotonic fluids, 20ml/kg when necessary, watching clinical markers (HR, BP, capillary refill time, urine output) as well as base excess and lactate.
 - Blood transfusion if Hb trigger reached 40ml/kg reached
- See Maitland paper of NEJM May 2011 and Duke editorial of Lancet 2011 – this population had a preponderance of malaria (57%), anaemia (32% had Hb<5)
- Summary
 - Prescribe fluids with care
 - Oral route preferable
 - Monitor and respond
 - Isotonic fluids to at-risk groups

Physiology and Pathophysiology of the Elderly: Does Anaesthesia Influence the Perioperative Phase? – Merwyn Maze – University of California, San Francisco

- We have a demographic problem: the World is getting older. 30% of the developed world population will be >65 years old
- We're all going to become geriatric anaesthetists!
- Pathophysiology of aging
 - Is it a disease process? (cf. Werner's Syndrome; telomere shortening)
 - Can it be cured? (cf. caloric restriction, Sirtuin Activators such as resveratrol)
 - CNS function – 20% reduction in brain weight from 20-80 years, decrease in grey matter, decrease in neurotransmitter system functions

- CVS – hypovolaemia common (decreased thirst), reduced cardiac output, orthostatic hypotension
- Resp – Decreased TLC, VC and PaO₂
- Renal – GFR declines 1-2% per year, but due to decreasing muscle mass the creatinine value remains steady
- Homeostatic control worsens with age (Eg. Blood glucose fluctuations). Less effective temperature control, poor pH buffering capacity, less baroreceptor response
- Baroreceptor: decreased cholinergic input to the heart due to diminished receptor density as well as less adrenergic activity
- Immune function and aging – decline in lymphocyte response, IL response, etc
- Pharmacokinetic considerations
 - Change in volume of distribution (decreased for hydrophilic, increased for lipophilic)
 - Decrease in albumin concentration (this increased free fraction)
 - Increase in alpha1 protein, thus MORE binding of certain drugs (eg morphine)
 - Decrease in liver function
- Pharmacodynamics
 - MAC decreases by 5% per decade >40 years
 - Benzo's have paradoxical effect
 - Decreased sensitivity to beta-adrenergic agonists
- Spinal anaesthesia
 - Reduced CSF volume and baricity
 - Decreased latency
 - Increased hypotension and urinary retention
- Post-op pain
 - Less likely to report
 - Multimodal approach is sound
- Drug-induced cognitive impairment = DELIRIUM
 - Anticholinergics
 - Benzodiazepines
- Poor sleep hygiene in hospital/ICU is very common. Alpha2 agonists are the best choice for sedation. See the MENDS trial.