



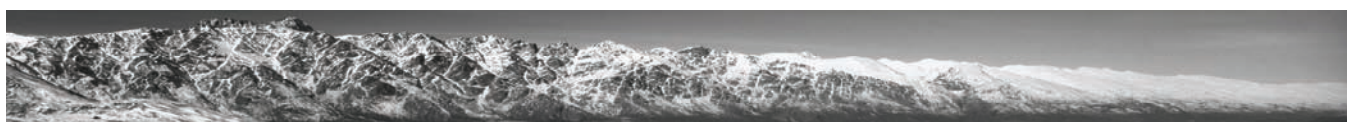
Annual Queenstown Update in Anaesthesia



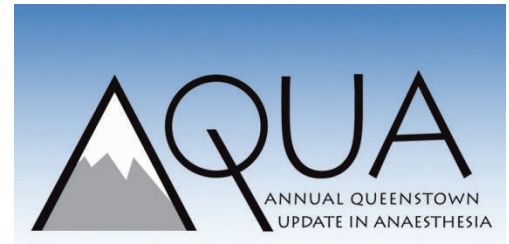
Programme and Abstracts, 2013

CONTENTS

Welcome	3
Social Programme	4
Scientific Programme	5
International Faculty	6
New Zealand Faculty	7
Abstracts	9
Perioperative Mortality	11
Post Op Pricklies	15
Aortic Stenosis – Truths and Myths	19
Paediatric Update – A Problem Based Approach	25
Nitrous Oxide – Does it Have a Future?	29
What to do at the Scene of a Road Crash	33
Trauma Care and the Anaesthetist – A Reality Check	37
Managing Diabetes	39
Trends in Vascular Surgery	43
A Cynic's Guide to Evidence Based Medicine	57
Emerging Issues in Orthopaedic Anaesthesia	59
Less Blood is More	63
An Antarctic Adventure – How Low Can You Go?	65
Sponsors	67







Welcome to the fifth Annual Queenstown Update in Anaesthesia. AQUA 2013 continues with the theme of the previous meetings, which is to provide anaesthetists with an annual update on core topics relevant to current clinical practice. Add in an excellent social programme and the setting of Queenstown in the ski season, and the ingredients are in place for a great conference.

AQUA 2013 features two international speakers who will add further depth and diversity to this year's scientific programme. The international faculty include – Professor David Story and Clinical Associate Professor Marcus Skinner. Between them they will cover topics of mortality, trauma, diabetes and surviving in the Antarctic.

Once again, we are indebted to our local faculty from around New Zealand for their hard work. Without their contribution, AQUA would not be possible. The organising committee would like to thank local speakers for their willingness to be involved.

AQUA 2013 will again capitalise on the attractions Queenstown offers for the social programme. The conference dinner will be held on Friday evening, at Walter Peak, reached by the spectacular trip on the TSS Earnslaw. A major challenge in the past has been to co-ordinate everyone to be at the dock on time... anyone arriving late this year will be made famous during the meeting on Saturday.

Although Coronet Peak has had a patchy season this year, we have arranged snow fall especially for the AQUA meeting. Night skiing will be held in conjunction with the AQUA BBQ on Saturday night we will conclude with live Bledisloe cup rugby.

We trust you will learn during the day, but also enjoy time off as well.

Once again we acknowledge the generous support of our sponsors. We have been well supported by industry this year and we would like to thank them all for being here. Please visit them in the exhibit area.

Enjoy.

Neil MacLennan

Martin Misur

Kerry Gunn

Karen Patching (KP)



SOCIAL PROGRAMME

Thursday, 22 August 2013

AQUA Welcome Drinks

5:00 p.m. – 7:00 p.m.

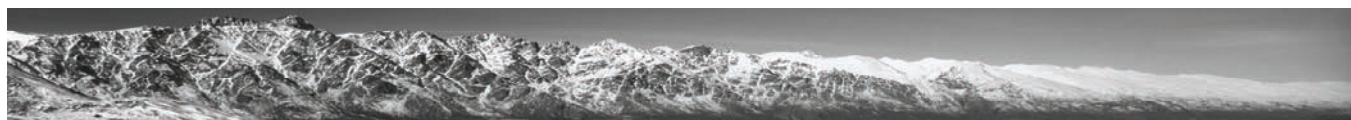
Friday, 23 August 2013

Ski bus to Coronet Peak departs Millennium Hotel	12:30 p.m.
Ski bus to Millennium Hotel departs Coronet Peak	4:15 p.m.
Make your way to Steamers Wharf, 88 Beach Street, Queenstown (Real Journeys)	5:15 p.m.
TSS Earnslaw commences boarding at Queenstown Wharf	5:45 p.m.
and departs Queenstown Wharf	6:00 p.m.
and departs Walter Peak	9:15 p.m.
and arrives Queenstown Wharf	10:00 p.m.

Saturday, 24 August 2013

Ski bus to Coronet Peak departs Millennium Hotel	12:30 p.m.
BBQ at Coronet Peak base building commences	6:00 p.m.
First bus to Millennium Hotel departs Coronet Peak (at conclusion of the rugby)	9:30 p.m.
Last bus to Millennium Hotel departs Coronet Peak	9:45 p.m.

NB – All transportation will leave promptly at the designated time. If you are late, you will be responsible for your own transportation.



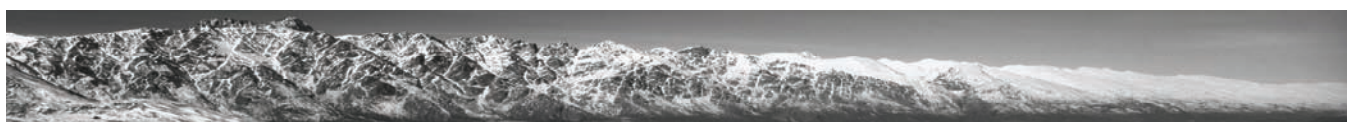
SCIENTIFIC PROGRAMME

Day 1 – Friday, 23 August 2013

0710-0755	Breakfast	Exhibitor Area
0755-0800	Welcome and Introduction – Rob Carpenter	Galaxy Room
Session 1:		
0800-0840	Perioperative mortality	David Story
0840-0905	Post op pricklies	John Foy
0905-0930	Aortic stenosis – Truths and myths	Joe MacIntyre
0930-0955	Paediatric update – A problem based approach	Ben van der Griend
0955-1025	Morning break	Exhibitor Area
Session 2:		
1025-1050	Nitrous oxide – Does it have a future?	Stuart Walker
1050-1115	What to do at the scene of a road crash	Tony Smith
1115-1145	Trauma care and the anaesthetist – A reality check	Marcus Skinner
1145	Close – Lunch packs available for you to pick up	Exhibitor Area
1230	Bus to Coronet Peak departs from Main Entrance, Millennium Hotel	

Day 2 – Saturday, 24 August 2013

0715-0800	Breakfast	Exhibitor Area
Session 3:		
0800-0830	Managing diabetes	David Story
0830-0855	Trends in vascular surgery	Andrew Hill
0855-0920	A cynics guide to evidence based medicine	Tony Smith
0920-0945	Emerging issues in orthopaedic anaesthesia	Gerard Willemsen
0945-1015	Morning Break	Exhibitor Area
Session 4:		
1015-1040	Less blood is more	Kerry Gunn
1040-1110	Medicolegal update	Denys Court
1110-1140	An Antarctic adventure – How low can you go?	Marcus Skinner
1140	Close – Lunch packs available for you to pick up	Exhibitor Area
1230	Bus to Coronet Peak departs from Main Entrance, Millennium Hotel	



INTERNATIONAL FACULTY

Professor David Story

Chair of Anaesthesia,
The University of Melbourne, Melbourne

Professor David Story is Chair of Anaesthesia at the University of Melbourne; and Head of the Anaesthesia, Perioperative and Pain Medicine Unit within the Melbourne Medical School. While he promotes research and teaching at the 14 hospitals affiliated with the University of Melbourne, he has two particular research interests – first, strategies to reduce complications after surgery and anaesthesia; and second, acid-base disorders and the underlying pathophysiology. He is a part time anaesthetist at the Austin Hospital Melbourne, providing anaesthesia and perioperative care for all surgical specialties including liver transplantation and cardiac surgery. He also engages the broader community about perioperative care.



Clinical Associate Professor Marcus Skinner

Clinical Director, Department of Anaesthesia and Perioperative Medicine,
Royal Hobart Hospital, Tasmania

Marcus did his basic medical training in Hobart graduating in 1984, sponsored by the Royal Australian Air Force through the medical Undergraduate program. Following his return of service obligations with the RAAF as a medical officer he undertook specialist anaesthesia training. It was during his time in the military that he became a Trauma instructor with the EMST programme and realised the need for trauma training in developing countries. He has undertaken a range of military deployments including military aid to Banda Aceh after the Sumatra Tsunami. He is widely experienced in aeromedical retrieval and recently conducted the earliest medical evacuation in winter from McMurdo ice runway in Antarctica.



Marcus was one of the cofounders of the Primary Trauma Care course and has been involved with teaching the PTC programs in a number of resource poor countries and has been undertaking PTC courses in Vietnam at Viet Duc University Trauma Hospital since 2004 and has returned each year since. In 2011 he also took a neurosurgical team to work at the Trauma hospital in Hanoi.



NEW ZEALAND FACULTY

Dr Denys Court

Auckland City Hospital
Auckland

Dr John Foy

Perioperative Services New Zealand Limited
Auckland

Dr Kerry Gunn

Auckland City Hospital
Auckland

Mr Andrew Hill

Auckland City Hospital
Auckland

Dr Joe MacIntyre

Nelson Hospital
Nelson

Dr Tony Smith

Medical Director, St John
New Zealand

Dr Ben van der Griend

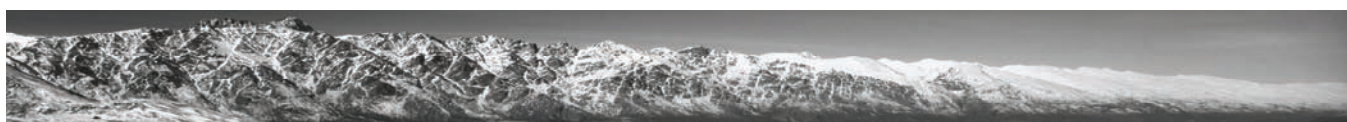
Christchurch Hospital
Christchurch

Dr Stuart Walker

Middlemore Hospital
Auckland

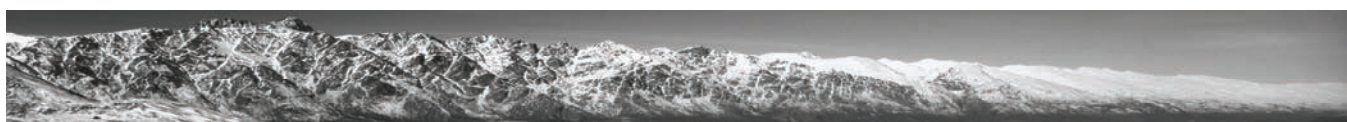
Dr Gerard Willemsen

Auckland City Hospital
Auckland





ABSTRACTS





PERIOPERATIVE MORTALITY

Prof David Story

Department of Anaesthesia, Perioperative and Pain Medicine Unit,
The University of Melbourne, Melbourne, Australia

This talk aims to discuss risk factors for perioperative mortality with particular emphasis on preoperative patient factors and postoperative complications.

Outcome Studies

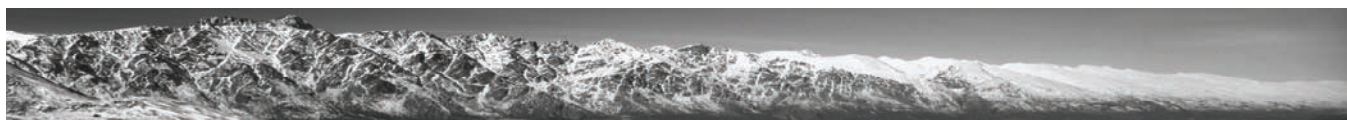
In 2010 we published a study of perioperative mortality and complications in Anaesthesia.¹ We called this study the REASON study; a slightly dodgy acronym: Research into Elderly Patient Anaesthesia and Surgery Outcome Numbers. The REASON study included around 4,100 patients. Based on previous work our primary hypothesis was that the overall rate of complications would be around 19%. The REASON study focused on elderly (70+ years) patients undergoing major non-cardiac surgery. Major surgery was defined as requiring at least one night's stay in hospital. While we excluded many patients undergoing day stay endoscopy and cataract surgery, some (often sick) inpatients requiring endoscopy were included. We were interested in the association of 30 day mortality with preoperative patient factors, a limited number of surgical factors (particularly non-elective surgery), and post-operative complications. Complications were pre-defined using definitions from previous Australian and New Zealand trials including the ENIGMA and MASTER trials. We followed up patients after 30 days, both in and out of hospital: we included more than just in-hospital mortality.

The most important findings of the REASON study were that, consistent with our hypothesis, 20% of the patients experienced complications within 5 days, and 5% died within 30 days. Because many patients developed more than one complication there were around 30 complications per 100 patients. Compared to patients who did not have a complication, patients who experienced one or more complications stayed a week longer in hospital. One in ten patients were admitted to critical care (ICU or HDU), half were admitted electively and the rest non-elective. Half the patients were ASA 3 and 13% were ASA 4. Compared to ASA 1 and 2 patients, the adjusted odd ratio (OR) for 30-day mortality was 3.0 for ASA 3 patients and 12.4 for ASA 4 patients. ASA physical status had a strong association with 30-day mortality. The value of the ASA status probably lies in it being a measure of global severity of co-morbidity. Many risk factors, such as diabetes are assessed in a yes / no dichotomous way without a measure of severity. This finding of the value of the ASA is in line with studies from the massive National Surgical Quality Improvement Program (NSQIP) run by the American College of Surgeons. The ASA scores stood out statistically over a number of individual comorbidities probably because of the global nature of the ASA score; for example both dialysis dependent renal failure and cardiac failure make a patient ASA 4.

Another grossly underestimated predictor of postoperative mortality is plasma albumin concentration. Decreased albumin is a marker of chronic disease and malnutrition. A NSQIP study suggested a curvilinear relationship between mortality and preoperative albumin concentration with an inflexion point at 30 g/L. That is the degree of hypoalbuminaemia reflects the severity of metabolic dysfunction. For simplicity we defined albumin less than 30 g/L as hypoalbuminaemia. Hypoalbuminaemia affected 1 in 6 patients and was associated with a more than doubled the risk of 30-day mortality.

Thoracic surgery had the highest mortality even when adjusted for patient factors. Increased mortality in thoracic surgery relative to other types of surgery is well known and important factor in the NSQIP data. The high mortality rate in thoracic surgery is likely to be multifactorial including factors such as: high incidence of aggressive cancer, frail patients, but also the possibility that intrathoracic surgery has greater systemic effects than abdominal surgery. When the odds ratios for mortality were adjusted for patient factors, the odds ratio for mortality in orthopaedics, plastics and urology fell considerably. These results highlight the importance of taking patient factors into account when looking at mortality rates; as patients become older and sicker and operative surgery becomes safer, patient factors are increasingly more important than the type of surgery.

In another study, the European Surgical Outcomes Study (EuSOS) group² examined mortality across Europe. Like REASON¹ they studied patients undergoing non-cardiac surgery requiring at least one night in hospital in



2011. Unlike REASON they studied all patients aged 16 years or older and looked at in-hospital rather than 30-day mortality. They studied 46,500 patients, of whom 1,855 (4%) died before hospital discharge. In the spirit of European unity the Researchers used the UK as a reference point (mortality 3%) and compared mortality between countries with odds ratios ranging from 0.44 for Finland (2% mortality) to 6.98 for Poland (18% mortality). Important factors for in-hospital mortality were, again: age, ASA status, urgency of surgery. An important finding was that three quarters of patients who died were at no point admitted to critical care services. Unfortunately, the EUSOS study did not measure complications.

Frailty

The 2010 National Confidential Enquiry into Patient Outcome and Death (NCEPOD)³ report on the elderly discussed assessing frailty, suggesting that this is another factor which should be considered when caring for elderly surgical patients. Frailty, like other things in life can be hard to define but vulnerability is an important factor: individuals with a diminished capacity to effectively compensate for external stressors, or more simply: easily broken. One approach is to view frailty as an overall global vulnerability.⁴ Some suggested indicators from the Canadian Veterans Heart Study for global frailty are: weight loss, exhaustion, slow walking speed and low physical activity, with a dichotomous yes/no with three or more. Another version, the Canadian Study of Health and Aging (CSHA) Frailty Scale is a bit like the ASA scale looking at dependence, with a severity component, however like ASA and anaesthetists the CSHA Frailty Scale is best in the hands of experts: our geriatrician colleagues.

CSHA Frailty Scale

1. Very fit – robust, active, energetic, well-motivated and fit; these people commonly exercise regularly and are in the most fit group for their age
2. Well – without active disease, but less fit than people in category 1
3. Well, with treated comorbid disease – disease symptoms are well controlled compared with those in category
4. Apparently vulnerable – although not frankly dependent, these people commonly complain of being “slowed up” or have disease symptoms
5. Mildly frail – with limited dependence on others for instrumental activities of daily living
6. Moderately frail – help is needed with both instrumental and non-instrumental activities of daily living
7. Severely frail – completely dependent on others.

Another way to consider frailty is deficit accumulation. In addition to their frailty scale The Canadian Study of Health and Aging also has the CSHA frailty index. This lists 70 deficits that can accumulate eg: falls, restlessness, and thyroid problems. In a recent surgical study using NSQIP data Velanovich et al⁵ found that a simple 11-point frailty index correlated with both mortality and morbidity for all surgical specialties. One advantage of the cumulative system in addition to its quantitative side is when considering limitations on treatment one point is the frailest older people can no longer accumulate deficits (ie become more frail); the next insult will cause failure of the system (ie death).

Unfortunately we do not have a simple robust quantitative measure of frailty that can be done by those with limited training eg orthopaedic registrars.⁶ Further, there may be an inverse relationship between frailty and anaerobic threshold, or VO_2 max for a given patient, which is an important area that requires further research. No matter how frailty is defined there is emerging evidence that it is an additional factor in assessing mortality risk as well as discharge destination for survivors.

Complications

In the REASON study the most frequent complications within five days of surgery were systemic inflammation and acute renal impairment. Systemic inflammation ranged from systemic inflammatory response syndrome (SIRS: inflammation without clear infection) to sepsis (inflammation with infection) to severe sepsis (inflammations with infection and organ dysfunction) to septic shock (inflammations with infection, organ dysfunction, and hypotension). Importantly after surgery there is often inflammation without infection (SIRS) that will depend on the site and extent of surgery. The definition used for acute renal impairment was 20% increase in creatinine. Patients with these types of complications included those at the more benign end of the disease spectrum, and yet they



were associated with marked increases in risk of mortality. These patients at the more benign end are likely to receive less attention in most surgical units, in part due to failure to recognise their association with mortality. An important study using NSQIP data looked at long-term outcome up to 5 years post-surgery. Patients who experienced either renal failure or systemic sepsis had increased mortality at 30 days, and even after 1 year and 5 years. This emphasises that these types of events around the perioperative period can have a significant long-term effect. Unplanned ICU admission was also a frequent and important independent predictor of mortality. Like preoperative ASA, unplanned ICU admissions are an indicator of the level of postoperative care required within an institution. We can identify preoperative variables that are particularly important patient factors for assessing risk: age, ASA, albumin, urgent surgery and emergency surgery. Importantly, these risks are increased when a patient has a postoperative complication.

As the safety of surgery has improved and rates of surgical mortality and risk of anaesthesia have decreased, patient factors, patient factors have become far more important in risk assessment. ASA status and low albumin are also associated with wound infections, that is a useful risk to communicate to orthopaedic surgeons in particular, as complications are associated with longer hospital stays.

Postoperative Care

Of the three phases of perioperative care: preoperative, intra-operative, and post-operative, the safest time is intraoperative with continuous anaesthesia care and high standard ANZ operative surgical care. Out of OR surgical care (surgical medicine) is an area for improvement, particularly postoperative care.

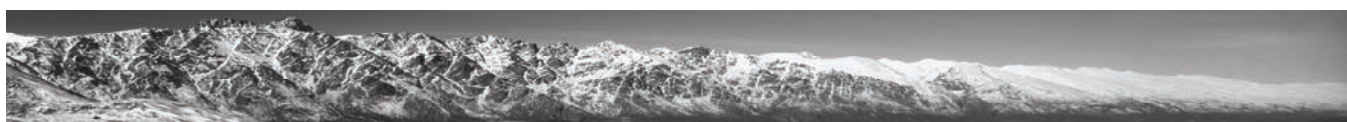
There are five domains in postoperative care –

- Surgical site management
- General medicine in the postoperative period
- Pain medicine
- Resuscitation
- Rehabilitation

In addition to allied health professionals, particularly nurses and physiotherapists, postoperative care is provided in the first few days by one or more of the following: surgeons, anaesthetists, ICU physicians, and internal medicine physicians. Currently, no medical craft group has expertise in all the domains of postoperative care.

Preventing and managing complications is the principal focus of postoperative care. This requires both surveillance and intervention. Using the NSQIP database Ghaferi et al⁷ reported in the New England Journal that among 80,000 patients in 150 hospitals in the United States mortality after complications almost doubled from 12.5 to 21.4% comparing the best 50 hospitals (lowest mortality) with the 50 worst hospitals (highest mortality). They called this “failure to rescue.” Two interrelated approaches to providing appropriate surveillance and intervention for appropriate rescue on the wards are co-management and critical care outreach. Unlike consultation which involves giving an opinion with the parent surgical unit conducting the suggested on-going management, co-management has been described as “...collaboratively managing patients with surgeons and specialists, sharing responsibility and authority.”⁸ Critical care outreach can be provided by the critical care specialties: anaesthesia, intensive care medicine, and emergency medicine. This can enhance both surveillance and intervention. Current critical care outreach includes – the respond blue team, medical emergency teams (MET), intensive care liaison nurses, and the most underappreciated, Acute Pain Services.

We have examined models of critical care outreach and co-management. In a pilot study of the Acute Pain Service⁹ providing added surveillance and interventions for identified high risk patients, we found that critical care outreach through the acute pain service was associated with decreased complications and mortality benefit but also found that this critical outreach was very resource intensive. We subsequently audited another clinical project, with a critical care registrar and nurse co-management team.¹⁰ While POST was popular with ward nursing staff¹¹ and surgical staff, length of hospital stay was increased rather than decreased as expected. This study had limitations and raises important questions about the reliability of hospital administrative data bases. In response to POST we are developing another model: the Surgical Medicine Fellow. This model differs from the POST in that the clinician is more senior (Fellow of the College of Intensive Care Medicine), and is involved in patient care for more of the inpatient stay. We plan to prospectively audit complications.



Future aims should be to improve both the accuracy and precision of risk assessments. This will help patients and medical teams decide on optimal care, which will increasingly include non-surgical options, or treatment limitation. Postoperative care requires on-going improvement with a particular focus on preventing, detecting and managing complications. Future education should focus on improving deficits in training.

References

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POST OP PRICKLIES

Dr John Foy

Perioperative Services New Zealand Limited
Auckland

Declarations

- Consultant to Southern Cross Healthcare
- Clinical Director of Southern Cross Hospitals Intermediate Care Units

Two Post Operative Chestnuts

- Hypoxia
- Hypotension

Hypoxemia

Firstly

- Urgent intervention?
- Increase the FiO_2
- Review events / history
- Focused examination
- Review investigations
 - CXR, ECG, ABG, Lung Fx

Causes to consider

- Inadequate PiO_2
 - eg Altitude
- Alveolar hypoventilation
 - eg Opiates
- Venous admixture
 - ie shunt
- Alveolar membrane disease
 - eg fibrosing alveolitis
- Hypermetabolism
 - eg MH, thyrotoxicosis

Venous admixture

- Airway obstruction
- Atelectasis
- Pneumonia
- Pulmonary Oedema
- Congenital conduits
- Pathologic conduits



Atelectasis is mostly what we deal with post operatively though...

- Problem: Collapsed Alveoli

Management – Get them open

- Incentive spirometry
- Sit them up, once safe to do so
- Chart turns
- Huffing, percussion, positioning
- Mobilise!
- F&P Airvo a modern option
- Non-invasive ventilation
- Failing that – consider the appropriateness and timing of IPPV

Hypotension

Firstly

- Urgent intervention?
- Fluid bolus if safe
- Review events / history
- Focused examination
- Review investigations
 - Hb, Creat, ECG, CXR

Focused cardiac ultrasound – You can learn this, you can do it!

- HART
- RACE
- FATE
- PGDipCU – Melbourne
- DDU – ASUM NSW
- ASCeXAM – USA

Tip – learn mostly from a cardiac sonographer

Information obtainable

- Volume status – LVEDD
- Volume status – IVC diameter
- LV systolic function
- Pressure state – atrial septal motion
- Segmental wall motion abnormality
- LV diastolic function
- RV systolic function – TAPSE
- $PASP = RAP_{ivc} + (TR_{Vmax})^2$

Prior management approach

- Volume
 - Like for like re losses
 - Until the patient is just about to drop their oxygen saturations, then stop volume 'maximised'
- Then vasopressor
 - eg metaraminol infusion 1-10mg/hour
- Then inotrope
 - eg noradrenaline in HDU / ICU



- While correcting the 'cause'

New option

- Diagnose the hemodynamic state
- Treat the state and cause
- Review treatment, with echo if necessary
- Maintain vigilance for more than one issue
- Management of hypotension
- Manage the hemodynamic state

Manage the hemodynamic state

- Hypovolemia = volume
- Primary LV systolic failure = inotrope +/- offload
- Primary LV diastolic failure = optimise lusitropy
- Mixed LV systolic / diastolic failure = inodilator
- RV failure = noradrenaline, off load ventricle
- Vasodilation = alpha constrictor of your choice
- Normal = tolerate and watch end organ function

As you manage / treat the cause as able

Intermediate Care

"Private, 76 year old, HT, post knee with epidural running, systolic 74mmHg, volume unresponsive" – old or new approach?

Approaches

Old

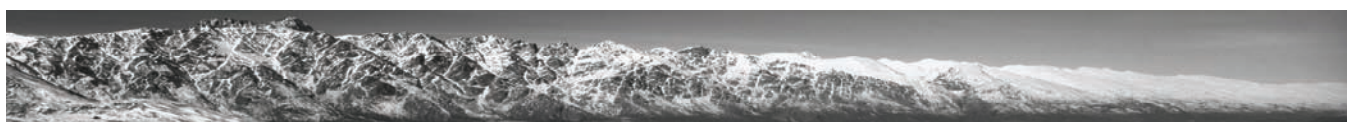
- No epidural
- More volume anyway
- IM metaraminol
- PO ephedrine
- Ward dopamine
- Tolerate it

New

- Intermediate Care
- 1:1 nursing
- Close observation
- Invasive monitoring
- Low dose vasoactive drugs
- Primary team responsible for medical care
- Clinical Director and Nurse Charge Nurse
- FCICM standard as a basis for quality

As instituted by Southern Cross Hospitals

- Not an HDU - HDU 21st century are closed, co-located, advanced cares
- Intermediate in capability to a ward bed and an HDU bed
- Same quality, based on FCICM, requires Level 2-3 ICU oversight
- Same policies, same management approach as oversight unit



Requires

- Clinical and nursing leadership
- Audit, and communication avenues
- Advanced training for a dedicated nursing team
- Guidelines and boundaries for safe care
- Ongoing training, and service improvements
- Ongoing liaison with oversight ICU

Key Requirement

- Strategic foresight by organisation
- Allocation of sufficient resources
 - Training, staff numbers, leadership

Results

North Harbour

- 250 patients in 3 years, no transfer from ICF to ICU / HDU
- All to ward
- Started to increase acuity of patients we care for
- Cardioversion training

Brightside

- 72 patients in a year, no transfers ICF to ICU / HDU
- All to ward
- Gaining confidence

Feedback

- Patients feel safe
- Nurses can do their job and get listened to
- Clinicians have a backstop they have control over
- ICU forget what a transfer to their HDU/ICU looks like
- MOH DAA 100% audit for both units NH 2012, BS 2013

Isn't it time you had one in your service?



AORTIC STENOSIS – TRUTHS AND MYTHS

Dr Joe MacIntyre

Department of Anaesthesia, Nelson Hospital,
Nelson

This talk reviews the preoperative risk stratification of patients diagnosed with aortic stenosis (AS) who are scheduled for non cardiac surgery. Understanding risk in these patients is important as it serves to guide decisions made by the clinician and the patient around the advisability of an operation.

The first study to discuss AS as a risk factor in non cardiac surgery was published in 1964.¹ A 10% mortality rate was described. In the intervening years little has changed with the 2009 updated ACC/AHA guidelines stating “severe AS poses the greatest risk for non cardiac surgery” with a “mortality risk of approximately 10%.”² Studies referenced in the guidelines are shown in Table 1.

Table 1 – AS and Risk in Non Cardiac Surgery – ACC/AHA Guidelines 2009

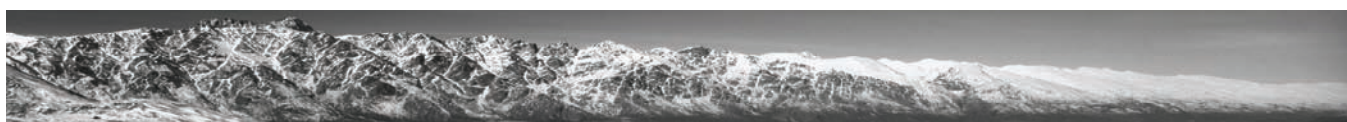
Study	Numbers	Study Type	Comment	Outcome
Goldman ³	23	Prospective case controlled	Clinical diagnosis of AS only. No stratification between asymptomatic and symptomatic patients	Goldman's Cardiac Risk Index – AS ranked 6th behind CCF, MI, PVC's, >70yrs, emergency
Raymer ⁴	55	Retrospective case controlled	Mix of moderate and severe AS	10% mortality in the AS and control group
Torsher ⁵	19	Case series	16 cases symptomatic	Two deaths 1. Elective bilateral TKJR 2. Emergency laparotomy
Zahid ⁶	5149	Retrospective case controlled	Unable to grade severity of AS or stratify for symptoms	No difference in mortality. Increased risk of AMI but only for low and intermediate risk surgery

In the same year European guidelines were produced which concluded “severe AS constitutes a well-established risk factor for perioperative MI and mortality.”⁷ The only new work referenced was a paper by Kertai in 2004.⁸ The results from this paper are shown in Table 2.

Table 2 – Aortic Stenosis and Cardiac Risk Factors (Kertai)⁸

Revised Cardiac Index Score	Cardiac Events in Controls	Cardiac Events in Patients with AS
0	0%	0%
1	3%	10%
2	3%	16%
≥3	6%	29%

AS in isolation had the same outcome as controls but increasing risk with RCRI suggests the risk with AS is not only dependent on severity and symptoms but on the interaction with other known risk factors. A detailed



examination of the remaining literature reveals further conflicting evidence and no clearer risk profile. A summary of this evidence is presented in Table 3.

Table 3 – Perioperative Risk Associated with AS in Non Cardiac Surgery

Study	Numbers	Study Type	Comments	Outcome
Agarwal ⁹	634	Retrospective case controlled	In patients with severe AS the only significant single complication was AMI – 71% in this group had coronary heart disease. In those with severe AS but without AMI only 40% had coronary heart disease Other variables measured – 30 day mortality, CCF, stroke were no different to controls	Primary outcome = 30 day mortality + AMI Significant factors – - High risk surgery (OR = 7.3) - MR (OR = 9.8) - Symptomatic severe AS (OR = 2.7) - Coronary heart disease (OR = 2.7)
Calleja ¹⁰	30 with severe AS	Retrospective case controlled	Compared outcome between asymptomatic moderate and severe AS > 75yrs undergoing non cardiac surgery	No increased AMI or death in patients with severe AS
O'Keefe ¹¹	48	Retrospective case series	75% symptomatic CCF 19% angina 7% syncope	No deaths but 7 (14%) perioperative events reported
McBrien ¹²	94	Retrospective case controlled	Mortality in hip fracture patients undergoing surgery	No increased risk of death with AS Sig difference in anaesthetic
Liebwitz ¹³	32	Retrospective case controlled	Patients undergoing surgery for hip fracture	No increased risk for cardiac events or death with AS
Adunsky ¹⁴	62	Retrospective case controlled	Patients admitted to hospital with hip fracture 19% were symptomatic	AS patients had surgical and non surgical treatments 6 AS patients treated non surgically. Two of these died 56 AS patients operated on. Two died. Mortality = 3.6% Non AS operated patients 94. Mortality = 3.3%

When trying to establish the risk associated with AS (or assessing the available evidence) what other factors do we need to take into account outside the evidence presented above. The natural history of AS alerts us to the fact once symptomatic patients cross into a higher risk group. It would seem sensible to translate this higher risk into the operative setting.

Presenting symptom	Mortality without AVR
Angina	50% die within 5 years
Syncope	50% die within 3 years
Dyspnoea	50% die within 2 years

Even if asymptomatic some patients are at increased risk with a small percentage of patients (approx. 4%) having a significant cardiac event / death or a rapid rate of progression to the symptomatic state requiring urgent AVR.^{15,16,17} Potential predictors of outcome in this group have been identified as –^{15,16,18,19,20 21,22,23,24,25,26}

- Coronary artery disease
- Functional status



- Exercise testing
- Severity of AS
- Aortic valve calcification
- Reduced left ventricular ejection fraction (LVEF)
- BNP/NT- BNP

Should patients undergo AVR, outcome is generally good with a mortality of around 2%. This applies to the “classic” AS patient with a good cardiac output and high flows across the valve producing high velocities. Some patients despite having severe stenosis determined by valve area have low gradients/velocities across the valve secondary to a low cardiac output.^{20,25,26,27,28} This group are classified as paradoxical low flow (PLF) and are mostly associated with a reduced LVEF. Stress echocardiography reveals subsets of aortic stenosis according to the effect of the dobutamine on LV contractility.^{27,28}

- Reduced LVEF but contractility reserve (CR) intact-dobutamine produces an increase in LVEF 20% and an increase in the mean aortic pressure gradient.
- Reduced LVEF no contractility reserve-dobutamine fails to produce an increase in LVEF or the mean aortic pressure gradient.
- Pseudo-severe AS – dobutamine produces an improvement in LV function increasing the mean gradient but also increasing the aortic valve area to a value $> 1.0\text{cm}^2$.

Collective perioperative mortality for patients with PLF is around 15%.^{16,17} In patients with preserved LV contractility reserve this reduces to 5%. In contrast, if contractility reserve is absent perioperative mortality can be as high as 30%. PLF is also seen in patients with a preserved LVEF. These patients have impaired LV relaxation, reduced stroke volume and reduced cardiac output suggesting significant impairment in intrinsic myocardial function. This “diastolic failure group” also do worse following AVR.^{29,30}

So what to do on Monday?

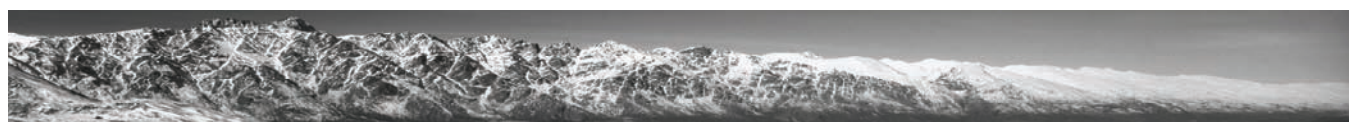
- Clearly define the “type” of AS. Is it high or low flow? If low flow consider systolic function / contractility reserve / pseudo-severe AS / diastolic function.
- Further determine cardiac status with BNP / NT-BNP
- Identify significant other cardiac pathology eg MR / RVSP
- “Characterise” the risk by evaluating the clinical picture – symptoms / NYHA / CCSAG / Lee Revised Cardiac Risk Index / function status / frailty.
- Adjust risk according to general risk factors for non cardiac surgery
- Add patient to AS database www.asdb.ac.nz!
- Calculate AortoScore – coming soon!

Summary

It seems severe AS is likely to increase operative risk, but questions remain as to how much and in what circumstances. The natural history of AS and the outcome data following AVR would suggest variable risk in this population based on symptoms, clinical factors, detailed echocardiographic and cardiac assessments. What translates from predictors of outcome in other fields of medicine to risk in non cardiac surgery has yet to be confirmed.

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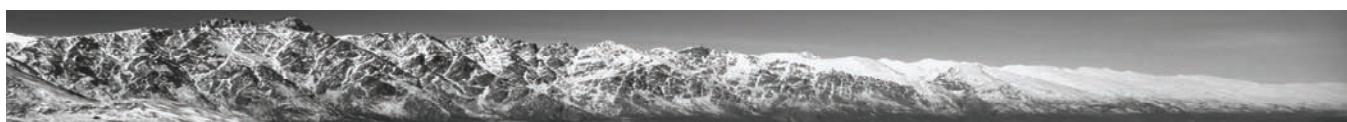
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PAEDIATRIC UPDATE – A PROBLEM BASED APPROACH

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Running late for your afternoon private list at St Elsewhere's, you meet little Johnny, a snotty-nosed three year old scheduled for an adenotonsillectomy for treatment of his snoring. His mother is very anxious and dad is outside having a cigarette. He weighs 12 kg.

Discussion Points:

1. Notes on obstructive sleep apnoea
2. Consent – Is anaesthesia safe?
 - a. Risk of death
 - b. Risk of neurotoxicity
3. ETT cuffed vs uncuffed
4. Intraoperative management
5. Fluids
6. Post operative analgesia – What's up with codeine?

1. Obstructive Sleep Apnoea (OSA)

OSA has incidence of 1-3% and in young children presentation differs compared to adults. Adenotonsillectomy improves >75% (cases with severe OSA or obesity are less likely to improve).¹

Looking at this case, risk factors include a history of snoring and the fact that he is a somewhat underweight for his age. Forty percent of snorers have OSA but OSA cannot be reliably diagnosed on history. Other risk factors (not present here) include congenital syndromes and cranial facial abnormalities, obesity, allergic rhinitis, etc.

On history, some good questions to ask include – does your child have difficulty breathing during sleep and are you worried about your child's breathing at night? Have you observed pauses in their breathing? Does your child have a restless sleep? Have you observed sweating? Does your child have behavioural problems?¹

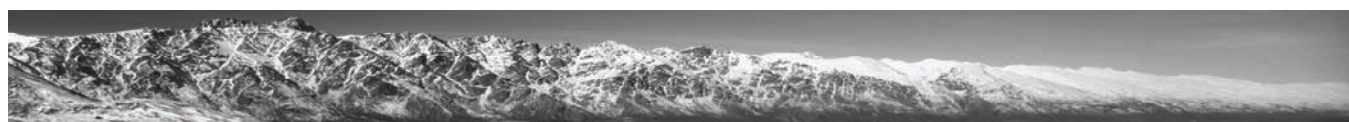
The gold standard for diagnosis is polysomnography (sleep study). However, practically very few children get a sleep study. It maybe useful for risk stratification or to rule out those with central apnoeas vs obstructive apnoeas (eg syndromic children). Echocardiography is reserved for children with signs of RV dysfunction.¹

Consider booking a HDU bed for children younger than 24 months, those that are very underweight (<3rd centile) or with morbid obesity and those with significant neuromuscular disease, syndromes prone to airway obstruction, complex congenital heart disease, or cor pulmonale / pulmonary hypertension.²

2. Consent – Is Anaesthesia Safe?

2a. Risk of Death

ANZCA produces a triennial report on anaesthesia-related mortality. Inevitably, there are a number of problems encountered with mortality estimation but despite limitations, ANZCA "guesstimates" the anaesthesia related mortality rate was 1:55,490 or 0.18 per 10,000 anaesthetics. Looking specifically at paediatric anaesthesia-related mortality there is very little data provided.³



A recent audit from the RCH, Melbourne (shameless plug for my own paper) looked at mortality from 101,899 anaesthetics administered to 56,277 individuals. This provided good data that did not rely on voluntary reporting.⁴

Overall 24 hour mortality was 14.7 per 10,000 anaesthetics delivered and mortality within 30 days was 34.5 per 10,000. Cardiac surgery had a higher incidence of 24 hour (131.5 per 10,000 anaesthetics) compared with non cardiac surgery (9.4 per 10,000 anaesthetics). The group of patients with the highest 24 hour mortality were neonates undergoing cardiac surgery (≤ 30 days old) with a 24 hour mortality of 194 per 10,000 anaesthetics.

There were 10 cases from 101,899 anaesthetics of anaesthesia-related death identified, ie those cases whereby anaesthesia or factors under the control of the anaesthetist, influenced the timing of death. Therefore, in this audit, the incidence of anaesthetic-related death is 1 in 10,190 or 0.98 cases per 10,000 anaesthetics performed. This is much higher than the ANZCA data and other data from overseas.

In all 10 cases, pre-existing medical conditions were identified as being a significant factor in the patients' deaths. Five of these had severe pulmonary hypertension (top of my list for patients to avoid). Importantly, this demonstrated that anaesthesia is safe in children with no or minor medical problems. Interestingly there were no airway-related anaesthetic deaths.

2b. Risk of Neurotoxicity

This whole debate was sparked by a discovery in 1999 that 7 day-old neonatal rats brains exposed to NMDA receptor antagonists (including ketamine) for some hours undergo apoptosis.⁵

Apoptosis is simply programmed cell death as opposed to necrosis which is cell death from some sort of injury. In the brain, redundant neurons undergo apoptosis. During the brain's 'growth spurt' period neurons undergo synaptogenesis - the neurons are trying to connect with each other. It is thought that those that don't make connections, the redundant neurons, undergo apoptosis. However, the apoptosis seen following exposure to ketamine is by far more prolific than what is normal.

Most of the anaesthetic agents have now been implicated including midazolam, isoflurane and propofol.⁶ There appears to be a synergistic effect when multiple agents are given together. Interestingly, there is not only histological evidence of neurodegeneration, neonatal rats subjected to anaesthesia displayed learning / memory disabilities that persists into their adolescence and adulthood.⁷ There may be some evidence that alpha-2 receptor agonists (clonidine, dexmedetomidine) have some protective effect.⁸

Human studies rely on retrospective cohort studies, which unfortunately have a number of limitations and therefore it is difficult to draw any definitive conclusions. A few large studies have suggested some association between prolonged accumulative / multiple exposure and learning difficulties or behavioural problems.⁹ Of course association does not prove causation. Other studies, typically looking at single brief exposure, are negative. Therefore, it is possible that there is association between anaesthesia exposure and adverse outcome, but this cannot be definitely confirmed or ruled out.⁸

3. ETT: Cuffed vs Uncuffed Tubes

My practice is to intubate kids for tonsillectomies although LMAs are used very commonly and very successfully. Historically, in paediatric anaesthesia, we have used uncuffed tubes. The rule being in children the correct sized tube should pass without resistance through the larynx and should have a slight leak at an inflation pressure of 20 to 25 cm of water.

However the disadvantages of an uncuffed tube include difficulties in ventilation with the presence of leak (especially with changing lung compliance), the need for higher gas flows, the difficulty in estimating the right tube size and subjecting patients to tube changes and, even in the presence of a leak, there is no guarantee against pressure lesions.

Microcuff have improved the design of paediatric ETTs by utilising a high volume / low pressure polyurethane cuff which is designed to sit below the level of the cricoid ring. The thin polyurethane cuff creates a seal at a lower cuff pressure and thus there is a low incidence of post intubation stridor.¹⁰



However, there are disadvantages. For example, a smaller internal diameter tube is required (increased resistance), there is no Murphy eye and they tend to kink when warm and humid. They are also more expensive but this is potentially off-set by cost savings from low-flow anaesthesia.

4. Miscellaneous Aspects of Intraoperative Management

Do I spray the cords? – No, because it probably leads to more problems than what it's worth with an increased incidence of desaturation during anaesthesia.¹¹

Dexamethasone is associated with reduced incidence of PONV and pain¹² and not associated with an increased incidence of bleeding.¹³

5. Fluids

“Super-hydration” with 30ml / kg Hartmanns during the case helps reduce the incidence of post-operative nausea and vomiting.¹⁴

If post-operative fluids are required (very rare), it is important to use a solution with an adequate amount of sodium.

The debate around post-operative fluids began following the publication of a number of cases of children dying or suffering from permanent brain damage as a result of hyponatraemia, primarily caused by a presumed increase in ADH activity followed by the administration of hypotonic fluids.¹⁵

As a reaction to deaths from hospital acquired hyponatraemia in the United Kingdom, The National Patient Safety Agency recommends that 0.18% saline solutions be removed from stock in areas that treat children.¹⁶ The minimum concentration of sodium to be used for maintenance infusions is 0.45% saline. However, there is increasing evidence that isotonic fluids reduces the risk of hyponatraemia^{17,18} and should therefore be used in the postoperative setting.

6. Post-Operative Analgesia (with a focus on Codeine)

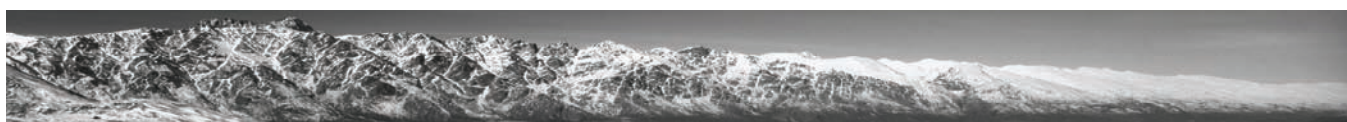
A recent case series¹⁹ reported on 2 deaths and one case of life threatening respiratory depression following the administration of codeine to children after adenotonsillectomy. Subsequent to this the FDA initiated an evaluation of the safety of codeine and identified 13 cases (including 10 deaths) associated with therapeutic codeine.²⁰ This has led to the FDA (in the USA) requiring manufacturers to add a boxed warning on the packaging (“black box”) describing the risk posed by codeine following adenotonsillectomy. A contraindication will also be added to restrict codeine use in these patients.

In the UK, the MHRA (UK) published a press release in June this year stating that codeine-containing medicines should not be used in children under 18 years with OSA post tonsillectomy and altogether in children under 12 years for any indication.

Here in NZ, Medsafe have taken a less dramatic approach and urged prescribers to “educate parents and caregivers of young patients about possible adverse effects associated with codeine use.”

Codeine is a prodrug – the activity of which depends on its conversion to morphine by the highly polymorphic CYP2D6 pathway. Poor metabolisers represent 5-10% of the population. Ultra-rapid metabolisers, represent roughly 1-10% of Caucasians and 15-30% of North African descendants. These patients are at risk of morphine toxicity, particularly dangerous in the setting of OSA and opioid sensitivity.

It is interesting to see that the US, UK and European regulatory bodies come out so strongly against codeine when the denominator (number of prescriptions) is huge compared to the handful of complications reported. It is unknown whether the alternatives (morphine, oxycodone) will prove any safer. My practice is to ensure regular paracetamol and non-steroidal analgesia is charted. I don't routinely chart an opioid but if I do, I chart oral morphine elixir.



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NITROUS OXIDE – DOES IT HAVE A FUTURE?

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Introduction

In order to have a future it is necessary, but not sufficient, to have had a past and to have a present. Nitrous oxide (N_2O) is the only anaesthetic agent which has a past and a present which has spanned the full extent of the age of anaesthesia. Even though the first official demonstration of N_2O was spectacularly unsuccessful, it rapidly established a place in anaesthetic practice which has been unrivalled and unsurpassed to this day. That this has occurred is testament to its efficacy and safety.

The general features of the pharmacology of N_2O including its major adverse effects have been known for a long time, and in spite of these it has continued to be in widespread use since its introduction. Nonetheless in recent years, a number of publications^{1,2,3,4} have highlighted the possible deleterious effects of N_2O . One of the most influential of these publications (ENIGMA 1) has come from The Australian and New Zealand College of Anaesthetists Clinical Trials Group, and has had a major influence on the use of N_2O in our region. The anticipated publication of ENIGMA 2 will, if positive add fuel to the pyre of N_2O , or if negative may allow a reprieve or even perhaps a resurgence.

This presentation will define the past to be from the introduction of N_2O and up to the publication of ENIGMA 1 in 2007. It will consider the present to be from ENIGMA1 to the publication of ENIGMA 2, and define the future as after ENIGMA 2 publication. This presentation will not predict any future past the year 2050. It will concentrate mainly on anaesthetic use, as its' use in obstetric and other forms of analgesia seem relatively secure,^{5,6} in spite of the fact that analgesic use is wasteful and polluting.

The Past

"I am sure the air in heaven must be this wonder-working gas of delight." Robert Southey, Poet (1774 to 1843).

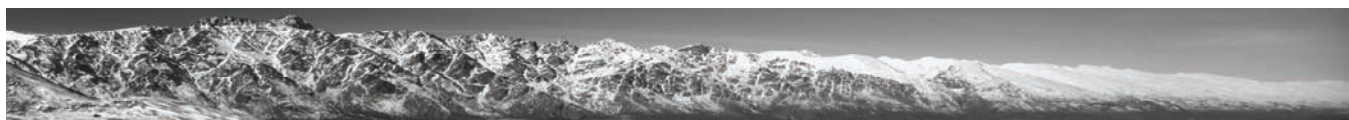
Many of the adverse effects of N_2O are dose related ie the effect is proportional to the degree (concentration and duration) of exposure.

Contrary to widespread belief there is very little evidence that for short, even ambulatory procedures there is any contraindication at all and in fact many under-appreciated advantages.⁷ Many of the alternatives, such as increasing concentrations of volatile agent or remifentanyl are not without concerns of their own. Ultra-short acting opioids carry the risk of opioid induced hyperalgesia, and chronic post-surgical pain (CPSP), which may actually be ameliorated by N_2O .^{8,9}

"In the 1990's pain physicians noticed an increasing number of patients being referred to pain clinics because of pain that had started after surgery and which continued for several months or years." (BJA editorial 2013 July). Perhaps the reduction in use of N_2O and the increased observation of CPSP are not coincidental?

The volatile anaesthetics have a greater risk of PONV than N_2O and all options are more expensive than N_2O . In a market report of medical gases, " N_2O followed O_2 in market share in 2011; however, N_2O 's market share is expected to plummet by 2018 due to the availability of anesthetic drugs with high profit margins and the growing use of medical air as an alternative anesthetic gas." See more at [Transparency market research](#).

For longer, inpatient procedures, in sicker patients there is less certainty. In a study of 250 inpatients randomised to 60% N_2O / isoflurane or air / O_2 / isoflurane, published in 1990, Eger found no difference in major (eg myocardial infarction, neuronal injury, hypoxaemia, infection, death) or minor (eg nausea, vomiting, headache, earache) untoward outcomes, between the groups, nor a trend to suggest that a larger data cohort would reveal a



significant adverse effect of N₂O. The duration of exposure was 1.5-4 hours.¹⁰ Their results support the continued use of N₂O.

Others have questioned this position. Badner⁴ showed N₂O-induced increased homocysteine concentrations are associated with increased postoperative myocardial ischaemia in patients undergoing carotid endarterectomy.

The Present – ENIGMA 1 (2007)

2,050 inpatients having major non-cardiac procedures were randomised to anaesthesia with or without N₂O. Expected exposure to N₂O of 2 hours. The primary outcome (length of hospital stay) was negative. There was increased “severe PONV” in the N₂O group. PONV has been the subject of much anaesthetic research. PONV is easy to study with many possible interventions, and it is clearly of some importance to patients. However it remains a short term problem and not one on which the future of a major plank of anaesthesia should be judged.

A meta-analysis of the effect of omitting N₂O on PONV concludes – “The clinically important risk of major harm (awareness) reduces the usefulness of omitting N₂O to prevent postoperative emesis.”¹¹

In ENIGMA 1 there were increased risks of “infective” complications (wound infection, pneumonia, pyrexia and atelectasis). There was a trend towards increased cardiac complications, which achieved significance when a subset of patients with cardiac risk factors was identified. This publication was preceded by editorials in the mass media^{12,13} and accompanied by an editorial in the same issue of *Anesthesiology*¹⁴ and almost certainly had profound effects on anaesthetic practice, especially in Australasia.

Subsequent to ENIGMA 1 there have been further publications arising from the original data, and also from other authors which have complicated the picture. In a long term follow up of all ENIGMA patients, Leslie et al¹⁵ found no difference in mortality (19%) or stroke (2.2%) but did show a difference in MI (overall rate 4.5%), the adjusted OR for MI with N₂O was 1.59 (95% CI 1.01-2.51 P0.04).

The publications of Professor Matthew Chan (from the Australian and New Zealand College of Anaesthetists Clinical Trials Group) support the continued use of N₂O because of its effects on chronic post-surgical pain¹⁶ and support the opposite because of its effects on leucocyte DNA and wound infection.¹⁷ Post-hoc analyses of large trials of anaesthesia such as POISE and GALA in patients with, or at risk of vascular disease, have both failed to show any cardiac risk attributable to N₂O.^{18,19} The VINO trial similarly has not shown any increased cardiac risk with N₂O.²⁰

Finally, N₂O is both a greenhouse gas and causes ozone depletion.

N₂O from medical use is less than 1% of anthropogenic N₂O which is in turn less than 5% of the anthropogenic climate change effect. Sadly, abolition of medical N₂O would have no effect on climate change.

The Future

An informal survey of 100 consecutive inhalational anaesthetics from our department in 2012 showed only 9 patients received N₂O.

We need to recognise that we are a tiny minority of the collective opinion of the global anaesthetic community. A recent analysis of the Cleveland clinic database showed that N₂O was used in 16,961 patients out of 49,016 between 2005 and 2009, with no deleterious effect. In spite of their conclusion that the results of this study do not support eliminating N₂O from anaesthetic practice there was a definite trend towards reduced use of N₂O over the 5 year period²¹ (see [Healthcare research analytics](#)).

Healthcare Research and Analytics Operating Room Audit (accessed August 13, 2012), also shows N₂O use has declined in the United States, utilised in 33% of all general anaesthetics given in 2009 but only 21% of those in 2011.

Data from the POISE re-analysis shows that the use of N₂O in patients at risk of cardiac complications varies from 5% of general anaesthetics given in Central and South America, up to 80% in India. Given the problems of providing affordable health care to a global population today of over 7 billion citizens, and even more importantly



projected to reach 9 billion by 2050, it would seem N₂O may well have a future even in patients at risk of cardiac events, regardless of the opinions of any of us.

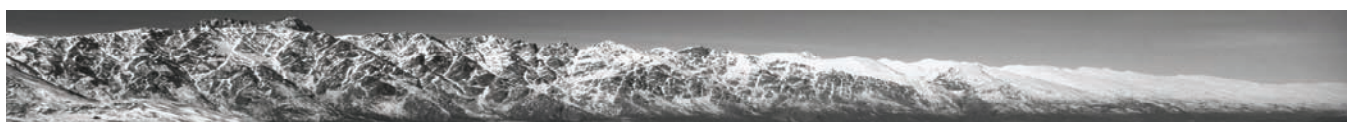
If ENIGMA 2 confirms all the findings of ENIGMA 1, then the dilemma that positive effects from N₂O on CPSP must be balanced against the risks of increased cardiac and infective complications remains. Anaesthetists will be left to consider the relative risks and benefits according to the patient's risk profile and the duration of exposure. It may be that N₂O may need to be given only for the first hour or two of anaesthesia in order to prevent central spinal cord sensitisation and limit the risk of infective complications and vascular risks of hyperhomocystinaemia.

With more information it may be we can use N₂O in a more knowledgeable way to gain the benefits and limit harms and retain N₂O as the anaesthetic “stock in the stew” in the foreseeable future. In patients at low risk of vascular events having moderate or short duration procedures, which still make up the vast majority of routine surgery, there should be no reason not to take advantage of the economic and clinical benefits of N₂O.

For those at high risk of PONV (Apfel score 4) it may be wise to avoid inhalational anaesthesia. For those at moderate risk of PONV, the decision to use N₂O would be more complex. For example, the risk of PONV with 2 prophylactic anti-emetics would need to be balanced against the risk of CPSP and perhaps the patient should be informed of the relative risks and benefits for full informed consent.

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WHAT TO DO AT THE SCENE OF A ROAD CRASH

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Most hospital staff will come across the scene of a road crash at some time in their lives. This can be a very disorientating environment. Sights (flashing lights, wreckage and blood), sounds (engines, generators and screaming) and smells (blood, vomit and petrol) all combine to overload your senses. It may be dark, wet and cold. They may have little or no equipment of their own and ambulance equipment is often unfamiliar. They often have unrealistic expectations of themselves and try to do the sorts of things they would do in a well-lit and well-staffed hospital. All of these features may combine to make it a difficult and stressful experience. Provided that you follow a systematic approach it need not be so.

When we review pre-hospital jobs that have not gone well the most common feature is that the scene has not been managed well as opposed to the clinical care of the patients not being performed well. In other words the key to successfully managing road crash is usually in managing the scene and not in managing the individual patients.

To Stop or Not

This is the first decision you need to make. It is always tempting to drive on past minor looking incidents but these may contain patients with significant injuries. If it does turn out to be minor then you can always get back in your car and be on your way. Be extremely careful stopping at motorway incidents if traffic is continuing to move past the incident at high speed. In general there is little benefit from stopping at metropolitan incidents if emergency services are already on scene, unless you have specific pre-hospital skills to offer. If emergency services are not on scene, or the scene is a rural one then you may be able to help.

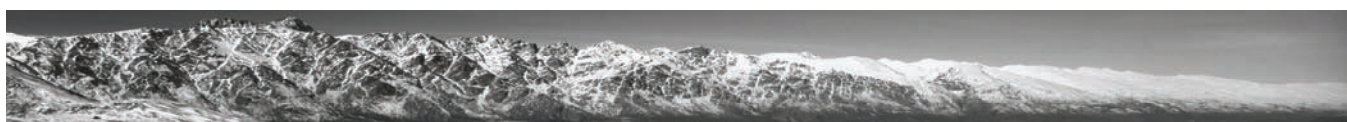
Remember SABC

S is for safety. Your safety and the safety of others is paramount. Park to protect the scene, leaving your hazard lights on. Leave your keys in the ignition if it is safe to do so (this facilitates emergency services subsequently moving your vehicle without having to find you). Wear protective and reflective clothing if it is available. Carry your cell phone. Pause as you approach the scene, look for and note all potential hazards. Do not approach if it is not safe. Delegate someone to control traffic.

A is for assess the scene. Assess the overall scene by walking from one end to the other, including a re-look for hazards. Turn off ignitions and put handbrakes on. Note the total number of patients including the number trapped. Make a visual estimate of patient's severity of injury. Remain hands off at this stage if at all possible. Utilise bystanders to provide initial care, providing these people with specific instructions, guidance and support. Do not let injured people wander away from the scene.

B is for broadcast to ambulance. Have the details written down if possible. Dial 111 and ask for ambulance. It won't feel like it at the time, but it is worth taking the time to do this yourself. If you delegate this task to someone else they invariably do not pass on the information that you ask them to. Give the exact details of the incident location, who you are and your assessment of the scene. Be prepared to answer a series of questions.

C is for communication and campaign plan. Triage and prioritise patients, sticking to the primary survey only at this stage. Continue to remain hands off if at all possible. Liaise with emergency services as they arrive and work together as a team. Concentrate on providing good first aid unless specifically trained and experienced in prehospital care.



The Keys to Success

The keys to success are remarkably similar to those in managing a single badly injured patient. They are –

- A team approach
- A team leader who ideally takes a hands off role and has a 'wider picture' perspective
- Clear, concise and explicit communication
- Forward planning
- Appropriate prioritisation and delegation

The Trapped Patient

Dealing with trapped patients can be a challenge and is best left to ambulance and fire personnel. Occasionally volunteer staff may ask a doctor to help. The keys to success are –

- Be safe. Protect yourself and patients from glass, sharp vehicle edges and undeployed airbags
- Assess the primary survey only initially. Keep patient interventions to a minimum and utilise bystanders to provide initial care. Provide these people with specific instructions, guidance and support
- Liaise with emergency services as they arrive. Work together as a team
- It is usually the fire service who will coordinate extrication. In general there are four stages to extrication
 - Vehicle stabilisation
 - Gaining access to the patients
 - Disentanglement of the patients from the vehicle
 - Removal of the patients
- Gaining access to the patients often involves removing the roof or door. Maximising access to the patients may require winching the vehicle away from whatever it has crashed into; this is usually easy and quick
- Reassess the primary survey. Continue to keep patient interventions to a minimum. Perform a very brief secondary survey looking for injuries that will alter the way in which you remove the patient
- Keep equipment and personnel within the working area to a minimum. Only those involved in hands on care or extrication should be in the working area. Be ruthless about keeping this area uncluttered. Monitors should only be taken into the working area in exceptional circumstances. They get in the way, they are easily damaged and they rarely alter what is done to the patient during extrication
- Use non-rebreather oxygen masks whenever possible. Try not to 'tie up' a pair of hands holding a ventilation bag and mask unless absolutely necessary. Check oxygen cylinders frequently – it is common for them to run out and for no-one to notice. IV lines are commonly snagged and removed during all phases of extrication. Avoid attaching fluid to lines unless shock is present in the primary survey
- Prior to removal re-examine the patient to ensure there is nothing you have missed that will affect the way you remove them (commonly missed are feet trapped under pedals). Ensure that all things that might snag (seat belts, clothing, oxygen tubing, IV lines etc.) are free and or secured
- Adequate pain relief, communication and coordinated removal are very important aspects of the extrication of patients with significant pain, particularly those with multiple limb fractures. Intensive Care Paramedics carry ketamine which is invaluable in this setting
- Have the stretcher positioned so that the patient is removed in one smooth move. Ensure that everyone involved in removing the patient knows where the patient is going and what position you want the patient placed in
- Except for exceptional circumstances most patients should be able to be extricated in under 20 minutes
- Once removed repeat the primary survey and perform a secondary survey

Helicopters

Be very cautious working around helicopters unless you are very familiar with doing so. Never approach a helicopter with the rotors turning unless you are signaled to do so (usually a thumbs up sign) by helicopter staff. Always approach a helicopter from the side in full view of the pilot or crewman and never approach a helicopter from the rear.

Helicopters continue to be commonly used in circumstances where there appears to be little benefit for the patient. The lack of space in the back of a helicopter significantly restricts the clinical care that can be provided en



route and this commonly compromises patient care. Thus a very significant time saving must be made in order for the benefit of a helicopter to outweigh the risks.

Which Hospital?

If the ambulance service has an established policy that dictates where patients are taken then this policy should be followed unless there are very strong reasons not to. In general, patients with major trauma should be taken, wherever reasonably possible, direct to a hospital capable of definitive care. There is increasing evidence from our trauma databases that this improves outcomes even when going direct to the hospital of definitive care involves bypassing a closer hospital. In general there is no role for 'stopping to stabilise' at a small local hospital unless the time and distance to definitive care are extraordinarily long (more than several hours).

Afterwards

If the crash is a significant one it is very common for the events to be played over and over in your mind afterwards. You are best to allow this process to occur and to allow it to 'play through your mind' whenever you find yourself thinking about it. In general, this results in a faster process (a shorter total time before you stop thinking about it) than trying to prevent yourself from thinking about it. Talk to your colleagues. Talk to the ambulance officers and consider attending their debrief. Follow up the patient's outcomes. Think about what went well and what didn't go well. Learn from your (and others) mistakes - we all make them.

What Equipment to Carry in Your Car

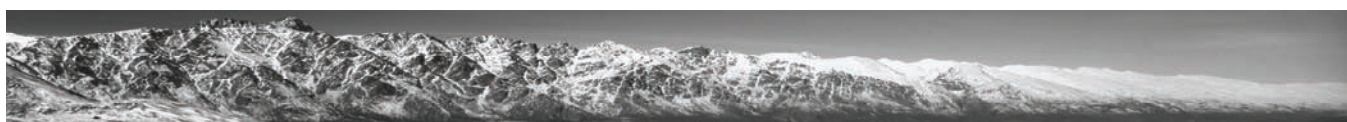
It is tempting to carry all sorts of things that might be useful. In reality, unless you are spending a lot of time in very remote areas, you are best to carry a small amount of gear that is cheap, takes up a small amount of space, doesn't matter if it has expired and is close at hand. I suggest the following –

- A high visibility vest
- Some gloves
- A small range of oropharyngeal airways
- A couple of dressing pads and elasticized bandages
- A couple of large long cannulae
- An ampoule or two of adrenaline
- A syringe and a needle
- A small torch

This can be put together for very little cost, takes up very little room and can fit in a small plastic bag under your seat. It will allow you to –

- Be visible
- Keep your hands clean
- Open an airway
- Compress external bleeding
- Decompress tension pneumothorax
- Treat anaphylaxis or life threatening asthma
- See in the dark

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TRAUMA CARE AND THE ANAESTHETIST – A REALITY CHECK

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“Reality is merely an illusion, albeit a very persistent one.”
Albert Einstein

Trauma anaesthesia is a significant part of the workload for many anaesthetists, particularly trainees. The management of trauma patients has matured significantly since a systematic approach to trauma care was introduced primarily by ATLS and the EMST programs, now nearly a half century ago. We are all aware that initial therapy begins with prompt pre-hospital medical care and efficient transportation to medical centres staffed by clinicians armed with a systematic approach to trauma management and well organised and designated trauma centres, where lifesaving procedures are immediately undertaken.

Trauma resuscitation with packed red blood cells and plasma, in parallel with surgical or interventional radiologic source control of bleeding, are the cornerstones of trauma management. Tranexamic acid is used in Europe with good results, but the drug is slowly being added to our pharmacy formulary. Recombinant factor VIIa can correct abnormal coagulation values, but its outcome benefit is less clear.

We ask questions such as “Does appropriately aggressive resuscitation with blood products, as well as adjunctive pharmacologic therapy, attenuate the systemic inflammatory response of trauma and improve outcome?” Such questions we hope, will be fully answered by appropriate trials and future investigations that determine whether for example, the concept of “early goal-directed therapy” such as used in sepsis may be applicable in trauma and if such an approach offers a similar survival benefit.

BUT...

How far have we come in a global sense? This talk is about Trauma and the anaesthetist – “a reality check.” For the vast majority of the world’s trauma cases patient transfer is not a therapeutic intervention, but a means of getting a patient to some form of care, with the reality that such care may be very limited. Oxygen and IV fluids, let alone blood, may not be available and the “golden hour” can be days. Often the anaesthetist is involved late in the patient’s management.

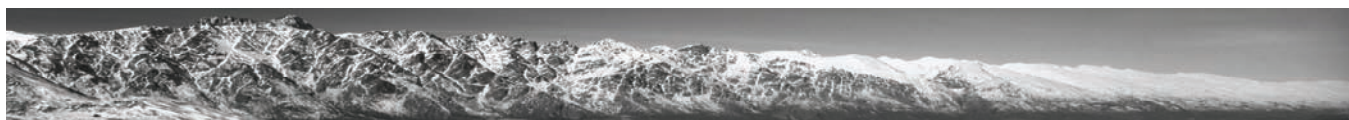
This talk hopefully will allow you a “Reality Check” on your busy “trauma” days.

In 2010/11, 1,367 New Zealanders died as the result of injury and someone is injured every 20 seconds. The Accident Compensation Corporation (ACC) received 1.5 million claims and paid \$2.2 billion in total claims costs.

Every year WHO reports there are 5.8 million deaths and more than 100 million people injured from violence and accidents. This causes a significant amount of disability and economic loss, especially considering that 90% of the problem occurs in low and middle-income countries (LMICs). Much of this burden could be decreased by prevention and improvements in trauma care. In 1990 road traffic accidents were the 9th leading cause of mortality worldwide with the 1.24 million deaths and 20-40 million people suffering injury. More than 90% of deaths that result from road traffic injuries occur in low- and middle-income countries.

It is projected that by 2030 road traffic accidents will be number 5. Regrettably the problem of road traffic trauma crashes and injuries does not “belong” to any specific agency, either at national or international level.

In May 2007 Ministers of Health from 193 WHO Member States met in Geneva for the Sixtieth World Health Assembly. The World Health Assembly is the governing body of the World Health Organization and WHA resolutions are the main policy mechanisms for directing WHO programs. These resolutions provide the WHO with a mandate to undertake activities in a specific area. The WHA assembly adopted resolution 60.22 on Trauma and Emergency Care Systems. The resolution was in 10 parts but its key message was to “urge” member states



to “do more in the area of trauma prevention and management.” There were a further 10 requests to the Director General of the WHO for various assistances to the member states so that they could in fact “do more.”

In order to promote such improvements globally, over 100 trauma care leaders from 39 countries from all WHO regions met two years later at a WHO Global Forum on Trauma Care in Rio de Janeiro. A large number of key stake holders attended, including 12 presidents and other officers from international professional societies, as well as 30 highly placed officials from national organisations.

Participants sought to “develop a strategy to promote greater political commitment to affordable and sustainable improvements in trauma care.” The summary statement from the Rio meeting was that “WHO should take the lead in developing a Global Alliance for Care of the Injured,” and the WHO’s Department of Violence and Injury Prevention and Disability (VIP) was to explore internally within WHO the steps needed to set up such an alliance.

In the meantime and for 10 years prior to that WHA plenary meeting in Geneva in 2007 (and the subsequent WHO Global Forum on Trauma Care in Rio in 2009), the Primary Trauma Care (PTC) organisation has been teaching and training trauma care responders and providers in low and middle income countries. (www.primarytraumacare.org). The PTC mission has been to provide health care workers with the necessary skills and knowledge to improve trauma management and the outcome from accident and violence. PTC has been vigorously promoting and propagating a strategy of affordable and sustainable improvements in trauma care all since the first PTC course was conducted in Fiji in 1997.

There are significant differences in outcome following injury in countries of different economic levels. One study reports mortality in the seriously injured increasing from 35% in the US, to 55% in middle income Mexico, to 63% in low income Ghana. Similarly injured people are nearly twice as likely to die in a low income setting than in a high income setting. The effect of improving organisation and planning of trauma care in high income countries have shown survival gains of 8–50% through the improved organisation and planning that comes with trauma systems.

Many injury deaths in low income settings could probably be treated well, and economic constraints are only part of the reason for the disparities in trauma outcomes between countries at different economic levels. Several programs in low income countries have already documented decreased mortality through the establishment of cost effective sustainable improvements in training, equipment, organisation and planning.

A Reality Check for us all – there is much that can be done to strengthen trauma and emergency care services through improved organisation and planning.

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MANAGING DIABETES

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This talk aims to discuss two areas of perioperative management of DM. The first is the changing role of the glycosylated haemoglobin (HbA1c) and the second is practical perioperative management of patients with diabetes.

HbA1c – Management and Diagnosis of Diabetes

While glycosylated hemoglobin (HbA1c) has been long associated with monitoring glycaemic control in patients with known diabetes, an important new use for HbA1c is to diagnose Type-2 diabetes.¹ Currently, blood sugar is being replaced by the (HbA1c) to diagnose many patients with Type-2 diabetes. Random blood glucose is still the best measure to diagnose Type-1. HbA1c is a form of haemoglobin that is measured to identify the average plasma glucose concentration over prolonged periods of time. The extent of haemoglobin glycosylation is related to the average plasma glucose concentration over time. Because the rate of undetected diabetes may exceed 10% in older patients, HbA1c will be an important screening tool for selected patients before surgery. This includes older patients and patients with vascular or cardiac disease. An HbA1c greater than 6.5% (associated with increased diabetic retinopathy) is one diagnostic threshold. An HbA1c of 6.0 to 6.4 indicates glucose intolerance or pre-diabetes; suggesting further investigation and monitoring glucose in the perioperative period. Importantly, patients with anaemia (shortened red cell life span), can have false negatives.¹

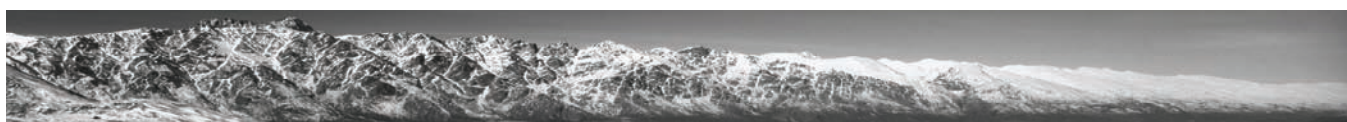
For many years the HbA1c has been an important marker of long term glycaemic control; with higher HbA1c being associated with increased diabetic complications. Further, HbA1c represents the average blood sugar over time. While the HbA1c signifies glucose control over the preceding three months, about 50% of the HbA1c is from the preceding month. Because there is a curvilinear relationship between HbA1c and the incidence of diabetic retinopathy and other complications, this relationship is used to define adequacy of control in established diabetes. An HbA1c less than 7% is seen as very good control while an HbA1c greater than 10% is seen as poor control.

In a move to SI units (and making our lives harder) the International Federation of Clinical Chemists (IFCC) are moving from the more familiar % units to mmol/mol.² If you think clinically in HbA1c as %: $\text{HbA1c in \%} = \text{HbA1c (mmol/mol)} / 10 + 2$. Americans are unlikely to adopt the IFCC recommendations on HbA1c and mmol/mol; they favour reporting an estimated average glucose. Average glucose has some benefits but has the risk of confusing average glucose and current glucose.

Using these new units in the perioperative setting, a rough guide is –

- HbA1c > 50 mmol/mol (7%) is strongly diagnostic for diabetes
- HbA1c > 70 mmol/mol (9%) indicates increased risk plus consider cancel for endocrine review
- HbA1c > 90 mmol/L (11%), only emergency surgery

There are limited studies, but with reasonably consistent results about HbA1c. First, HbA1c appears to be a reasonable screening tool for diabetes in hospitalised patients to diagnose unrecognised diabetes and to assess glucose management in patients with diagnosed diabetes.³ Current evidence is limited but it would seem reasonable to use HbA1c to test older patients being admitted for surgery, and patients undergoing surgery for conditions associated with diabetes: cardiac and vascular. Higher HbA1c is associated with increased infection and mortality, particularly with cardiac surgery. A patient with newly diagnosed diabetes is unlikely to be optimised, and risks minimised, for elective surgery. Similarly a patient with known diabetes but with poor glycaemic control is likely to have an increased risk of infection as well as other complications and mortality.



Elective joint replacement surgery is one area where risk may be reduced through screening with HbA1c. These suggestions are based on limited evidence. Importantly, observational trials are needed to further identify risk with intervention trials to assess the value of intervention, ie delay surgery and medically optimise the patient.

Practical Perioperative Management⁴⁻⁶

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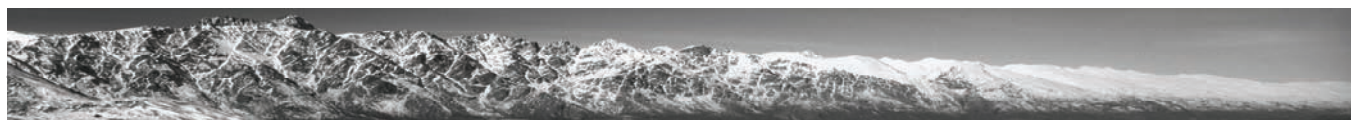
The perioperative period combines the factors of stress, fasting, nausea and vomiting, pain and analgesia, and any underlying chronic or acute illnesses. While these factors tend to drive up the blood sugar level, low blood sugar is the major risk for the patient. Hyperglycaemia, however, is associated with increased complications, particularly infections. Anecdotal reports suggest that some endocrinologists over estimate their understanding of perioperative medicine which can lead to conflict. Younger, motivated and (internet) educated patients (more typical of Type-1 patients) will expect a coherent plan. In the absence of a collaborative plan the next best option is hospital perioperative guidelines (or breadth of acceptable practice) produced by a collaborative group. Management regimens with oral drugs with or without insulin, or mixed short and longer acting insulins are relatively inflexible, and are often inappropriate for inpatient care, in part because fasting or nil by mouth is difficult to manage. Patients on these inflexible regimens are often better switched to basal / nutritional/corrective insulin or insulin infusions.

The key to perioperative management of diabetes is MEASURING THE BLOOD SUGAR.⁵ A good analogy is measuring blood pressure. It may be possible to detect hypotension (read hypoglycaemia) clinically, particularly in awake patients but rarely during anaesthesia and the early postoperative period. Similarly hypertension (and hyperglycaemia) is difficult to detect without measurement but is rarely an emergency and overtreatment should be avoided or at least recognised. The target perioperative blood sugar is around 8 mmol/L with a range of 5 to 10 mmol/L.⁷ Blood sugar outside this desired ranges can be called dysglycaemia. For patients with poor control undergoing urgent surgery this target may be higher, eg aim for 10 mmol/L with a range of 7 to 12 mmol/L. Like blood pressure, low blood sugar is almost always worse than high. Blood gas machines provide more accurate point-of-care testing than hand held devices. Hand held devices have an error of at least +/- 10% and are unreliable under a blood sugar level of 3 mmol/L and over a blood sugar level of 25 mmol/L, that is clinically emergent hypoglycaemia and important hyperglycaemia.

The approach most likely to minimise risk, particularly hypoglycaemia, is the simplest. Patients with diabetes should be first on an AM operating list when possible. Tell patients to not take any insulin or oral agents in the morning. Patients should measure their blood sugar, ideally after midnight, to reduce the risk of hypoglycaemia, with apple juice a good solution for possible hypoglycaemia. On admission, medical staff should be notified about patients with a blood sugar greater than 10 mmol/L, and more importantly, less than 5 mmol/L. Patients can bring their long acting insulin and if blood sugar is 5 mmol/L or greater take the long acting AM dose if due, either supervised or nurse administered, reducing the risk of wrong dosing. As patients with diabetes are done further down lists, particularly in the afternoon, management becomes more complex and dysglycaemia more likely, as patients will need food and then drugs to limit their hyperglycaemia.

Patients with continuous subcutaneous insulin infusions (CSII)⁸ can either continue their infusion on basal setting which may pose a risk if staff do not understand the device; which is quite possible with many devices and many settings. Diabetes educators are a good resource. Because the pumps administer only short acting insulin (eg aspart) any malfunction or disconnection that is undetected can lead rapidly to hyperglycaemia and eventually ketosis. However, malfunction with hypoglycaemia is more important. Another option is to switch with patients with pumps to an intravenous regular insulin (Actrapid) infusion set at the same basal rate as the subcutaneous pump. Once again the importance of actually measuring the blood sugar cannot be overemphasised.

Treating hypoglycaemia (blood sugar level < 5 mmol/L) has several components. First treat the blood sugar. This is done with 3 ml/kg of 5% dextrose (5g/100 ml) or 200 ml (10g) for most adults. The alternative is 0.3 ml/kg or 20 ml (10g) of 50% dextrose (50g/100ml). Profound hypoglycaemia (blood sugar level < 3 mmol/L), like profound hypotension, should be treated urgently but not excessively. The aim is to keep the blood sugar over 5 mmol/L. Like treating hypotension with pressors you don't need the whole bag or syringe to do this and if you do something is seriously wrong. The second aim is to explain the hypoglycaemia. Importantly medication errors need to be excluded, particularly those involving long acting drugs such as glibenclamide and glargine.⁸ In the absence of IV access but with appropriate level of consciousness, oral glucose, including 5% dextrose can be used. Subcutaneous glucagon injection can be used if IV or oral glucose is not an immediate option. Level of consciousness should improve over the next five to ten minutes.



While hypoglycaemia is more important, hyperglycaemia (blood sugar level > 10 mmol/L) is more frequent. A blood sugar level between 10 and 15 mmol/L requires attention but is relatively unimportant. A blood sugar level > 15 mmol/L has greater risk. The most effective way to treat hyperglycaemia is intravenous regular (Actrapid) insulin.

A rule of thumb for adults is IV Actrapid dose, units = measured blood sugar level – 8, with 8 mmol/L as the target.

eg blood sugar level 15 mmol/L = Actrapid dose of 7 units IV.

For patients with either Type-1 or Type-2 diabetes, keeping the blood sugar level in the suggested range of 5 to 10 mmol/L is a primary aim. Avoiding ketosis is important in patients with Type-1 diabetes is important but would require a fair amount of mismanagement to achieve in elective patients.

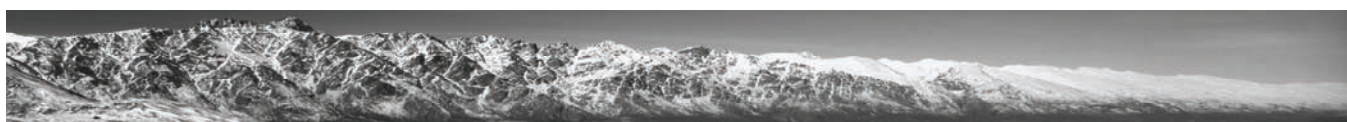
Postoperative in-patient management, particularly for patients requiring postoperative insulin should be done collaboratively with physicians – endocrine or general. There is still a strong tradition of insulin infusions. Insulin infusions often involve concomitant glucose infusions either as separate infusions, combined insulin-dextrose, or combined glucose-insulin-potassium (GIK) infusions. Insulin infusions have until recently been the mainstay for perioperative management of patients with Type-1 diabetes and many with Type-2. There are no comparative effectiveness studies however of insulin infusions and basal / bolus approaches with the newer insulins such as glargine (basal) and aspart (bolus).⁹ Bolus doses are divided into nutritional and corrective. It is clear that both infusions and basal / bolus are superior to traditional sliding scales which involve corrective insulin only, and described as a combination of myth and insanity.¹⁰

All efforts need to be made to avoid iatrogenic dysglycaemic injury: surveillance and intervention. Postoperative nausea and vomiting can increase the risk of a disconnect between consumed (or absorbed) glucose and insulin therapy. Aggressive prevention and management of nausea and vomiting may help avoid hypoglycaemia. Dexamethasone may increase the blood sugar level¹¹ but the antiemetic benefits may be more important. Patients with diabetes can have the usual maintenance fluids, accepting that 5% dextrose is not a maintenance fluid. In the past there has been concern that Hartmann's may be associated with hyperglycaemia from gluconeogenesis of lactate but this is not supported by more recent studies. Saline may have adverse renal effects and is best avoided in patients with Type-2 diabetes, particularly those with kidney disease.¹²

Finally – MEASURE THE BLOOD SUGAR!!!

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TRENDS IN VASCULAR SURGERY

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There is a general trend to endovascular management in both aortic and peripheral vascular surgery. Carotid intervention has moved back to surgical intervention for symptomatic lesions with less intervention for asymptomatic lesions. There is more emphasis on medical management, risk factor modification and selective non-operative treatment. Combined surgical and endovascular intervention has led to the development of the endovascular or hybrid theatre which is useful in both aneurysmal disease (thoracic and abdominal), peripheral arterial occlusive disease (PAOD) and trauma.

Aortic Aneurysm

There is a trend to endovascular AAA repair with most units offering stent graft repair in preference to open repair. Approximately 50-80% of aneurysms will be treated with stent grafts. Relative indications include "hostile abdomen" and increased medical risk especially respiratory disease. Relative contra-indications include "good" medical risk and young age. All randomised studies show a 50% reduction in 30 day mortality. The mortality advantage is not sustained at 2-3 years, with most of the advantage lost due to late re-intervention (DREAM and OVER). Quality of life advantages are also degraded with on-going surveillance and re-intervention. A number of devices are now available commercially. Improvements include lower profile delivery systems, more flexible limbs and re-positionable devices. Promising results have been seen with the Nellix sac anchoring prosthesis, which fills the aneurysm sac with a polymer filled gel, and has an extremely low incidence of type 2 endoleaks (residual aneurysm sac perfusion).

The use of truly percutaneous repair has been increasing. Large-hole closure techniques (pre-close) have allowed this. Not all femoral arteries are suitable though, with femoral calcification, re-operative groins and ectatic femoral arteries a problem.

Iliac aneurysms remain a problem for endovascular treatment. Acute occlusion of IIA is a persisting problem with a 20-40% incidence of buttock claudication. Iliac aneurysms can be treated with preservation of internal iliac perfusion using Iliac Bifurcation devices. These are mini-branch devices with a short covered stent being introduced into the IIA usually from the other femoral.

Challenges also remain in the treatment of aneurysms with a juxtarenal and thoracic aneurysms. Juxtarenal aneurysms can be treated with fenestrated devices, using covered stents into the renal and visceral vessels. There is additional renal artery risk with both open and endovascular repair. Only one company (COOK Medical) manufactures these and currently 6-8 weeks is needed to manufacture and sterilise. Off-the-shelf fenestrated devices have been proposed (COOK p-branch, Endologix Ventana) but these remain under research protocols. Branches, as opposed to fenestrated devices are also available, extending treatment to thoraco-abdominal aneurysms. These are used when the stent graft does not oppose the wall of the aorta at the level of the branch intended to be re-perfused. Again covered stents are used, with access from the axillary or brachial artery. Long sheaths are required from the upper body with multiple sheaths and two endovascular teams working from above and below often simultaneously. Multiple covered stents are required. These branches have had a pleasing short and medium term patency with 5-year freedom from branch intervention of 84%.

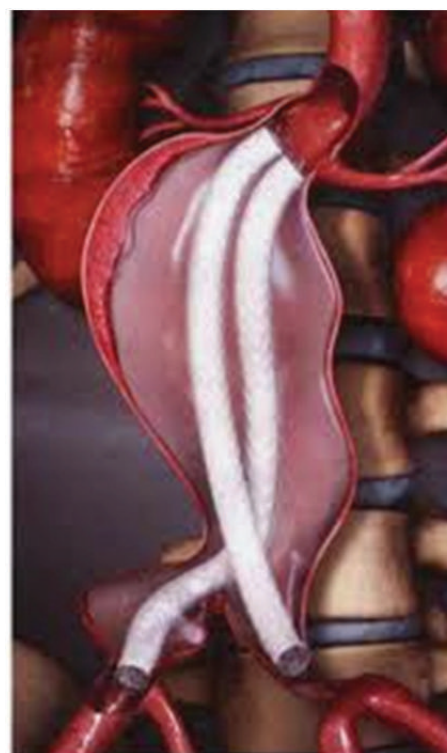


Figure 6. Endologix's Nellix AAA device.



There is additional risk of spinal cord ischemia (SCI) with these extensive repairs. Techniques to reduce the risk of SCI include preservation of the left subclavian artery and the internal iliac artery, the use of CSF drains and peri-procedural blood pressure manipulation to avoid hypotension. Additional risk factors include previous aortic repair, long lengths of coverage and coverage of the T8-L1 region (artery of Adamkiewicz).

The proximal extent of thoracic endovascular repair can be limited by the head and neck vessels but increasingly arch de-branching can be used, including de-branching from the ascending aorta.

Arch repair requires increasing co-operation between cardiac and vascular services although most are still done with Vascular Surgery lead due to endovascular expertise. There is additional cerebral risk, both anterior and posterior circulation, with arch manipulation. Parallel grafts (snorkel, chimney, and periscope) can be used when fenestrated technology is not available.

New Zealand runs a prospective audit of thoracic endovascular techniques, under the auspices of the Vascular Society of New Zealand.

Medical therapy and indications for aneurysm repair remains unchanged at 5-5.5cm for AAA and 6cm for thoracic aneurysms. The UKSAT and ADAM showed that among patients with a small abdominal aortic aneurysm there was no long-term difference in mean survival between the early-surgery and surveillance groups. Non-intervention for high-risk patients remains very valid.

Aortic Dissection

Urgent intervention for Acute Type A Dissection remains mandatory to prevent tamponade, aortic valve disruption and coronary artery ischaemia. This remains the preserve of cardiac surgery.

Acute type B thoracic aortic dissection is an important pathology often requiring repair. Intervention for complication is usually advised (branch vessel ischaemia, rupture and acute false lumen dilatation). Aims of intervention with proximal stent grafting include cover of the proximal fenestration, re-expansion of the true lumen and branch vessel stenting (STABLE trial). Uncovered bare metal stents distal to the covered stent can be used to help true lumen re-expansion. Intervention in "uncomplicated" dissection remains under investigation (INSTEAD trial).

There is limited ability to intervene in Chronic Type B Thoracic Aortic Dissection with late aneurysm formation being treated mostly with open surgery. Stent grafts have a much smaller role.

Vascular Trauma

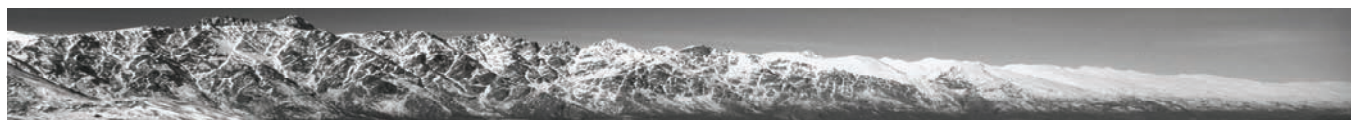
Virtually all blunt aortic trauma is treated with stent grafting. Endovascular techniques can also be used to treat difficult to access bleeding for both blunt and penetrating trauma (pelvis, abdominal, subclavian, renal).

Genetics

High-level genetic research is being done in Australasia (Dunedin and Cairns) to determine genetic inheritance and risk profiles. Millions of data analysis points using Genome Wide Association Screening (GWAS) can identify previously unknown correlation to genes and specific phenotypes.

Peripheral Artery Occlusive Disease (PAOD)

There is much time spent in diagnosis of PAOD helped with specific Vascular Laboratories. Non-invasive testing (pulse volume recording, plethysmography, ankle brachial indices, exercise testing) and ultrasound form the backbone for this.



Most centres will look towards investigation of PAOD in Rutherford class 3-6, although diagnostic dilemmas, young age and the suggestion of iliac disease (better results with intervention as compared with infra-inguinal) may be investigated earlier.

Symptoms	Rutherford classification
Asymptomatic	0
Mild claudication	1
Moderate claudication	2
Severe claudication	3
Rest pain	4
Minor tissue loss/ulcer	5
Major tissue loss	6

Risk factor modification has become vastly more important with smoking cessation, anti-platelet therapy, statin therapy, exercise regimes and medical treatment of diabetes and hypertension featuring. Statin therapy appears to have pleotropic advantages over and above its cholesterol lowering effects, leading to most vascular patients being prescribed therapy regardless of lipid profile.

Ideal Lipid Profile – New Zealand Heart Foundation Guidelines	
Total cholesterol	Less than 4 mmol/L
LDL cholesterol (bad)	Less than 2.0 mmol/L
HDL cholesterol (good)	Greater than 1.0 mmol/L
TC / HDL ratio	Less than 4
Triglycerides	Less than 1.7 mmol/L

PAOD Intervention

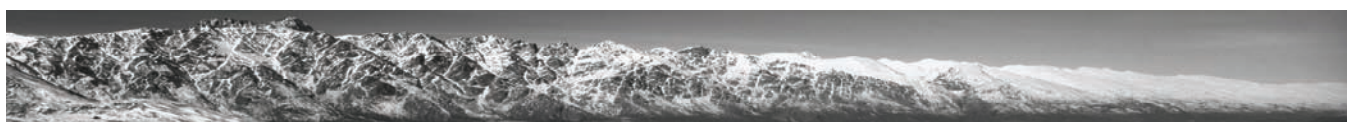
Most intervention is endovascular as a first line if there is felt to be advantage over open therapy (shorter lesions, stenosis rather than occlusion, not involving branches, more proximal lesions especially iliac). Plain Old Balloon Angioplasty (POBA) still remains the mainstay. Stents have found favour recently in the SFA with the BASIL trial suggesting that “In patients presenting with severe limb ischaemia due to infra-inguinal disease and who are suitable for surgery and angioplasty, a bypass-surgery-first and a balloon-angioplasty-first strategy are associated with broadly similar outcomes in terms of amputation-free survival, and in the short-term, surgery is more expensive than angioplasty.”

Options for leg endovascular intervention –

- Plain Old Balloon Angioplasty (POBA)
- Stents
- Drug eluting balloons
- Cutting balloons
- Drug eluting stents
- Covered stents
- Bio-absorbable stents

Mid and late re-stenosis has hampered intervention in the lower leg with SFA and popliteal lesions struggling to achieve less than 50% re-stenosis rates at 2 years. For critical limb ischaemia particularly with tissue loss this is thought to be less important as short-term healing with mid-late re-stenosis less important ie re-stenosis doesn't matter if the foot has healed.

Equipment which is available through smaller sheaths is very helpful with much femoral work being able to be done through 5Fr and even 4Fr sheaths. Groin closure devices have significantly helped the groin complications and early ambulation.



Tibial intervention has markedly increased with major advances in inventory and technique. Included in this is the angiosome concept where specific parts of the foot are supplied by specific tibial arteries and endovascular intervention should be guided somewhat by this –

- Low profile balloons
- Tibial / CTO wires (0.014/0.018 gauge rather than 0.035)
- Smaller access sheaths
- Longer balloons
- Dedicated stents with and without drug coating
- Retrograde and safari access
- Angiosome concept



Bypass surgery has reduced significantly although remains the mainstay for longer lesions and failed endovascular intervention. Combined procedures in hybrid theatres are increasingly common in this area particularly as management of the common femoral artery disease remains mostly surgical. There is no advantage for Dacron over PTFE but there has been a vast reduction in prosthetic graft use. Vein confers a 10-50% advantage over prosthetic (TASC) more marked the more distal (tibial) the bypass. The use of alternative sources of vein (contralateral great saphenous vein, small saphenous vein, arm vein) and the use of spliced vein (multiple vein segments of vein) can make these procedures more challenging and lengthy from a surgical point of view. There is no clear benefit of reversed vs non-reversed vein.

Rigorous surveillance of bypass and endovascular repair is useful. However there is less utility in US surveillance of iliac intervention (surgical or endovascular) and tibial endovascular intervention (due to calcification).

Renal Artery

There has been a significant decline in surgical renal intervention in the last 10 years. Almost all intervention is endovascular with surgery being limited to failed endovascular intervention and some procedure in combination with open aortic surgery.

There has also been a decline in renal angioplasty and stenting for ostial atherosclerotic disease. Both ASTRAL and CORAL have shown mixed results with only sub-group benefit. However there are a number of people with hypertension and some with ischaemic nephropathy who may benefit. There may be benefits in endovascular advances with low profile wires (0.014) and stents, the use of “no touch” vessel cannulation and covered stents to minimise the risk of athero-embolisation.

Renal angioplasty without stenting is the mainstay for fibro-muscular disease which is a smaller subset of hypertension patients typically the younger female.



Renal artery denervation has been trialled with promising results in the non-atherosclerotic hypertensive patients. This requires direct renal artery cannulation and then delivery of a noxious stimulus such as radio-frequency ablation to both renal arteries (SIMPLICITY). Early results are promising with results showing “Catheter-based renal denervation can safely be used to substantially reduce blood pressure in treatment-resistant hypertensive patients.” These are similar to older surgical techniques of renal artery denervation, which was abandoned due to procedural morbidity and some mortality.

Carotid

Some changes have occurred. There is increasing evidence for the use of medical services to detect patients at high risk of recurrent stroke after a cerebral event. This has been led by the Oxford Group who has championed the ABCD2 scoring system but also re-analysis of older studies (NASCET and ECST), which randomised medical therapy vs carotid endarterectomy (CEA). Early intervention will reduce the recurrent event rate although there is still some debate about the timing. Most units aim to have intervention within 2-3 weeks of the sentinel event. This often requires hospitalisation to achieve. No-referral or open access stroke clinics may assist. Significant advantages exist for CEA in symptomatic carotid stenosis with NNT about 5.

There have been major changes with the type of intervention for symptomatic carotid stenosis with a retrenchment for carotid angioplasty and stenting (CAS). Several trials including ICSS have shown a doubling in stroke rate after CAS. Many of these have been smaller strokes. MRI sub-studies have shown and even higher incidence of cerebral infarcts after CAS when compared with CEA.

High-risk patients may still be referred for CAS and results in this group are encouraging (SAPPHIRE). Specific indications include anatomic variants with high bifurcation, previous surgery or radiotherapy and possibly high medical risk. Some reservations exist in the post-radiotherapy group with a much higher incidence of common carotid lesions, which can be longer, more fibrotic and prone to embolisation. The radiation “arteritis” in the low neck can also be treated with bypass such as subclavian-to-carotid bypass. The major option with the higher risk patients is local anaesthetic CEA. This was shown in GALA to be equivalent and some enthusiasts use this routinely. A slick procedure, both anaesthetically and surgically, is required. A co-operative patient who is able to lie quietly for several hours is required. The incidence of shunt dependence is much lower than with other indirect methods of measuring cerebral perfusion (between 4-10%).

Intervention for lower grade stenosis (50-69%) is beneficial in some cases but patients much have a greater life expectancy. Stroke rate is reduced from 11% to 5% over 5 years for those with a 50-69% stenosis, compared with 26% to 9% over 2 years for those with >70% stenosis.

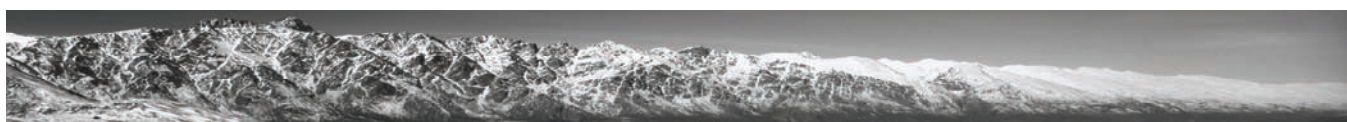
Mesenteric Revascularisation

Chronic gut ischaemia remains a challenge with most intervention using endovascular attempts first. Success and longer-term results are limited with a high re-intervention rate, poorer results in the coeliac artery and those with heavily calcified vessel origins. Surgical revascularisation has excellent patency (>90% at 5 years) but is more lethal with an 8-10% 30-day mortality. There remain diagnostic challenges in this area. There is also a small subset of young patients (almost exclusively female) with coeliac artery compression syndrome, some of which respond to surgical intervention. These should not have endovascular intervention due to poor results and stent compression.

Acute mesenteric ischaemia is a true surgical emergency and most are treated surgically, although catheter directed thrombolysis has been used. A laparotomy to determine gut integrity is useful.

Imaging

There is a major swing to non-invasive diagnostic imaging and few diagnostic catheter studies are done. There are advantages for and against each modality including Nephrogenic Systemic Fibrosis (NSF) and contrast-induced nephropathy (CIN). Most of the non-invasive studies allow interventional planning with marked reductions in contrast dose.



Modality	Advantages	Disadvantages
Ultrasound	Safe, no radiation, repeatable, reliable, excellent for surveillance, able to be done with anti-coagulated patient	Operator dependent, still has difficulties with calcification, especially tibial, time consuming
MRA	Safe, no radiation, able to do dynamic studies, edits out calcium, able to be done with anti-coagulated patient	Operator dependent, Some risk of NSF. Limited in patients unable to co-operate, claustrophobic and those with pacemakers, Not reimbursed in Australia
CTA	Safe, available, excellent for aorto-iliac pathology and aneurysms (lumen, wall and peri-arterial information), able to be done with anti-coagulated patient	Some risk of CIN, radiation, operator dependent with significant time periods of post processing time, difficulties with calcium
Catheter angiography	Remains gold standard, ability to intervene	Involves arterial puncture, radiation, risk of CIN, problems with hypertension, anti-coagulated patients and calcified femoral arteries

NSF – “Nephrogenic systemic fibrosis is a new disease whose incidence has peaked and receded over the past decade. It occurs in the presence of significant renal impairment, either acute or chronic (MDRD creatinine clearance of $<30 \text{ mL/min/1.73m}^2$), and is associated with the administration of gadolinium-based contrast (GBC). Since 2006, the incidence of this disease has decreased markedly in patients with renal impairment, mainly owing to protocols that have not administered GBC to patients with creatinine clearances of less than $30 \text{ mL/min/1.73m}^2$, and in some cases with the use of less toxic and lower doses of GBC.”

CIN prevention has focused on consideration of alternate imaging modality, normovolaemia, discontinuation of nephrotoxic agents (such as NSAIDs), contrast dose reduction, use of low-osmolal contrast media or iso-osmolal contrast media, IV fluids and N-acetylcysteine. “Over the past half-decade, clinical trials have compared the efficacy of IV sodium bicarbonate (bicarbonate) with IV sodium chloride (saline). Although several trials showed bicarbonate to be more effective than saline for the prevention of CIAKI, other trials reported no difference between these two IV fluids. Clinical trials investigating the efficacy of N-acetylcysteine also have been inconsistent in their results. Consequently, there remains clinical equipoise regarding the superiority of bicarbonate (compared with saline) and the role of N-acetylcysteine.”

Newer intervention fusing the pre-procedural CT or MR with the angio table looks promising. The ability to do on-table angio-CT (360° spin) allows greater precision with angio-intervention (eg fenestrated stent grafting, neuro-intervention) and body intervention (liver, renal, aortic).

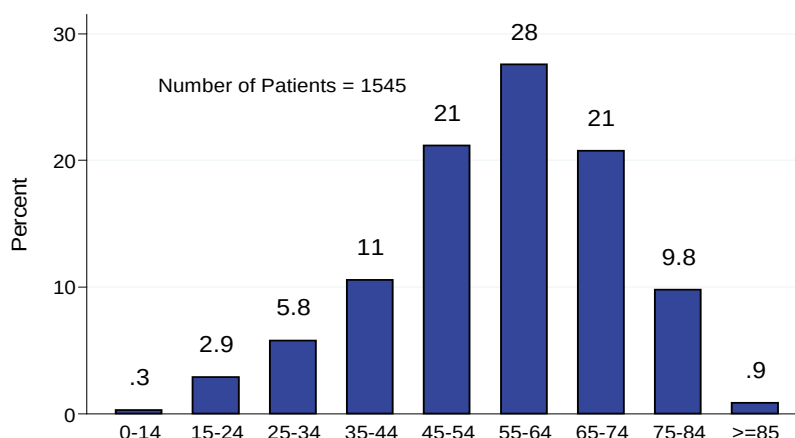
Dialysis Access

In many units this represents up to 20% of work with a steady increase in patients on dialysis. There is a trend to older sicker patients and a specific emphasis on trying to create an autogenous fistula. One of the main drivers of this is the “Fistula First” initiative in the US with further guidelines at National Kidney Foundation (www.kidney.org/professionals/kdoqi/).

Local data is available on the ANZDATA website which has free public access at ANZ data (www.anzdata.org.au/v1/). And the following tables are taken from the ANZDATA 2010 report.

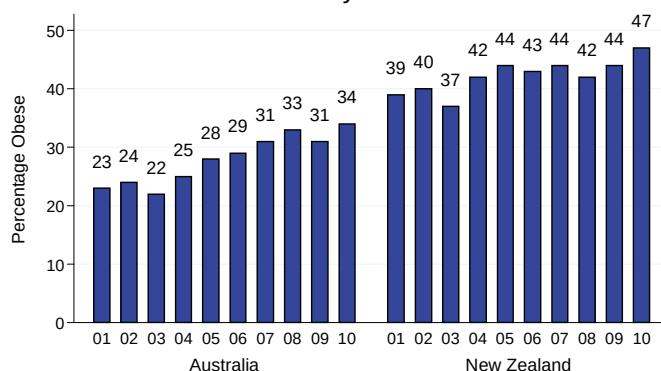


Age (%) of Current Haemodialysis Patients New Zealand 31-Dec-2010

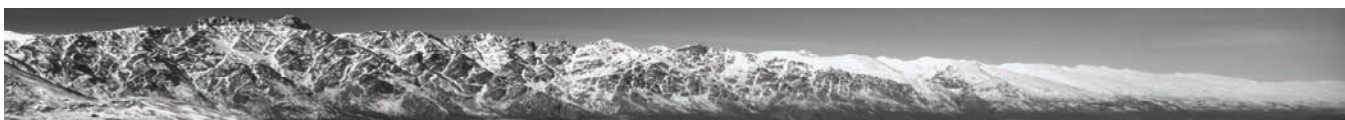


Obesity is an increasing problem with more than 45% of patients in New Zealand starting dialysis obese. Obesity for these analysis is defined as a BMI $> 30\text{kg/m}^2$. Morbid obesity is defined as $\geq 25\text{kg/m}^2$.

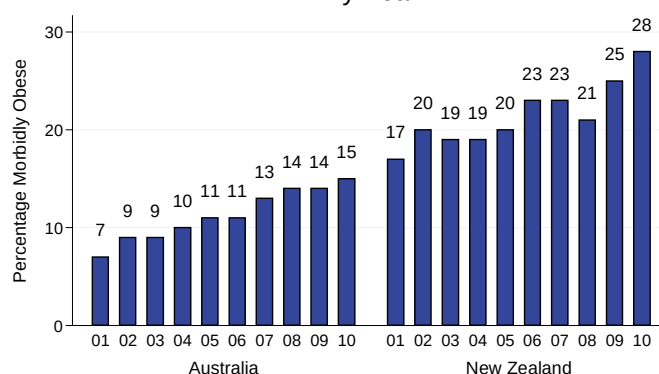
Obesity in Incident Haemodialysis Patients By Year



Obesity in Incident Haemodialysis Patients By State and Country - 2010

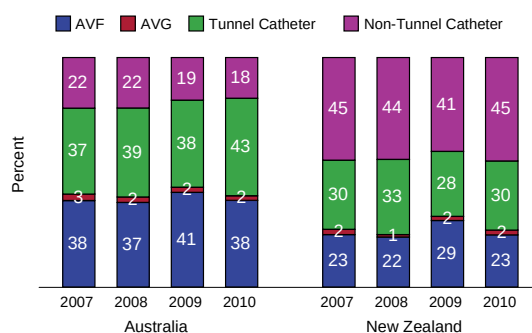


Morbid Obesity in Incident Haemodialysis Patients
By Year



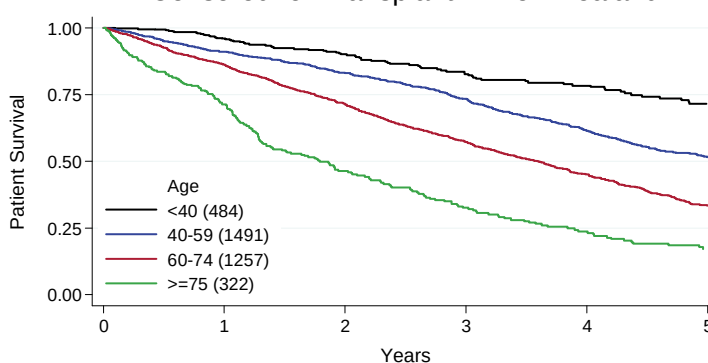
Many patients start dialysis with a central venous line and this confers a significant burden of catheter related sepsis central venous thrombosis and central venous stenosis.

Vascular Access - Initial RRT
Haemodialysis at Initial Modality



Graft / line in older risk can be considered due to poor life expectancy and longer times to achieve a functioning fistula. Currently at Auckland Hospital a minority of patients have a functioning fistula at 6 months after referral.

Patient Survival - Haemodialysis at 90 Days
1999 - 2010
Censored for Transplant - New Zealand



Venous Intervention

Venous intervention has made its way back to public hospitals and is riding the “gadget” wave. Techniques for thermal (RFA or EVLT) and chemical (Ultrasound Guide Foam Sclerotherapy, USGFS and Clarivein) ablation of the saphenous vein. “Currently available clinical trial evidence suggests RFA and EVLT are at least as effective as surgery in the treatment of great saphenous varicose veins. There are insufficient data to comment on USGFS.” These are local anaesthetic day stay procedures. EVLT and RFA utilise “tumescence” anaesthetic, which



is ultrasound guided peri-venous delivery of relatively large volumes of dilute local anaesthetic. This acts as an analgesic but also as a heat sink.

Hyperhydrosis

Thorascopic sympathectomy remains used for palmar sweating but popularity has waned due to compensatory flushing and hyperhidrosis on the trunk and face.

Audit

Both the Australian and New Zealand Society of Vascular Surgery (ANZSVS) and the Vascular Society of New Zealand have made full practice audit a condition of membership. The Australasian Vascular Audit (AVA) is a case based on-line data entry system fully funded and owned by the ANZSVS. Index procedures are aortic surgery, fistula patency at discharge, fempop patency at discharge and stroke/death rates for carotid intervention.

Aortic Surgery

Start Date: 1-Jan-2010
Consultant: auckland

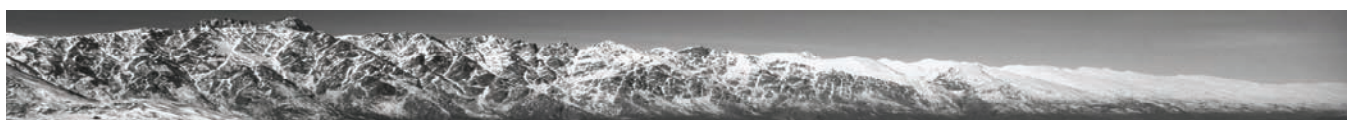
End Date: 31-Dec-2012
Hospital: Auckland City Hospital - Auckland -

	auckland @ Auckland City Hospital - Auckland - Auckland	ANZSVS
Open Elective Cases	58	2008
Survival Rate %	96.55%	96.76%
Open Emergency Cases	80	1283
Survival Rate %	85%	79.73%
EVAR Elective Non-Fen Cases	85	3897
Survival Rate %	100%	99.38%
EVAR Emergency Non-Fen Cases	11	289
Survival Rate %	90.91%	86.51%
EVAR Fenestrated Cases	23	302
Survival Rate %	100%	96.03%

Training

There is a dedicated Vascular Surgery training programme and this has been in place with an exit exam since 1997. Currently there are about 50 trainees over 5 years in Australia and New Zealand. There are specific requirements for surgical procedures, US, endovascular procedures, Research/Publication and sign off each run and pre-exam.

Multi-disciplinary teams have had some growth in complex areas including Trauma, Placenta Accreta and high-risk foot clinics.



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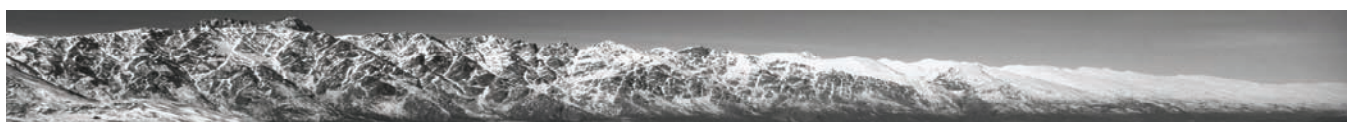
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A CYNIC'S GUIDE TO EVIDENCE BASED MEDICINE

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Introduction

Treating patients is as much an art as it is a science. Even allowing for a large 'art' component, a significant amount of what we do has little or no evidence to support it. Even a common and simple practice such as oxygen administration has been subject to little in the way of appropriate research.

Why Do We Do What We Do?

Much of what we do is either based either on history or theory. The problem with this is that there are multiple examples of clinical practice based on history or theory that turned out to be harmful when subject to an appropriate randomised trial. For example –

- Prophylactic hyperventilation in traumatic brain injury
- Steroids for traumatic brain injury

Why Don't We Have Good Evidence?

At the moment we have an evidence pyramid with a large amount of clinically meaningless evidence at the bottom and a small amount of clinically meaningful evidence at the top. The evidence at the bottom is contributed to by –

- Too many underpowered studies
- Too many single centre studies run by enthusiasts in tertiary centres
- Too many studies with surrogate outcome markers
- Too many studies with results that are statistically significant, but clinically insignificant
- Too many studies sponsored by companies with financial interests in the study
- Pressure to publish as a result of an inappropriate focus on the number of personal publications

What About The Clinically Meaningful Evidence That We Have?

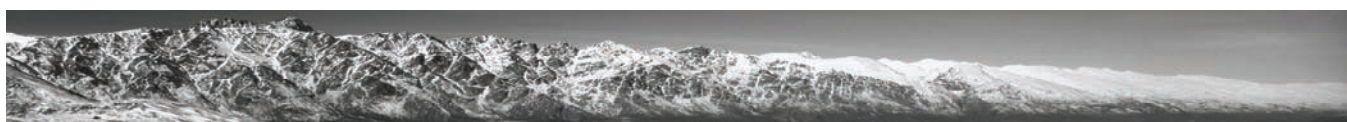
We do have some clinically meaningful evidence, but there are problems here too –

- Some ignore it
- Some take the evidence and apply it to a wider group of patients than those entered in to the study

Meta-analyses

Meta-analyses are fraught with problems including selection bias and statistical 'smoke and mirrors.' Meta-analyses are probably useful at summarising the evidence that we have but they are not useful for guiding clinical decisions. There are multiple examples of the results of meta-analyses being overturned by an appropriately powered randomised trial. Examples include –

- Dopamine for patients at risk of renal failure
- Albumin containing fluid for resuscitation from shock



What Can We Do?

It is time we changed the shape of the evidence pyramid. It is time we –

- Largely abandoned single centre studies
- Abandoned surrogate outcome markers
- Largely abandoned meta-analyses
- Focused on a collaborative approach to multicentre, randomised trials

The Future

Our workload is going to grow at 4-6% per year and our funding is going to grow at 1-2% a year. Part of the future is performing research on how we can deliver healthcare more efficiently. This is unlikely to come from researching new treatments but researching how to use existing treatments (or combinations of treatments) more effectively.



EMERGING ISSUES IN ORTHOPAEDIC ANAESTHESIA

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This talk aims to highlight some emerging concepts and research in orthopaedic anaesthesia that will have an impact on our execution of patient care.

Enhanced Recovery After Orthopaedic Arthroplasty Surgery

A recent audit of primary total knee arthroplasty carried out at Auckland City Hospital demonstrated an average length of inpatient hospital stay of seven days, with an average time to mobilisation (walking 10 metres) of three days. This was consistent with data from the United Kingdom, prior to the institution of an enhanced recovery for surgery program in primary joint arthroplasty which has yielded very encouraging results.¹ At the time of this meeting, a Ministry of Health coordinated nationwide program for ERAS is being developed for New Zealand.²

The precise patient care model will vary between institutions – as several key influential parameters such as patient demographics, levels of social support, the existence of a pain service etc will be inconsistent between institutions. Notwithstanding this, momentum is building towards developing a multi-disciplinary infrastructure where initial mobilisation of hip arthroplasty patients can be achieved on day zero, with knee arthroplasty patients on day one and hospital discharge on day three.

A perioperative care pathway is likely to involve all or some of –

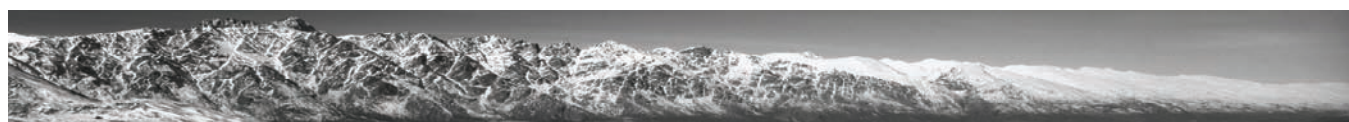
- Optimisation of pre-op haematocrit
- Managing expectations by formalised pre-op education
- Pre op carbohydrate drink³
- Gabapentin to commence preoperatively and to continue into the post op period
- Low to moderate dose spinal anaesthesia, with adjuvant general anaesthesia or sedation as required
- Avoidance of intrathecal morphine⁴
- Routine use of tranexamic acid
- Avoidance of drains, and urinary catheters where possible
- High Volume Local Anaesthetic Infiltration (HLVAI) by surgeon, with or without an indwelling joint infusion catheter for post-op management
- Regular simple analgesia with oxycodone for breakthrough
- Prophylactic antiemetics

Conventional femoral nerve blockade (with or without an indwelling catheter) is avoided in most published protocols as the associated residual motor blockade is frequently sufficient to preclude mobilisation even on day 1. However a 2013 study has demonstrated that the addition of adductor canal nerve block was associated with even further improvements in early ambulation.⁵ Routine insertion of lumbar epidurals will not have a role in these pathways.

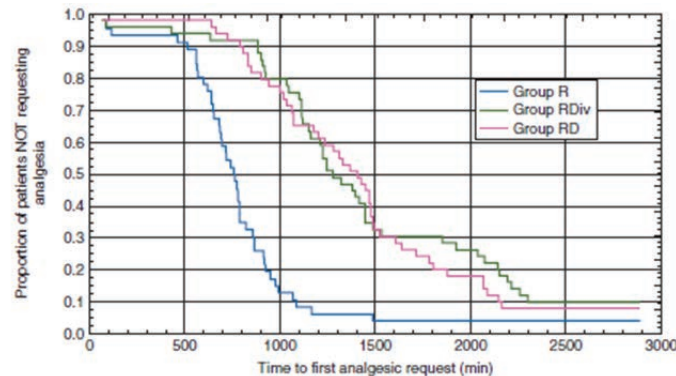
The ideal technique providing minimal analgesia with excellent side effects has yet to be established. However newer techniques including HVLAI are demonstrating equivalence in patient satisfaction and encouraging earlier mobilisation with fewer side effects than currently used regimens.

Adjuvants to Local Anaesthetics

A double blind randomized trial by Cummings et al in 2011 demonstrated an enhanced duration of analgesia by combining dexamethasone with both ropivacaine and bupivacaine for elective shoulder surgery,⁶ compared with either drug administered alone. This paper has led to some enthusiasm in combining dexamethasone with local



anaesthetic solutions in regional blockade. However a more recent investigation in 2013 by Desmet et al⁷ compared three different patient groups having arthroscopic shoulder surgery – interscalene block (ISB) with ropivacaine 0.5% only; ISB with ropivacaine and 10mg perineural dexamethasone; and ISB with ropivacaine alone but with intravenous dexamethasone (10mg) administered. These authors found nearly a twofold increase in time to first postop supplemental analgesic requirement in both the dexamethasone groups compared to the non-dexamethasone group.



Kaplan-Meier survival plot, reproduced from BJA April 2013.⁷

Therefore this study demonstrated dexamethasone as an effective analgesic adjuvant. However this effect was independent of whether it was deposited perineurally or intravenously. The authors concluded that because perineural dexamethasone remains unlicensed, intravenous dexamethasone should be considered as an alternative to perineural dexamethasone when prolongation of post op analgesia is sought.⁷ Whether the main mechanism of action of glucocorticoids is a systemic anti-inflammatory effect or a local effect on peripheral nerves remains unknown.

A 2013 meta-analysis combined nine RCTs comparing the effect of dexmedetomidine (3-15mcg) as a local anaesthetic adjuvant. Five trials investigated intrathecal dexmedetomidine and four investigated dexmedetomidine as part of a brachial plexus block. Sensory block was prolonged by 150min with intrathecal dexmedetomidine, but did not reach statistical significance in brachial plexus block. The authors concluded that there is currently insufficient safety data to support perineural dexmedetomidine in the clinical setting.⁸

Postoperative Visual Impairment in Spinal Surgery

This is obviously a catastrophic complication of medical care. The incidence is quoted as 0.2% for spinal surgery and is probably increasing. There is fortunately an improving awareness of its mechanisms and therefore practices which will reduce the likelihood of it occurring. The optic nerve is a watershed zone which has impeded perfusion in the prone position, further complicated by periorbital oedema with impaired venous drainage which occurs in the prone position. A recent review by Zimmerer et al⁹ defined the at risk patient as “patients who suffer from a pre-existing cardiovascular disease and additional metabolic diseases, where a prolonged duration of surgery in prone position (eg > 2h) or an increased blood loss is expected.” Further, a 2012 multi-centre case-control study¹⁰ identified obesity, male sex, Wilson frame use, duration of surgery, greater estimated blood loss and decreased percentage colloid administration as independent risk factors for ischaemic optic neuropathy.

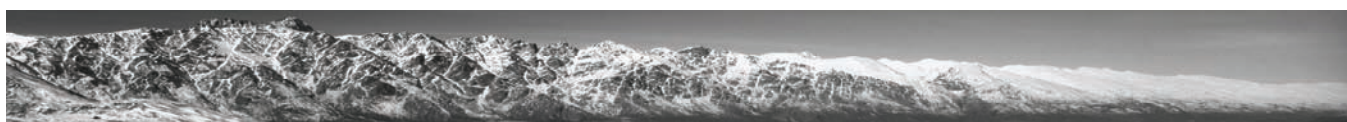
Recommendations according to the literature –⁹

- Avoid direct pressure on the globes
- Avoid perioperative hypotension
- Avoid perioperative anaemia
- Consider 10 degrees of reverse Trendelenburg during prone surgery
- Lower transfusion threshold to keep haematocrit above 30% in at-risk patients
- Avoid infusions of large amounts of crystalloid
- Consider staging long spinal surgeries (above 8h)
- Maintain mean arterial pressure at patient's baseline
- Avoid changes in any perfusion-related medication shortly before surgery
- Perform a postoperative visual exam as early as possible in at-risk patients



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LESS BLOOD IS MORE

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There have been a significant number of papers published in the last two years questioning the benefits of liberal transfusion of red cells and coagulation products, but also indicating the place for a more systematic approach to their use. This talk will cover some of the more significant ones.

Massive Haemorrhage

Despite widespread adoption of Massive Transfusion Protocols, there has been no randomised controlled trial proving their benefit. More importantly the “mixture” within them has not been validated. Holcomb et al reported a prospective study in 10 major trauma centres in the USA. They recruited patients who received greater than three units of red cells with major trauma and showed a 3-4 times reduction in 24 hour mortality. Further subgroup analysis confirmed the validity of a score to predict the benefit of initiating an MTP, using HR > 120/min, systolic BP < 90mmHg, positive FAST, BE < -6 and INR > 1.5. While the survivorship of initiating an MTP with a ratio of plasma to red cells greater than 1:2 is confirmed, the exact constituents of the mixture are still controversial. European trauma units, without access to FFP or cryoprecipitate have developed alternate MTPs using fibrinogen and prothrombin complex concentrates (Prothrombinex). They suggest better maintenance of fibrinogen, less bleeding, less need for platelets and improved survival compared to “conventional FFP based” MTPs. Generally the critical aspect seems to be the ability to maintain a fibrinogen above 2g/L through bleeding. For Australasian anaesthetists this means using more cryoprecipitate. Updated MTPs reflect this.

Following on from the CRASH-2 study, tranexamic acid use has increased in critical bleeding. Subgroup analysis of the trial indicates that the benefits of a 1g bolus are lost if the drug is given more than three hours after injury. There is a drive to give it early in trauma bleeding, and a study is commencing for its use in prehospital care. A parallel study is currently recruiting in obstetric bleeding.

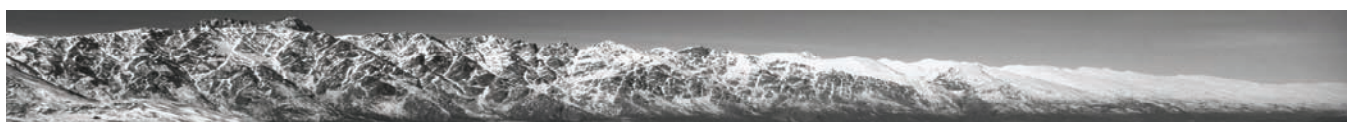
A study by Morrison in combat casualties in Afghanistan used tranexamic acid with a massive protocol guided by ROTEM. It confirmed a greater likelihood of a patient being non-coagulopathic at the conclusion of surgery, but a slightly increased risk of thrombotic complications. This counters the negative finding of risk in the CRASH-2 study. It is important to consider this, and introduce postop DVT prophylaxis in trauma patients after bleeding has ceased. Overall however, it showed a reduced mortality in massive haemorrhage by using TXA.

Limiting Red Cell Use

Many Western countries have seen a reduction in red cell use over the last five years. New Zealand and Australia are experiencing a sharp decline. In New Zealand a 9.2% reduction of sales occurred from NZBS since 2010, and the reduction shows no signs of stopping.

It is uncertain what the cause of this is, but effective blood management programs, a move to less open and more minimally invasive surgical procedures, more rational management of bleeding, and the use of medicines instead of red cells are certainly a major factor in the reduced demand.

Patients bleeding from gastrointestinal haemorrhage were studied by Callcut who showed a restrictive policy of transfusion improved outcome in patients, with a mortality of 9% at 45 days if transfusion was commenced at a Hb < 90g/L vs a mortality of 5% if the Hb was allowed to drop to 70g/L before transfusion. It follows recent analyses of the NQIP surgical database indicating Hb should be supported with a haematocrit of 0.28 if bleeding more than 1,500ml in the case, but 0.24 if there is less than 500ml blood loss. In most cases less transfusion is more.



Exceptions to this rule seem to be in Acute Coronary Syndrome. A recent small series by Carsons indicated increased mortality in patients with an acute MI having a Hb below 100g/L. This was underpowered to be definitive, but indicates caution in allowing severe anaemia to persist in a patient with chest pain. Similar trends are present in severe sepsis and acute stroke. These however are the exceptions to the general rule that a Hb > 70g/L in non-bleeding patients should not need red cell transfusion.

On-going controversy persists with the effect of older blood on outcome. Currently a move to 14 day old blood being disposed of would mean too little red cells being available for patient care. However, if the restrictive transfusion policies continue, 20-25 day expiry may be possible. Currently an ANZICS trial is looking at the effect of the age of blood.

Management of Anaemia

A new and exciting area of interest is anaemia. Preoperative anaemia increases perioperative mortality and complication rates. One third of patients present with anaemia if using the WHO criteria (Hb < 130g/L if male, and 120g/L if female). Half of that population has iron deficiency (ferritin < 40g/L) and respond to oral or IV iron. Evidence suggests, with treatment, 80% will raise their Hb by 20g/L in 8 weeks if taking oral iron, or 22 days if given a single dose of IV iron.

If this algorithm is applied to a population that has a perioperative blood loss of 1,000mls or greater this reduces the likelihood of red cell transfusion by 40% (from 52% to 12% in TKJR). This is a logical response to dealing with patients with preoperative anaemia. There may be an increased risk of infection with iron given intraoperatively but this is controversial.

To work effectively preoperative anaemia must be diagnosed and investigated at least one month before surgery is planned. This creates difficulties with most patients only having it noted immediately preoperatively or often never at all. New systems are being developed to assist anaesthetists to manage patients presenting for blood loss surgery to reduce the likelihood of transfusion.

Currently in NZ, iron polymaltose is the only preparation available for total dose, one time IV infusion. It traditionally has been given over a three hour infusion. Faster rates seem safe. Iron carboxymaltose can be given as a single bolus, and may be available on the PML if costs can be justified.

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AN ANTARCTIC ADVENTURE – HOW LOW CAN YOU GO?

Clinical A/Prof Marcus Skinner

Clinical Director, Department of Anaesthesia and Perioperative Medicine,
Royal Hobart Hospital, Tasmania

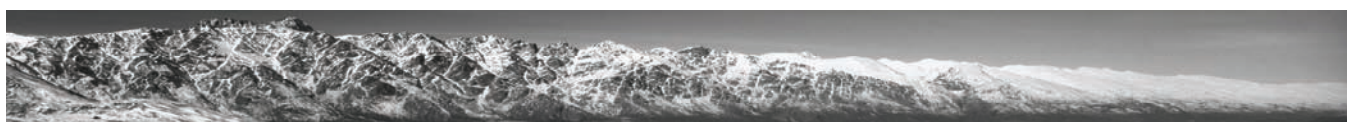
“Below the 40th latitude there is no law; below the 50th no god; below the 60th no common sense and below the 70th no intelligence whatsoever.”
Kim Stanley Baker

This talk gives a documented account and outlines the logistical and operational requirements to successfully complete an aero-medical evacuation of a “critically ill patient” from Pegasus Ice Runway at the McMurdo Base Antarctica (Latitude 77.51S) in August 2012.

The Australian Antarctic Division (AAD) conducts and supports collaborative research programs with other Australian and International organisations in Australian Antarctic and Sub-Antarctic territories.

The AAD headquarters is based in Kingston, Tasmania, just south of Hobart. The division’s headquarters houses laboratories for science, electronics and equipment stores, communications and other operational and support facilities and the AAD maintains three permanently manned stations on the Antarctic continent, and one on Macquarie Island in the sub-Antarctic –

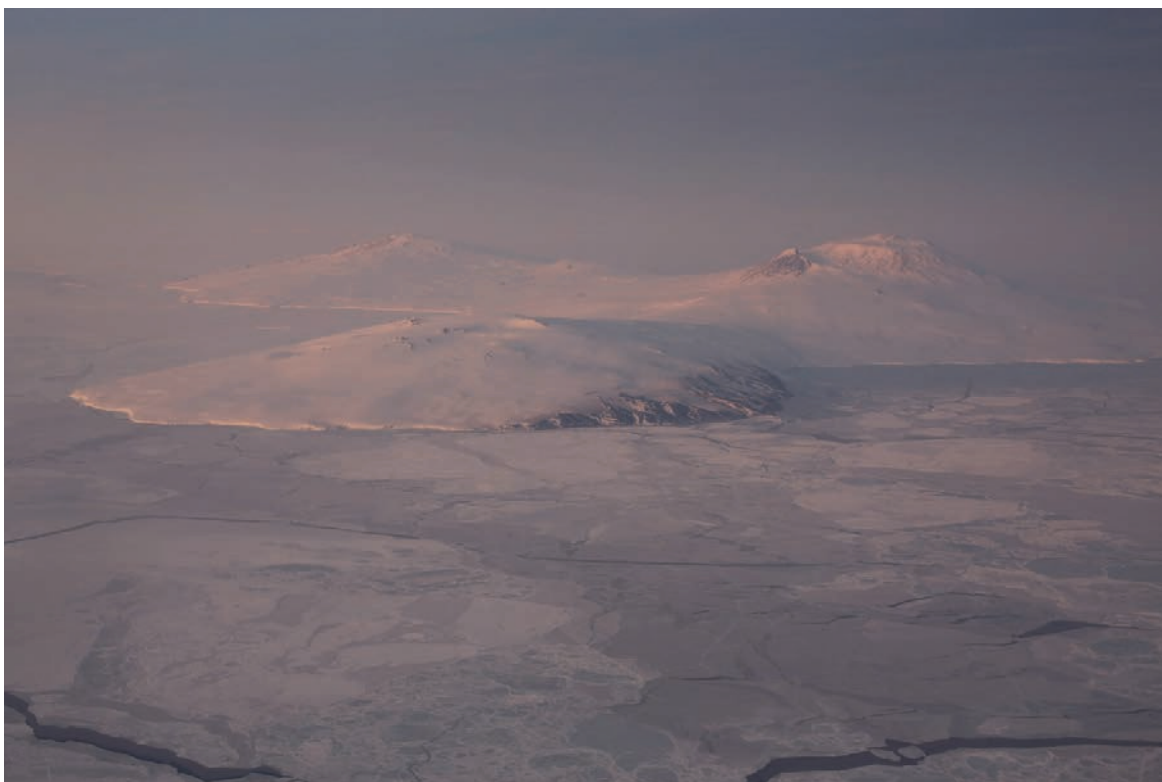
- Casey Station (including the seasonal camp at Wilkins Runway)
- Davis Station
- Mawson Station
- Macquarie Island Station



The AAD utilises an air transport system, both for transport to and from Antarctica, and for transport within the continent. Services to and from Antarctica are provided normally between November and February each year, by an Airbus A319 long range aircraft. This aircraft normally operates to and from the Wilkins ice runway, situated some 65 kilometres (40 mi) from Casey Station.

The Royal Hobart Hospital has had a long term clinical and educational supportive role with the Australian Antarctic Division Polar Medicine Unit located in Hobart. Our Anaesthesia and Peri-operative Service has for many years provided a “Lay-Surgical” in theatre education role for expeditioners and placement for up-skilling of the AAD Doctors.

An urgent request was received to undertake an aero-medical retrieval. Staff undertaking this remote location retrieval required a supportive set of skills in addition to clinical specialist skills in Anaesthesia and Intensive Care required to accomplish this mission.



Elements of the mission that will be presented and discussed include –

- Operational activation, mission directive and time critical constraints
- Logistical and clinical needs
- Cold weather medical and aviation operational limitations
- Principles of Aeromedical Retrieval from an isolated, remote “hostile” environment
- “What if” contingencies that were considered
- “Real Risk” issues / operational duration
- Particular issues of landing on ice runways
- A brief look at anaesthesia implications in such an environment



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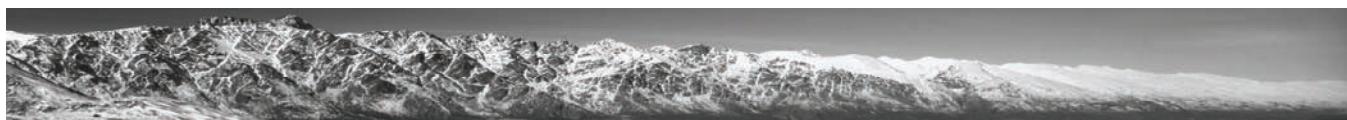
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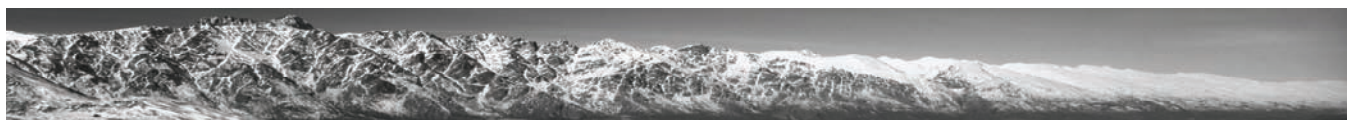
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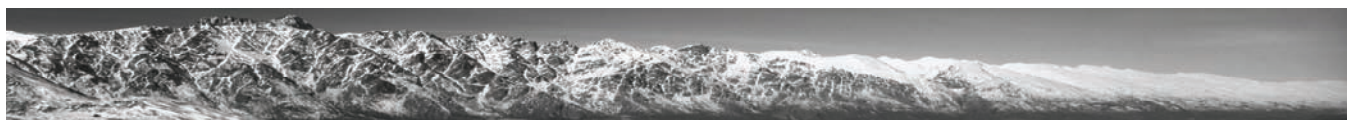


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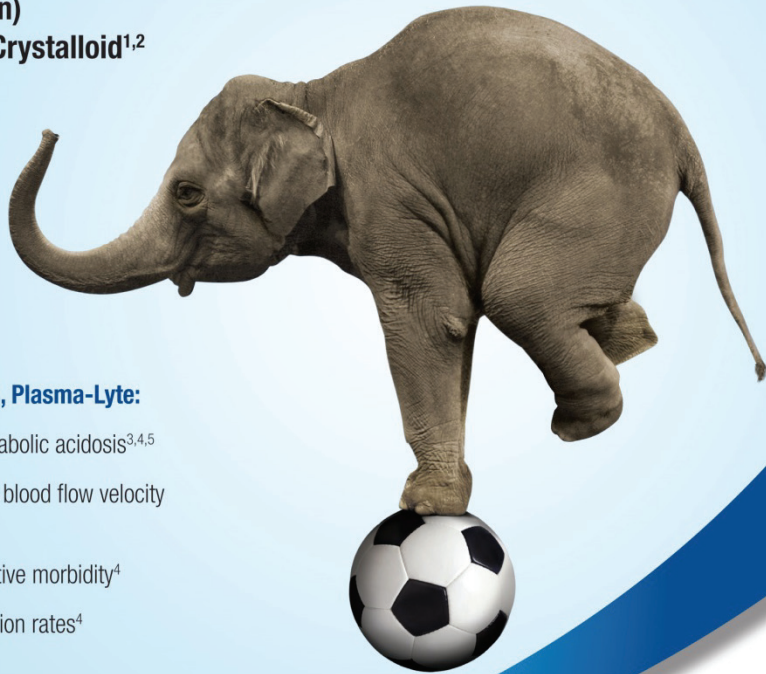
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Name of the Medicine Plasma-Lyte 148 Replacement IV Infusion (Multiple Electrolyte Injection). **Indications** Plasma-Lyte 148 Replacement IV Infusion is indicated as a source of water and electrolytes or as an alkalinising agent. **Contraindications** None known. **Precautions** Plasma-Lyte 148 Replacement IV Infusion should be used with great care, if at all, in patients with congestive heart failure, severe renal insufficiency, and in clinical states in which there exists oedema with sodium retention. Plasma-Lyte 148 Replacement IV Infusion should be used with caution if at all, in patients with hyperkalemia, severe renal failure, and in conditions where potassium retention is present. **Interactions with other Medicines** Caution must be exercised in the administration of Plasma-Lyte 148 Replacement IV Infusion to patients receiving corticosteroids or corticotropin. **Drug/Laboratory Test Interactions** There have been reports of positive test results using the Bio Rad Laboratories Platella Aspergillus EIA test in patients receiving Baxter gluconate containing Plasma-Lyte solutions. These patients were subsequently found to be free of Aspergillus infection. Therefore, positive test results for this test in patients receiving Baxter gluconate containing Plasma-Lyte solutions should be interpreted cautiously by other diagnostic methods. **Adverse Effects** Reactions that may occur because of the solution or the technique of administration include febrile response or infection at the site of infusion. Other reactions that may occur include: Circulatory effects, Extravasation, Hypervolemia, Venous thrombosis, Phlebitis extending from the site of injection. If an adverse reaction does occur, discontinue the infusion, evaluate the patient, institute appropriate therapeutic countermeasures, and save the remainder of the fluid for examination if deemed necessary. **Dosage** As directed by the physician. **Warning:** Do not use plastic containers in series connections. Such use could result in air embolism due to residual air being drawn from the primary container before administration of the fluid from the secondary container is complete. Do not administer unless solution is clear and seal is intact. **Preparation for Administration** Suspend container from eyelet support. Remove plastic protector from outlet port at bottom of container. Attach administration set. Refer to complete directions accompanying set. **Date of Approval** Approved by the TGA: 01/12/2005. Date of the most recent Amendment: 28 December 2007. Vialflex and Plasma-Lyte are trademarks of Baxter International Inc.

PBS Information: PLASMA-LYTE 148 REPLACEMENT IV INFUSION is listed on the PBS as a IV infusion for electrolyte replacement.

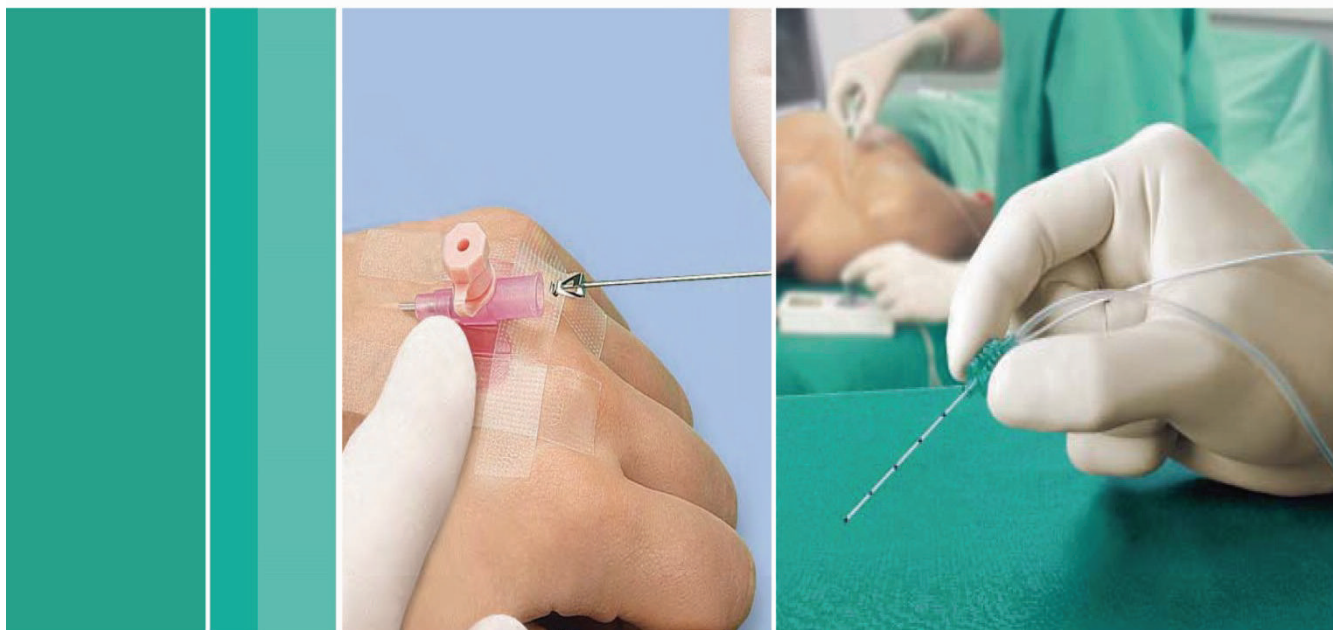
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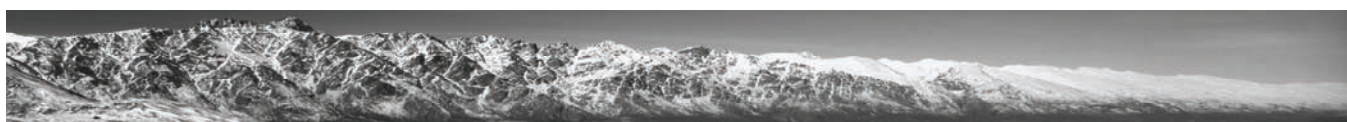
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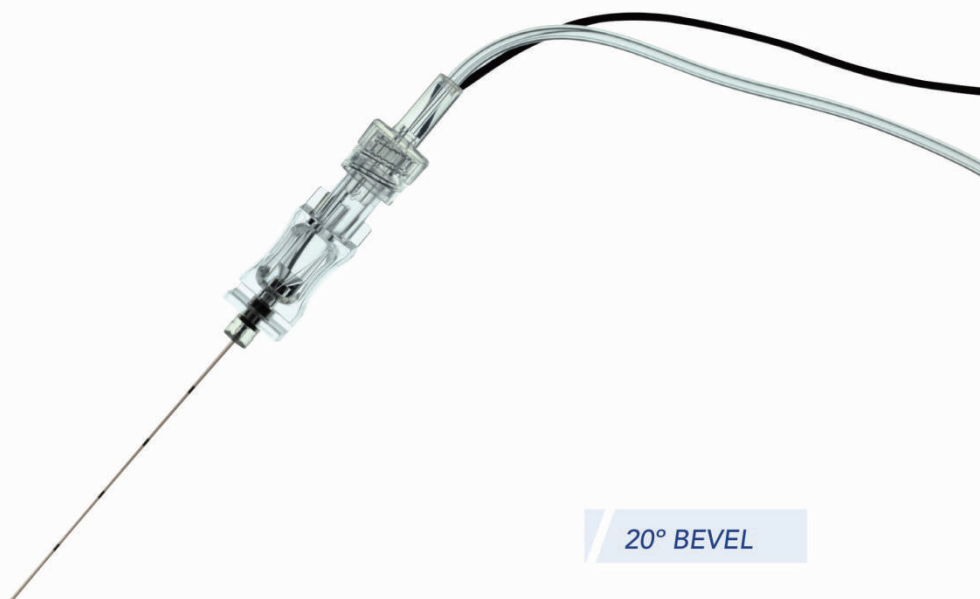


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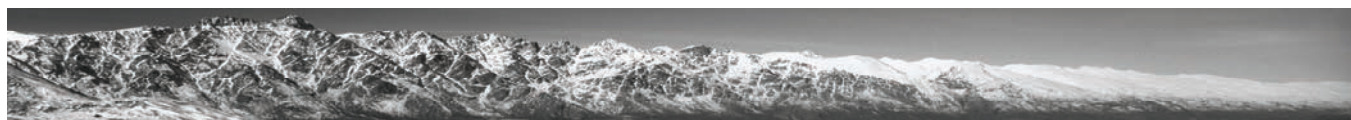
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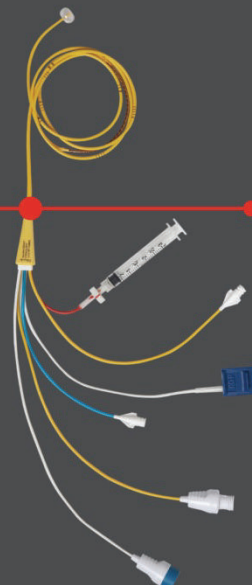
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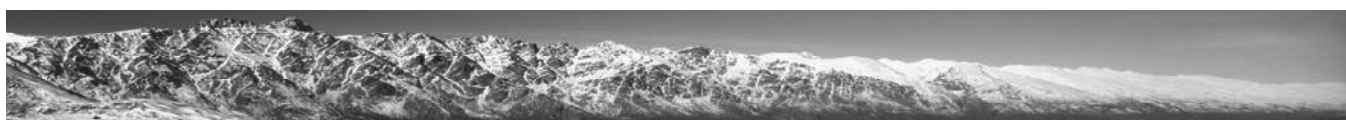
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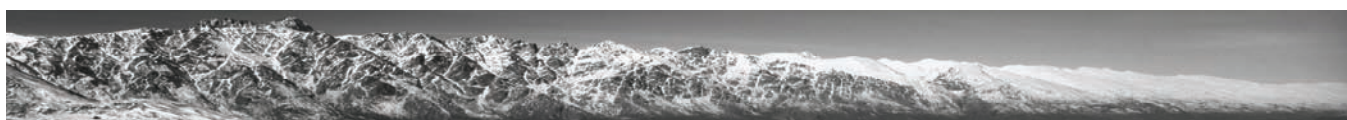
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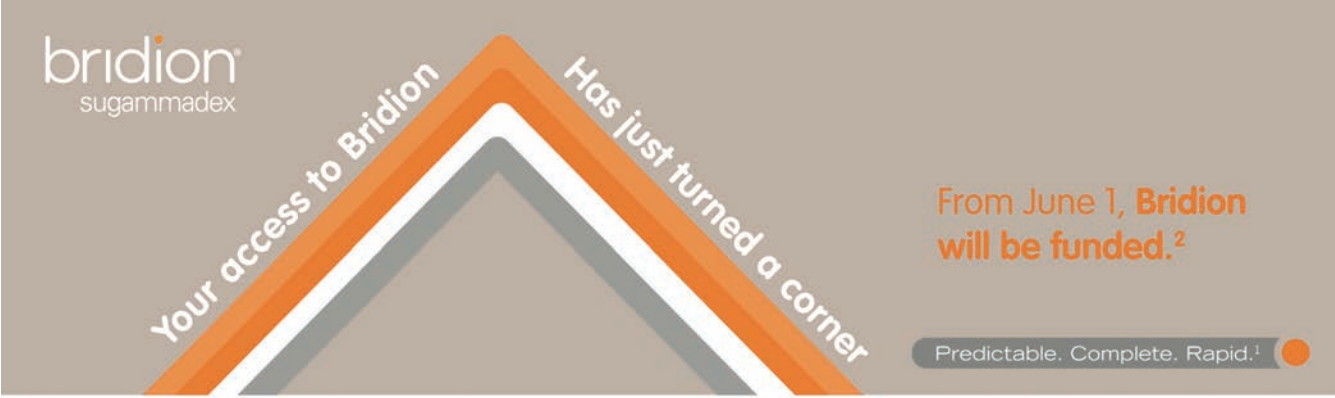
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From June 1, Bridion will be funded.²

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References: 1. BRIDION NZ Data Sheet. 2. Pharmaceutical Schedule, www.pharmac.govt.nz/Schedule. **Bridion® (Suggamadex) 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 suggamadex 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; 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, fusidic acid, flucloxacillin, 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). Severe hypersensitivity reactions can be fatal. Events associated with surgical procedures under general anaesthesia. **Marketed by:** Merck Sharp & Dohme (NZ) Ltd., Newmarket, Auckland. Based on Medsafe-approved Data Sheet, prepared January 2013, available on www.medsafe.govt.nz. © BRIDION is a registered trademark. ANES-1083263-0000. TAPS DA1613MW

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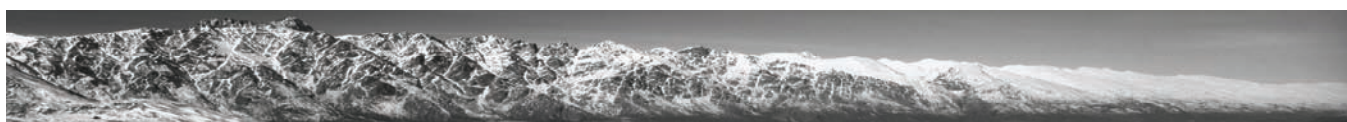
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