

SASA 2014

Annual Congress of the South African Society of Anaesthesiologists,

Sun City, March 14-19 2014

Ross's Rough Notes

This set of notes is incomplete, published as an interim collection before the end of the congress.

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 – Saturday 15 March 2014

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** - Population, Intervention, Comparison, Outcome
- 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 angiogenesis.
 - 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
 - Syndrome 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 Bickard

- 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 solvent, 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 monotonic (as the patient gets deeper, the number gets smaller)
- Technosense – 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, remifentanyl...)
- Future: Electroencephalogram? (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 aggressive 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
- $MAP = Q \cdot SVR$
- 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 – inotrope
- 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 Dilution”
- 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

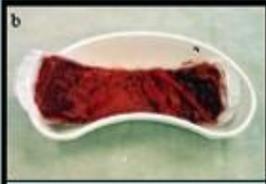
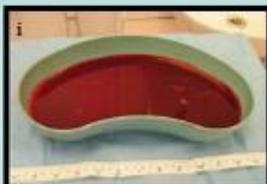
- Sepsis remains a major, costly, deadly problem worldwide
- Original guidelines 2004: only 5 of 52 recommendations made on the basis of strong evidence
- Revision in 2008: “no industry involvement”. 72 recommendations made; 8 on the basis of strong recommendation. Took out low-dose dopamine and activate 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

A Pictorial Reference Guide to Aid Visual Estimation of Blood Loss at Obstetric Haemorrhage: Accurate Visual Assessment is Associated with Fewer Blood Transfusions
Dr Patrick Bose, Dr Fiona Regan, Miss Sara-Paterson Brown

		
Soiled Sanitary Towel 30ml	Soaked Sanitary Towel 100ml	Small Soaked Swab 10x10cm 60ml
		
Incontinence Pad 250ml	Large Soaked Swab 45x45cm 350ml*	100cm Diameter Floor Spill 1500ml*
		
PPH on Bed only 1000ml	PPH Spilling to Floor 2000ml	Full Kidney Dish 500ml

***Multidisciplinary observations of estimated blood loss revealed that scenarios (e-f) are grossly underestimated (> 30%)**
For Further Information please contact Miss Sara Paterson-Brown
Delivery suite, Queen Charlottes Hospital, London

- 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
- Haemocue 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)

Refresher Course, Day 2 – Sunday 16 March 2014

Phaeochromocytoma: An Update – Andrew Levine, Stellenbosch University

- 50% present with CAD (accelerated?), 50% without
- Symmetric LVH very common. Protective – normalises wall tension. Consequence: increased backward pressure with PHPT and pulmonary oedema
- Takotsubo-like cardiomyopathy. ST and enzyme changes; ballooning on angio
- Can also cause a dilated cardiomyopathy or catecholamine cardiotoxicity
- Inotrope responsiveness in catecholamine cardiotoxicity is greatly reduced. ECMO has been used to salvage these cases in case reports, as they seem to be reversible with time.
- Phae can present with a septic/cardiogenic shock type picture, or with signs of catecholamine excess.
- Vasculature in these patients is similar to that in patients with longstanding hypertension, with thickened media. They behave like hypertensives in response to stimuli and anaesthetics
- Due to 'hypertensive' vasculature, they have disturbed autoregulation. Blood pressures must be maintained at adequate levels.

- Preparation: see Nat Rev Endocrinol 2010 - Alpha-1 selective blockade essential. Prazocin/doxazosin may be preferable to phenoxybenzamine
- Ca⁺⁺ channel blockers touted in the literature (nicardipine) but if magnesium is used intraoperatively this is a very dubious practice
- Mg⁺⁺ infusions preoperatively have been used (ref: Mike James)
- Beta-blockers should only be instituted after adequate alpha blockade
- Ready for theatre: Roizen's criteria (No BP <160/90 for 24hrs, etc)
- Drugs to avoid: sympathomimetics, antidepressants, myocardial sensitizers (halo/enflurane), anticholinergics, steroids, histamine release, suxamethonium, contras (non-ionic is safe), epidural local anaesthesia, dopaminergic antagonists
- Potential crisis periods: induction, intubation, incision, dissection, IVC compression, blood loss, "we've got the main vein", "it's already out"
- Intraop haemodynamic management: Magnesium is the mainstay of management. Cheap, available, predictable, wide therapeutic index, measurable, reversible with calcium, renal excretion rapid. Beware if the patient is on a calcium channel blocker! Can potentiate neuromuscular blockade. Vasopressin analogues can be used to control hypotension.
- When the tumour is really out, the magnesium is antagonised with calcium. Preload is important due to rapid expansion of the vascular space.
- Haemodynamic monitors – PAC or TOE

Delirium after Anaesthesia – David Duthie, Leeds

- Consciousness of self and environment; disturbance in arousal, wakefulness and cognition
- NICE July 2010: www.nice.org.uk/guidance/CG103/guidance :
 - Disturbed consciousness, cognitive function or perception
 - Acute onset (1-2 days), fluctuating course
 - Hyperactive/hypoactive/mixed
 - Serious, poor outcomes
 - May be prevented and treated if dealt with urgently
- www.icudelirium.co.uk is a useful resource
- Prolonged delirium (>3 months) is a predictive factor for PTSD, dementia and death
- Motoric subtypes: hyperactive, hyperaroused, agitated, hypoactive, hypoalert, lethargic, mixed or alternating. EEG useless. Can use Richmond Agitation-Sedation scale to classify
- CAM-ICU diagnostic tool www.youtube.com/watch?v=6WyJ0zL7Vkl
- Aetiology: None proven. Decreased oxidative metabolism affecting neurotransmission due to inflammation and chronic stress implicated. Neuronal aging predisposes to delirium (hence more common in the elderly)
- Management: see NICE guidelines (link above)
 - Treat cause
 - Communicate and re-orientate
 - De-escalate distress and violence
 - Consider short-term haloperidol or olanzapine
 - Sort out basic care issues – oxygenation, pain control, prevent constipation, sleep hygiene, etc.
 - Benzodiazepines more likely to induce delirium than treat it

- Alpha-2 agonists are the most recent additions to the regime (clonidine, dexmedetomidine)
- Haloperidol 1-5mg as a single dose after checking ECG for QT prolongation; repeat after 30 minutes if necessary. Consider fentanyl/propofol/alpha-2 if unsuccessful
- Statins unconvincing.
- Dexmedetomidine did not decrease incidence in some studies, but duration of delirium and ventilation reduced. Other studies did show decreased incidence but may be too good to be true.

What Becomes of a Broken Heart: Perioperative Cardiac Failure - Eric Hodgson, University of KwaZulu Natal

- Adverse cardiac remodelling – stroke volume is reduced in both dilated and hypertrophic cardiomyopathy.
- See new cardiac failure guidelines – link in SAJAA article published in congress edition
- Perioperative assessment of systolic CCF: Presence of creps in the lungs implies shunt and therefore hypoxia. Ask about METs and examine the chest. Measurement of BNP (>87.5pg/ml) and 6 minute walk test are useful.
- Minimise catecholamine use in cardiac failure – increased dose and duration are associated with worse outcomes.
- Effective therapies for systolic cardiac failure: cardiac resynchronisation with triple-chamber pacing, IABP for bridge, LVAD (beware: they will be anti-coagulated!), transplant.
- Diastolic heart failure: decreased LV cavity size, increased myocyte size, impaired filling with reduced stroke volume but normal ejection fraction.
 - Reversible - must decongest to improve function
 - Lusitropic (relaxation) action is reduced
 - Recognisable on mitral inflow Doppler flow velocity profile (E:A wave relationship)
 - Fixed cardiac output state.
 - Any disturbance (eg. dysrhythmia) can precipitate flash pulmonary oedema
 - Easily palpable apex with heaving.
 - ECG: Qs from previous MI; strain pattern
 - BNP's elevated
 - Dobutamine stress echo useful if 6MWT inconclusive
 - Rx: decongest using B-blocker and nitrate then ACEI/ARB. Diuretic is 3rd line – very limited role in perioperative period. Patients are crucially dependent of preload.
 - Delay surgery wherever possible to improve function with medical therapy (especially if you need to start Beta-blockers – give 2-4 weeks minimum)
 - Intraop opioids and/or labetalol
- BNP levels pre-discharge predict readmission and mortality
- Slides are at www.tinyurl.com/SASAHF14

Peri-operative Ultrasound – Marta Kot

- Previously we had sonographer -> reporter -> decision maker; now we have sonographer = reporter = decision maker
- Focussed assessment with ultrasound is part of the clinical examination in the modern world

- Portable, non-invasive, can be used frequently, useful for guidance for high-risk interventions (lines, blocks, centesis, etc)
- Can be used as part of our ABCs (airway, breathing/lung/chest, cardiac, ocular/neuro, etc.)
- eFAST: Hepatorenal, subcostal, splenorenal, pelvic, etc.
- Lung ultrasound being incorporated into multiple protocols. Artefacts give diagnostic clues!
- Winfocus group: Ultrasound ABCDE - <http://trauma.winfocus.org>
- “Echodynamics” A-PLUS (Airway-Pleura-Lung UltraSound). Determines the cause in shock and/or hypoxia
 - Pericardial effusion - ?tamponade
 - Preload – source of bleeding
 - Contractility - ?LV ?RV ?interaction
 - Valve function
 - Pleura – ?pneumo ?haemo ?effusion
 - Lung - ?consolidation ?oedema
 - Intubation - ?difficult airway ?position of tube
- Various excellent examples given
- Presence of lung sliding excludes pneumothorax with 100% negative predictive value!

Perioperative Wound Infection Prevention – Dan Sessler, Cleveland Clinic

- Surgical site infections are common, costly, increase length of stay and double mortality
- All wounds become contaminated
- Infections are established within 2 hrs of contamination. Host defence and prophylactic antibiotics make the difference. ABx should be given one hour before incision; single dose is okay unless op is very long. Should never be continued longer than 24hrs. Our mission is to make sure they get given in time.
- Oxygen:
 - Host defence is the other big factor – oxidative killing by neutrophils. Requires molecular oxygen contained in lysosomes (‘good’ free radicals). Amount is determined by tissue partial pressure. Oxygen also promotes angiogenesis and healthy scar formation.
 - Hopf et al 1996 show low tissue oxygen leads to more infections than expected, and visa versa. Supplemental oxygen is easy to provide by the concentrations provided is somewhat random (30% in Europe, 40% in SA, seemingly random in US)
 - No important difference in atelectasis between 30 and 80% O₂
 - Supplemental oxygen doubles tissue oxygen
 - Early studies showed that supplemental O₂ reduced infections...but later studies (PROXI, Meyhoff et al, Lancet 2009 and Akca, unpublished) refuted this. Current trial underway (Kurz et al)
- Hypothermia:
 - Definitely leads to more wound infections
 - Reduced host defence
- Transfusion:
 - Definitely increase the risk of infection
- Smoking:

- Reduces tissue oxygenation, within the clinically significant effect. One cigarette decreases tissue oxygenation by about 20mmHg for an hour. This is only found if tissue oxygenation is measured directly
- Increase risk of infection in smokers
- Smoking cessation decreases mortality
- Hyperglycaemia:
 - Tight glucose control improves immunity (multiple studies – Gallacher, Athos, Van Den Bergh)
 - Tight glucose control showed no difference in infections! (Abdelmalak, BJA 2013)
- Fluid management:
 - High vs. moderate fluid volume replacement increased tissue oxygenation, but there was no significant difference in wound infection in a small-ish study (N=250)
 - Doppler-guided fluid management show reduce composite complications, but not a reduction in wound infection risk
- RECOMMENDATIONS:
 - Timely antibiotics
 - Maintain normothermia
 - Reduce transfusions where possible.
 - Stop smoking (surgery is a ‘teachable moment’)
 - Euglycaemia
 - Guided fluid management

Blood is thicker than water: Perioperative coagulation abnormalities – Palesa Motshabi Chakane

- Coagulation relies on coagulation proteins, platelets and the endothelium
- The endothelium is an immense ‘organ’ which plays a pivotal role in coagulation
- Recent publication in NEJM highlights the role of the red cells (Hunt BJ, Bleeding and Coagulopathies in Critical Care, NEJM). However, isolated reduction in haematocrit has not been shown to compromise *in vitro* coagulation (see Iselin et al, BJA 2001; 87(2))
- Strong correlation with low haemoglobin, platelet count and fibrinogen with bleeding... no duh?
- PoC devices are leading the role in perioperative coagulation testing (TEG, ROTEM)
- Multiplate TEG uses ADP or arachidonic acid (AA) for platelet mapping to measure platelet aggregation in antiplatelet therapy
- ROTEM can also do INTEM, EXTEM, FIBTEM, ABTEM, HEPTM and EXTEM-A to look at specific components.
- INTEM emulates the contact phase of haemostasis, analysing the ‘traditional’ intrinsic pathway. Influenced by coag factors, platelets, fibrinogen and heparin. Guides administration of FFP, coag factors, fibrinogen or platelets
- HEPTM is an INTEM in the presence of heparinise. In comparison with INTEM, this allows the effects of heparin to be seen and controlled for.
- EXTEM activates haemostasis using tissue factor. This looks at the ‘traditional’ extrinsic pathway, looking at coag factoes, platelets and fibrinogen. NOT influenced by heparin. Forms the baseline for FIBTEM and APTM
- FIBTEM is EXTEM-based withch uses cytochalasin-D to eliminate the platelet contribution to clotting, allowing examination of the fibrin function. This detects fibrin deficiency, allowing replacement of fibrinogen.

- APTM is EXTEM-based assay for fibrinolysis
- Fibrinogen concentrate can be kept in theatre and given to correct fibrin/fibrinogen deficiency
- Prothrombin complex concentrates can also be used to replace factors II, VII, IX and X
- Anticoagulants in the perioperative period:
 - Warfarin is stopped 7 days preop and recommenced postop. Must be covered with heparin in the interim if this is done. Another option in an emergency is to reverse it with 10-30ml/kg FFP. Prothrombin complex can be given at 25u/kg and guided by PoC testing
 - Unfractionated heparin is commonly used. Half-life of 40min. Easily reversible. Can be monitored with HEPTM or ACT if necessary.
 - LMWH – can use HEPTM to monitor
 - Antoplatelet drugs: Clopidogrel is the most common. Should be stopped 5-7 days in advance, with bridging using glycoprotein IIb/IIIa agents. Can monitor with EXTEM-A/Multiplate/VerifyNow to map platelet activity in emergencies or urgency
 - Xa inhibitors (eg. rivoraxaban) have half-lives ranging from 7-14 hours. Xa assays take time but can be used to guide reversal with PCCs
 - Thrombin inhibitors (eg. Dabigatran): Can use thrombin time to monitor. Can be dialyzed out in dire emergencies (yikes!)
- Pro-coagulant drugs:
 - Tranexamic Acid and Epsilon-amino-caproic Acid – CRASH2 trial gives us some confidence. ATACAS shows that there is not the expected increase in thrombotic events.
 - DDAVP – stimulates release of vWF and FVIII. Useful in haemophilia, maybe in other bleeding. Has issues and significant side-effects.
 - Protamine – used to reverse heparin. Too much increases bleeding.
 - Aprotinin – out of favour currently due to high risk of thrombotic events
 - Tissue glues – can be used by surgeons (various agents)
- Massive transfusion protocols
- ROTEM in massive haemorrhage:
 - APTM to measure hyperfibrinolysis
 - EXTEM-A to measure thrombocytopenia
 - FIBTEM to measure fibrin levels
- Summary: 5P approach: Prolene, Protamine, Prothrombin, Patience, Prayer

Extubation: Old problem, new solutions – Ellen O’Sullivan, Dublin

- Intubation is one of the most important anaesthetic skills...but what about extubation?
- BJA 1998 Asai: Resp complication incidence at intubation 4%, at extubation 8%, in recovery 4% -> 12% happen when/after the tube comes out!
- NAP4 has 9 recommendations on extubation. 28% of events occurred during emergence and recovery. Majority related to airway obstruction. Contributory factors: lack of equipment, planning, judgement, training in extubation.
- Extubation planning:
 - See DAS Extubation guidelines (2004, currently being revised)
 - Published in Anaesthesia in 2012. (6215 articles considered!)
 - They come with flow charts (Thank heavens!)

- Available here: <http://www.das.uk.com/content/das-extubation-guidelines>
- Airway risk factors:
 - Was the airway normal at induction, and has it remained so?
 - Is there change due to surgery?
 - Is there aspiration risk?
- Discussion of algorithms ensued
- Advanced techniques:
 - Awake remifentanyl technique – profound analgesia and suppression of airway reflexes. Remi used throughout case and then continued at 8-12ng/ml until patient is awake, breathing and tube out.
 - Bailey manoeuvre – ETT replaced with LMA while asleep. LMA insertion before ETT removed to help keep position of the epiglottis. Useful for thyroid surgery, where FOB can then be used to inspect the vocal cords before awakening.
 - Airway exchange catheter technique – CardioMed ETVC, Sheridan and Cook. See research by Cooper (CJA 1996), Loudermilk (Chest 2007) and Mort (A&A 2007;105). Good practical points. Know your kit!
 - Soon to come: Cook Extubation Set.
- Appropriate location for patients with risky extubation (ICU/HCU/PAHCU). Capnography should be available. Full difficult airway equipment in recovery.
- NAP4 recommendations also include guidelines for post-op pulmonary oedema and laryngospasm

The Future and Safety of Ambulatory Anaesthesia – Peter Glass, New York

- History of ambulatory surgery briefly described
- Drivers for growth:
 - Economics
 - Patient preference
 - Technology and anaesthesia advances
 - Safety/Quality
- Barriers:
 - Availability of in-patient beds
 - Economies of scale
 - Job loss at hospitals
 - Inconvenience to surgeons
 - Not all patients prefer ambulatory surgery
 - Tight regulatory controls
- Present state of ambulatory surgery:
 - Much occurs on a day-case basis in the main hospital OR
 - Free-standing ambulatory centres
 - Office-based procedures
 - Surgical technologies using minimally-invasive methods have greatly increased the range of surgery which can be done on a same-day basis (total hips as day cases !?!)
 - Outcomes are remarkably good (only 1-1.5% require admission after day-case surgery). Major morbidity rare. This is due to careful patient selection.
 - Emphasis has moved to prevention and treatment of more common but less severe problems, like PONV, pain, prolonged sedation and lethargy.
- Patient satisfaction:

- Preoperative visit with testing, education, online and DVD materials
- Preoperative call and insurance issues sorted out
- “Meeter Greeter” on arrival to smooth over practicalities.
- Private rooms, nice gowns, dignity and support
- Get well card signed by everyone involved
- All post-op info provided in printed form to take home
- Recovery follow-up
- Future:
 - Anaesthesia service providers – lots of nurse anaesthetists in the USA, including ‘sedation nurses’, and SedaSys machines...
 - Preoperative evaluation – use of electronic medical records, telemedicine, and smart-phone based technologies
 - New minimally-invasive surgical approaches. Hospital -> day case surgery -> office-based surgery. Catheter development, robotics, etc.
 - Office-based surgery: Poorly regulated area at the moment. Lowest overhead costs. Surgeons freed from credentialing issues...but more regulation likely to come soon.
 - Non-surgeons doing procedures with non-anaesthetists providing anaesthesia...
- New drugs
 - NMB – Sugammadex
 - Non-opiate analgesics with greater efficiency
 - Ultra-short acting esterase-metabolised benzodiazepine! CNS 7056 (remimazolam) (Ross says: see <http://www.ncbi.nlm.nih.gov/pubmed/17585216>)
- Outcomes research underway (SAMBA Clinical Outcomes Research) in ambulatory anaesthesia. SAMBA = Society for AMBulatory Anaesthesia.

Point-of-Care Testing – Richard von Rahden

- Big disadvantage is cost...but in the ‘big picture’ it is often cheaper
- Glucometers:
 - Hypoglycaemia kills brains
 - Hyperglycaemia damages the glycocalyx, impairs leucocyte function, impairs fibroblast function and causes osmotic diuresis
 - Glucose control targets have changed over time; around 8mmol/l is probably appropriate. Rapid readings are required.
 - PoC devices work rapidly, only require a drop of blood, and incorporate the same measurement methods as lab glucose measurements.
 - HOWEVER, PoC glucose has some potential problems:
 - Glucose changes with site – arterial>capillary>venous. We must be consistent. Capillary sampling requires consistency.
 - [Blood glucose]<[plasma glucose] due to ‘dilution’ caused by red cells. This is corrected for mathematically by the analyser. Beware anaemia and/or high lipid levels, as they can increase the error.
 - PO₂, icodextrin (from dialysis), ascorbic acid and paracetamol can interfere with the various reactions in different ways.
- Haemoglobinometers:
 - Oldest technique = Haematocrit.
 - Measures actual volume, so not always accurate if cells are abnormal.

- Absorption methods (blood gas or Masimo) all subject to interference.
- Haemoccue very good, but can vary up to 1g/dl.
- ACT:
 - Heparin is essential for many CPB, ECMO, CRRT, vascular surgery.
 - ACT has linear relationship, which allows you to work out reversal doses.
 - Numbers from different devices aren't necessarily the same.
- TEG/ROTEM:
 - Discussed elsewhere at length.
 - Dynamic clot strength measurement.
 - Similar; either is effective.
 - Needs extra hands to run, and needs QC process.
- Lactate:
 - Anaerobic metabolism byproduct.
 - Also formed when there is lots of glucose breakdown (eg. Adrenaline infusion).
 - Now part of goal-directed therapy. Type A/B.
 - Problems with site, contamination, comparability of absolute levels.
 - Are trends good enough, or do we need specific levels?
- Cartridge-based diagnostics:
 - Can do full panels on small volumes of blood.
 - Lots of cardiac tests and even hormone levels.
 - Some queries about whether tests from different machines are comparable.
 - Beware rule-in/rule-out thresholds.
- Remember:
 - Understand the strengths and weaknesses of the different devices.
 - Beware of mixing results
 - Beware of published threshold levels
 - Do the technical stuff right (QC/pre-analytical technique)

New Innovations in Interventional Cardiac Procedures – Jeorg Ender, Leipzig

- Transcatheter valve replacement (TAVI/TAVR)
 - Issues with paravalvular aortic regurgitation. This has an impact on mortality.
 - JenaValve now released to prevent this, which fixes the native valve leaflets to the prosthesis.
 - Engager valve also has control arms to capture native valve leaflets. Sapien valve has a skirt to prevent leakage. These new generation valves thus don't need to be oversized, thus reducing complications from oversizing.
- Strokes:
 - Transcatheter valves have higher peri-procedure stroke rates.
 - Claret device has filters which are placed in left carotid and brachicephalic arteries to catch debris shed during balloon valvuloplasty and valve implantation.
- Some discussion of valve-in-valve procedures and 'effective orifice area'
- Aortic valve implantation mainly angio-guided; mitral valve implantation mostly echo-guided.
- Transapical TAVI approach now being used with Sapien AV for mitral valve stenosis.
- Dumbbell technique for echo-guided valve implantation.

- Transcatheter mitral clips (Alfaeiri) from groin through atrial septum! Can only be done with echo guidance. Needs three-plane guidance using 3D echo/x-plane. Atrial septum puncture site also selected using echo.
 - Neochord system for transapical placement of artificial chordae in cases of mitral prolapse. Also echo guided.
 - Field is continuously growing - First human percutaneous mitral anuloplasty with Neochord placement performed in the last month.
 - Indications are still being determined; patient selection is the most critical part of the entire procedure.
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