SASA Congress 2012

Refresher Course & Congress Sat 18 – Wed 22 February 2012

Ross's Rough Notes

Disclaimer – these notes were typed during the presentations I was able to attend and have been minimally edited. I cannot guarantee that they are correct; they are certainly not complete. Please use at your own discretion!

Refresher Course Day 1 – Saturday 18 February

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 Hopsital/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 diseas and clinical risk factos (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 getign 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% or 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 (O_2 is required for Na absorption, in a direct relationship).
- Furosemide may be effective in shutting down sodium absorption and increasing medullary pO_2
- 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.

Remifentanil 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. Oxymetaziline 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 antisyalogogue in preference to atropine due to it's 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

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- 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 Californa, San Francisco

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- 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 = DELERIUM
    - Anticholinergics
    - Benzodiazepines
- Poor sleep hygiene in hospital/ICU is very common. Alpha2 agonists are the best choice for sedation. See the MENDS trial.

Refresher Course – Day 2

Intubation through the Supraglottic Airway – Ellen O'Sullivan – Dublin, Ireland (and Chair: Difficult Airway Society)

- Common pattern – repeated attempts at intubation with gradually worsening face-mask ventilation until CICV develops
- DAS Guidelines of 2004 refer – 4 step plans
    - Plan A: initial tracheal intubation plan
    - Plan B: secondary tracheal intubation plan
    - Plan C:
    - Plan D:
- First rescue airway use of cLMA – prototype in 1983!
- LMA route guide to intubation – Blind/Bougie/FO/Aintree catheter with fiberscope
- Blind success rate 28-74% success; FO success rate 75-95%
- Aintree catheter technique – Anaesthesia 1994;49:543. Can oxygenate throughout.
- LMA/Aintree/Fiberscope technique can be done awake with local
- Limitations = limited mouth opening
- "Dedicated Airway" – able to ventilate while intubation is in progress
- ILMA introduced 1997
- Ventilation successful 73-100% on first attempt
- See Ferson DZ et al
- See McNeillis et al Eur J Anaesthesiol
- Proseal – better seal pressures -FOI described by Brain and thereafter others
- Brimacombe 2004 – railroading proseal over GEB placed deliberately in oesophagus – 100% first-time success
- Second-generation SADs – LTA, proseal, supreme, slippa, etc
- Which SAD is best for facilitating intubation? cLMA, iLMA, pLMA all described.

Obstetric Catastrophes – Warwick Ngan Kee – Chinese University of Hong Kong

- Airway disasters, massive haemorrhage, and (pre-)eclampsia
- Airway
    - Confidential Enquiries over the years…. AIRWAY, AIRWAY, AIRWAY
    - Obstetric failed intubation rates – 1:240 to 1:280
    - All units must have a difficult airway plan/drill/algorithm
- Massive haemorrhage
    - Case report of undiagnosed placenta acreta presented
    - Should we be doing routine cross-matches? -> if there are risk factors!
    - Moving to 1:1 (or 1:1:1) transfusion protocols
    - Should we have obstetric massive transfusion protocols?
    - Cell salvage – reduces allogenic blood use; amniotic fluid is less of a problem than anticipated; costs/staffing/setup are bigger limiting factors.

    - Other areas of interest to watch
        - Topical haemostatic agents
        - Antishock garments
        - POC coagulation monitors
        - Real-time non-invasive Hb monitors
    - Use of interventional radiology in obstetric haemorrhage is growing, but there are controversies: resources, skilled and experienced radiologists, suitable locations, lack of RCTs, differences in techniques. Potential complications: ischaemia, thrombosis, foetal complications
- Pre-eclampsia/eclampsia
    - Various diagnostic criteria from around the world
    - Possible DDx: cocaine intoxication; phaeochromocytoma
    - Pathogenesis – form of immune maladaptation leading to systemic endothelial dysfunction
    - Definite difference between those who present early and those who present late. Late = high CO, low SVR. Early = low CO, high SVR.
    - Major cause of death is intracranial haemorrhage
    - A shift in focus towards managing systolic blood pressure has occurred, as this is the factor that reduces risk of ICH
    - Another shift in focus -> uncontrolled cerebral perfusion pressure is the core problem
    - Any SBP over 160mmHg should be treated
- Several case examples presented.
- Final warning: Case of chlorhexidine accidentally administered in the epidural space. Separate the processes of cleaning the back and preparing the epidural!

Norepinephrine in Septic Shock: Why and When? – Xavier Monnet – Bicetre Hospital, Paris

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- Vasodilation is a feature of septic shock, leading to decreased arterial pressure, which in turn results in organ dysfunction
- NE increased MAP and decreased lactate in several studies
- Why NE specifically?
    - Dopamine = beta1 and alpha1 activities
    - NE = prominently alpha1; beta effects are minimal and mostly inotropic (not chronotropic)
    - NE is the most potent vasopressor
    - Arrhythmias much less common with NE
- When should we administer NE?
    - NE is aimed at restoring organ perfusion by increasing the arterial tine; therefore it must be used in cases of decreased vasomotor tone
    - DAP is related to arterial tone.
    - **NE should be used in patients with low MAP associated with low DAP and poor organ perfusion.**

- Clinical case presented
    - NE rapidly restores MAP and DAP
    - Potentials volume loading by increasing venous return (alpha receptors also on venous circulation!). NE increases cardiac preload. The volume of the venous "tank" is decreased, so fluid administration is more effective at smaller volumes.
    - Recall the MCFP vs RAP vs CO graph!
- What are the effects of norepinephrine on the microcirculation?
    - De Backer examined effects on splanchnic circulation and found that the perfusion was IMPROVED after administration of NE
    - Cutaneous blood flow studies showed that microcirculation was preserved.
    - Dubin et al in Crit Care 2009 reported improved microcirculation
- Take-home messages:
    - NE is the most potent vasopressor and should be used early in septic shock
    - Use when low MAP is associated with low DAP
    - NE recruits part of the unstressed volume and potentiates volume loading
    - NE is not deleterious to the microcirculation

Current Concepts in Pain Management – Milton Raff – Pain SA, Cape Town

- Understanding of the physiology essential
- 2 types of pain – nociceptive and neuropathic
- Nociceptive pain responds to NSAIDS, paracetamol and opioids… as does the pain of inflammation
- Most acute and perioperative pain is nociceptive
- Limbic system controls the affective aspects of pain – fear, anxiety, poor sleep, etc.
- We have at least 6 levels of defence:
    - Step 1: Peripheral activation can be counteracted by the NSAIDs and coxibs
    - Step 2: Nerve conduction can be blocked by sodium channel blockers – ie. Well-placed local anaesthetic!
    - Step 3: Calcium and NMDA channels can be influenced by gabapentin and ketamine
    - Step 4: Central activation can be again countered by the NSAIDs and paracetamol
    - Step 5: CNS opioid receptors are at our disposal
    - Step 6: Limbic modulation of the spinal cord can be influence by alpha2 drugs
- So, what's new in SA?
    - Hydromorphone – works on the central opiate receptors. Old drug elsewhere; new to us. Once-a-day formulation using OROS system (osmotic pump in hard capsule) providing 24-hour profile. Absorption is predominantly in the colon, and is thus unaffected by feeding patterns. Many metabolites; none active. Typical opiate side effects. Dose is approximately 1:5 with morphine (1mg hydro:5mg morphine)
    - Oxycontin – well known in USA, new to us. Predominantly kappa and then mu effects. Controlled-release short-acting preparation. Tablets must never be crushed or chewed.

    - Buprenorphine transdermal patch – partial agonist; highly lipid soluble, low molecular weight. Partial mu-opioid agonist. 7-day preparation. High potency. 3 doses with different size patches. May cause local reaction.
    - MOR-NORI class – mu-opioid as well as noradrenergic effects -> tapentadol

Predicting Outcome – Bruce Biccard

- Applying population risks to individual patients
- Is risk prediction warranted?
    - Prognosis vs disease vs test properties vs treatment
    - Test must be better than clinical risk factors to be useful
    - Treatment must be effective to warrant stratification of risk
- Is the patient predisposed?
    - Risk factors alone do not indicate disease in a given patient
    - Biomarkers bridge this gap
- What perioperative (dynamic) factors increase or decrease risk?
    - Almost always a function of the *type of surgery* (highest=vascular)
    - It's pointless to risk stratify patients who are undergoing low-risk surgery
    - Risk stratification should be an on-going process – changes in treatment and response modify risk continuously.
    - Blood loss and surgical duration are surrogate markers for the only true intra-operative risk: difficult surgery!
- What is an appropriate test?
    - Before the event = screening test. In CVS surgery, BNP is commonly used.
    - After event = diagnostic test.
    - If post-op diagnostic test is negative -> continue to risk stratify.

TIVA Modelling: Where are we going? – Jeff Coetzee – University of Stellenbosch/Tygerberg Hospital

- Can we predict the combined effect of multi-drug TCI? Pharmacokinetics and dynamics of multiple agents
- Clark receptor occupancy theory – response is proportional to the ratio of bound to unbound receptors.
- A dose-response curve can be thought of as an effect site concentration-effect probability curve…but we can't chase 100% probability, as certainty of effect is where toxicity lurks
- Mixing additive or synergistic drugs moves the area of certainty away from the area of toxicity
    - Nitrous oxide + volatiles = additive
    - Propofol + midazolam = synergistic
    - Volatile + sufentanil = synergistic
    - Propofol + opioid = synergistic

- Administering two drugs simultaneously produces a multitude of dose-response curves. Plotting the relationship between two drugs in three dimensions creates a "dose-response surface" for the "new" drug (the combination).
- We can find the right combination of drugs to promote the most rapid recovery from the infusion.
- We will soon be seeing "live" isoboles on graphical displays on our TCI pumps (cf. Drager SmartPilot View)
- George Box: "Remember that all models are wrong; the question is how wrong they have to be to no longer be useful."

Outcomes in Anaesthesia in South Africa – Christina Lundgren

- Most fundamental measure = mortality
- Morbidity = defined complication or a critical event
- Closed claims study will soon commence in SA
- What do we mean by "Anaesthetic Death"?
    - Four basic categories
    - 1 – Death solely due to anaesthetic
    - 2 – Anaesthetic contributory
    - 3 – Anaesthetic associated
    - 4 – Death solely due to surgery
    - No agreement in the international literature/practice
- The literature in SA:
    - 1930's in SA: 1 in 1000 anaesthetics
    - 1950's in SA: 1 in 1000 surgeries; 5 in 10 000 AAD/ACD
    - 1956-1987: ACDs decreased from 0.43 in 1000 to 0.07 in 1000
    - Good analysis: Coetzee & Du Toit 1987
- The "24-hour cut-off" does not exist anywhere in SA law
- SA data from Lundgren study presented.

Enhanced Recovery – Wilson & Mark Daugherty (UK)

- Google it. There is lots of info ;) NHS has very good patient information.
- Consistent clinical pathway
- Pre-assessment & preparation for surgery
- Pre-op fluids – high energy drinks
- No bowel prep
- Targeted fluids
- No NG tube
- Early feeding
- Early mobilisation
- Early removal of drips and drains

- Avoidance of opiates
- What peri-op analgesia (Where is the evidence?)
- Rectus sheath blocks are taking over from epidurals, and there is evidence to suggest that they improve outcomes.
- Multiple sets of audit data provided.

Congress Day 1

Very few notes I'm afraid – during the opening morning session I was absorbed with the registrar's stream, and then spent the whole afternoon in various the airway workshops. Between the two I attended these two sessions:

Clinical haemodynamic monitoring – Ivan Joubert – Department of Critical Care, University of Cape Town & Groote Schuur Hospital

- The numbers themselves are not the target!
- Goal is perfusion – harmony at a cellular level
- What is shock? Physiologic -> inadequate tissue perfusion
- The tools we traditionally use:
    - ECG – rate and rhythm
    - BP – a surrogate for perfusion
    - Urine output
- These are blunt tools! More confounds the issue:
    - Relationships are almost never linear!
    - Resuscitating never gets us back to the starting point – to get the numbers the same we tend to over-resuscitate
- We need better end-points!
- FIRST trial used as a discussion point – what resus endpoints to use?
- HR, CVP and MAP did not correlate well with improvements in lactate.
- The problem with CVP…
    - Has been used as a "Gold Standard" for optimising fluid volume
    - CVP is a function of posture, ventricular compliance, systemic venous tone, intrathoracic pressure and fluid volume.
    - Many pitfalls in interpreting CVP
    - Data suggests it is not reliable in the critically ill NOR is it reliable in healthy patients!
    - PCWP suffers the same drawbacks
    - See Kumar et al Crit Care Med 2004;32:691-699 for study of CVP in healthy patients
    - LVEDV/RVEDV/stroke volume index – NONE of these had a correlation with CVP before and after fluid loading.
    - CVP in critically ill – see Lichtwarck-Aschoff et al, Intensive Care Med 1992
    - The use of monitoring changes in CVP in response to fluid challenges was proposed initially based on tenets of cardiac physiology, but has never been proven through study

    - See Chest 2008 – Does CVP predict fluid responsiveness – A Systematic Review and A Tale of Seven Mares
    - The receiver-operator curve for CVP/fluid responsiveness follows the line of equal probability.
- What are good goals?
    - Good perfusion is the ultimate goal
    - Warm peripheries (toes and nose)
    - Clearance of lactate
    - Improvement in the blood gas (pH and standardised base excess)
    - Pulse oximeter
        - What is the plethysmograph? It's a volume/flow change detector!
        - Why look at the trace? It tells us about peripheral capillary perfusion!
        - Use of ventilation-induced plethysmographic variations to optimise patient fluid status (See Desebbe & Cannesson, Curr Op Anaes 2008 21:722-778)
        - Monnet & Teboul Crit Care 2005 – Do we have our finger on the solution?
- The big focus?
    - Dynamic indicates such as…
    - Systolic pressure variation
    - Pulse pressure variation
    - Etc.

Xavier Monnet – Paris Bisect Hospital

- Starling is a curve, not linear – we cannot expect a linear response. There are many Starling curves depending on the patient's age, condition and pathology.
- Giving too much fluid to patients increases mortality (see ARDSnet study!)
- How do we predict fluid responsiveness?
    - CVP has been disproven
    - Responders and no-responders cannot be separated by CVP value (See Osman 2007)
    - Repetitive fluid challenges can be used, but over time this results in fluid overload
    - Respiratory variation of pulse pressure does not require fluid administration, and has been shown by multiple studies to be an effective measure
    - PP variation does have important limitations:
        - Can't be used if the patient has arrhythmias
        - Can't be used if the patient has spontaneous breathing efforts
        - Can't be used in ARDS/low VT/poor compliance
    - Only a small portion of ICU patients are suitable for PPV testing
    - End-expiratory occlusion (EEO) allows increased venous return, and can thus predict fluid responsiveness
    - This can be demonstrated very easily if the patient is on a cardiac output monitor, and works even better if patients are on high PEEP (such as ARDS)
    - The passive leg raise (PLR) test also predicts fluid responsiveness well without risk of fluid overload. 10% increase in cardiac output is important; using arterial pressure results in false negatives.

Congress Day 2

WHO Checklist – Iain Wilson – Royal Devon and Exeter, UK (President: AAGBI)

- Example given of RAF Phantom which shot down RAF Jaguar in training exercise
- System failure, human errors, communications
- Examples given of medical failure
- Wrong site surgery is catastrophic but rare
- More common issues
    - Thromboembolic phenomena
    - Infections
    - Unexpected haemorrhage
- Could a checklist help?
    - Effective in other industries
    - Either as an aide memoire
    - Or as a mandatory tool
- Good example from medicine – Pronovost Central Line paper for preventing CLABSI
- What is the evidence that the checklist makes a difference?
    - Pilot study – inpatient complications from 11 down to 7%, mortality from 1.5 down to 0.8%
    - Neily et al JAMA 2010 – teamwork training and checklist
    - Ann Surg 2012;255:44-9 – where checklist is used properly, greatest reductions in mortality are seen
- Pre-list briefing is a brilliant strategy
- Communication issues are involved in 60% of theatre issues
- Challenges to checklist
    - Incomplete
    - Hurried
    - Tick box culture
- Being safe vs. documenting safety
- Where next – crisis checklists?

Drug Errors – Peter Gordon – Department of Anaesthesia, Groote Schuur Hospital/University of Cape Town

- Any error, irrespective of whether harm is caused
- Prescribing/administration/dispensing
- Mistakes vs. Slips vs. Lapses
- Prescribing errors – lack of knowledge, illegible handwriting, confusion of name (diltiazem vs. diazepam), decimal points, abbreviations (AZT?)
- Results of local and national survey presented
    - 94 % of anaesthetists had given drug error

    - Muscle relaxants and vasoactive drugs most common
    - 4 attributable deaths (lignocaine, propranolol, esmolol, adrenaline)
    - Centralised reporting agency suggested
- Prospective study, 3 teaching hospitals, 30000 anaesthetics
    - Incidence of errors: 1:274
    - Approximately 6 errors per month per hospital
    - Misidentification and similar looking ampoules most common cause
    - Morbidity fortunately uncommon
- Similar studies in New Zealand; 1 error per 200 anaesthetics (Merry & Peck)
- Reason's "Swiss Cheese" model – system vs. people errors
- Reading errors – often due to pattern (mis)recognition
- Far greater incidence of errors after midnight
- Education – we must create a safety culture from medical school onwards
- "We should ensure we do not read what we expect, but inspect what we read."
- Adopt a universal colour-coded labelling system in theatres
- Improve drug storage
- Reduce fatigue
- Develop a "reporting culture"
- See Camire E et al. CMAJ 2009; 180:936-943

The GO (Global Oximetry) Project – Ian Wilson – President AAGBI

- Anaesthesia in countries such as SA, UK very strong and physician lead
- Driving force in patient safety
- Safety of anaesthesia has improved dramatically over the last 6 decades
- Safety improved by 18x around the 1980's – this was largely due to the widespread introduction of pulse oximeters
- AIME study (Australia) – 82% of incidents could have been presented with oximetry
- Survey in Uganda 2007-8: no hospital met WHO standard; minimal staffing; minimal equipment
- Outcomes in anaesthesia in many areas of the developing world are desperately poor (mortality 1:200!)
- How do we improve outcomes? Personnel, equipment, monitoring
- Concept of "Global Oximetry"
    - Walker et al Anaesthesia 2010 - Gap data, ideal oximeter, training etc.
    - Barriers to oximetry – EXPENSE, EXPENSE, EXPENSE, robustness
    - Pulse oximeter added to the WHO checklist
    - About 77000 theatres worldwide without oximetry
    - New branding: "Lifebox"
    - $250 robust oximeter
    - Fundraising webpage
    - Coverage on WHO and other webpages
- Pulse oximeter that connects to cell phone under investigation

- www.lifebox.org
- Worthwhile read: Atul Gawande – "Checklist Manifesto"

The Anaesthetist and the Environment – Robert Sneyd – Peninsula Medical School, Plymouth, UK

- The greatest medical crisis on Earth are the effects of the looming climate change disaster
- Lancet commission – managing the health effects of climate change
- Doctors must take a lead in being advocates for heeding effects of climate change
- Beyond CO2… we (anaesthetists) are prolific users of disposal devices, particularly plastics
- Leaching of "Gender Bender" compounds from plastic administration sets (particularly into lipid emulsions such as Propofol)
- Bisphenol A (BPA) is associated with higher rates of heart disease
- NHS (UK) carbon footprint is 18 million tonnes CO2 per annum (energy 22%, transport 18%, and procurement 60%)
- Sustainability = "ensuring a long-term strategy that does not preclude our ability to deliver high-quality health care tomorrow"
- Why to sign up? Save money, legal compliance, enhanced reputation, resilience, better patient care into the future
- Moving patients (under their own transport or that of the system) is a huge environmental cost – improving local access to care (and preventing complication/readmission) reduces this.

Ergonomics and the Anaesthetist – Ian Findlay

- Ergonomics is the interaction between the anaesthetist and technology
- The anaesthetic machine is the major point of interaction
- Interaction with the flow meters requires a small percentage of our time (3% visual, 2% physical) but is rated as the 3rd most critical safety interaction.
- Introduction of new types of flow meters (electronic controlled) has never been tested scientifically against the older (mechanical rotameter) technology.
- This was studied for the first time by SA researchers using a standardised accepted (NASA) task load index questionnaire
- Objective workload was higher for electronic meters, while subjective workload was lower.
- Inconsequential clinical errors were high in the mechanical group; clinically relevant errors were high in the electronic "low flow" group.
- A second study under stressful conditions found that the majority of errors occurred with electronic flow meters.
- Model of human performance: Perception -> Situational Awareness (interpretation, comprehension, projection) -> Response selection -> Response Execution -> System Environment Feedback -> Perception and so on…
- Low-flow meter adjustment usually occurs at times of low task density, and is accomplished using closed loop feedback.

- Electronic device have device-specific action sequences, which, if completed in the incorrect order or left incomplete, result in a return to the baseline settings.
- What can we conclude from this assessment?
    - Subtasks required differ between the two types of flow meter
    - Sources of workload differ
    - Error types differ – manual = calculation; electronic = perception of state
    - Electronic systems particularly sensitive to action sequence and post-completion errors
    - Context-sensitive assessment of new technology should be undertaken.
- Resources available from ianfindlay7@gmail.com

Automated Anaesthesia Record Keeping: Pros and Cons – Zane Farina – Pietermaritzburg, SA

- History
    - First anaesthesia record – A.E. Codman at MGH 1894
    - "Record the pulse" – FB Harrigan at MGH
    - First published – Cushing 1902
    - BMJ 1896: "By recording blood pressure we pauperise our senses and weaken clinical acuity"
- What is an anaesthetic record?
    - Medicolegal - An accurate record is more important than a "nice looking record". AARK – more accurate data, improved completeness and legibility, automatic alerts
    - Training tool
    - Checklist (airway, ventilation, antibiotics, positioning, etc.)
    - Audit tool
    - Situational awareness tool – "writing as cognition"
- What should the AARK do?
    - Facilitate above uses
    - Improve situational awareness
    - Free time for complex problem solving
- Achieving the aims
    - Every surface a computer
    - Able to track all aspects of anaesthetic (eg. Drug administration; airway devices)

Using TCI to Deliver TIVA to Children – Graeme Wilson – University of Cape Town/Red Cross War Memorial Children's Hospital

- First commercial TCI device = Diprifusor, 1996
- Rapid acceptance everywhere (except the US!)
- Millions of TIVA's given safely around the world
- Introduction of TIVA/TCI into paediatric anaesthetic practice has been slow
- Propofol infusion syndrome has been a concern

- TIVA in children is not easy to administer (and standardised manual TIVA protocols don't really exist for kids)
- Paediatric TCI software is not ubiquitous
- Why TIVA in children?
    - Contra-indications (eg. Dystrophies, MH, etc)
    - Locations remote from OT
    - More specialised surgeries being attempted (esp. neuro)
    - Quality of recovery
- Goals:
    - Desired, stable effect
    - Considerable PK/PD variation
- How does TCI work? Loading dose + infusion to match redistribution + infusion to match clearance (…expand for as many compartments as you are for…)
- Kids vary – LOTS! Growth and development bring about profound changes
- Increased volume of distribution and higher clearance – infusion rates are much higher (about 50% more on a per-kg basis)
- Context-sensitive half-times are thus much longer
- Using adult models in kids results in much lower plasma concentrations than predicted
- Commercial models are not available for children under 12 months
    - Hepatic metabolism immature
    - Reduced metabolic elimination of Propofol (glucoronidation; CYP isoenzymes)
    - Postconceptual vs. postnatal age
- Distributional clearance in l/hr/kg decreases with age in a non-linear relationship (in adults this becomes quite linear)
- Allometry – non-linear relationship between the size of the organism and function (eg. Head size in relation to body weight as an individual ages). Allometric scaling better describes the relationship between weight and metabolic functions (using a power scale of ¾)
- The smaller the child, the higher the cardiac output and clearance
- See Myburgh et al Int Care Med 2001 for effects of increasing CO with inotropes on plasma Propofol concentration
- Currently there are two commercial available paediatric models in SA (neither perfect, each with strengths and weaknesses):
    - Paedfusor
        - Based on Marsh model
        - Validated from children 1-15 years
        - Uses weight-adjusted model for children up to 12 years
        - Limited allometric scaling
        - Gentle
        - Plasma targeting
    - Kataria
        - Older model, from 1994. Based on small sample (53 kids, 3-11 years)
        - Compartmental volumes are a linear function of body weight
        - Clinically it works surprisingly well
        - Heavy handed

        - Plasma targeting
    - Very little to generate a significant preference
    - Titration to effect is the most important!
- Hopefully newer models will incorporate better allometric scaling
- Practical use:
    - EMLA if absolute contra-indication to gas, otherwise gas induction
    - Use an adjuvant
    - Paedfusor model if possible
    - Plasma targeting, so be patient during induction and emergence
    - Similar plasma targets to adults
    - Use a dedicated cannula with a one-way valve if possible, and avoid dead space
    - Beware mixing drugs
    - Beware refilling of syringes (profofol causes rubber stopper to become sticky)
- Open TCI initiative – www.opentci.org
- Closed loop TCI coming sometime in the future?
- New drugs?
    - AZD 3043
    - Esterase metabolised hypnotic agent
- "The only principle of drug dosage that survives is that the drug dose must be adapted to the individual patient" (1940!)

Computer controlled technologies in anaesthesia – Robert Sneyd – Peninsula Medical School, Plymouth, UK

- Electronic records and fancy displays
    - E-records = improved quality and perhaps savings
    - Fancy displays = ?
    - Interaction models on your monitoring systems = cool for specific cases, great for teaching, fairly useless for day-to-day work
    - Lack of compatibility between different manufacturers systems is a huge issue
- TCI is great.
- Closed loop systems are the next stage
    - Well established concept in engineering (from air conditioning to aviation!)
    - Mostly still experimental in medicine!
    - Pump -> patient -> EEG monitor -> analyser -> controller -> pump
    - Research on rats with EEG monitor explained
    - Research in humans (using BIS) is on-going
    - Commercial systems are not far away
- SEDASYS – automated closed-loop sedation system. Works well in study of 1000 patients
- TCI can also be applied to volatiles (Drager Zeus)
- Automated control of ICU ventilation (ASV, eg Hamilton Intellivent) and weaning
    - Adaptive Support Ventilation

    - Otis relationship (change in TV/RR with changes in WOB)
    - Monitors EtCO2, FiO2, SpO2, PEEP, Paw and adjusts as necessary
    - See Lellouche et al AJRCCM 2009
- Continuous closed-loop glucose control – sensors are a problem at the moment, but systems are developing rapidly

Cardiac causes of weaning failure – Xavier Monnet – Hospital Bicetre, Paris

- Increased LV preload, increased cardiac work -> possible cardiogenic pulmonary oedema
- Anxiety induced hypertension adds to LV afterload
- Vicious cycle!
- Weaning failure from cardiac origin was 87% in one study
- Suspect if:
    - Obvious other causes of failure have been excluded (eg. ongoing pneumonia)
    - There is pre-existing COPD and/or LV dysfunction
- T-piece trial is the most sensitive test to detect cardiac failure in weaning, as the negative pressures are the greatest.
- How do we pre-diagnose cardiac-origin weaning failure?
    - PA catheter can demonstrate increased PAOP (by definition required for pulmonary oedema). However, measuring PAOP can present a significant challenge (and requires a PAC, which are becoming much less common).
    - Cardiac output must increase during weaning – if it does not, the patient will fail
    - Decrease in SvO2 may be a marker as well – demonstrated on one study
    - Echocardiography allows estimation of PAOP can gives real-time information on cardiac function and fluid status. E and E/Ea measurements are required to predict weaning failure – they are not, however, perfect in correlation with PAOP. Echo also requires a very experienced operator as it is very challenging in these patients.
    - BNP is a well-known marker of pulmonary oedema. Grassno (Crit Care Med 2007) used expert opinion to determine patients in cardiac failure; NT-proBNP increased to a much greater extent in the patients with failure. The ROCurve was remarkably good, but the study has important limitations (no PAC hence not "gold standard"; renal failure patients excluded).
    - Plasma protein concentration: Pulmonary oedema is the result of a huge migration of plasma into the interstitium of the lung, and must therefore cause a haemoconcentration effect. (See Figeuras & Weil, Circulation 1978). Monnet and Teboul measured [plasma protein] in t-piece weaning (with PAC in place) in 46 patients, showing no significant change in concentration in those who did not develop PE, but a large increase (>6%) in patients who did. This could be a very practical method, although it has only been demonstrated in this one study so far. **Look at patients with new pulmonary oedema for an increased [plasma protein], and watch the concentration fall as treatment is commenced.**
- Take-home:
    - Weaning failure from cardiac origin is not rare

- Expect it in patients with COPD and LV dysfuncton
- All weaning trials are not equivalent; the t-piece trial is the most challenging for the heart
- The PAC is the gold standard, but it's invasiveness is problematic in this context
- Echo is powerful but highly technical
- BNP/NT-proBNP is promising but requires further study
- [Plasma protein] is very promising but requires validation

Permissive Hypoxaemia – Jenna Piercy – University of Cape Town/Groote Schuur Hospital

- Lots of research, lots of questions…but lots of conjecture
- "This first atmospheric pollutant…rusts a person in 100 years or less…if introduced today may never achieve FDA approval." Severinghaus
- Paracelsus – it's all about the dose. Oxygen is a drug!
- Should we be dosing oxygen on SpO2 or PaO2?
- Is the quest for normoxaemia damaging and dangerous?
- Permissive hypoxaemia:
  - Lung protective strategy
  - Minimise pulmonary and systemic damage
  - Balance improved SpO2 against risk of barotrauma, volutrauma, toxicity, haemodynamic changes, etc
  - ARDSnet (2000) – low Vt, low Pplat -> reduced mortality and ventilator days. Not just about the lungs, however: these patients also had less circulatory, renal and coagulation failure. Concept of biotrauma and orhan cross-talk… "extrapulmonary organ failure"
  - High FiO2 exposure can cause ARDS-like lesions in the lung, including hyaline membrane formation, pulmonary artery adaptation.
  - Neonates resuscitated with 21% O2 had less mortality than those resus'd with 100%
  - Neonatal retinopathy caused by high O2 concentration
  - Severe TBI – extreme hyperoxaemia (PaO2 >64kPa) had higher mortality
  - Nontraumatic cardiac arrest – higher mortality in the hyperoxaemic group than normoxaemic AND hypoxaemic!
- Reactive oxygen intermediates are considered to be a likely cause of these deleterious effect (eg superoxide anion, hydrogen peroxide, hydroxyl radical)
- Hypoxia increases adenosine, which acts on the A2A receptor, downregulating inflammation. Reduced adenosine worsens inflammation, exacerbates ARDS and accelerates death (animal data – Theil et al PLoS Biol 2005)
- Hyperoxia + aggressive ventilation is worse than either separately (Animal data)
- Arterial hypoxia does not imply tissue hypoxia – this depends of oxygen DELIVERY
- How do we manipulate O2 delivery?
  - Think of DO2 equation – SaO2, Hb, CO
  - Think of VO2 – reduce oxygen consumption
    - Ensure good sedation

        - Relax/paralyse if we need to
        - Cooling if necessary
        - Avoid ventilator asynchrony
    - Determining CO is very difficult in critically ill patients
    - Inotropes alter the balance of DO2 and VO2
    - Driving haemoglobin – what values to target?
- What evidence is there that hypoxaemia is safe? EVEREST! See Grocott NEJM 2009
    - SaO2 relatively well maintained due to left shift of OHDC, increase in Hb, and likely an increase in CO (not measured on the South Col ;))
- Clinical trials:
    - Adequate DO2 is adequate; more is not better
    - Permissive hypoxaemia is complemented my permissive hypercapnoea
- In the pipeline:
    - OXYGEN-ICU study (Italy)
    - BRAINOXY study (Finland)
- What should we give?
    - FiO2< 0.4 is non-toxic
    - FiO2 > 0.5 harmful
    - Titrate to achieve SpO2 88-92%
    - Supranormal delivery of O2 is NOT advantageous

How to deal with the haemodynamic effects of PEEP – Xavier Monnet – Hospital Bisetre Paris

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- Is it important?
    - Yes! We should apply high PEEP to our ARDS patients (ARDSnet trial)
    - PEEP has some deleterious effects on haemodynamics
    - PEEP increases fluid volume requirements
- How does PEEP alter haemodynamics?
    - PEEP increases both the intrathoracic and transpulmonary pressures
    - 2 main (possible) consequences:
        - Decreased right cardiac preload (intrathoracic pressure adds to the RAP, limiting flow back to the heart and thus decreased preload). However, this has been difficult to demonstrate in studies and may not be that important. This may be because it is only seen when the right heart is preload/volume dependant. Administering fluid prevents this problem.
        - Increased right cardiac afterload - increased lung distension causes and increase in pulmonary vascular resistance (by forcing more areas of the lung into West Zone 3) Over time this may cause a right ventricular distension, and at the most serious stage an acute cor pulmonale.
- How do we limit the deleterious effects of PEEP on the RV afterload?
    - Limit over-distension by limiting the plateau pressure
    - Ensuring that the patient is volume replete increases the proportion of West Zone 1 or 2 rather than Zone 3
    - Limit hypocapnoea and hypoxia (as these can cause pulmonary vasoconstriction)

- Provided that we respect the safety limit of plateau pressure (30cmH20), PEEP along does not increase the incidence of acute cor pulmonale.
- How do we detect PEEP-induced haemodynamic impairment?
    - Suspicion of RV failure if combination of
        - Decreased SV
        - Raised CVP
        - Decreased ScvO2
    - Decreased cardiac index/output
    - Echocardiography -> RV dilatation of acute cor pulmonale (RV larger than LV)
- Take-home:
    - Respiratory benefit of increasing PEEP is counterbalanced by haemodynamic impairment
    - PEEP-induced haemodynamic impairment is mainly related to increased RV afterload
    - PEEP-induced RV afterload increase may be attenuated by limiting Pplat and restoring volume status
    - Detect PEEP-induced impairment through monitoring and/or echo

Sense and Sensibility in Transfusion for Trauma Surgery – David Muckart – Inkosi Albert Lithuli Hospital/University of KwaZulu Natal

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- Massive blood transfusion – many different contrasting definitions!
- Replacement with what?
- To what end point?
- What about component therapy?
- "I shall not attempt further to define it, but I know it when I see it."
- Concerns
    - Tissue injury and CoT (decreased thrombin, increased fibrinolysis)
    - Acidosis (decreased FVII activity, platelet function, clotting function; increased fibrinolysis)
    - Hypothermia (decreased platelet function and coagulation enzyme function)
- Hb to aim for in trauma – 10g/dl is sensible (TRICC data applies to stable critically ill patients)
- PRBC:FFP – Duchesse et al – if less than 10u transfused, 1:1 ratios don't really make a big difference. If more than 10u, the mortality benefit of 1:1 begins to emerge.
- Kashuk et al (J Trauma 2008) showed the ratio is likely to be closer to 1:2 (some data suggest 1:1.4)
- 1:1 does seem to cause an increase in complication (TRALI, immunosuppression?)
- PRBC:platelets 1:1 is important
- What you do in the first 6 hours counts most
- Ideal ration PRBC:FFP:platelets this possible 1:2:1
- rFVIIa seems to be dead in the water; perhaps the studies should have looked at using it much earlier in resus

- CRASH2 – Muckart is a non-believer, casting aspersions on the whether the data is too good to be true; another study to confirm or refute would be good.
- How to go about this practically:
    - <6 PRBC: Guided by TEG if you have it, otherwise PRBC only
    - 6-10 PRBC: Platelets 1:1, plasma 1:2-3
    - >10 PRBC: Platelets 1:1, plasma 1:2, cryo

Congress Day 3

Is there a role for pre-emptive analgesia – Janneke van Nugteren – Groote Schuur Hospital/University of Cape Town

- "Treatment initiated before and operational during the surgical procedure in order to block the physiological consequences of painful stimuli"
- 2 approaches – PRE vs. NO and PRE vs. POST
- Basic physiology: Tissue damage -> peripheral sensitisation -> altered transductions and increased conduction to CNS -> hyperalgesia & allodynia
- Central sensitisation = pain memory in the dorsal horn. Good article: Latremoliere A et al J Pain 2009;10:895-926
- First-line sensitisation = normal response to early sensitisation. Protective mechanism to reduce ambulation and promote healing.
- Second-line sensitisation = ongoing peripheral inflammation and nerve injury. Spontaneous peripheral ectopic generation of action potentials, structural changes in synaptic function, apoptosis of inhibitory interneurons, etc.
- Woolf CJ, Central sensitisation; Pain 2011;152:S2-15
- Clinical evidence for pre-emptive analgesia:
    - 6 Systematic reviews, fraught with issues.
    - Biggest = Moiniche et al 2002 & 2004. Very contradictory results. NSAIDs more promising, LA/epidural no good.
    - Next biggest = Ong: LA/epidural useful!
- What about ketamine?
    - Remerand et al AA 2009 – significant reduction in ketamine group
    - Sen H et al AA 2009 – reduction in ketamine and gabapentin groups
    - Ryu HG et al Clin Pain – ketamine no good
    - Duale et al Eur J Pain – no difference
- What about gabapentinoids?
    - Sen Het et al Eur J Anaes – reduction in gabapentin group
    - Buvanendran A et al AA 2010 – No neuropathic pain in pregabalin group, 5% in control group
    - Burke et al AA 2010 – decreased VAS with pregabalin
- Why have studies failed us?
    - Inadequacy of the animal experimental model

    - Insufficiency of pre-emptive analgesic techniques (we don't know what we should be using, for how long, and how much)
    - Operations are not equal; surgical techniques differ, etc.
    - Complex physiology
    - Difficult outcome measures
    - Patient factors
- Should we be abandoning the term "pre-emptive" analgesia and aim for "preventative" analgesia (adequate duration, adequate intensity). See Dahl & Kehlet
- Further strategies:
    - Good study design – detailed pre-op assessments, identifying high risk patients, documenting surgical handling of nerves, assessing functional consequences of pain
    - Procedure-specific pain guidelines (See www.prospect.org)
    - New drug designs – blocking nerve growth factors, modulating microglial activation, transient receptor potential antagonists, cytokine antagonists.

Opioids and Respiratory Depression: PCA – Eric Hodgson – UKZN

- Classify patient and surgical risk and try to match the two to achieve adequate pain control
- Assess pain control and sedation – AVPU scale useful. Pain can only be rated by patients who are spontaneously awake. Patients who have to be woken to ask them about pain don't need more analgesia!
- Graded response to pain stimulus: glabella tap -> trapezius pinch -> jaw thrust
- Asleep patients: if RR>10, leave to sleep. If RR<10, assess LOC
- VAS is a research tool. Just ask your patients to assess their pain (need drugs or not?)
- Premedication/night sedation:
    - Benzo's can cause paradoxical reactions and be ant-analgesic
    - Amitriptyline synergistic and sedative
- PCA drugs:
    - Morphine – long on and off-set. Active metabolite (M6G) which accumulates with renal dysfunction
    - Fentanyl – gaining favour. No active metabolites
    - Pethidine (meperidine) – absolute no
    - Tramadol – anecdotal success
    - Ketamine – limited efficacy in unselected patients. Good in opiate resistance and chronic PCA use
    - Alpha2 agonists – synergistic and antiemetic.
    - Ketamine + dexmedetomidine as "opioid resensitisers"
    - Antiemetics – metoclopramide doesn't help and risks EPSEs. 5-HT3 antagonists good. Don't bother if the patient doesn't complain of nausea
- IM injections – either ineffective or too effective! Subcutaneous is possibly better, using frequent small boluses.
- PCA does have risks of adverse effects
    - Excessive sedation -> coma -> death

    - Local anaesthetic toxicity
    - Medication errors
    - Accumulation
- Principles of PCA safety:
    - Avoid IV PCA; use SC
    - Use disposable pumps for opioids; mechanical for local
    - If using a lager bolus, use a longer lockout.
    - Background infusions only in HDU
    - In the wards use only PCA (not NCA)
    - Morphine still the most widely used; fentanyl in elderly/OSF
- Local techniques
    - Keep it CIMPLE
    - Field infiltration, catheters in wounds, nerve or plexus blocks
- PCEA is gradually becoming less popular as the rate of peripheral catheter use increases
- Future
    - wound and US-guided catheters
    - PCRA (patient controlled regional anaesthesia)
    - Liposomal bupivacaine (Exparel)
- Beware: BTTWWADI (But That's The Way We've Always Done It!)
- www.riskybusinessafrica.co.za
- http://tinyurl.com/817px9y

The Link between Acute Postoperative Pain and Chronic Pain Syndromes – Gillian Lamacraft

- Pain is a common feature before surgery and almost always follows surgery
- Postsurgical pain is the second most common cause of chronic pain in Pain Clinics (most common = degenerative cause)
- Macrae and Davies (1999) – pain lasting more than 2 months after surgery, if other causes (eg. malignancy/infection) have been explored and excluded
- Incidence of Chronic Post-Surgical Pain (CPSP)
    - Likely to be linked to the level of damage (but can occur with minor surgery)
    - Well recognised after certain types of surgery (eg. amputation)
    - Also happens after other types - hernias, breast augmentation, vasectomy!
    - Incidence 30% after Pfannenstiel caesarean section!
- Basic physiology – see earlier talk. See also CMAJ 2006
- Current interest in the epsilon isoform of protein kinase C
- Risk factors for CPSP
    - Modifiable – pre-operative pain; long-term opioid use
    - Non-modifiable – age, genetics
- Hyperalgesia from chronic opioid use can be addressed with use of ketamine (evidence not all positive), NSAIDs, Gabapentin, Nitrous oxide (reduced CPSP)
- Epidural & perineural catheters
- Pre-emptive vs. preventive analgesia

    - Reduce chronic pain
    - Improve functional status
- Depression can cause and be caused by pain
- Pre-op depression causes increased incidence of CPSP
- Rx with antidepressants often effective

Practical Paediatric Pain Management – Jenny Thomas – Red Cross War Memorial Children's Hospital & University of Cape Town

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- Pain management at RCWMCH
    - Multidisciplinary
    - Anaesthesia-directed, nurse driven
    - Physio, OT
    - Creative therapies – volunteers (psychotherapy, aroma therapy, art, music)
    - Child life specialists
    - Weekly meeting
- What is needed?
    - All in the mind!
    - Believe in yourself: you can do this in your environment
    - Teamwork
    - Coach (you?)
    - Knowledge is power – learn and teach
    - Know the drugs available and use them judiciously
    - Balance confidence with humility
- In the beginning (20 years ago)
    - Morphine, NSAIDs, paracetamol
    - No assessment tools
    - Minimal use of LA regional techniques
    - Surgeons: "Sedate with panc"
    - Opioid phobia
    - "They [the children] will not remember"…and other misconceptions
- Where are we now?
    - Enlightened parents
    - Safety
    - Efficacy
    - Pathology
    - Improved techniques
    - Appropriate medications
    - Sedation/analgesia techniques outside of the OR
    - Audit: critical adverse events
    - Science-based evidence in paediatrics
    - Assess -> Solve -> Implement -> Measure
- If you can't talk to the patient, talk to the parent.

- Case reports & ideas presented
- Practical points:
    - Give drugs in Coca-cola (sugar & bubbles)
    - Rescue medications: Valoron (tilidine HCl) SL and/or ketamine
    - Perfalgan (IV paracetamol) prior to induction if the patient has an IV
    - Weaning of drugs
    - Value of preparation (talking, explaining, child life specialists)
    - Anxiety
    - Role of mother/parents

Making a Difference – Robert Sneyd – Peninsula Medical School

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- Making a difference… for patients and their outcomes, for institutions and healthcare systems, for strategic leadership, political engagement, international and environmental stewardship.
- Good start: get as good as you can through study, CPD, research, etc.
- Leadership challenges:
    - Self-leadership – be open to the evidence
    - Challenge your colleagues to put it into evidence
- Bad stuff:
    - Are anaesthetics bad for you?
        - cumulative deep hypnotic time - current evidence inconclusive -> no reason to change practice now
        - Beware the "triple low"
        - Watch the literature
    - Can anaesthetics cause cancer?
        - Serum from patients receiving regional blocks inhibited cancer cells; morphine caused proliferation
        - Regional anaesthesia may suppress cancer!
    - Are we damaging babies' brains?
        - No difference in cerebral blood flow
        - Mice show inattentive behaviour after neonatal sevo exposure
        - Be aware that we may be damaging baby brains…
- Changing institutional behaviour changes institutional outcomes (think of ERAS)
- Examples: Hip Fracture Network and Emergency Laparotomy Network
- Evidence-based practice is not discretionary – it's a duty!
- Data=Knowledge=Power
- Political leadership: Engage! There is no "them" in society, there is only "us".
- Get involved in clinical leadership
- Anaesthetists are equipped with the ideal skills to lead
- Helsinki Declaration for Safety in Anaesthesia
- "Stick your head out and be a pain in the arse!"

The Disrptive Doctor – Sean Kaliski – Forensic Mental Health Services – University of Cape Town & Groote Schuur Hospital

- Increasing awareness of bad behaviour; corresponding increase in ethical guidelines
- HPCSA – No supercession, no casting aspersions, reporting impairment.
- AMA 2010: "Inappropriate behaviour" = conduct that is unwarranted and is reasonably interpreted to be demeaning or offensive.
- Sexual harassment; racial/ethnic slurs; intimidation; abusive language; aggressive; persistent lateness; etc.
- Staff leave, and patient care quality decreases.
- Loss of professionalism in the profession.
- Not a lot of data exist
- 3-5% of medical personnel display a pattern of disruptive behaviour
- 75% are in the surgical disciplines
- Difficult to differentiate from justified behaviour
    - Difficult circumstances
    - Justified complaints
- 2 types:
    - "Impaired schmucks"– drugs, alcohol, psychiatric disorders, poor levels of competence. Generally reported/disciplined for incompetence or bad outcomes.
    - "Competent bastards" – Often excellent technicians, but horrible persons.
        - The Old Guard – prominent member of medical fraternity, but has hair trigger and known "issues"
        - The Trauma Drama – young, energetic, excellent… but fly out of proportion
- All doctors are narcissists (inflated sense of self-importance)… but those who are more narcissistic than you are a problem
- Prevention -> Code of conduct -> Create organisational ethos
- Treatment -> Reports committee -> Investigate -> Meet with doctor -> advise treatment -> Impose sanctions if required -> Legal action if necessary.

EBM – Pro Con Debate – Dean Gopalan (Pro) & David Muckart (Con) (Both UKZN)

Pro:

- EBM stands accused of misleading the medical worlds
- EBM is the integration of best research evidence, clinical expertise and patient values.
- EBM is for everyone – doctors, patients, health authorities, funders, and societal/regulatory bodies.
- Evidence in its broadest sense is a currency by which one fulfils the burden of truth
- All evidence below critically appraised articles is unfiltered
- What is your "personal P value"?
- "Doubt is not a pleasant condition, but certainty is absurd." (Voltaire)
- "More important than the quest for certainty, is the quest for clarity." (Gautier)

Con:

- Phenomenal bias in medical publications
    - Positive results bias – positive trials 3x more likely to be published
    - Obfuscation – detrimental results deliberately supressed
    - Funding bias – 5x more likely to support a drug funded by for-profit organisations
    - Guidelines/consensus frequently supported by industry
    - Academic bias – 60% of medical school chairs receive departmental or personal income from industry
    - Ghost and guest authorship
- Statistical (In)Significance
    - p=0.05 means a 1:20 error, or 5% chance of error.
    - Would we accept 5% failure in the aviation industry?
    - Mathematical significance does not imply clinical significance
- NNT vs. NNH is possibly the only relevant clinical comparison
- Meta-analysis minimises random errors, but does nothing for and all forms of bias
- Ahmed et al BMJ 2012: 29% of metal-analyses did not use unpublished data; 52% did not obtain individual data; 30% reviewer selection bias was a problem.
- "The good physician treats the disease; the great physician treats the patient." (Osler)

The End ☺