SASA Congress 2012 – Refresher Course – Day 2 – Sunday 19 February

Ross's Rough Notes

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

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

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

- Airway disasters, massive haemorrhage, and (pre-)eclampsia
- Airway
    - Confidential Enquiries over the years…. AIRWAY, AIRWAY, AIRWAY
    - Obstetric failed intubation rates – 1:240 to 1:280
    - All units must have a difficult airway plan/drill/algorithm
- Massive haemorrhage
    - Case report of undiagnosed placenta acreta presented
    - Should we be doing routine cross-matches? -> if there are risk factors!
    - Moving to 1:1 (or 1:1:1) transfusion protocols

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

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

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

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

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

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- Understanding of the physiology essential
- 2 types of pain – nociceptive and neuropathic
- Nociceptive pain responds to NSAIDS, paracetamol and opioids… as does the pain of inflammation
- Most acute and perioperative pain is nociceptive
- Limbic system controls the affective aspects of pain – fear, anxiety, poor sleep, etc.
- We have at least 6 levels of defence:
    - Step 1: Peripheral activation can be counteracted by the NSAIDs and coxibs
    - Step 2: Nerve conduction can be blocked by sodium channel blockers – ie. Well-placed local anaesthetic!
    - Step 3: Calcium and NMDA channels can be influenced by gabapentin and ketamine
    - Step 4: Central activation can be again countered by the NSAIDs and paracetamol
    - Step 5: CNS opioid receptors are at our disposal
    - Step 6: Limbic modulation of the spinal cord can be influence by alpha2 drugs
- So, what's new in SA?
    - Hydromorphone – works on the central opiate receptors. Old drug elsewhere; new to us. Once-a-day formulation using OROS system (osmotic pump in hard capsule) providing 24-hour profile. Absorption is predominantly in the colon, and is thus unaffected by feeding patterns. Many metabolites; none active. Typical opiate side effects. Dose is approximately 1:5 with morphine (1mg hydro:5mg morphine)

    - Oxycontin – well known in USA, new to us. Predominantly kappa and then mu effects. Controlled-release short-acting preparation. Tablets must never be crushed or chewed.
    - Buprenorphine transdermal patch – partial agonist; highly lipid soluble, low molecular weight. Partial mu-opioid agonist. 7-day preparation. High potency. 3 doses with different size patches. May cause local reaction.
    - MOR-NORI class – mu-opioid as well as noradrenergic effects -> tapentadol

Predicting Outcome – Bruce Biccard

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

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

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

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

Outcomes in Anaesthesia in South Africa – Christina Lundgren

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

Enhanced Recovery – Wilson & Mark Daugherty (UK)

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

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

End of Refresher Day 2