SASA 2013 – Port Elizabeth

Ross's Rough Notes

NB – This is not yet the complete set. This version is Refresher Day 1 (incomplete) and Day 2, and Congress Day 1 (of 3)

Refresher Day 1 (Saturday)

Session 1 – Cardiovascular

Aging and the cardiovascular system – Prof Patrick Wouters, University of Ghent, Belgium

- Maximum age achieved by a human = 122 years. Human idealised total life span is 120 years. Life expectancy is an empirical estimate and evolves rapidly depending on the living conditions and environment.
- Little consensus on what is considered 'elderly' – previously 65 years, now 85 suggested. Elderly portion of the population is increasing rapidly. This is going to massively impact on health economics.
- The older patient presents a significant increase in risk of perioperative morbidity/mortality.
- "Normal" aging is the progressive degeneration of structures and functional reserves. Mechanism is debated; worsening in cell repair systems and/or accumulation of free radicals.
- Vascular aging involves degradation of the elastic connective tissue with collagen, which has 100-1000x the stiffness of elastin, and dysfunction of the endothelium. Accumulation of Advanced Glycation End products (AGE's).
- Stiffening of the arterial conduit arteries – analogy of the 'windkessel' system to change interrupted pulsatile flow into a continuous (sinusoidal) flow. The higher the compliance, the lower the pulse pressure and more consistent the flow. Pulse pressure is a measure of compliance!
- Pulse pressure is also determined by wave refraction. The reflected pressure wave plays a role in diastolic coronary perfusion; arterial stiffening leads to rapid refraction, adding to increased systolic pressure and decreased diastolic perfusion. Double hit to the old heart…
- Increased pulse pressure causes
    - Increased systolic load to ejection
    - High cardiac oxygen consumption
    - LVH with decreased capillary refill
    - Reduced diastolic perfusion pressure
    - Inefficient organ perfusion
- Pulse pressure is an age-independent predictor of stroke (controversial)
- Chronic heart failure is a disease of the elderly. There are two phenotypes – reduced ejection fraction (reduced LV function) and preserved EF. Both types result in similar mortality

- Diastolic HF has preserved EF and accounts for approximately half of cases.
- Very easy to diagnose LV inflow patterns with echo. The early inflow is reduced; maximum filling speed decreases with age, and the atrial kick becomes essential.
- What is the basis of diastolic dysfunction? Replacement of elastic tissue with collagen tightening/stiffening of the sarcomeres.
- Decreasing compliance of the ventricles results in an increase in the gradient of the pressure/volume curve; the elderly heart is emptier at the same pressures as the young heart; conversely, adding the same volumes to an old heart as one would to a young heart will overload it easily.
- There are also the problems of impaired baroreceptor response, impaired parasympathetic control, decreased responsiveness to beta agonists, reduction in effectiveness of ischaemic preconditioning (Nguten et al 2008), etc.
- Risk assessment! Comorbidity, pulse wave velocity, pulse pressure, telomere length, exercise. Can we reduce risk?
- Atrial fibrillation is also a degenerative disease, provoked by atrial enlargement (secondary to diastolic dysfunction)
- Summary:
    - Increased vascular stiffness
    - Increased cardiac stiffness
    - Narrow therapeutic goals – maintain diastolic pressure, restrictive fluid status but enough to do the job
- "Youth is a wonderful thing, but what a crime to waste it on children." GBS

Understanding Cardiomyopathies – Prof Justiaan Swanevelder, University of Cape Town

- Cardiomyopathies – 'heart muscle disease', "intrinsic disease of the myocardium associated with cardiac dysfunction". WHO classification:
    - Category I – specific and unclassified cardiomyopathies
    - Category II – established cardiomyopathies with intrinsic disorders of the myocardium
        - DCM
        - HCM
        - RCM
        - ARVD
    - Other classifications – AHA 2006 (Maron BJ Circulation 2006), European Society of Cardiology 2008 (Elliot et al Eur Heart J 2008).
- Specific causes:
    - Ischaemic/hypertensive/valvular
    - Inflammatory
    - Toxic
    - Metabolic
    - Neuromuscular (Duchenne's, Friedrich's ataxia)
- Unspecified – vertricular noncompaction, Tako-Tsubo

- LV non-compaction:
    - LV 'stops growing' in the fetal state (about 8 weeks developmental stage)
    - Deep recesses, spongiform 2-layer appearance (Rehfeldt KH et al 2008)
- DCM – exclusion criteria: abnormal loading conditions, congenital heart disease, excessive alcohol, etc.
- Doppler pattern changes dramatically – very short E (early) wave, prominent A (atrial kick) wave). Atrial fibrillation occurs frequently. 3 functions of the atrium – reservoir, conduit and atrial kick. In cardiomyopathy, the heart becomes more dependent on the atrium.
- RCM is the least common. Primary and secondary causes; amyloidosis is the most common infiltrative type. Atria often larger than ventricles. Interesting paper out of Maputo – see Mocumbi AO et al. Heart 2008. NB – difference between restrictive cardiomyopathy and restrictive pericarditis.
- HCM – good review – Elliot & McKenna, Hypertrophic Cardiomyopathy. Lancet 2004;363:1881-91. Cause of young adult exercise-related death. Familial, autosomal dominant. Can occur at different levels within the heart! Typical 'dagger' shape on pulse wave dopper.

Session 2 – Drugs and Fluids

Oxygen: Good or Bad? – Dr Bill Wilson, Adelaide, Australia

- Hypoxia not only stops the machine, it wrecks the machinery (Haldane)
- High saturation past a point only increases the dissolved fraction, not the saturation.
- Oxygen toxicity
    - All mammals are sensitive to exposure to hyperoxaemia
    - Pulmonary toxicity at 1 ATM at 100% - bronchoscopic evidence at 6 hours; cough, pain and lung injury at 24 hours
    - CNS – Hyperbaric air at 8 ATM causes convulsions
- Reactive oxygen species (ROS) – free radicals (unpaired electron) and intermediates (H_2O_2)
- Free radicals are easily converted to hydrogen peroxide in the mitochondria under normal conditions. We have good defence mechanisms (enzymatic and non-enzymatic). Poorly developed in neonates and begin to fail in the elderly
- Massive production of free radicals if there is high dissolved oxygen tension. Reperfusion and mitochondrial injuries follow.
- Physiological role of ROS –
    - Activate guanylate cyclase
    - Control of apoptosis
    - 'Oxygen sensor' in the carotid bodies and for erythropoietin
    - Role in ischaemic preconditioning
- Nunn editorial in BJA Dec 2007 refers
- Preoxygenation – increases O_2 in FRC and reduces the time for desaturation. However, oxygen in a drug and should be treated as such. Concern for ROS, absorbtion atelectasis,

etc. Degree of alveolar collapse is significantly increased if 100% O2 is used. Atelectasis with 100% O2 – 15% atelectasis; 80% - 3%; 60% - 0.6%

- Grocott HP Anesthesiology Clinics – avoid excessive [O2] in one lung ventilation
- Austin BMJ 2010 – mortality of 9% in high-flow vs. 4% in titrated O2 (target SpO2 88-92%) in patients with COPD
- AVOID trial (underway) – Prehopsital O2 may WORSEN outcome in ischaemic heart disease/ACS/MI
- McNulty et al – coronary blood flow measured by intraluminal Doppler showed significant DECREASE in CBF with 100% O2 as compared to 40%; also showed significant decreased vessel diameter on 100% O2 with angio
- Kilgammon JAMA 2010 – Improved survival with normoxia as opposed to hyperoxia in retrospective analysis of post-cardiac arrest patients.
- Hyperoxaemia increases surgical site infections (although this was a small effect that is minimal compared to other factors).
- NO benefit of supplemental O2 during Caesarean section for elective/routine procedures; no evidence of harm in emergencies (but no real evidence of benefit).
- Neonates resuscitated on oxygen potentially do worse than those rescuscitated on air
- Retinopathy of prematurity, bronchopulmonary dysplasia, functional MRI changes all occur in neonates who become hyperoxic.
- Summary: Oxygen is a drug, hypoxia is BAD; hyperoxaemia may be bad too.

Making PCA Safe – Jeff Coetzee

- Relieving pain decreases the surgical stress response, which in turn decreases the incidence of complications
- Postoperative pain management is still largely dependent on opioids, which have a very variable dose requirement based on individual factors. The MEAC (minimum effective analgesic concentration) is very consistent in INDIVIDUAL patients, but very variable between individuals.
- Avoid peaks and troughs… hence the principle of PCA
- BUT…there are many, many examples of respiratory depression DESPITE the use of PCA
- Causes: lack of training/knowledge, improper prescribing, inadequate monitoring.
- Respiratory depression caused by opioids has three components: decreased respiratory drive, sedation and airway obstruction. Together these cause OIVI: “Opioid Induced Ventilatory Insufficiency”
- Opioids affect the genioglossus muscle, which plays a crucial role in maintaining a patent airway
- Pain ‘protects’ against the respiratory effects of opioids; opiated patients who then have a nerve block to address their pain rapidly develop respiratory depression.
- The water-soluble nature of morphine creates a powerful concentration gradient; blood concentrations and effect site concentrations can thus differ dramatically.
- Basic components of PCA:
    - Load the patient properly!

    - Achieve the MEAC before commencing the PCA
    - Choose an appropriate PCA demand dose – optimal is generally considered 1mg
    - Out of practice due to no good evidence and/or evidence of harm – background infusions and 1 or 4 hour limits.
- Managing PCA:
    - Morphine loading dose 2-4mg every 5-10min until pain <= 4/10
    - Start PCA with demand dose 1mg, lockout 6-8min
    - If analgesia is successful, continue
    - If the analgesia is not successful, evaluate. If the patient is sedated, use an alternative strategy (add NSAID or regional etc). If the patient is not sedated, improve your loading dose with titrated boluses
    - If this is still ineffective, an infusion with continuous monitoring is advised.
- Standardise dilution (1mg/ml)
- Premixed syringes (from pharmacy) advisable
- Dedicated IV line preferable; non-return valves essential; don't allow 'add-a-line' to be connected to the PCA IV!
- Monitoring PCA –
    - Monitoring respiration rate alone is not sufficient; low rates signal a problem, but insufficient volumes at an adequate rate is a far more common occurrence. Nurses should be trained to assess volume and adequacy of breathing as well as rate
    - Sedation level/score should also be assessed and monitored
    - Sedation – the sixth vital sign?
    - Future monitoring – capnography; breath sounds by automated system?
- Nurses are the cornerstone for safe PCA. Perhaps we should have training and certification in PCA. Must be able to recognise OIVI; inadequate ventilation, etc.
- The time and place to educate the patient about the PCA is not in the recovery room! Warn patients that the device cannot take the pain away completely.
- "We must live with the fact that morphine analgesia is accompanied by respiratory depression; our patients must live in spite of it."

EBM: Do we need gelatins and starches? – Andy Parrish

- Revised Starling equation and the glucocalyx model of transvascular fluid exchange – see Woodcock & Woodcock
- Is there a sub-glycocalyx space?
- Efficacy: choose an appropriate effect measure! Is it surrogate vs. patient-relevant (live longer, get well, get well quicker, less resource-intensive)
- The real hard outcome is only one thing… mortality
- Beware the hazards of interpreting Forest plots
- Gelatins – no demonstrated mortality advantage in any of the trials
- Cochrane groups (prior to the 4 big new trials) showed no advantage in mortality
- Big trials: VISEP, CHRYSTMAS, 6S, CHEST, SAFE, BASeS
- 6S – caveat infusor – subgroup analysis!

- Safety data:
    - VISEP was stopped early for safety
    - CHEST mortality rate was lower than expected
    - Short follow-up misses evidence of harm
- Cognitive psychology and practice patterns…
    - 'Group think' and the 'we all do it' phenomenon
    - Substitution and solving of an evidence problem with a simpler plausibility/belief problem
    - Availability bias: many trials; few good trials
    - Framing: a search for the "right" colloid when we should be searching for whether to use them at all
- Conclusions:
    - Biological models in flux
    - No convincing evidence of mortality benefit
    - Convincing evidence of mortality disadvantage in severe sepsis
    - Concern for harm in terms of increased renal replacement therapy
    - Change in practice is appropriate

Make it Zero – Edwin Turton

- Introduction to the LifeBox product – www.lifebox.org
- Pulse oximeter is the only monitoring device that has been featured on the WHO Surgical Safety Checklist

Session 3 - Paediatrics

The Child with a Murmur – Kotie Bester, Red Cross Children's Hospital

- 50-80% of children have a murmur at some time
- Only a fraction of these are pathological
- Murmurs are caused by turbulent flow. Normal flow is laminar. Turbulence occurs if there is abnormal density, diameter, velocity or viscosity (recall Raynaud's Number)
- Children have relatively brisk flow, small vessels, acutely angled branches, and little intervening tissue, so murmurs are more commonly heard.
- Hx – look for family history of congenital heart disease (3-5x increase in risk if there is a family member with CHD), sudden cardiac death or sudden infant death.
- Pre- and perinatal Hx – think of drugs (lithium, SSRIs), alcohol, toxins, intra-uterine infections (NB rubella), maternal diabetes and prematurity
- Associations – chromosomal disorders, syndromes, rheumatic fever, etc
- Symptoms – poor feeding, fatigue, diaphoresis, poor exercise tolerance, failure to thrive, poor growth, history of swelling (eyes in the morning), cyanosis, dizziness, palpitations, chest pain, syncope or near-syncope. Chest pain and syncope are very uncommon, but when they

occur they are associated with AS and HCM. Respiratory symptoms – recurrent chest infections.

- Most common symptoms in the emergencu room – dyspnoea, fatigue, nausea & vomiting, dizziness
- Examination tips:
    - You cannot auscultate a crying child
    - Aim for the target – aim for the heart first. Ignore the murmur initially – listen for everything else that will help you make the call (clicks, extra sounds, diastolic sounds, etc).
    - Examine the abdomen to learn about the heart – situs inversus can be indicated by a liver on the wrong side; hepatomegaly is a common feature of failure.
    - Is the child dysmorphic? 25% with heart problems have other dysmorphic features.
    - Check pulses in all limbs; try to check gradient between arms and legs (>20mmHg is abnormal) and check the capillary refill
    - ECG and CXR are not particularly useful….unless the child has LVH, which is associated with AS and HCM.
- Physiological murmur:
    - Intensity changes with changes in flow (valsalva, exercise)
    - Most systolic murmurs are benighn
    - Most pansystolic and diastolic murmurs are pathological
    - 7 S's: Sensitive (changes in position, respiration), systolic (ejection), short, single, small (not radiating), soft (grade 1 or 2), sweet (not harsh).
- Red flags: HCM:
    - Increased intensity on standing or valsalva
    - Decrease or no change on passive leg raise
    - Decrease or no change when going from standing to squatting
- More red flags:
    - Abnormal S2
    - Systolic clicks
    - Increased precordial activity (may be ASD/VSD/PDA)
- Seven innocent murmurs:
    - Still's Murmur – most common, probably LV outflow. LLSB, early systolic, decreases on standing, typically at 2-6 years. Twanging/tuning fork character.
    - Aortic systolic murmur – over aortic area (URSB), ejection systolic.
    - Pulmonary flow murmur – LSB 2-3rd interspace, peaks in mid-systole, dissonant quantity, loudest supine (decreases when upright). Exclude ASD, PS.
    - Peripheral pulmonary stenosis – axilla or back, grade 1-2, low pitch, early to mid-systolic, infants. Exclude PA stenosis
    - Mammary artery soufflé – Caused by arteries and veins running to and from the breasts, high pitch, along chest wall, stretches from systole into diastole.
    - Venous hum – over low anterior neck, lateral to SCM, resolves or changes when supine, continuous whirring quality, accentuated in diastole.
    - Supraclavicular systolic murmur – early brief systolic murmer
- VSD: Holosystolic, 20-25%
- ASD: 8-14%, ejection systolic, ULSB, wide fixed split S2, may have very soft diastolic rumble

- PDA: 6-11%, continuous murmur, ULSB, can radiate to back
- PS: 7.5-9%, ESM, ULSB, radiates to infraclavicular regions, axilla or back
- Coarct: 5-8%, heard at back
- AS: 5-6%, ESM, URSB
- Approach:
    - Consider risk, complications to avoid, haemodynamic aims, endocarditis prophylaxis, paradoxical embolism
    - Probably innocent (nothing on history, asymptomatic, good effort tolerance) -> Proceed
    - Ominous:
        - Less than 1 year (esp < 6 weeks). Under 1 year may be asymptomatic even if the defect is significant.
        - High anaesthetic risk
        - Extracardiac abnormalities
        - Cyanosis, PHT, arrhythmias, PS
    - Uncertain about lesion, but asymptomatic, uncomplicated lesion in the differential, and minimal risk procedure -> Probably safe to proceed, but cardiology referral postoperatively.

Muscle disorders – Dr Rebecca Gray, Red Cross War Memorial Children's Hospital, Cape Town

- Weakness can be as a result of problems at any level from the brain to the muscle tissue itself, but this talk will focus on the muscle diseases specifically
- Myotonias:
    - Channelopathies
    - Triggered by pain, cold, stress and diathermy
    - Can be dystrophic or non-dystrophic
    - Steinert's Muscular Dystrophy – respiratory insufficiency, cardiac involvement (DCM) and mental retardation.
    - Assess preoperatively – establish disease severity, avoid triggers, check electrolytes.
    - Muscle relaxation is a challenge. Sux is out! Neostigmine might trigger contraction. Volatiles useful but can cause a lot of respiratory depression. Good analgesia is the key.
    - NOT associated with MH
- Muscular dystrophies
    - X-linked, but up to 1/3 are spontaneous
    - Most common is Duchenne's. 1/3 mentally retarded.
    - Death is usually due to cardiomyopathy.
    - Female carriers are prone to scoliosis and cardiomyopathy
    - Restrictive lung disease picture with poor cough and recurrent infections. Think about pre-op optimisation and post-op support. BiPAP is a useful modality.
    - Cardiac problems are easy to underestimate (patients usually sedentary). Almost impossible to do stress testing, and scoliosis makes echo difficult.

(Notes pending completion)

Ketamine: New Tricks for Old Dogs – Dr Graeme Wilson, Red Cross War Memorial Children's Hospital, Cape Town

(Notes pending transcription)

Refresher Day 2 (Sunday)

Session 4 – AGMs and other meetings

Session 5 – Patient Care: Ancient and Modern

Changes in obstetrics in the last 15 years: Is there anything new under the sun? – Prof Rob Dyer, University of Cape Town

- Massive amount of literature – this talk to focus on the areas of new developments and practice
- Sophistication of epidural anaesthesia: potency and motor block, methods of administration, methods of infusion and combined spinal-epidural
- Patient-controlled epidural anaesthesia – bolus administration of local works better than infusion, reduces total dose, and requires less interventions
- Epidurals cause…
    - No increased risk of Caesarean
    - Duration of first stage unaltered
    - Unaltered cervical dilation when administered
    - Second stage prolonged, but only about 15 minutes on average
    - Increased incidence of assisted delivery
    - Improved cord gases
    - Temperature effects are real
- CSE: Faster analgesia, less rescue analgesia required, but more pruritis, fetal bradycardia. No effect on labour outcome; little basis for using CSE over conventional epidurals
- Continuous spinal: PDPH incidence high, risk of infection, technical difficulties – for specific indications only (severe cardiac disease, morbid obesity)
- Good review: Butwick and Carvahlo ?2010
- Alternatives to regional – IV remifentanil: Kinetics favourable, crosses placenta favourably, mixed successes in trials, recent fatality. Interesting with sufficient monitoring.
- Arterial circulation very important in the modulation of spinal hypotension
- What influences the degree of hypotension? Case selection, dose, aortocaval compression, fluids, vasopressors.
- Saving mothers report – 79% of fatalities in SA were under spinal anaesthesia; management of spinal hypotension was a core problem.
- See Gelman S – Venous Function and Central Venous Pressure (pretty much required reading)
- The patient who has bled has a high stressed volume and spinal anaesthesia can be fatal; the elective patient has plenty of volume and the spinal is (relatively) safe.
- 8mg is the critical minimum for safe anaesthesia with spinal; less results in a high risk of additional analgesic supplementation with subsequent side effects
- Mercier 2012 – Cesarian delivery fluid management: Crystalloid co-load is better than preload. Colloid preload better than any co-load, but is expensive.
- Haemodynamic responses to spinal anaesthesia –
    - Hypotension with tachycardia (95%) – SVR decreased 35% on average, so treat with pressor (not inotrope), ie. Phenylephrine (not ephedrine)

    - Hypotension with bradycardia (3%) – attributed to Bezold-Jarisch Reflex, or (Reverse) Bainbridge Reflex. Final common pathway is the vagus nerve, so the treatment is anticholinergic.
- Excessive ephedrine causes fetal acidosis
- Categories of urgency of Caesarean section – see RCOG guidelines (www.rcog.org.uk)
- Pathophysiology of preeclampsia and peripartum cardiomyopathy
    - Increasing understanding into the pathophysiology of preeclampsia – 2 stage disease, first the development of an ischaemic placenta, and then the effects of sFlt-1 and oxidative stress resulting in clinical signs of preeclampsia (see Fernando Anaesthesiaology 2007)
    - LV stroke work index in the untreated patient is raised – the so-called 'inovasoconstrictor state' (see Dennis AT PhD study)
    - Spinal anaesthesia is safe in preeclamptic women – has been demonstrated well in the literature in the last 15 years.
    - See BJA 2009;102(3) for interesting commentary
    - Nature, May 2012: Cardiac angiogenic imbalance leads to peripartum cardiomyopathy. Angiogenesis in pregnancy leads to physiological hypertrophy. Mice without the growth factor developed cardiomyopathy. There may be a link with preeclampsia. Hypothesis developing research!
- Regional anaesthesia for cardiac disease
    - Here to stay!
    - Safe with careful use – several recent papers
- General anaesthesia:
    - Failed intubation drill has improved organisation and outcomes (see Vaida et al J Clin Anesth 2009)
    - Remifentanil has found a place in attenuation of intubation responses, but there is a high incidence of requirement for neonatal resuscitation
    - Awareness – major progress has been made. See Robins et al Anesth Analg 2009
    - Thiopentone up to 7mg/kg
    - Overpressure inhalation (iso or sevo)
    - Target 0.8 MAC or 0.5 if used with N20
- Obstetric haemorrhage:
    - Management strategy/protocol are required
    - Massive transfusion protocol
    - Effective surgical intervention
    - Landmark paper – Phaneuf et al 2000 – down-regulation of oxytocin receptors
    - Oxytocin remains the gold standard. ED50 is very low (0.35u). Standard bolus 3u over 60 sec
    - Fibrinogen level of <2.0g/L is an early predictor of severity of PPH. See IJOA 2013 editorial by Butwick: Postpartum haemorrhage and low fibrinogen levels
    - Antifibrinolytics: WOMAN trial is on the go.
    - rFVIIa: See Mercier and Bonnet 2010 for a good revew
    - Choice of anaesthesia in placenta praevia? Only one paper (Usta Am J Obstet Gynecol 2005) shows accreta increases after 2 previous caesarean sections.
    - Cell salvage is here to stay. See Yentis IJOA 2008

- Post Caesarian analgesia:
    - High safety profile of neuraxial opioids in the obstetric setting
    - Multimodal; 100 mcg intrathecal morphine with systemic paracetamol and NSAID
    - TAP block: Intrathecal morphine is better, limited duration, should be ultrasound guided
- South African audit – largest in the world after NCEPOD. See more at the congress
- Obstetric anaesthesia in low-resource settings – see Dyer, Reed & James 2009

Introduction to Point-of-Care Ultrasound – Prof Massimiliano Meineri, Toronto, Canada

- Point-of-care testing can be performed at the bedside. Spock/Star Trek refers…
- Hand-held pocket ultrasound is already in use; USB ultrasound is here; smartphone ultrasound has been developed and is going on sale
- Read about the "ICU-sound" Protocol in Anaesthesiology Oct 2012 (and accompanying editorial)
- POCUS is used in many applications – Heart, lung, FAST, DVT, vascular access, etc.
- Complete TTE requires 40 tasks; FOCUS TTE looks at 3 sites and only 5 views (PLAX, PSAX, A4C, IVC, S4C). This can assess for effusion, function, dilation, volume, and place a pericardial drain if required.
- The FATE protocol is similar to FOCUS, but has a slightly different order and includes checking for a pleural effusion.
- There are many other protocols: BLEEP, CLUE, FAST, RUSH, RACE, HEART, INBU
- Focussed assessment is supported by a large body of literature, especially in critical care. Changes in diagnosis and/or management occurs in 50-65% of cases.
- US has a role in resuscitation during CPR – see Breitkreutz et al Resuscitation 2010. Echo altered management in 78% of cases.
- FATE predicts cardiovascular morbidity (see Cowie, J Cardiothroacic & vascular anaesthesia 2012) – however, the protocol used was more extensive than normal FATE
- Lung ultrasound can be used for assessment of pneumothorax, pleural effusion, consolidation, interstitial disease, etc. Lung sliding after central line placement can exclude pneumothorax – sensitivity is HIGHER than CXR with similar specificity (Alrajhi, Chest 2012)
- Many courses and online modules for learning POCUS are available. There are also a number of smartphone applications. FATE application is free on iTunes. Toronto website is excellent (and free) – http://pie.med.utoronto.ca/TTE or http://pie.med.utoronto.ca/TOE
- Is preoperative FOCUS TTE ready for prime time? See Manecke article!

The Variability of Brain Protection Measures – Prof Tony Figaji, University of Cape Town

- "The most important strategy is profound knowledge on cerebral physiology and homeostasis in health and disease." Klein & Engelhard 2010 Best Practice Clin Anes
- Malapropism of the day: "There was a great deal of interest in trying to protect the brain from the pharmaceutical industry" (Tony Figaji in the presentation)…. HAAA!

- Pharmacological means have suffered a failure to translate from the animal research
- Prevention of secondary injury remains the key focus.
- Trouble with trials of TBI Therapies:
    - BP, hypothermia, decompressive craniectomy, intracranial pressure monitoring… All trials in the last 2 decades have failed
    - Is there something systematically wrong?
    - Outcomes in the best centres are consistently better than the rest. What is it that they are doing? Is it just high-intensity therapy?
    - Our thinking in management approaches in the brain may be too simplistic (example of the thought process in treating infection given)
    - Failure in places may be due to the way we've been performing the EBM; sample size often dilutes the benefit in the patients that were suited in the first place (but we didn't know who will and won't benefit).
- Heterogeneity presents on many levels
    - Pathology
    - Pressure-volume curve
    - Pressure autoregulation
    - Metabolic regulation/CBF coupling
    - CO2 reactivity
    - O2 reactivity
- High ICP is bad…
    - CBF and oxygenation drop in an exquisitely sensitive relationship
    - BUT ICP monitoring is not being shown to help.
    - There is a huge degree of inter-individual heterogeneity
    - We cannot approach all causes of raised ICP in the same way! A mass lesion does not behave in the same way as cerebral oedema or subclinical seizures, for instance
- Blood pressure:
    - Pressure (auto)regulation maintains a fairly steady CBF under normal conditions, but in the injured brain it becomes unpredictable
    - A patient with impaired autoregulation can suffer an _increase_ in ICP with an increase in BP due to increase intracranial blood volume
    - Cannot state that "higher BPs are better" – we must individualise the management to optimise CBF – monitoring helps in this regard.
- Haemoglobin:
    - Blood transfusion usually improves cerebral oxygenation, but not in all patients. Practically, if there is a high risk of cerebral ischaemia, transfusion trigger should perhaps be raised to 9 or 10 g/dL.
- Oxygenation:
    - Increased Fi02 increases PaCo2 which increases PbtO2…but this is only TENSION not CONTENT. Increasing FiO2 can cause decreased CBF via hyperoxic vasoconstriction.
- Carbon dioxide:
    - Hyperventilation leads to worse outcomes – decreasing cerebral blood volume decreases ICP, but also drops CBF which reduces total delivery

    - When ICP is high, decreasing CO2 be useful, but when it is low there is no additional benefit. When ICP is low, allowing CO2 to increase may be helpful to a point. These points are very heterogeneous.
- Steroids:
    - Big studies say they don't work.
    - Microdialysis work (at UCT) suggests that the inter-individual variability may be the fault; there is a huge difference in the reaction to steroids in different patients.
- There are some events that we know exist but don't yet understand… see papers on 'spreading depressions'
- Interventions in ICU can cause severe changes in ICP and brain oxygenation without the patient showing any clinical evidence of stress
- New developments and/or becoming more common in intraoperative brain monitoring:
    - Motor evoked potentials
    - Somatosensory evoked potentials
- Patients with focal pathology don't tolerate drops in pressure below normal
- Likely the best strategies for neuroprotection:
    - Physiology is complex and variable
    - Best way to deal with it is monitoring, not assumption
    - Neuroprotection is not futile, just very, very hard.

Session 6 - Cardiopulmonary

Haemodynamic monitoring – refresher & update – Prof Patrick Wouters, University of Ghent, Belgium

- Not going to discuss echo (lots of other lectures) nor the PA catheter (the sun is setting)
- Many new devices have emerged on to the market for varying degrees of non-invasive haemodynamic monitoring
- It is crucial to understand the underlying assumptions to understand the limitations
- Four major groups:
    - Fick principle – indicator dilution
    - Pulse contour analysis
    - Doppler principle: flow velocity
    - Thoracic bioimpedance/bioreactance
- Fick principle:
    - Mass out = mass in + mass added
    - Used for bolus thermodilution using the Stewart-Hamilton equation
    - PICCO plus setup: Pulse contour plus temperature thermodilution
    - A large bolus makes this technique as accurate as SG catheter
- Pulse contour analysis
    - Fluctuation around the mean is proportional to the volume
    - Compliance of the system must be known to perform the calculation accurately – non-linear relationship makes this difficult to perform. PICCO relies on the thermodilution.

    - Arterial blood pressure signal must be good – over-dampening or excessive resonance will result in inaccuracy
    - The more distal the measurement, the more inaccurate.
    - PulseCO/LiDCO uses lithium for calibration; some related issues such as delay in performing repeat measurements, cross-reaction with quarternary ammonia drugs (eg. NDMRs) and adjustment for haemoglobin and sodium levels.
    - Vigileo does not use calibration; special transducer; must enter data for normogram (weight/height)
    - System does not work accurately in patients with low SVR states; new algorithms have improved this, but frequent calibration is required, especially if there are changes in SVR
    - Inaccurate with AV insufficiency, irregular heart rhythms, IABPs, sepsis, dampening, poor calibration, etc.
    - Extra-vascular lung water measurement is a highly calculated value – many presumptions. Perhaps useful for trends, but treat with a degree of caution.
    - These monitors are designed to work on patients with mechanical ventilation; they are inaccurate when the patient is breathing spontaneously
    - Fingertip non-invasive pulse contour analysis machines are now available (NEXTFIN, VISMO); one uses the ECG signal as well to calculate transit time. These use more layers of assumption and calculation – trends may be useful though.
- NIRS (Near Infrared Spectroscopy):
    - Closest estimation of oxygen consumption – has been validated with mixed venous O2 saturation in patients undergoing OPCAB
    - Allows examination of balance between oxygen supply and demand
- Conclusions:
    - Non-invasiveness => less accuracy
    - Knowledge of physics and maths required to make informed decisions
    - Straight leg raise is still a good test
    - NIRS is underutilised – window to global hemodynamics

Anaesthesia after Heart/Lung Transplant – Adriaan van Rensberg, University of Toronto, Canada

- More than 20 000 transplants per year in USA today. Survival is becoming very good – 1 year 'all comers' survival is 80%. Heart transplant 5-year survival is 75%.
- Always anticipate organ dysfunction in the graft and other major organs.
- All have immune suppression; most are on prophylaxis
- Frequent side-effects of immunosuppression and medication:
    - Infection and malignancy (48%)
    - Peripheral vascular disease (12%)
    - Diabetes
- Interactions between medications common
- Disruptions in the perioperative period can upset the balance of immunosuppressive therapy
- All these patients have an altered inflammatory response

- Overall 4-fold increase in cancer risk
- Neuropsychiatric complications = 33% ! Seizures, neurotoxicity, encephalopathy, infection, cerebrovascular disease, etc.
- Most patients develop some form of renal insufficiency regardless of the type of graft
- Surgery after previous heart transplantation:
    - Most common indication for transplant is IDCM or ICM
    - Technique is important; recently the caval-caval anastomosis has become popular
    - All transplanted organs have pathophysiology
    - Denervation is complete (Sympathetic, parasympathetic, sensory)
    - Must use direct-acting drugs
    - Re-innervation can occur but timing is not well elucidated
    - Baseline rate is 90-100
    - Little or no response to intubation, light anaesthesia, carotid artery massage, etc.
    - No pain from ischaemia
    - Normal contractility
    - Increase cardiac output – not through increased HR reflex, so must increase preload or give positive chromotropes
    - 40% of transplant patients will present later for non-cardiac surgery
    - Emergency surgery carries a higher risk than elective surgery
    - Beware preload dependency when using neuraxial anaesthesia!
- Cardiac allograft vasculopathy:
    - Diffuse distal coronary artery narrowing
    - Intimal thickening
    - Etiology multifactorial
    - Major cause of death after 1 year
- Lung transplantation:
    - Most frequent indication is COPD, followed by cystic fibrosis
    - Bronchial-bronchial anastomosis, with cuff of donor atrium connected to PA's
    - Cough reflex is maintained, but there is denervation below the level of the bronchial anastomosis
    - RV remodelling occurs. There is a disruption of lymphatic and cilliary clearance
    - Rejection is characterised by bronchiolitis obliterans. Obstructive pattern on PFT.
    - Single lung transplantation creates differential ventilation, perfusion and compliance. Very challenging to ventilate.
    - 18% of patients will present for non-lung surgery. Emergency surgery carries significant mortality. Beware blocks in the neck for patients who have phrenic nerve palsy.
    - Caution intubation past anastomoses – use direct vision if you have to
    - Use a lung-protective strategy

Intra-Operative Management of the Patient with Severe COPD – Peter Slinger, University of Toronto, Canada

- Case presentation – 60F, laparotomy for bowel obstruction, FEV_1 27% predicted, treated with inhalers, occasional steroids.
- CXR is essential to recognise bullae!
- Pressure in a bulla is equal to mean alveolar pressure. In spontaneous breathing, this is around -2cm H_2O. Positive pressure ventilation is almost certainly going to cause inflation, with potential sequelae.
- Get an ABG: is the patient a CO_2 retainer or not? When you're trying to get the patient off the ventilator post-op, you want to know where the baseline is! (Get the girl's address if you want to drive her home after the dance…)
- Excessive O_2 will increase physiological dead space through reducing hypoxic pulmonary vasoconstriction in the unhealthy lung units. This causes an 'intrapulmonary steal', upsetting the fragile balance that has developed as the disease has developed.
- Don't fear the CO_2 retention; CO_2 is 'relatively' good for you; the body can store vast quantities. Apnoeic oxygenation will keep you alive for a long time (until acidosis finally kills you).
- Dynamic hyperinflation and the Lazarus phenomenon
    - Allow time for deflation
    - Bronchodilators
    - Small volume of PEEP (around 50% of autoPEEP – see Caramez MP et al. Crit Care Med 2005;33)
- Thoracic epidurals:
    - Aim for sensory (not motor) block – 0.25% bupivacaine has shown to be safe
    - MASTER trial showed thoracic epidurals reduce respiratory failure, which is the most costly (and second most common) complication of abdominal surgery. COPD patients get the most benefit from this effect (3-fold decrease in respiratory complications after abdominal surgery).
- Summary:
    - Think of bullae
    - Think of CO_2 retention
    - Think of dynamic hyperinflation
    - Get great analgesia (thoracic epidural definitely indicated).

Congress Day 1 (Monday)

Session 7 – Invited Speaker Plenary

Welcome by Adrian Wentzel

- Delegates welcomed by the congress organiser

Understanding NHI: Is it really our only option? – Theuns Botha, Western Cape MEC for Health

- Good health and access to an equal quality health care system is the ultimate goal
- NHI in its current proposed format would require a 26% personal tax increase
- Countries in which NHI works well have a good proportion of tax payers… RSA has 1 in 10 citizens paying tax (5 of 51 million) and nearly 1 in 3 (16 of 50 million) on some form of government grant.
- Various criticisms of the various Green Paper recommendations.
- Huge discrepancy between primary and tertiary level costs noted.

A Positive and Stress Free Environment: Managing the Work Environment and Stress – Alan Glover

- Life is about the way you talk to yourself and the actions you take
- Awareness -> Thinking -> Decision -> Behaviour -> Results -> Reflection
- Welch – four principles: success recipe; methods of behaviour; candour; differentiation
- Critical success factors: strong leaders, great people, quality products and services
- Qualities of a great leader: Visionary person, self-belief and confidence, courage to risk, humility, willingness to serve.

Session 8 – Visitor Plenary Session

Chronic Heart Failure: The Next Challenge in Anaesthesia? – Patrick Wouters, University of Ghent, Belguim

- Incidence of heart failure in patients presenting for surgery >60 years = 20%, CAD 34% (Anaesthesiology 2008;108:558). 30d mortality significantly raised; this has mostly been addressed for CAD but not yet for CHF
- Is our diligence with giving CAD patients beta blockers harming our CHF patients?
- CHF prevalence at age >70 years = 10-20%
- CHF Etiology: CAD 70%, cardiomyopathies, IHD, EtOH, etc
- Definition: Symptoms of heart failure, signs of heart failure, and evidence of cardiac dysfunction
- Classification: NYHA (Class I-IV) or ACC/AHA (Stage A-D)
- Treatment by class as per Circulation 2009;110:1911-2016

- Survival after diagnosis (@ age 75) remains low (1yr = 50%)
- EF is the best predictor of 30d outcome in CHF (low EF reflects poor functional reserve), although good EF (>50%) is no better than >45%
- 2 phenotypes of heart failure – those with preserved LV function (good EF, AKA diastolic chronic heart failure) and those with poor LV function (poor EF)
- Diastolic heart failure:
    - Usually older patients
    - Usually women
    - 3 essential criteria – clinical signs and symptoms of HF, normal EF, small ventricle, abnormality in LV filling and/or diastolic function (See E J Heart 2007;28:2539)
    - Pathophysiology entirely different from systolic failure; increase in contractile performance, but EDV-P relationship shifts upwards. Filling pressure must be increased dramatically to maintain ventricular filling
    - Mitral LA-LV pressure gradient is decreased; early diastolic filling is decreased; atrial kick becomes more important. E:A ratio begins to reverse. Mitral valve blood velocity is decreased. Proportionally greater input of the atrial kick (50 rather than the normal 25%); if these patients lose sinus rhythm they lose 50% of cardiac output!
    - Pressure rise in late filling increases as the ventricle becomes stiffer (compliance decreasing, such as in a hypertrophic ventricle)
    - Left atrial pressures rise over time to try and improve filling gradient; pattern of filling becomes normalised once again, although high pressures remain. Circulatory volume thus plays a crucial role in these patients.
    - Mitral annular motion can be used in conjunction in pulse waveform Doppler to gain a more complete picture. Mitral annulus movement is directly proportional to ventricular compliance. E= mitral inflow = pressure gradient; E' = annular velocity = complicance. E:E' ratio is the key!
- Specific treatment?
    - Majority of trials failed
    - Phosphodiesterase inhibitors improve systolic function and therefore may assist diastolic function, but there is no specific agent available yet.
    - Two situations – small empty hearts, or high pressures in big hearts
    - Rate control: Prolong diastole
    - Rhythm control: keep sinus to maintain atrial kick
    - Focus on oxygen balance – hypertrophic ventricles have relatively hypodense vascular distribution and limited reserve
    - Be restrictive with inotropes and vasodilators – this is the REVERSE of managing hearts with systolic dysfunction
    - Narrow limits for optimal fluid status
    - Maintain diastolic blood pressure at the awake levels
- Systolic HF
    - Maintain low dose beta-blocker
    - ACR-I?ARB/diuretics – cancel on day of surgery
    - Aldosterone agonists? – pending study
    - Limit NSAID (?)

- Different approaches – not all CHF is equal!
    - LV systolic HF: vasodilation > inotrope > vasopressor
    - LV diastolic HF: vasopressor > inotrope > vasodilation
    - RV systolic HF: inhaled vasodilator > inotrope > vasopressor

What's New in Regional Anaesthesia? – Prof Vincent Chan, University of Toronto, Canada

- Trend towards more outpatient procedures has increased interest in regional anaesthesia
- Trend towards more minimally invasive (laparoscopic etc.) surgery
- Trend from central to peripheral nerve blocks
- Shift from blind/landmark techniques to ultrasound guided
- Shift to use RA as part of a multimodal approach
- Attention being given to prolonged analgesia
- Benefits of RA in terms of surgical outcome?
- A&A 2005;101:1634: shorter PACU times, faster discharge in ambulatory anaesthesia
- Use procedure-specific RA techniques and don't forget that local with monitored anaesthesia care is suitable for many procedures (eg. Inguinal herniorrhaphy, ambulatory anorectal surgery)
- Increasing use of anticoagulants has also driven the use of peripheral nerve blocks rather than neuraxial block
- Ultrasound has been shown to be better than nerve stimulation in numerous studies
- Single shot techniques:
    - use long-acting LA
    - consider long release formulation (liposomal bupivacaine). Cumulative pain scores and opioid use still significantly reduced after 72 hours. Clinical safety profile of liposomal bupivacaine – even 600mg dose remained below toxic blood levels. Onset may be slower than normal bupivacaine.
    - Use adjuncts – adrenaline, bicarbonate, alpha 2 agonists, steroid.
    - Evidence for alpha-2 agonists has become more robust in the recent literature; few studies on dexmedetomidine yet but those that have been completed are promising.
    - Ketamine no good
- Continuous PNB:
    - Does improve pain on post-op day 1 (eg. Anesthesiology 2008;108:703)
    - BUT problems with quadriceps weakness in lower limb blocks (see BJA 2013 – Falls and major orthopaedic surgery with peripheral nerve blockade)
- Lipid emulsion – nice review in Anaesthesiology 2012 (?Weinberg)
- New techniques:
    - More distal femoral nerve blocks (Saphenous nerve block) 8-12cm distal to inguinal crease
    - Adductor canal block
    - Catheter with intermittent boluses rather than infusion

    - Local infiltration analgesia – see Acta Orthop 2008;79:174-83 Australian article on knee replacements with two intra-operative injections by the surgeon +- catheter left next to the prosthesis! Nice review – J Bone Joint Surg Br 2012;94:1154-1159. Safe and effective, can reduce hospital stay.
- TAP block:
    - Somatic (but no visceral) pain relief
    - Good review recently shows that optimal technique, mixture and volume is still not known.
    - Large volumes and/or two injections work better than small volumes and/or one injection.
    - More posterior injections seem to work better
- Continuous wound infusion catheters are very effective; may be as good as epidural for certain types of surgery
- RA and cancer recurrence:
    - 2 main studies quoted: Breast cancer (Anaesthesiology 2006;4) and prostate surgery (Anaesthesiology 2008)
    - Look to cell-mediated immunity for possible mechanisms: NK cells, B-helper and B-cytotoxic cells. Apoptosis inducted and angioneogenesis prohibited
    - Surgery may enhance cancer metastasis, anaesthesia can depress cell-mediated immunity. Scientific evidence is exciting but inconclusive. (See BJA 2012:105;106-115). Opioid sparing effects also important. Reduction of peri-operative stress must be reduced with as little systemic drugs as possible
    - These effects may depend on both the type of surgery and the type of cancer (eg. Survival benefit for epidural in rectal Ca surgery, but not for colonic Ca surgery).
    - We need a lot more prospective studies.

Session 9 – Registrar Research Prize

Efficacy of transverse adbominis plane blocks for total abdominal hysterectomy – Adri Marais, University of Cape Town

- Still a fairly novel techniques; only 2 other RCTs of TAP for TAH
- Null hypothesis – USG TAP block does not improve analgesia
- Prospective double-blind RCT, N=30 (15 in each group). Malignancies as indication for surgery excluded.
- Primary outcomes = morphine requirements and pain scores
- Secondary outcomes = nausea, vomiting, pruritis
- Ethics and trial registration performed; written informed consent as well as PCA demonstration.
- Random allocation, blind sealed envelope, 20ml bupivacaine vs 20ml 0.9% saline
- Bilateral TAP blocks done post-induction
- Blocks done by single experienced operator with US guidance; anterior approach.

- Postoperative management with morphine/droperidol disposable PCA. Pain scores with UPAT at rest and with movement. All patients received standard analgesic regiment: paracetamol, indomethacin regularly and antiemetic when required.
- Single investigator (PI) did all pain scores
- Power analysis showed 9 patients required for each group.
- Results:
    - Data from 2 patients in TAP block group due to necessity for relook laparotomy and technical failure of PCA
    - One protocol violation after analgesics altered
    - Morphine requirements were decreased 47% in TAP block group at 6 hours and 49% at 12 hours.
    - 4 patients complained of nausea (2 in each group). No vomiting or pruritis
- Limitations: Not powered to assess difference in opiate side effects. Could not assess after 24 hours
- Conclusions:
    - TAP blocks reliable and safe for TAH

Degradation of paracetamol and other constituents in Perfalgan – Catherine Jackson

- Can Perfalgan vials be used as a multi-dose vial over 24 hours?
- Perfalgan is sold as single-use only and discarded after 2 hours
- Various constituents including mannitol, cysteine, hydrochloric acid
- Expensive in South Africa but very useful in paediatrics
- Many centres using single vial for many children to save costs
- Paracetamol stability testing:
    - High performance liquid chromatography with UV absorption used
    - Chromatogram peak unchanged at 24 hours – paracetamol in solution stable at this period
- Excipient stability testing:
    - Hydrogen NMR used to fingerprint compounds
    - Paracetamol/mannitol/cysteine peaks analysed – superimpose perfectly EXCEPT for the cysteine which was oxydised to cystine, which is the purpose of the additive (preventing oxidation of the other constituents)
- Lipid solubility testing:
    - Octanol:water partition coefficient compared
    - UV spectrums at 0 and 24 hours of suspension showed almost identical penetration
    - Octanol:water partition coefficients remained within normal limits over 24 hours
- Therefore, NO significant change over 24 hours!
- This validates the current practice for cost-saving and prevents fear of underdosing.

Desflurane potentiation of rocuronium and cisactracurium – Pamela Scheepers, University of Stellenbosch

- Older data suggests that desflurane potentiation of rocuronium is less than that of cisatracurium
- Time to spontaneous recovery was compared with a 3xED95 dose
- 4 groups: Roc-Des, Roc-TIVA, Cis-Des, Cis-TIVA
- TOF-GUARD used for TOF 90% target
- Specific measures taken to prevent signal drift and inaccuracy.
- Data analysed with 2-way ANOVA
- Recovery clearly more rapid with propofol rather than desflurane
- A statistically significant difference exists between propofol and desflurane (need to use more NDMR with TIVA).
- NO statistical significant difference between rocuronium and cisatracurium found
- Post-hoc power analysis showed 104 patients would be needed to disprove this; clinical relevance is doubtful.
- Desflurane prolongs mean time to recovery after rocuronium and cisatracurium, but there is no demonstrable interaction between the two muscle relaxants.

Audit of Post-Operative Pain in Orthopaedic Patients - Neil Hauser, University of Cape Town

- Why pain? Relatively poorly controlled, limited data in our patients, aggressive pain control has numerous clinical benefits
- Why orthopaedics? Painful procedures and patient group frequently reporting severe pain.
- 2-cycle audit process
- Visual analogue score of 4 chosen as target.
- Interventions between audits
- Intervention immediately if pain score ever higher than 7 (for ethical reasons)
- 4 time-points used: 0, 12, 24 and 48 hours
- Patient satisfaction also rated in four categories
- Common theme – pain scores dropped significantly at all times.

Use of Respiratory Rate as an Indicator of Post-Operative Analgesia – Magdalena Jaworska

(No notes taken)

Obstetric Anaesthesia at District and Regional Hospitals in KwaZulu Natal – Caseloads, Human Resources and Experience – Anette Theron

(No notes taken)

Session 10 – Smiths Travel Award

The Groote Schuur Emergency Surgery Triage System: A Tool for Prioritising Emergency Surgical Cases in a Busy Tertiary Hospital – Luis Felipe Montoya-Pelaez, University of Cape Town

- Increasing focus in managing emergency surgery systems in the last 10 years. Various policy documents. Underfunding of emergency surgery in favour of elective surgery is a worldwide phenomenon.
- Most takes place after hours, often by trainees, frequently unsupervised, on the sickest patients.
- Focus falls on improving the resource allocation and staffing levels
- Emergency surgery: an acute surgical intervention during the admission, or "an unplanned manner"
- 2006 situation – poor prioritisation, very limited resources, poor outcomes attributed
- Prospective audit was performed to assess needs at GSH
- Bulk of emergency work at GSH is general and orthopaedic surgery
- Location of pre-operative assessment (first contact with an anaesthetist/registrar) was performed in the operating theatre in 87% of cases!
- 52% by SR, 21% by JR, 20% by consultants (1 in 5 cases supervised)
- First case start time was before 08h00 on only 3 days during the audit period
- Audit recommendations:
    - Develop a triage tool
    - Introduce a dedicated triage registrar to perform triage and improve pre-op management
    - Establish an Electronic Emergency Case Booking System
- No study data on triage for emergency surgery (although lots in prehospital and emergency department)
- NCEPOD 2004 triage tool: Immediate, urgent, expected or elective
- Australian tool – specific times allocated (<15 min, <1 hour, <4 hours etc)
- SATS (South African Triage Score) used in emergency units throughout the country. Colour coding (Red/Orange/Yellow/Green/Blue). Not designed for OT.
- Tool developed combining colours of SATS with NCEPOD ratings with Australian times -> Groote Schuur Emergency Surgery Triage System (GSESTS)
- Management of GSESTS:
    - Prioritisation/colour coding is responsibility of surgeon
    - Cases delayed longer than the time-to-theatre are reassessed and re-coded (up or down)
    - Final arbiter of disputes is the anaesthetist
- Triage registrar – appears to have been successful. Not supposed to have other clinical duties (Haaaa!)
- Electronic Emergency Case Booking System (SurgiBank):
    - Created by IT company
    - Colour coding on a large monitor outside emergency OT
    - Immediate visual impression of case load and urgency
    - Space in record for surgical notes
- Timeline:
    - June 2009 – implementation of triage system and triage registrar
    - May 2010 – Electronic system in place
- How are we doing?

    - Electronic system has streamlined and made transparent the bookings
    - Triage has enhanced communication between surgeons and anaesthetic staff
    - Delays due to poor or no workup are infrequent
- Some data:
    - Two years (Jan 2011-Jan 2013)
    - 10815 cases operated; 12798 booked. Average 14.8 cases operated per day
    - Red 549, Oragne 1788, Yellow 4068, Green 6297, Blue 96.
    - Significant improvement in the percentage of time that red cases are operated within the time limit has improved. Total average cases per day dropped from 15.6 in 2011 to 14.0 in 2012
    - Definite lull in cases happening between 0700-0800, and 1800-2000

Drug Errors in Anaesthesia – John Roos, GF Jooste Hospital, Cape Town

- Mechanisms, consequences and avoidance (individuals and systems)
- Risk of dying from medical error is 1:300; odds of dying in hospital from human error is 33 000 times greater than dying in an airline crash (British data)
- 1:10 patients treated will suffer preventable harm (WHO)
- 7000 deaths from medical error per annum in USA
- 1 error per 133 anaesthetics in NZ; 1 in 274 in SA (is this number underreported?)
- Catchpole et al – errors are common, severe errors are uncommon
- 1% error rate extrapolated to the airline industry would be 30 airline crashes at Heathrow a day. 1% is not a small number!
- RSA study (Llewellyn & Gordon et al) – one error per week; experience not important; >50% are substitution errors; dose errors common in paediatric; no difference between emergency and elective surgery
- Substitution errors; wrong route; wrong dose. Omissions are also errors!
- Similar drug packaging is a very serious concern (eg. Mannitol/dextrose/voluven, midazolam 1mg/ml and 5mg/ml, propofol/etomidate, etc)
- Some 'horror stories' related:
    - Suxamethonium and fentanyl
    - Protamine sulphate given while on bypass
    - Bupivacaine injected into CVP rather than epidural
    - Bupivacaine injected into IV rather than epidural
    - Unexplained tachycardia in the paeds tonsillectomy patient – previously prepared 200ml bag of adrenaline that was not used was put back into the warmer and used the next day on a child. Child died as a consequence.
    - Adrenaline drawn up as an 'emergency drug' – chances of incorrect administration are greater than the chances of getting to use it.
    - Phenylephrine drawn up as ephedrine (1 ampoule into 10ml…200x the normal dose!)
    - Calcium chloride instead of normal saline mixed with lignocaine in a Bier's Block
    - Eusol mistaken for saline and injected into the epidural space

    - Ephedrine and morphine (similar ampoules) substituted – "morphine" administration led to increased BP and rate every time it was given
    - The strange case of cefuroxime coma… "Cefuroxime" given as pre-op prophylactic antibiotic in the passage waiting for her procedure collapsed. "Cef" was actually a syringe of thiopental sodium…
- Error mechanisms
    - Busy workload
    - Multi-tasking ("doing everything badly at the same time")
    - Anaesthesia "by committee"
    - Fatigue
    - Distractions
    - Personal stress
    - Medications (NB – ARVs)
    - Memory failure
    - Unfamiliar drugs (eg. Amiodarone)
    - Unfamiliar environment
    - Latent conditions – "organisational pathologies" built into the system that set up mistakes
- Error management:
    - Build a "safety culture"
    - Drug errors WILL happen
    - Safe staff report more errors
    - If you are not reporting you error, you are not managing your risk
    - Those that admit to nothing have the most to hide
    - Robust reporting system
    - Blame-free culture
    - Learn and share safety lessons
    - "Free lessons" – if you can learn a lesson without cost to your patient, it is "free", so learn from the mistakes of others
    - Labels: An absolute no-brainer. Actively read each label, and actively label each syringe. Labels should be colour-coded. Cross-check the syringe against the ampoule
    - Tactile reinforcement: fluted knobs on the anaesthetic machine…but you can also use different sizes of syringes for different drugs.
    - Visual reinforcement with different needles and drug labels
    - Labelled ports and covered/sealed epidural ports
    - Epidural strapped on the opposite side to the CVP
    - Don't strap IV lines and arterial line's 3-way taps on the same arm, and label them
    - Keep your vials and ampoules until the end of the case to allow cross-checking
    - Store similar ampoules away from each other
    - Don't store drugs in the wrong packaging
    - Store dangerous drugs separately – make people fetch them with intention
    - Keep poisons (eg. Bupivacaine) away from IV drugs
    - Calculators in paediatric theatres
    - Antidotes available

- Predetermined protocols for infusions and unfamiliar drugs
- Be aware of fatigue – no non-essential surgery late at night