Contents

Re-Examining RSI 2
Which Video Laryngoscope 4
Tracheal Extubation: Strategies for Managing Difficult Airway in Non-Obstetrics 6
Muscle relaxation in laparoscopic surgery: How deep is deep enough? 12
Preoperative iron deficiency anaemia - review with a regional flavour. 14
Peri-operative nutrition 17
Modern metal implant toxicity 20
Anaphylactic shock under anaesthesia: a reappraisal of the pathophysiology and management 21
Pulmonary hypertension: an overview for the “non-cardiac” anaesthetist 23
Strategies to reduce emergence agitation in children 25
Intraoperative awareness and general anaesthesia for caesarean delivery: A fresh look at an ongoing problem 28
Goal-directed transthoracic echocardiography – a translational education program 33
Pain management for trauma: Time to embrace regional anaesthesia? 35
The role of regional anaesthesia in clavicle fracture surgery 38
Re-Examining RSI

Intro
- classic = preoxy, cricoid, thio & sux, no BMV, intubation, inflate cuff, EtCO2 then remove cricoid
- proposed advantages:
  - ↓aspiration
  - if unable to intubate then short acting induction drugs mean spont ventilation should soon return
- disadvs:
  - ↑risk of awareness - as doses predetermined
  - haemodynamic instability due to non titration of drugs
  - sux disadvantages & conditions where unable to use eg spinal cord injury, burns, cholinesterase def, penetrating eye injury)

Propofol vs Thiopentone
- ↓bp:
  - 5mg/kg thiopentone may cause less hypotension than 2.5mg/kg propofol
  - although 5mg/kg thio vs 2mg/kg propofol equivocal
- airway grade:
  - satisfactory intubation conditions with either as long as relaxant used
  - propofol better at blunting airway reflexes & improving laryngoscopy blade in absence of relaxant
- speed of onset:
  - thio & propofol have similar time to loss of consciousness
  - time to BIS <50 quicker in thio 52sec vs 65sec
  - BIS levels lower with propofol than thio maybe suggesting ↓risk of awareness with propofol
- cost:
  - propofol x13 cheaper than thio ($9.73 vs $0.76)
- obstetrics:
  - growing evidence prop safe in obstetrics
  - common drug error of thio vs antibiotics ∴ NAP5 recommended prop for induction in obstetrics

Sux vs Rocuronium
- benefits to avoiding sux:
  - muscle pain - 50% occurrence
  - allergy - 1:5.500
  - MH
  - atypical cholinesterases - 0.01%
- cochrane r/v:
  - sux = excellent intubating conditions more reliably than roc
  - roc 1.2mg/kg does provide acceptable views
  - recommend roc 2nd line only
- CICO:
  - sugammadex reduces disadv of roc in CICO although administration can be troublesome
  - 1mg/kg sux in modelling shows critical desat occurs prior to 50% twitch height recovery

Short acting Opioid to Supplement Induction Agent
- fentanyl if given with RSI induction drugs will not work at time of intubation
- fentanyl given prior to RSI induction drugs leads to risk of airway problems
- alfentanil benefits:
• same time to onset as induction drugs
• ↑ depth of anaesthesia without haemodynamic instability
• ↓ awareness - NOT proven
• ↑ intubating conditions - proven esp if using roc & not sux
• no evidence suggesting ↑ risk of brady cardia if alfentanil added to sux & prop

Conclusion
• suggested 1st choice = prop & sux
• addition of alfentanil could be considered esp if roc is used instead of sux
• if sux Cl’ed then roc is fine
Which Video Laryngoscope

Selection Criteria
- include:
  - type of blade
  - operating environment
  - portability - seize, power source, video display
  - reliability
  - robustness
  - integration
  - expense - capital, disposables, maintenance

Blades
- types of:
  - Macintosh:
    - advs: traditional view, adjuncts in difficult intubation, educational for trainees
    - trainee uses direct view while educator looks at video screen & provides direction
  - hyperangulated:
    - = blade of choice after failed DL with Mac blade & bougie
    - do not use R side of mouth but use midline point & shoot with styletted ETT
    - bougie use is difficult
    - can make easy intubations (gd I & II) actually more difficult
    - two types:
      - channelled - used as a guide to make this different intubating technique easier
      - non-channeled

Imaging
- some can record video for documentation, legal record, training
- robustness & reliability impt

Standardisation
- learning curve - everyone must know what is where
- remote anaesthesia should ideally use same system
  - portability is impt
- Recommend all hospitals should have a hyperangulated device in addition to standard Mac blad Vls

Cost
- should factor in cost of disposables over time eg
  - single use entire unit
  - single use blades only
  - entirely reusable unit

Pre-Hospital
- very different environments:
  - needs to work in direct sunlight
  - not fog in winter
  - work in sub zero temp
  - be waterproof in rain
  - light, quick start up time
  - tolerate vibrations from flying devices
• rapid start up
• if electronics fail - can still function as DL device
• suitable for paeds & adults
• quick learning curve impt - limited exposure of personnel ⇒ common to use Mac blade devices
• recommend method: look in mouth ⇒ screen ⇒ mouth ⇒ screen four step approach to ↓ iatrogenic injury
• portable capnography vital

Conclusions
• dont forget awake techniques
• must document what type of device used & grade of view obtained:
  • POGO scoring system for VL:
    - =percentage of glottic opening score
    - replaces grade I & 2 of Cormache & Lehane
    - 100% = see entire glottic opening from ant commissure to post cartilages

• Fremantle score for VL:
  - = stand alone score for using VL
  - can be easy to get a good view, but difficult to pass tube
  - some VLs designed not to get a full view of glottic opening (esp if channeled devices)
  - 3 parts to score:
    • Best view obtained =
      • Full
      • Partial
      • None
    • Ease of passing tube:
      • 1 = easy
      • 2 = modified eg had to use adjuncts, or alternate action
      • 3 = unachievable eg unable to pass ET or had to abandon technique
  • Name of device & blade used

Figure 5. The Fremantle score

<table>
<thead>
<tr>
<th>View:</th>
<th>Full</th>
<th>Partial</th>
<th>None</th>
</tr>
</thead>
</table>

Blue Book 2015 Summary - 5
Tracheal Extubation: Strategies for Managing Difficult Airway in Non-Obstetrics

- ASA closed claims on intubation - showed 27% decrease from 80s to 90s
- Extubation claims have not changed - extubation complications may now exceed intubation
- ↑ morbidity at extubation
- ICU extubation complications = 2-25% although may be related to other factors eg
  - duration of vent & prolonged sedation
  - severity of disease
  - critical illness myopathy
  - CVS & neuro complications

Predicting Which Patients are At Risk for Extubation

- 3 main predictor classifications:
  - patho-physiological predictors
  - anatomical predictors
  - Airway risk factors from intubation

Patho-Phys Factors

<table>
<thead>
<tr>
<th>Table 1. Conventional extubation parameters (modified from Francon D et al14.)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Respiratory criteria</strong></td>
</tr>
<tr>
<td>Patent airway, spontaneous and regular breathing, no inspiratory chest wall retraction</td>
</tr>
<tr>
<td>Tidal volume &gt;5-8 ml.kg⁻¹</td>
</tr>
<tr>
<td>Minute ventilation &lt;10 l.min⁻¹</td>
</tr>
<tr>
<td>Respiratory frequency 12-25breaths.min⁻¹</td>
</tr>
<tr>
<td>Negative inspiratory pressure &gt;-20cmH₂O against a closed glottis</td>
</tr>
<tr>
<td>Swallowing/cough reflexes intact</td>
</tr>
<tr>
<td><strong>Gas exchange</strong></td>
</tr>
<tr>
<td>Spo2 &gt;95 per cent with Fio2 &lt;0.5 and Peep &lt;5cmH₂O</td>
</tr>
<tr>
<td>PaO₂ &gt;60mmHg</td>
</tr>
<tr>
<td>Or values appropriate for the individual patient</td>
</tr>
<tr>
<td><strong>Neuromuscular</strong></td>
</tr>
<tr>
<td>Awake, following simple order commands, obtain a verbal response</td>
</tr>
<tr>
<td>Reversal of neuromuscular blocking agent (T₄/T₁ &gt;0.9)†</td>
</tr>
<tr>
<td><strong>Cardiovascular</strong></td>
</tr>
<tr>
<td>Haemodynamic stability (blood pressure and pulse rate ±20 per cent of preintubation levels)</td>
</tr>
<tr>
<td>No vasopressor or inotropic support</td>
</tr>
<tr>
<td><strong>General</strong></td>
</tr>
<tr>
<td>Core temperature ≥36 degrees Celsius</td>
</tr>
<tr>
<td>Normoglycaemia</td>
</tr>
<tr>
<td>Good analgesia (VAS ≤3)*</td>
</tr>
<tr>
<td>Absence of anaesthetic or surgical complications</td>
</tr>
</tbody>
</table>

*VAS; visual analogue score
†T₄/T₁; train-of-four ratio
Anatomical Predictors
• can classify difficult airway anatomy using a 3 column approach
• allows targeted plans based on location of problem”

Posterior Problem
• eg
  • Cx spine esp occipito-atlanto-axial complex
  • Anky spond
• may be fine to extubate if risk of airway otherwise deemed low:
  • place LMA behind ETT while ETT still in situ
  • remove ETT ⇒ LMA as airway conduit

Anterior Problem
• eg
  • retrognathia
  • mandible, tongue & submandibular tissues
• may be fine to extubate if risk of airway otherwise deemed low:
  • place LMA behind ETT while ETT still in situ
  • remove ETT ⇒ LMA as airway conduit

Middle Column Problem
• e.g.:
  • = airway passage
  • influences:
    • external ie ant & post
    • internal eg loss of pharyngeal mm tone, mucosal oedema, airway tumours delayed extubation recommended
• 2 situations:
  • difficult airway with long operation requiring large doses of opioids:
    • risk of depression of genioglossus & pharyngeal mm tone ⇒ loss of airway reflexes
    • tone loss in conjunction with other column problem ⇒ dangerous extubation
  • anaesthetic/surg factors eg prolonged head down, massive fluid resus, soft tissue trauma, airway mucosa swelling (↑hydrostatic pressure, ↑capillary permeability, ↓lymph drainage)
    • anatomical distortion of middle column ⇒ delay extubation

Airway risk factors from intubation
• From DAS guidelines:
  • Known difficult airway
  • Airway deterioration (trauma, oedema, bleeding)
  • restricted airway access
  • obesity/OSA
  • Aspiration risk

Physical Assessment of Airway
• should consider 1 or all of these:
  • DL
  • cuff leak test
  • fibreoptic visualisation
Direct Laryngoscopy
- may provide useful info on supra-glottic & glottic structure & function
- if head & neck surgery problems to consider:
  • sub mandibular infection
  • thermal injury
  • air mucosal swelling
- problems with DL:
  • DL grading is a snap shot of airway - not dynamic
  • ETT in situ may make grade look better than when it is removed
  • ETT may be compressing mucosal swelling

Cuff Leak Test
- procedure:
  • carefully suck sub glottic & supraglottic areas
  • cuff deflated
  • tube lumen is occluded
  • Ax of leak around tube:
    - measure volume quantitatively or just listen for leak
    - resp effort by spont ventilation
    - or 20mmHg through ETT
    - leak measured by difference between:
      • insp tidal volume - CUFF UP
      • exp tidal volume - CUFF DOWN
    ⇦ if leak volume is <110ml (ie 10-20%) then test +ve = fail
- Test = 80% PPV for for failed extubation in ICU 24hrs prior to extubation after extended ICU intubation
- That said relying solely on cuff leak test may ⇒ unnecessary extra days of ventilation
- Rx: fluid management & steroids may help in suitable cases

Fibreoptic
- fibreoptic bronchoscope - can assess anatomy & function at many levels
- nasoendoscopy -
  • assess supraglottic swelling & oedema
  • interpretation is subjective
  • can be useful if borderline cuff leak test
  • good positioning & anti-sialogue impt
  • ETT may prevent adequate Ax

Extubation Plan
- 3 major options:
  • use of extraglottic device
  • airway exchange catheter
  • Cannula cricothyroidotomy

Extraglottic Device ie LMA
- device used as an airway catheter
- flex bronch advanced through device to inspect laryngeal anatomy & function
  ⇦ good for vocal cord Ax
- can place Aintree catheter through bronch then rail road ETT over catheter if reintubation is required
• method may incur risk esp if periglottic oedema from middle column ⇒ difficult placement of extra glottic device

**Airway Exchange Catheter**

- = small, hollow semi rigid plastic. radio opaque with wire
- design:
  - ↑safety while changing an ETT
  - maintain oxygenation
  - allow guide to reintubation if needed
- use has been well demonstrated in studies:
  - ↓hypoxaemia
  - ↓bradycardia with hypotension
  - ↓oesophageal intubations
- size of catheters - 11, 14, 19FG
  - ↑largest (19) only tolerated in 50% of pts
- method:
  - Decide on AEC size:
    - for exchange: select largest which fits in ETT
    - for placeholder: select small size (tolerated better)
  - AEC placed through ETT to tip of ETT only
  - remove ETT over AEC with care not to deepen its position in the trachea
  - mark on AEC depth position at teeth - with permanent pen!!
  - use O2 via FM
    - ↓O2 via lumen of AEC is NOT recommended due to pressure risk
  - fasten AEC to pts forehead
  - occlude AEC tip and label to prevent it being used as a feeding tube!!!
- length required:
  - 1 study maxfax/neck surgery- 10.4 hours (range 4-24hrs) = 4 of 36pts needed reintubation
  - peri-glottic oedema most common in first 45mins but reported up to 8hrs post extubation
  - ↓recommend up to 12hrs AEC if CVS/resp/neuro compromise
- advs of AEC:
  - ↑ed first pass success rate at reintubation
  - ↓complications related to prolonged intubation
  - method for continuous oxygenation
  - monitor CO2
  - jet ventilation
  - ↓CVS instability during re-intubation
- disadvs:
  - inadvertent removal
  - unable to advance an ETT over the AEC in airway oedema
  - barotrauma risk if jet ventilation used
  - lung abscess
  - direct airway trauma
  - lung laceration

**Cannula Cricothyroidotomy**

- identification can be difficult - although easy with ultrasound
- cannula placement may be possible but:
  - difficult to secure in place
  - dislodgement an issue
Monitoring following Extubation

- early signs of extubation failure are impt:
  - early signs:
    - sore throat
    - hoarse/weak voice
    - poor cough
    - poor swallow or drooling
  - late signs:
    - stridor
    - orthopnea
- NAP 4 - use of capnography in recovery would have led to earlier detection of airway obstruction in several cases

Suggested Extubation Strategy

see diagram below
**Conclusion**
- staged strategies for identification & management may improve safety
- early warning signs for problems vital
- AECs can be very useful but not fail safe
Muscle relaxation in laparoscopic surgery: How deep is deep enough?

Intro:
Anaesthesia is “balance” of hypnosis, analgesia and muscle relaxation.
Most anaesthetists aim for moderate depth of block of 1-4 twitches on TOF. Most agree some degree of paralysis necessary for laparoscopic surgery, depth largely unknown. Recent studies have suggested only deep neuromuscular block achieves best operating conditions during laparoscopic surgery.

Definitions:
- Intense block - no twitch in TOF nor PTC. Depth of intense block cannot be measured.
- Deep block - ≥1 twitch in PTC, but no twitch on TOF
- Moderate block/surgical depth of block - 1-3 twitches on TOF
- Shallow block - >3 twitches on TOF - seamlessly progress into recovery.

Conflicting Recent reviews:
Maden et al. concluded neuromuscular block per se marginally improved operating conditions, and that deep block was superior to moderate block.
Kopman and Naguib found little or no evidence for improved surgical conditions under deep neuromuscular blockade.

If only analyse studies meeting below criteria (<5%) difference largely resolves.
Criteria:
- distinguish between studies comparing no vs moderate or deep block, and those comparing different depths of block with use no block as control
- clear description of outcome parameters
- whether neuromuscular block monitored
- randomised allocation to treatment arms, and observer blinding

Just looking at studies meeting above criteria:

No block vs deep block:
3 studies: small numbers of lap choles or lap gynae procedures:

- Deep muscle relaxation improved surgical space at a given intra-abdominal pressure, or allowed a reduction in IAP without impairing operating conditions.

Deep block resulted in less undesirable effects such as sudden movement or coughing.

Moderate/shallow vs deep block:
3 studies: lap choles, lap prostatectomy or nephrectomy, lap hysterectomies:

- All studies found better operating conditions under deep vs moderate or shallow block.

Difference small, but may be important in high risk patients (bariatric, signify adhesion) or procedures (robotic surgery) where even small movement may cause catastrophic consequences.

Effect of IAP on post-operative patient outcomes: (not investigated in above studies)
High IAP - adverse haemodynamic effects, impaired organ perfusion, via either macro- or micro-vascular changes (latter possible resulting in organ dysfunction).
High IAP linked to post-laparoscopy pain.
Low IAP found to significantly reduce problems such as renal and liver dysfunction, splanchnic hypoxia, cardiac dysfunction, haemodynamic suppression, autonomic and general inflammatory stress response.

Discussion:
Recognised that nerve stimulation at adductor pollicis brevis muscle may reflect pharyngeal muscle function, but is poorer descriptor of diaphragmatic muscle relaxation (due difference in NMDA sensitivity between different muscle groups). Recovery of adductor pollicis brevis may take significantly longer than diaphragm
following full paralysis, hence diaphragmatic contractions may impair surgical working conditions at much deeper level of neuromuscular block than usually maintained.
In order to achieve adequate paralysis of abdo wall and diaphragm for lap surgery, many anaesthetists combine modest/shallow block with deep anaesthesia to facilitate NMDA reversal at end of surgery. In this case, ideally combine with volatile agents (known muscle relaxant properties). Depth of anaesthesia however not sufficiently predictive to maintain satisfactory surgical conditions due to inter-individual variation.

**Conclusions:**
- good evidence for beneficial effects of deep muscle relaxation on surgical working conditions, however ideal depth not yet defined.
- Recent studies show small but significant benefit for deep vs moderate/shallow block.
- Continuous monitoring and maintained deep-to-moderate block appears superior to one-off dose of NMBA with subsequent recovery.
- Deep block may be advisable for difficult cases or when sudden moment could be catastrophic. Impact of deep block on post-op patient outcomes less investigated.
Preoperative iron deficiency anaemia - review with a regional flavour.

Summary of epidemiology and pathophysiology of IDA, therapeutic options, rationale for process of haematinic optimisation in peri-op and maternity setting at Launceston General Hospital (LGH), and prospects for future in promoting system for appropriate patient blood management in obstetrics and surgery at LGH.

PBM (patient blood management):
Primary goal to improve patients clinical outcomes (also minimises or avoids blood transfusion). RBC transfusion in peri-op period associated with increased mortality (debate whether cause is transfusion per se or series of physiological insults that lead to transfusion) and morbidity (incr post-op infections and length of hospital stay; TRIM (transfusion-associated immune modulation) describes range of immune changes that occur due to transfusion). Blood transfusion is expensive. Paradigm shift away from blood transfusion in Australia - PBM programs implement peri-op strategies that are built on 3 conceptual ‘pillars’ promoting approach aimed at:
• optimising red cell mass,
• minimising blood loss and bleeding, and
• harnessing and optimising physiological reserve of anaemia.

Anaemia and Iron Deficiency:
Anaemia: men <130g/L; non-pregnant women <120g/L, pregnant (>20/40) women <110g/L

Anaemia is independent RF for morbidity, mortality, hospitalisation and decreased QOL. Pre-op anaemia is independent RF for post-op morbidity and mortality, and associated with increased likelihood of blood transfusion.

IDA most common cause of anaemia in patients presenting for major elective surgery. Diabetes, cardiac failure, cancer and chronic renal impairment are associated with greater risk of concurrent anaemia that are other co-morbidities. IDA is late feature of iron deficiency.

Assessing iron deficiency:
FBC (Hb and blood film), as well as indices such as MCV, MCH and red cell count to diagnoses microcytic anaemia. In areas where haemoglobinopathies are prevalent (microcytosis), iron studies are surrogate marker for IDA.

Degree of iron deficiency classified according to ferritin level:
• severe: serum ferritin <15-20microg/L
• moderate: ferritin 30-50microg/L
• mild: ferritin 50-100microg/L.

Low transferrin saturation (<15-20%) also indicates iron deficiency. Ferritin is a APP, therefore raised in inflammation/infection. Should check CRP if anaemia with raised ferritin to exclude reactive cause.

Hepcidin (plasm protein) also increased in inflammation. Hepcidin impairs absorption of dietary iron and inhibits use of stored iron, and can lead to iron sequestration disorder in which iron storage may be normal (as per ferritin level0, but the transferrin saturation is low. These patients not iron deficient per se, but unable to access iron stores appropriately.

Iron Therapy:
Oral - commonly fails due non-compliance (due s.e. - GI disturbance). Usually ferrous salts - low and variable absorption rates, limited by ingestion of certain foods or mucosal luminal damage, and need acidic medium for
optimum absorption. Newer Ferric compounds avoid these obstacles, but generally less soluble with poor bioavailability.

Usual oral ferrous sulphate dose for Rx of iron deficiency is 80mg/day of elemental iron.

IV - First-generation preparations (dextran complexes) had serious se (incl anaphylaxis) limiting use. Iron sucrose safer but limit by max weekly dose. Recent formulations (iron polymaltose and carboxymaltose complexes) are better tolerated and can be used for rapid repletion of iron stores.

**Evidence:**

Iron therapy in obstetrics:


Pre-op iron therapy:

Shown to be effective in treatment of IDA (most data from ortho patients). Timeframe for IV transfusion to be effective is unclear. Small ortho trial suggest NOF fixation, joint replant and spinal surgery, iron infusions less than 4 days pre-op decreased need for blood transfusion and decreased rates of wound infection.

Pre-op erythropoiesis-stimulating (ESA) agents:

Usually in combo with iron therapy.

No advantage over iron therapy alone in non-cardiac surgical patients (with respect to post-op Hb levels and blood transfusion).

Chronic kidney disease patients may benefit from pre-op ESA therapy.

Post-op iron therapy:

Erythropoietic response to blood loss blunted by systemic inflammatory response to surgery in post-op period. Hepcidin impairs absorption or oral iron, such that oral iron therapy fails to incr Hb conc. Contradictory evidence for IV iron therapy in treatment of post-op anaemia.

POC testing:

2010 LGH trial - POC Hb check on arrival to pre-assessment clinic. If low Hb - sent for urgent FBC and iron studies, if anaemic and iron deficient referred to day procedure unit for iron infusion.

Audit of LGD data:

<table>
<thead>
<tr>
<th></th>
<th>2010</th>
<th>2015</th>
</tr>
</thead>
<tbody>
<tr>
<td>pre-op anaemia prevalence</td>
<td>20%</td>
<td>18%</td>
</tr>
<tr>
<td>pre-op iron studies requested</td>
<td>2%</td>
<td>76%</td>
</tr>
<tr>
<td>blood transfusion rate</td>
<td>13%</td>
<td>6%</td>
</tr>
<tr>
<td>iron infusion in setting of IDA</td>
<td>N/A</td>
<td>9/14</td>
</tr>
</tbody>
</table>

Where to from here?

Pre-op identification and treatment of IDA as part of blood conservation strategy has potential to benefit patients undergoing major elective surgery, however changing medical practice in a sustainable manner is challenging.

Plans at LGH: PBM and haematinic optimisation

- expand transfusion nurse role
- emphasis on GP contact so patients screened for IDA in community to avoid surgical delays
- encourage major elective surgical patients to be seen by Anaest. at least 4/52 before surgery
- re-introduce POC testing in pre-op assessment unit
- automated checking of pre-op blood tests, with abnormal results flagged for intervention.
- revised clinical guidelines for interpretation of pre-op iron studies (incl transferrin saturation) to improve diagnosis.
• mandatory FBC and iron studies for all urgent cancer surgery at time surgeon books procedure, results directed to anaesthetic clinic.
• stocking and use of IV iron carboxymaltose in day procedure unit (more expensive)
Peri-operative nutrition

Optimisation of nutrition during peri-op period recognised for it potential contribution to improving clinical outcomes.

**Peri-op metabolism:**
Key metabolic processes of concern are hyperglycaemia and protein catabolism.

Surgical insult initiates post-op metabolic response by:
- hypothalamus to release GHRH and CRH,
- → pituitary gland to release GH and ACTH,
- → adrenal gland to release cortisol.

Cortisol promotes gluconeogenesis, hence causing hyperglycaemia.
GH regulates protein catabolism, increasing rate of lipolysis and inhibiting effects of insulin (further contributes to hyperglycaemia).
Usual negative feedback mechanism to regulate cortisol is ineffective post-op, hence ongoing cortisol production and increase in stress response.

Insulin and glucagon key hormones in regulating blood glucose.
Insulin produced by pancreas in response to increased blood glucose. Facilities movement glucose into muscle and adipose tissue, promotes conversion excess glucose into glycogen and TGs. Mitigates protein catabolism and lipolysis in muscle.
During surgery, anaesthesia interferes with insulin secretion. In addition, apparent insulin resistance of cells develops in post-op period, hence post-op hyperglycaemia in common occurrence.
Hyperglycaemia may lead to delayed wound healing and adverse outcomes (infection, sepsis and mortality).

Protein catabolism in muscle related to cortisol production (elevated in peri-op period due to stress response). End-products of muscle breakdown are amino acids, which are then metabolised into other energy substrates (glucose, fatty acids, ketones). Process occurs in all patient undergoing major surgery, greater extent in patients receiving suboptimal energy and protein.

CHO stored as liver glycogen is rapidly deleted within first 24hours if starvation. Beyond this time frame, shift in energy source, muscle stored broken down to meet energy demand of vital organs.

**Peri-op evaluation.**
Identifying “high-risk” patients usually involves assessment by anaesthetist focusing on cardiac and respiratory issues. No mandate from College to assess nutrition.
Two key risk factors that predispose to adverse peri-op outcomes are obesity and under-nutrition.

**Under-nutrition:**
Association with increased risk of surgical complications, related to impact on immune system and subsequent risk of infection.

Screening: often overlooked, despite acknowledged importance.
Many screening tools, NRS-2002 (Nutritional Risk Screening 2002) only one validated in surgical patients in predicting outcomes. Not as sensitive and specific in detecting malnutrition as malnutrition-screening tool (MST) - simple tool validated in a acute setting
- have you lost weight without trying?
- if yes, how much (kg)?
- have you been eating poorly because of a decreased appetite?

Nutrition intervention: implementation of tailored nutrition plan (consideration of current nutrition intake, expected surgical insult and subsequent metabolic demand).
Adequate optimisation in undernourished patient not always possible, depending on clinical situation (i.e. urgent cancer surgery). Benefit in as little as 5-7 days of pre-op nutritional support. Intention not to correct months/years of malnutrition, but to prepare patient to endure surgical insult from a metabolic perspective.

**Obesity:**
Associated with increased intra- and post-op complications. Use of strict low-calorie diets to facilitate rapid pre-op with loss becoming commonplace despite lack evidence - in attempt to reduce health cost associated with obesity. Limited evidence for very-low calorie diet pre-op in cancer surgery - primary concern is rapid loss of lean muscle mass. Obese patients often have poor muscle store anyway. Inadequate muscle store linked to suboptimal outcomes therefore should avoid.

**Peri-op Nutritional Management:**
1. **Parenteral nutrition in peri-op patient.**
   Debate surrounding PN and its role in peri-op and/or critically ill patients. Perceived increased risk of infection - data from old studies (poor glycemic control, poor asepsis, lipid formulations high in polyunsaturated fat (potentially pro-inflammatory)).

   Newer studies:
   - Caesar et al (2011), compared early initiation PN with late initiation supplement insufficient EN. Early PN showed increases in: risk of infection, no. patients requiring mechanical ventilation >2 days, duration renal replacement therapy, cost to healthcare. Limitations: large glucose load in first 24-48hr of intervention arm, premixed PN low dose protein:energy. Some patient received PN with no clinical indication.
   - Doig et al (2013), standard care vs early PN in patients with relative CI to EN. Found decr length mechanical ventilation in PN group; but no difference in mortality, infectious complications length of stay. Limitations: narrow subset of patients.
   - Harvey et al (2014) compared early initiation and optimisation of enteral vs parenteral nutrition and found no difference in mortality, infectious complications; and reduction hypoglycaemic episodes and N+V. Limitations: target calorie delivery not reached in majority of patients in either group.

   ASPEN (Am Society of Parenteral and Enteral Nutrition) recommend consideration of PN in malnourished patients undergoing GI surgery (commence pre-op for 5-7 days, continuing post-op in patients unable to tolerate EN)

2. **Immuno-nutrition/pharmaco-nutrition:**
   Enrichment of EN with specialised nutrients such as arginine, omega-3-fatty acids, glutamine and other antioxidants. Nutrients positively modulate immune response, influence gut function and attenuate inflammatory response post-op in GI cancer patients. Improvement in short-term outcomes (reduction in post-op infection and shorter length of hospital stay).

   ESPEN (European Society Parenteral and Enteral Nutrition) endorse use immune-modulating formula in elective upper GI surgical patients. Debate regarding efficacy of peri-op vs pre-op immno-nutrition - similar beneficial effect with just pre-op, therefore ?no need to continue post-op. Canadian guidelines recommend against use of arginine-enriched formations in critically ill patients due increased mortality in some studies.

3. **ERAS:**
   Implementation of best practice by means of multimodal surgical care; protocol accounts for various aspects of care in pre-, intra- and post-op phases of surgery, including anaesthetics, nutrition, mobility and fluid control:
• discourage use of mechanical bowel prep,
• adequate glycaemic control,
• reduction in fasting duration to 6 hours for solids and 2 hours for clear fluids,
• CHO loading 2 hours pre-op, and
• early introduction of EN incl oral nutrition supplements for at least 4 days post-op.
Nutritional components of ERAS have lead to improved glycemic control, lowered level of insulin resistance, more rapid return of bowel function and reduced length of stay.
Exceptions to ERAs include diabetics (don’t CHO lead) and those with delayed gastric emptying (need longer fasting period).

4. Glycemic control:
Limited data available to confirm benefit of peri-op glycemic control.
Consensus that conventional approach to glycaemic control (aim BSL <12mmol/L) better than tight control/intensive insulin therapy (due risk hypoglycaemia).

Post-Op Enteral Intake:
Historically, post-op nutrition delayed until patient passed flatus or a bowel motion - incr risk of malnutrition and delayed post-op recovery.
In 3 recent meta-analyses looking at EN within 48 hours of surgery, none reported increased incidence anastomotic leak; no adverse effects noted.
Hospitals slow to implement change in practice despite recent evidence.

Collaborative Approach to Peri-op Nutrition:
Martindale suggests ‘prehabilitation’ period for patients undergoing elective surgical procedures:
• early nutritional assessment to identify at-risk patients,
• an exercise physiologist to establish adequate physical activity in order to attenuate any muscle wasting,
• assessment and optimisation of glycemic control and
• smoking cessation program 30 days prior to surgery.

Identify ‘at-risk” patients - undernourished or obese.
5-7 days nutritional optimisation pre-surgically if able in malnourished.
Consider pre-op immune-enhanced formulas in patients undergoing GI surgery for malignancy.
Trial En when viable in patent requiring specialised nutrition support post-op. Limit PN to patients unable to have EN.
Multidisciplinary approach recommended.
Modern metal implant toxicity

-The clinical concern is corrosion (electrochemical degredation), structural failure or reduced implant integrity may result in increased local and systemic concentrations of metals leading to patient morbidity.

-How? The various ions, Na, Cl, Mg, HCO3 in water may undergo reduction/oxidation reactions
-titanium (most commonly used) is relatively inert
-corrosion rate of <0.02mm/year

What? Geno-toxicity and carcinogenicity
-evidence that chronic exposure to metals including titanium may effect every organ system via hypersensitivity reactions, granuloma formation, accumulation leading to end organ dysfunction

-there is no evidence that elevated serum levels of these metals directly influence either inhalational or IV anaesthesia agents, it is clear that PK and PD can be affected by end organ impairment as a result of metal toxicity (obviously!!)

-there is a growing number of patients presenting for joint revision with local or systemic toxicity.
-emerging area of concern
Anaphylactic shock under anaesthesia: a reappraisal of the pathophysiology and management

Types of anaphylactic reactions ACID
Type I Anaphylaxis
Type II Cytotoxic-mediated
Type III Immune-complex
Type IV Delayed hypersensitivity

Pathophysiology: Type 1 Hypersensitivity reaction: requires prior exposure and sensitization, the important cells are basophils (in the blood) and mast cells (in the tissues), both express high affinity receptors (FcRI) on their surfaces, these bind IgE antibody which also binds allergen and then cross-link with another similar cell causing degranulation and release of mediators of anaphylaxis
- this is not an all or nothing phenomenon reflected in the variability of clinical severity
- the grade of anaphylaxis is measured by the mast cell tryptase
- the number of FcRI is increased by higher amounts of serum IgE
- pholcodine-containing cough suppressant shave been shown to increase levels of circulating IgE

Unusual characteristics of NMBD anaphylactic reactions is that it commonly occurs on first exposure and the molecules are small. This suggests that an environmental exposure and sensitization to substituted ammonia groups in commonly used chemicals such as pholcodine or those used by hairdressers.
- because the Ab in the sensitized individual have not been specifically raised to a single NMBD but merely recognize the substituted ammonia groups, one would expect all muscle relaxants to behave similarly which is not the case. Therefore you cannot assume that someone who has a reaction to benzylisoquinoline will NOT have a reaction to a steroidal NMBD

Mediatory release and termination of response
FIRST GROUP: pre-synthesised mediators include histamine, serotonin and various proteases including tryptase, carboxypeptidase
- resulting in Cardiovascular, respiratory and skin changes

- SECOND GROUP: phospholipid metabolites generated as a consequence of the release of phospholipase A2 including platelet-activating factor and the eicosanoids causing bronchospasm and angiodema.

- THIRD GROUP: cytokines and chemokines, involves up regulation of gene expression and result in cell signaling and chemotaxis.

- there is variation in physiological responses but even the most severe clinical reactions under anaesthesia do not last longer than 20 minutes

- what terminates the reaction?
- negative intracellular signaling
- endocytosis of activated Ag-Ab-FcRI complex
-the FcRI are bound to ubiquitins and some are recycled after disassociation

There does not appear to be a role for giving sugammadex in rocuronium anaphylaxis as a means of disengaging the Ag (roc) from FcRI. The reaction has already occurred within minutes therefore clinically sugammadex is being given too far down the physiological line.

Rapid desensitization can be achieved with escalating doses of the agent over 3-4 hours. ?how. But re-sensitization will recur if drug is not being administered after 1 half life.

The haemodynamic response
- histamine is the most important mediator
- the most common manifestation occurring in 205 out of 227 patients was CV depression characterized by profound hypotension, sinus tachycardia, loss into interstitial space of up to 35% of blood volume and low cardiac filling pressures. –note that severe bronchospasm was rare and only really seen in patients with other conditions that predispose to chronic airway obstruction such as smoking or asthma
- hypotension occurs not only due to a fall in SVR but also because of a failure of VR due to both interstitial losses and sequestration of blood in the peripheral venous system
- therefore pressors alone are not effective unless cardiac output is maintained with volume repletion

Treatment
- at the onest of anaphylaxis endogenous catecholamine release is massive therefore not usually a primary pump problem therefore adrenaline will have little effect on the heart if it’s empty
- give fluid: colloids have been shown to be better than crystalloids
- trendelenburg potions is useful in aiding VR
- adrenaline is 1st line agent but if response is not as good as predicted then there is a role for trying metaraminol or vasopressin (50mcg boluses of Ad if not cardiac arrest)
- sugammadex (in the case of rocuronium) will reverse muscle relaxation and increase tone which will compress intramuscular and abdominal vessels
- minimising anaesthetic drugs during shock is useful also as it will increase tone and sympathetically mediated increase in venous tone (up to 500ml is seen in exercise)
- there are cases in which sugam has been reported to be of benefit when the anaphylaxis has subsequently been proven to have been caused by an antibiotic.
- with conventional treatment, anaphylaxis is often rapidly curtailed and the surgical procedure completed. Administration of sugammadex may preclude this option
Pulmonary hypertension: an overview for the “non-cardiac” anaesthetist

-Definition: defined according to mean pulmonary artery pressure at rest as assessed by right heart catheterization (RHC)

-ohms law V=IR OR R=P/Q

-RHC is invasive

-remmeber a failing right heart may be unable to generate high pressures, hence the presence of a low pulmonary artery pressure does not guarantee a normal right heart and pulmonary vasculature

<table>
<thead>
<tr>
<th>Mean pressures:</th>
<th>Normal</th>
<th>Mild PH</th>
<th>Moderate PH</th>
<th>Severe PH</th>
</tr>
</thead>
<tbody>
<tr>
<td>RAP</td>
<td>2-5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mPAP (mmHg)</td>
<td>12-15</td>
<td>25-40</td>
<td>40-55</td>
<td>&gt;55</td>
</tr>
</tbody>
</table>

mPAP= mean pulmonary artery pressure

TTE criteria

-from Doppler interrogation of the velocity of any tricuspic regirgitation jet, a modified Bournoulli equation can be used to estimate RV systolic pressure

-RVSP=RAP+(tricuspid regurgitation jet velocity x 4)

TR jet velocity >3.4m/s, PA systolic pressure >50mmHg with or without echo features suggestive of PH then Pulmonary Hypertension is likely

Classification of pulmonary hypertension

-no longer primary or secondary

-5 classifications

1.1 idiopathic

1.2. heritable

1.3. drug and toxin induced

1.4. associated with

   a) connective tissue disease
   b) HIV infection
   c) portal hypertension
   d) congenital heart disease
   e) schistosomiasis, pulmonary veno-occlusive disease, haemangiomatosis, persistent pulmonary HTN of the newborn

2. Pulmonary HTN due to left heart disease

3. Pulmonary HTN due to lung diseases and/or hypoxia

4. Chronic thromboembolic pulmonary HTN

5. Pulmonary HTN with unclear multifactorial mechanisms

Pulmonary vascular resistence
-the pulmonary vasculature can accommodate massive increase in flow without increasing pressure
-factors that influence PVR can be broadly classified into mechanical or neurohormonal

**Mechanical:** PVR is lowest at FRC, rises with inspiration and full expiration
-as CO rises, PA pressure may rise however it causes recruitment and distension of capillary beds which plays a role in reducing PVR
-if pulmonary blood flow remains elevated for a prolonged period, such as systemic to pulmonary shunting (eg PDA, ASD or VSD) arteriolar smooth muscle becomes hyperplastic, this will eventually lead to a fixed rise in PVR that is poorly responsive to vasodilator therapy
-once PVR is >SVR there will be a permanent right-to-left shunt allowing venous blood to enter the systemic circulation= eisenmengers syndrome

**Neural:** SNS innervation, alpha-1 receptors causing NA mediated vasoconstriction and beta-2 receptors causing AD-mediated vasodilation
Cholinergic system (via vagus nerve) muscarinic receptors increase NO causing vasodilation

**Humoral:** prostaglandins, prostacyclins, endothelin, amines (histamine, serotonin)

Hypoxic pulmonary vasoconstriction: locally mediated

**Right ventricular function**
-low pressure, low resistance
-not suited to sudden rises in PVR
-in RV hypertrophy atrial systole becomes very important for ventricular filling
-IPPV may have profound effects on RV performance as it impedes VR and RA filling causing an immediate fall in RV output

-interventricular dependance descirves the syenrgisit relationship between the RV and LV
- the two important elements are the pericardium and the LV septum
-if the RV fails and enlarges, pericardium is fixed so LV will also fail
-the IVS becomes concave to the right in systole rather than the left, septal dyskinesis affects the tricuspid valve leading to TR

**Coronary blood flow**
-RV muscle mass increases due to hypertrophy
-RV wall strain becomes similar to that of the left and may even exceed it
-RCA takes on left-sided flow dynamics, that is, flow in diastole only
-therefore in severe pulmonary HTN, hypotension is poorly tolerated as the blood flow through RCA falls leading to RV ischaemia (intra-aortic ballon may be useful for this)
Strategies to reduce emergence agitation in children

INCIDENCE/DEFINITION

- EA common, recent Cochrane review of about 6000 children showed approximately 1/3 of children will experience EA after sevofurane anaesthesia.
- Emergence agitation not same as emergence delirium (ED). ED is a subset of EA. No universally agreed definition of EA.
- The most widely accepted scale currently, and the only validated scale is the Paediatric Anesthesia Emergence Delirium (PAED) scale (see below).
- Research currently supports a threshold PAED score >12 to describe presence of EA.
- There's also the simpler 'WATCHA' score. EA = score of 3 or greater.

AETIOLOGY AND RISK FACTORS

- Risk factors include: preschool aged group, pre-operative anxiety, certain temperaments (for example, poorly adaptable), ophthalmological and ear, nose and throat (ENT) procedures, sevofurane or desfurane anaesthesia, and inadequate analgesia at the time of emergence.
- 'Sevo washout' may be possible mechanism, although exact detail is unknown. We know that delaying emergence from sevo by various drugs have been observed to reduce EA.

CONSEQUENCE OF EA

- EA is usually self-limiting with resolution within 15-30 minutes of emergence. However, the two main issues with EA are self-injury and dissatisfaction.

INTERVENTIONS TO REDUCE EA

- The greatest body of evidence exists for propofol, fentanyl, α2-agonists (particularly dexmedetomidine) and halothane.
- 2014 Cochrane systemic review showed table summary:

<table>
<thead>
<tr>
<th>Figure 1. PAED scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not at all</td>
</tr>
<tr>
<td>The child makes eye contact with the caregiver</td>
</tr>
<tr>
<td>The child's actions are purposeful</td>
</tr>
<tr>
<td>The child is aware of his/her surroundings</td>
</tr>
<tr>
<td>The child is restless</td>
</tr>
<tr>
<td>The child is inconsolable</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Figure 2. Watcha scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Score</td>
</tr>
<tr>
<td>Calm, quiet</td>
</tr>
<tr>
<td>Crying, but can be consoled</td>
</tr>
<tr>
<td>Crying, cannot be consoled</td>
</tr>
<tr>
<td>Agitated and thrashing around</td>
</tr>
</tbody>
</table>
PROPOFOL
- cf w sevo induction/maintenance, prop is effective as either TIVA (reduce incidence by 1/3), as infusion during maintenance after gas induction, as transition at the end (3mg/kg over three minutes reduced incidence to 1/4 cf with control, but emergence time was longer by 8 mins), or as a bolus at the end (1mg/kg; this however isn't as effect as TIVA; prolongs emergence by 4 mins).
- It may be possible to reduce emergence time by starting transition to propofol earlier.
- ineffective if it's used for induction only, or as a 1mg/kg bolus shortly after induction. This is not surprising given the pharmacokinetics of propofol (Cochrane).

FENTANYL
- effective in both prevention and treatment of EA, even in absence of pain, IV or IN.
- optimal dose IN = optimal dose of 2mcg/kg
- IV: 1mcg/kg at end of sevo anaesthesia. this is not associated with increased side effects.
- cf propofol, equally effective, but may see more PONV with fentanyl.

CLONIDINE
- Clonidine effect in reducing EA seems limited to the setting regional blocks rather than ENT surgery.
- In terms of IV clonidine, the majority of studies have used a dose of 2mcg/kg but this can result in sedation, delayed PACU discharge. (dose-dependent prolongation of emergence half-time: 25mins with 2mcg/kg cf 10.8 mins without clonidine).
- clonidine 1.5mcg/kg IV was ineffective in reducing EA in children having adenoidectomy.

DEXMEDETOMIDINE
- extensively, results are far better than clonidine in reducing EA.
- Multiple benefits too: reduction in EA in many different surgeries including ENT, reduction in rescue analgesia, reduction in PONV and a minimal increase in emergence time (which is statistically signifcant, but not necessarily clinically signifcant).
- IV dose range between 0.15-1mcg/kg has been studied, given either early or late stages of anaesthesia.
- One group found, the 95 per cent effective dose was 0.38mcg/kg, to prevent EA in Adenoidtonsillectomy.
- intranasal route works too as a premed: a very recent RCT cf IN 1mcg/kg vs 2mcg/kg vs placebo given 45 minutes prior to induction and found a dose-dependent reduction in EA, in MAC-LMA, emergence time prolonged six and eight minutes respectively but no difference in time in PACU.
- A predictable drop in heart and blood pressure is reported in RCTs and is typically described as being not clinically significant (despite statistical significance), but this obviously depends on an individual patient's comorbidities and the clinical scenario.
- It's also very expensive.
OTHER VOLATILE AGENTS VERSUS SEVOFLURANE

- Halothane vs sevofurane, compared in more than 30 RCTS and meta-analysis shows a halving of the rate of EA.
- No difference between halogenated ether.

OTHER ADJUNCTS

- Ketamine either as a bolus of 0.25mg/kg at the end anaesthesia or as premedication may reduce EA
- Other options may help but more studies needed. These include: other opioids, tramadol, ketorolac, regional, antihistamine, magnesium, nitrous oxide to washout sevo before emergence.

ConCLUSION – A GENERAL APPROACH FOR A CALM EMERGENCE

- Many pharmacological interventions to reduce EA. But there's no one-size fits all magic bullet.
- Rather, a context-specific, multimodal approach to EA prevention can result in reduced EA and greater satisfaction. IE similar to PONV prophylaxis.
- A recommended approach is to stratify the risk factors (patient and procedure) and decide on the number and nature of prophylactic EA interventions.
  1. Ensure adequate analgesia for painful procedures at the time of emergence, including IV or intranasal fentanyl.
  2. Emerge on propofol rather than a volatile agent if there is IV access – choose one of the following: TIVA, switch to propofol maintenance after gas induction, transition from volatile maintenance to propofol late in the procedure, or a propofol bolus at the end. Emergence on propofol may have additional benefits of reduced airway reactivity and reduced PONV.
  3. Consider an α2-agonist, particularly dexmedetomidine if available (either IV intraoperatively or as premedication in the anxious child).

<table>
<thead>
<tr>
<th>Effective</th>
<th>Ineffective</th>
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<tbody>
<tr>
<td>Propofol</td>
<td>Desflurane</td>
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<tr>
<td>Fentanyl</td>
<td>Isoflurane</td>
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<tr>
<td>Dexmedetomidine</td>
<td>Midazolam premedication</td>
</tr>
<tr>
<td>Clonidine</td>
<td>Parental presence at emergence</td>
</tr>
<tr>
<td>Halothane</td>
<td>Gradual sevoflurane cessation</td>
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<tr>
<td>Ketamine</td>
<td>Lower sevoflurane concentration during maintenance</td>
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<tr>
<td>IV Midazolam at end</td>
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<tr>
<td>Analgesia</td>
<td></td>
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<tr>
<td>Various sedatives</td>
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<td>N2O washout</td>
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Intraoperative awareness and general anaesthesia for caesarean delivery: A fresh look at an ongoing problem

Intro:
• Traditional recommendations for GA in operative obstetrics advocate for heavy paralysis and light anaesthesia.
• However there's concern of accidental awareness during GA in obstetric surgery = ie 'explicit recall of operative events during GA'.
• Recent NAP5 showed 1/670 incidence, more than 10x risk across all anaesthesia subspecialties. In ANZ, risk is estimated to be 1/382.
• multiple risk factors exist for awareness in operative obstetric: rapid sequence induction, higher incidence of airway difficulty, pharmacokinetic changes of pregnancy, and often urgency.
• This article question some of the traditional recommendations, aiming to reduce risk of accidental awareness.

Comparison between propofol and thio:
• Thiopentone: its place in obstetrics recently challenged and there seems a growing favour towards propofol, likely due to human/institutional factors. eg. more distinct appearance propofol, less potential for accidental drug error
• Re: haemodynamics between thio/prop: thio may be marginally better than propofol in maintaining haemodynamic stability after induction dose is given.
• Re: speed of onset: comparable times at 43-46 secs respectively; but time to return of consciousness is longer for prop: 530 sec vs 330 sec (9 mins vs 5.5 mins).
• Re: amnesia/sedative effect: studies shown propofol has more profound amnesia for a given degree of sedation cf thio. ie equi-sedative concentration is 4 x higher for thio, but equi-amnestic concentration is 7 x higher for thio.
• Re: Key measures of neonatal/maternal outcomes, including neonatal wellbeing and maternal blood loss, are comparable between prop/thio.

Re: inhalational agent:
• On uterine tone: an impaired response to oxytocin becomes apparent around 0.75 MAC in vitro.
  o A Cochrane meta-analysis noted a greater fall in haematocrit and a greater estimated blood loss (by 127mL) in GA LSCS, cf w regionals. But there was no greater need for blood transfusion.
• Impact on fetus: A 2012 Cochrane review cf GA vs regional ELCS found no statistically significant difference in Apgar scores, cord blood gases, neurological adaptive scores or need
for oxygen resuscitation. Suggesting that “the effect on the foetus of anaesthetic agents is innocuous and reversible”

Re: N2O + important ANZCA trials

- ENIGMA cf N2O with 80% oxygen group: No difference in the primary endpoint (duration of hospital stay), however, the secondary endpoints of wound infection, pneumonia, atelectasis and severe nausea and vomiting (PONV) were increased.
- but ENIGMA-II 2014 said N2O is safer than ENIGMA I suggested. This larger trial supported the observation of an increased incidence of PONV (15 per cent in the nitrous oxide group versus 11 per cent in the no nitrous oxide group), but demonstrated no increase in other adverse outcomes, including cardiorespiratory morbidity.
- A follow-up study of 640 ENIGMA patients observed a significant reduction in chronic postsurgical pain in the nitrous oxide group.
  - but this analgesic effect was not demonstrated in another recent, as-yet unpublished study (by Matt Chan) exploring this.

Re: opioids

- MAC sparing effect of opioid is less on cp50 (response to command) for propofol, but a profound reduction on cp50 for movement.
  - Ie Opioids is better in reducing need for muscle relaxants, but not as good in provision of amnesia, nor reducing risk of awareness.

Re: BIS:

- Recommended in GA CS
- BIS has been shown to reduce the incidence of awareness when compared to clinical signs; but it’s not superior to Et monitoring with alarms set at <0.7 age-adjusted MAC.
- BIS does have other benefits: helps to achieve rapid wake-up, shorter recovery times and a lower total anaesthetic drug usage.
  - Note: there’s observational evidence correlating cumulative deep hypnotic time (BIS <40) with increased mortality and morbidity; BALANCE trial is investigating this.
- but beware, BIS is drug sensitive, with nitrous oxide and ketamine having paradoxical effects;
- BIS does not prevent all cases of awareness

Conclusion:

- rate of accidental awareness in GA CS is known to be high
- Re: propofol vs thio
- Re: inhaled agents: overpressure so to achieve effect-site concentration sufficiently in timely fashion; particularly relevant in pregnant women considering their increased cardiac output.
  (although there's also MAC reduction in pregnancy, balancing the risk of underdosing inhaled agent).
- balance volatile effect vs. reducing uterine tone;
- Re: N2O/opioids, valuable in reducing need for muscle relaxant, but be careful with N2O’s high MAC awake / MAC ratio and the small contribution of opioids to reducing MAC awake
Neonatal effects of GA appears to be transient and reversible.
Minimising anaesthetic delivery is not a suitable substitute for adequate resuscitation in the hypovolaemic patient.

**Other interesting numbers:**
- MAC awake to MAC ratio of 0.64 for nitrous oxide is relatively high. Anaesthetic agents with a higher ratio of MAC awake/MAC are poorer providers of amnesia.
- Rate constant for equilibration $t_1/2_{keo}$ are 4.3 minutes for sevofurane and isofurane and 2.3 minutes for desfurane.
- My favourite quote of useless piece of information: "At typical induction doses, neither thiopentone nor propofol demonstrate a sufficient plasma level to reliably prevent awareness by the time of skin incision and delivery during elective caesarean delivery7,16,21" - 3 papers to tell us that???? and published in BJA, anesthesiology?????
Remifentanil patient-controlled analgesia (PCA) on the delivery suite – past, present and future

Intro:
Remi PCA works:
• according to patient satisfaction, remifentanil PCA ranks highly
• according to pain scores, there’s only modest reductions during remifentanil PCA use.
• the above may be explained by a combination of sedation, euphoria and a rewarding effect which made the pain more manageable
• Comparisons of pain score in meta-analyses: remifentanil PCA better than nitrous oxide and pethidine, however epidural is best (despite similar satisfaction score between remi PCA and epidural).

Safety profile
• 2 main concerns are sedation and respiratory depression. May occur more often than we think.
• Studies have reported incidence of desaturation (sats <90% for 5% or more time) of 7/19 (~35%).
• Epidurals have consistently shown to result in better maternal oxygen saturations and sedation score, cf with remi PCA.
• mode of delivery appear to be unaffected by remifentanil PCA.
• Neonatal data is reassuring: no difference in neonatal HR, sats, BP in first 24 hours after birth, by maternal remi PCA use.

Can have serious morbidity
• 3 case reports of maternal cardiacresp arrest with remi.
• remi PCA creates another potential for drug error which could have dire consequence.
• Subsequently in an editorial, it’s recommended to have continuous (5 things): presence of a midwife during PCA use, continuous oxygen saturation monitoring, continuous cardiotocography (CTG) monitoring, a minimum three minute lockout for the PCA protocol (which is not commonly followed, including CCDHB) and a rigorous internal audit process.

Dosing/delivery
• variable by studies. Majority used: bolus of between 20-50mcg, a lockout of between one and two minutes and no background infusion

novel ideas
• a vital signs-controlled, patient assisted intravenous analgesia delivery system was proposed. In a closed-loop interactive system the remifentanil bolus is titrated according to the number of demands made in 15 minutely periods. If the oxygen saturation fell below 95% for greater than 15 seconds or maternal heart rate fell below 60 beats per minute for greater than 15 seconds, the pump automatically paused for five minutes then restarted with a smaller demand dose. If the desaturation continued for longer than five minutes the pump stopped and alarmed.

Blue Book 2015 Summary - 31
• efficacy was demonstrated by a case where during a seven-hour period of use, the pump paused for transient oxygen desaturations on 17 occasions and the lowset recorded oxygen saturation was 90%.

**Conclusion**

• Remi useful alternative to epidural in obstetrics
• better than IM pethidine, worse than epidural
• approx 1/10 woman using remi PCA will still elect to have epidural
• effect to newborn is reassuringly small
• minority of women may get sedation/resp depression, and we rely on midwifery care to monitor these patients
• safe delivery of remi must be achieved, ways to ensure that include: midwifery education, guidelines on management of complications, continuous monitor of patient, regular audit of PCA/sats/patient satisfaction, concerns.
• decision to offer remi PCA must take into account the above safety concerns.

Some interesting numbers: remi onset time 30-60sec, peak effect 2.5 mins, average labour contraction = 70sec.

**CCDHB’s way:**

• only for established labour in 3rd trimester, 100% attendance by midwife at all times and no recent opioids and with patient consent (as unlicensed indication for remi); continuous SpO2, give O2 if sats <94% and continue for PCA use; inform Paeds. Monitor RR/BP/HR at least Q15-30mins. Record sedation score. Keep naloxone, ambubag in the room. Be particularly careful if there’s increased risk of apnoea eg OSA. Consider O2 with capnography for monitor.
• method: press button just before or at start of contraction ie try to anticipate contraction. Through dedicated cannula.
• regimens:

  1. variable bolus with no basal – use this first. Change to 2nd regimen below if still in pain once maximum bolus dose is reached or if peak effect of bolus is after contraction is over (inadequate analgesia with over-sedation between contractions).
     • Start bolus = 0.25mcg/kg and increase in 0.25mcg/kg increments at 15mins intervals until VAS 4 or below. Max dose = 0.75mcg/kg.
     • 2min lockout. Hourly max = 12mcg/kg/hr (equivalent to infusion of 0.2mcg/kg/min).
  2. fixed bolus with variable basal.
     ♠ Bolus fixed at 0.25mcg/kg.
     ♣ Infusion start at 0.025mcg/kg/min. increase rate by 0.025mcg/kg/min increments at 30min intervals until VAS 4 or below. Max rate = 0.1mcg/kg/min. 2 min lockout. Hourly max dose = 12mcg/kg/hour; equivalent to infusion of 0.2mcg/kg/min.
• Stop PCA if hypotension, bradycardia, RR<8, hypoxaemia despite O2.
Goal-directed transthoracic echocardiography – a translational education program

The use of transthoracic echocardiography, (TTE) as opposed to transoesophageal echocardiography, by anaesthetists is increasing.

Only a few hospitals in Australasia have established and sustainable education programmes incorporating quality assurance and outcome of TTE teaching and training into daily clinical practice.

This article looks at an educational program that may fulfil the requirements for a sustainable program in TTE so this tool can be integrated into daily clinical practice.

Able to answer questions at point of care.

In the skilled hands can estimate PA pressures, LA/LV pressures and CO accurately and start treatment as appropriate.

Formal education in TTE is not well defined in Anaesthesia. Novice operator and Expert Operator.

Novice operator is able to perform emergency TTE and recognise life-threatening clinical conditions e.g. hypotension caused by hypovolaemia, reduced ejection fraction, right ventricular failure or cardiac tamponade.

The advanced or expert operator has the ability, after training in a stepwise fashion from basic to advanced skills and knowledge, to perform a full cardiac assessment.

Goal-directed TTE = abbreviated / shortened TTE performed at the point of patient care, rules out the presence of major abnormalities as the cause of the acute physiological disturbance. Involves acquisition, recording, storage of a reproducible, easy to obtain, clinically relevant minimum data set by a trained operator.

Principles of goal-directed scans are:

Acceptable and applicable
Bedside test
Comfortable and concise examination – limited views
Diagnosis and response to therapy – contractility status and volume status
Embolism (air, blood, amniotic fluid) – right heart function/relative size
Foetal heart rate assessment (in the case of a pregnant woman)

ANZCA PS46 - guidelines for the training and practice of perioperative cardiac ultrasound in adults.

It recommends – at least 20 supervised TTE, at least 20 additional unsupervised TTE with full review by a supervisor, and at least 50 additional goal-directed TTE (with review by supervisor as necessary), be performed. After basic training is achieved, standards are maintained by audit and peer review and performing at least 50 studies annually.

TTE education must have quality assurance, but PS46 does not
include the need to record outcome data.

Kirkpatrick's framework provides 4 levels of outcome assessments and evaluating their impact

Level 1 – Reaction – participant's reaction to the intervention.
Level 2 – Learning – the degree to which the learning occurs as a result of the intervention.
Level 3 – Behavioural change – the transfer of learning to behaviour at work.
Level 4 – Organisational performance – the impact of learning on patient outcomes

Ideally, educational programs satisfying these four areas of evaluation would result in a positive reaction by participants to the education program (Kirkpatrick level 1), improved learning by the participant (level 2), the transfer of skills and knowledge to the clinical environment by the participant (level 3) and an improvement in patient outcomes (level 4).

Education programme outline for a 12 month continuing program in Echo (see figure !)

Assessment 1 (preeducation) initiital training (four months) Assessment 2 (post initial education)
Probationary training 4 months Assessment 3 (post probationary training) Independent training (four months) Assessment 4 (post independent training) Completion of training and commencement of maintenance of skills and knowledge
Pain management for trauma: Time to embrace regional anaesthesia?

For decades trauma management has defaulted to GA and systemic analgesia alone, despite multiple recognised drawbacks. Regional anaesthesia is established as an integral component of high-quality, evidence-based perioperative anaesthetic care.

ATLS focuses on simultaneous assessment and management with the immediate focus being preservation of life. Pain management is given a lower priority.

Unrelieved acute pain is an independent risk factor for progression to chronic pain. Surgeries associated with significant nociceptive inputs and nerve injury e.g thoracotomy, amputations have high incidences of chronic pain, 6-65% and 50-85% respectively.

“Stress response” - complex physiological response to tissue injury. This involving activation of neural, metabolic, endocrine, haematological and immunological systems. The magnitude of this catabolic state corresponds with the extent of tissue damage. Attenuation of the stress response is a meaningful treatment objective. Plausible advantages of avoiding “stress” include improvement of the myocardial oxygen supply/demand ratio, maintenance of gut and immune function, and reduced thromboembolism risk.

Specific issues in trauma - coagulopathy, potential secondary nerve injury and perceived delayed diagnosis of acute compartment syndrome (ACS).

Table 1. Advantages and limitations of regional anaesthesia for trauma

**Advantages**
- Superior pain control
- Chronic pain protection
- Avoidance of airway management (for example, difficult/failed intubation, bleeding, oedema, dental damage, cervical spine movement)
- Stable haemodynamics
- Satisfaction
- Ease of transport
- Facilitation of physiotherapy
- Reduced nursing requirement
- Attenuation of stress response
- Reduced opioid dosage

**Limitations**
- Competition with resuscitation objectives
- Acute compartment syndrome
- Secondary nerve injury
- Coagulopathy
- Training, infrastructure, education, attitudes
- Polytrauma
• Positioning – e.g spinal cord injury
• Local Anaesthetic Systemic Toxicity (LAST) risk
• Management mandating general anaesthesia (for example, sternotomy, craniotomy)

Combative patient

Why Regional for Trauma?
Regional anaesthesia is highly desirable for many trauma injuries.
Avoids side effects of systemic opiates.
Growing body of evidence from battlefield trauma.

Examples of regional techniques in Trauma:

- continuous catheter infusions for NOF
- Paravertebral and thoracic epidural blocks for multiple rib # - reduce respiratory morbidity and possibly mortality, (paravertebral/thoracic epidurals are gold standard following thoracotomy)
- Recuts sheath and TAP blocks for anterior abdominal wall surgery
- Brachial plexus and interscalene blocks for shoulder reductions, axillary blocks for distal radius #

Tibial shaft # - resistance to regional anaesthesia is high due to perceived risk of Compartment syndrome

Compartment syndrome
Uncommon but feared consequence of trauma. A critical pressure increase in a confined myofascial space causing microvascular ischaemia of nerves and vessels traversing the affected compartments treatment = emergency fasciotomy.
Pain disproportionate to clinical situation is a cardinal feature.
Fear that regional technique may mask pain and delay dx based on a few case reports. These had other contributory factors for delayed diagnosis.
Systematic review by Mar et.al – no association between regional interventions and delay in detection of CS, rather dx may be assisted where breakthrough pain is reported in presence of previously satisfactory perineural block.

Nerve Injury
Recognised complication of trauma.
Mechanisms include laceration, axial stretch, compression and vascular compromise
Neurologic deficit may be aggravated by iatrogenic causes e.g surgical fixation, poor positioning, regional anaesthesia intervention

Double crush phenomenon described in 1973 – association between cervical radiculopathy and carpal tunnel syndrome. Unifying explanation to this is that individual lesions in a nerve increases the risk of injury at a 2nd location along the same nerve.
There is continued reluctance to undertake regional anaesthesia due to this reason.
Individual risk assessment is crucial. Performing thorough neurological exam and documentation is paramount.

Coagulopathy
Common in trauma. Acute coagulopathy in trauma is a complex process initiated by tissue trauma, hypoperfusion, hypothermia and acidaemia, compounded by large volume autologous blood transfusions.
Once haemorrhage control has been achieved, the balance of risks shift towards thrombosis. Guidelines exist for regional techniques in the presence of thromboprophylaxis but in the severe trauma setting this is less clear. Where complex risk-benefit balance exists, two consultants should agree the risk/benefits are justified.
The role of regional anaesthesia in clavicle fracture surgery

INTRODUCTION
commonly injured bone in the human body accounting for 2.6 per cent to 4 per cent of all adult fractures. Of all clavicle fractures, shaft # - 69 -84%, lateral # less common 21-28%, medial # 2-3%.

Typical patient is a young male involved in a sporting injury. Second small peak in elderly following fall and medial # is more likely in this case.
Owing to mechanism of clavicle fracture, other associated injuries e.g rib # with pneumothorax have anaesthetic implications.

TREATMENT OPTIONS
Treatment options – operative and non operative
Traditional approach to mid shaft # was conservative tx with sling or figure of eight bandage.
There are concerns regarding non union and delayed healing with conservative approach therefore surgical fixation rate is increasing.

Surgical techniques: intramedullary fixation or plate fixation.

They both have similar success rates in range of shoulder movement and bone union. Post operative pain is similar in both techniques.

Plate insertion – extensive soft tissue dissection and risk of supraclavicular nerve injury, prominence just under skin which patient may find annoying

Intra medullary fixation – less invasive, two small cuts but technically more challenging and increased risk of failure especially in rotationally or axially unstable #

Innervation of Clavicle

Poorly defined
Widely accepted that innervation to skin overlying clavicle is from supraclavicular nerve – a branch of superficial cervical plexus
Bony innervations unclear
Some text books say that innervations to clavicle is from brachial plexus via the following nerves alone or in combination: subclavian nerve, suprascapular nerve, long thoracic nerve
Hiton’s law could also be applied – ‘a joint is innervated by same nerves that supply the muscles acting on the joint, which also supply the skin overlying the articular insertions of those muscles. Hence it would be expected that nerves to subclavius (subclavian nerve), pec major, clavicular head (lateral pectoral nerve) and deltoid (axillary nerve) also contribute to clavicular innervations.
Note: these nerves arise from upper brachial plexus (C5,6,7)

Good regional anaesthesia for clavicle has been achieved with these blocks

- Inetescale block using high dose of bupivacaine 200mg in 22 patients with midshaft clavicle #
- Superficial cervical plexus block in 1 patient
- Combined superficial cervical plexus block and C5 nerve root block in 3 patients (one patient with lateral clavicle # and in the other two patients, location of clavicle # was not specified)

It appears that single regional technique can’t reliably be used to achieve surgical anaesthesia or perioperative analgesia for clavicle # surgery

Risk of nerve injury from surgery
- Injury to supraclavicular nerve as a result of surgery is high
- Performing superficial cervical plexus block or supraclavicular block may implicate anaesthetist as a potential cause of post op altered sensation in the distribution of supraclavicular nerve (significantly this can affect breast and nipple area)
- Injury to brachial plexus is well recognised post clavicle #, however direct injury to brachial plexus from bony fragments of clavicle is <1%

Conclusions:
Best anaesthesia mx for clavicle # surgery is yet to be defined

It is likely that clavicle is innervated by both superficial cervical plexus (medial clavicle) and brachial plexus (lateral clavicle)

Multi blocks are needed if regional alone is used
- superficial cervical plexus block (performed at level of C4 the level of thyroid cartilage) + interscalene block
- for patients where avoiding interscalene block is important – use superficial cervical plexus block + C5 nerve root block