PACSA 2012 – Ross's very rough notes

Supplementary to the congress abstract book

Friday 2 November 2012

Anaesthetic Neurotoxivity in Children – Randolph Flick – Mayo Clinic

- Ikonomidou et al, Science 283:70, 1999.
- Wilder paper is the most widely cited paper in the field in the last decade.
- Millions of children are exposed to anaesthesia each year
- Concern for developmental apoptosis
- Synaptogenic period – 3rd trimester to 4 years
- GABA and NMDA agents
- Jevtovic-Todorovic et al
- Surgical stimulation is not protective (recent work)
- Is any of this clinically significant in children? Difficult to study prospectively!
- Research now focussing on nonhuman primates. Same findings in monkeys as in rats; both pathological and behavioural
- Observational studies in children – all retrospective, all significantly flawed.
- Association not causation!
- Twin study – "No evidence of a Causal Relationship" – no difference in unexposed twin.
- Hernia study – 2-fold higher frequency of development or behaviour. No control for patients who had other procedures.
- DiMaggio C study 2 – looks like a dose-response effect
- Hanson (Netherlands) – no difference in large national cohort (2500 hernia repairs vs. 5% sample of population). No control for patients who had other procedures; not all patients had a general anaesthetic.
- Five published Mayo studies – all from an old cohort (1976-1982)
- Wilder study (Anaesthesiology 110:796, 2009): 1 exposure, no difference. Increasing exposures show increasing risk of learning disability
- Are kids who's moms had an epidural smarter? -> Robust testing shows the answer is NO. Unsure where the confounders crept in
- Co-morbidities cause learning disabilities; illness exposes kids to doctors who may then diagnose previously unrecognised LDs
- Co-morbidity, however, does not seem to be the driver for the LDs associated with anaesthesia/surgery
- Pattern carried over in speech and ADHD disabilities; not in emotional learning abilities.
- Worthwhile reading – Flick & Warner, "A user's guide to interpreting observational studies of pediatric anesthetic neurotoxicity"
- Low hazard ratios (<3) are likely caused by confounders…but the scope of the problem if there is a true causation is huge.

- What do we tell parents? Very little evidence; we are doing research; no reason to be concerned based on what we do know.

Peri-op analgesia for the neonate and prem – Rebecca Grey – Red Cross War Memorial Children's Hospital, Cape Town

- Why are we nervous? Altered body composition, immature metabolism, technical challenges, questions regarding neuronal damage, off-label drug use.
- Off-label: common in paediatrics. Must have evidence to support choice of drug and dose, preferably in the international literature and guidelines
- Safety: Choose appropriate dose and placement of child (ICU/ward)
- Compassion
- Fulfil basic needs (feeding, temperature, sources of discomfort)
- Multimodal approach to analgesia
- Non-pharmacological: non-nutritional sucking; 2% glucose; swaddling; swaying; massage
- Regional: Neuraxial, truncal, specific nerve blocks. Advantages – less respiratory complications, good analgesia. Problems – risk of catastrophic neurological injury or toxicity
- Truncal blocks may be safer; catheter placement makes them very useful. US guidance makes these blocks much more effective.
- In a resource-limited setting, regional anaesthesia is EXTREMELY useful. Beware – areas that aren't covered by the block. Add simple analgesics.
- Systemic analgesia – stepwise and tailored.
- Simple (paracetamol etc) -> Opiates -> opiate-sparing agents (a2/ketamine/gabapentin)
- Only oral NSAIDS available in this age group (rectal doses too high)
- Opiates: TITRATION! Prem/neonate needs at least a HCU setting
- Oral (valeron), intramuscular (not recommended); IV (morphine infusion)
- Beware of withdrawal
- Ketamine – 0.25-0.5mg.kg. Higher doses assosciated with apnoea
- Clonidine – 1mcg/kg PO 8 hourly
- Gabapentin – 2mg/kg TDS (up to 70mg/kg/day!)

Fluid management in the paediatric patient – Karmen Kemp – Red Cross War Memorial Children's Hospital, Cape Town

- Paediatric brain has higher intracellular sodium concentration and is much more sensitive to changes in tonicity
- Safety guidelines should be widely propagated; hypotonic solutions should be removed from perioperative wards
- High incidence of suspicion and alertness.
- Glucose – traditionally 5% was used in very hypotonic solutions. Patients regularly became hyperglycaemic. Should be reserved for patients at high risk of hypoglycaemia. Beware of patients with extensive regional blocks – no surgical stress, so they become hypoglycaemic.

Beware TPN pumps – decrease rate based on regular insulin checks (can run at 2/3 of original rate)

- Cf. notes for fluid composition of neonates and renal maturation.
- Maintenance about 3ml/kg/hr on day one, increasing to 6ml/kg/hr over the first few days.
- Low threshold for using colloids like SHS in neonates
- Regular electrolyte, haemoglobin and glucose checks if giving large volumes
- Which colloid? Albumin very expensive. Data exists for the safe use of 3rd generation starches in children; caution in those at very high risk of coagulopathy. (Sumpelmann et al, Paediatric Anaesthesia; Liet et al, Pediatr Crit Care Med 2003). None of these studies used doses more than 20ml/kg however, and cardiac patients were excluded.
- SHS is approximately 20x more expensive than Voluven in SA State Sector
- Haas et al – TEGs – MA decreased in gelatin, MA and a-Angle decreased in HES
- Fluid shift: Impossible to measure, makes estimation of fluid requirements very difficult. We can't see what is going on at the level of the endothelial glycocalyx! Mechanical stress, endotoxin, ischaemia reperfusion injury, inflammation, glycocalyx destruction by fluid overload.
- Practice *conservative, demand driven* fluid administration
- See "The concept of the glycocalyx" by Brettner, Chappell and Jacob
- Fluid -> ANP release -> cGMP -> glycocalyx breakdown
- The future looks like it will feature a lot more of the word "Glycocalyx"…
- Further reading –
    - Cote Ch. 8 "Fluid management" in A Practice of Anesthesia for Infants and Children.
    - Bailey 2010 – Perioperative crystalloid and colloid management in children: Where are we and how did we get there?

Peri-operative Ventilation of Neonates – Maria Reyneke, University of the Orange Free State, Bloemfontein

- Why are they so vulnerable? Impaired gas exchange, high metabolic rate, shunt physiology, poor lung mechanics
- Effects of anaesthesia – post-op apnea, periodic breathing, reduced FRC, VQ mismatch, worsened Vt:Vd, worsened compliance and more resistance, more prone to laryngospasm
- Ventiator-assosciated lung injury (VALI) has multiple causative mechanisms – volutrauma, barotrauma, atelectrauma, biotrauma (TRALI; oxygen toxiticy -> fibrosis)
- How to prevent VALI:
    - Avoid hypoxia and hypercarbia – maintain oxygenation (Pmean and FiO2) and ventilation (Alveolar MV and frequency). Low volume, high frequency ventilation is usually less pathological to the lung
    - Lung-protective ventilation (Safe window between closing volume and lower inflection point on the volume-pressure curve)
    - Recruitment manoeuvres should take the pressure to the higher inflection point on the v-p curve

    - Adapt to specific pathophysiology and surgical requirements (eg. heterogenous ventilation, higher or lower PEEP, lower respiratory rates and permissive hypercarbia)
    - Focus on lung mechanics
        - Monitor capnography and blood gasses frequently
        - Extreme caution with laparoscopy (IAP<6mmHg, VCV, be alert for ETT displacement into RMB)
        - Choose mode carefully – decide whether PCV or VCV will suit your patient's pathology better. Beware PCV when the IAP is changing frequently. Newer machines have much more sensitive control of VCV; newer modes (such as pressure-controlled volume-guaranteed ventilation) have distinct advantages.
- Decide…then monitor, monitor, monitor! SpO2, EtCO2, ABGs, CXRs, etc.
- Ventilator problems: Mnemonic DOPES (Displacement, Obstruction, Pneumothorax, Equipment failure, Splinting of the diaphragm) {S added by Ross}
- Extubation should be a team decision with surgeon, ICU, etc. Patient must be stable, normothermic, normal electrolytes, no inotropes, etc. Beware the known difficult airway. Prems must have prophylaxis against apnoea and be monitored (HCU!).
- Transport on vent support: Prevent accidental extubation; maintain temperature; take emergency equipment; sort out sedation and pain control; take t-piece/neopuff/neonatal BVMR. Extreme caution if <1500g
- Surgery in NICU:
    - Challenging, uncomfortable
    - Paralysis and high dose opiate
    - TIVA (no vapouriser)
    - Keep the lung open!
    - Consider HFOV
- HFOV
    - Transport almost impossible
    - Akin to open lung strategy with ling protection
    - Very small Vt at very high frequency
    - Trend towards reduced mortality and BPD (literature inconclusive)
    - Decouples oxygenation (controlled by FiO2 and Pmean) and ventilation (controlled by pressure difference and frequency)
    - Alveolar pressure just above the closing pressure
    - Bias flow of warm humidified air at 20 litres/min at the frequency of oscillation (3-10Hz; 180-600/min!)
    - Cycle volume = tidal volume delivered = amplitude
    - Decreasing the frequency increases the amplitude, thus decreasing PaCO2
    - Chest wiggle factor – clinically useful measure. Visible movement of the patient's chest while on the oscillator.
    - "HFOV = CPAP with a wiggle"
    - Practically:
        - Never disconnect the circle!
        - Sedation and analgesia important

- Inhalational anaesthesia is not possible – use TIVA
- Consider the intravascular volume carefully
- Increase bias flow to 40l/min if suctioning has to be performed
- Tolerate hypercarbia if there is no contraindication

Oesophageal Atresia & Tracheoesophageal Fistula –Priorities in Resource-Limited Environments – Andrew Levin, University of Stellenbosch & Tygerberg Academic Hospital, Cape Town

- Lots of anatomical variants (Sub-type 3b has 20 sub-subtypes)
- Embryology probably of little importance to the busy clinician
- Incidence between 1:1500 and 1:4500 – TBH sees about 8 a year
- VATER and VACTERL association – cardiac anomalies are the very big problem
- Many other associated congenital abnormalities (CHARGE, etc)
- Presentation – antenatal (polyhydramnios; enzyme tests), at birth (scaphoid, gasless abdomen), frothing, feeding problems, coughing, cyanosis, etc
- Routine echo and renal ultrasound should be a standard of care
- Prognosis depends on anomaly, genetic background, and cardiac morbidity
- Initial management – upright position, replogle tube on suction, antibiotics, urgent surgery
- Surgical approach – excision and anastomosis over a *very well fixed* NGT
- Interposition if the defect is very long
- Thoracotomy on the opposite side to the aortic arch -> extrapleural dissection -> azygous divided -> Divison of trachea and oesophagus -> Repair -> Closure
- Have blood in theatre
- Thoracoscopy is becoming a reality
- Surgical complications – anastomotic leak, anastomotic stricture, recurrent fistula)
- Anaesthetic management:
    - Difficult ventilation (on induction 7%, during maintenance 15%)
    - Positive pressure support is needed if the lungs are compromised
    - Gentle bagging will usually ventilate the lungs
    - Stomach inflation will aggravate ventilation problems
    - During thoracotomy, IPPV is essential
    - Lots of shunt during thoracotomy
    - Moving the tube may help occlude the fistula – this clearly has problems if the fistula is at the level of the carina, which makes a trifurcation in which the fistula is indistinguishable from the main bronchi
    - USE BRONCHOSCOPY TO IDENTIFY THE FISTULA...*if* you have a bronchoscope suitable for neonates.
    - Use a cuffed endotracheal tube
    - Consider using a Fogarty catheter down the fistula to occlude it. This also facilitates location of the fistula by palpation. This prevents extensive dissection in the tracheoesophageal grove, preventing recurrent laryngeal nerve injury.
- If no flexible fiberoscope:
    - A urology cystoscope with a 30 degree lens can be used

    - Must be very careful – rigid, sharp device!
- Main priority is control of ventilation

Paediatric Massive Transfusion Protocols – Graeme Knottenbelt, Starship Hospital, Auckland

- Situation of crisis – physiology, leadership, systems all challenged
- J Trauma 2006 Malone et al – first comprehensive adult MTP
- Transfusion, June 2012 – first comprehensive paediatric MTP
- Dente J Trauma – MTP decreased mortality
- O'Keefe 2008 – MTP reduced costs
- Why should there be a benefit after MTP implementation? Crisis resource management, systems improvement, evidence based practice
- Focus on "Human Factors" – non-technical skills (eg. communications)
- Enticott JC – Review on decision support for massive transfusion
- Change from a reactive to a proactive system of blood provision
- What we know about massive transfusion
    - Acidosis
    - Hypothermia
    - Coagulopathy
    - Crystalloids avoided
    - 1:1:1 RBC:FFP:platelets
    - MTP essential
    - Complications of transfusions
- SHOT – Serious Hazards of Transfusion in Children (Ped Anaes 2011)
- What we're unsure about…
    - Fibrinogen (BJA review recently)
    - rFVIIa (Drug in search of evidence of benefit… Cochrane: Indications should be restricted to clinical trials)
    - Tranexamic acid (Cheap, available, minimal complications)
    - Goal-directed therapy using TEG/ROTEM – absence of evidence (so far)
    - Paediatric management (most information is taken from adult studies)
- See ADHP Paediatric Massive Transfusion Protocol; Royal Children's Hospital (Queensland) MTP; Princess Margaret Hospital (Perth) MTP; various UK protocols (Birmingham, Bristol); Seattle Children's Hospital; Boston Children's Hospital
- Create an MTP for your hospital!
    - Create
    - Institute
    - Audit
    - Research
    - Contribute to consensus
- "Whatever you do, always give 100%... unless you're donating blood."