AQUA
ANNUAL QUEENSTOWN
UPDATE IN ANAESTHESIA

AQUA 2017

Annual Queenstown Update in Anaesthesia

Programme and Abstracts

www.aqua.ac.nz

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AQUA
ANNUAL QUEENSTOWN UPDATE IN ANAESTHESIA

JAFA
Joint Anaesthesia Faculty Auckland

New Zealand Society of Anaesthetists

Welcome to Queenstown

Dear Colleague

Welcome to AQUA 2017, our 9th and biggest ever AQUA.

Our Australian speakers are Dr Nicholas Chrimes and Dr Chris Thompson. Dr Chrimes is well known for his work on emergency airway management and a systematic team approach to crisis management. He will present on both these topics. Chris Thompson, a previous AQUA speaker is talking on developments in ventilation and also on cerebral protection in the neurosurgical patient. Chris has offered to run a ventilation workshop and further details can be found on the AQUA website.

Our usual strong local faculty are providing updates on a broad range of topics including paediatrics, regional anaesthesia, diabetes and blood management. There is an interesting perspective on the opioid overdose epidemic in the US and its implications for New Zealand. And we will be re-visiting the THRIVE talk from 2016 with our speaker actually present in Queenstown this time! Thanks to our speakers for their hard work in preparing their talks and joining us in Queenstown.

The AQUA dinner on Friday night is at the Skyline Restaurant. The AQUA BBQ is on Saturday evening at Coronet Peak. We will be screening the second Bledisloe cup rugby match with kick-off at 1935.

A big thanks to our sponsors who are again supporting our meeting and make it viable.

Finally, we do have some snow at Coronet Peak, so we are all set for another great meeting.

We look forward to seeing you there.

Kerry Gunn
Neil MacLennan
Karen Patching

AQUA Conveners

Social Programme

THURSDAY, 24 AUGUST 2017

1700 – 1900

Registration & Welcome Function
Exhibitor Area, Pounamu Room, The Heritage

Browns Fitting Service – Foyer outside the Pounamu Room, the Heritage

FRIDAY, 25 AUGUST 2017

1800 onwards

AQUA Conference Dinner

Skyline Gondola and Restaurant

SATURDAY, 26 AUGUST 2017

1800 onwards

AQUA BBQ & Bledisloe Cup Rugby Function

Coronet Peak Base Building, Queenstown

International Faculty

Dr Nicholas Chrimes

Specialist Anaesthetist, Monash Medical Centre
Melbourne, Australia

Dr Nicholas Chrimes is a Specialist Anaesthetist at Monash Medical Centre in Melbourne. He has clinical interests in obstetric, bariatric and neurosurgical anaesthesia. He has extensive experience in healthcare education and simulation having previously been head of education for both Monash Simulation and the Monash Health Medical Education Unit. He is currently an instructor at Monash Simulation and a visiting instructor at the Sydney Clinical Skills and Simulation Centre. Dr Chrimes is the creator of the Vortex Approach to airway management and the Triad Approach to crisis management. He is an instructor on the Effective Management of Anaesthetic Crises (EMAC).

Mr Reza Nouraei

Specialist ORL Surgeon, Auckland City Hospital
Auckland, New Zealand

Reza Nouraei is an ORL surgeon with interests in Laryngology and Tracheal surgery. He is currently a locum specialist at Auckland City Hospital and will soon be joining the Centre for Airway, Voice and Swallowing at Poole Hospital in England. He was an academic ORL trainee in London and a co-founder of the National Centre for Airway Reconstruction.

In 2015 he described Transnasal Humidified Rapid-Insufflation Ventilatory Exchange (THRIVE) with Anil Patel and has since studied and presented extensively on the underlying physiology of trans nasal humidified oxygen insufflation and its clinical application in a range of circumstances including procedural sedation, rapid-sequence intubation, and management of complex and acutely-compromised airway patients.

Dr Chris Thompson

Specialist Anaesthetist, Royal Prince Alfred Hospital
Sydney, Australia

Dr Thompson is a senior staff specialist at RPA in Sydney. He has a sub-specialty interest in neurosurgical anaesthesia and cerebral monitoring and cerebral protection. He has a long-term interest in the design of anaesthesia equipment, particularly human interface issues for new anaesthetic machines.

New Zealand Faculty

Dr Sara Allen	Specialist Anaesthetist, Auckland City Hospital
Dr Kerry Gunn	Specialist Anaesthetist, Auckland City Hospital
Dr Justin Holborow	Specialist Anaesthetist, Dunedin Hospital
Dr Graham Knottenbelt	Specialist Anaesthetist, Starship Children's Hospital
Dr Joe MacIntyre	Specialist Anaesthetist, Nelson Hospital
Jim Moriarty	Actor, Registered Psychiatric Nurse, Wellington
Dr Chris Nixon	Specialist Anaesthetist, Auckland City Hospital
Dr David Sidebotham	Specialist Anaesthetist, Auckland City Hospital
Dr Jay van der Westhuizen	Specialist Anaesthetist, Auckland City Hospital

Airway Workshop

Dr Rose Duckett	Specialist Anaesthetist, Auckland City Hospital
Dr Paul Gardiner	Specialist Anaesthetist, Auckland City Hospital
Dr Sarah Scott-Brown	Specialist Anaesthetist, Auckland City Hospital

Scientific Programme

Friday, 25 August 2017

Session 1		**Icon Conference Room**
0725	Behind the Scenes: Forum Update	Kaye Ottaway
0755	Welcome and Introduction	David Kibblewhite, President NZSA
0800	What's happening with ventilation?	Chris Thompson
0835	Update on Blood and Iron	Kerry Gunn
0900	Paediatric Anaesthesia Update	Graham Knottenbelt
0925	Point of Care Ultrasound in the Perioperative Setting	Sara Allen

Session 2		**Icon Conference Room**
1020	A Short Degustation of Regional Anaesthesia	Justin Holborow
1050	Perioperative risk associated with aortic stenosis in non-cardiac surgery	Joe MacIntyre
1115	Opioid epidemic in the USA. Lessons learned…	David Sidebotham
1145	Close	
1300	Airway Workshop 1	Paul Gardiner, Rose Duckett &
1530	Airway Workshop 2	Sarah Scott-Brown

Saturday, 26 August 2017

Session 3		**Icon Conference Room**
0800	The Vortex approach to airway management	Nicholas Chrimes
0835	How to THRIVE with a difficult airway patient	Reza Nouraei
0900	Being too sweet… 'to siphon honey'	Jay van der Westhuizen
0925	Protecting the brain	Chris Thompson

Session 4		**Icon Conference Room**
1020	The Triad approach to crisis management	Nicholas Chrimes
1050	Towards a better understanding…	Jim Moriarty
1120	Forty years of anaesthetic practice	Chris Nixon
1150	Close	
1300	Advanced Ventilation Skills Workshop (1 hour)	Chris Thompson

The AQUA Conference 2017 can be claimed under the ANZCA CPD Knowledge and Skills category under the following activities: Lectures 1 credit/hour. Small group discussions 2 credits/hour.

What's happening with ventilation?

Chris Thompson

Royal Prince Alfred Hospital, Australia

This talk will cover a wide range of topics related to ventilation, providing a physiologically based perspective on which to base rational adjustment of the settings on a modern ventilator.

Topics such as optimal PEEP, ideal I:E ratio, when to go fast vs slow, pre-oxygenation, understanding CO_2, Thrive, recruitment and outcome will be covered in some detail.

Update on Blood and Iron

Kerry Gunn

Auckland City Hospital, New Zealand

Massive Haemorrhage: Shock Trauma and Coagulopathy

Trauma has few benefits. But for the study of the response of the human's physiology to shock it provide a unique model to explain changes that have troubled clinicians for decades in understanding why patients continue to bleed when normally they do not.

If a patient has severe trauma defined by evidence of shock and ongoing, uncontrolled bleeding they have a 20% mortality, which increases to 40-50% if in addition they have a coagulopathy. They are 8 times more like to die in the next 24 hrs with a coagulopathy than not, and results from the PROPPR and PROMMTT [1]studies suggest that rapid resuscitation with fibrinogen rich blood products may reduce bleeding, improve short term survival, but not such that in hospital mortality is reduced.

The development of a coagulopathy has been recognised for many years since Cannon [2]recognised the delirious effect of resuscitation of patients with clear fluids in battlefield trauma. The dilutional coagulopathy does not explain the profound blockade in coagulation in shock. Evidence currently points to poorly perfused endothelium, stimulated by a hyper adrenergic sympathetic system exuding thrombomodulin and activated Protein C into the microcirculation.[3] This effects PAI-1 to promote fibrinolysis, inhibit FV and FVII to stimulate thrombin, and thus limit clot forming in the microcirculation. While this may preserve the organ if perfusion is re-established, the systemic effects of this are to induce non-surgical bleeding that increases mortality in the trauma patient.

Thus, and in tandem with this the previously intact glycocalyx is damaged. [4]When large crystalloid resuscitation fluids are used the protein and heparan matrix within the extra-endothelial layer loses its integrity. Fluid loss through the basement membranes increases, and he effectiveness of the circulation is impaired.[5]

Indicators of increased mortality using coagulation parameters show that they are the result of profound shock. Elevated Protein C levels, Syndactin-C levels (indicating glycocalyx destruction) and elevated adrenaline levels all are associated with abnormalities in coagulation parameters (INR, aPPT), and TEG abnormalities.[6] Similar changes in platelet aggregation occur.

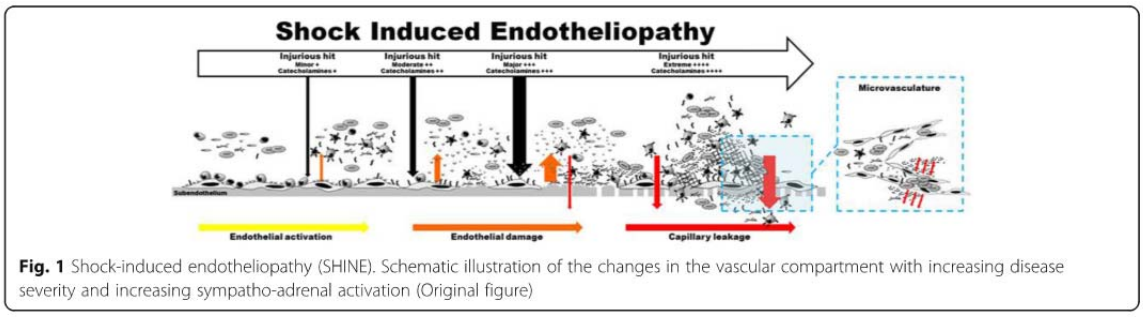


Fig. 1 Shock-induced endotheliopathy (SHINE). Schematic illustration of the changes in the vascular compartment with increasing disease severity and increasing sympatho-adrenal activation (Original figure)

Johansson *et al. Critical Care* (2017) 21:25

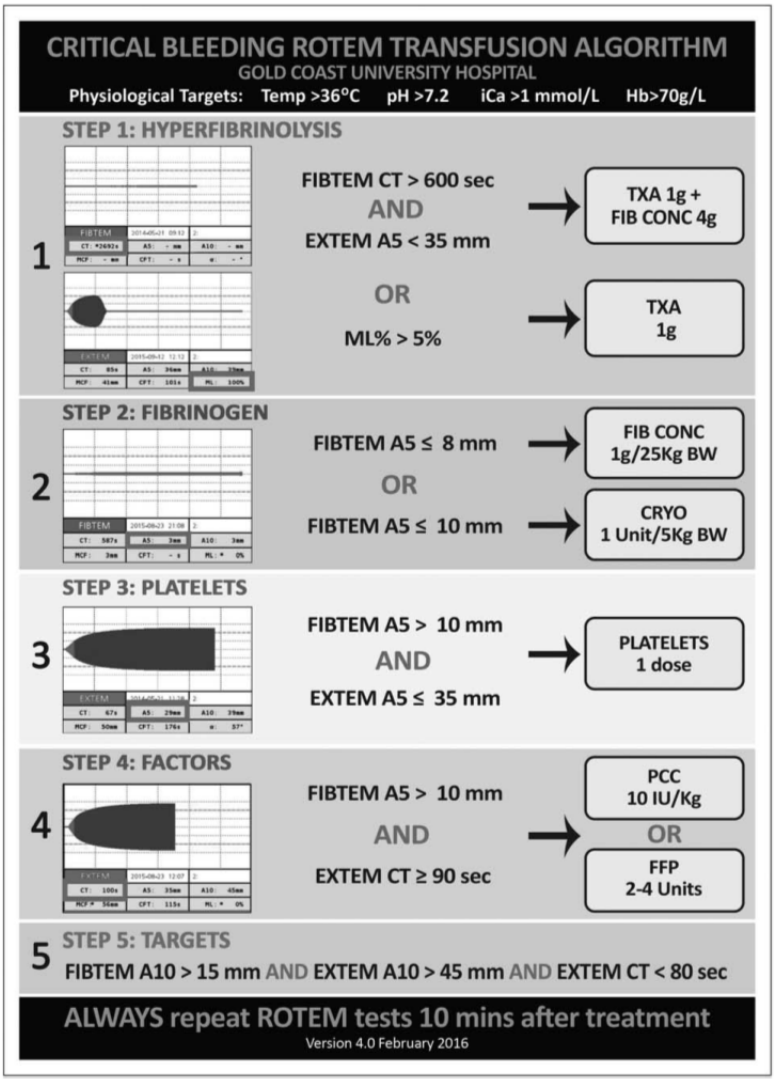
The resulting clinical problems are a patient in shock with bleeding from non-surgical wounds, that continues to bleed after the trauma pathology is fixed. This leads to abdominal compartment syndrome, Multisystem organ failure and death.

Empiric responses to this have been a rapid recognition of patients at risk, rapid transport to a definitive site of bleeding control (operating room or interventional radiology), damage control surgery, which involved rapid surgery limited to stopping bleeding, then stopping, Damage control resuscitation which involves limiting crystalloid, empiric use of Tranexamic acid at a dose of 15mg/kg bolus plus an infusion over 1 hrs, blood given in either a 1:1:1 fixed ratio, or targeted to a TEG or ROTEM, and sometimes permissive hypotension. Patients with persistent acidosis and hypothermia are managed in the ICU until stabilised before definitive trauma surgery

Together these bundles of care have reduced mortality form massive haemorrhage in trauma substantially.[7]

The question is where these lessons can be applied in other surgical areas. While the principles are logically applied to any surgery that includes shock and uncontrolled bleeding, in normal high blood loss surgery evidence is lacking to aggressive resuscitation along these lines. A warm, not shocked patient with limited tissue trauma behaves differently and focused therapy is more logical. In Cardiothoracic surgery, the effect of drugs that are anticoagulant and antiplatelet need to be specifically reversed rather an empirically treated.

The concept of Goal directed therapy where abnormalities are corrected only in bleeding patients has the advantage of focussing therapy on laboratory abnormalities. The most validated of these is using a TEG or ROTEM. It further allows treatment with less exposure to allogenic blood products, and less system waste. [8,9] But it usually needs specialist skills and a dedicated person controlling the resuscitation.



The question in the future is if we need to add a person to the team. There has usually been an airway specialist, should we add a bleeding specialist?

Evidence supporting the use of PBM

Improved outcomes and reduced costs with PBM – the Australian experience

The implementation of the world's first comprehensive health-system-wide PBM programme in Western Australia has reduced transfusion rates and improved patient and economic outcomes.[10] Prior to the implication of the programme, this Australian state already had one of the world's lowest RBC issuance rates; 31.8 per 1000 population in 2008-9, compared with rates in Germany, Denmark, the UK and the US of 57.3, 60.0, 36.3 and 48.8 per 1000 population, respectively. [10]
Patient and economic outcomes investigated over 6 years in 605,046 inpatient admissions at four major Western Australian adult tertiary care hospitals partaking in the PBM programme during the period 2008-14, revealed a 41% (RR 0.59; 95% CI 0.58-0.60, $p < 0.001$) decrease in units of RBCs, fresh-frozen plasma and platelet units transfused per 1000 discharges when baseline values were compared with end of study data (**Figure 5**). [10] During this period, the mean RBC pre-transfusion haemoglobin level decreased from 7.9 g/dL to 7.3 g/dL ($p < 0.001$) and the proportion of single-unit RBC transfusions increased from 33.33% to 63.69% ($p < 0.001$). There was a significant reduction in the proportion of elective surgical patients admitted with anaemia (20.81% vs 14.42%; $p = 0.001$), a 28% risk-adjusted reduction in hospital mortality (OR 0.72; 95% CI 0.67-0.77; $p < 0.001$), a 21% risk-adjusted reduction in hospital-acquired infections (OR 0.79; 95% CI 0.73-0.86), a 31% risk-adjusted reduction in acute myocardial infarction/stroke (0.5% vs 0.4%; OR 0.69; 95% CI 0.58-0.82; $p < 0.001$), and an adjusted 15% reduction in mean length of hospital stay (5.9 days vs 5.3 days; incidence RR 0.85; 95% CI 0.84-0.87; $p < 0.001$)

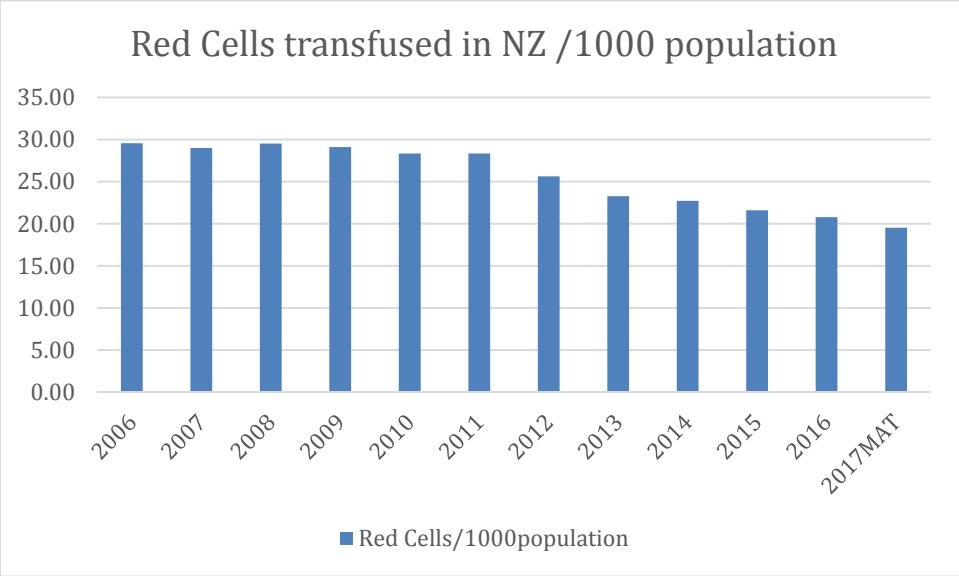
These reductions translated to a product-acquisition cost saving of AU$18,507,092 and an estimated activity-based saving of between AU$80 million and AU$100 million during the 6-year study period. The risk of all-cause emergency readmissions rose from 11.4% to 12.4% during the study period (OR 1.06; 95% CI 1.02-1.10; $p = 0.001$); this finding is contrary to the findings of other studies. [10]
The findings of the Western Australian study are consistent with those of smaller studies investigating the implementation of individual PBM strategies in selected patient groups.[24-30] A systematic review and meta-analysis of RCTs by Salpeter et al., demonstrated that trials with more restrictive transfusion thresholds demonstrate significantly reduced infection, cardiac events, rebleeding and mortality rates compared with those using less restrictive thresholds.[32] Furthermore, there are numerous risk-adjusted observational studies showing independent dose-dependent associations between RBC transfusion rates and increased morbidity and mortality.[10]

The European Union follows suit

In April 2017, the European Commission announced the publication of two PBM guides recommending PBM as the standard of care for the European Union.[30] The guides were modelled on the "impressive results" of the Western Australian PBM programme. Furthermore, the World Health Organisation has endorsed and promoted PBM and it is widely accepted as current best practice.[32]

Local experience

'Blood as a gift' has been an ongoing initiative in NZ at Auckland City Hospital since 2010. The initiative was developed with the mission of introducing and embedding blood management principles and practice, in an aim to improve patient blood safety and reduce unnecessary transfusions. Between October 2010 and December 2013, the initiative improved the utilisation of RBC units, with an overall reduction of approx. 18% in mean consumption. In addition to the associated financial savings, there was a significant time saving for both patients and staff.



The status of PBM in NZ

There are a number of subtle differences between NZ, Australia and other counties with regard to PBM. The costs of blood products are borne by the District Health Boards, unlike federally paid products in Australia. It may be that micromanagement of the effective use of NZ blood products have always lead to more rational use. Saying that, audits in the ADHB in 2003 showed 20% of prescribed red cells had no logical indication on modern criteria (as in the NBA guidelines), and a program to encourage better transfusion practice lead to a 17% reduction in use. Similar programs with the use of fresh frozen plasma, platelets and group and antibody screens have led to reduced use. In fact in absolute terms, FFP use in 2017 is 50% of that in 2005, and red cell use has been dropping in the last four years, despite an increasing population and more complex surgery. While the more restricted use of blood products are logical in anaemic patients, or those with deranged coagulation screens, but not bleeding, the use of a MTP has lead to reduced deaths due to exsanguination. This paradoxically is associated to a more liberal delivery of coagulation factors (predominantly fibrinogen dominant) to the patient rapidly. As these products are usually frozen, early thawing often leads to waste. The key to using these products wisely is to understand the indications for the activation of the MTP, use rapid assays (often the thromboelastogram [TEG]) to diagnose coagulopathy, and to stop the MTP at the correct time. Alternative approaches with a goal directed approach that differs in not following the empiric formula of an MTP are also being investigated.
The development of the NBA guidelines involved collaboration with NZ and Australian groups, and the endorsement of them by Colleges in Anaesthesia, Critical Care, Surgery and Haematology. They are relevant to NZ practice.

The optimal timing of iron treatment

Oral iron still should be the first drug used in treating iron deficiency. In patients tolerant of the side effects, and with intact gastrointestinal absorption, replacement is near equivalent to a single bolus of IV Iron. Unfortunately many of the patients presenting with low ferritins have a body deficit of iron of >1G. With daily absorption of oral iron approx. 5mg, replacement will take 2-3 months. Thus with a patient intolerant to oral iron, poor absorption due to elevated hepcidin, and surgery within the next month, the place of IV Iron has become better defined. Modern preparations have a low side effect profile, unlike the older dextran preparations. They are able to be given as an undiluted slow IV injection in GPs' surgeries, and are effective. Peak elevations in haemoglobin are in the 2-4 week period, dependent of the nature of the deficiency, and the degree of concomitant bleeding. Post-operative oral iron has very limited absorption.

Implementing PBM in NZ

The implementation of a PBM strategy in NZ has started, but has much promise to reduce further unnecessary blood transfusions, and consider reducing wastage. This has put considerable pressure of the New Zealand Blood Service (NZBS), who is adapting their donor population to meet the product demands of more fractionated products (IVIG, Prothrombinex®, albumin and possible fibrinogen concentrate) and reduced FFP and red cell use. We do not have a well-funded overarching body like the NBA in NZ, but we do have an effective network of committed clinicians, mainly through blood transfusion committees to introduce systems to reduce variance in blood transfusion practices. We also need to ensure the public, who are the donors and recipients of blood at the same time, believe the decisions we make on their behalf are well founded in fact, and are not wasteful.

The recently developed 'Simplified International Recommendations for the Implementation of Patient Blood Management' which include a series of simple cost-effective, best-practice, feasible and evidence-based measures, is a useful resource aimed at enabling any hospital to reduce both anaemia prevalence on the day of surgery or intervention and anaemia-related unnecessary transfusion in surgical and medical patients.

Educational resources on PBM

BloodSafe eLearning Australia has excellent online courses relating to PBM and clinical transfusion practice for health professionals. https://bloodsafelearning.org.au
The National Blood Authority provides a range of online tools to aid in the implementation of the PBM Guidelines at a heath provider level. https://www.blood.gov.au/implementing-pbm

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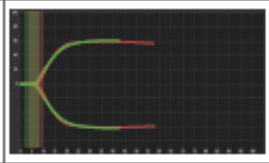
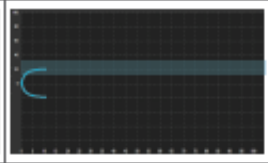
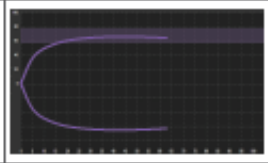
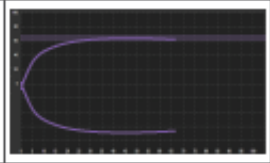
TEG® 6s Deficiency Assessment

	Clot Rate	Clot Strength	Clot Strength	Clot Stability
Hemostatic Component	Coagulation factors & heparin	Fibrin clot	Platelet & fibrin clot	Fibrinolysis
Test - Parameter	CK / CKH - R	CFF - MA	CRT - MA	CRT - LY30
Normal Tracings *Shaded Reference Ranges for illustration only*				
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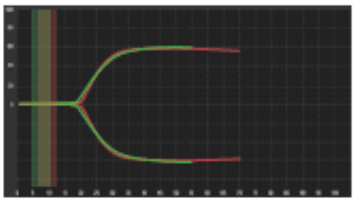
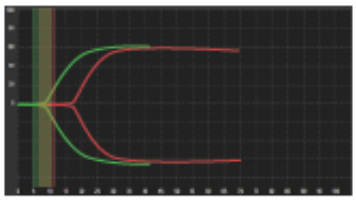
Hypercoaguable	↓ R_{CK} (min)	↑ MA_{CFF} (mm)	↑ MA_{CRT} (mm)	N/A

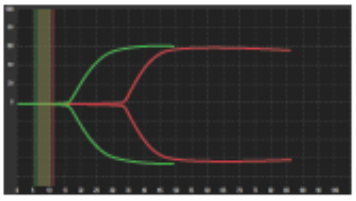
Clot rate



Factor Deficiency

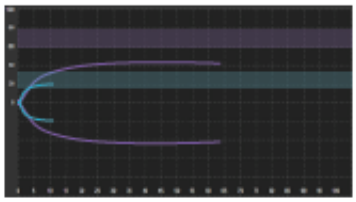


Heparin Effect

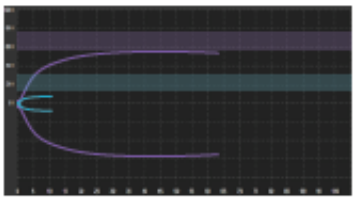


Factor Deficiency & Heparin Effect

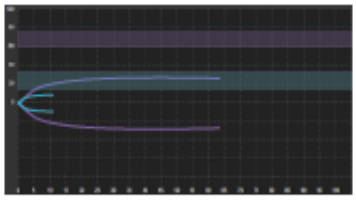
Clot strength



Platelet Deficiency

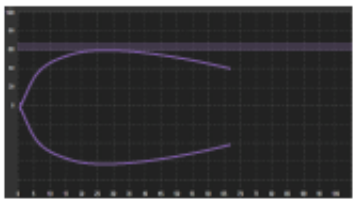


Fibrinogen Deficiency

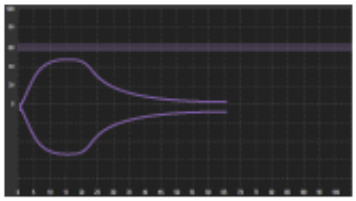


Platelet & Fibrinogen Deficiency

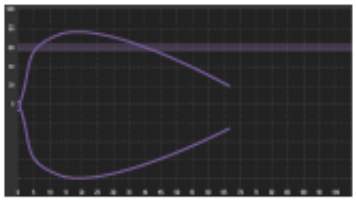
Clot stability



Hyperfibrinolysis



Primary Fibrinolysis



Secondary Fibrinolysis

Shaded reference ranges are shown for illustrative purposes only

CK - R reference range

CKH - R reference range

CFF - MA reference range

CRT - MA reference range

CRT - LY reference range

Results from the TEG 6s analyzer should not be the sole basis for a patient diagnosis, but should be evaluated together with the patient's medical history, the clinical picture and, if necessary, other coagulation tests.

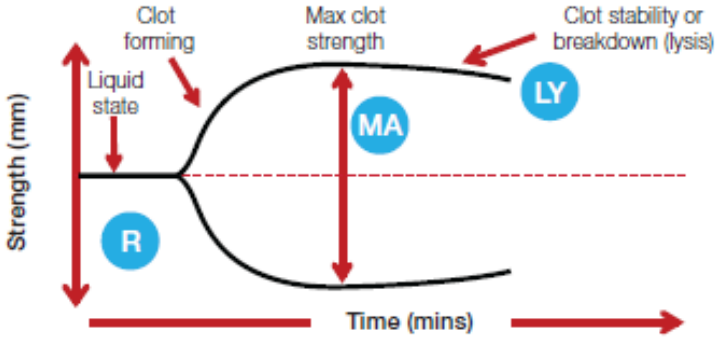
HAEMONETICS®

TEG® 6s Deficiency Assessment

TEG tracing results

Thromboelastography measures clot strength over time, providing information relative to:

- Clot rate (rate R, in mins)
- Clot strength (Maximum Amplitude MA, in mm)
- Clot stability (LYsis LY30, as a %)



Viewing cartridge results

The TEG 6s analyzer runs four tests simultaneously, providing the most specific and timely information.

The greatest sensitivity to clotting factors and heparin is achieved with the R parameter of the CK and CKH tests.

Clot strength is most rapidly assessed with the MA parameter of the CRT test, while CFF isolates fibrinogen conribution.

Deficiency assessment guide

Results from the TEG 6s analyzer should not be the sole basis for a patient diagnosis, but should be evaluated together with the patient's medical history, the clinical picture and, if necessary, other coagulation tests.

Test	Parameter	Deficiency
CK	↑ R	Clotting factors *
CKH	R < CK-R	Heparin effect
CFF	↓ MA	Fibrinogen
CRT	↓ MA	Platelets **
CRT	↑ LY30	Fibrinolysis

* In presence of heparin (CK-R > CKH-R) refer to CKH-R for adequacy of clotting factors
** If CFF-MA normal

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HAEMONETICS®

Paediatric Anaesthesia Update

Graham Knottenbelt

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Hypotension in Neonates Under Anaesthesia

What we know

- Up until recently, the observation that 'the infant woke up fine after my anaesthetic' has been the standard by which we have assessed neurocognitive outcomes.
- There is little evidence to support our definitions of hypotension of infants/neonates in common practice. Blood pressure can be a poor surrogate for perfusion.
- Infants have less cerebral auto regulatory reserve and are at higher risk for inadequate cerebral perfusion.

What this update adds

- Hypotension is not uncommon during neonatal anaesthesia.
- Intraoperative hypotension has been linked to poor postoperative neurological outcomes.
- While 'neurotoxicity of anaesthesia' grabs the headlines, cerebral oxygenation and perfusion changes associated with anaesthesia may also be a contributing factor to the neurological outcome. Indeed, hypotension and hypocapnia may be the most common avoidable complications occurring during anaesthesia in neonates.
- Cerebral desaturation detected by Near Infrared Spectroscopy (NIRS) is linked to neurological damage in humans for both extent of desaturation and duration.
- The association between blood pressure changes in infants having anaesthesia and changes in regional cerebral oxygen saturation (rScO2) using (NIRS) is described, with cerebral desaturation occurring in more than 90% of whose BP drops by more than 35% of baseline.

Management recommendations

- Anaesthetic management should focus on optimising organ perfusion and not merely on maintaining a particular blood pressure.
- Paediatric anaesthetists need to be vigilant in managing blood pressure variability in neonates, limiting substantial reductions in all neonates to no more than 20% lower than baseline.
- NIRS is a simple addition to intraoperative monitoring to estimate cerebral perfusion, especially when looking at within-patient trends.
- Though not necessary for all infants, NIRS is helpful is monitoring at-risk patients like the premature neonate, infants with cardiac abnormality or having cardiac surgery, those having extensive or prolonged surgeries like diaphragmatic hernia or tracheooesophageal repair, and severely sick infants like those with necrotising enterocolitis.

Fasting

What we know

- Due to the concern regarding aspiration, most fasting guidelines suggest a 6, 4, 2 rule (6 hours of starvation for food, 4 hours for breast milk, and 2 hours for clear fluids).
- Fasting for prolonged periods in children has been shown to increase thirst and irritability, result in greater reductions in systolic blood pressure on induction and to induce a catabolic state.

- Shortened fasting times for clear fluids improves patient perioperative experience and comfort with regards behaviour, anxiety, hunger, thirst, haemodynamic conditions and vascular access.

What is new in this review

- A 2-hour clear fluid fasting policy, even when proactively managed, results in an actual fasting time of between 4 and 13 hours.
- More paediatric hospitals are considering a liberal clear fluid regime for children not at risk for aspiration, allowing free clear □uids until called to the operating suite.
- Incidence of pulmonary aspiration is no higher with a liberal fluid regimen the than with a 2-hour rule.
- Institutional Quality Improvement (QI) processes has shown to be effective in improving fasting times.

Management recommendations

- Suggestion for a more liberal fasting regime (6-4-0 fasting regime) for low aspiration-risk cases.
- Fasting in children is an important patient-focused quality factor for care and should be incorporated into a QI process. A liberal fasting time for fluids needs to be married with other QI methodology, measuring times, targeting specific key drivers and interventions, educating and empowering ward staff, reducing confusion over procedure start times, giving parents accurate information, and reducing variation.

Tramadol and Codeine

What we know

- Children with known respiratory disease have increased opioid sensitivity. Of particular concern are those with obstructive sleep apnoea secondary to tonsillar hypertrophy or obesity.

Codeine

- Codeine is a prodrug with no analgesic effect. It must □rst be metabolised in the liver to its active metabolite, morphine.
- There genetic variability in its hepatic conversion, some children being poor metabolisers who will have less analgesic bene□t, and others who are extensive or “ultra-rapid” metabolisers who will produce higher levels of morphine.
- Within the past several years, an increasing number of case reports have illustrated clinically important respiratory depression, anoxic brain injuries and death among children receiving appropriate weight-based dosages of codeine for analgesia at home, particularly following tonsillectomy.
- Several national and international organizations (WHO, EMA, FDA) have issued advisories on the use of codeine in paediatrics, based on CYP2D6 pharmacogenetics.

Tramadol

- Tramadol is a weak opioid agonist with active analgesic activity.
- Tramadol is mainly metabolised to an inactive metabolite, and minimally through CYP2D6 to O-desmethyltrama-dol, which has a 200-fold greater affinity for opioid receptors than the parent drug.
- FDA has issued advisories on the use of tramadol in children.

What this update adds

- The warnings on the use of codeine in paediatrics are justified.
- Tramadol is not codeine and, despite the FDA warning, tramadol still a reasonable choice of analgesia for children.
- Tramadol overdose is a greater danger than CYP2D6 variants. None of the reported tramadol deaths were related to the metabolites, but rather over dosage itself, OSA and obesity.
- The droplet formulation of tramadol should no longer be available in New Zealand and has been replaced with a more preferable tramadol 10 mg/ml elixir.
- One drug that could replace tramadol is tapentadol, a drug with similar mechanism of action to tramadol but analgesic activity that is independent of enzyme systems and that has no active metabolites.
- Pharmacogenetics is an exciting and rapidly expanding □eld. The clinical availability, affordability, and practicality of personalised pharmacogenetics and prescribing remains to be seen.

Management recommendations

- Codeine offers no unique benefits over other opioids and has several well-documented negatives. It should not be used in children under 12 years old.
- A multimodal opioid-sparing analgesia strategy reduces the need for perioperative opioid use.
- Tramadol is still a reasonable choice of analgesia.
- Tramadol dose should be limited for acute pain after tonsillectomy (e.g. dose 1 mg/kg 6-8 hourly, max 400 mg/day). Children with OSA who have undergone tonsillectomy should be monitored in hospital overnight.

Anaesthesia-Induced Developmental Neurotoxicity

What we know

- There is compelling evidence from animal and laboratory studies suggesting that early exposure to general anaesthesia is detrimental to normal brain development, leading to structural and functional impairments of neurons and long-lasting impairments in normal emotional and cognitive development.
- The evidence from human studies is inconsistent and not conclusive at present. Up until recently, the vast majority of human studies were retrospective cohort studies. The findings were mixed but generally show weak evidence for an association though all have major weaknesses of confounding factors.
- In December 2016, the FDA issued a warning statement regarding the use of anaesthesia or sedation in young children highlighting potential risk of anaesthetic procedures that last longer than 3 hours or multiple procedures required in children less than 3 years of age. Evidence to support such warning is currently insuf□cient and incomplete.

What this update adds

- Some of the most important studies have appeared recently. Discussion focuses on new developments in translationally relevant NHP animal studies of anaesthesia-induced developmental neurotoxicity and summary of five recent human clinical studies: three large population-based studies; and two prospective human trials, the General Anaesthesia compared to Spinal anaesthesia (GAS) trial and The Paediatric Anaesthesia Neurodevelopment Assessment (PANDA) trial.

- These studies do allow us to draw some cautious conclusions: that short-term single exposure of 60 minutes or less to surgery and anaesthesia is not associated with measurable long-term neurodevelopmental problems.

Management recommendations

- Neurotoxicity of anaesthetic agents is an important issue for anaesthetists, but we need to place it into context of other confounding stresses on brain development and how the potential risk of anaesthesia could change practice. Should we modify the anaesthetic technique, delay surgery until older, or treat with drugs that may attenuate any harmful effects?
- The ESA/ESPA/EACTA/EuroSTAR consensus statement amount to a pragmatic and sensible two statements:

    1. No child or pregnant woman should ever undergo any medical procedure that is not necessary or done for trivial reasons.

    2. Established safe anaesthetic techniques delivered by trained and experienced staff in a paediatric environment supported by the necessary clinical organisation are essential factors for the delivery of safe anaesthesia and sedation in children.

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Tramadol/Codeine

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Point of Care Ultrasound in the Perioperative Setting

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The use of point of care ultrasound (POCUS) in the perioperative setting is rapidly increasing. Indications are expanding, and a growing body of literature supports its efficacy for improving patient management and outcomes.[1] The use of POCUS has become standard practice for vascular access, regional anaesthesia, cardiac, and chest assessment in the intensive care and anaesthesia environments, and in the emergency department. Training in POCUS is now mandatory for intensive care medicine and emergency medicine, and is supported in anaesthesia, but not yet mandated. POCUS encompasses a wide range of modalities and scope of practice – and as such, multiple professional societies and colleges have proposed guidelines and standards of training. [1 2 3 4 5] As machines become smaller, more portable, and less expensive, and the utility of POCUS is further demonstrated, use is expected to become nearly ubiquitous. This talk will focus on the current use of echocardiography and ultrasound by non-cardiac anaesthetists, in the perioperative setting.

Transoesophageal Echocardiography

Whilst transoesophageal echocardiography (TOE) skills are generally the domain of cardiologists and cardiac anaesthetists, the role of TOE in the perioperative setting for diagnosis of unexplained haemodynamic instability, and in the management of high risk and prolonged surgeries for co-morbid patients is well established. Non-cardiac anaesthetists may make use of TOE by referral to colleagues (e.g., a combined intra-operative approach with two anaesthetists), or with training and interpretation at the basic level. As with all modalities in POCUS, defined scope of practice and appropriate level of training and credentialing are essential.[6] Basic and advanced levels of TOE certification have been established in the United States by the National Board of Echocardiography (NBE), and in Canada by the Canadian Cardiovascular Society and Canadian Society of Echocardiography, whilst in Europe and the United Kingdom certification for TOE is at an advanced perioperative level. Guidelines for the performance of TOE are available from the American Society of Echocardiography (ASE), and The European Association of Cardiovascular Imaging (EACVI).[7] TOE is useful for both limited and comprehensive examination of the heart, great vessels, lungs and pleura.

Transthoracic Echocardiography and Focused Cardiac Ultrasound (FOCUS)

Transthoracic echocardiography (TTE) is used more frequently than TOE, and is less invasive, often more portable, and associated with less cleaning and sterilisation costs. TTE probes and machines can be used not only for cardiac ultrasound, but for imaging of the great vessels, the chest and airway, and abdominal structures. Suitably qualified and trained clinicians can perform TTE in the perioperative setting, and guidelines exist once again for training and qualification. [5] FOCUS refers to use of surface echocardiography and can encompass a broad range of studies and uses, from a comprehensive diagnostic echocardiogram ("formal TTE") to a Focus Assessed Transthoracic Echo (FATE) protocol echocardiogram, which examines five basic views, and is used to specifically rule in or rule out limited pathology or conditions. Diagnosis of gross LV and RV pathology (dilatation, severe systolic impairment), pericardial collections, severe valvular pathology (in particular severe aortic stenosis) can be made reliably using FOCUS. The interpretation of volume status and afterload has also been described using FOCUS, and whilst in patients with previously normal cardiac status and supporting physiologic variables this can be reliable, there are several caveats that are important to understand to avoid misdiagnosis or incorrect interpretation of echocardiographic findings. [8 9] These will be discussed during the presentation.

Lung and Airway Ultrasound

Chest pathology such as pleural collections, pneumothorax, pulmonary oedema, lung atelectasis and airway pathology may be diagnosed (and the response to management such as drainage via catheters or needle aspiration, assessed) with POCUS. Ultrasound has been used to identify the trachea and cricothyroid membrane in patients with difficult anatomy (such as obesity), and to identify vascular abnormalities prior to front of neck access (planned or unplanned). Ultrasound has proven utility in these areas also, as a rapid and readily available, non-invasive and reliable method of assessment. [10] [1]

Other Areas

Point of care ultrasound is used for other diverse tasks. Optic nerve ultrasound allows diagnostic information relating to intracranial pressure, whilst ultrasound is also used to detect intraocular foreign bodies, retinal detachment, vitreous haemorrhage, and retrobulbar haemorrhage. [1] Vascular uses are well beyond the scope of this paper, however are well documented and include assessment of vessels, confirmation of anatomy, and use for guidance during placement of catheters or intravascular procedures. Use during regional anaesthesia is of proven benefit, lowering the number of needle passes required, improving block onset time, and avoiding vascular injury, and is now a standard of care. [11] POCUS for abdominal diagnosis and management is increasingly described, and can be used to examine indwelling urinary catheters placement and efficacy, abdominal collections and fluid, gastric volume and aspiration risk, and the vena cavae and other vessels for information on volume status, and right heart function. [1] [12] [13]

Summary

The use of POCUS is increasing, due to the wide variety of clinical applications and the proven efficacy, as well as the relatively short and steep learning curve for novice practitioners to become competent in basic and limited assessments. Using ultrasound is a standard of care in several areas of anaesthetic practice currently, and this is likely to expand (along with training requirements) in the future.

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A Short Degustation of Regional Anaesthesia

Justin Holborow

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At AQUA I have chosen to discuss 1) IV vs perineural dexamethasone for postoperative pain 2) an anatomical update for the adductor canal block, 3) phrenic nerve sparing regional anaesthesia of shoulder surgery and 4) a useful tip with lumbar spinal sonography. Other hot topics in regional anaesthesia include ultrasound education, stop before you block, awake vs asleep blocks, paravertebral alternatives for breast surgery, and epidural alternatives (trunk block) regional anaesthesia for abdominal surgery including Erector Spinae, Quadratus Lumborum and TAP blocks.

Dexamethasone: IV or Perineural

For those regionalists who are not routinely placing perineural catheters, there is always interest in finding local anaesthetic additives to increase the longevity of a single shot block. Dexamethasone has emerged as the most promising of the potential additives currently available. There are now many studies showing its benefit over placebo prolonging analgesia by an extra 10 hours **Choi BJA2014;112(3):427-39**. This is highly significant because it allows a patient to get through the first night post-operatively without requiring transitional analgesia at an unfavourable time. IV dexamethasone is known to have an anti-inflammatory and significant analgesic effect. **DeOliviera Anaesthesiology 2011; 115:575-588**. Interestingly, it then emerged that dexamethasone given intravenously was just as effective as dexamethasone administered perineurally. ***In 2013 Desmet. BJA 2013;111(3):445-53, Abdallah et al RAPM 2015;40(2)125-132.*** This created uncertainty as to the optimal route of dexamethasone administration particularly since perineural dexamethasone is an off licence indication and has an unproven long term safety record. Recently more light has been shed on this topic with a meta-analysis by **Chong et al RAPM 2017;42(3)319-326.** They found that the time to first analgesic request was 3.77 hrs longer in the perineural group. However, subgroup analysis showed no difference in the interscalene group. The decision to use perineural dexamethasone should be on a case by case basis based on surgery location, type, transitional pain timing, discharge planning and surgical and anaesthesia culture and perceived safety.

Adductor Canal Block: Anatomical update

There has been much interest recently in the adductor canal block for post knee arthroplasty analgesia either as a stand-alone analgesic technique or in combination with local anaesthetic infiltration. The movement away from the usual quadriceps paralysing femoral nerve block is due to a greater emphasis on early post-operatively mobilisation, and has been made possible by the availability of ultrasound.

Studies show the adductor canal block is equi-analgesic for post knee arthroplasty, with less quadriceps weakness. **Jaeger RAPM 2013;38(6):526-532** To date there is limited evidence that this has made a difference in falls or improved function **Elkassabany Anesth Analg 2016; 122:1696-703** as significant multi-factorial post-op quadriceps weakness still occurs independent of anaesthetic technique.

Initial studies describe a “mid-thigh” sub sartorial injection half way between ASIS and the base of the patella calling it an “adductor canal block”. **LUND Acta Anaesthesiol Scand 2011; 55:14-19** However this is technically in the low femoral triangle above the beginning of the adductor canal. Case reports of local anaesthetic spreading cephalad and causing a true femoral nerve block have been described. **Chen RAPM 2014; 39:170-171. Veal. Acta Anesthesiol Scand 2014; 58:362-364.** Despite this anatomical fact being pointed out in 2014 **Bendtsen et al. RAPM 2014 39(3):253-254, RAPM 2014;39(5):442-443** there has been subsequent confusion in the literature about the terminology for the regional anaesthesia being provided, the definition of the adductor canal block, its contents and their relative significance in sensory innervation of the knee joint. This has made meta-analyses on the topic difficult and further

definition of the anatomy needs to be confirmed to allow useful comparisons. (**Hussain RAPM2016;41(3):314-320**. Recently there have been several anatomical studies conducted trying to shed light on this issue. (**Bendtsen RAPM 2016;41(6)711-719, Burkett RAPM 2016;41(3):321-327, Wong RAPM 2017;42(2):241-245**

The adductor canal begins at the intersection of the medial border of the sartorius and adductor longus muscles. This point is easily determined using ultrasound landmarks **Fig 1** (**Wong RAPM 2017;42(2):241-245)** and is distal to the midpoint of the thigh previously explained. **Fig2**. (**Wong RAPM 2017;42(2):241-245)** It finishes at the adductor hiatus where the vessels dive deep into the popliteal fossa. It is bound anteromedially by vasto-adductor vastus medialis and posterolaterally by adductor longus then adductor magnus. **Bendtsen RAPM;39(5):442-443.**

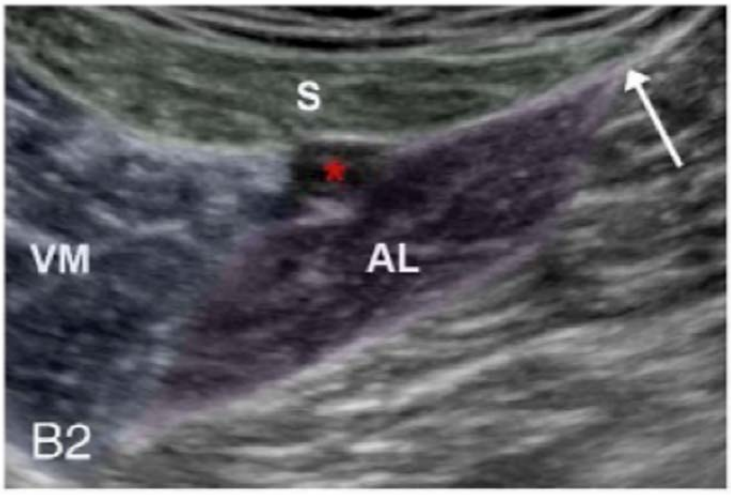


Fig 1: Depicts the beginning of the AC defined as the apex of the FT, where the medial border of the sartorius muscle intersects the medial border of the adductor longus muscle (white arrow in B2). This level corresponds to the blue arrows in Figure 2. AL (purple), adductor longus muscle; AM (orange), adductor magnus muscle; asterisk (red), femoral artery; F, femur; S (green), sartorius muscle; SM (yellow), semimembranosus muscle; VM (blue), vastus medialis muscle.

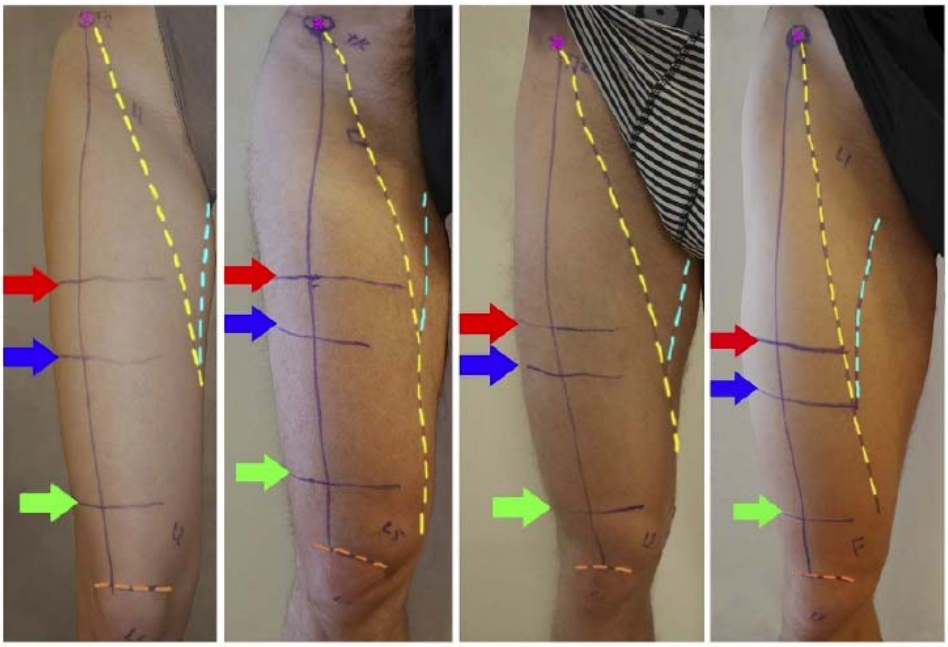


FIGURE 2. The figure shows the thighs of 4 volunteers. The midpoint of the thigh (red arrow) is defined as half the distance between the ASIS (pink asterisk) and the base of patella (orange stippled line) corresponding to the ultrasound images in Figure 1A. The proximal end of the AC (blue arrow) is defined by the intersection of the medial border of the sartorius muscle (yellow stippled line) and the medial border of the adductor longus muscle (cyan stippled line) corresponding to the ultrasound images in Figure 1B. The distal end of the AC is defined as the adductor hiatus (green arrow) corresponding to the ultrasound images in Figure 1C.

There is debate and inter-individual variability about which nerves travel through the adductor canal and the significance of these nerves in post knee arthroplasty nociception.

These nerves include the saphenous nerve, the nerve to vastus medialis and the obturator nerve.

Local anaesthetic injected into the low femoral triangle above the beginning of the true adductor canal will reliably block the saphenous nerve and the nerve to vastus medialis however volumes >20mL run the risk of causing quadriceps weakness. **Jaeger P. Br J Anaesth 2015;115(6): 920-926**
A distal adductor canal block just proximal to the adductor hiatus runs the risk of missing the nerve to vastus medialis, and potentially the infrapatellar branch of the saphenous nerve in a small group but also spreading through the adductor hiatus posteriorly and causing a sciatic nerve block with resultant foot drop.

The optimal adductor canal block is a low volume <20mL proximal adductor canal or low femoral triangle block. The best way to determine this location is to use ultrasound landmarks **Fig 1** rather than traditional external anatomical landmarks.

Phrenic nerve sparing regional anaesthesia for shoulder surgery

The interscalene block is the gold standard regional anaesthetic technique for shoulder surgery. Unfortunately despite dose reduction it cannot reliably be provided without a coinciding phrenic nerve block and hemiparalysis of the diaphragm. Most of the time this is extremely well tolerated and of no clinical significance. Unfortunately in a small subset of the population with minimal respiratory reserve, a phrenic nerve block can be a significant clinical problem.

The phrenic nerve is derived from C3,4,5 and runs down the ventral surface of the anterior scalene muscle. Its proximal end lies very close to the brachial plexus and interscalene groove, but its path diverges from the brachial plexus as it descends. The innervation of the shoulder joint significant in post-operative nociception includes the suprascapular and axillary nerves with a lesser contribution from the lateral pectoral, upper and lower suprascapular nerves. The suprascapular nerve leaves the brachial plexus proximally at the level of the trunks. Therefore, a single injection intending to block these nerves needs to be near the trunks i.e. a interscalene or supraclavicular block.

Recently there has been increased interest in solving this problem with a number of excellent review articles. **El-Boghdadly Anaesthesiology 2017;127(1)173-191, Tran RAPM 2017;42(1):32-38.** All of these strategies aim to deposit local anaesthetic more peripherally away from the phrenic nerve.

The strategies presented include

a) **Low dose interscalene block:** Interscalene block causes phrenic nerve block via cephalad or ventral spread. Ultrasound has allowed smaller doses to be used. Despite many attempts the lowest incidence was 27% with 5ml Ropivacaine 0.75% **Studner BJA2016;116(3):405-412.**

b) **Extra-fascial interscalene block**: 20ml Bupivacaine 0.5% deposited 4mm lateral to C5+6 nerve roots in belly of middle scalene muscle has been shown to reduce the incidence to 21% **Palhais BJA 2016;116(4):531-537.**

c) **Low dose C7 nerve root block**: In 2009 Renes et al showed that 10mL Ropivacaine 0.75% posterolateral to the C7 nerve root reduced phrenic nerve block to 13%. **Renes RAPM 2009; 34:498-502.** The C7 foramen is known to a highly vascular area so further studies need to confirm its safety as well as efficacy for surgical anaesthesia.

d) **Supraclavicular brachial plexus block**: The supraclavicular approach has a lower incidence of phrenic nerve palsy however it is still significant with high volume multi-injection techniques affording an incidence of phrenic nerve blockade of up to 34%. This incidence can be reduced to 0% with reduced volume 20ml and targeting of local anaesthetic posterolateral to the brachial plexus. **Renes RAPM 2009; 34:595-599** Unfortunately this was not in patients undergoing shoulder surgery so its utility in providing

complete shoulder anaesthesia is still untested. Cornish et al achieved similar results by depositing local anaesthetic lateral and inferior to the brachial plexus with a novel bent needle technique. **Cornish et al Anaesthesia. 2007;62:354-358.**

e) **Combined suprascapular and axillary nerve blocks:** This combination has the advantage of no phrenic nerve blockade, minimal motor block, less transitional analgesia issues however less reliability at providing complete anaesthesia for major shoulder procedures. **Dhir RAPM 2016;41(5):564-571.**

f) **Variations of Suprascapular nerve and posterior and lateral cord blocks:** Recently there have been several new approaches to anaesthetise the suprascapular nerve and posterior and lateral cords. **Rothe** (**Casanova BJA 2016;117(6):835. Taha BJA 2017;119(1)110-171 Sondekoppam BJA 2016; 117(6):831-832: not studied.** Theoretically they should provide excellent shoulder anaesthesia with no phrenic nerve blockade. Further studies are required to see whether this is true and how they stack up against the interscalene block.

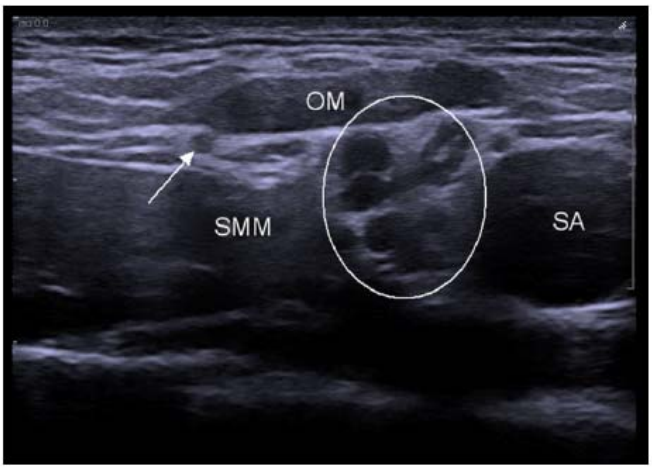


Fig. 1. Ultrasonographic image of the supraclavicular fossa. The transducer is positioned in an oblique sagittal plane. The left side of the image is oriented posterolaterally. Important landmarks are the subclavian artery (SA), the omohyoid muscle (OM), the scalenus medius muscle, and the supraclavicular part of the brachial plexus (encircled). The arrow marks the suprascapular nerve (SSN) in a fascia layer under the OM in close relation to the brachial plexus.

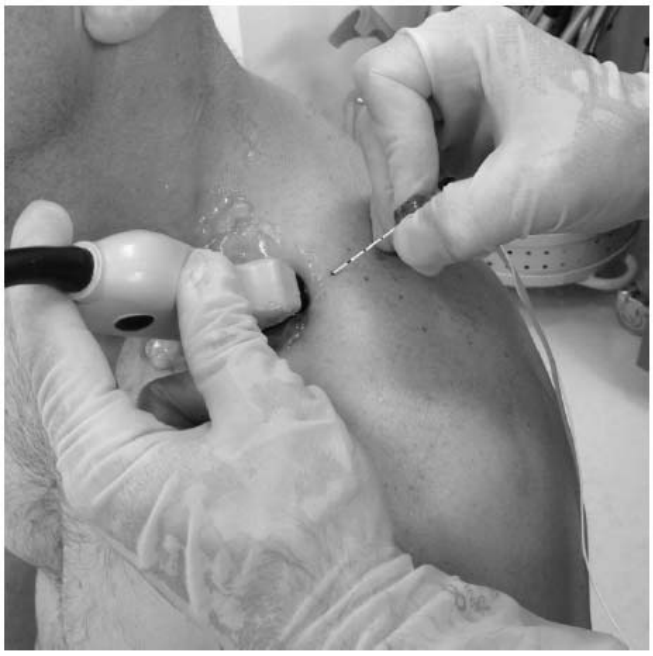


Fig. 2. Volunteer, transducer, and needle position. Lateral view of the shoulder region demonstrating in-plane needle insertion at the upper margin of the trapezius muscle. The transducer is orientated in the oblique sagittal plane and the hand of the operator is resting on the clavicle. The shoulder joint is in neutral position and the hand of the volunteer is resting on the thigh.

Lumbar Spine Sonography

Clinical palpation is the mainstay for performance of lumbar neuraxial blockage despite lumbar spine sonography being available for over a decade. There is good evidence that in patients with unpalpable or abnormal anatomy, lumbar spine ultrasound pre-scan reduces overall procedure time, needle passes, allows accurate interspace selection and depth calculation**. Perlas RAPM 2016; 41:251-260** Despite this, it is my impression this tool is rarely practiced amongst colleagues. I am finding myself using an ultrasound pre-scan more and more useful and now do it routinely for anyone with unpalpable or abnormal anatomy.

My approach was initially developed in Perth under the guidance of Dr Chris Mitchell. This is the same technique described by **Carvalho. (anaesth clinics 2008;26:145-148)** with greater emphasis on the anterior wall of the spinal canal.

My Routine

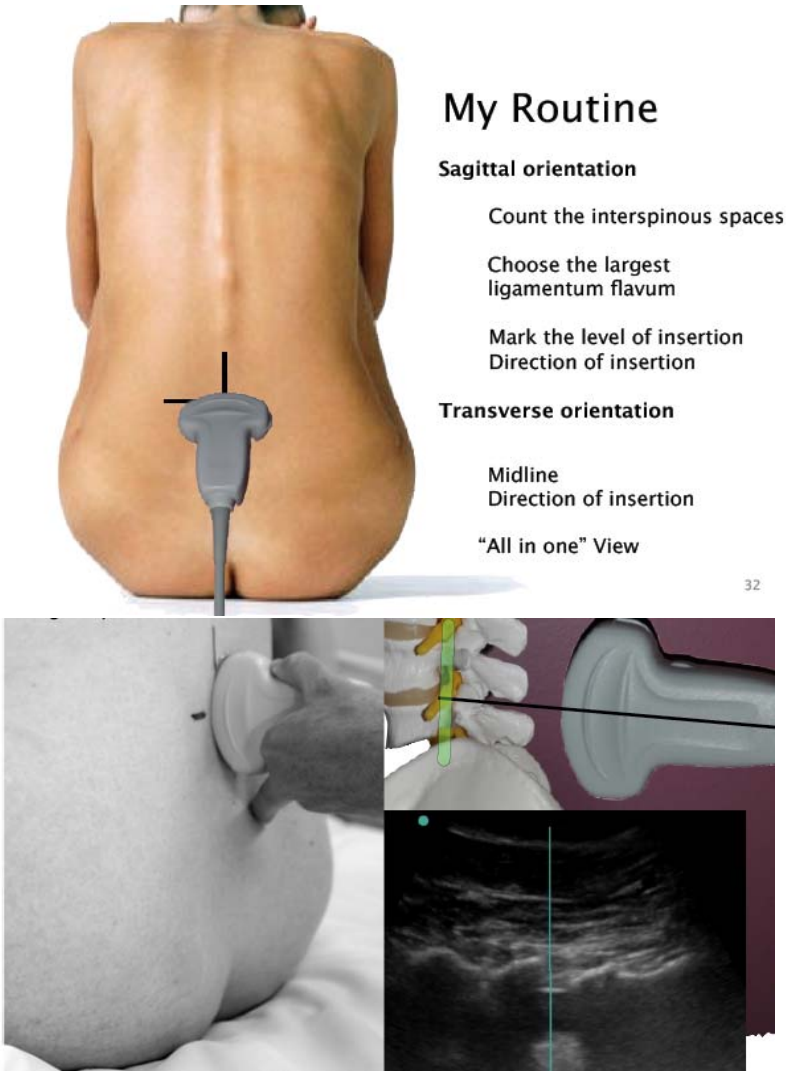
Firstly, I make sure I have an ultrasound with a low frequency curvelinear probe, sterile probe cover and sterile pen. I then get scrubbed, gowned, gloved, prepared and position the patient for the procedure. I think this is important to scan the patient in exactly the position of the procedure and immediately before.

Saggital paramedical view (see figure)

With the probe in the saggital paramedian orientation the smooth line leading into the saw tooth pattern of the lamina can be seen. This allows accurate inter vertebral space numbering. I focus my attention on looking through these spaces to the reflection of the anterior wall of the spinal canal. The size of this acoustic signal is proportional to the size of the boney acoustic window. This indicates which interspace is largest and most favourable. In a young patient, there may be many favourable spaces. In an older patient or one with abnormal anatomy there may be fewer choices.

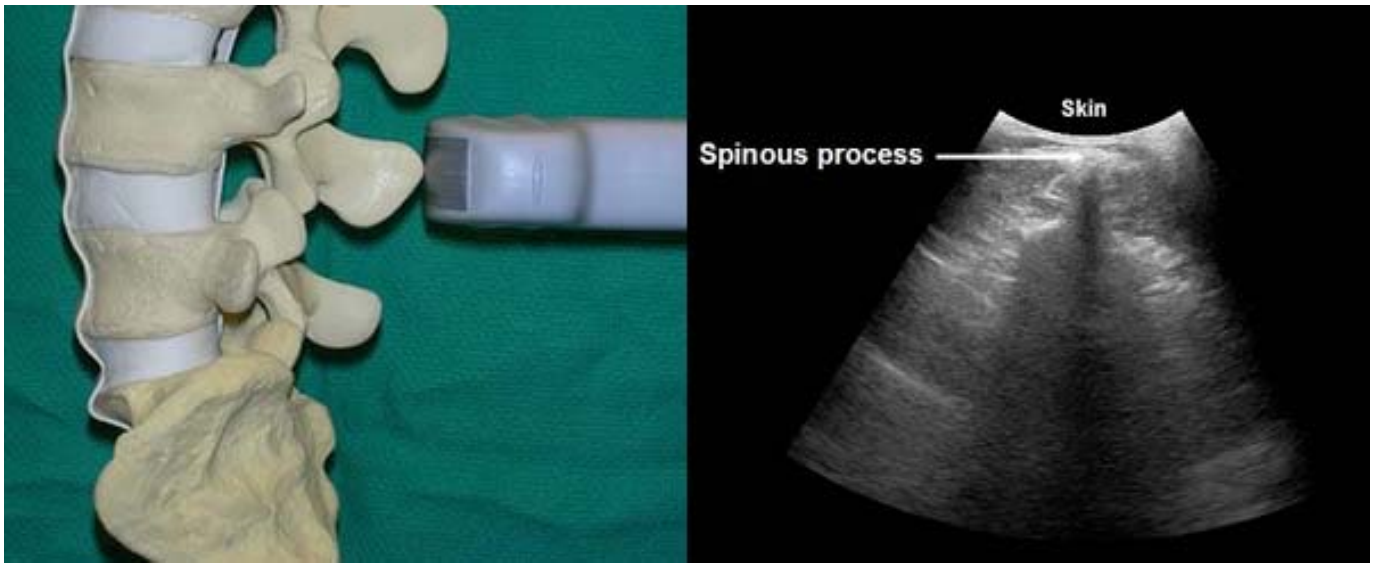
Transverse medial view

With the probe in the coronal view, one can assess the midline, angle of insertion in the horizontal and vertical plane, and depth of the epidural space. Determining the midline and interspace in best done by firstly finding a spinous process. This forms a bright hyperechoic signal close to the surface in the midline with a cone shaped acoustic shadow underneath. The probe can then be slowly moved cephalad or caudad off the spinous process into the intervertebral space where the acoustic shadow is replaced by bat head shaped shadow of the facet joints. . The anterior wall of the spinal canal is then visible in the midline. One visible this gives the “all in one view”

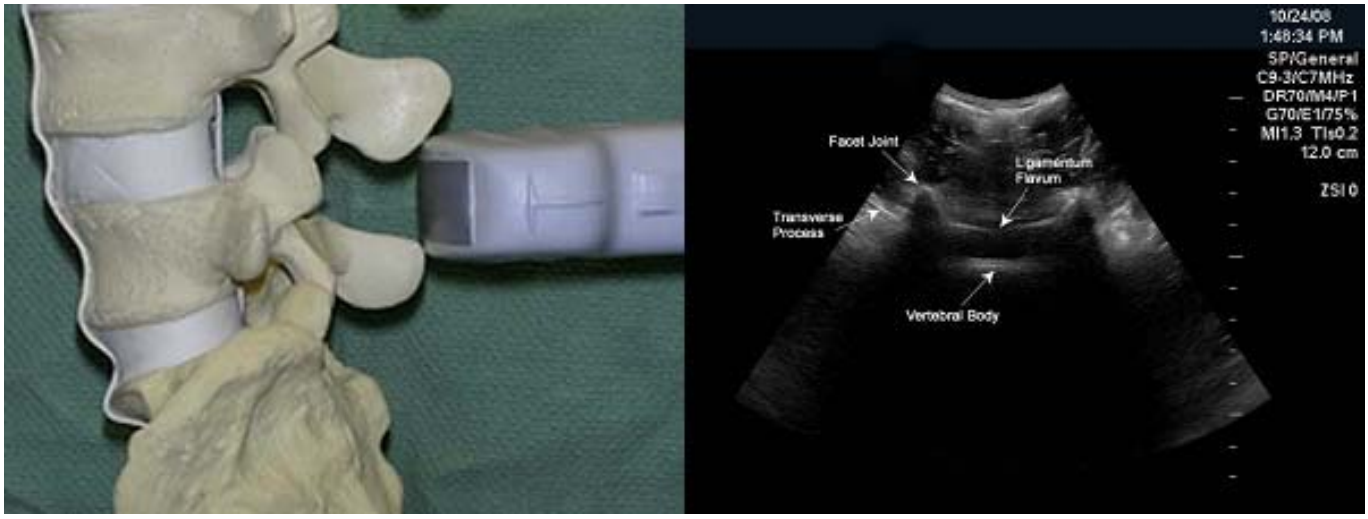


Saggital paramedical view. The green line in the ultrasound image passes down between the saw tooth appearing laminae, through the intervertebral spaces and spinal canal and shows the acoustic signal from the anterior wall of the spinal canal

Transverse medial view of spinous process



Transverse medial view showing bright line of vertebral body aka anterior wall of the spinal canal. I use this as an indicator of a large acoustic window.



Conclusion

Perineural dexamethasone prolongs the time for first analgesic request compared to intravenous dexamethasone by about 3.5 hours. However there is no difference with interscalene blocks.

The adductor canal can be defined using ultrasound landmarks at the intersection of the medial borders of adductor longus muscle and sartorius muscle. This is distal to the usual mid-thigh location traditionally used when performing an adductor canal block. The exact components and relative contribution of nerves to post knee arthroplasty nociception is still being defined.

Phrenic nerve blockade cannot reliably be avoided with a single block regional anaesthetic technique for shoulder surgery. There has been an advance in different approaches for blocking peripheral nerves to provide complete anaesthesia for shoulder surgery.

When performing lumbar spinal ultrasound pre-scan for epidural and spinal anaesthesia, emphasis on the acoustic signal of the anterior wall of the spinal canal helps choose the most favourable space.

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Perioperative risk associated with aortic stenosis in non-cardiac surgery

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Introduction

The magnitude of the perioperative risk associated with AS is relatively unknown. The last prospective trial was Goldman's derivation of the cardiac risk index in 1977[1]. The study had 23 patients with clinically diagnosed AS. Recent work has been retrospective with varying results. In three relatively large retrospective controlled trials two found no difference between the controls and asymptomatic patients with severe AS, and one an increase in major cardiac adverse events (MACE) but not mortality[2,3,4.] Apart from the retrospective nature, the trials had variable control criteria and different surgical populations in particular with regard to emergency and elective surgery. Our aim was to record the risks associated with AS in non-cardiac surgery and to try and identify pre-operative variables associated with the risk.

Method

Five centres in NZ agreed to participate in the study (Nelson, Auckland, Christchurch, Whangarei, Wellington). Patients were recruited pre operatively and information collected on a number of variables including demographics, co-morbidities, frailty, symptoms, echo parameters and laboratory investigations. Post operatively, 30-day mortality, one year mortality and major adverse cardiac events were recorded as outcomes. Regression analysis was used to identify univariate and then significant multivariate variables associated with these outcomes.

Results

147 patients were entered into the study. Severe AS was present in 61 (41.5%) and 86 (58.5%) had moderate AS. 104 patients (70.8%) had elective surgery and 43 (29.2%) emergency surgery. 31 patients (21%) patients died within 1 year and 13 (9%) of these within 30-days. 33 (22%) had a MACE. Significant univariate variables associated with all 3 outcomes were age, ASA, emergency surgery, NYHA classification, albumin, frailty and a history of congestive heart failure. Aortic valve area, surgical risk and MR were associated with mortality only. On multivariate analysis only emergency surgery was associated with all 3 outcomes. Aortic valve area and albumin were associated with 30-day mortality. Symptomatic AS was associated with increased MACE but not mortality.

Discussion

The incidence of AS in first world populations is largely related to advanced age, particularly over the age of 75 years. As the size of the elderly population increases so too will the influence of AS on perioperative outcome. This study identified AS is associated with 30-day mortality after non cardiac surgery. The increased risk is related to valve area with increasing valve area reducing risk (OR 0.03, CI 0-0.4). Emergency surgery was the only variable associated with 30-day mortality, one year mortality and MACE. Patients with low albumin had increased MACE's and were more likely to die at 30 days post

operatively. Translating these findings into a clinically useful approach can be done in a number of ways. At a simplistic level the crude rates of 30-day mortality (9%) and MACE (22%) can be used as a starting point and adjusted according to surgery type, symptomatic status, valve area etc. A more complex approach would be to calculate the expected mortality using existing morbidity and mortality calculators and then adjust to aortic valve area with each reduction in 0.1cm^2 increasing mortality by 10%.[5].

Summary

The study encourages anaesthetists to view reduction in valve area as a risk factor rather than categorisation into moderate and severe AS and focuses outcome on mortality. It also defines the role of emergency surgery, symptoms and albumin in this patient population.

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Opioid epidemic in the USA. Lessons learned…

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Case and Deacon

On 8th December 2015 a remarkable paper was published in the *Proceedings of the National Academy of Sciences*.[1] The authors were Anne Case and Angus Deacon, a husband and wife team from the Woodrow Wilson School of Public and International Affairs, Princeton University. Anne Case is a Professor of Economics and Public Affairs and Sir Angus Deacon is the Dwight D. Eisenhower Professor of International Affairs and Professor of Economics. He is also the winner of the 2015 Nobel Prize in Economics. Their paper, entitled, "Rising Morbidity and Mortality in Midlife Among White, Non-Hispanic Americans in the 21st Century", identified an increasing death rate amongst white Americans – particularly whites with limited education in their middle years –a trend beginning in the late 1990s and continuing to the present day. Over the same time period, death rates for Hispanics and blacks in the United States (US) continued to decrease, as did death rates for adults in other first world countries. Case and Deaton identified three causes of the increase: chronic liver disease, suicide, and, most importantly, poisonings. While such a trend in a first world country during peacetime is not unprecedented – it happened in Germany following the Second World War and in Russia after the fall of the Soviet Union – it is, nevertheless, highly unusual. The authors commented that, while the causes are incompletely understood, there was a strong link to the increased availability of opioid prescriptions during the same time period.

Case and Deacon's paper received considerable attention in the media. "The Dying of the Whites" (Ross Douthat, *New York Times*, November 7, 2015), "Middle-Aged White Americans are Dying of Despair" (Olga Khazan, *The Atlantic*, November 4, 2015), and "Why Are So Many Middle-Aged White Americans Dying?" (Olga Khazan, *The Atlantic* January 29, 2016) are three examples, but there are 100s of such essays to be found online.

Just how bad is it?

Over the last two years, in both the medical literature and the media, the excess mortality amongst poorly educated whites, first identified by Case and Deacon, has been increasingly linked to opioid overdose. One of the most compelling articles addressing rising mortality from drug overdose is from Josh Katz. In an article "Just How Bad is the Drug Overdose Epidemic?", published in the *New York Times* in April of this year, Katz quotes figures from the National Center for Health Statistics, Centers for Disease Control and Prevention.[2] Katz identifies 52,404 deaths from drug overdoses in 2015, up from 8413 in 1990, a 500% increase. The figure of 52,404 compares to 37,757 deaths from car accidents, 35,763 from guns, and 6,465 from HIV/AIDS in the same year. Given that approximately 60% of gun deaths are from suicide, in 2015 deaths from drug overdoses exceeded that of gun homicide and car accidents combined, and the rate of increase in overdose deaths is similar to the death rate increase during the worst period of the HIV/AIDS epidemic in the late 1990s. Drug overdose is now the leading cause of death in people under 50 in the US. In short, the current drug epidemic is the worst in US history.

Unlike other recent drug crises, such as crack cocaine in the 1980s and methamphetamine in the early 2000s, this epidemic involves opioids, most commonly taken in the form of prescription drugs – oxycodone (OxyContin, Percocet) and hydrocodone (Vicodin) – or as heroin. More recently, illegally imported fentanyl and other synthetic opioids have become more common.
The epidemic has a very uneven geographic distribution.[2,3] The worst affected regions are the rural, socially disadvantaged, parts of Appalachia (West Virginia, Kentucky, Pennsylvania, and southern Ohio)

and New England (New Hampshire, Massachusetts, and Maine). In the worst affected state, West Virginia, the death rate during 2014-2015 was approximately 40 per 100,000 people.[3] During the same time period, the death rate in Nebraska, the 25th worse state, was approximately 7.5 per 100,000 people. Within West Virginia itself the death rate during 2014-2015 varied from county to country, from less than 10/100,000 to more than 130/100,000 deaths per persons, being concentrated in poorer, more rural communities. Again, the concentration in rural communities is in contradistinction to urban-based drug problems of the past: The New York heroin scene of the 1960s and 1970s and the crack cocaine epidemic of the 1980s, which occurred predominantly in poor parts of large urban centres, such as Chicago and Detroit.

Porter and Jick

In 1980, a one paragraph letter appeared in the *New England Journal of Medicine* under innocent-sounding title, "Addiction Rare in Patients Treated with Narcotics".[4] In it, Jane Porter and Hershel Jick, from the Boston Collaborative Drug Surveillance Program, briefly outlined how they encountered only four documented cases of addiction (only one of which was "severe") amongst 40,000 hospitalised patients, of whom 12,000 received at least one opioid prescription. The authors didn't describe any post-hospital follow-up (there wasn't any) and did not describe their methodology. However, the figure – less than 4 in 12,000 patients – transmogrified into a widely quoted figure of "less than 1% risk of addiction".

Six years later Russell Portenoy and Kathleen Foley, writing in the journal *Pain*, described treating 38 patients with opioid analgesics for chronic, non-malignant pain,[5] a practice that had hitherto been rare. These two papers were picked up by the media. Ronald Melzack, writing in *Scientific American* in 1990 under the title, "The Tragedy of Needless Pain",[6] wrote, "Contrary to popular belief, morphine taken solely to control pain is not addictive. Yet patients worldwide continue to be undertreated and to suffer unnecessary agony". Sam Allis, writing in *Time* magazine in 2001 in an article entitled "Less Pain, More Gain" described Porter and Jick's one paragraph letter as a "landmark study" and claimed it demonstrated that, "The exaggerated fear that patients would become addicted to opiates was basically unwarranted".[7] Russell Portenoy, being interviewed in the *New York Times* in 1993 claimed, "There is a growing literature showing these drugs (opioids) can be used for a long time with few side-effects and that addiction and abuse are not a problem".

And so, from these humble beginnings, and in the absence of any meaningful evidence of safety, opioid prescribing for chronic non-malignant pain commenced in earnest. Prescriptions for opioid analgesics increased three-fold between 1999 and 2010, a 10-year period (data from the US Annual review of Health). During the same time period, deaths from opioid overdoses increased four-fold and admissions for opioid treatment increased three-fold.

As befitting a bona-fide treatment, in 2009 clinical guidelines for the use of chronic opioid therapy for chronic non-cancer pain were published.[8] While taking a relatively favourable position to the use of opioids for chronic, non-cancer pain, the guidelines have very little to say on the risks associated with such therapy. They do state, however, that, "Estimates of aberrant drug-related behaviours, drug abuse, or misuse in patients range from 0% to 50%" (!). In the disclosures, 11 of the 20 authors report receiving industry-sponsored research funding and 12/20 list having received industry honoraria, including the now famous, Russell Portenoy.

In fact, chronic pain *is* a serious unmet need in the US. In a 2010 survey of 90,000 patients, one in five respondents – equating to 39 million US citizens – described having persistent pain, defined as pain on most days for longer than three months.[9] Unfortunately, it is increasingly apparent that using opioids to treat chronic non-cancer pain is harmful, over and above the risk of overdose. In a recent article in *JAMA* patients treated with long-acting opioids for chronic, non-cancer pain had an increased mortality

compared to propensity matched controls, including for non-overdose deaths (Hazard ratio 1.72, 95% confidence interval 1.24-2.39).[10]
By 2011, in the thick of the prescription opioid crisis, Russell Portenoy had had a change of heart. In an interview released by Physicians for Responsible Opioid Prescribing he had this to say: "None of the papers presented any real evidence, and yet what I was trying to do was create a narrative so that the primary care audience would look at this information *in toto* and feel more comfortable about opioids…because the primary goal was to destigmatise we often left the evidence behind".

Purdue Pharma: an American success story

Purdue Pharma is a privately owned pharmaceutical company founded in 1952 and based in Stamford Connecticut. Purdue began life innocuously enough producing laxatives, earwax remover, and antiseptics. However, in 1991 Purdue began focussing on pain management, in particular oral formulations of opioid analgesics. It has on its books MS Contin (morphine), Dilaudid (hydromorphone), Butrans (buprenorphine), and – the 900-lb gorilla of them all – OxyContin (sustained release oxycodone). Concurrent with the shift to 'pain management' Purdue's fortunes have blossomed. By 2015 *Forbes* magazine estimated Purdue was worth USD $35 billion. Growth has come almost exclusively from sales of opioid analgesics, most importantly OxyContin. The company is privately owned by the Sackler family. Again in 2015, *Forbes* 'conservatively' estimated their wealth at USD $14 billion, money made almost exclusively from Purdue and its sale of OxyContin.

OxyContin was approved by the FDA in 1996 and marketed as a safe alternative to other opioids for patients with moderate and severe pain. The long-acting formulation was touted as avoiding the intense high sought by addicts and it was claimed to be 'nearly addiction proof'. In its first year of release, US sales of OxyContin generated USD 44 million, a figure that had increased to a whopping USD 1.5 billion/year by 2002.[11,12] These figures represent only US sales; the drug is sold in more than 35 countries world-wide.

Unfortunately, OxyContin is not as safe as initially advertised. It turns out that crushing the tablets released up to 70% of the free oxycodone, information that had long been known from Purdue's in-house testing. Given the high tablet strength (up to 160 mg) the drug became a ready source of strong opioid for snorting or injecting. Recreational use, addiction, overdose, and death became widespread.

The methods by which Purdue went about marketing OxyContin have been well documented, both in in the medical literature[13] and the mainstream media.[14] In an article from *American Journal of Public Health*, entitled, "The Promotion and Marketing of OxyContin: Commercial Triumph, Public Health Tragedy", Art Van Zee lays bare the techniques used by Purdue. First and foremost, OxyContin was promoted for non-cancer pain. While the number of patients with cancer pain is relatively static, as noted above, 39 million Americans suffer persistent pain. Second, Purdue targeted primary-care physicians. Previously, long-term opioid prescribing was the domain of palliative care physicians and oncologists. However, by 2002 half of all opioid prescriptions were by written by primary care physicians, many of whom had little training in the safe prescribing of opioids. To achieve these goals, Purdue funded all-expenses paid 'pain management' conferences to resorts in California, Arizona, and Florida. More than 5,000 doctors, nurses and pharmacists attended these meetings, where they were 'trained' in pain management by Purdue's bureau of experts. Marketing giveaways included soft toys, fishing hats, multi-tools, and, curiously, a compact disc of swing music ("Get in to the Swing of it with OxyContin"). Between 1996-2002 the OxyContin sales force doubled and sales representatives were paid bonuses averaging USD $70,000/year. Some received bonuses as high as high as USD $250,000/year. Patients were given free OxyContin 'starter packs' of between one week and one month's supply. In total, 36,000 starter packs were distributed. Throughout it all, Purdue systematically downplayed the risk of addiction, frequently citing the inherent safety of the slow release formulation and the "less than 1% risk of addiction" misquote from Porter and Jick. As Mike Mariani wrote in *The New York Times*, "It

was the most aggressive marketing campaign ever by a pharmaceutical company for a narcotic painkiller".[14]

However, by the early 2000s concerns were increasing about problems of addiction, overdose, and death related to prescription opioids, and with OxyContin in particular. In 2003 Purdue Pharma was the subject of a Congressional investigation by the US General Accounting Office.[11,12] Its report found Purdue had pursued an aggressive marketing campaign promoting the drug to treat a range of conditions to physicians who may not have been adequately trained in pain management. Purdue was cited for false and misleading advertising, particularly relating to minimising the abuse potential and risk of overdose associated with OxyContin. In 2007, this led to a federal law suit, in which Purdue entered into a plea agreement, admitting charges that it misled doctors and patients about the addictive properties of OxyContin and misbranded the product as "abuse resistant." Purdue was fined USD $634 million, and various executives including Michael Friedman (president) and Paul Goldenheim (medical director) were subject to multi-million dollar personal settlements. The figure of USD $634 million, while substantial, pales when compared to the more than USD $10 billion Purdue made from OxyContin during its first six years of sales and the estimated USD $72.5 billion annual cost to health insurers of the opioid epidemic. Subsequently, tight restrictions have been placed on opioid prescribing in the US and there have been numerous high-profile convictions of doctors found guilty of reckless prescribing. Sentences have involved lengthy prison time. OxyContin itself has now been re-formulated by Purdue to reduce the release of free drug when crushed. Ironically, this has allowed Purdue prolonged patent protection for OxyContin. Despite a lull during the years 2004-2006, sales of OxyContin subsequently increased and have remained above USD $2 billion annually. To date, OxyContin have generated in excess of USD $25 billion for Purdue.

The ascendancy of heroin and fentanyl

Since 2011 deaths from prescription opioids has decreased slightly. However, overall deaths from drug overdose have continue to increase, largely on the back of a dramatic increase in deaths related to heroin and fentanyl. A key reason for this change has been the fall in price and increased availability of heroin and fentanyl. In inflation-adjusted dollars, the price of heroin fell from USD $3,260/gram in 1981 to USD $465/gram in 2012 (data from US Offices of National Drug Control Policy).

One contributor to the falling price of heroin is a softening of the approach to cannabis by US lawmakers. Unlike most of the world, which sources its heroin from Afghanistan, only 4% of US heroin comes from Central Asia. Most of the US heroin comes across its southern border, either directly from Mexico or indirectly from Columbia. In particular, there has been a dramatic rise in the cultivation of opium poppies in the Sierra Madro region of Mexico. Increased legal production of cannabis in the US has dramatically reduced the profitability of Mexican-grown cannabis, causing farmers and cartels in the hill country of Sinola state (of the Sinola cartel and El Chappo fame) to shift to heroin production.

In drug addicted America, cheap heroin has filled the gap left by the reduced availability of prescription opioids. Today in the US, it is easier and cheaper to purchase an equivalent dose of heroin than 'oxy'.

And not just heroin. Fentanyl and other synthetic opioids, such as U-47700 ('Pink'), furanyl fentanyl, and carfentanil, have begun appearing in the US. These drugs are virtually all purchased online (via the so-called 'dark web' or TOR (the onion router) network), largely from China, and brought into the US by the postal service. The move from prescription drugs to heroin and fentanyl has come at significant cost in terms of inadvertent drug overdose and death. While not ideal, at least when crushing OxyContin, addicts are using a product that has been prepared to an industry standard by professionals with a reasonable training in pharmacology and weights and measures. No such guarantee is available when dealers cut 'oxy' and heroin with fentanyl and carfentanil.

With a potency of 10,000 times that of morphine, carfentanil, is a particularly dangerous drug. Carfentanil is available commercially for use by vets for anaesthetising large animals, such as elephants. The human LD 50 of carfentanil is as little as the equivalent of one grain of salt. Carfentanil is easily absorbed through the skin, and there have been several deaths of law enforcement officers (and vets) following inadvertent skin exposure. Even the clandestine labs in China who manufacture the drug state on their websites that carfentanil should only to be taken by 'experienced fentanyl users'.

Social decay, unemployment, and Trumpland

The communities that have been worst affect by the opioid epidemic are some of the poorest in the country. Blue collar counties that have suffered greatly from the decline of industry in the US over the last three decades. Communities affected by factory closures, shuttered-up businesses, and social decay. Unemployment is high, rates of health insurance are low, and alcoholism, suicide, and drug addiction are rife. These are the same communities that overwhelmingly voted for Donald Trump in the 2015 presidential election.

A multi-faceted approach is required to solve the current crisis, including good jobs, decent wages, increased educational achievement, and a much greater availability and funding of drug rehabilitation programmes. Whether or not building a wall along its southern border, trade tariffs aimed at bringing back American jobs, and repeal of the Affordable Care Act will achieve these ends is a matter of debate. To this observer, at least, such an outcome seems unlikely.

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The Vortex approach to airway management

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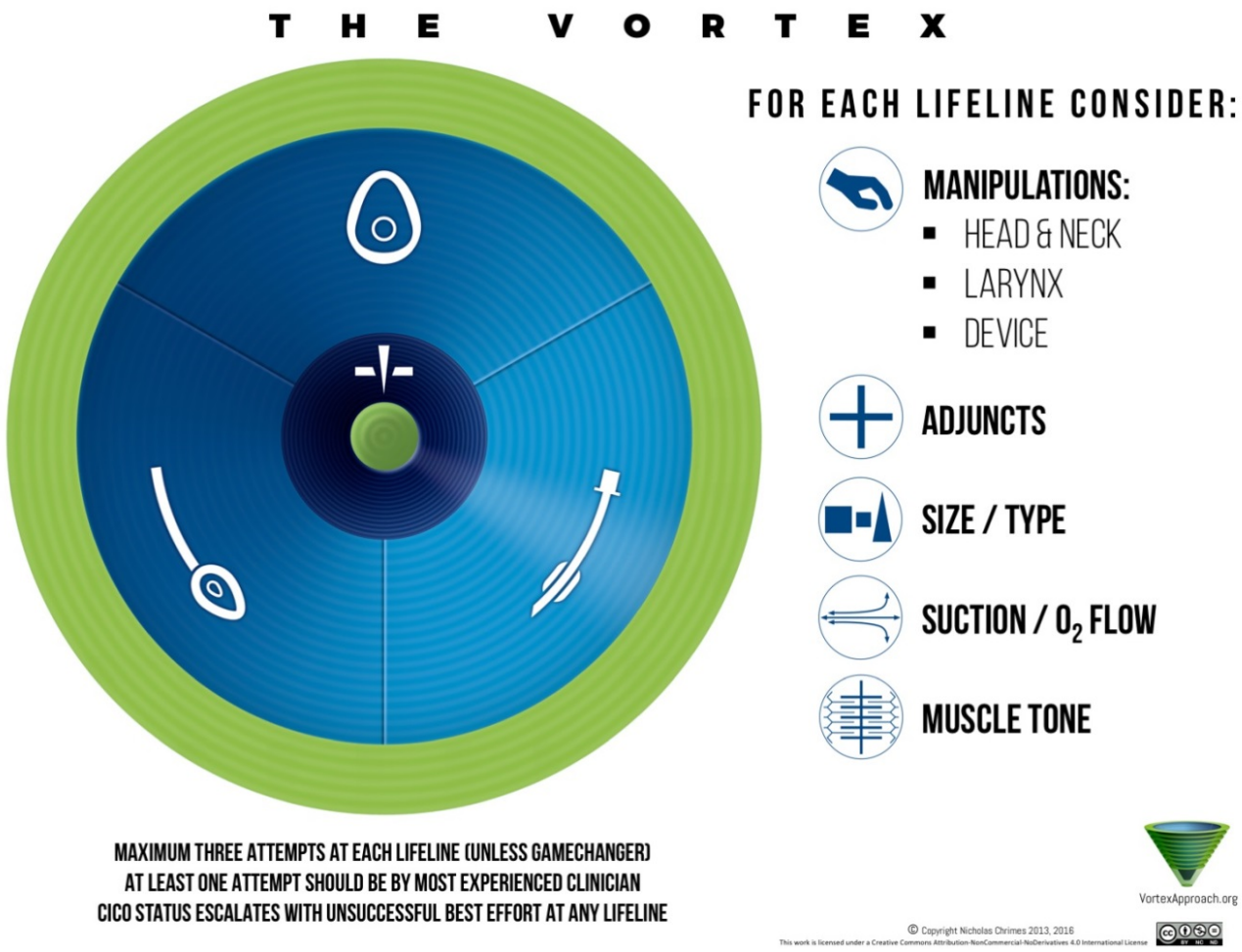
While technical competence and adequate planning are crucial to effective airway management, it is well recognised that even well prepared airway clinicians can sometimes fail to perform basic interventions under stress. The major airway algorithms are valuable training tools to familiarise clinicians with an approach to emergency airway management *prior* to the occurrence of an airway crisis. They are not, however, usually presented in a format that makes their content readily accessible in real-time to teams of potentially highly stressed clinicians *during* the process of managing a challenging airway. In addition, they typically provide guidance which is predominantly directed at anaesthetists and is usually restricted to the circumstance where the primary plan for airway management is endotracheal intubation.

The Vortex Approach, in contrast, is based around a "high acuity implementation tool", specifically designed to be used during the high-stakes, time critical situation of an evolving airway emergency. It is intended to help clinical teams perform under pressure by providing a simple, consistent template that can be taught to all clinicians involved in advanced airway management, irrespective of critical care discipline and whether they are from a medical, nursing or paramedical background.



It is also able to be used in any context in which an airway management takes place.

The Vortex implementation tool is based on the premise that there are only three upper airway 'lifelines' (non-surgical techniques) by which alveolar oxygen delivery can be established and confirmed: face mask, supraglottic airway and endotracheal tube. If a 'best effort' at each of these three lifelines is unsuccessful then a can't intubate, can't oxygenate situation (CICO) situation exists and 'CICO Rescue' (emergency front-of-neck access) must be initiated.

Completion of a 'best effort' at any of the three upper airway lifelines without being able to restore alveolar oxygen delivery mandates spiral movement inward towards the next lifeline. The circular arrangement of the three lifelines on the tool means that airway management can be initiated using any lifeline and proceed to the remaining ones in whatever sequence is judged most appropriate in the clinical circumstances. A list of five categories of optimisation, applying equally to each of the three lifelines, is provided to prompt consideration of the available options for maximising success during a best effort at any lifeline.

Completion of best efforts at all three lifelines without restoring alveolar oxygen delivery culminates in spiral movement to the centre zone of the tool, representing the need to initiate CICO Rescue. Conversely, confirmation of alveolar oxygen delivery using any of the three lifelines, results in outward movement into the circumferential 'Green Zone'. The Green Zone prompts recognition of the opportunity to re-oxygenate, gather resources and make a plan, that arises whenever alveolar oxygen delivery is able to be established. The Green Zone is also visible in the centre of the tool to remind clinicians that, when all three lifelines have been unsuccessful, CICO Rescue also restores alveolar oxygen delivery and provides the same opportunities.

The Green Zone refers to any situation in which adequate alveolar oxygen delivery can be confirmed and the patient is no longer at imminent risk of critical hypoxia. This provides the clinical team with time to pause and consider the opportunities available to them before further instrumenting the airway.

Inability to intubate is an inconvenience. It is the loss of alveolar oxygen delivery resulting from repeated airway instrumentation that creates an emergency. Declaration that the Green Zone has been entered emphasises a key moment of situational awareness to the team. This has the potential to interrupt the process of repeated airway instrumentation that can convert the 'can oxygenate' situation into the 'can't oxygenate' situation.

The Vortex implementation tool is the core of the broader Vortex Approach which provides a comprehensive array of resources to facilitate all phases of airway advanced airway care including airway assessment, development of an airway strategy and performance of airway interventions in both the routine and emergency setting. The focus of the Vortex Approach is on providing "implementation tools" for real-time use during the process of airway management. In addition, it provides "foundation resources", to be referred to prior to undertaking airway management, that teach clinical teams how to use to use the Vortex Approach.

Implementation Tools – The Vortex Approach incorporates a suite of implementation tools designed to facilitate both the preparation and intervention phases of advanced airway care. These aim to present information in a manner that is simple enough to be accessible to teams during clinical practice. These adjunctive tools work in an integrated fashion with the primary Vortex tool using the same concepts and language.

Foundation Resources for the Vortex Approach – "implementation" tools will not be effective without prior familiarity and training to lay a foundation for their use. The Vortex Approach therefore provides a number of "foundation" resources that establish understanding of the

background principle specific to the Vortex Approach and proficiency in the team behaviours required for its successful implementation.

Technical Foundation Material – effective airway management requires that clinicians have a foundation in the requisite technical knowledge, skills and attitudes that make them competent to make appropriate decisions in response to the prompts provided by the Vortex and implement the chosen interventions. Although the resources of the Vortex Approach provide a limited amount of technical material, the bulk of this technical content should be derived from other recognised airway management resources and formal airway training programs. The Vortex Approach then provides a template to prompt team recall and application of this technical background material in real-time. Thus the Vortex Approach should not be viewed as an alternative to the major airway algorithms but as a complementary resource, designed to facilitate implementation of the management recommendations outlined by these training tools and improve the performance of clinical teams.

The Vortex serves to maximise opportunities to establish alveolar oxygen delivery by:

- Facilitating effective planning for airway management
- Facilitating efficient best efforts at each of the three upper airway lifelines.
- Encouraging appropriate decision making when any of these are successful and the Green Zone is entered.
- Promoting early priming for CICO Rescue as an airway crisis evolves.
- Facilitating rapid recognition of the need for CICO Rescue.

More free resources relating to the Vortex Approach can be found at VortexApproach.org

How to THRIVE with a difficult airway patient

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No abstract submitted.

Being too sweet… 'to siphon honey'

Jay van der Westhuizen

Auckland City Hospital, New Zealand

Guidelines for Peri-operative management of the Adult Diabetic patient.

Scope of the problem…

Diabetes is a growing global problem, the prevalence has more than doubled since 1980, and predicted to reach epidemic proportions by 2040[i]. One in eleven people has been diagnosed with diabetes and 90% of those are type 2, with a large proportion being obesity related. The incidence in New Zealand is 240 000 with 50 newly diagnosed daily[ii], in Australia 1.2 million with 280 patients newly diagnosed daily - that is one person every five minutes[iii]! It is the fastest growing chronic condition in Australasia. The financial burden on health is staggering and furthermore a large proportion of these patients presents for surgery.

10-15 % of the surgical population is diagnosed with diabetes, and 40% of those presenting for intermediate or high-risk surgery are yet to be diagnosed. There is a growing reasoning that all those presenting for intermediate or high-risk surgery should be screened.

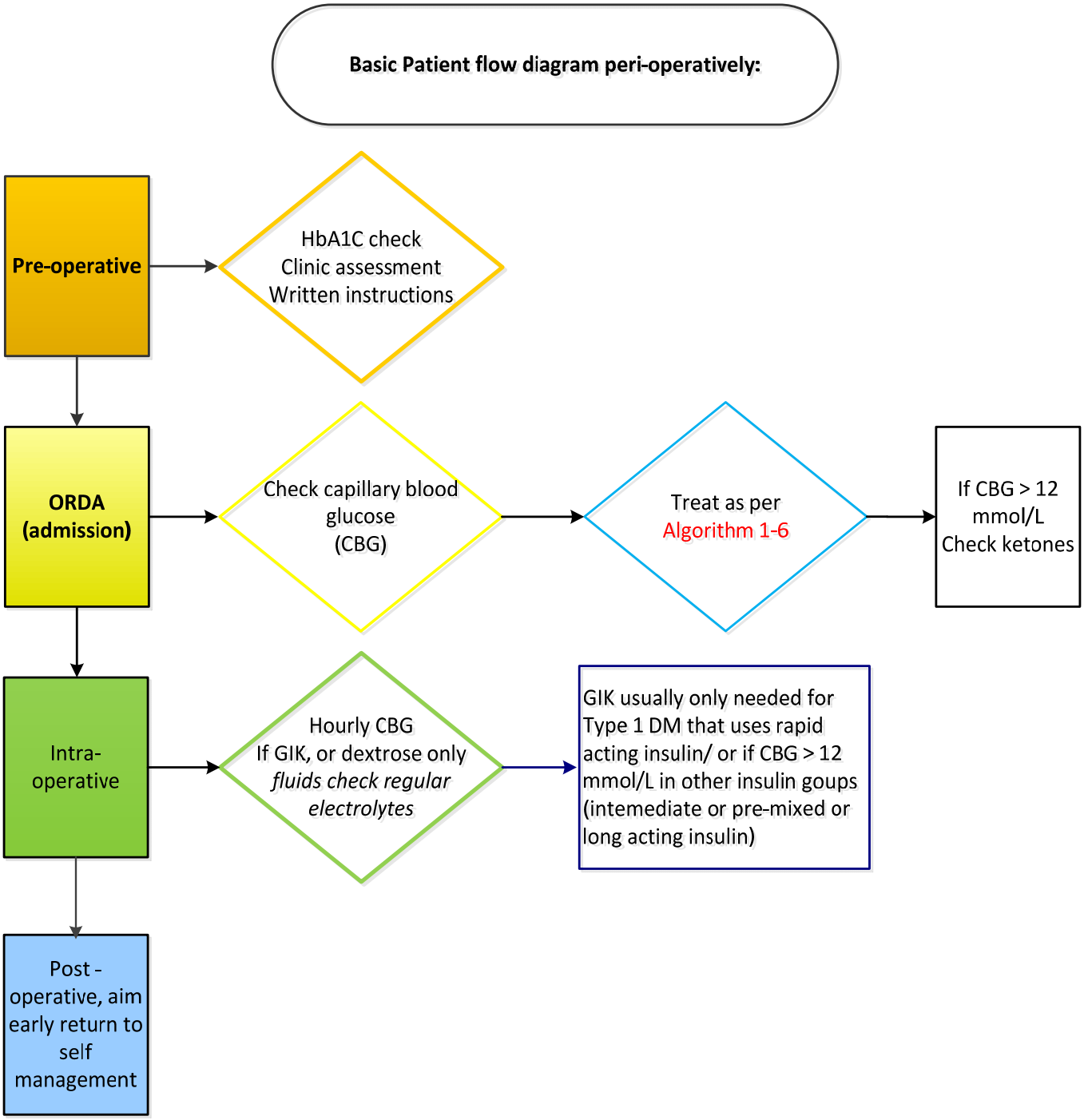
The significance…

The implication of post-operative complications has a significant impact on the outcome morbidity and mortality in this group[iv].

In poorly-controlled diabetics there is a **greater than 50% increase in mortality, a 2.4-fold increase in the incidence of postoperative respiratory infections, a doubling of surgical site infections, a threefold increase in postoperative urinary tract infections, a doubling in the incidence of myocardial infarction, and an almost twofold increase in acute kidney injury**[v].

Key points for modern management of the surgical patient:

- Optimal pre-operative HbA1c < **69mmol/mol**
- The target blood glucose range perioperative is **6-10 mmol/L** (but acceptable range is 5-12mmol/L) *If a patient's usual BSL level is high normal, aim for high normal peri-operatively*
- **Hyperglycaemia defined as >12mmol/L**
- **and check for ketones if >12 mmol/L (> 15mmol/L in Australia)**
- For patients with Type 1 Diabetes: **NEVER STOP** the subcutaneous ***basal insulin*** (e.g. Lantus®, Protaphane® or Humulin NPH®). The dose may need adjusting in some population groups e.g. 'grazers'
- Insulin pumps - if the patient is able to 'self-manage' during the perioperative period, the insulin pump therapy should be continued using the **'sick day regime**'. If not able to self-manage, the pump should be discontinued and diabetes managed with a GIK infusion
- Those patients with poor glycemic control (**HbA1c > 69 mmol/mol**) should be **deferred** if possible and referred to a diabetologist /diabetes nurse specialist before elective surgery
- Note**: Risk of hyponatremia** with use of only 5% or 10% Dextrose as IV fluid, if running a GIK (Glucose/ Insulin/ Potassium Infusion) require at least daily checks of electrolytes, especially sodium
- Written advice for all surgical patients including hypo- and hyperglycemia management advice


Basic Patient flow diagram peri-operatively:
Pre-operative
HbA1C check
Clinic assessment
Written instructions
ORDA (admission)
Check capillary blood glucose (CBG)
Treat as per Algorithm 1-6
If CBG > 12 mmol/L
Check ketones
Intra-operative
Hourly CBG
If GIK, or dextrose only
fluids check regular electrolytes
GIK usually only needed for Type 1 DM that uses rapid acting insulin/ or if CBG > 12 mmol/L in other insulin goups (intemediate or pre-mixed or long acting insulin)
Post - operative, aim early return to self management

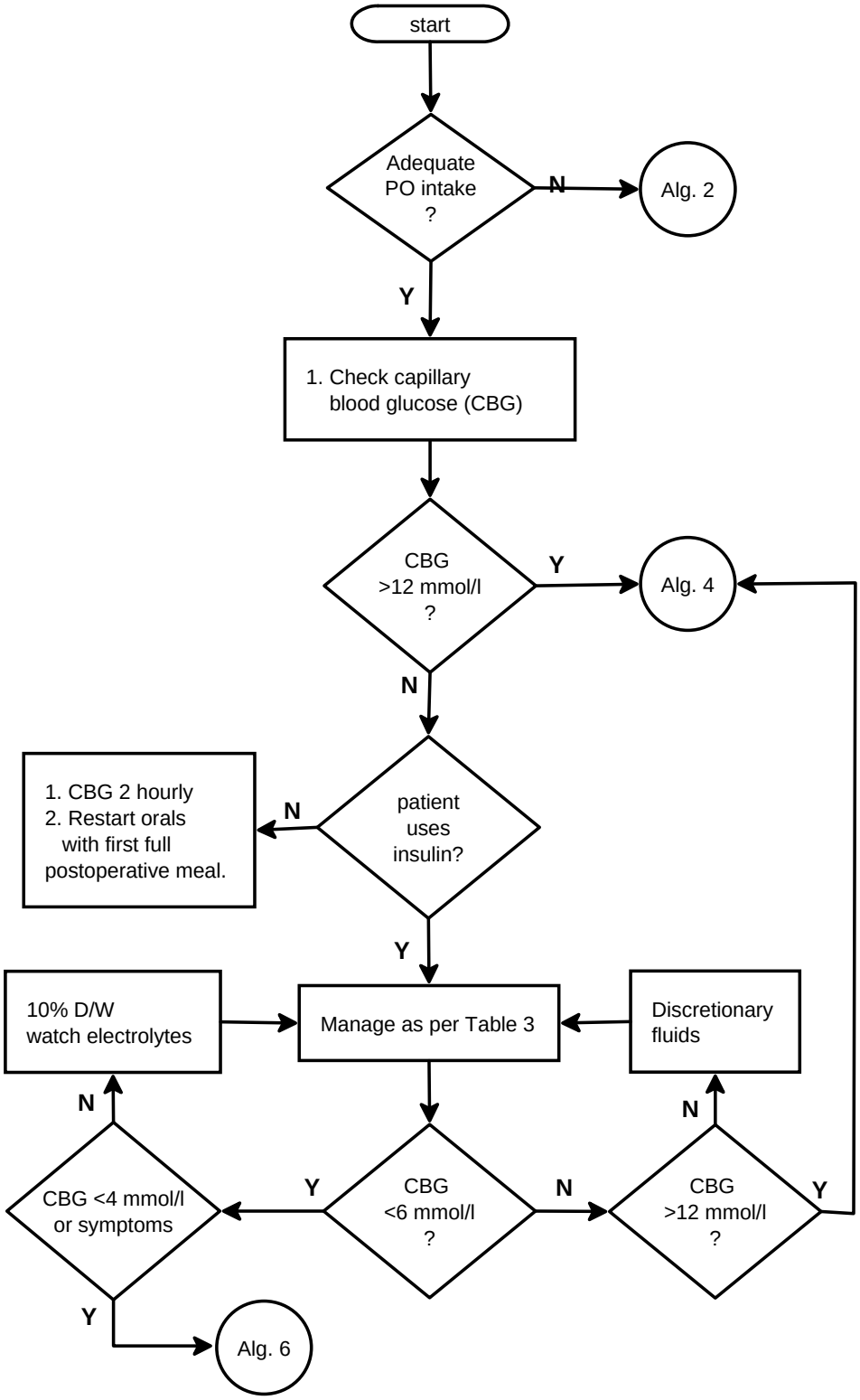
Algorithm 1

PATIENTS WHO CAN EAT WITHIN 6 HRS AFTER SURGERY

Special considerations

1. Patients on insulin pump *must attend* a preoperative anaesthetic clinic. On the day of surgery, use 'sick day' or basal 'sleep' rate.

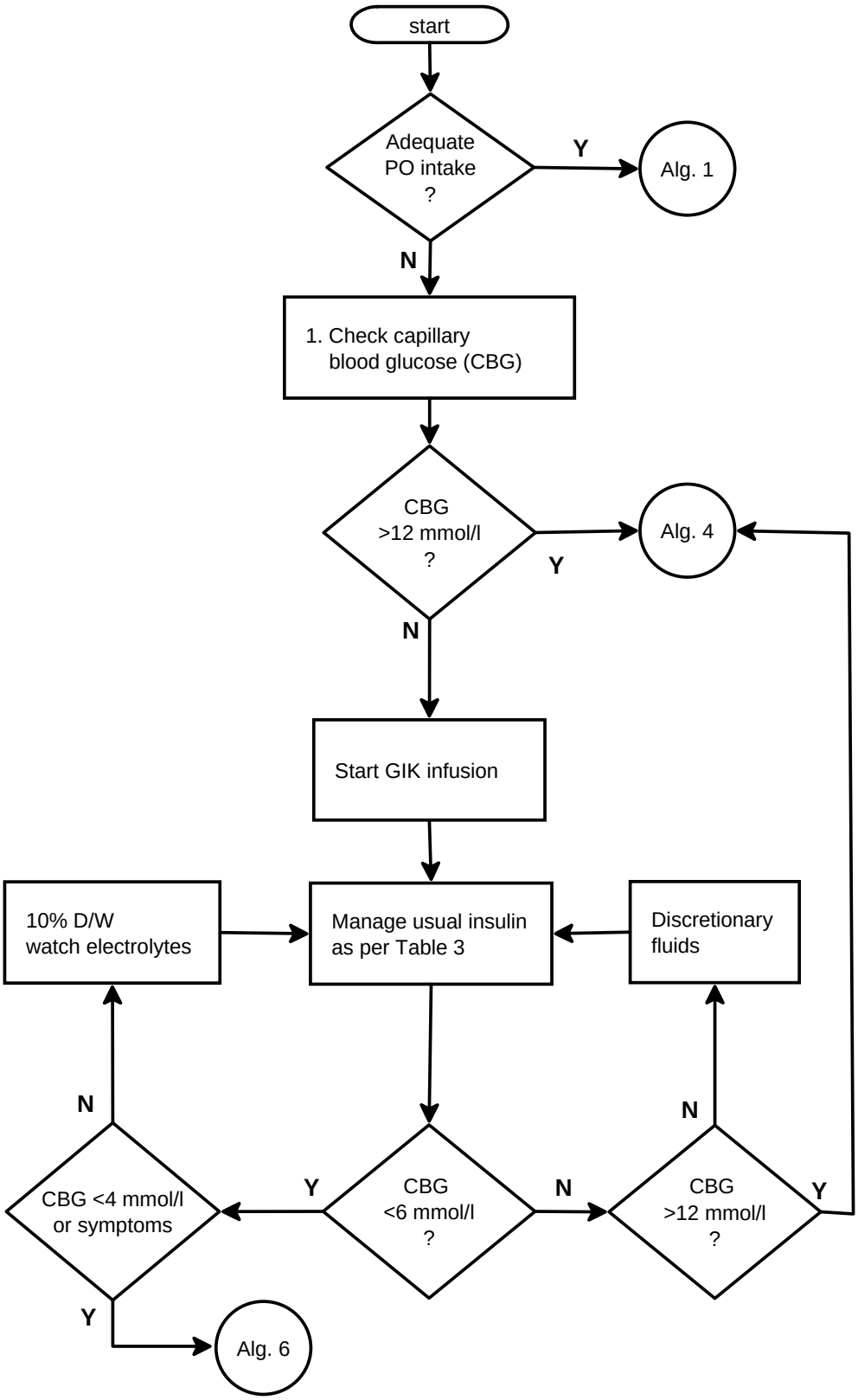
2. A patient on long acting glargine (Lantus™) or detemir (Levemir™) can have their usual basal dose irrespective of the type of surgery. Use in conjunction with GIK, if GIK is required peri-operatively.



Algorithm 2

THE PATIENT CAN'T EAT WITHIN 6 HOURS

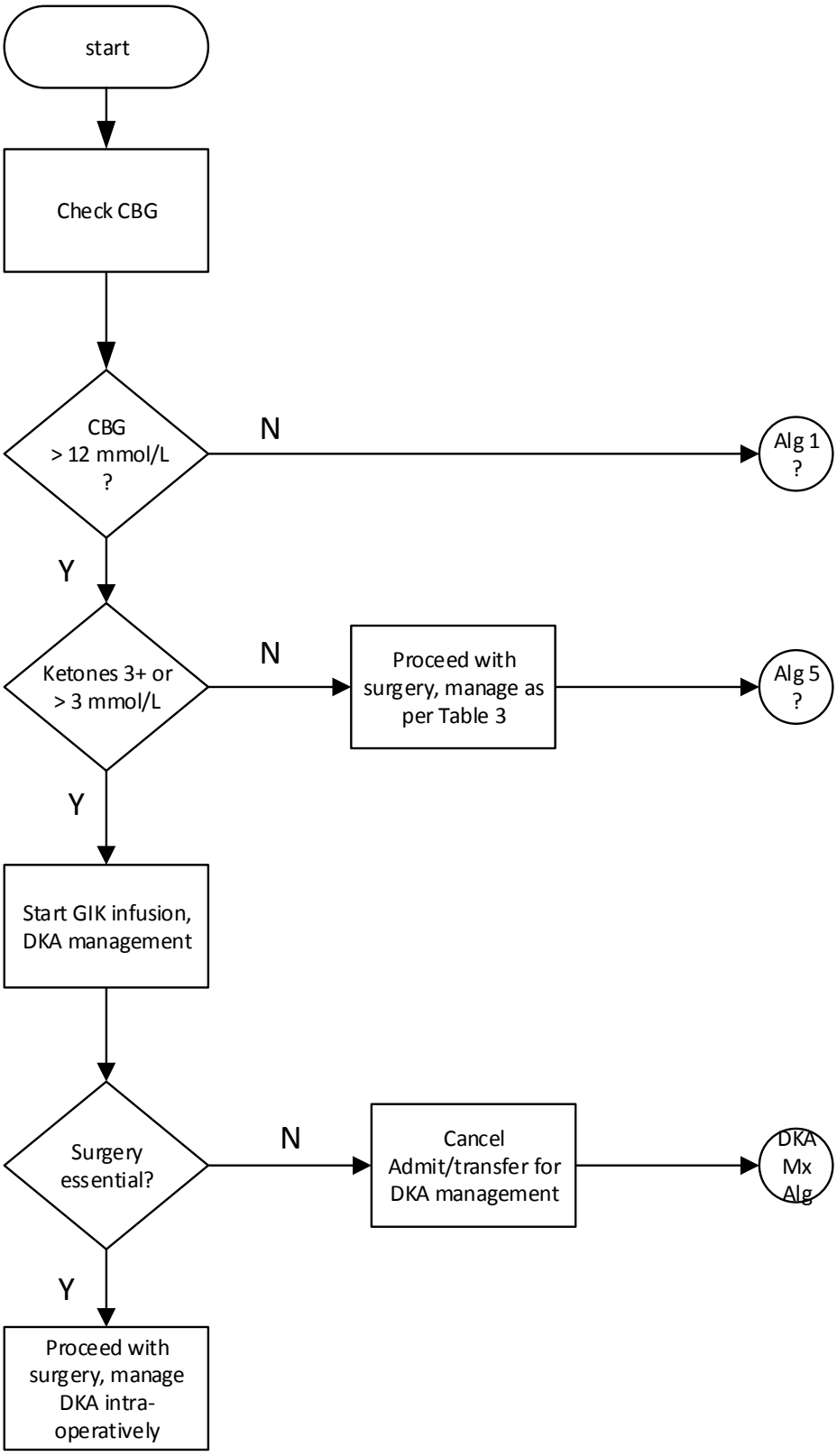
This section applies to a diabetic patient undergoing any procedure for which they will be NBM pre- or post-procedure. It does not apply to ophthalmology patients (see perioperative management - ophthalmology). **NB. Starvation starts at point of first missed meal.**



Algorithm 3

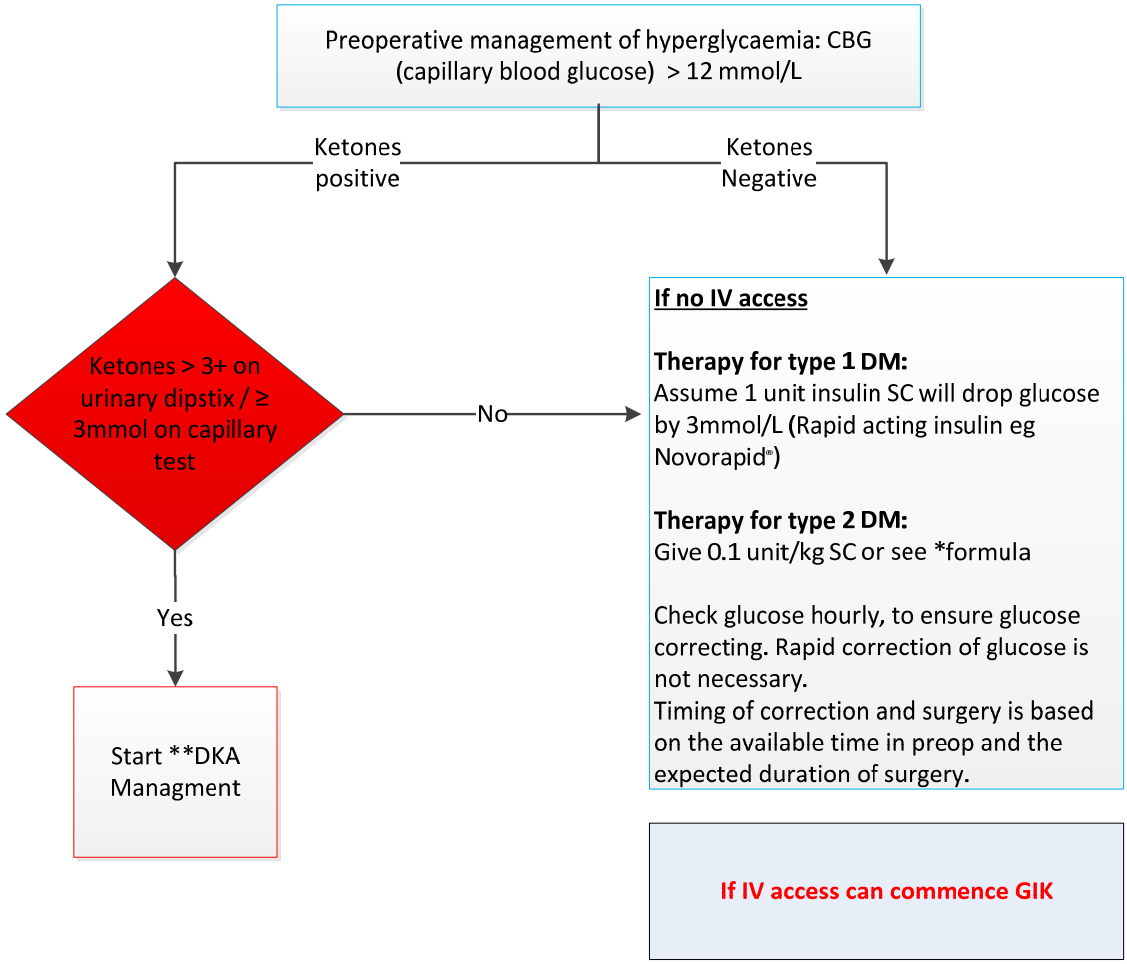
POOR GLYCAEMIC CONTROL (HBA1C > 69)

Poor pre-operative glycaemic control



Algorithm 4

HYPERGLYCEMIA



Choice of route of administration and other issues
Rapid acting insulin is for subcutaneous use only
Subcutaneous insulin every 1-2 hours provides similar glucose control as IV infusion in patients with DKA
Regular insulin by IV bolus half-life is <10min so will require larger doses by IV for adequate control
Dose determination – traditional sliding scale assumes that all patients have similar insulin sensitivities or that there is no change in insulin sensitivity during different stages of acute illness – which is incorrect.

Determine insulin sensitivity/resistance for dose determination: 80/100 rule
Calculate total daily insulin requirement then
If using regular insulin – 80/total daily dose = expected glucose decrease/unit insulin
If using rapid acting insulin – 100/total daily dose = expected glucose decrease/unit insulin.
(Example: daily dose of 60 units - 1 unit insulin will reduce glucose by 1.5 mmol/L)

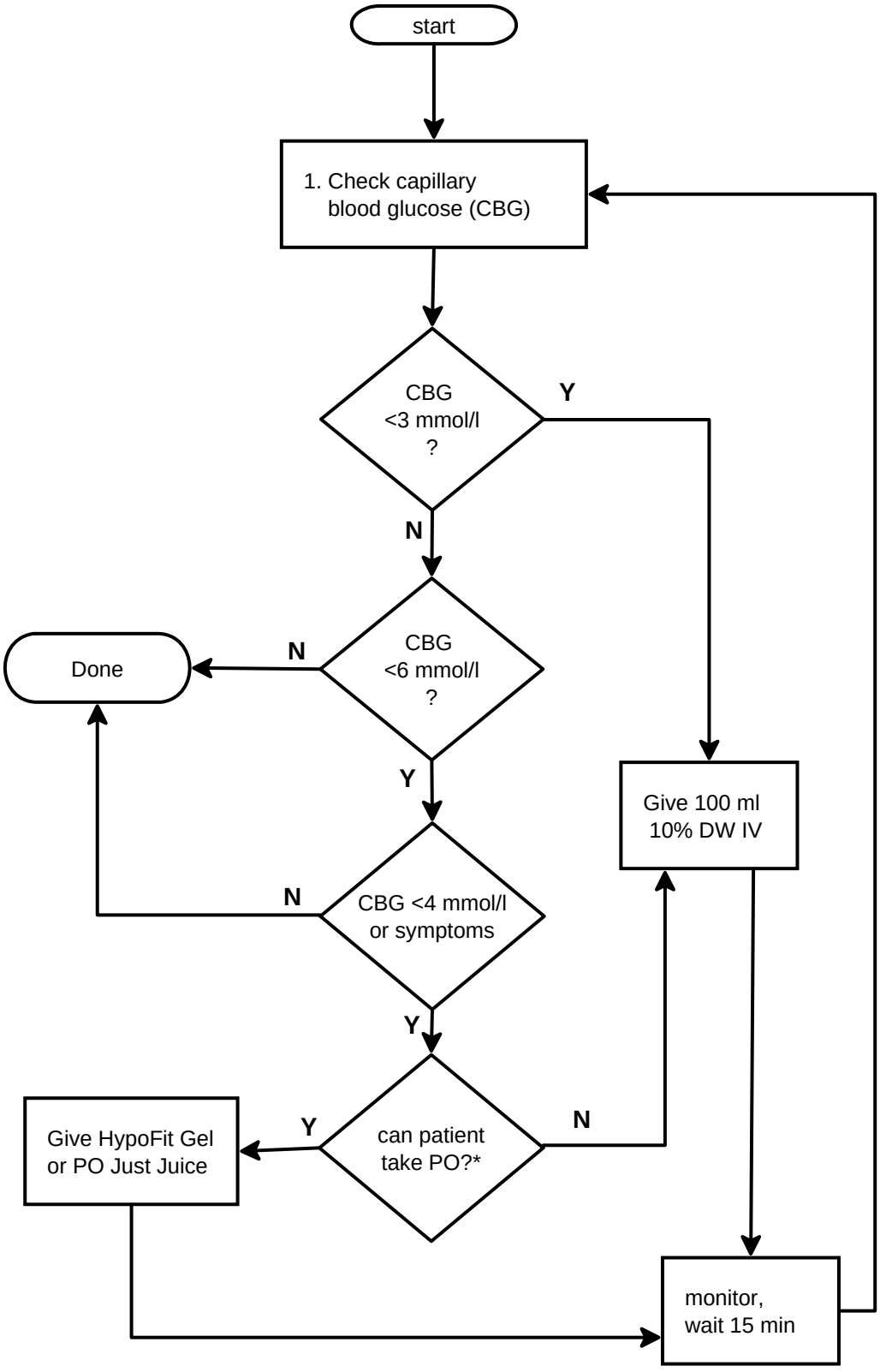
***DKA = Diabetic Ketoacidosis*

*

For Type 2 DM correction with 0.1mg/kg SC up to a maximum of 6U of rapid acting insulin.

Algorithm 5

HYPOGLYCEMIA



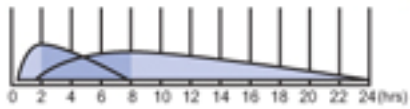
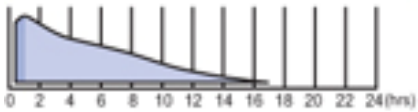
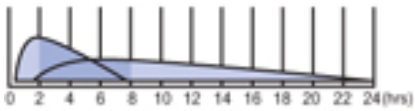
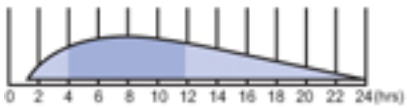
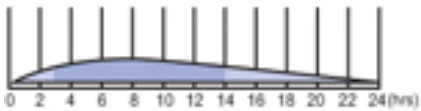
Management of Oral Hypoglycaemic agents –

Tablet	AM surgery	PM surgery
Acarbose	Omit morning dose	Take morning dose if eating
Metformin	Take as usual *if able to eat within 6 hours* post-operatively*	Take as usual *if able to eat within 6 hours* post-operatively*
	*__OMIT__ if impaired renal function (eGFR < 50mL/ min/m 2) or if having cardiac surgery/procedures requiring contrast, or if starvation period continue for more than 6 hours post-operatively.	
Pioglitazone	Take as normal BUT omit **for Cardiothoracic surgery**	Take as normal BUT omit **for Cardiothoracic surgery**
Sulfonylureas (glibenclamide, glipizide, gliclazide)	Omit (restart when eating normally)	Omit (restart when eating normally)
DPP4 inhibitors (saxagliptin, sitagliptin)	Omit	Omit
GLP-1 agonist (exenatide)	Omit	Omit

Note: *If patient is ill with sepsis, has cardiac failure, renal failure or diarrhoea then metformin needs to be withheld - risk of lactic acidosis.*

DPP4 inhibitors: dipeptidylpeptidase-4 inhibitors
GLP-1 receptor agonist: glucagon-like peptide-1

Management for patients on insulin –

Types of Insulin Available	Brand Name	Activity (may vary between patients)	Profile
Rapid Acting	Humalog®* Apidra® Novorapid®	Onset: up to 20 minutes Peak: 1-2 hours Duration: 2-5 hours	
Short Acting	Humulin R® Actrapid®	Onset: 30 minutes Peak: 2-4 hours Duration: 6-8hours	
Withhold dose while NBM, normal dose the night before surgery. Check BSL 2 hourly while NBM.			
Premixed insulin	Humulin 30/70® Penmix 30® Mixtard 30® Penmix 50®	Onset: 30 minutes Peak: 2-8 hours Duration: Up to 24 hours	
Rapid Acting Premixed Insulin	Humalog Mix 25® Novomix 30®	Onset:0-15 minutes Peak: 30-70 minutes Duration:16-18 hours	
	Humalog Mix 50®	Onset:0-15 minutes Peak: 30-70 minutes Duration:16-18 hours	
If **AM surgery**: give 50% of usual dose on admission to hospital with hourly CBG checks** If **PM surgery**: 50% of am dose with light breakfast			
Intermediate Acting	Humulin NPH® Protaphane®	Onset:1-2 hours Peak: 4-12 hours Duration: Up to 24 hours	
If AM surgery: give 80% of dose on admission to hospital with hourly CBG checks** **If PM surgery:** give 80% of AM dose with light breakfast at home and advice for hypoglycaemia management.			
Long Acting	Detemir (Levemir®) Glargine (Lantus®)	No pronounced peak Duration: Up to 24 hours	
Administer usual dose regardless of timing of surgery or starvation status.			

**If CBG < 4 mmol/L and ≥2 hours pre-operative give 200 mL clear apple juice/tropical just juice or hypo-fit gel, if less than 2 hours commence glucose 10% infusion at 80 mLs/hr if CBG 4 - 6 mmol/L and if < 4mmol/L at 120 mL/hour. Still administer basal insulin for Type 1 DM.

If CBG > 12 mmol/L, ensure if on long acting insulin to take usual dose or 80% of usual dose of intermediate acting insulin and start GIK, in addition check for ketones and if 3+ start DKA management.
Patients should have 80 mL/hr of glucose 10% whilst starvation ongoing once they are administered their usual insulin and CBG < 6mmo/L (fluid management is at the discretion of the procedural anaesthetist in theatre).

These, and international guidelines takes a pragmatic approach to the peri-operative management of the diabetic patient having surgery. The diversity of patients makes it a very difficult topic, clearly there will be individuals who do not fit a specific pathway. Most diabetic patients have vast experience in managing their own diabetes, and the aim is now to manipulate 'normal' insulin regimes, moving away from using the GIK or VRII (variable rate insulin) and aiming for early return to self-management.
Admission on day of surgery, having been assessed and optimised in the pre-assessment clinic. All patients should have a written plan*, and ideally listed first to minimise disruption to glycaemic control and minimise starvation time.

Other considerations...

If Dexamethasone is used as post-operative nausea and vomiting prophylaxis blood glucose level should be checked hourly for four hours. If possible use alternative anti-emetic.
ERAS (Enhanced recovery after surgery) – this group of patients are likely ideal ERAS however use of carbohydrate drink should not be used for Type 1 DM with a is short starvation time, it can be used for Type 2 DM and for all types DM if GIK or VRII used. Evidence in this population scant and gastric emptying may be delayed[vi].

Controversies*...*

Contrary to previous understanding the risk of lactic acidosis with metformin is low. However it should still be withheld in the patient with renal impairment or if contrast used intra-operatively[vii]. It was found that those who continued with metformin, often inadvertently, had improved outcomes compared with those patients who omitted metformin as instructed[viii].

Safety*...*

Insulin was listed as one of the top five 'high risk' medications in a National Audit in the UK (NaDIA). Half of patients treated with insulin had a medication error on their drug chart[ix]. The VRII is still overused and patients are at risk of DKA (diabetic ketoacidosis) during transition from intravenous to sub-cutaneous insulin management.

There is also risk of hyponatremia associated with the GIK.

*Advice pamphlets

Advice for diabetic patients who use insulin to manage their diabetes before surgery

Please bring all your usual medication, tablets and Insulin to hospital on the day of surgery.

If you use **Insulin** to manage your diabetes please discuss this with your **anaesthetist** or pharmacist.

If you use Lantus (Glargine) or Detemir (Levemir) Insulin you may continue to administer your usual dose.

If your surgery is in the morning: administer your own insulin when you get to hospital. But if you use long acting insulin Detemir (Levemir) or Lantus (Glargine), you can administer it at home.

If your surgery is in the afternoon: administer your own insulin at home after having a light breakfast.

Advice for low blood sugar management (blood sugar level < 4 mmol/L) or if you having symptoms of low blood sugar on the morning of surgery:

If your blood sugar level is < 4 mmol/L two hours before surgery you can have 200mls clear apple juice, tropical just juice or hypo-fit gel. Recheck your blood sugar level and proceed to hospital accompanied.

If it is less than two hours before your surgery and you are at hospital the staff should start a glucose infusion.

If your blood sugar level is > 12 mmol/L please advise staff at the hospital and they will start treatment and check for ketones in your urine or blood as soon as you arrive.

Other instructions:

If you unsure what to do, do not take anything BUT bring all your medication to hospital.

For more information contact:

Instructions for your type of Insulin

	Types of Insulin Available	Lilly Brand Name	Novo Brand Name	Day of surgery	Calculated dose for day of surgery
☐	***Rapid Acting/Short Acting**	Humalog*/ Humulin R	Novorapid /Actrapid	Do not have a dose	
☐	**Intermediate Acting**	Humulin NPH	Protaphane	Have **80% of your normal dose** (ask the pharmacist or anaesthetist to work out the correct dose) and check your blood sugar every hour for first two hours. For **morning surgery** only administer Insulin once you've arrived at hospital. For **afternoon surgery** administer insulin after light breakfast at home.	
☐	**Long Acting**		Detemir (Levemir)	Have your usual dose regardless of time of surgery, and check your blood sugar as usual.	
☐	**Long acting**	Glargine	Lantus	Have your usual dose regardless of time of surgery, and check your blood sugar as usual.	
☐	***Premixed Insulin/ Rapid Acting Insulin**	Humulin 30/70 Humalog Mix 25 Humalog Mix 50	Penmix 30 Mixtard 30 Penmix 50 Novomix 30	If **morning surgery**: half of usual dose on admission to hospital with hourly blood sugar checks. If **afternoon surgery**: half of morning dose with light breakfast and check blood sugar levels hourly for two hours then as usual.	

*Do not confuse Humalog (rapid acting) with Humalog Mix 25 and Humalog Mix 50 (premixed preparations)

Advice for diabetic patients who take oral medication and are undergoing surgery.

Please bring all your usual medication, tablets and Insulin to hospital on the day of surgery.

Please do not take **any** of your usual diabetic medication unless told by the Anaesthetist or Pharmacist to do so. **If you are unsure what to do, take nothing and bring all your medication to hospital.**

Instructions for your oral diabetic medication have been ticked, highlighted or circled below:

	Tablet	Having surgery in the morning:	Having surgery in the afternoon:
□	Acarbose	Do not take usual morning dose if not having breakfast.	Take your usual morning dose if having light breakfast.
□	Metformin	**Take your usual morning dose,** But **do not take** dose if: □ You won't be eating for more than six hours after surgery, for example having bowel surgery. □ Having contrast studies (Radiology). □ Having heart or lung surgery.	**Take your usual <u>morning and afternoon</u> dose,** But **do not take afternoon** dose if: □ You won't be eating for more than six hours after surgery, for example having bowel surgery. □ Having contrast studies (Radiology). □ Having heart or lung surgery.
□	Pioglitazone	Take as normal. **Except do not take if having heart or lung surgery.**	Take as normal. **Except do not take if having heart or lung surgery.**
□	Suphonylurea (Glibenclamide,Glipizide, Gliclazide)	Do not take. Only start taking again when eating normally.	Do not take. Only start taking again when eating normally.
□	DPP4 inhibitor (Saxagliptin, Sitagliptin)	Do not take.	Do not take.
□	GLP-1 analogue (Exenatide)	Do not take.	Do not take.

Other instructions:

If you have any questions or enquiries please contact:

Welcome *Haere Mai* | Respect *Manaaki* | Together *Tūhono* | Aim High *Angamua*

Aiming for better care for the Surgical diabetic patient.

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Protecting the brain

Chris Thompson

Royal Prince Alfred Hospital, Australia

This talk will focus on blood pressure management and cerebral monitoring during at-risk surgical procedures, such as carotid endarterectomy and shoulder surgery in the recumbent sitting position.

Strategies to reduce the chance of a catastrophic neurological outcome after shoulder surgery will be discussed.

The Triad approach to crisis management

Nicholas Chrimes

Monash Medical Centre, Australia

Clinical crisis management frequently requires key priorities to be initiated in a time-critical fashion to avoid significant morbidity or mortality to patients. This can be a stressful situation and, even in the hands of highly competent and experienced staff, this stress can result in time-critical interventions not being implemented in an appropriate time frame and important priorities being delayed or overlooked, thereby compromising patient care. The use of properly designed cognitive tools to prompt clinicians to perform these basic tasks, could improve management of these situations but the amount of information that can be processed by stressed individuals in the initial phases of managing a crisis may be very low.

Whilst algorithms & clinical guidelines usually present information that is *technically* correct regarding how to manage a medical crisis, they tend to be text based, information dense documents. Whilst such documents have an important part to play in *preparing* clinicians to deal with a crisis *prior* to a crisis occurring they are less suited to use *during* the initial phase of a crisis, when clinicians may have limited information processing ability due to stress. The term "High Stakes Cognitive Tool" was coined by Dr Nicholas Chrimes & Dr Peter Fritz, in relation to the Vortex Approach, to refer to a tool which is designed specifically for use during time-critical emergencies. In order to achieve this, not only must the *content* of the tool address the *technical factors* required to manage the crisis, but the *design* of the tool must address the *human factors* aspects that will allow it to be used in "real time" during a crisis. A high acuity implementation tool must therefore be simple enough to be used by teams of potentially highly stressed clinicians during the initial stages of managing a time-critical emergency and flexible enough that the same tool can be consistently applied to any context in which a particular crisis might arise. The purpose of a high acuity implementation tool is to act as a prompt for recall of prior training and planning, in order to allow clinical teams to perform under pressure.

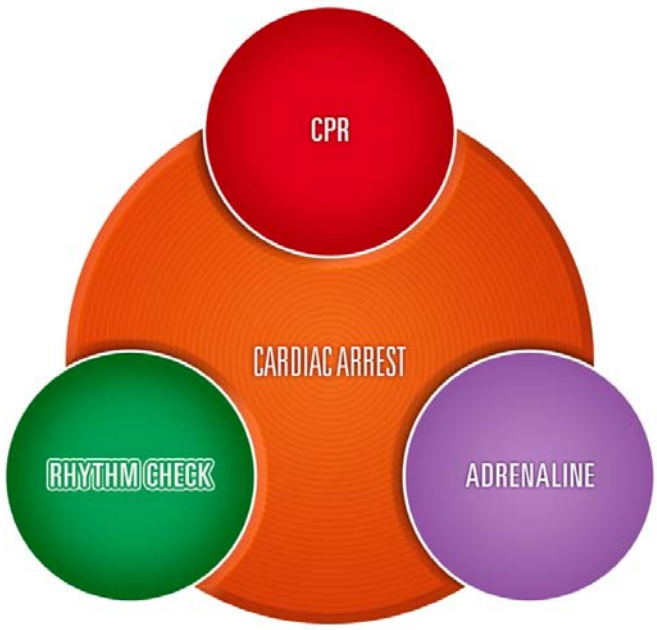
It is well documented in emergency airway management that even experienced clinicians are vulnerable to making significant, fundamental errors when they are under pressure and that such errors can lead to serious adverse patient outcomes. The Vortex Approach was conceived by Dr Nicholas Chrimes as a high acuity implementation tool to assist in addressing these issues during management of the unanticipated difficult airway.

Although the issues relating to airway emergencies apply to the management of other clinical crises, adapting the principles of the Vortex Approach for use beyond airway management poses some additional challenges. In general, clinicians engaged in independent advanced airway management can be assumed to have a baseline level of competence with the requisite technical skills (where this is not the case the issue should be addressed by improving training, not via use of a cognitive tool). These skills tend to be well maintained even in situations of stress, provided clinicians are provided with appropriate cues to implement them using prompts to remind the team of the available options and to improve situational awareness. In contrast to difficult airway management, other clinical emergencies may require significant information recall, complex decision making and tracking of multiple parallel processes in order for to be managed effectively. The amount of information which needs to be provided by a cognitive tool to enable clinicians to manage a crisis effectively may vary widely according to their level of training, experience and exposure – all of which contribute to their level of familiarity with management of that crisis. Even for a particular individual managing a specific crisis, information recall may vary widely in different contexts according to their level of stress. The factors which may induce stress in a clinical situation are numerous but include foreseeability, complexity, availability of assistance, urgency to intervene, severity of potential harm, feelings of responsibility for causing the crisis, fatigue, prior emotional state and familiarity with the crisis itself. The dynamic nature of many of

the factors which contribute to the stress produced by a particular clinical circumstance means, that there is potential for a clinician, no matter how experienced and familiar with a clinical event, to be become stressed to the point that cognition becomes significantly compromised.

Traditionally, cognitive aid design has addressed the above issues by creating tools with detailed technical content that cater to the "lowest common denominator" in terms of knowledge and cognitive capacity. The rationale is that provision of exhaustive instructions ensures that even someone with limited familiarity or information recall will have access to sufficient technical information to successfully manage the crisis. Conversely it is assumed that more experienced, well-functioning clinicians who do not need to access this detail can "skip over" it, selecting only the information they need. Whilst this may intuitively seem to make sense, this approach ignores the fact that stress can not only impair a clinician's ability to access their own existing knowledge but can also interfere with their ability to process the information presented to them in a cognitive aid. Thus these information laden tools do not address the cognitive challenges produced by stress, diminishing their utility in "real time" management of clinical crises.

The Triad Approach addresses the above challenges by providing a hierarchy of resources of increasing complexity which can be accessed according to a clinician's information needs and cognitive capacity. The first tier (the Triad) simply presents management priorities, the second tier (the Task List) identifies tasks needed to address these priorities and the third (the Intervention Guide) provides more detailed background information on the specific interventions required to complete the tasks. The intention is that each tier provides prompts at an appropriate level of detail for the cognitive capacity of the clinicians involved in managing the crisis. The simpler prompts of the upper tiers ensure time critical interventions are recognised and initiated, even by highly stressed individuals. This not only provides urgent therapy to the patient in a timely fashion but the sense of achievement in progressing management of the crisis will hopefully decrease the level of stress ("success to destress"), thereby improving their cognitive capacity so that they can process the more detailed information presented in the lower tiers. Thus to achieve the desired balance between maintaining simplicity and providing the varying level of technical information required by a clinician to manage any crisis, the Triad Approach is designed so as to provide increasingly detailed information that can be accessed according to the requirements of a specific situation.



More free resources relating to the Triad Approach are available at

Towards a better understanding…

Jim Moriarty
New Zealand

No abstract submitted.

Forty years of anaesthetic practice

Chris Nixon

Auckland City Hospital, New Zealand

It occurred to me last year that I gave my first anaesthetic over 40 years ago in 1974 as a medical student. In the time I have available I will, through a series of stories give you a little insight into the changes in Anaesthesia, academically and professionally. I will talk about Wooley and Roe, Pearl Harbor, the first cardiac transplant in England, my first cardiac transplant, Digoxin babies, Pipeline swaps, vigilance, old drugs, new drugs, bullying, obsessionality, and Teflon I have given parts of this talk before and never finished and this will be no exception.

The first public demonstration of Anaesthesia in 1846 was not the first anaesthetic, with prior claims made for W Crawford Long in 1842. In 1823, Henry Hill Hickman anaesthetised animals with carbon dioxide and amputated limbs to assess the effects. He wrote about his findings to Sir Humphrey Davy, then President of the Royal Society. Apparently, the work was never read, and later an article in the Lancet rejected his ideas as "Surgical humbug". Despite support from Napoleon's field surgeon, Barron Larry, the French also did not pursue this idea. Hypoxic anaesthesia nevertheless was practiced for many years for Dental anaesthesia, the early McKesson apparatus able to deliver 100% Nitrous oxide which was used for induction. A variety of fail-safe devices, gas failure alarms and indexing of delivery systems have improved safety. Oxygen analysers in the breathing systems were suggested in the 1970s although not essential and most hospitals did not have these. My first anaesthetic was given in 1974 using a Boyles machine. Monitoring was simple, a finger on the pulse and a blood pressure cuff attached to a von Recklinghausen's oscillotonometer(1). At this stage almost all theatres relied on gas cylinders which were colour coded and attached to the machine using a pin index system (1952) and bodok seal. A mechanical oxygen failure device, the Ritchie whistle, was present on many but not all machines.

In 1981, I travelled to Rotterdam for a two-week locum. On my first day, the first patient was a Surinamese for hysterectomy and after 30mins the oxygen alarm signaled a falling oxygen level despite rotameters suggesting a 1:2 mixture of Oxygen:Nitrous oxide. At the same time the pipeline oxygen pressure alarm showed increased pressure. The oxygen level continued to fall to 18% before I opened the valve on the oxygen cylinder, and disconnected the pipeline. The oxygen immediately returned to 33%. Engineers had incorrectly connected the high pressure air line into the oxygen pipeline whilst doing renovations replacing the oxygen with air. Three years earlier, the UK had instituted a permit system for work on gas pipelines, including a physician sign-off prior to use. I wrote a letter to Anaesthesia suggesting the cost of defense of a hypoxia claim would be less than the cost of making analysers mandatory but was told that point had been made many times. Nothing changed really until pulse oximeters were available which actually monitored the patient. They were developed in Japan in 1972 by Aoyagi and Kishi and became available commercially from 1981. I would not see one until 1985. Capnometry and end tidal agent monitoring was available and in an effort to control escalating legal costs for medical malpractice, a minimum monitoring standard was adopted at Harvard and subsequently endorsed on 21st October 1986 by the ASA(2) (US). So in the space of ten years we had gone from total reliance on pulse, blood pressure and patient colour, to electronic devices capable of displaying physiological signals. A recent pipeline swap in Sydney reminds us of the requirement for anaesthetic involvement in checking pipelines after engineering work.

Epidural analgesia for labour in Sheffield was introduced at the Jessop Hospital by David Nicholas against a tide of midwife opposition. In 1978 I was personally instructed by David, and in order to become proficient we were resident on call 24/7 for one week during which we were expected to have the opportunity to insert 20-30 epidurals. Often the call came as the patient was nearing delivery, the patient was by this stage in severe pain and had worn the midwife down. Caesarian sections were in the

main performed under general anaesthesia. There was no anaesthetic technician, which required careful preparation of the machine and equipment so it was all within reach. Failure to intubate required a switch to spontaneous breathing via a mask using ether via an EMO draw-over inhaler. The Confidential enquiry into perioperative deaths in Scotland (1972-5) published in 1978 found anaesthesia had now become the leading cause of death in women delivered by Caesarian section (5 of 13 deaths). Maternal mortality from all causes had declined significantly whilst that for anaesthesia had not fallen. The two main causes of anaesthesia related death were aspiration and hypoxic cardiac arrest principally due to oesophageal intubation. These statistics resulted in a revaluation of general anaesthesia and a shift towards regional anaesthesia.

My introduction to Anaesthesia at the Jessop Hospital in Sheffield, as a medical student was at least with a trained anaesthetist, Dr David Edbrooke. At this time, Sheffield was one of the few medical schools where undergraduates were given instruction in anaesthesia. In the same year, a colleague of mine, Tim Shaw, also started anaesthesia but in a very different way. As a medical house officer, he was informed that his role was to give anaesthetics for patients in Casualty who required fracture manipulation or abscess drainage. His 'instruction' was given by the Casualty consultant and consisted of a brief introduction to the Boyles machine and how to operate it using nitrous oxide, oxygen and ether.

Specific training programs in anaesthesia were introduced after the second world war, and Pearl Harbor is in some way partly responsible for that. In December 1941 the Japanese bombed the 5th fleet at anchor in Pearl Harbor resulting in mass casualties. Anaesthetists were told that "iv anaesthesia was the cause of more fatal casualties among the servicemen at Pearl Harbor than were the enemy bombs".(3). Was it fair to blame thiopentone? The hospitals were overwhelmed by patients, had inadequate fluid and blood replacement, oxygen or ventilator support and pressed unskilled personnel to administer iv anaesthesia sometimes in over dosage. It became clear that better training was required and it was 'no longer appropriate for any junior doctors or nurses to administer sophisticated anaesthetic drugs'.(4). Prior to world war 2, there were few career anaesthetists, often the surgical dresser (intern) was pressed to give anaesthetics. The University of Sydney began a Diploma of Anaesthetics course in 1945 and in 1948 the Faculty of Anaesthetists at the Royal College of Surgeons of London was established. A similar Faculty of Anaesthetists at the Royal Australian College of Surgeons began in 1952. The UK Fellowship examination started in 1953.

When I began as an SHO in Anaesthetics, surgeons would limit surgical time as mortality increased with duration of anaesthesia, and often excluded patients on the basis of age, weight and smoking. Almost all patients over the age of 30 were edentulous and came to the operating theatre without teeth after premedication with Omnopon and Scopolamine. Vomiting on induction was fairly common and we were taught how to manage this very early in our careers. An oral airway was always used, and depth of anaesthesia was judged by the signs described by Guedel.

The Woolley and Roe case. As a junior anaesthetist training in Sheffield there was considerable reluctance to perform in spinal anaesthesia following the Wooley and Roe case in nearby Chesterfield. Two consecutive patients, namely Wooley and Roe became paraplegic following spinal injections of tetracaine for relatively minor surgery. The devastating complication was investigated and court proceedings ensued. Professor Sir Robert Macintosh was called upon for expert opinion and formulated the theory accepted at the time, that phenol had leaked into and contaminated the ampoules resulting in neurotoxicity. The anaesthetist, Dr Graham was acquitted based on the Bolam principle that he could not be expected to know this and had the standard of knowledge of competent anaesthetists in 1947. Dr Graham did not believe the 'invisible crack theory'. The case was revisited by Roger Maltby and more believable cause was found(5). It seems that the theatre sister responsible for sterilizing the needles had left acidic descaler in the sterilizing water boiler. No compensation was awarded to Wooley or Roe. A third patient severely ill from intestinal obstruction died a few days after operation, had probably also suffered neurological consequences. Interestingly, Lord Denning reviewed the case in 1955 and said there was no way compensation could be given. The case changed the legal judgements in medical

negligence to pro-doctor rather than pro-plaintiff. It is suspected that Lord Denning had in mind a no blame system akin to New Zealand's.

In the early 1974 there were six different different induction agents available to me and five muscle relaxants. Now at Auckland City Hospital we have refined this to 3 induction agents (Propofol, Etomidate and Methohexitone) and 4 relaxants (Atracurium, Rocuronium, Vecuronium, and Suxamethonium). The use of Suxamethonium has waned and Tetrahydraminacrime has disappeared. Methohexitone was widely held to be associated with seizure activity and promoted for ECT therapy due to its lowering of seizure threshold. Administration was associated with limbs movements and the literature, mainly anecdotal letters suggested this implied seizures. I was asked by Neurologists at GOS to give methohexitone to terminate a seizure which it did. Looking back I found that seizures attributed to the drug were merely the excitation it produced and this was based on a letter to a journal. This old pearl was handed down as being true from then on. Articles in journals usually used Student's t test for statistical analysis and 20 patients were the usual number needed to provide an answer. Nowadays, outcome research is based on thousands of patients. We tend to believe what we have learned in based on good foundation research but often that is not true.

I spent one year on fellowship at 'Sick kids' in Toronto. Only a few years earlier in 1980-81 Toronto was rocked by a murder enquiry as babies on Ward 41 Cardiology died unexpectedly during the night. When the story got out, immense pressure was put on the local police chief to solve this apparent crime(6). The pall of this event still hung over the hospital in my time there in 1985. So what did happen? In January 1981 the hospital mortality committee reviewed 22 cases aged between 9 days and 12 months who had died and found 15 cases with unaccountable causation. By March 1981, 32 more babies and 3 children would die on the unit – a 625% increase over similar periods, most dying from midnight to 6am. A post mortem examination of one baby showed higher than normal levels of digoxin and similar findings were discovered in other deceased babies. The Toronto police were called in to investigate. Under significant press coverage, the Mayor pressured the police department to rapidly control this and make early arrests. They began interviewing nurses on the night shift, who generally were surprised, tearful and exclaimed lack of knowledge, but when interviewing Susan Nelles, she merely asked to speak to an attorney. The police immediately took this to imply guilt and arrested her on March 25th 1981. After a four month preliminary hearing, Susan Nelles was released as she was not on duty for all cases. She successfully sued for false arrest and received $190,000. The witch hunt then turned on Phyllis Traynor, who the US center for disease control in Atlanta identified as being present in all 29 suspicious deaths. Charles Smith was the pathologist involved, now disgraced. The cases remained unsolved. In April 2010, Nurse Lucia de Berk was cleared on seven charges of murder in a remarkably similar story to that of the Toronto. Gavin Hamilton believes no murders were committed. His book The nurses are innocent, believes the deaths were anaphylaxis to MBT, present in the rubber of pre-packaged syringes and which tests as Digoxin(7). The tests used to measure digoxin misread MBT as digoxin. At the same time as the Sick kid's deaths, Australian researchers warned of MBT's lethal effects especially in babies. Corporate governance is at the heart of a book by Sharon Stone, whose daughter died in another incident at Sick kid's hospital. The worrying feature is protection of institutional reputation perverting the availability of information and truth(8).

I was fortunate to be appointed as the only Registrar in the National Heart Hospital in 1981, working with Donald Ross and Magdi Yaccoub, famous cardiac surgeons. Donald was a contemporary of Christian Barnard at the University of Cape Town(9). A gifted surgeon he pioneered aortic homografts for right ventricular outflow tract reconstruction, developed methods of hypothermia and a pump oxygenator. In 1968 he performed the first cardiac transplant in the United Kingdom. The patient a 45 year old man survived 46 days before dying from sepsis. He performed 2 more heart transplants before halting further operations until rejection management was resolved. Two facts are of interest in this story. The first is the planning for the operation included practice on a pig. Unfortunately during preparations the pig escaped and was chased though the hospital. The second is that the anaesthetist involved, Alan Gilston, slept at the patient's bedside for the 46 days. His name is not mentioned in the

press reports. Alan was an inventor of the Gilston T piece and the Wallace cannula, and the founder of the UK Intensive Care Society in 197(10). During my time at the National Heart Hospital, Donald Ross decided a patient with critical multi-vessel coronary disease could be revascularised only by removing the heart to allow multiple grafts to be sutured. This was my only heart transplant case.

It was no doubt experimental in nature, and not repeated, but also probably marks the end of the era of surgical 'freedom'. I don't know the ethical situation surrounding the case, but I don't recollect any discussion on that particular issue.

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- For single shot nerve blocks performed under ultrasound.
- Safety code with high visibility X-pattern reflectors.
- Well recognised 30° bevel.
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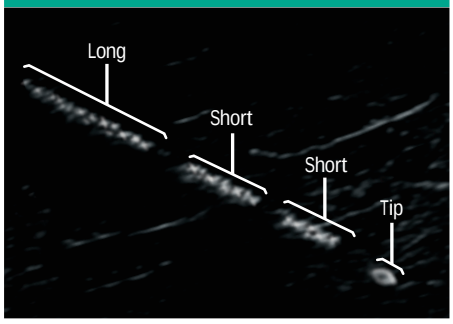
- 50 cm long DEHP-free injection tubing.

Safety Code



*Please note that the visualisation of the safety code may depend on a number of factors such as proper setting of the ultrasound unit, patient related conditions of the tissue, artefacts and proper alignment of the US probe and the needle.

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Ultraplex® 360 single shot needle is configured to meet the needs of clinicians who perform peripheral nerve blocks under ultrasound visualisation. Due to high visibility of the X-pattern, the needle is easy to visualise with minimum artefacts, even at steeper penetration. The 30° bevel together with a high quality coating provides excellent feel when advancing through tissue.

STIMUPLEX® ULTRA 360

Stimuplex® Ultra 360 has all the properties of the Ultraplex 360, as well as the capabilities for nerve stimulation.

SAFETY CODE

The B. Braun Ultraline 360 range features the safety code designed to help locate needle tip in challenging nerve block conditions. The X-pattern is strategically applied on the first 20 mm of the needle shaft to create the three segmented code “short-short-long”, distinguishing between the needle tip and the upper needle shaft.*

User benefits

- Highly visible echogenic X-pattern facilitates clear needle visualisation.
- Safety code enables easier identification of the needle and needle tip position.
- 30° bevel and insulation for optimal puncture and gliding behaviour.
- 360 degree X-pattern reflectors ensuring visibility from all sides.

References
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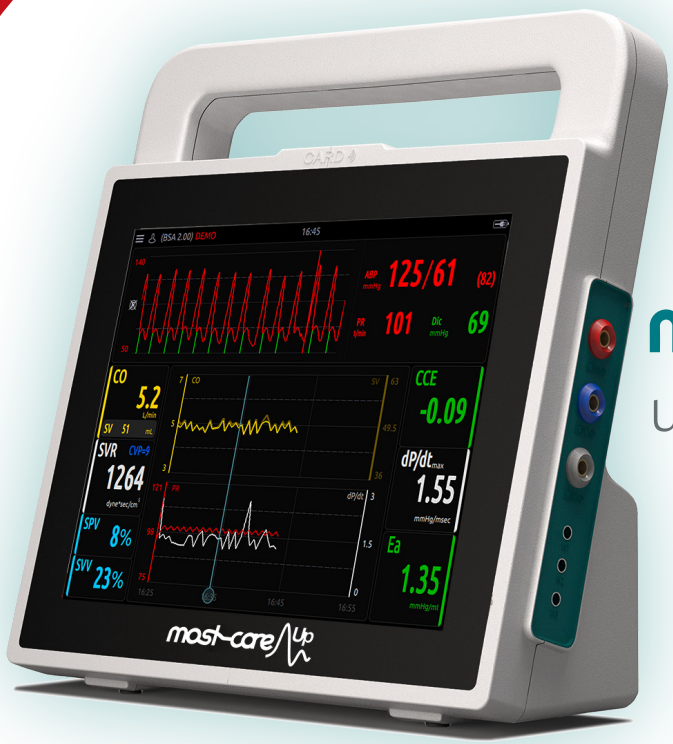

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1. Patel A & Nouraei S. *Anaesthesia*. 2015
2. Miguel-Montanes R. et al. *Crit Care Med*. 2015
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CONSIDER BRIDION (SUGAMMADEX) FOR SPECIFIC PATIENTS AND PROCEDURES

PATIENTS

- Cardiovascular risk[1]
- Obesity[2,3,4]
- Pulmonary disease[5,6,7]

PROCEDURES

- ENT Surgery[8,9]
- Open abdominal surgery[8,9,10]
- Laparoscopy[8,9,11,12]
- Procedures ending sooner than expected or short procedures.[13,14]

In New Zealand BRIDION is listed for the following scenarios:[15]

Where surgery duration is unexpectedly short

Unexpectedly difficult airway that cannot be intubated and requires rapid reversal of anaesthesia and Neuromuscular Block

Partial residual block after conventional reversal

Reversal of profound Neuromuscular Block from rapid sequence induction using rocuronium

Neostigmine or a neostigmine/ anticholinergic combination is contraindicated e.g. IHD, morbid obesity, COPD

Severe neuromuscular degenerative disease where Neuromuscular Block is required

References: 1. Norton. Residual neuromuscular block as a risk factor for critical respiratory events in the post anesthesia care unit Revista Española de Anestesiología y Reanimación 2013;60(4): 190-196. **2.** Leykin Y, Pellis T, Lucca M, et al. The pharmacodynamic effects of rocuronium when dosed according to real body weight or ideal body weight in morbidly obese patients. Anesth Analg 2004;99(4):1086-1089. **3.** Meyhoff CS. Lund J, Jenstrup MT, et al. Should dosing of rocuronium in obese patients be based on ideal or corrected body weight? Anesth Analg. 2009;109(3):787-192. **4.** Weinstein JA, Matteo RS, Ornstein E. et al. Pharmacodynamics of vecuronium and atracurium in the obese surgical patient. Anesth Analg. 1988;67(12):1149-1153. **5.** Amao R, Zornow MH, Cowan RM, et al. Use of sugammadex in patients with a history of pulmonary disease. J Clin Anesth. 2012;24(4):289-297. **6.** Murphy GS, Szokol JW, Marymont JH, et al. Residual neuromuscular blockade and critical respiratory events in the postanesthesia care unit. Anesth Analg. 2008;107(1)130-137. **7.** Hogg RMG, Mirakhur RK. Reversal of neuromuscular blockade: current concepts and future developments. J Anaesth Clin Pharmacol. 2009;25(4):403-412. **8.** Meretoja OA. Neuromuscular block and current treatment strategies for its reversal in children. Pediatr Anesth. 2010;20(7):591-604. **9.** Lemmens HJM, El-Orbany MI, Berry J, et al. Reversal of profound vecuroniunn-induced neuromuscular block under sevoflurane anesthesia: sugammadex versus neostigmine. BMC Anesthesiol. 2010;10(1):15. **10.** Welliver M, McDonough J, Kalynych N, et al. Discovery, development, and clinical application of sugammadex sodium, a selective relaxant binding agent. Drug Des Devel Ther. 2008;2:49-59. **11.** Tammisto T, Olkkola KT. Dependence of the adequacy of muscle relaxation on the degree of neuromuscular block and depth of enflurane anesthesia during abdominal surgery. Anesth Analg. 1995;80(3):543-547. 1**2.** Ogunnaike BO, Jones SB, Jones DB, et al. Anesthetic considerations for bariatric surgery. Anesth Analg 2002;95(6)1793-1805. **13.** Irvine M, Patil V. Anaesthesia for robot-assisted laparoscopic surgery. Contin Educ Anaesth Crit Care Pain. 2009;9(4):125-129. **14.** J. E. Caldwell. Clinical implications of sugammadex Anaesthesia 2009;64: 66–72. **15.** Pharmac. Section H Bridion Listing Pharmac 2014.

BRIDION® (sugammadex) is a Prescription Medicine, fully funded under Section H of the Pharmaceutical Schedule from 1 June 2013. Indications: Reversal of neuromuscular blockade induced by rocuronium or vecuronium. **Dosage & Administration:** Immediate reversal of intense block. 16.0 mg/kg IV, three minutes following administration of rocuronium (1.2 mg/kg) in adults, (including: elderly, obese patients, patients with mild and moderate renal impairment and patients with hepatic impairment). Routine reversal of profound block. 4.0 mg/kg IV following rocuronium- or vecuronium induced block when recovery has reached 1-2 post-tetanic counts; in adults. Routine reversal of shallow block. 2.0 mg/kg IV following rocuronium- or vecuronium-induced block when recovery has occurred up to reappearance of T2; in adults; 2.0 mg/kg IV following rocuronium in children and adolescents (2-17 years). **Contraindications:** Hypersensitivity to sugammadex or to any of the excipients. **Precautions:** Repeated exposure in patients; respiratory function monitoring during recovery; use for reversal of neuromuscular blocking agents other than rocuronium or vecuronium; coagulopathy; severe renal impairment; severe hepatic impairment; marked bradycardia, use in ICU; hypersensitivity reactions (including anaphylactic reactions); pregnancy (Category B2); lactation; infants less than 2 years of age including neonates; prolonged neuromuscular blockade (sub-optimal doses) and delayed recovery. Interactions: Potential identified with toremifene, hormonal contraception. Could interfere with progesterone assay and some coagulation parameters. **Adverse Reactions:** Dysgeusia, prolonged neuromuscular blockade, anaesthetic complication (restoration of neuromuscular function), hypersensitivity reactions varying from isolated skin reactions to serious systemic reactions (i.e anaphylaxis), bronchospasm and pulmonary obstructive events. Severe hypersensitivity reactions can be fatal. Events associated with surgical procedures under general anaesthesia. Isolated cases of marked bradycardia and bradycardia with cardiac arrest. **Marketed by:** Merck Sharp & Dohme (NZ) Ltd., Newmarket, Auckland. Based on Medsafe-approved Data Sheet, prepared 14 February 2014, available on **www.medsafe.govt.nz**
ANES-1125902-0002 TAPS DA4814MW BCG2-H BRI0003 08/2014.

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- *Menstrual cramps or period pain*[1]
- *Pain after surgery*[1]

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References: 1. CELECOXIB PFIZER® Data Sheet.

CELECOXIB PFIZER® Celecoxib 100 mg and 200 mg THERAPEUTIC INDICATIONS symptomatic treatment of pain & inflammation in osteoarthritis, rheumatoid arthritis & ankylosing spondylitis, management of acute pain & treatment of primary dysmenorrhoea. **CONTRAINDICATIONS** hypersensitivity to celecoxib or other excipients; allergy, asthma or urticaria with sulphonamides, aspirin, NSAIDs or COX-2 specific inhibitors; concomitant use of other NSAIDs; peri-operative use in cardiac or major vascular surgery; unstable/significant established IHD, PAD or cerebrovascular disease; active peptic ulceration; GI bleeding; estimated creatinine clearance <30 mL/min; CHF; severe hepatic impairment. See Data Sheet for details. **SPECIAL WARNINGS AND PRECAUTIONS FOR USE** Suspected or known CV disease or risk factors; history of CV disease; history of, or at risk of, GI ulcer disease or bleeding; asthma and rhinitis, with or without nasal polyps; renal and liver dysfunction; dehydration; serious skin reactions including exfoliative dermatitis, erythema multiforme, Stevens-Johnson syndrome, toxic epidermal necrolysis; concomitant use with ACE inhibitors, angiotensin receptor antagonists, digoxin, diuretics, beta blockers, corticosteroids, oral anticoagulants, cyclosporin or methotrexate; fluid retention & oedema; hypertension; cardiac failure; may mask fevers; reversible infertility, pregnancy & lactation; children. Discontinue at first appearance of skin rash, mucosal lesions or any sign of hypersensitivity. See Data Sheet for details. **UNDESIRABLE EFFECTS** More common: headache, dyspepsia, URTI, diarrhoea, sinusitis, abdominal pain, nausea. Rarely or Serious: drug rash with eosinophilia and systemic symptoms (DRESS, or hypersensitivity syndrome), syncope, CHF, ventricular fibrillation, pulmonary embolism, cerebrovascular accident, MI, GI perforation, GI bleeding, pancreatitis, liver failure, thrombocytopenia, agranulocytosis, aplastic anaemia, pancytopenia, hypoglycaemia, suicide, aggravated epilepsy, acute renal failure, Stevens-Johnson syndrome, toxic epidermal necrolysis, exfoliative dermatitis, sepsis, sudden death, angioedema, anaphylactoid reaction intracranial haemorrhage, myositis, hallucination. See Data Sheet for details. **DOSE AND METHOD OF ADMINISTRATION** use lowest effective dose for shortest duration possible. 200 400 mg daily. Maximum recommended dose is 400 mg per day. See Data Sheet for details. **MEDICINES SCHEDULE** Prescription Medicine Celecoxib Pfizer is a funded medicine – a prescription charge will apply. Before prescribing, review Data Sheet available from Medsafe (www.medsafe.govt.nz) or Pfizer New Zealand Limited (www.pfizer.co.nz) or call 0800 736 363. ® Registered trademark. V10517. PP-CEL-NZL-0037. TAPS NA9187. SPITFIRE J000757. 06/17. © Pfizer 2017.

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OASIS 2018 is heading to Blenheim!

6-7th April 2018, Marlborough Conference Centre

On behalf of the organising committee, it is my great pleasure to invite you to join us in Blenheim from 6th - 7th April for the Obstetric Anaesthesia Special Interest Symposium (OASIS) 2018. Our scientific sessions will draw on the combined knowledge of anaesthetists, obstetricians and maternal medicine physicians from Auckland's National Women's Hospital and other centres in New Zealand, and will address a broad range of topics relevant to peripartum anaesthetic care. Workshops include Anaphylaxis and Obstetric Haemorrhage Emergency Response workshops and two PBLD workshops focusing on challenging obstetric topics.

Our social functions kick off with a meet and mingle with friends and colleagues at the B. Braun Welcome Reception on Friday and a chance to test your skills at a Blind Wine Tasting Competition. Our Gala Dinner at the Omaka Aviation Heritage Centre is a rare opportunity to enjoy fine food and wine surrounded by the recently unveiled 'Dangerous Skies' World War II exhibition - an event not to be missed!

I look forward to warmly welcoming you to Blenheim next year.
Jo Doa, OASIS 2018 Convener

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