SASA 2014

Annual Congress of the South African Society of Anaesthesiologists,

Sun City, March 14-19 2014

Ross's Rough Notes

Please note that these notes are taken during the lectures at high speed and thus are neither complete nor comprehensive. I try to get the gist of the messages, and the bits that I find most valuable. There are almost certainly errors and they should be attributed to me and not the speakers. Please feel free to share the link if you find them useful.

Refresher Course, Day 1

Confined Space Intubation – Ross Hofmeyr, University of Cape Town

- My talk is available online at www.wildmedic.co.za/presentations A review article on the subject is in press, and I'll post the link when it is available.

Evidence-based Anaesthesia – John Carlisle, NHS

- Combining research with clinical experience, in the framework of the patient's context.
- Find it -> Interpret it -> Communicate it
- Asking the question: **PICO - P**opulation, **I**ntervention, **C**omparison, **O**utcome
- You'd need to read 95 papers a day just to keep up with randomised controlled trials published across the medical literature.
- "Best" evidence depends on the type of question
- Systematic reviews are based on a formal system; meta-analysis pools the actual numbers of the individual trials
- Cochrane publish the protocol for the review before it is begun
- Beware confusing relative risk and odds ratios

Cardiopulmonary Interaction – Justiaan Swanevelder, University of Cape Town

- We cannot influence cardiac or respiratory function independently.
- Ventricular interdependence is well describe but underappreciated.
- Pressure-volume loops are an effective way to understand the relationships
- The left side of the heart can tolerate increases in afterload, but the right ventricular fails rapidly if pressures are increased quickly.
- As long as the pulmonary pressures/resistance stays low, the patient can survive well with a single ventricle
- Hypoxic pulmonary vasoconstriction increases pulmonary vascular resistance, but despite the 'wonder drugs' there is little we can do once the right ventricle is failing

- Increased airway pressures and volume (see Pinsky et al Am Rev Resp Dis 1992;146:681-7):
    - Decreased preload on left and right sides
    - Increased afterload on RV; decreased compliance on LV
    - Reduced RV contractility; variable effects on RV
    - Compression of heart in the cardiac fossa
- PEEP has variable effects
- Numerous clinical examples with echocardiography provided

Anaesthesia for Space Travellers – Johan van der Walt, University of Cape Town

- Brief review of the history of the space race – the governments of the world are taking a back seat, and private enterprise is now running in the lead
- Virgin Galactic plans to have paid low earth orbit flights departing soon
- We are inevitably going to see an increase in space travellers needing anaesthesia, either on their return, or later in orbit
- Risks to our physiology are still somewhat unknown and exploration is continuous
- Greatest risk to astronauts on extended missions is still trauma
- Zero gravity makes out endothelium more vulnerable to stress.
    - ANP, NO and Mg are decreased, with impaired angioneogenesis.
    - Decreased diurnal blood pressure variation with progressive hypertension
    - Mg physiology impaired, influencing >300 enzymatic processes
    - Decreased EPO levels leading to anaemia
- Autonomic dysfunction with orthostatic hypotension
    - Cephalad fluid shift with subsequent volume constriction
    - Sydrome of inadequate sympathetic response
    - Alteration in alpha-receptor distribution -> down-regulated in space
    - Baroreceptor reflexes which are altered/decreased
    - Neuraxial anaesthesia could be a problem just after return
- Cardiac atrophy caused by microgravity environments
- Decrease in CVP due to cephalad shift of fluid, causing diuresis
- Plasma volume reduction = Class 1 ATLS haemorrhage before any bleeding has even occurred!
- Potential airway and ventilation difficulties
- Increased half-lives and bioavailability of drugs due to physiological changes
- Many potential causes of trauma in space…
- Fluids in bags and vials will separate and form foam
- Could suspended animation be the future paradigm in space (and on Earth)?

Myocardial Injury after Noncardiac Surgery - Bruce Biccard

- Landmark paper published recently – Anaesthesiology 2014;120(3)564-578 (Paper seems to be free access at the moment)
- 85% of patients suffering perioperative cardiac injury are asymptomatic (and thus missed)
- VISION study undergo – 40000 patients >45yrs requiring non-cardiac surgery with a night in hospital. Sufficient power reached after 15000 patients!

- This constitutes a 'new' diagnosis – MINS (Myocardial Injury after Noncardiac Surgery). Involves cardiac myocardial ischaemic injury, but not necessarily infarction.
- Outcomes are the same for patients with a peak TnT >= 0.03, regardless of the presence or absence of symptoms (remember 85% asymptomatic).
- Myocardial morbidity is high – predicted 30-day cardiac mortality of 10%; composite mortality of ~20%
- So, should we monitor postoperative troponins?
    - Missed diagnosis (Asymptomatic)
    - 30-day risk of mortality
    - 3rd universal definition of MI
    - Potential for simple therapy
- Surveillance monitoring may prove cost-effective
- VISION signal has been shown in a number of prospective cohorts
- Rx includes statin, aspirin, ECG

Thermoregulation in Anaesthesia – Dan Sessler, Dept Outcomes Research, Cleveland Clinic

- Body temperature is tightly regulated within a tight range
- Three important regulatory responses are sweating, vasoconstriction or vasodilation and shivering
- The shivering threshold is a full degree below vasoconstriction
- Anaesthesia impairs thermoregulation, delaying response temperature changes. Decrease is non-linear with increasing concentration of IV anaesthetics and linear with volatiles.
- Temperature drop under anaesthesia is well documented and consists of three phases:
    - Initial rapid core drop (1-1.5°C) – internal core to peripheral redistribution (loss of vasoconstriction causes movement of heat from the core to the usually cooler periphery)
    - Slower linear decrease – heat loss to the environment exceeds production
    - Plateaux – patient becomes sufficiently cold (~34C) to vasoconstrict again
- Neuraxial anaesthesia causes…
    - A central inhibition of thermoregulatory control (much like general anaesthesia). Magnitude is smaller, but cause is unknown.
    - Peripheral loss of regulatory mechanisms (due to neural control of sweating, vasoconstriction and shivering)
- Monitoring sites for accurate core temperature: Nasopharynx, oesophagus, tympanic thermocouple. No good: Anything infrared (too inaccurate); rectal (too slow).
- Consequences of hypothermia:
    - Each degree of hypothermia increases blood loss by 20%
    - Increased surgical site infection
    - Prolonged hospitalisation
    - Decreased drug metabolism
    - Enzyme dysfunction
    - Patient discomfort
- How do you keep people warm?
    - Thermal insulators (plastic sheets, surgical drapes, paper towels) – a single layer makes a 30% difference (trapping an insulating air layer against the patient)

- More layers does not work better
- Forced-air warmers work, work well, and are safe
- Warming fluids doesn't help to warm patients much, but it does definitely prevent heat loss. One unit of cold blood or one litre of room-temperature fluid cool the core by 0.25°C

Never trust a drug pronounced three different ways, or: Drug Errors in Anaesthesia - Rob Raw, Iowa

- Discovered vs. undiscovered
- Wrong drug, wrong dose, wrong label, wrong use, wrong soluent, wrong patient, wrong time, wrong...
- Many stories shared…all true and all scary ☹
- Communication: Verbally verify the action – Speak the drug
- RTARTS – Read The Ampoule, Read The Syringe
- Save the ampoules until the end
- We must collect data on drug errors – and this information must be protected/indemnified to protect the process from legal attacks.
- Drop-down lists are a frequent source of error on electronic administration/charting systems
- Regional anaesthesia additive errors are very serious but quite uncommon
- See: Cooper 2013 in Anaesthesia Clinics
- Also see: Llewellyn 2009 AIC (SA article about interns)

Paediatric Anaesthetic Emergencies – Jenny Thomas, Red Cross War Memorial Children's Hospital, Cape Town

- Emergency = serious, unexpected and often dangerous situation requiring immediate action
- Errors and emergencies happen to everyone, and do not imply a fault
- Crisis in practice: relying on intuition without knowledge leads to poorer outcomes. Learned and practiced algorithms produce a standardised response when a catastrophe occurs.
- "To err is human, to forgive, divine"
- Recognise as early as possible. Listen to your suspicions.
- Reduce distractions
- Adverse events should all be reported and recorded for learning purposes – 'free lessons'
- SAJAA still publishes case reports – use this forum!
- At RXH, the burns theatre has the highest incidence of anaesthesia-related critical incidents
- Numerous cases presented.
- Severe hyperkalaemia is a consequence of both malignant hyperthermia and anaesthesia-induced rhabdomyolysis. Resuscitation may be prolonged and may require bypass/ECMO to maintain circulation while potassium is being removed.
- Ped Anaes Sept 2013 – entire issue on paediatric muscle disorders
- RXH perioperative workup protocol for muscle biopsy is working well and is available for dissemination/use at other centres.

Depth of Anaesthesia and Awareness – Ellen O'Sullivan, Dublin

- Patients who have had awareness during surgery can suffer permanent psychological sequelae
- Anaesthesia is a continuum with no limits. Levels have been defined in various publications
- Approximate incidence 0.15% (1% in high-risk patients) according to the Bryce questionnaire
- NAP5 – Accidental awareness. NAP5 Baseline incidence ~1:15 000
- Are depth of anaesthesia monitors a waste of time?
    - How big is the problem anyway?
    - You've always managed without them
    - They are expensive
    - The evidence is not very strong
- Various monitors based on processed EEG. Many passive, some active (AEPs)
- Linearised (100->0) and montonic (as the patient gets deeper, the number gets smaller)
- Technonsense – we don't know how it works (eg .BIS monitor)
- BIS scale: awake (90), moderate sedation (70), general anaesthesia (50), deep anaesthesia (30), isoelectric (10)
- Isolated forearm technique:
    - Invented by Mike Tunstall in Aberdeen and further developed in Hull by Ian Russell
    - Cuff on opposite arm to IV inflated before muscle relaxant given
    - Small but evangelical following
- What is the relationship between unresponsiveness, unconsciousness and connectedness?
- B-Aware (Lancet 2004): 80% reduction in awareness (2 vs 11 cases if awareness out of 2465 patients total)
- B-Unaware (NEJM 2008): BIS vs ETAG – 0.21% awareness (2 out of nearly 1000 total) in each arm
- BAG-RECALL (NEJM 2011): ETAG outperformed BIS in preventing awareness (N~5500)
- Deep anaesthetic state (<45%) has a poorer outcome (Monk A&A 2005) – is a low BIS a sign of poor protoplasm, or is our management making patients sicker?
- Sessler – Beware the "triple low" (low BP, low MAC, and low BIS)
- The cost of a BIS electrode will buy a lot of isoflurane
- Many things make you look anaesthetised when you are not… hypothermia, abnormal EEGs, loss of contact, decreased muscular activity (muscle relaxants!) etc
- Some anaesthetic drugs don't affect BIS (Xenon, ketamine, remifentanil…)
- Future: Electroecephalone? (EEG converted to sounds)
- NAP5 will bring a lot more answers

Time to Face the Book: Unfriending IV Fluids – Zane Farina, Pietermaritzburg

- Paracelsus: All things are poison, and nothing is without poison; only the dose permits what is poisonous.
- Fluids are a drug, and the dose must be specific. Different patients have different 'compliance' to cope with relative over- or underhydration
- What should be in our fluids?
    - Crystalloid vs colloid
    - Electrolyte composition
    - Osmolality

    - Charge buffer
- When and how do we decide to give fluids? What are our end-points?
- Good friend: Woodcock & Woodcock glycocalyx model
- NEJM 2013 Myburg and Mythen – Resuscitation fluids
- Glycocalyx model is a potential surrogate endpoint for research into mechanisms of damage and options for therapy
- Starch data is confused; lumping and splitting the studies to show absence of benefit. Retracted data is not always excluded (eg. Joachim Bolt)
- 3:1 ratio is definitely out. 1.5:1 is more likely to be beneficial.
- Starches will be back, likely with much stronger package limits.
- What does FEAST tell us?
    - Bolus groups (saline and albumin) had 10% mortality, no bolus group only 7%
    - Fluid bolus did not help these children!
    - Issues with study design
    - Large bolus (40ml/kg) increased later in the trial!
    - Lots of anaemia amongst the children in the study
    - Use syringe drivers for bolus resuscitation of children
    - Use more agressive endpoints for resuscitation – peripheral perfusion, lactate clearance
- Chloride is not a benign anion for electrical neutrality (Yunos in JAMA)

Vasoactive Agents: Synergy in Shock Management – Richard von Rahden

- Shock is a state of deficit in oxygen delivery vs oxygen consumption
- The CVS is a system of pipes, pumps and fluid
- Flow is a lot of fluid moving; pressure is the driving force moving it.
- Flow in the human is cardiac output; pressure is MAP
- MAP = Q*SVR
- In terms of delivery of O2, both flow and pressure are required
- Must deliver CI >2.2 l/min/m2 and MAP >65
- Pump fail – intrope
- Vasodilation – vasopressor
- Hypovolaemia – fluid!
- Just enough fluid, vasoconstrictor and inotrope…Goldilocks!
- Divergent abnormalities mask each other
- Significant interactions, competitive and synergistic actions of different vasoactive drugs
- Inotropes:
    - Use for pump failure (isolated or as part of a mixed shock state)
    - Dobutamine: beta-1 and -2, good inotrope but also vasodilator (inodilator), so good for isolated failure. Disasterous in cases of fluid depletion and distributive shock (eg. hypovolaemic shock or sepsis) "Dobutamine is Deadly in Dilation"
- Vasopressor
    - Alpha-1
    - Phenylephrine, ornipressin, etc
    - Beware overconstriction causing raised afterload and decreased cardiac output/flow
    - "Phenyl Finishes off Failing Hearts"

    - Acceptable use – vasodilation from regional anaesthesia
    - Potentially deadly – use where myocardium is depressed
- Mixed agents:
    - Dopamine, Noradrenaline, Adrenaline
    - All beta effects at lower doses, alpha effects at higher doses
    - Noradrenaline touted as best for "warm shock"…but we don't have it in SA
    - Dopamine – renal dose is an outdated concept and has been disproved. Causes release of endogenous noradrenaline, which is a problem once supplies are depleted. Several other issues – suppressed TSH, prolactin, immunity, causes nausea
    - Adrenaline – we have it, it is cheap, it definitely works, it is tiratable. Cons: too much inotropy, too little vasopressor effect, hyperglycaemia, hyperlactataemia. May cause myocardial ischaemia due to tachycardia and increased metabolic demands.
    - Make sure you are fluid replete when using lots of inotropy
- (Comments and discussion after the lecture regarding the use of phosphodiesterase inhibitors, especially milrinone. Most in SA don't have access; generally limited to special permission in dedicated cardiothoracic surgery units)

Effective Pain Relief in Children – Johan Diedericks, University of the Orange Free State

- Pain management is required not just for humanitarian reasons, but also to decrease sympathetic response, postoperative complications, and lifelong pain responses and endorphin tone.
- Children (and neonates) definitely feel real pain
- Clinicians tend to respond to 'objective' rather than the patient's reported pain.
- Nursing decision-making is essential to effective pain management on the wards postoperatively.
- Van Hulle Vincent et al (Stellenbosch) – Online training effective for nurses, and resulted in better pain scores in paediatric patients after the intervention.
- 0-1yrs: Little emotional impact; pain perception worse?
- 1-5yrs: Emotional attachment to primary caregiver; pain worse or overridden by fear
- 5-10yrs: Abstract fear of mutilation or death; understanding may alleviate pain
- >10yrs: Fear of losing control; may hide pain
- Girls react more with fear and anxiety; boys react more with anger
- Various rating scales discussed (eg. VAS, Faces, CHEOPS, FLACC, OPS)
- Multiple non-pharmacological modalities (play, music therapy including music entrainment, clowns, distraction with various means, etc)
- Adjuvants to our normal analgesics: Corticosteroids, topical/local/regional anaesthesia, neuroleptics, benzodiazepines (maybe for muscle spasm)
- New meta-analysis questions the use of ketamine in post-op pain, but it is accepted and commonly used in some centres (eg. Red Cross)
- Extensive use of opiates, including PCA
- Overall approach:
    - Comprehensive, multimodal approach
    - Put children in control (give info, use PCA)

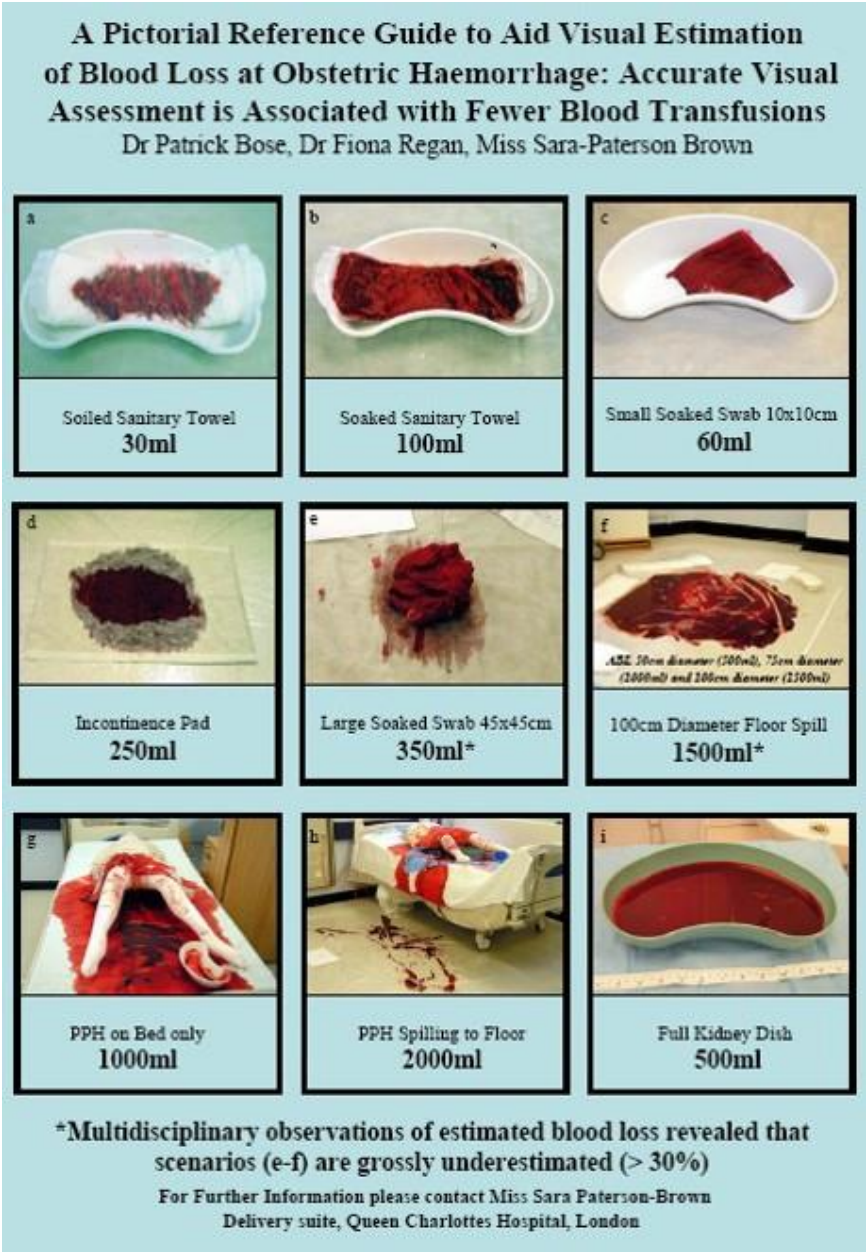
    - Interdisciplinary team
    - WHO analgesic ladder – useful for creating protocols
    - Specific surgical scenarios
    - Administration techniques
- Neonatal procedural pain: make use of breast feeding, suckling, sucrose. Not a lot of evidence for topical local anaesthetics in this age group.
- EMLA and paracetamol premed should be liberally used in paediatric patients
- Extensive use of regional anaesthesia/analgesia wherever possible

The Surviving Sepsis Guidelines Campaign: Where are we now? - Dean Gopalan, University of KwaZulu Natal

- Sepsis remains a major, costly, deadly problem worldwide
- Original guidelines 2004: only 5 of 52 recommendations made on the basis of strong evidence
- Revision in 2008: "no industry involvement". 72 recommendations made; 8 on the basis of strong recommendation. Took out low-dose dopamine and activate protein C. Added mechanical prophylaxis for DVT
- Revision in 2012: 85 recommendations, 10 ungraded. Only 6 strong evidence: No low-dose dopamine; low tidal volume ventilation, weaning protocol; DVT prophylaxis; ulcer prophylaxis
- GRADE system (quality and level of evidence) used
- Not going to go through the individual recommendations
- Protocol-driven resuscitation targeting CVP, MAP, urine output and CVSpO2
- Early antibiotic administration and source control are key facets.
- Implementation of 'bundles' is the current paradigm; several studies have showed a mortality benefit in the same period, but it seems to be working.
- "Opinion-based vs evidence-based…should be discarded"?
- Must be applied as suitable to the local setting

New trends in the management of postpartum haemorrhage during Caesarean section – Robert Dyer, University of Cape Town

- UCT Anaesthesia covers 3 obstetric units with ~35 Caesarean sections per day
- Maternal death is still due to haemorrhage in 25% of cases (2008-2010 confidential enquiry data)
- How can anaesthetists save lives?
    - Assess blood loss
    - Modify practice – anticipate, refer, coordinate resuscitation
    - Monitor haemoglobin and coagulation
    - Transfuse according to a protocol
- Massive haemorrhage = 1 blood volume or 10u PRBCs within 24 hours
- Rule of 30's: heart rate increase by 30/min, respiratory rate >30/min, 30% blood loss, etc
- Visual estimation of blood loss is usually too little – periodic estimates are more effective than a single estimate



- Abruption, praevia, multiple pregnancy and preeclampsia all increase the risk of haemorrhage dramatically (5-13x). Even emergency C/S has a 4-fold increase.
- Less blood loss has been shown with regional anaesthesia for C/S in placenta praevia, but cases must be carefully selected.
- Increased placenta accrete rate is linked to increased use of C/S
- Rapid and adequate use of blood coagulation products (eg. fibrinogen, platelets, etc)
- We should see the coagulation system as a whole, recognising initiation and amplification
- Fibrinogen is the major contributor in obstetric coagulation, and is the best marker for developing a coagulation abnormality in PPH. However, turnaround time on fibrinogen levels is long, and baseline values vary dramatically.
- More emphasis on point-of-care coagulation testing (eg. ROTEM, TEG). ROTEM perhaps more suited due to additional analysis (INTEM, APTEM, EXTEM, FIBTEM). All have demonstrated hypercoagulability of pregnancy.

- Non-invasive measurement of Hb may be useful, but concerns for the accuracy remain
- Haemoccue measurements remain reliable and should be used for PoC testing
- Massive transfusion protocols:
    - Anticipate uncontrolled haemorrhage
    - Anticipate more than 10u PRBCs
    - 6:4:6 PRBC:FFP:Platelets is current recommendation in obstetric haemorrhage
    - Consider fibrinogen strongly – fibrinogen levels are increased in early pregnancy, but decrease due to fibrinolysis later in pregnancy. Fibrinogen <2g/L has a 100% positive predictive value for PPH.
    - Early measurement in haemorrhage -> if low, supplement early.
    - FFP has approximately 400mg fibrinogen in 200-250ml; cryoprecipitate has 2500mg in 150ml!
    - 3ml/kg cryoprecipitate should increase fibrinogen levels by 1g/L
    - FFP can actually decrease your levels!
    - FIB-PPH trial underway at the moment
- Antifibrinolytics: WHO recommendation since 2009 despite lack of RCT evidence
- WOMAN trial in progress (1g TXA given if >500ml bleeding in vaginal or >1000ml in C/S delivery)
- rFVIIa does not have evidence and is not in use in most centres. See Curr Opin Anest 2012;25:309-314
- Cell salvage – theoretical problems have not yet materialised in clinical practice, but not being used widely (or at GSH/UCT)