AQUA
ANNUAL QUEENSTOWN
UPDATE IN ANAESTHESIA

AQUA 2015

Annual Queenstown Update in Anaesthesia

Programme and Abstracts

www.aqua.ac.nz

Table of Contents

AQUA
ANNUAL QUEENSTOWN UPDATE IN ANAESTHESIA

JAFA
Joint Anaesthesia Faculty Auckland

NZSA
SALUS PER SCIENTIAM

Welcome to Queenstown

Dear Colleague,

Welcome to AQUA 2015, the Annual Queenstown Update in Anaesthesia.

This year we follow the structure we have in the past but at a new venue. I trust you enjoy the change to the Heritage Hotel. We have been lucky to encourage a particularly strong and quality group of speakers this year which we know you will enjoy.

Professor Kate Leslie will be well known to you, but will address the topic of depth of anaesthesia and the relation to outcome. In addition, she has recently been working on health equity and costs. Kate has some interesting data of comparative costs of healthcare between our two countries. To continue our guests from Melbourne we have Roman Kluger from St Vincent's Hospital, to discuss the cardiac risk profile of patients, and how to manage this in non-cardiac surgery. He will also discuss his first love, echo, for the non-cardiac anaesthetist. To round off we have Michael Barrington continuing his review of regional anaesthesia from the Bats on Ice meeting.

We are lucky also to have a strong local faculty, both anaesthetists, and other medical colleagues to discuss progress in areas of interest to you. We are particularly indebted to them for taking time out from their busy schedules.

Despite the attempts of the All Blacks to reschedule their Bledisloe Cup away from AQUA, we still have a great dinner at Prime, with guest speaker Steve Gurney, and the famous AQUA BBQ night at Coronet Peak. As in past years, it's a great time to think about what you have learnt during the day over a quiet wine, while dodging your colleagues attempting to show you how graceful they are on skis.

This is the 7th Annual Queenstown Update in Anaesthesia meeting that we have presented in this framework, and we are grateful to you for supporting the meeting. As always we are keen to keep it fresh and in-keeping with what you need as a good educational meeting, in a fun place to meet and learn.

We hope you have a great meeting and enjoy the beautiful area of Queenstown in all its winter splendour.

Kerry Gunn
AQUA Co-convenor

Neil MacLennan
AQUA Co-convenor

Social Programme

THURSDAY, 20 AUGUST 2015

1700 – 1900

Registration & Welcome Function
Pounamu Room, Heritage Hotel, Queenstown

FRIDAY, 21 AUGUST 2015

1800 onwards

Conference Dinner
Prime Waterfront Restaurant, Queenstown

SATURDAY, 22 AUGUST 2015

1800 onwards

AQUA BBQ Function
Coronet Peak, Queenstown

International Faculty

Prof. Kate Leslie

Head of Research, Royal Melbourne Hospital

Professor Leslie is Head of Research at Royal Melbourne Hospital's Department of Anaesthesia and Pain Management, where she is also staff anaesthetist, has published more than 140 research papers and made more than 170 research presentations.

Dr Roman Kluger

Specialist Anaesthetist, St Vincent's Hospital, Melbourne

Dr Kluger is a senior staff anaesthetist at St. Vincent's Hospital in Melbourne, Victoria. He is a member of the Steering Committee of the Postgraduate Diploma in Perioperative and Critical Care Echocardiography, University of Melbourne and on the executive of the CVP (Cardiovascular and Perfusion) Special Interest Group of the Society and College of Anaesthetists. He has special interests in cardiac anaesthesia, transoesophageal echo-cardiography, perfusion, myocardial ischaemia, perioperative beta-blockade and use of antifibrinolytic agents. He has a number of publications and has lectured widely on perioperative TOE in Australia.

Dr Michael Barrington

Specialist Anaesthetist, St Vincent's Hospital, Melbourne

Dr Barrington is a specialist anaesthetist from St. Vincent's Hospital in Melbourne. His major interest is regional anaesthesia, and in particular ultrasound use in regional anaesthesia. He is the founder of the important Australian and New Zealand International Registry of Regional Anaesthesia which has generated important information about outcomes for regional anaesthesia. He is the current chair of the Regional Anaesthesia Special Interest Group.

New Zealand Faculty

Dr Conrad Engelbrecht	Specialist Anaesthetist, Waikato Hospital
Prof. Alan Merry	Specialist Anaesthetist, Auckland City Hospital
Dr Laura Young	Consultant Haematologist, Auckland City Hospital
A/Prof. Simon Mitchell	Specialist Anaesthetist, Auckland City Hospital
Dr Colin Marsland	Specialist Anaesthetist, Wellington Hospital
Dr Indu Kapoor	Specialist Anaesthetist, Wellington Hospital
Dr Peter Hicks	Intensivist, Wellington Hospital
Dr Sally Roberts	Microbiologist, Auckland City Hospital
Dr Maurice Lee	Specialist Anaesthetist, North Shore Hospital

Scientific Programme

FRIDAY, 21 AUGUST 2015

Session 1

0800	**Depth of Anaesthesia and Outcomes**	Kate Leslie
0835	**Acute Pain Update**	Conrad Engelbrecht
0900	**Regional Anaesthesia: What matters?**	Michael Barrington
0935	**Anaesthesia, the Health Quality and Safety Commission, and Us**	Alan Merry

Session 2

1030	**Cardiac Risk of Non-Cardiac Surgery**	Roman Kluger
1105	**Anticoagulants for Anaesthetists... 2015 Update**	Laura Young
1130	**Atom bombs, circle circuits, and CO_2: links between diving and anaesthesia**	Simon Mitchell

SATURDAY, 22 AUGUST 2015

Session 3

0800	**Airway Update**	Colin Marsland
0825	**Update in Paediatric Anaesthesia**	Indu Kapoor
0850	**Transthoracic Echocardiography (TTE) for Non-Cardiac Anaesthetists**	Roman Kluger
0920	**Intensive Care Update**	Peter Hicks

Session 4

1015	**Reducing post-operative infections; the role of anaesthetists**	Sally Roberts
1040	**Competing with Australia: How does our health system compare?**	Kate Leslie
1110	**Nepal Earthquake Response**	Maurice Lee
1140	**Closing comments**	

Depth of Anaesthesia and Outcomes

Kate Leslie

Department of Anaesthesia, The Royal Melbourne Hospital

The current practice of general anaesthesia emphasises giving adequate doses of anaesthetic drugs in order to ensure that all patients are unconscious. Additional sedative and analgesic drugs are often then given to prevent or treat haemodynamic instability and in response to changing surgical stimulation. This approach is effective and safe for most of our patients.

However, the dose of anaesthetic that ensures unconsciousness for all patients means those who are sensitive to anaesthetics receive significantly more drug than necessary.[1] Reducing the dose towards the threshold for consciousness becomes a matter of judgement, unless monitors that process the frontal lobe electroencephalograph (EEG) to track anaesthetic depth are used. These monitors make it possible to titrate anaesthetic dose more precisely according to individual patient requirements. Marketing of these monitors, such as the bispectral index monitor (BIS) (Covidien Inc, Colorado, USA), has emphasised their use to ensure sufficient anaesthetic administration to prevent awareness. Their use to reduce unnecessarily deep anaesthesia is more controversial.

An association between relatively deep anaesthesia and increased post-operative mortality has been demonstrated in a number of observational studies.[2-11] The majority of these studies have been *post hoc* analyses of studies undertaken for other purposes, for example determining whether the use of processed EEG monitors reduces the incidence of awareness. All have used the BIS monitor to measure anaesthetic depth and most report statistically significant associations between BIS values <45 and death.

All the randomized trials to date have used the BIS monitor to measure anaesthetic depth. One reported increased mortality in a BIS=50 group than in a BIS >80 group[12] and two reported no difference between BIS = 35 and BIS = 50-55 groups.[13,14]

The ANZCA Clinical Trials Network (http://www.anzca.edu.au/fellows/Research/clinical-trials-network.html) is leading an international randomized controlled trial of deep (BIS = 35) and light (BIS = 50) volatile-based general anaesthesia (the Balanced Anesthesia Study) (Australian New Zealand Clinical Trials Registry No: 12612000632897). In total 6500 patients will be required to explore our hypothesis of a 20% relative risk reduction for 1-year mortality in the BIS = 50 group. The choice of BIS targets for light and deep anaesthesia was based on previous studies where BIS has been blinded, and the manufacturer's recommendations.[15] Anaesthetists will also be asked to maintain MAP between patient-specific limits that they identify before randomization.

What should you do as you read the literature on this subject and wait for definitive evidence? Our recommendation is that you strive for the optimal anaesthetic depth: deep enough to avoid intraoperative responsiveness and postoperative recall but light enough to avoid intraoperative hypotension and postoperative side effects. Titration of anaesthetic depth using a processed EEG monitor, in addition to clinical signs, haemodynamic responses and anaesthetic delivery indices, will allow identification of patients with anaesthetic sensitivity.

References

1. Leslie K, Short T. Low bispectral index values and death: the unresolved causality dilemma. Anesth Analg 2011;113:660-3.
2. Monk T, Saini V, Weldon B, Sigl J. Anesthetic management and one-year mortality after noncardiac surgery. Anesth Analg 2005;100:4-10.
3. Lindholm M, Traff S, Granath F, Greenwald S, Ekbom A, Lennmarken C, Sandin R. Mortality within 2 years after surgery in relation to low intraoperative bispectral index values and preexisting malignant disease. Anesth Analg 2009;108:508-12.
4. Leslie K, Myles P, Forbes A, Chan M. The effect of bispectral index monitoring on long-term survival in the B-Aware trial. Anesth Analg 2010;110:816-22.
5. Kertai M, Palance B, Lin N, Searleman S, Zhang L, Burnside B, Finkel K, Avidan M. Association of perioperative risk factors and cumulative duration of low bispectral index with intermediate-term mortality after cardiac surgery in the B-Unaware Trial. Anesthesiology 2010;112:1116-27.
6. Kertai M, Palance B, Pal N, Burnside B, Zhang L, Sadiq F, KJ F, Avidan M. Bispectral index monitoring, duration of bispectral index below 45, patient risk factors and intermediate-term mortality after noncardiac surgery in the B-Unaware Trial. Anesthesiology 2011;114:545-56.
7. Watson P, Shintani A, Tyson R, Pandharipande P, Pun B, Ely E. Presence of electroencephalogram burst suppression in sedated critically ill patients is associated with increased mortality Crit Care Med 2008;36:3171-7.
8. Lindholm M, Granath F, Eriksson L, Sandin R. Malignant disease within 5 years after surgery in relation to duration of sevoflurane anesthesia and time with bispectral index under 45. Anesth Analg 2011;11:778–83.
9. Lindholm M, Brudin L, Sandin R. Cumulated time with low bispectral index values is not related to the risk of new cancer or death within 5 years after surgery in patients with previous or prevailing malignancy. Anesth Analg 2104;118:782–7.
10. Willingham M, Ben Abdallah A, Gradwohl S, Helsten D, Lin N, Villafranca A, Jacobsohn E, Avidan M, Kaiser H. Association between intraoperative electroencephalographic suppression and postoperative mortality. Br J Anaesth 2014;113:1001–8.
11. Sessler D, Sigl D, Kelley S, Chamoun N, Manberg P, Saager L, Kurz A, Greenwald S. Hospital stay and mortality are increased in patients having a 'triple low' of low blood pressure, low bispectral index and low minimum alveolar concentration of volatile anesthesia. Anesthesiology 2012;116:1195-203.
12. Brown IV C, Azman A, Gottschalk A, Mears S, Sieber F. Sedation depth during spinal anesthesia and survival in elderly patients undergoing hip fracture repair. Anesth Analg 2013;118:977–80.
13. Abdelmalak B, Bonilla A, Mascha E, Maheshwari A, Wilson Tang W, You J, Ramachandran M, Kirkova Y, Clair D, Walsh R, Kurz A, Sessler D. Dexamethasone, light anaesthesia, and tight glucose control (DeLiT) randomized controlled trial. Br J Anaesth 2013;111:209-21.
14. Short T, Leslie K, Campbell D, Chan M, Corcoran T, O'Loughlin E, Myles P. A pilot study for a prospective, randomized, double-blind trial of the influence of anesthetic depth on long term outcome. Anesth Analg 2014;118:981-6.
15. Short T, Leslie K, Chan M, Campbell D, Frampton C, Myles P. Rationale and design of the Balanced Anesthesia Study: a prospective randomized clinical trial of two levels of anesthetic depth on patient outcome after major surgery. Anesth Analg 2015;accepted 21 January 2015.

Acute Pain Update

Conrad Engelbrecht

Department of Anaesthesia, Waikato Hospital

When I was asked to talk about "Acute Pain: An Update", I experienced first hand what psychologists call "the distant elephant phenomenon". This phenomenon refers to situations where something in the distance appears small, non-threatening and quite manageable. However, the closer you move to it (the elephant), the bigger and less manageable it becomes until you are really up close, stare it in the eye and just realise how an intimidating beast it is.

Why is acute pain such an intimidating topic? I believe the reasons are many. Firstly, the definition of pain is very subjective. Secondly, pain may have an emotional component and the pain experience may not be the same for any two patients. Our ability to measure pain is not particularly sophisticated and this is especially true when patients use our "tools" for pain assessment to verbalise anxiety and distress in addition to their pain. Add to this the fact that the literature on acute pain medicine is often conflicting, the studies suffer from significant heterogeneity and even the findings of good studies may not be implementable in your institution for a variety of reasons and "voilà": You have a distant elephant! It is not possible to offer an exhaustive update on the literature concerning acute pain. Instead I wanted to focus on some topics that seem relevant to our daily practise. Our understanding of these topics is evolving and in discussing this, the difficulties in navigating the literature will be highlighted.

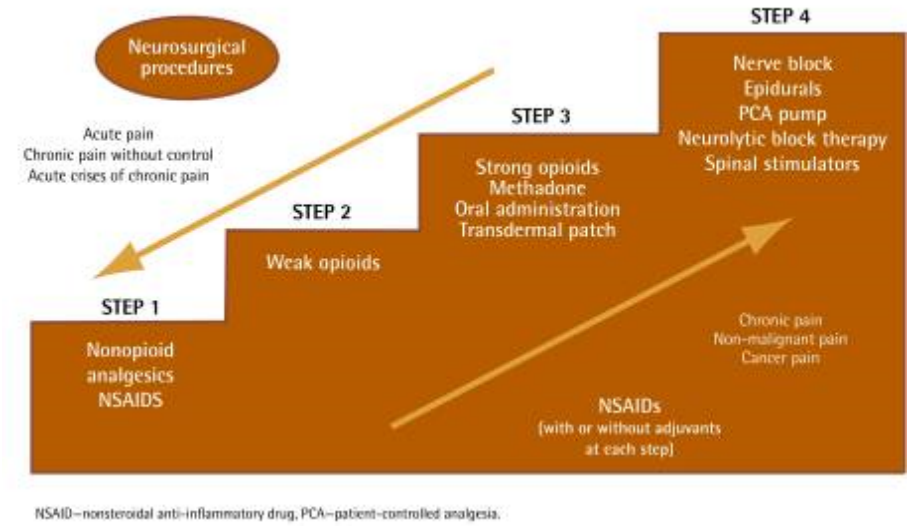
These topics are:

1. Has the basic framework for pain management changed?
2. Cancer and the analgesics we use.
3. Pre-emptive and preventative analgesia and treatment of established pain.
4. Ketamine is good for pain! Right?
5. Methadone. Can you teach an old drug new tricks?
6. Other "Pain stuff".

Has the basic framework for pain management changed?

The World Health Organization (WHO) developed the analgesic ladder as a guide for managing cancer pain in 1986 and revised it in 1997 with the addition of "adjuvants". The key principles were "step-up" analgesia as required or "step-down" analgesia as stronger drugs were no longer needed and it advocated "by the clock" administration of analgesics rather than "on demand" dispensing. The "right drug; at the right dose; at the right time" was inexpensive and 70-80% effective. [1, 2] It was widely adopted across the world with many subsequent modifications that have seen its implementation in both acute and chronic pain. In 2010 one such a modification of the analgesic ladder was published in the Canadian Family Physician. [2]

Figure 2. New adaptation of the analgesic ladder



(Reproduced with permission. Grisell Vargas-Schaffer. Is the WHO analgesic ladder still valid? Twenty-four years of experience. Can Fam Physician. 2010 Jun; 56(6): 514–517)

Combined with the key principles underpinning the original analgesic ladder, it is still highly relevant and a useful frame for the management of acute pain. As anaesthetists we typically start at "step 4" with a sophisticated pain management modality and then "step-down" as patients' needs changes post-operatively. Similarly we "step-up" analgesia when pain is poorly controlled.

Cancer and the analgesics we use

Opioids

One of our roles as anaesthetists is to facilitate surgery for our patients but our involvement in patients' journeys are, relatively speaking, short lived. Is it possible that our actions and drugs could have an effect on the long term outcomes that were traditionally thought to be primarily determined by the scalpel of a surgeon and the drugs of a physician? This question dates back to as far as 1962 and in the last 10 years questions on the longer term effects of commonly used agents, especially opioids and their potential role in cancer recurrence, have intensified. [3]

Opioids have been shown to affect tumour cell proliferation and tumour apoptosis. Opioids modulate angiogenesis and processes involved in tumour growth and invasion through direct and indirect mechanisms such as alteration of the inflammatory micro-environment. Opioids are immunosuppressive with good evidence pointing to inhibition of Natural Killer (NK) cells in particular. This is important as NK cells are implicated in cancer protection by "mopping up" rogue disseminating cancer cells. [4, 5]

Complicating matters is the fact that not only morphine, but pain can also strongly inhibit NK cells and as a result, poorly controlled pain may influence cancer spread and recurrence. It is interesting that endogenous opioids seem to improve NK cell activity which likely confers protection against cancer cell dissemination. [4]

Retrospective studies have shown reduced cancer recurrence after regional anaesthesia and reduced opioid use in prostate, breast and ovarian adenoma carcinoma but it is not clear whether the benefit arises from reduced opioid use or protection through a mechanism conferred by regional anaesthesia. [6, 7, 8] Interestingly, regional anaesthesia and reduced morphine use does not seem to confer the same protection in colonic cancer [5] or hepatocellular cancer where patients receiving epidural analgesia with morphine had higher cancer recurrence rates and higher mortality than patients receiving intravenous fentanyl. [9]

In vitro studies showed enhanced cancer cell proliferation with low doses of morphine but with high doses or prolonged exposure morphine suppressed cancer cell proliferation and caused apoptosis. [10] Remifentanil and fentanyl did not seem to affect cancer cell growth at any dose and tramadol suppressed the immune system less than morphine and suppression was only temporary. [10, 11]

Nonsteroidal anti-inflamatory drugs (NSAID)

A number of human and animal studies showed cyclooxygenase-2 (COX-2) overexpression was associated with cancer recurrence and was an unfavourable prognostic factor. [12, 13]

Treatment with a NSAID reduced cancer recurrence rates and time to recurrence in obese patients with breast cancer. [14] However; treatment with celecoxib 200mg twice daily for 12 months showed no benefit in bladder tumour recurrence. [15] Muddying the waters further were studies showing that COX-2 expression in proliferative breast cancer resulted in poorer survival outcomes but that COX-2 expression was protective, and considered a favourable prognostic factor, in non-proliferative breast cancer leading to better outcomes. [16]

Ketamine has anti-inflammatory properties through NMDA antagonism, reducing COX-2 and Il-6 expression, with animal studies showing anti-tumour effects in mice. [17] Evidence exists that ketamine reduces opioid consumption; even in opioid tolerant patients [18]. Human studies are still lacking.

There is almost a universal call to perform larger multi-centred studies to examine the relationship between cancer recurrence, opioids and other drugs. I fear that larger studies may prove counterproductive as the evidence suggests finer nuances in the relationship between cancer cells, the immune system and the drugs we use. This may be a case where bigger is not better as smaller, more specific studies could prevent these finer important nuances being lost in larger data sets.

In summary:

- There is a growing body of evidence that suggests that morphine (in particular) may be bad for you if you have cancer.
- COX-2 expression is generally seen as an unfavourable prognostic factor.
- Non-steroidal anti-inflammatories may confer some protection through COX-2 inhibition but evidence suggests that this may be specific to the cancer type or even subtype.
- The type of NSAID used to inhibit COX-2 does not appear to be important.
- There is established literature that pain and surgical stress suppress the immune system and diminish the body's natural defences against cancer.

A suggested practical approach from the available literature may include:

- Control acute pain and the surgical stress response with adequate anaesthesia, multimodal analgesia (including NSAIDS) and regional anaesthesia or a combination of these tailored to the needs and comorbidities of the patient.
- With respect to the use of morphine and opioids in cancer surgery a recent consensus statement from the BJA Workshop on Cancer and Anaesthesia found no conclusive evidence to change clinical practice.
- Keep current with the literature and be prepared to change practice as ongoing investigation and studies shape our understanding.

Pre-emptive and preventative analgesia and the treatment of established pain

Preventative analgesia is defined as an analgesic intervention that has an effect on postoperative pain and/or analgesic consumption that exceeds the expected duration of action of the drug. Pre-emptive analgesia refers to the timing of an intervention being performed pre-incisional. [19]

Paracetamol

The safety profile, ease of administration and cost effectiveness of paracetamol has seen it become a popular premedication that is well established in anaesthetic practice. Does the timing of paracetamol administration matter? In a recent study comparing pre-emptive intravenous (IV) paracetamol with

intra-operative paracetamol in functional endoscopic sinus surgery (FESS), time to first analgesic requirement was longer and rescue analgesic consumption and post-operative VAS pain scores were lower in the pre-emptive group up to 24 hours after surgery. [20]

Gabapentin and Pregabalin

Gabapentin is an interesting drug. It was originally approved in December 1993 by the FDA for use as an adjuvant medication to control partial seizures in adults. Our understanding of its mechanism of action continues to evolve and in addition to an impressive list of "off-label" uses [21] it seems to have established itself in anaesthetic and pain management practice.

A number of studies have been published with respects to gabapentin and pregabalin's use as a pre-emptive or preventative drug and acute pain adjunct. Results were conflicting with two studies showing the efficacy of single doses of 600mg gabapentin and 150mg pregabalin for increasing the duration of postoperative analgesia, reducing the requirement for rescue analgesia, decreasing postoperative pain scores and opioid use [22, 23] but gabapentin 600mg as premedication had no opioid sparing effect compared to placebo for abdominal hysterectomy in another study. [24] Gabapentin 900mg or 1200 mg, but not 600mg, administered either pre- or post-incision, was found to be effective in pain management following lumbar laminectomy [25] and gabapentin and pregabalin was effective in preventing chronic pain at one year following lumbar discectomy. [26]

A recent review (2014) on non-opioid analgesic use for pain prophylaxis and established pain helps to clarify the efficacy of individual drugs in these areas. [27]

Paracetamol (oral and IV) was opioid sparing (up to 20%) when given as pain prophylaxis and oral paracetamol had a number needed to treat (NNT) of 3.6 (95% CI 3.4, 4.0) for "established" pain. [27]

The evidence for NSAIDs was convincing for the treatment of "established" pain with a NNT of 2.5 (2.4, 2.6) and 2.7 (2.3, 3.3) for a single 400mg or 500mg dose of ibuprofen or naproxen respectively. NSAIDs were also opioid sparing leading to reduced opioid related side effects and complications, especially sedation and post operative nausea and vomiting (PONV). [27]

COX-2 selective drugs had comparable NNT to other NSAIDs for the treatment of "established" pain and also had an opioid sparing effect. However, this did not seem to translate to reduced opioid related adverse effects. [27]

A meta-analysis places the NNT for gabapentin (200-500mg) at 11 for the treatment of acute pain. Its opioid sparing effect (13-32mg morphine/24hrs) was consistent and clinically significant, resulting in reduced urinary retention (NNT=7) and vomiting (NNT=6), but not nausea. [27]

Pregabalin was opioid sparing 24 hrs after surgery at all doses <75mg – 300mg but no differences were identified on acute pain outcomes between single and multi-dose regimes. The review concluded that pregabalin compared to placebo improved post-operative analgesia at the expense of sedation and visual disturbance. [41]

The authors concluded that the numbers needed to treat should be interpreted with care, as the definition of NNT suffers from the potential of not including substantial pain relief in major surgery that just does not amount to a 50% reduction on the visual analogue scale (VAS).
In summary:

- It would appear that paracetamol and NSAIDs (including COX-2 selective drugs) are useful in the context of treating acute established pain.
- Both NSAIDs and paracetamol used for "pain prophylaxis" are opioid sparing and reduce opioid related side effects. The opioid sparing effect of COX-2 selective inhibitors does not appear to reduce opioid related side effects.
- The evidence for gabapentin's role in treating acute pain is less established and it would appear that doses less than 600mg are not very effective. Gabapentin is opioid sparing but sedation is potentially problematic.

- Is appears that pregabalin may be a useful adjunct in treating acute post surgical pain, but at the expense of sedation and visual disturbances. There appears to be no difference in acute pain outcomes between single and multi-dose regimens.

Data on combinations of the drugs discussed previously is scarce. [27]

Ketamine is good for pain! Right?

The evidence published in the last six years for the use of ketamine in acute pain and the prevention of chronic pain is conflicting.

There was no difference in early and late postoperative pain and morphine consumption with ketamine administered at doses of 0.25, 0.5, and 1 mg/kg in women undergoing Caesarean delivery under general anaesthesia [28] and diclofenac 100mg suppositories were more effective and had fewer troublesome side effects that 0.15mg/kg ketamine for elective gynaecological laparoscopy. [29] A number of studies have failed to show evidence for the use of ketamine to prevent chronic pain or for its use as an adjunct to acute pain management in the context of thoracotomy surgery. [30, 31, 32]

In contrast, a single dose of 15mg ketamine in combination with hydromorphone provided substantial analgesia for severe acute pain (NRS > 9) in the emergency department [33] and low dose ketamine (0.5mg/kg bolus followed by 0.1mg/kg infusion for 24hrs) significantly reduced post-op pain and opioid consumption in limb fracture surgery. [34] The pre-induction administration of intramuscular ketamine between 25-40mg showed significantly lower 48hr pain scores and approximately 35% less morphine use, again in the context of lower limb fracture surgery. [35]

An important study compared the effect of ketamine (0.3mg/kg bolus plus an infusion of 0.05mg/kg/hr till the end of surgery) versus gabapentin (1200mg pre induction with a bolus and infusion of saline) on acute pain, opioid consumption and the prevention of chronic pain. Gabapentin and ketamine were similar in improving early acute pain and both gabapentin and ketamine reduced morphine consumption (35% and 42% respectively). At six months post surgery gabapentin, but not ketamine, seemed to have reduced chronic pain after hysterectomy. [36]

In hip arthroplasty Ketamine decreased morphine consumption at 24h and at day 30, patients receiving ketamine were more mobile needing less assistance. At day 180 patients receiving Ketamine had clinically and statistically significant less pain (8% vs 21%; p=0.036). [37]
In summary:

- It would appear that Ketamine can be a useful adjunct in controlling acute pain especially in limb fracture surgery.
- Ketamine is opioid sparing.
- The evidence for Ketamine protecting against the development of chronic pain is conflicting.
- It would also seem that in thoracic surgery its use as an adjunct to acute pain management is doubtful.

Methadone. Can you teach an old drug new tricks?

The literature on the use of methadone in acute pain is surprisingly scant. A single dose of methadone (0.2mg/kg) for multilevel complex thoracolumbar surgery reduced postoperative opioid requirement by approximately 50% (25 mg morphine equivalents) at 48 and 72 hours compared to sufentanil (63mg morphine equivalents). In addition, pain scores were lower by approximately 50% in the methadone group at 48 hours after surgery and the incidence of side effects was comparable in both groups. [38]
I use IV methadone regularly when I am concerned about post-operative pain or where long lasting analgesia is required. Conceptually I think of methadone 0.1-0.2mg/kg IV as an easily titratable, "long acting morphine" with a hint of ketamine when used in theatre or recovery. It has complex pharmacology when given orally, with repeated dosing or with long term use and therefore; in the context of acute post surgical pain, I tend to avoid it in the ward in favour of fentanyl or morphine patient controlled analgesia (PCA) or oral sevredol or oxycodone. Anecdotally, I have found methadone useful as a "rescue analgesic" in the recovery room on a number of occasions.

Other "pain stuff"

1. Cebranopadol

Cebranopadol is a first in its class opioid and nociceptin/orphanin FQ receptor agonist (NOP). It is unique in being a single molecule with agonistic properties at MOP and NOP receptors with a 3- to 38-fold lower affinity for KOP in humans and the rat and ~20-fold lower affinity for DOP in humans.

Phase II trials show promise for its use in acute nociceptive and neuropathic pain as well as chronic pain and Phase III trials are underway.

Favourable pharmacology appears to include a time to peak effect of 20min when administered IV and 90min for oral administration with anti-nociceptive effect evident at 7 hours. Tolerance to equi-analgesic doses of cebranopadol developed at day 26 compared to morphine at day 11. [39]

2. Tramadol use in the immediate post-operative period. How good is it really?

A recent systematic review and meta-analysis (2015) suggest that combining tramadol with morphine in the immediate postoperative period appears to be limited to slightly lower levels of morphine use after surgery compared to placebo or non-opioid analgesics. Pain intensity at rest at 24 hours was no better and there was no_detectable benefit on the incidence of opioid-related adverse effects. [40]

3. Opioid-induced hyperalgesia (OIH). Is it real?

In a review published in 2009 it was concluded that there was not sufficient evidence to support or refute the existence of OIH in humans except in the case of normal volunteers. [42] A more recent review and meta-analysis suggested that the clinical impact of remifentanil-induced hyperalgesia in the immediate postoperative period appeared to be limited to a slight increase in pain intensity at rest persisting for 24 h. It also showed a moderate increase in morphine use without influencing opioid-related side-effects. [43]

In summary: It would appear to be a real phenomenon mostly associated with high dose remifentanil. It does not appear to be clinically problematic.

References

1. http://www.who.int/cancer/palliative/painladder
2. Grisell Vargas-Schaffer. Is the WHO analgesic ladder still valid? Twenty-four years of experience. Can Fam Physician. 2010 Jun; 56(6): 514–517
3. Nomura Y; Kawaraguchi Y; Sugimoto H; Furuya H; Kawaguchi M. Effects of morphine and fentanyl on 5-fluorouracil sensitivity in human colon cancer HCT116 cells. Journal Of Anesthesia [J Anesth] 2014 Apr; Vol. 28 (2), pp. 298-301
4. Young MJ; Alston TA. Does anesthetic technique influence cancer? Journal Of Clinical Anesthesia [J Clin Anesth], 2012 Feb; Vol. 24 (1), pp. 1-2
5. Afsharimani B; Cabot P; Parat MO. Morphine and tumor growth and metastasis. Cancer Metastasis Reviews [Cancer Metastasis Rev] 2011 Jun; Vol. 30 (2), pp. 225-38]
6. Biki B, Mascha E, Moriarty DC, Fitzpatrick JM, Sessler DI, Buggy DJ. Anesthetic technique for radical prostatectomy surgery affects cancer recurrence: a retrospective analysis. Anesthesiology 2008;109(2):180–7.
7. Exadaktylos AK, Buggy DJ, Moriarty DC, Mascha E, Sessler DI. Can anesthetic technique for primary breast cancer surgery affect recurrence or metastasis? Anesthesiology. 2006;105(4):660–4.
8. Lin L, Liu C, Tan H, Ouyang H, Zhang Y, Zeng W. Anaesthetic technique may affect prognosis for ovarian serous adenocarcinoma: a retrospective analysis. Br J Anaesth. 2011;106(6):814–22.
9. Cao L; Chang Y; Lin W; Zhou J; Tan H; Yuang Y; Zeng W. Long term survival after resection of hepatocellular carcinoma: a potential risk associated with the choice of post operative analgesia. Anesthesia and Analgesia [Anesth Analg] 2014 Jun; Vol. 118(6):1309-16.

10. Nomura Y; Kawaraguchi Y; Sugimoto H; Furuya H; Kawaguchi M. Effects of morphine and fentanyl on 5-fluorouracil sensitivity in human colon cancer HCT116 cells. Journal Of Anesthesia [J Anesth] 2014 Apr; Vol. 28 (2), pp. 298-301
11. Sacerdote P, Bianchi M, Gaspani L et al. The effects of tramadol and morphine on immune responses and pain after surgery in cancer patients. Anesth Analg 2000; 90: 1411–4
12. Kim, Hee Sung; Moon, Hyeong-Gon; Han, Wonshik; Yom, Cha Kyung; Kim, Woo Ho; Kim, Jun Ho; Noh, Dong-Young. COX2 overexpression is a prognostic marker for Stage III breast cancer. Breast Cancer Research and Treatment. Feb 15, 2012, Vol. 132 (1):51-9
13. Yoshikawa R; Fujiwara Y; Koishi K; Kojima S; Matsumoto T; Yanagi H; Yamamura T; Hashimoto-Tamaoki T; Nishigami T; Tsujimura T. Cyclooxygenase-2 expression after preoperative chemoradiotherapy correlates with more frequent esophageal cancer recurrence.World Journal Of Gastroenterology: WJG [World J Gastroenterol] 2007 Apr 28; Vol. 13 (16), pp. 2283-8
14. Bowers LW; Maximo IX; Brenner AJ; Beeram M; Hursting SD; Price RS; Tekmal RR; Jolly CA; deGraffenried LA. NSAID use reduces breast cancer recurrence in overweight and obese women: role of prostaglandin-aromatase interactions. Cancer Research [Cancer Res] 2014 Aug 15; Vol. 74 (16):4446-57
15. Sabichi AL; Lee JJ; Grossman HB; Liu S; Richmond E; Czerniak BA; De la Cerda J; Eagle C; Viner JL; Palmer JL; Lerner SP. A randomized controlled trial of celecoxib to prevent recurrence of nonmuscle-invasive bladder cancer. Cancer. Prevention Research (Philadelphia, Pa.) [Cancer Prev Res (Phila)] 2011 Oct; Vol. 4 (10):1580-9
16. Park BW; Park S; Park HS; Koo JS; Yang WI; Lee, JS; Hwang, H; Kim, SII; Lee, KS. Cyclooxygenase-2 expression in proliferative Ki-67-positive breast cancers is associated with poor outcomes. Breast Cancer Research and Treatment. June 1, 2012, Vol. 133 Issue 2, p741, 11
17. Snyder GL, Greenberg S. Effect of anaesthetic technique and other peri-operative factors on cancer recurrence. BJA 105 (2):106-115 (2010)
18. Loftus RW; Yeager MP; Clark JA; Brown JR; Abdu WA; Sengupta DK; Beach ML. Intraoperative ketamine reduces perioperative opiate consumption in opiate-dependent patients with chronic back pain undergoing back surgery. Anesthesiology [Anesthesiology] 2010 Sep; Vol. 113 (3):639-46
19. Macintyre PE; Scott DA; Schug SA; Visser EJ; Walker SM. Acute Pain Management Scientific Evidence 2010 3rd Edition. ("Blue Book")
20. Koteswara CM. A Study on Pre-Emptive Analgesic Effect of Intravenous Paracetamol in Functional Endoscopic Sinus Surgeries (FESSs): A Randomized, Double-Blinded Clinical Study. D S, Journal Of Clinical And Diagnostic Research: JCDR [J Clin Diagn Res] 2014 Jan; Vol. 8 (1): 108-11
21. Mack A. Examination of the evidence for off-label use of gabapentin. J Manag Care Pharm 2003 Vol. 9 (6): 559–68.
22. Bafna U; Rajarajeshwaran K; Khandelwal M; Verma AP. A comparison of effect of preemptive use of oral gabapentin and pregabalin for acute post-operative pain after surgery under spinal anesthesia. Journal Of Anaesthesiology, Clinical Pharmacology [J Anaesthesiol Clin Pharmacol] 2014 Jul; Vol. 30 (3): 373-7
23. Pandey CK; Karna ST; Tandon M; Pandey VK; Singh A. Comparative evaluation of prophylactic use of pregabalin, gabapentin and diclofenac sodium for prevention of succinylcholine-induced myalgia: a randomized, double-blinded study. Journal Of Postgraduate Medicine [J Postgrad Med], 2014 Jan-Mar; Vol. 60 (1): 16-20
24. Joseph TT; Krishna HM; Kamath S. Premedication with gabapentin, alprazolam or a placebo for abdominal hysterectomy: Effect on pre-operative anxiety, post-operative pain and morphine consumption. Indian Journal Of Anaesthesia [Indian J Anaesth], 2014 Nov-Dec; Vol. 58 (6): 693-9
25. Khan ZH; Rahimi M; Makarem J; Khan RH. Optimal dose of pre-incision/post-incision gabapentin for pain relief following lumbar laminectomy: a randomized study. Acta Anaesthesiologica Scandinavica [Acta Anaesthesiol Scand], 2011 Mar; Vol.55 (3): 306-12
26. Dolgun H; Turkoglu E; Kertmen H; Gurer B; Yilmaz ER; Comoglu SS; Sekerci Z. Gabapentin versus pregabalin in relieving early post-surgical neuropathic pain in patients after lumbar disc herniation surgery: a prospective clinical trial. Neurological Research [Neurol Res], 2014 Dec; Vol. 36 (12):1080-5

27. Dahl JB; Nielsen RV; Wetterslev J; Nikolajsen L; Hamunen K; Kontinen VK; Hansen MS; Kjer JJ; Mathiesen O. Post-operative analgesic effects of paracetamol, NSAIDs, glucocorticoids, gabapentinoids and their combinations: a topical review. Scandinavian Postoperative Pain Alliance (ScaPAlli), Acta Anaesthesiologica Scandinavica [Acta Anaesthesiol Scand], 2014 Nov; Vol. 58 (10):1165-81
28. Bilgen S; Köner O; Türe H; Menda F; Fiçicioğlu C; Aykaç B. Effect of three different doses of ketamine prior to general anaesthesia on postoperative pain following Caesarean delivery: a prospective randomized study. Minerva Anestesiologica [Minerva Anestesiol], 2012 Apr; Vol. 78 (4), pp. 442-9
29. Vosoughin M; Mohammadi S; Dabbagh A. Intravenous ketamine compared with diclofenac suppository in suppressing acute postoperative pain in women undergoing gynecologic laparoscopy. Journal Of Anesthesia [J Anesth], 2012 Oct; Vol. 26 (5), pp. 732-7
30. Yazigi A; Abou-Zeid H; Srouji T; Madi-Jebara S; Haddad F; Jabbour K. The effect of low-dose intravenous ketamine on continuous intercostal analgesia following thoracotomy. Annals Of Cardiac Anaesthesia [Ann Card Anaesth], 2012 Jan-Mar; Vol. 15 (1):32-8
31. Hu J; Liao Q; Zhang F; Tong J; Ouyang W. Chronic post-thoracotomy pain and perioperative ketamine infusion. Journal Of Pain & Palliative Care Pharmacotherapy [J Pain Palliat Care Pharmacother], 2014 Jun; Vol. 28 (2):117-21
32. Dualé C; Sibaud F; Guastella V; Vallet L; Gimbert YA; Taheri H; Filaire M; Schoeffler P; Dubray C. Perioperative ketamine does not prevent chronic pain after thoracotomy. European Journal Of Pain (London, England) [Eur J Pain], 2009 May; Vol. 13 (5):497-505
33. Ahern TL; Herring AA; Stone MB; Frazee BW. Effective analgesia with low-dose ketamine and reduced dose hydromorphone in ED patients with severe pain.The American Journal Of Emergency Medicine [Am J Emerg Med], 2013 May; Vol. 31 (5):847-51
34. Deng GF; Zheng JP; Wang S; Tian B; Zhang SG. Remifentanil combined with low-dose ketamine for postoperative analgesia of lower limb fracture: a double-blind, controlled study. Chinese Journal Of Traumatology Chinese Medical Association [Chin J Traumatol], 2009 Aug; Vol. 12 (4):223-7
35. Rakhman E; Shmain D; White I; Ekstein MP; Kollender Y; Chazan S; Dadia S; Bickels J; Amar E; Weinbroum AA. Repeated and escalating preoperative subanesthetic doses of ketamine for postoperative pain control in patients undergoing tumor resection: a randomized, placebo-controlled, double-blind trial. Clinical Therapeutics [Clin Ther], 2011 Jul; Vol. 33 (7):863-73
36. Sen H; Sizlan A; Yanarates O; Emirkadi H; Ozkan S; Dagli G; Turan A. A comparison of gabapentin and ketamine in acute and chronic pain after hysterectomy. Anesthesia And Analgesia [Anesth Analg], 2009 Nov; Vol. 109 (5):1645-50
37. Remérand F; Le Tendre C; Baud A; Couvret C; Pourrat X; Favard L; Laffon M; Fusciardi J. The early and delayed analgesic effects of ketamine after total hip arthroplasty: a prospective, randomized, controlled, double-blind study. Anesthesia And Analgesia [Anesth Analg], 2009 Dec; Vol. 109 (6):1963-71
38. Gottschalk A; Durieux ME; Nemergut EC. Intraoperative methadone improves postoperative pain control in patients undergoing complex spine surgery. Anesthesia And Analgesia [Anesth Analg], 2011 Jan; Vol. 112 (1):218-23
39. D. G. Lambert, M. F. Bird and D. J. Rowbotham. Cebranopadol: a first in-class example of a nociceptin/orphanin FQ receptor and opioid receptor agonist. British Journal of Anaesthesia 114 (3):364–6 (2015)
40. V. Martinez, L. Guichard and D. Fletcher. Effect of combining tramadol and morphine in adult surgical patients: a systematic review and meta-analysis of randomized trials. British Journal of Anaesthesia 114 (3): 384–95 (2015)
41. B. M. Mishriky, N. H.Waldron, A. S. Habib. Impact of pregabalin on acute and persistent postoperative pain: a systematic review and meta-analysis. British Journal of Anaesthesia 114 (1): 10–31 (2015)
42. Fishbain DA, Cole B, Lewis JE, Gao J, Rosomoff RS. Do opioids induce hyperalgesia in humans? An evidence-based structured review. Pain Med 2009; 10: 829–39
43. D. Fletcher, V. Martinez. Opioid-induced hyperalgesia in patients after surgery: a systematic review and a meta-analysis. British Journal of Anaesthesia 112 (6): 991–1004 (2014)

Regional Anaesthesia: What matters?

Michael Barrington

Department of Anaesthesia and Acute Pain Medicine, St. Vincent's Hospital

Training and Education

Training in regional anaesthesia is often conducted in an ad-hoc manner related to the availability of both surgical cases appropriate for regional anaesthesia and individuals with appropriate expertise in the field. Many studies on training in regional anaesthesia have focused on a minimum number of procedures required to achieve competency. In many training scenarios, the recommended number of cases is simply not available. An alternative approach is to structure training so that it follows valid educational principles that are relevant to motor skills training in regional anaesthesia.[1]

A. **Creating the pre-trained novice:** Ultrasound-guided nerve block procedures comprise discreet component tasks that when deconstructed provide a basis for teaching, learning and assessment of skills. In the motor skill learning literature, fractionization refers to practising a discreet component of a complex motor skill separately.[2] Acquiring relevant sonograms and identifying anatomical structures is a discreet component skill required for ultrasound-guided regional anaesthesia. A second key component skill is needle-guidance under ultrasound. Both sonography and needling skills can be practiced in a non-clinical environment and then combined with other skills and knowledge to perform a clinical procedure.

   Learning motor skills in an environment remote from the operating room and clinical responsibilities has merit. As a motor skill, sonography skills required for regional anaesthesia are relatively complex requiring a high level of cognitive involvement. During an early stage of their training, trainees should become familiar with task demands and acquire and develop a degree of automaticity in the core psychomotor skills required for specific procedures before performing them on patients. When a trainee then performs the procedure in the clinical environment it then requires less attentional capacity and information processing for the core skills as these would have been developed during their prior part- task training. The trainees' attention could then be more fully committed to higher-order processes and developing key attributes including communication, team-working, planning and decision making. [3]

B. **Deliberate practice:** is required to create the pre-trained novice ready for regional anaesthesia and to further develop practitioners with wide levels of expertise. It is likely that anyone, regardless of expertise will benefit from deliberate practice. The key point is that an expert will be practicing a different task to that practiced by the novice. The validity of deliberate practice has been demonstrated in many fields including music, chess, sport and medicine. The term deliberate practice does not appear commonly in the anaesthetic literature, however in a recent edition of Anesthesia and Analgesia, there is a review article on deliberate practice.[4]

   An example of the benefits of deliberate practice is demonstrated in the following example. Ten University of Melbourne Doctor of Medicine students, all novices to ultrasound, had their sonographic proficiency assessed in a structured teaching environment. Proficiency was measured by the novices ability to acquire and interpret sonograms required for ultrasound-guided axillary brachial plexus block. Figure 1 demonstrates that within 8 – 10 supervised scanning sessions that included feedback; novice participants achieved sonographic proficiency (maximum score possible, 18) at this anatomical site.

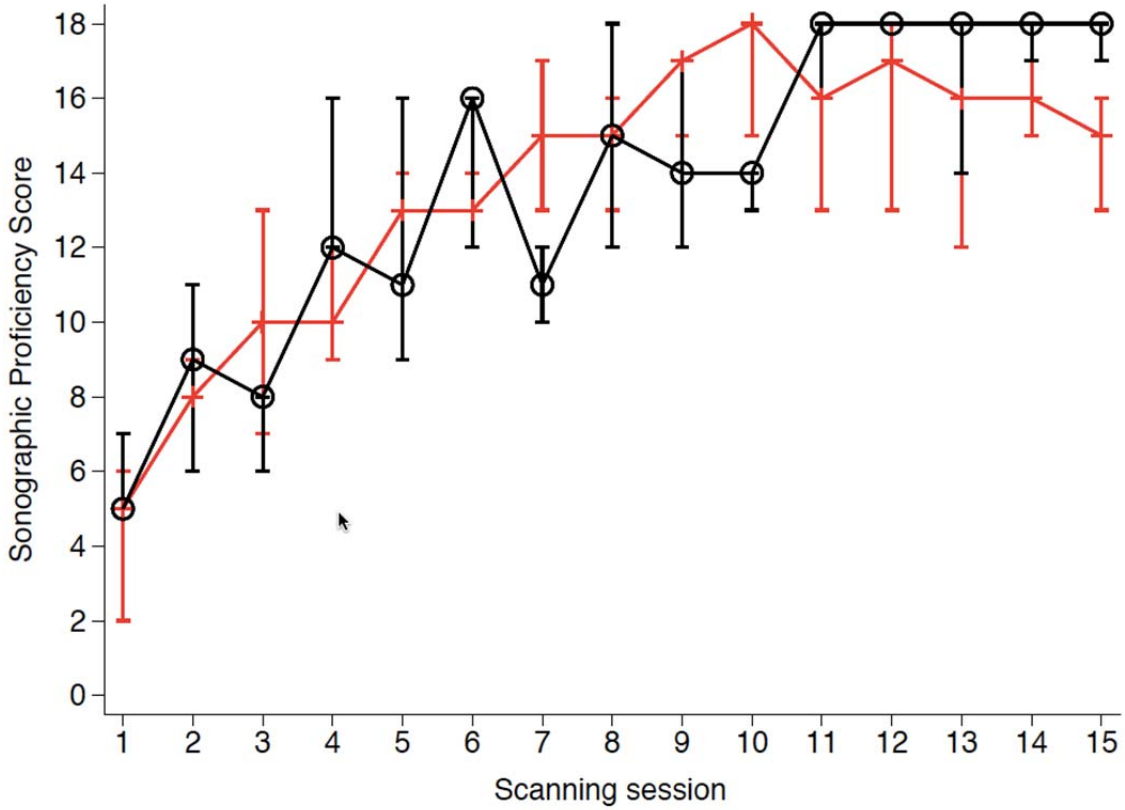


Figure 1. Group median (and interquartile range) of 'Sonographic Proficiency Scores' versus Scanning Session for massed (—+—) and distributed (—⊠—) groups.

C. **Preoperative briefing/debrief:** this can set the goals for the learning exercise (procedure) and set out the boundaries for what tasks will be done by whom. The briefing can assess the trainee's knowledge and a standard dialogue can be used. This is a dialogue that I commonly use for a registrar before performing a procedure. This assesses the trainees knowledge in advance of assessment of procedural skills required for trainee Work-based assessments.[5]

Pre-procedure interview (away from patient)

1. What is the name of the block and what is the indication for the block?
2. What are the most important anatomical landmarks?
   (Provide clues if necessary: Nerves, vessels, muscles, fascial planes if relevant)
3. What sensory and motor blockade will the block produce?
4. Assume I am the patient. Please provide me with information that I require to make an informed decision and be able to consent (or not) to this block procedure.
   (Provide clues if necessary: Side effects, risks and complications)
5. What monitoring are you going to use during the block?
6. What probe are you going to use?
   (If trainee states anything other than high or intermediate frequency/ linear probe, ask for further explanation).
7. Patient is …kg:
   What are you going to inject and what volume and concentration? What needle type?
8. Post-procedure management:

How are you going to test the success of the block?

If the block is unsuccessful (or incomplete) what are you going to do (assuming you still plan to proceed for surgery)?

If the block is successful what instructions are you going to give the patient and the nurse looking after the patient, after the operation?
The preoperative briefing can be used to discuss a wide range of logistical, generic clinical and hospital-specific issues relating to a particular case. Feedback given to a trainee when a procedure is completed can track back to learning objectives.

D. Educational concepts to consider during and immediately following a procedure:

a. Learning versus performance - performance of a motor skill is a measure of immediate aptitude. If a learning session leads to a sustained improvement in performance then learning has occurred. During a teaching session, improvement in a measurable aspect of a registrar's performance does not necessarily reflect learning. For learning to occur the improvement must persist or be transferrable to other skills.

b. Concurrent feedback - Feedback delivered during the performance of a skill. Concurrent feedback may be required for a novice however; if feedback is delivered during the procedure it may inhibit learning of an intermediate practitioner. In this setting concurrent feedback may represent coaching and inhibit progress towards independent practice.

c. Motor skill level of trainee - recognising the level of motor skill development is helpful in determining the required level of supervision and content of feedback. Motor skill stages have behavioral and neuropsychological correlates. Ideally the pre-trained novice has reached the intermediate stage of motor skill development. At the intermediate stage there is a degree of automaticity in the learners actions. We are more relaxed, have increased confidence and efficiency improves.

d. Zone of proximal development - When a registrar performs a task that is difficult enough that they require help from a tutor, it is described as being in the zone of proximal development. Tasks performed in this zone promote learning.

e. Scaffolding – is a term used to describe the support given to a registrar when they are performing a task in the zone of proximal development. Scaffolding also refers to support provided to control and monitor the clinical environment so that the trainee can focus on a specific skill.

f. Summary feedback – Feedback provided shortly after the procedure is completed. One method of initiating a discussion is to ask: "How do you think you performed?" Feedback should be targeted and focus on 2 -3 points including constructive feedback, positive feedback and identification of weaknesses.

Standardized pathways and the Perioperative Surgical Home

It is essential that regional anaesthesia is integrated into the modern paradigm of perioperative care. The modern paradigm of care includes programs such as Enhanced Recovery After Surgery (ERAS) and the Perioperative Surgical Home (PSH). The American Society of Anesthesiologists has proposed the PSH as a potential solution to the variability in both the cost and quality of perioperative care in the United States.[6] There is increasing demand to demonstrate value to patients, insurers and our patients. ERAS and PSH call for standardized, evidenced-based interdisciplinary perioperative pathways for commonly performed surgical procedures. Although PSH is likely to include ERAS pathways, the conceptual framework for PSH is more encompassing than ERAS. According to Kain, the PSH is a practice model that emphasizes superior coordination of care from the minute a decision to operate is made until 30 days after discharge. PSH aims to implement evidenced-based preoperative, intraoperative and postoperative protocols with minimal variability across an institution. Protocols will vary based on surgical services and will be tailored to the local environment. PSH will leverage data as a means to evaluate and improve all interventions in a continuous manner. Opportunities exist because of the vast amounts of information being collected from existing information technology infrastructure.[7] Length of stay and other metrics relating to cost are outcome measures that the PSH aims to collect. For example, in a total joint pathway developed for the PSH, the median length of stay for both total knee and hip arthroplasties was 3 (2-3) days, median (95% confidence interval).[6] Of relevance to our

current practice is that, these results (for length of stay) were obtained without the use of regional anaesthesia techniques traditionally used by anaesthesiologists.

In many scenarios, the lack of controlled trials relevant to a clinical pathway make it difficult to definitively recommend a specific technique for a pathway and often, there are several anaesthetic and analgesic options. Therefore, we may rely on consensus statements and expert opinions. As far as possible, in the PSH all therapies that the patient are exposed to will be based on evidenced-based best practice. In addition, the goal of PSH is to minimise variability within a given institution in the perioperative therapies and interventions that patients are exposed to. The PSH therefore requires development of standardized pathways for each stage of the perioperative care for commonly performed operations.

In the table below are examples of components of a pathway for total joint arthroplasty. This structure contains content that is not meant to be definitive and in no way implies that there is consensus on many of the components of the pathway.

Anaesthetic pathway for total joint arthroplasty
Goals of the pathway The goals of the pathway should be clearly stated, and may include reduction in mortality, morbidity, and costs, increases in patient satisfaction and improved pain control.
Patient selection Modifiable risk factors, such as smoking, poorly controlled diabetes, obesity, and recreational drug use may affect rates of surgical complications.[8] The pathway may address when surgery should be delayed to address these factors. Non-modifiable co-morbidities that create an unacceptable surgical risk may be included in a pathway.
Preoperative education and preadmission planning This provides an opportunity to identify patient characteristics that conflict with the default elements in the anaesthetic pathway and address them preoperatively. If the pathway includes continuous peripheral nerve catheters, this may provide an opportunity for patient education. Patients should be coached on use of multimodal analgesia. Preoperative physical therapy should be considered.
Pre-procedure checklist This pathway element frequently includes patient identification, site marking, confirmation of allergies, comorbidities, availability of blood products, coagulation status, and final checks of blood results.

Multimodal analgesia

Agent	Benefits	Drawbacks
Paracetamol	Reduce postoperative pain scores, opioids sparing.[9]	Hepatotoxicity.
Gabapentin / pregabalin	Reduce postoperative pain scores, opioid sparing,[10] may reduce incidence of chronic postsurgical pain [11] and benefit patients with chronic pain.[12]	Increased sedation, particularly in elderly, increased respiratory depression with doses > 300 mg (when combined with general but not neuraxial anaesthesia).[13]
Cyclooxygenase - 2 inhibitors	Reduce postoperative pain scores, opioids sparing.[14]	Renal impairment.
Oral opioids (e.g. sustained release oxycodone)	Reduce postoperative pain scores.[15]	Increased risk of respiratory depression with oxycodone dose > 10 mg (when combined with general but not neuraxial anaesthesia).[13]

Use of regional anaesthesia for postoperative pain control

The choice of technique may affect other elements of the pathway. Approaches are evolving as new techniques, equipment and drugs become available. The following table lists common sites used for regional anesthesia, along with benefits and drawbacks.

Technique	Benefits	Drawbacks
Epidural block	Considered the gold standard for postoperative analgesia for a range of surgeries.	The side-effect profile may interfere with modern care pathway and rare risk of catastrophic outcome (e.g. epidural hematoma).[16,17]
Femoral nerve block	Effective for knee surgery without drawbacks of epidural;[18] associated with improved outcomes at 6 weeks in one trial.[19]	Quadriceps weakness may interfere with rehabilitation. Small risk (2 – 4 per 10,000) of long-term nerve injury [20] but overall choice of anaesthetic does change risk of nerve injury.[21]
Sciatic nerve block	Reduced posterior knee pain.[22]	Risk of neuropathy similar to femoral nerve block. May improve analgesia and early mobilization [23] or add little analgesia to existing femoral block. [24,25], unlikely to improve long-term outcomes.[26]
Selective tibial nerve block	Reduced likelihood of foot drop.[27]	Injection close to popliteal crease, risk of peroneal nerve injury with lateral to medial approach or vascular injury.
Adductor canal	Similar (but probably not as effective) pain relief to femoral nerve block with reduced muscle weakness,[28,29] effective in treating existing severe pain.[30]	Closer to surgical site, evolving technique.
Local infiltration analgesia	Easy and quick to perform, no muscle weakness.	Evolving evidence for efficacy.[31] However, experts point to poor quality of some of the existing studies.[32] Success of technique, likely operator-dependent. Associated with transient peroneal nerve palsy.[33]

Other variables to consider for regional anaesthesia.

Technique	Benefits	Drawbacks
Single shot	Quick to perform, low cost, effective.[18]	Shortest duration (may be benefit if rapid recovery of muscle strength is required for physical therapy).
Nerve catheter	Improved analgesia compared to single –injection technique.[34] Longest duration of analgesia, relatively titratable.	More difficult and time consuming to perform, more expensive, requires postoperative surveillance.
Extended release formulations of local anaesthetic (e.g. liposomal bupivacaine)	As quick to perform as single shot block, with longer block duration	Compared with bupivacaine, currently little evidence of efficacy [35]. Costly. Limits ability to redo block. Safety and side-effect profile currently emerging.[36]

Surgical anaesthesia

Options for surgical anesthesia are summarized in the table below.

Anaesthesia	Benefits	Drawbacks
Spinal	Associated with improved outcomes including a mortality benefit in some[37,38] but not all studies.[39]	Technically difficult on certain patients. Rare catastrophic outcomes. Duration of spinal anaesthesia may be inadequate for surgery. Patients may be reluctant to be 'awake' for surgery.
Epidural	Similar benefits as spinal, but can be used for postoperative analgesia and longer duration surgeries.	Can be technically difficult on certain patients. Rare catastrophic outcomes, e.g. epidural hematoma.[16,17]. Patients may be reluctant to be 'awake' for surgery.
General	Complete amnesia	Rare catastrophic outcome (e.g. difficult airway), increased risk of respiratory depression.[13]

Neuraxial anaesthesia is associated with improved outcomes [37,40] however this modality is not always preferred.[41] If neuraxial anaesthesia is employed, decisions regarding the inclusion or exclusion of long- or short- acting opioids are relevant as it may impact on subsequent pathway elements (postoperative monitoring, rehabilitation, etc.). Even patients who receive neuraxial anaesthesia usually require sedation and some pathways may specify the desired level of sedation.

Intraoperative drugs

This may include first- and second-line antibiotics, preferred antiemetics for spinal anaesthesia versus general anaesthesia, and preferred sedatives for spinal or epidural anaesthesia. Anticoagulation is usually started in the postoperative period by the surgery team, but may be commented upon here. Intraoperative dexamethasone appears to improve postoperative pain scores as well as proving an effective antiemetic.[42]

Intraoperative transfusion goals and blood conservation options

Blood transfusion has risks[43] and one of the goals of the pathway may be to minimize blood loss and hence transfusion requirements. A wide variety of techniques are available to minimize blood loss, some of which are summarized in the table below.

Technique	Benefits	Drawbacks
Intraoperative hypotension	Reduced blood loss	Increased vigilance and monitoring required. Risk of end-organ ischaemia. Under-resuscitation may contribute to postoperative orthostatic intolerance impairing early mobilisation
Tourniquet use	Reduced blood loss and protocols exist on appropriate use. [44]	Risk of ischaemic injury or axonal neuropathy [45-47] or effect on quadriceps function. [48]
Appropriate thermoregulation	Reduced blood loss via maintenance of coagulation cascade, improved recovery	
Cell scavenging	Reduced exposure to allogenic blood products	Added cost and complexity
Reinfusion drains	Reduced allogenic blood product requirements	Added cost and complexity
Tranexamic acid	Reduced blood loss due to antifibrinolysis. [49]	Association with seizures.[50] No known increased risk of thrombotic events but has only recently come into use in this surgical population.

Postoperative pain control

Pathways often address pain control for patients with chronic pain or opioid use, as well as those without. Generally this section will comment on both the expected infusion regimens for continuous peripheral nerve blockade,[51] as well as adjuvants such as patient control opioid administration, ketamine, or other drugs.

Considerations regarding orthopedic surgical pathway

Anaesthetic pathways need to comment upon ways in which they interact with the surgeon's pathway, and make it clear why particular recommendations are made.

What outcomes matter

Likely our sub-specialty will need to focus on long-term, more definitive and patient-centred outcomes including: 30-day mortality,[37] disability free survival,[52] validated Quality of Recovery Scores,[53] robust measures of postoperative pain such as the Brief Pain Inventory,[54] hospital length of stay, re-admission rates and persistent post-surgical pain.[55,56]

Having a broader perspective

Patient outcomes may benefit from anaesthetists' having a broader perspective, collaborating outside of their specialty, incorporating a diverse set of viewpoints, backgrounds and skills. Vincent has eloquently outlined the benefits of multidisciplinary work, collaboration and exchange.[57] Dr Neuman (USA) has been awarded the 2015 American Society of Anesthesiology Presidential Scholar award.[58] Recently, he also received $11.9 million funding from the Patient Centered Outcomes Research Institute for a major, multicenter pragmatic trial of spinal versus general anaesthesia for patients having surgery for fractured neck of femur. This proposal required engagement with a wide array of patients and stakeholders, including local elder advocacy organizations, the American Society of Anesthesiology and the Anesthesia Quality Institute, the U.S. Centers for Medicare and Medicaid Services, other organisations and professional societies; as well as 37 academic and community hospitals in the United States, Canada, and Australia.[58]

References

1. Slater RJ, Castanelli DJ, Barrington MJ: Learning and teaching motor skills in regional anesthesia: a different perspective. *Reg Anesth Pain Med* 2014; 39: 230-239
2. Schmidt R. Supplementing the Learning Experience In. In: *Motor Learning and Performance - A Situation-Based Learning Approach 4th Edition.* 4th edn. Champaign, IL, USA: Human Kinetics; 2008: 218 - 253.
3. Fletcher GC, McGeorge P, Flin RH, Glavin RJ, Maran NJ: The role of non-technical skills in anaesthesia: a review of current literature. *Br J Anaesth* 2002; 88: 418-429
4. Hastings RH, Rickard TC: Deliberate practice for achieving and maintaining expertise in anesthesiology. *Anesth Analg* 2015; 120: 449-459
5. Watson MJ, Wong DM, Kluger R, et al.: Psychometric evaluation of a direct observation of procedural skills assessment tool for ultrasound-guided regional anaesthesia. *Anaesthesia* 2014; 69: 604-612
6. Garson L, Schwarzkopf R, Vakharia S, et al.: Implementation of a total joint replacement-focused perioperative surgical home: a management case report. *Anesth Analg* 2014; 118: 1081-1089
7. Cyriac J, Cannesson M, Kain Z: Pain management and the perioperative surgical home: getting the desired outcome right. *Reg Anesth Pain Med* 2015; 40: 1-2
8. Peersman G, Laskin R, Davis J, Peterson M: Infection in total knee replacement: a retrospective review of 6489 total knee replacements. *Clin Orthop Relat Res* 2001: 15-23
9. Zhou TJ, Tang J, White PF: Propacetamol versus ketorolac for treatment of acute postoperative pain after total hip or knee replacement. *Anesth Analg* 2001; 92: 1569-1575
10. Mishriky BM, Waldron NH, Habib AS: Impact of pregabalin on acute and persistent postoperative pain: a systematic review and meta-analysis. *Br J Anaesth* 2015; 114: 10-31
11. Clarke H, Bonin RP, Orser BA, Englesakis M, Wijeysundera DN, Katz J: The prevention of chronic postsurgical pain using gabapentin and pregabalin: a combined systematic review and meta-analysis. *Anesth Analg* 2012; 115: 428-442
12. Sawan H, Chen AF, Viscusi ER, Parvizi J, Hozack WJ: Pregabalin reduces opioid consumption and improves outcome in chronic pain patients undergoing total knee arthroplasty. *Phys Sportsmed* 2014; 42: 10-18
13. Weingarten TN, Jacob AK, Njathi CW, Wilson GA, Sprung J: Multimodal Analgesic Protocol and Postanesthesia Respiratory Depression During Phase I Recovery After Total Joint Arthroplasty. *Reg Anesth Pain Med* 2015; 40: 330-336
14. Lin J, Zhang L, Yang H: Perioperative administration of selective cyclooxygenase-2 inhibitors for postoperative pain management in patients after total knee arthroplasty. *J Arthroplasty* 2013; 28: 207-213 e202
15. Rothwell MP, Pearson D, Hunter JD, et al.: Oral oxycodone offers equivalent analgesia to intravenous patient-controlled analgesia after total hip replacement: a randomized, single-centre, non-blinded, non-inferiority study. *Br J Anaesth* 2011; 106: 865-872
16. Pumberger M, Memtsoudis SG, Stundner O, et al.: An analysis of the safety of epidural and spinal neuraxial anesthesia in more than 100,000 consecutive major lower extremity joint replacements. *Reg Anesth Pain Med* 2013; 38: 515-519
17. Bateman BT, Mhyre JM, Ehrenfeld J, et al.: The risk and outcomes of epidural hematomas after perioperative and obstetric epidural catheterization: a report from the Multicenter Perioperative Outcomes Group Research Consortium. *Anesth Analg* 2013; 116: 1380-1385
18. Paul JE, Arya A, Hurlburt L, et al.: Femoral nerve block improves analgesia outcomes after total knee arthroplasty: a meta-analysis of randomized controlled trials. *Anesthesiology* 2010; 113: 1144-1162
19. Carli F, Clemente A, Asenjo JF, et al.: Analgesia and functional outcome after total knee arthroplasty: periarticular infiltration vs continuous femoral nerve block. *Br J Anaesth* 2010; 105: 185-195
20. Neal JM, Barrington MJ, Brull R, et al.: The Second ASRA Practice Advisory on Neurologic Complications Associated With RegionalAnesthesia and Pain Medicine Executive Summary 2015. *Reg Anesth Pain Med* 2015; Volume 40, Number 5, September-October 2015
21. Jacob AK, Mantilla CB, Sviggum HP, Schroeder DR, Pagnano MW, Hebl JR: Perioperative nerve injury after total knee arthroplasty: regional anesthesia risk during a 20-year cohort study. *Anesthesiology* 2011; 114: 311-317

22. Sato K, Adachi T, Shirai N, Naoi N: Continuous versus single-injection sciatic nerve block added to continuous femoral nerve block for analgesia after total knee arthroplasty: a prospective, randomized, double-blind study. *Reg Anesth Pain Med* 2014; 39: 225-229
23. Cappelleri G, Ghisi D, Fanelli A, Albertin A, Somalvico F, Aldegheri G: Does continuous sciatic nerve block improve postoperative analgesia and early rehabilitation after total knee arthroplasty? A prospective, randomized, double-blinded study. *Reg Anesth Pain Med* 2011; 36: 489-492
24. Abdallah FW, Chan VW, Gandhi R, Koshkin A, Abbas S, Brull R: The analgesic effects of proximal, distal, or no sciatic nerve block on posterior knee pain after total knee arthroplasty: a double-blind placebo-controlled randomized trial. *Anesthesiology* 2014; 121: 1302-1310
25. Safa B, Gollish J, Haslam L, McCartney CJ: Comparing the effects of single shot sciatic nerve block versus posterior capsule local anesthetic infiltration on analgesia and functional outcome after total knee arthroplasty: a prospective, randomized, double-blinded, controlled trial. *J Arthroplasty* 2014; 29: 1149-1153
26. Wegener JT, van Ooij B, van Dijk CN, et al.: Long-term pain and functional disability after total knee arthroplasty with and without single-injection or continuous sciatic nerve block in addition to continuous femoral nerve block: a prospective, 1-year follow-up of a randomized controlled trial. *Reg Anesth Pain Med* 2013; 38: 58-63
27. Sinha SK, Abrams JH, Arumugam S, et al.: Femoral nerve block with selective tibial nerve block provides effective analgesia without foot drop after total knee arthroplasty: a prospective, randomized, observer-blinded study. *Anesth Analg* 2012; 115: 202-206
28. Jaeger P, Zaric D, Fomsgaard JS, et al.: Adductor canal block versus femoral nerve block for analgesia after total knee arthroplasty: a randomized, double-blind study. *Reg Anesth Pain Med* 2013; 38: 526-532
29. Kwofie MK, Shastri UD, Gadsden JC, et al.: The effects of ultrasound-guided adductor canal block versus femoral nerve block on quadriceps strength and fall risk: a blinded, randomized trial of volunteers. *Reg Anesth Pain Med* 2013; 38: 321-325
30. Jaeger P, Grevstad U, Henningsen MH, Gottschau B, Mathiesen O, Dahl JB: Effect of adductor-canal-blockade on established, severe post-operative pain after total knee arthroplasty: a randomised study. *Acta Anaesthesiol Scand* 2012; 56: 1013-1019
31. den Hartog YM, Mathijssen NM, van Dasselaar NT, Langendijk PN, Vehmeijer SB: No effect of the infiltration of local anaesthetic for total hip arthroplasty using an anterior approach: A randomised placebo controlled trial. *Bone Joint J* 2015; 97-B: 734-740
32. Andersen LO, Kehlet H: Analgesic efficacy of local infiltration analgesia in hip and knee arthroplasty: a systematic review. *Br J Anaesth* 2014; 113: 360-374
33. Tsukada S, Wakui M, Hoshino A: Postoperative epidural analgesia compared with intraoperative periarticular injection for pain control following total knee arthroplasty under spinal anesthesia: a randomized controlled trial. *J Bone Joint Surg Am* 2014; 96: 1433-1438
34. Bingham AE, Fu R, Horn JL, Abrahams MS: Continuous peripheral nerve block compared with single-injection peripheral nerve block: a systematic review and meta-analysis of randomized controlled trials. *Reg Anesth Pain Med* 2012; 37: 583-594
35. Schroer WC, Diesfeld PG, LeMarr AR, Morton DJ, Reedy ME: Does Extended-Release Liposomal Bupivacaine Better Control Pain Than Bupivacaine After TKA? A Prospective, Randomized Clinical Trial. *J Arthroplasty* 2015
36. Ilfeld BM, Viscusi ER, Hadzic A, et al.: Safety and Side Effect Profile of Liposome Bupivacaine (Exparel) in Peripheral Nerve Blocks. *Reg Anesth Pain Med* 2015
37. Memtsoudis SG, Sun X, Chiu YL, et al.: Perioperative comparative effectiveness of anesthetic technique in orthopedic patients. *Anesthesiology* 2013; 118: 1046-1058
38. Memtsoudis SG, Sun X, Chiu YL, et al.: Utilization of critical care services among patients undergoing total hip and knee arthroplasty: epidemiology and risk factors. *Anesthesiology* 2012; 117: 107-116
39. Neuman MD, Rosenbaum PR, Ludwig JM, Zubizarreta JR, Silber JH: Anesthesia technique, mortality, and length of stay after hip fracture surgery. *JAMA* 2014; 311: 2508-2517
40. Pugely AJ, Martin CT, Gao Y, Mendoza-Lattes S, Callaghan JJ: Differences in short-term complications between spinal and general anesthesia for primary total knee arthroplasty. *J Bone Joint Surg Am* 2013; 95: 193-199
41. Fleischut PM, Eskreis-Winkler JM, Gaber-Baylis LK, et al.: Variability in anesthetic care for total knee arthroplasty: an analysis from the anesthesia quality institute. *Am J Med Qual* 2015; 30: 172-179

42. Backes JR, Bentley JC, Politi JR, Chambers BT: Dexamethasone reduces length of hospitalization and improves postoperative pain and nausea after total joint arthroplasty: a prospective, randomized controlled trial. *J Arthroplasty* 2013; 28: 11-17
43. Goodnough LT: Risks of blood transfusion. *Crit Care Med* 2003; 31: S678-686
44. Fitzgibbons PG, Digiovanni C, Hares S, Akelman E: Safe tourniquet use: a review of the evidence. *J Am Acad Orthop Surg* 2012; 20: 310-319
45. Nitz AJ, Dobner JJ, Matulionis DH: Pneumatic tourniquet application and nerve integrity: motor function and electrophysiology. *Exp Neurol* 1986; 94: 264-279
46. Kornbluth ID, Freedman MK, Sher L, Frederick RW: Femoral, saphenous nerve palsy after tourniquet use: a case report. *Arch Phys Med Rehabil* 2003; 84: 909-911
47. Weingarden SI, Louis DL, Waylonis GW: Electromyographic changes in postmeniscectomy patients. Role of the pneumatic tourniquet. *JAMA* 1979; 241: 1248-1250
48. Saunders KC, Louis DL, Weingarden SI, Waylonis GW: Effect of tourniquet time on postoperative quadriceps function. *Clin Orthop Relat Res* 1979: 194-199
49. Kagoma YK, Crowther MA, Douketis J, Bhandari M, Eikelboom J, Lim W: Use of antifibrinolytic therapy to reduce transfusion in patients undergoing orthopedic surgery: a systematic review of randomized trials. *Thromb Res* 2009; 123: 687-696
50. Murkin JM, Falter F, Granton J, Young B, Burt C, Chu M: High-dose tranexamic Acid is associated with nonischemic clinical seizures in cardiac surgical patients. *Anesth Analg* 2010; 110: 350-353
51. Ilfeld BM, Moeller LK, Mariano ER, et al.: Continuous peripheral nerve blocks: is local anesthetic dose the only factor, or do concentration and volume influence infusion effects as well? *Anesthesiology* 2010; 112: 347-354
52. Shulman MA, Myles PS, Chan MT, McIlroy DR, Wallace S, Ponsford J: Measurement of disability-free survival after surgery. *Anesthesiology* 2015; 122: 524-536
53. Stark PA, Myles PS, Burke JA: Development and psychometric evaluation of a postoperative quality of recovery score: the QoR-15. *Anesthesiology* 2013; 118: 1332-1340
54. Ilfeld BM, Madison SJ, Suresh PJ, et al.: Treatment of postmastectomy pain with ambulatory continuous paravertebral nerve blocks: a randomized, triple-masked, placebo-controlled study. *Reg Anesth Pain Med* 2014; 39: 89-96
55. Karmakar MK, Samy W, Li JW, et al.: Thoracic paravertebral block and its effects on chronic pain and health-related quality of life after modified radical mastectomy. *Reg Anesth Pain Med* 2014; 39: 289-298
56. Ilfeld BM, Madison SJ, Suresh PJ, et al.: Persistent postmastectomy pain and pain-related physical and emotional functioning with and without a continuous paravertebral nerve block: a prospective 1-year follow-up assessment of a randomized, triple-masked, placebo-controlled study. *Ann Surg Oncol* 2015; 22: 2017-2025
57. Vincent C, Batalden P, Davidoff F: Multidisciplinary centres for safety and quality improvement: learning from climate change science. *BMJ Qual Saf* 2011; 20 Suppl 1: i73-78
58. Fleisher LA: Mark D. Neuman, M.D., M.Sc., Recipient of the 2015 Presidential Scholar Award. *Anesthesiology*; 123

Anaesthesia, the Health Quality and Safety Commission, and Us

Alan Merry

Department of Anaesthesia, Auckland City Hospital

The Health Quality and Safety Commission (HQSC) was formed in 2010 as a crown agent (and therefore independent of the Ministry of Health) to monitor, guide, support, influence, and encourage improvement in health and disability services in New Zealand (NZ) [1]. In absolute terms, we have one of the least expensive and best value health care systems in the world, and patients have good access to effective and reasonably timely care. Unfortunately, as with most countries, our spending on health care has increased as a proportion of GDP every year since 1999[2]. Despite this, there are persistent challenges in providing high quality services safely to all our patients, and adverse events continue to be reported. Social determinants of health are as important as healthcare services, and inadequate housing and child poverty are of increasing concern.

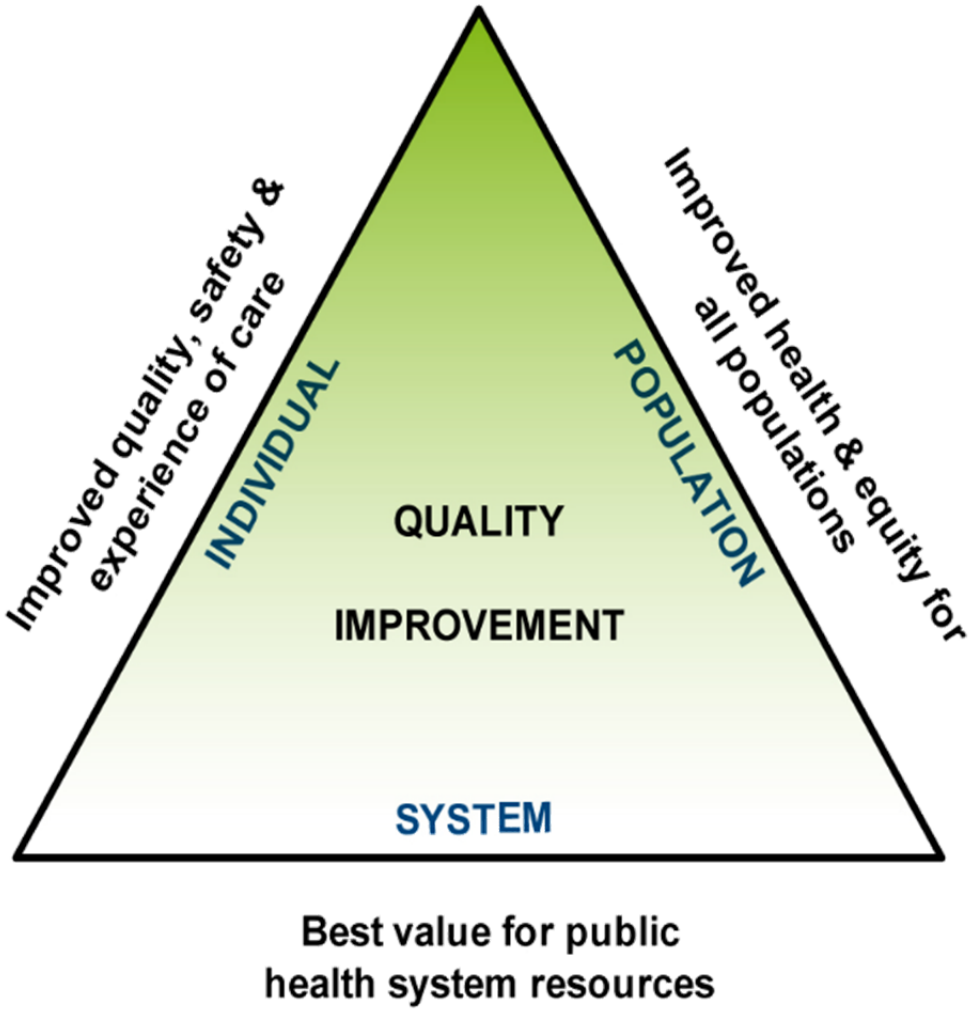


Figure 1. The New Zealand Triple Aim (source, HQSC).

The New Zealand Triple Aim[3-5] (figure 1) has now been widely adopted as the overarching goal of our health and disability services, promoting the Government's two high level outcomes:

1. New Zealanders living longer, healthier and more independent lives; and
2. New Zealand's economic growth is supported.

The three aims are:

1. improved quality, safety and experience of care;
2. improved health and equity for all populations; and
3. best value for public health system resources.

These are underpinned by two fundamental objectives:

1. doing the right things; and
2. doing things right, first time.

Measurement is integral to improving quality in any endeavour, including healthcare[6]. However, there are costs in measurement, and with limited resources it is important for every aspect of measuring the quality and safety of healthcare that the burden is kept low and that the value is clear. In this light, the HQSC has introduced a framework of interacting measures [5]. These include:

1. quality and safety indicators (QSIs), which are measures of the whole system;
2. quality and safety markers (QSMs), which are measures of specific interventions to improve the quality or safety of particular aspects of our services;
3. the New Zealand Atlas of Healthcare Variation;
4. Quality Accounts with which District Health Boards report on the quality and safety of their services in parallel with their financial reports;
5. the reports of the four mortality review committees; and
6. annual reports of serious and sentinel adverse events.

The QSMs are unique to NZ. In general they consist of a measure of process (e.g., compliance with the World Health Organization's five moments of hand hygiene) and an indicator measure of one related outcome (e.g., the rate of staphylococcus aureus infection in hospitals). The initiatives evaluated by these QSMs have direct relevance to specific problems in our services, but they also serve to engage healthcare professionals in quality improvement and thereby build capability and capacity in improvement and implementation science. Local context is critically important in the delivery of healthcare, and improvement depends on the engagement of local practitioners. This is certainly true in the operating room generally and for anaesthetists in particular. There are several examples of outstanding engagement and leadership by anaesthetists in the Commission's work, and also in quality improvement more generally. The work of Simon Mitchell on the effective use of the WHO Surgical Safety Checklist[7-10], of Leona Wilson leading the Perioperative Mortality Review Committee[11], and of John Barnard chairing the Medication Safety Expert Advisory Group illustrates the former point, and of Kerry Gunn on the rational use of blood exemplifies the latter.

The HQSC is expected to provide advice to the Minister and to others within the sector. Recently requests have been made under the official information act for the release of the following information for the past five calendar years, broken down first by each surgical department, and then by each individual surgeon (including their name and area of expertise): the number of procedures/operation performed and the numbers...

- with consequent complications;
- with consequent infections;
- which consequently required secondary corrective surgery; and
- with consequent deaths arising from the surgery.

DHB CEOs have been reluctant to comply, and the Health Ombudsman has been asked to rule on this dispute. The HQSC has undertaken an extensive review of the relevant issues. These include:

- Statistical considerations in the identification of outlying performance of individual practitioners on the basis of outcomes such as perioperative mortality 12.
- The contribution of other practitioners to perioperative mortality, notably anaesthetists 13 14 and intensivists.
- The potential contribution of other practitioners to postoperative infections, notably anaesthetists 15-28, junior doctors and nursing staff 29.
- The importance of driving teamwork rather than idiosyncratic behaviour 30,31.

The HQSC is presently consulting with the sector and consumers before formulating advice on this matter. The contribution of members of the Australian and New Zealand College of Anaesthetists' NZ National Committee to this process of consultation has been particularly helpful.

I am convinced that the culture prevailing in NZ healthcare is overwhelmingly positive, notably amongst anaesthetists. This contributes to the excellent results achieved for the vast majority of our patients at very modest cost. The Commission's role is to work with practitioners, including anaesthetists, to make good healthcare better.

References

1. Shuker C, Bohm G, Bramley D, et al. The Health Quality and Safety Commission: making good health care better. N Z Med J. 2015;128(1408):97-109.
2. Merry AF, Hamblin R. More for less: Best patient outcomes in a time of financial restraint. Journal of Extra-Corporeal Technology. 2012;44(4):178-185.
3. The IHI Triple Aim. 2012; http://www.ihi.org/offerings/Initiatives/TripleAim/Pages/default.aspx accessed 7th August 2015.
4. *Bisognano M, Charles K.* Leadership for the triple aim. Three-pronged framework helps executives lead quality initiatives. Healthcare Executive. 2012;27(2):80, 82-83.
5. *Hamblin R, Bohm G, Gerard C, et al.* The measurement of New Zealand health care. N Z Med J. 2015;128(1413):50-64.
6. *Merry AF, Weller J, Mitchell SJ.* Improving the quality and safety of patient care in cardiac anesthesia. Journal of Cardiothoracic & Vascular Anesthesia. 2014;28(5):1341-1351.
7. *Hannam JA, Glass L, Kwon J, et al.* A prospective, observational study of the effects of implementation strategy on compliance with a surgical safety checklist. BMJ Quality & Safety. 2013;22(11):940-947.
8. *Merry AF, Mitchell SJ.* The world health organization safe surgical checklist: It's time to engage. N Z Med J. 2012;125(1362):11-14.
9. *Vogts N, Hannam JA, Merry AF, et al.* Compliance and quality in administration of a surgical safety checklist in a tertiary New Zealand hospital. N Z Med J. 2011;124(1342):48-58.
10. *Haynes AB, Weiser TG, Berry WR, et al.* A surgical safety checklist to reduce morbidity and mortality in a global population. New England Journal of Medicine. 2009;360(5):491-499.
11. *The Perioperative Mortality Review Committee.* Perioperative mortality in New Zealand. Inaugural report of the Perioperative Mortality Review Committee 2011. Wellington: Health Quality and Safety Commission; 2012.
12. *Walker K, Neuburger J, Groene O, et al.* Public reporting of surgeon outcomes: low numbers of procedures lead to false complacency. The Lancet. 2013;382(9905):1674-1677.
13. *Merry AF, Ramage MC, Whitlock RML, et al.* First-time coronary artery bypass grafting: The anaesthetist as a risk factor. British Journal of Anaesthesia. 1992;68(1):6-12.
14. *Glance LG, Kellermann AL, Hannan EL, et al.* The impact of anesthesiologists on coronary artery bypass graft surgery outcomes. Anesthesia & Analgesia. 2015;120(3):526-533.
15. *Gargiulo DA, Sheridan J, Webster CS, et al.* Anaesthetic drug administration as a potential contributor to healthcare-associated infections: A prospective simulation-based evaluation of aseptic techniques in the administration of anaesthetic drugs. BMJ Qual Saf. 2012;21(10):826-834.
16. *Koff MD, Corwin HL, Beach ML, et al.* Reduction in ventilator associated pneumonia in a mixed intensive care unit after initiation of a novel hand hygiene program. Journal of Critical Care. 2011;26(5):489-495.
17. *Koff MD, Loftus RW, Burchman CC, et al.* Reduction in intraoperative bacterial contamination of peripheral intravenous tubing through the use of a novel device. Anesthesiology. 2009;110(5):978-985.
18. *Korb ML, Hawn MT, Singletary BA, et al.* Adoption of preoperative oral antibiotics decreases surgical site infection for elective colorectal surgery. Am Surg. 2014;80(9):e270-273.
19. *Loftus RW, Brindeiro BS, Kispert DP, et al.* Reduction in intraoperative bacterial contamination of peripheral intravenous tubing through the use of a passive catheter care system. Anesthesia and Analgesia. 2012;115(6):1315-1323.
20. *Loftus RW, Brown JR, Koff MD, et al.* Multiple reservoirs contribute to intraoperative bacterial transmission. Anesthesia and Analgesia. 2012;114(6):1236-1248.
21. *Loftus RW, Brown JR, Patel HM, et al.* Transmission Dynamics of Gram-Negative Bacterial Pathogens in the Anesthesia Work Area. Anesthesia & Analgesia. 2015;120(4):819-826.
22. *Loftus RW, Koff MD, Birnbach DJ.* The Dynamics and Implications of Bacterial Transmission Events Arising from the Anesthesia Work Area. Anesthesia and Analgesia. 2015;120(4):853-860.
23. *Loftus RW, Koff MD, Brown JR, et al.* The Epidemiology of Staphylococcus aureus Transmission in the Anesthesia Work Area. Anesthesia & Analgesia. 2015;120(4):807-818.
24. *Loftus RW, Koff MD, Brown JR, et al.* The Dynamics of Enterococcus Transmission from Bacterial Reservoirs Commonly Encountered by Anesthesia Providers. Anesthesia & Analgesia. 2015;120(4):827-836.

25. *Loftus RW, Koff MD, Burchman CC, et al.* Transmission of pathogenic bacterial organisms in the anesthesia work area. Anesthesiology. 2008;109(3):399-407.
26. *Loftus RW, Muffly MK, Brown JR, et al.* Hand contamination of anesthesia providers is an important risk factor for intraoperative bacterial transmission. Anesthesia & Analgesia. 2011;112(1):98-105.
27. *Loftus RW, Patel HM, Huysman BC, et al.* Prevention of intravenous bacterial injection from health care provider hands: the importance of catheter design and handling. Anesthesia and Analgesia. 2012;115(5):1109-1119.
28. *Loftus RW, Yeager MP, Clark JA, et al.* Intraoperative ketamine reduces perioperative opiate consumption in opiate-dependent patients with chronic back pain undergoing back surgery. Anesthesiology. 2010;113(3):639-646.
29. *Pittet D.* WHO First Global Patient Safety Challenge: saving lives in healthcare through clean hands. Infection. 2010;38(2):79-80.
30. *Merry AF, Weller JM.* Teamwork and minimizing error. Oxford Textbook of Cardiothoracic Anaesthesia. 2015;2003:461.
31. *Leslie K, Merry AF.* Cardiac surgery: all for one and one for all. Anesthesia & Analgesia. 2015;120(3):504-506.

Cardiac Risk of Non-Cardiac Surgery

Roman Kluger

Department of Anaesthesia, St. Vincent's Hospital

Is it a problem?

Yes – death directly due to anaesthesia is less than 1 in a few hundred thousand. However 30 day mortality and morbidity following surgery is common and much of it is cardiac. Worldwide more than 200 million adults have major non-cardiac surgical procedures annually. Millions of these patients will have a major vascular complication (vascular death, nonfatal myocardial infarction [MI], nonfatal cardiac arrest, or nonfatal stroke) within 30 days after surgery. Myocardial infarction is the most common major peri-operative vascular complication. For example in the original POISE (PeriOperative ISchemic Evaluation) study[1] 1.6% died of vascular causes, 0.7% had a stroke, 0.5% had a non-fatal cardiac arrest, and 5.0% had an MI in the first 30 days. Overall postoperative vascular events account for one third of perioperative deaths and are associated with increased hospital stay and long-term mortality rates.

Why estimate risk?

1. To inform patient and surgeon decision making. Most surgery is elective and often can be postponed or even cancelled. Less invasive surgical alternatives may be available and more appropriate.

2. To inform peri-operative management. Risk reduction strategies must aim at high risk patients. For example pre-operative troponins, BNP levels and stress testing; invasive haemodynamic monitoring and tighter blood pressure and heart rate control during surgery; and postoperative monitoring in high dependency /ICU, postoperative troponins and long term follow-up and prophylactic cardiac medications. However at this stage none of these strategies have been convincingly shown to safely decrease morbidity or mortality.

Peri-operative Myocardial Infarction

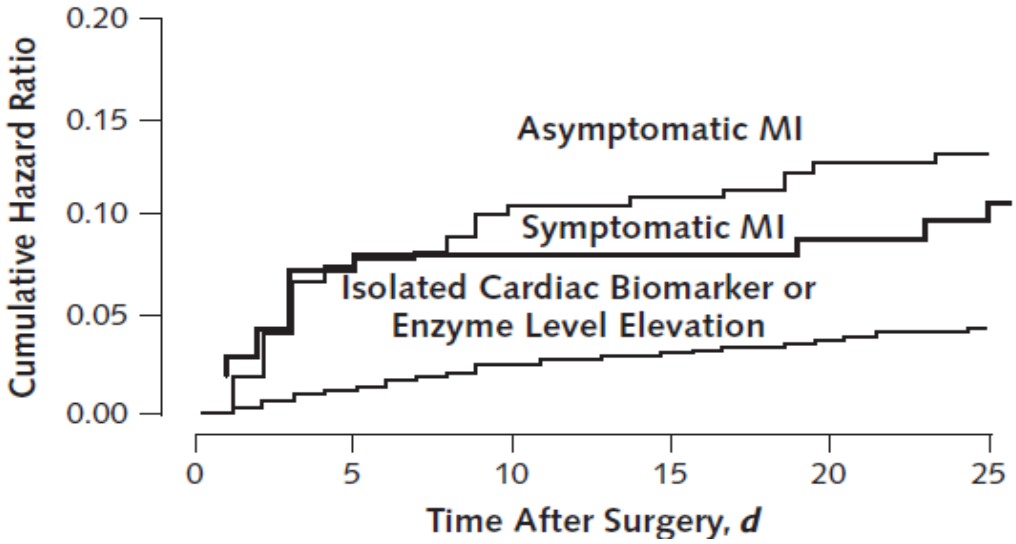
The risk of an MI in the postoperative period is far higher than at any other period and its prognosis is worse (approximately double the mortality of an outpatient MI) because the postoperative period is a period of inflammation, hypercoagulability, stress, hypoxia and anaemia.

Analysis of the POISE data, where all patients had daily cardiac enzymes, ECGs and clinical evaluations provided much information regarding postoperative MIs[2].

5% of the patients had a postoperative MI and the 30 day mortality was higher for patients who had an MI (11.6%) than for those who did not (2.2%). 74% of MIs occurred within 48 hours of surgery. 58% of patients who had an MI died with 48 hours of the event.

Of the patients who had an MI 65% did not have ischaemic symptoms, and the mortality rate was similar between those who did and did not have symptoms.
8.3% of patients had elevated levels of cardiac enzymes (but did not fulfil MI definition) and were also at higher risk of adverse cardiac events. See figure 1.
Only 10.6 % of the MIs had ST elevation.

Figure 1: Cumulative hazard ratios for mortality among patients who had symptomatic or asymptomatic MI or an isolated elevated level of a cardiac biomarker or enzyme after surgery.



Pre-operative Risk Prediction

Several risk indices have been developed during the past 30 years, based on multivariate analyses of observational data, to represent the relationship between clinical characteristics and peri-operative cardiac mortality and morbidity. The indices developed by Goldman et al. (1977), Detsky et al. (1986), and Lee et al. (1999) have become well-known.

RCRI

The Lee index or 'revised cardiac risk' index (RCRI)[3], a modified version of the original Goldman index, was designed to predict major adverse cardiac outcomes (defined as post-operative myocardial infarction, pulmonary oedema, ventricular fibrillation or cardiac arrest, and complete heart block) and until recently was considered by many to be the best and most widely used index in non-cardiac surgery (ref x). It was developed on a cohort of nearly 3000 patients aged >50 years who underwent non-emergent non-cardiac procedures with an expected length of stay 2 or more days at Brigham and Women's Hospital in early 1990s and validated on another cohort.

This risk index comprises six variables:

1. High risk surgery - intra peritoneal, intrathoracic or supra-inguinal vascular
2. History of IHD - history of myocardial infarction, positive exercise test result, current ischemic chest pain or nitrate use, pathological Q waves on ECG (patients who had undergone prior coronary bypass surgery or angioplasty were included only if they had such findings after their procedure)
3. History of heart failure - defined as a history of heart failure, pulmonary oedema, paroxysmal nocturnal dyspnoea; an S3 gallop or bilateral rales on physical examination; or chest radiograph showing pulmonary congestion
4. History of cerebrovascular disease, stroke or transient ischemic attack
5. Pre-operative treatment with insulin
6. Pre-operative creatinine > 175 mmol/L (> 2.0 mg/dL)

Estimated risk of a major adverse cardiac event in based on predictors in the Lee index

No. of risk factors	Risk of major perioperative cardiac event, % (95% CI)
0	0.4 (0.05-1.5)
1	0.9 (0.3-2.1)
2	6.6 (3.9-10.3)
≥3	11.0 (5.8-18.4)

This index is poorer at estimating risk in vascular surgery and especially open aortic procedures.

The following website provides these RCRI risks when the predictors are input:
http://www.mdcalc.com/revised-cardiac-risk-index-for-pre-operative-risk/

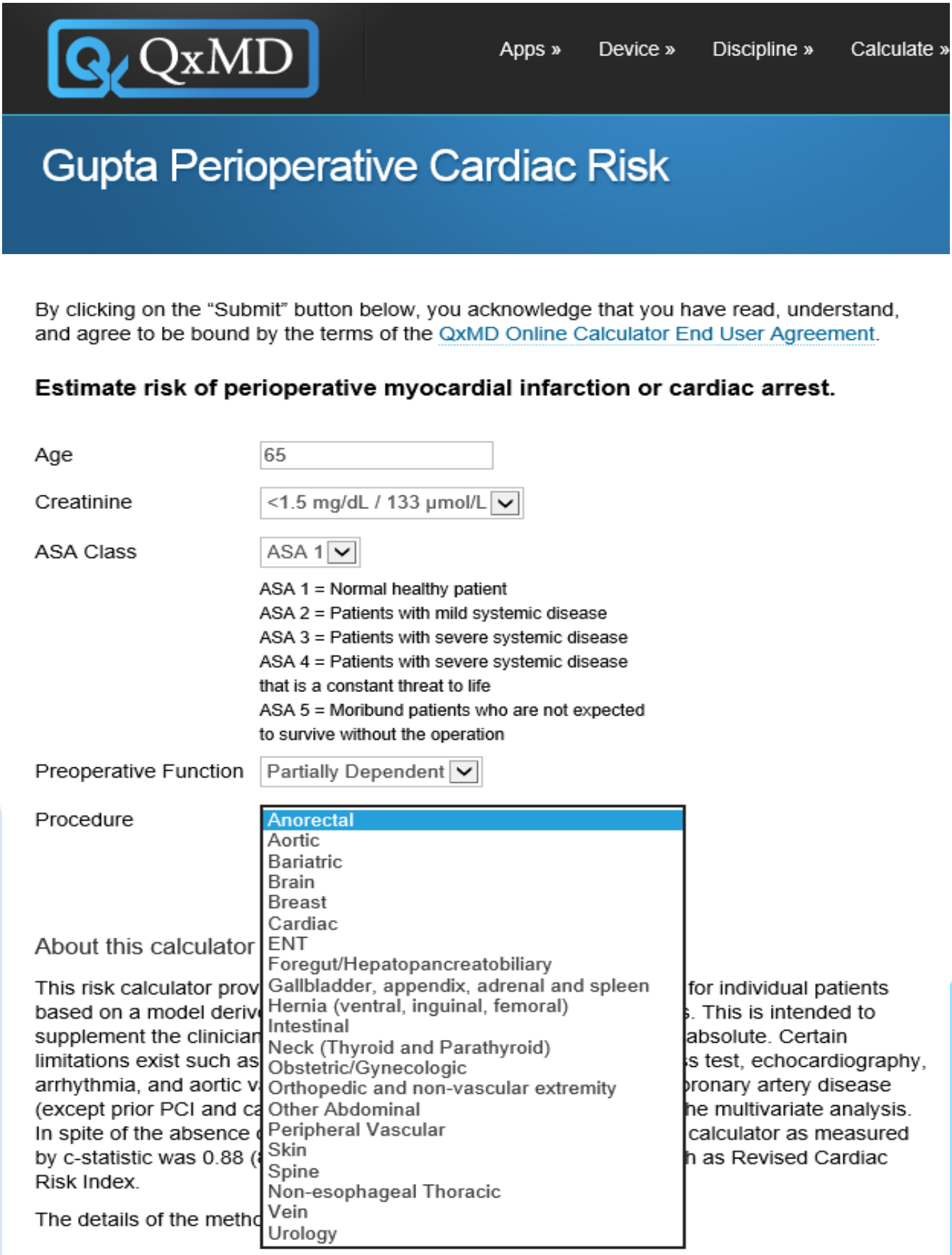
NSQIP MICA model[4]

A new predictive model was recently developed using the American College of Surgeons National Surgical Quality Improvement Program (NSQIP) database. This NSQIP MICA model was built on the 2007 data set, based on patients from 180 hospitals, and was validated with the 2008 data set, both containing 200,000 patients. The primary endpoint was intra-operative/post-operative myocardial infarction or cardiac arrest (MICA) up to 30 days after surgery. Five predictors of perioperative myocardial infarction/cardiac arrest were identified:

1. Type of surgery,
2. Functional status,
3. Elevated creatinine (>130 mmol/L or >1.5 mg/dL),
4. American Society of Anesthesiologists (ASA) Physical Status Classification
5. Age.

This model is presented as an interactive risk calculator at http://www.surgicalriskcalculator.com/miorcardiacarrest. This tool is also available on a mobile app 'Calculate by QxMD'. Unlike the Lee index it is not a simple scoring system, but outputs a probability of MICA for an individual patient depending on values of the 5 variables above.

Figure 2: A screen shot from the NSQIP MICA model website.



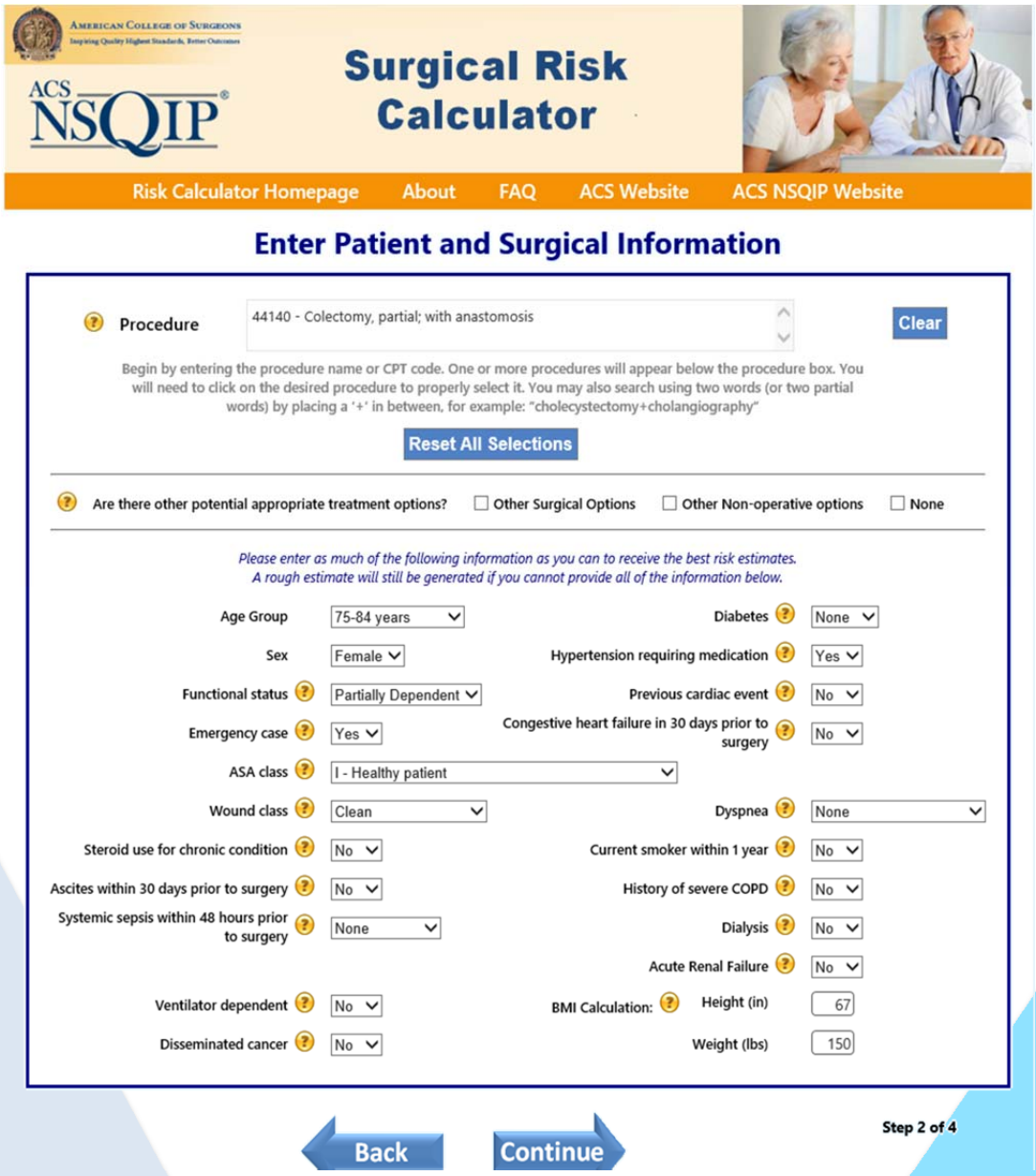
It performed better than the Lee risk index, with some reduction in performance in vascular patients, although it was still superior. However, some perioperative cardiac complications of interest to clinicians, such as pulmonary oedema and complete heart block, were not considered in the NSQIP model because those variables were not included in the NSQIP database. Furthermore daily postoperative ECGs and troponins are not mandated (cf. Lee's study creating the RCRI, and the POISE studies). In other words silent MIs could be missed, so rates may be underestimated.

NSQIP Surgical Risk Calculator[5]

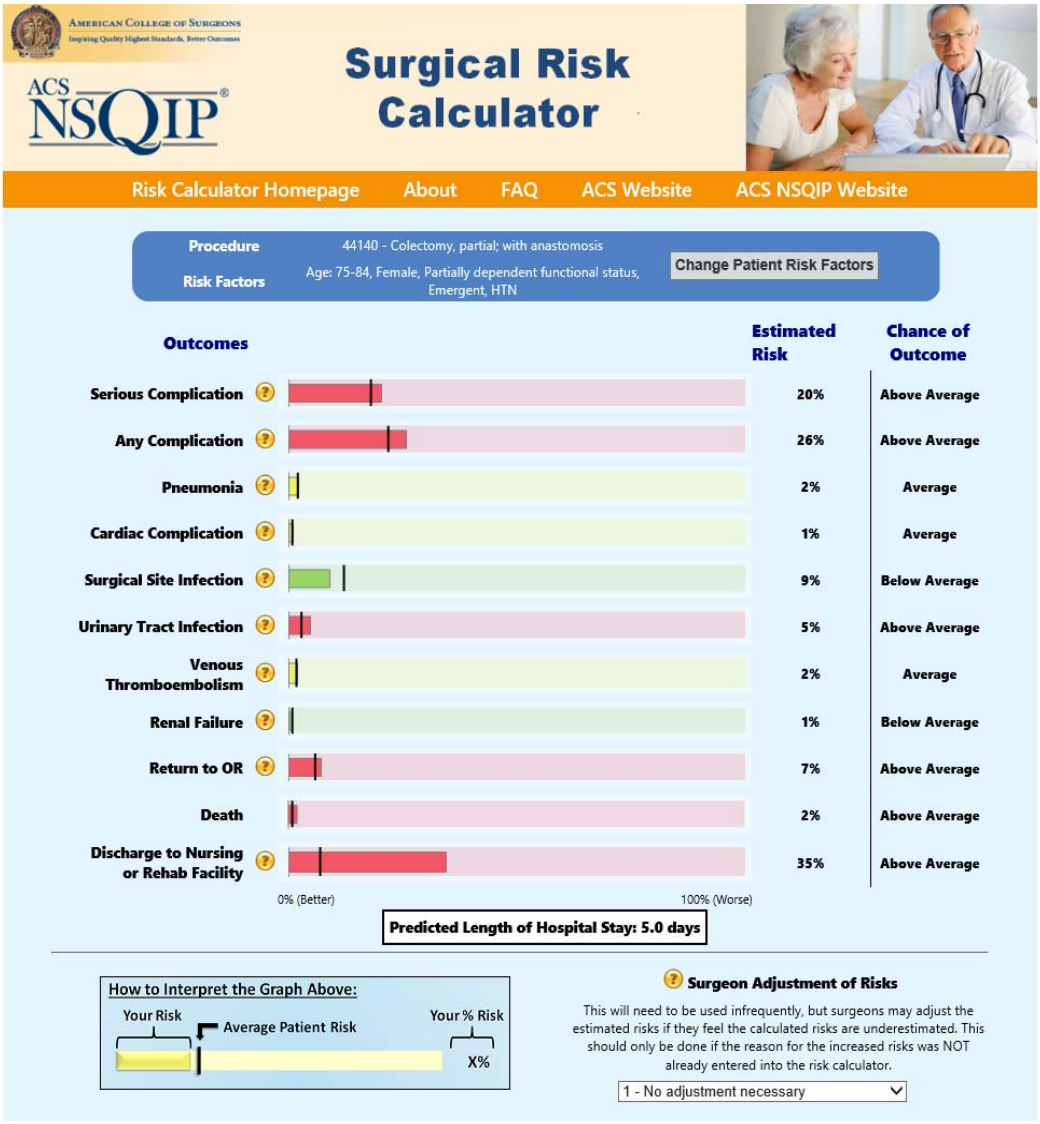
Over 1.4 million patients in the NSQIP database have been used to create probably the best estimator of surgery-specific risk of MICA, death and 8 other adverse outcomes. The NSQIP Surgical Risk Calculator (found at http://riskcalculator.facs.org) calculates these risks based on 21 patient specific variables (eg, age, sex, body mass index, dyspnoea, previous MI, functional status) and the actual operation.

Figures 3: Screenshots of the ACS NSQIP Surgical Risk Calculator website

(A) Risk factor entry screen.



(B) Report screen



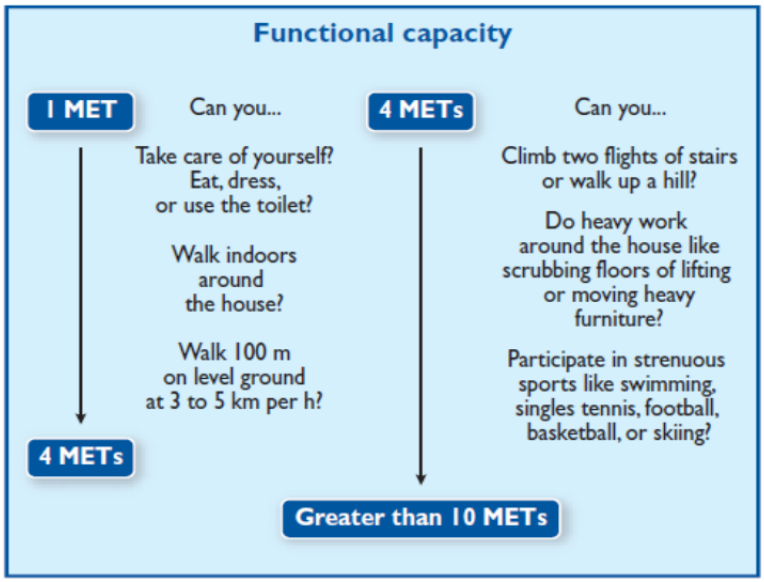
Reference Information for National Surgical Quality Improvement Program (NSQIP) such as data field definitions can be found at the following website:

http://www.google.com.au/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&sqi=2&ved=0CB0QFjAAahUKEwiGtpfO_IPHhXDF5QKHdPSDVE&url=http%3A%2F%2Fbcpsqc.ca%2Fdocuments%2F2012%2F11%2FNSQIP-FAQ-2012-forSurgeons.doc&ei=iq66VYbJEsOv0ATTpbeIBQ&usg=AFQjCNGQah6KT7xmo6HcOPNoOj53Clxhiw&bvm=bv.99261572,d.dGo

Functional status

The two risk calculators above take into account patient's functional capacity as a 3 point scale (independent, partially dependent and totally dependent) and indirectly via the ASA physical status. Furthermore in both the European[6] and American Heart Association guidelines[7] determination of functional capacity is an important step in preoperative cardiac risk assessment algorithms and is measured in metabolic equivalents (METs). One MET equals the basal metabolic rate. Exercise testing provides an objective assessment of functional capacity. Without testing, functional capacity can be estimated from the ability to perform the activities of daily living (see figure 4). Climbing two flights of stairs demands 4 METs, and inability to achieve this indicates poor functional capacity and is associated with an increased incidence of post-operative cardiac events. However, this appears to be a poorer predictor than other inputs into the risk models such as ASA status.

Figure 4: Estimated energy requirements for various activities.



The European guidelines use the grading of surgical risk shown in Figure 5 in their algorithm. The American guidelines use risks estimated from one of the NSQIP models discussed earlier in their most recent algorithm.

Figure 5: Surgical risk estimate

Low-risk: < 1%	Intermediate-risk: 1–5%	High-risk: > 5%
• Superficial surgery • Breast • Dental • Endocrine: thyroid • Eye • Reconstructive • Carotid asymptomatic (CEA or CAS) • Gynaecology: minor • Orthopaedic: minor (meniscectomy) • Urological: minor (transurethral resection of the prostate)	• Intraperitoneal: splenectomy, hiatal hernia repair, cholecystectomy • Carotid symptomatic (CEA or CAS) • Peripheral arterial angioplasty • Endovascular aneurysm repair • Head and neck surgery • Neurological or orthopaedic: major (hip and spine surgery) • Urological or gynaecological: major • Renal transplant • Intra-thoracic: non-major	• Aortic and major vascular surgery • Open lower limb revascularization or amputation or thromboembolectomy • Duodeno-pancreatic surgery • Liver resection, bile duct surgery • Oesophagectomy • Repair of perforated bowel • Adrenal resection • Total cystectomy • Pneumonectomy • Pulmonary or liver transplant

CAS = carotid artery stenting; CEA = carotid endarterectomy.
[a]Surgical risk estimate is a broad approximation of 30-day risk of cardiovascular death and myocardial infarction that takes into account only the specific surgical intervention, without considering the patient's comorbidities.
[b]Adapted from Glance *et al.*[11]

Pre-operative Biomarkers

Cardiac troponins T and I (cTnT and cTnI, respectively) are the preferred markers for the diagnosis of myocardial infarction because they demonstrate sensitivity and tissue specificity better than other available biomarkers. Troponin release and thus "myocardial injury" can be the result of non-coronary factors such as a high adrenergic drive, sepsis, inflammation, myocarditis, renal failure or right ventricular failure secondary from aetiologies such as pulmonary embolism. As more sensitive troponin assays become available it is clear that even normal individuals have detectable levels and elevations above the 99th percentile can be due to structural heart disease in the absence of any acute process. Hence the latest definition of acute MI[8] mandates ischaemic symptoms or ECG changes in addition to troponin levels above the 99th percentile.

Troponin levels detectable by the novel high sensitive troponin assays are 10 times lower than those detected by conventional troponin assays, and even only slightly elevated values have been found to be associated with increased cardiovascular risk. Recent studies demonstrated that among individuals in

the general population and among patients with chronic vascular disease, measurable circulating TnT and TnI levels reflect chronic sources of subclinical myocardial injury, rather than acute processes, and predict long-term adverse cardiac outcomes and death.

Existing evidence suggests that even small increases in cTnT in the peri-operative period reflect clinically relevant myocardial injury with worsened cardiac prognosis and outcome. In one series of 1,041 consecutive patients undergoing endovascular peripheral revascularization measurable cTnT levels (> 0.01 ng/ml) were detected pre-operatively in 21.3% of individuals and were strongly associated with adverse cardiovascular event rates and mortality, independent of impaired renal function[9]. In another series of 979 patients undergoing non-cardiac surgery pre-operative hsTnT levels above 0.014 g/ml provided strong prognostic information incremental to Lee's RCRI[10].

B-type natriuretic peptide (BNP) and N-terminal pro-BNP (NT-proBNP) are produced in cardiac myocytes in response to increases in myocardial wall stress. This may occur at any stage of heart failure, independently of the presence or absence of myocardial ischaemia. Multiple studies[11-14] have demonstrated that elevated pre-operative NP concentrations are powerful independent predictors of perioperative cardiovascular complications (i.e. mortality, MI and heart failure). In vascular surgical cases, it appears pre-operative BNP risk stratification outperforms traditional clinical risk stratification[15].

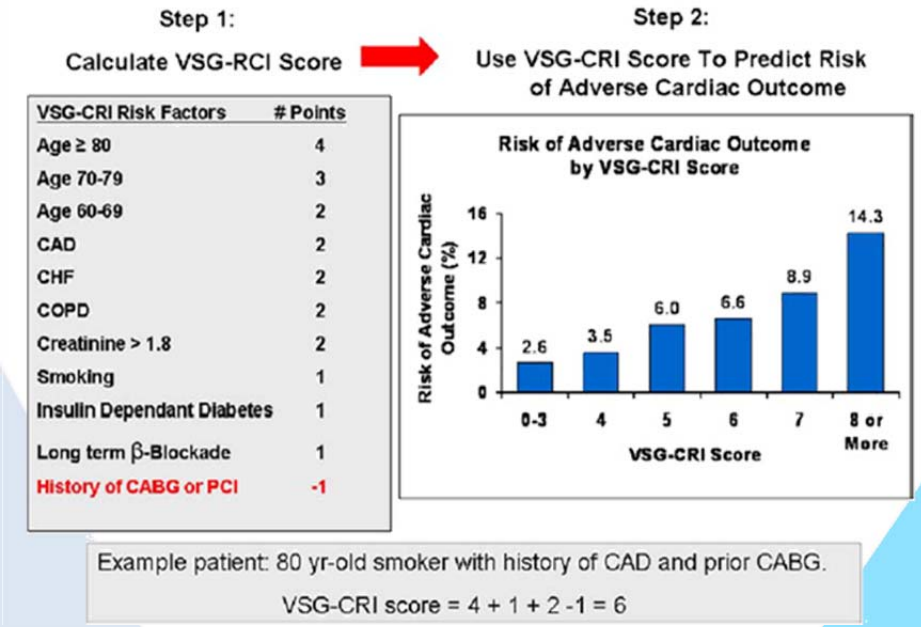
American and European guidelines suggest that based on the existing data, assessment of serum biomarkers for patients undergoing non-cardiac surgery cannot be proposed for routine use, but may be considered in high-risk patients.

Miscellaneous

Frailty is an increasingly used concept in the geriatric medicine and a number of 'frailty scores' exist. Increasing frailty has been associated with increased complications (including cardiac) and length of hospital stays in medically stable older adults undergoing non-cardiac surgery[16].

Although not mentioned in the American or European guidelines the Vascular Study group of New England developed a cardiac risk index based on over 10,000 patients undergoing vascular surgery[17] (see figure 6). This Vascular Study Group of New England Cardiac Risk Index (VSG-CRI) more accurately predicts in-hospital cardiac events after vascular surgery than the RCRI which substantially underestimates in-hospital cardiac events in patients undergoing elective or urgent vascular surgery, especially after lower extremity bypass, endovascular abdominal aortic aneurysm repair and open infrarenal abdominal aortic aneurysm repair.

Figure 6: Vascular Surgery Group Cardiac Risk Index



Intra-operative risk prediction

Anaesthesia providers have long assumed that a smooth intra-operative course is associated with better postoperative outcomes. Tachycardia[2], hypotension[18], bleeding [2,18], anaemia and transfusion have all been independently associated with adverse cardiac events. However they may just reflect patient characteristics or the physiological impact of surgery and may not be amenable to modification.

Two recent large studies have looked at impact of hypotension on outcomes. Monk et al[19] (over 18,000 patients) found that SBP < 70 mmHg, MAP < 50 mmHg, and DBP < 30 mmHg for over 5 minutes, were associated with increased 30 day mortality. In over 100,000 patients Mascha et al[20] found that cumulative duration of MAP less than 50, 55, 60, 70, and 80 mmHg was associated with increased odds of 30-day mortality.

Gawende et al[21] published an interesting approach to using intra-operative parameters to predict adverse outcomes. They developed a 10-point (Apgar like) score based on a patient's estimated amount of blood loss, lowest heart rate, and lowest mean arterial pressure during general or vascular operations. This score (see figure 7) was significantly associated with major complications or death within 30 days. For example, those with scores of 4 or less had a greater than 50% risk of major complications, including a 14% mortality rate.

Figure 7: A 10-Point Surgical Outcomes Score

	0 points	1 point	2 points	3 points	4 points
Estimated blood loss (mL)	> 1,000	601–1,000	101–600	≤ 100	—
Lowest mean arterial pressure (mmHg)	< 40	40–54	55–69	≥ 70	—
Lowest heart rate (beats/min)	> 85	76–85	66–75	56–65	≤ 55†

Surgical score = sum of the points for each category in the course of a procedure.
*Based on model 1 from cohort 1.
†Occurrence of pathologic bradyarrhythmia, including sinus arrest, atrioventricular block or dissociation, junctional or ventricular escape rhythms, and asystole also receive 0 pts for lowest heart rate.

Post-operative prediction

Increases in postoperative levels of troponins and BNP are both independently associated with adverse cardiac outcomes and mortality.

Troponins

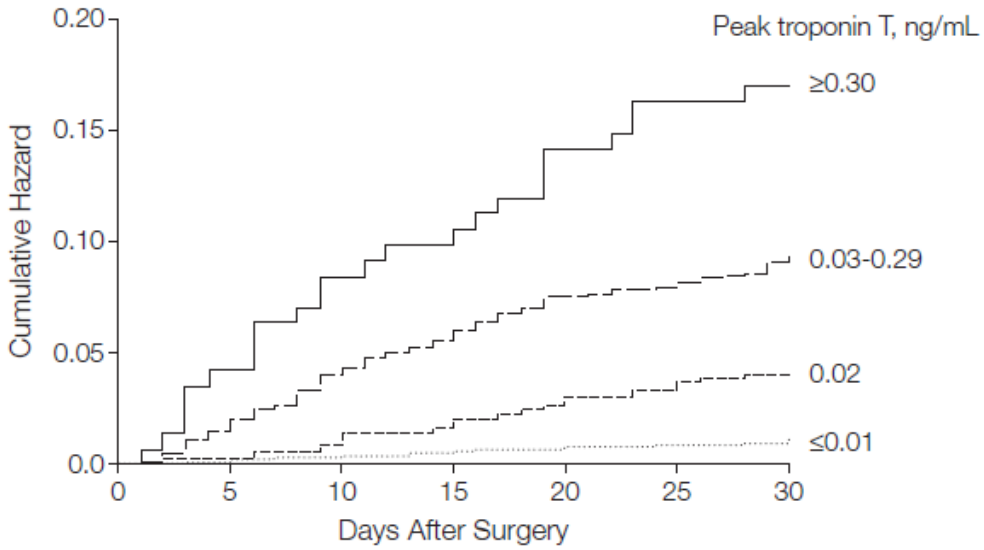
Many studies have confirmed the association between elevated troponin levels and adverse outcomes in a variety of settings, however the results from the first 15,000 patients in the VISION study have provided the most extensive and generalisable information regarding postoperative troponins and adverse vascular outcomes and mortality.

The VISION Study (Vascular Events in Non-cardiac Surgery Patients Cohort Evaluation) [22] is a large international prospective study evaluating major complications after non-cardiac surgery. Eligible patients are aged 45 and over undergoing non-cardiac surgery requiring at least overnight admission. Patients' TnT levels were measured 6 to 12 hours after surgery and on days 1, 2, and 3 after surgery. 24 pre-operative variables were documented and there were daily clinical assessments. The primary outcome was mortality at 30 days and whether the cause was vascular or non-vascular. 45% of deaths were vascular. They found that Peak TnT measurement added incremental prognostic value (to the clinical variables) to discriminate those likely to die within 30 days for both vascular and nonvascular mortality. Peak TnT values after non-cardiac surgery proved the strongest predictors of 30-day mortality, and the population attributable risk analysis suggested elevated TnT measurements after surgery may explain 41.8% of the deaths.

Multivariable analysis demonstrated that peak TnT threshold values of 0.02 ng/mL, 0.03 ng/mL, and 0.30 ng/mL were independently associated with 30-day mortality (the commonly accepted upper limit for this assay is 0.04 ng/ml). See table below and figure 8.

Peak TnT measurement	% of cohort	30 day mortality	30 day vascular mortality	Time to death
≤ 0.01 ng/ml	88.4%	1%	0.4%	
0.02 ng/ml	3.3%	4.0%	1.4%	13 days
0.03 - 0.29 ng/ml	7.4%	9.3%	4.5%	9 days
≥ 0.3 ng/ml	0.9%	16.9%	9.2%	6.5 days

Figure 8: Kaplan-Meier Estimates of 30-Day Mortality Based on Peak Troponin T Values



Among patients who experienced a TnT elevation of 0.02 ng/mL or greater, this occurred at 6to12 hours after surgery, post- operative day 1, postoperative day 2,and postoperative day 3 in 45.9%, 28.3%, 17.7%, and 8.2% of these patients, respectively

A weakness of the VISION study was that they did not measure pre-operative troponins so cannot assess how a pre- operative value would impact risk prediction.

The VISION investigators published a follow-up paper [23] in which they used the VISION data to determine properties of "Myocardial Injury after Non-cardiac Surgery" (MINS) which they defined as prognostically relevant myocardial injury due to ischaemia. To do this they analysed the ischaemic symptoms and ECGs of patients who has a troponin level above 0.04 ng/ml (elevated 'abnormal' lab threshold). They also excluded 95 patients who were adjudicated to have a non-ischaemic cause for their troponin rise (e.g. sepsis, pulmonary embolism, cardioversion). Multivariable analysis (including preoperative predictors) revealed that an elevated troponin (≥ 0.03 ng/ml) after non-cardiac surgery, irrespective of the presence of an ischemic symptom or ECG, independently predicted 30-day mortality. Therefore, the authors' diagnostic criterion for MINS was a peak troponin T level of 0.03 ng/ml or greater judged due to myocardial ischemia (i.e. not sepsis etc.). MINS was an independent predictor of 30-day mortality (adjusted hazard ratio, 3.87; 95% CI, 2.96–5.08). Twelve hundred patients (8.0%) suffered MINS, and 58.2% of these patients would not have fulfilled the universal definition of myocardial infarction. Only 15.8% of patients with MINS experienced an ischemic symptom. 9.8% of MINS patients died within 30 days of surgery and an additional 9% had a non-fatal cardiac arrest, stroke or congestive cardiac failure.

BNP

Using individual patient data meta-analysis (on over 2000 patients) Reitze N et al[24] found that an elevated postoperative BNP independently associated with increased mortality, myocardial infarction, and cardiac failure at 30 days and more than 180 days after non-cardiac surgery,

In a further analysis Rodseth RN et al[25] found that post-operative BNP measurement enhanced risk stratification for the composite outcomes of death or nonfatal myocardial infarction at 30 days and more than 180 days after non-cardiac surgery compared with a pre-operative BNP measurement alone.

However the extent to which post-operative NP elevation correlates with post-operative troponin elevation and whether measuring NP provides additional information to that provided by post-operative troponin alone is unknown.

What can we do?

There is no good evidence for a safe and effective way to prevent postoperative cardiac complications or how to treat them and improve their long term prognosis. However recent large trials have clarified some issues regarding preoperative management. Clonidine is of no benefit and increases risk of hypotension (POISE-2)[18]. Aspirin is of no benefit and increases risk of bleeding (POISE-2[26]). It should be stopped prior to surgery unless minimal bleeding risk and major thrombosis risk. In general aspirin should be continued in patients with stents and recent infarcts.

Evidence regarding statins is still evolving but AHA and European guidelines suggest continuing statins in patients currently taking them and that the pre-operative initiation of statins is reasonable in patients undergoing vascular surgery and maybe other high risk situations.

A recent review of perioperative beta-blockade[27] concluded that beta blockade started within 1 day or less before non-cardiac surgery prevents nonfatal MI but increases risks of stroke, death, hypotension, and bradycardia. Without the controversial DECREASE studies, there are insufficient data on beta blockade started 2 or more days prior to surgery. Multicenter RCTs are needed to address this knowledge gap. AHA and European guidelines state that pre-existing beta-blockade should be continued and suggest that they may be initiated in high risk situations (high risk patient, high risk surgery, tachycardia) in a titrated manner.

Nitrous oxide has been cleared of increasing cardiovascular risk (ENIGMA 2).

The VISION study has confirmed the importance of measuring post-operative troponin levels to identify a poor short and long term prognosis population for which preventative strategies must be studied.

Foucrier et al[28] performed an interesting (non-randomised) study in which they compared one year freedom from adverse cardiac events in vascular surgery patients who had a peri-operative MI or significant troponin rise and had intensification of their previous medical therapy (addition of anti-platelet drug, statin, beta blocker or ACE inhibitor) to a group who had no change in their medical therapy following their MI / troponin rise. Patients not receiving intensified cardiovascular therapy had a higher risk of an adverse cardiac event compared to patients with no MI / troponin rise and those whose therapy had been intensified. Patients whose therapy was intensified had similar risk to those who had no MI / troponin rise. They concluded that in patients with elevated troponin I levels after non-cardiac surgery, long-term adverse cardiac outcomes may likely be improved by following evidence-based recommendations for the medical management of acute coronary syndromes.

Another recent study (non-randomised)[29], which reinforced the potential importance of optimal post-operative patient management, found that postoperative delay in resuming angiotensin receptor blockers (ARBs) is common, particularly in patients who are frail after surgery. However withholding ARBs was strongly associated with increased 30-day mortality.

Devereuax et al[2] also found that a substantial proportion of patients with a peri-operative MI did not receive cardiovascular medications known to be effective in managing patients with non-operative MI.

The issue of how to treat postoperative MI / troponin elevations is being studied in a large multicentre trial: Management of Myocardial Injury After Non-cardiac Surgery Trial (MANAGE). This was started in 2013 and expected to be completed by 2017. This is a factorial design in which patients who have suffered an MI or an elevated troponin measurement after surgery with no alternative explanation (e.g., pulmonary embolism, sepsis) to myocardial injury are being randomised to placebo, omeprazole (20 mg daily), dabigatran (110 mg bd) or both.

There have also been some promising small trials[30] on the pre-operative use of the anti-anginal drug, ivabradine. This is a specific inhibitor of the I_f current in sinoatrial node myocytes which reduces heart rate independently of sympathetic activation and does not cause hypotension. A large randomised trial is required.

Finally I would just to bring you attention to a recently published study. Using clinical data from the New York State Cardiac Surgery Reporting System Glance et al[31] found that the rate of death or major complications among patients undergoing coronary artery bypass graft surgery varies markedly across anesthesiologists , controlling for confounders such as patient factors and hospital quality. These findings suggest that there may be intra-operative opportunities to improve outcomes among high-risk surgical patients. This study did not collect details of peri-operative management so which aspects of anaesthesia management may have impacted outcomes is unknown.

Summary

The following 3 recommendations from the European guidelines summarise much of the material in this literature review:

1. Clinical risk indices such as the RCRI or NSQIP model are recommended for peri-operative risk stratification.
2. Assessment of cardiac troponins in high risk patients, both before, and 48-72 hours after major surgery should be considered.
3. NT-proBNP and BNP measurements may be considered for obtaining independent prognostic information for perioperative and late cardiac events in high-risk patients.

There is promising data from existing studies and extensive on-going research regarding interventions to improve peri-operative and longer term cardiac outcomes in our high risk patients.

Statistical note

Many papers quote a "c statistic" of "C-statistic" to compare models or risk indices. The c statistic, is a measure of how well a model can be used to discriminate subjects having the event from subjects not having the event. Values range from 0.5 to 1.0. A value of 0.5 indicates that the model is no better than chance at predicting the outcome. A value of 1.0 indicates that the model perfectly identifies those with and without the outcome. Models are typically considered reasonable when the c-statistic is higher than 0.7 and strong when it exceeds 0.8. The c-statistic is the same as the area under the receiver-operating characteristic curve, formed by taking the predicted values from the regression model as a diagnostic test for the outcome event in the data.

References

1. Devereaux PJ, Yang H,Yusuf S, POISE Study Group. Effects of extended-release metoprolol succinate in patients undergoing non-cardiac surgery (POISE trial): a randomised controlled trial. Lancet 2008; 371: 1839-47.
2. Devereaux PJ; Xavier D; Pogue J; Guyatt G; POISE (PeriOperative ISchemic Evaluation) Investigators. Characteristics and short-term prognosis of perioperative myocardial infarction in patients undergoing noncardiac surgery: a cohort study. Annals Intern Med 2011; 154: 523-8.
3. Lee TH, Marcantonio ER, Mangione CM, Thomas EJ et al. Derivation and prospective validation of a simple index for prediction of cardiac riskof major noncardiac surgery. Circulation 1999; 100: 1043–1049.
4. Gupta PK, Gupta H, Sundaram A et al. Development and validation of a risk calculator for prediction of cardiac risk after surgery. Circulation. 2011; 124: 381-7.
5. Karl Y Bilimoria, Yaoming Liu, Jennifer L Paruch et al. Development and Evaluation of the Universal ACS NSQIP Surgical Risk Calculator: A Decision Aid and Informed Consent Tool for Patients and Surgeons. J Am Coll Surg 2013; 217: 833-842.
6. 2014 ESC/ESA Guidelines on non-cardiac surgery: cardiovascular assessment and management European Heart Journal 2014; 35: 2383–2431.
7. 2014 ACC/AHA Guideline on Perioperative Cardiovascular Evaluation and Management of Patients Undergoing Noncardiac Surgery: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation 2014; 130: e278-e333.
8. Third Universal Definition of Myocardial Infarction Consensus Statement. Circulation 2012; 126: 2020-2035
9. Birgit Linnemann, Thilo Sutter, Eva Herrmann ET AL. Elevated Cardiac Troponin T Is Associated With Higher Mortality and Amputation Rates in Patients With Peripheral Arterial Disease. J Am Coll Cardiol 2014; 63: 1529–38
10. Weber M, Luchner A, Seeberger M et al. Incremental value of high-sensitive troponinT in addition to the revised cardiac index for peri-operative risk stratification in non-cardiac surgery. Eur Heart J 2013; 34: 853–862.
11. Karthikeyan G, Moncur RA, Levine O et al. Is a pre-operative brain natriuretic peptide or N-terminal pro-B-type natriuretic peptide measurement an independent predictor of adverse cardiovascular outcomes within 30 days of noncardiac surgery. A systematic review and meta-analysis of observational studies. J Am Coll Cardiol 2009;54:1599–1606.
12. Rodseth RN, Lurati Buse GA, Bolliger D, et al. The predictive ability of pre-operative B-type natriuretic peptide in vascular patients for major adverse cardiac events: an individual patient data meta-analysis. J Am Coll Cardiol 2011; 58: 522–9.
13. Farzi S, Stojakovic T, Marko T, et al. Role of N-terminal pro B-type natriuretic peptide in identifying patients at high risk for adverse outcome after emergent non-cardiac surgery. Br J Anaesth. 2013; 110: 554–60.
14. Editorial by Fox, Amanda. Perioperative B-type Natriuretic Peptide/N-terminal pro-B-type Natriuretic Peptide Next Steps to Clinical Practice. Anesthesiology 2015; 123:246-8.
15. Biccard BM, Lurati Buse GA, Burkhart C et al. The influence of clinical risk factors on pre-operative B-type natriuretic peptide risk stratification of vascular surgical patients. Anaesthesia 2012; 67: 55–9.
16. Monidipa Dasgupta, Darryl B. Rolfson , Paul Stolee et al. Frailty is associated with postoperative complications in older adults with medical problems. Archives of Gerontology and Geriatrics 2009; 48: 78–8316.
17. Bertges DJ, Goodney PP, Zhao Y et al. The Vascular Study Group of New England Cardiac Risk Index (VSG-CRI) predicts cardiac complications more accurately than the Revised Cardiac Risk Index in vascular surgery patients, Vascular Study Group of New England. J Vasc Surg 2010; 52: 5-12.
18. Devereaux PJ; Sessler DI; Leslie K et al. Clonidine in patients undergoing noncardiac surgery. POISE-2 Investigators. N Engl J Med 2014; 370:1504-13.
19. Michael Walsh, Philip J. Devereaux, Amit X. Garg, et al. Relationship between Intraoperative Mean Arterial Pressure and Clinical Outcomes after Noncardiac Surgery Toward an Empirical Definition of Hypotension. Anesthesiology 2013; 119: 507-15.
20. Terri G. Monk, Michael R. Bronsert, William G. Henderson et al. Association between Intraoperative Hypotension and Hypertension and 30-day Postoperative Mortality in Noncardiac Surgery. Anesthesiology 2015; 123: 307-19.

21. Edward J. Mascha, Dongsheng Yang, Stephanie Weiss et al. Intraoperative Mean Arterial Pressure Variability and 30-day Mortality in Patients Having Noncardiac Surgery. Anesthesiology 2015; 123: 79-91.
22. Atul A Gawande, Mary R Kwaan, Scott E et al. An Apgar Score for Surgery. J Am Coll Surg 2007; 204:201–208.
23. The Vascular Events In Noncardiac Surgery Patients Cohort Evaluation (VISION) Study Investigators. Association Between Postoperative Troponin Levels and 30-Day Mortality Among Patients Undergoing Noncardiac Surgery. JAMA 2012; 307: 2295-2304.
24. The Vascular Events In Noncardiac Surgery Patients Cohort Evaluation (VISION) Study Investigators. Myocardial Injury after Noncardiac Surgery: A Large, International, Prospective Cohort Study Establishing Diagnostic Criteria, Characteristics, Predictors, and 30-day Outcomes. Anesthesiology 2014; 120: 564-78.
25. Reitze N. Rodseth, Bruce M. Biccard, Rong Chu et al. B-type Natriuretic Peptide for Prediction of Major Cardiac Events in Patients Undergoing Noncardiac Surgery: Systematic Review and Individual Patient Meta-analysis. Anesthesiology 2013; 119: 270-83.
26. Rodseth RN, Biccard BM, Le MY, et al. The prognostic value of preoperative and post-operative B-type natriuretic peptides in patients undergoing noncardiac surgery: B-type natriuretic peptide and N-terminal fragment of pro-B-type natriuretic peptide: a systematic review and individual patient data meta-analysis. J Am Coll Cardiol. 2014; 63: 170–80.
27. Devereaux PJ, Mrkobrada M, Seesler DI et al. Aspirin in patients undergoing noncardiac surgery. POISE-2 Investigators. N Engl J Med, 2014; 370:1494-1503.
28. Perioperative Beta Blockade in Noncardiac Surgery: A Systematic Review for the 2014 ACC/AHA Guideline on Perioperative Cardiovascular Evaluation and Management of Patients Undergoing Noncardiac Surgery. Circulation 2014; 130: 2246-2264.
29. Foucrier A, Rodseth R, Aissaoui M et al. The long-term impact of early cardiovascular therapy intensification for postoperative troponin elevation after major vascular surgery. Anesth Analg 2014; 119: 1053–63.
30. Susan M. Lee, Steven Takemoto, Arthur W. Wallace. Association between Withholding Angiotensin Receptor Blockers in the Early Postoperative Period and 30-day Mortality. Anesthesiology 2015; 123: 288-306.
31. Patrizia Lo Sapio , Gian Franco Gensini, Sergio Bevilacqua et al. The role of ivabradine in the incidence of perioperative coronary complication in patients undergoing vascular surgery Int J of Cardiology 2013; 168: 4352–4353.
32. Laurent G. Glance, Arthur L. Kellermann, Edward L. Hannan et al. The Impact of Anesthesiologists on Coronary Artery Bypass Graft Surgery Outcomes. Anesth Analg 2015; 120: 526–33.

Anticoagulants for Anaesthetists… 2015 Update

Laura Young

Haematology Department, Auckland City Hospital

The uncertainties of management of emergency surgery or acute bleeding in patients taking NOACs (non-vitamin K oral anticoagulants) are finally becoming clearer. Whereas we previously have debated the use of various prohaemostatic agents, now direct reversal strategies are in phase III clinical trials. The front runner is idarucizumab which is a monoclonal antibody that directly binds irreversibly to dabigatran. A large number of New Zealand patients have been included in the recently published initial results and the local clinical experience of use of this agent is positive. Andexanet is a decoy factor X molecule which aims to reverse all Xa inhibitors, both direct and indirect. Phase III data was recently presented in abstract form. In the meantime for Xa inhibitor patients (such as rivaroxaban) we continue to recommend standard measures and possibly prothrombinex, acknowledging the limited data available.

Finally in elective surgery, the use of bridging strategies for those taking warfarin will be discussed. A clinical trial comparing no bridging versus bridging with low molecular weight heparin in warfarinised patients with intermediate to high risk AF was published last month and did not show any reduction in stroke, at the expense of increased bleeding complications, with dalteparin. A simplified elective surgical algorithm recently developed for Auckland City Hospital will be presented.

Atom bombs, circle circuits, and CO_2: links between diving and anaesthesia

Simon Mitchell

Department of Anaesthesia, Auckland City Hospital

There are many reasons why the specialties of anaesthesia and diving medicine are complimentary, not least of which is the strong relevance of respiratory physiology to both fields. This presentation will describe several of our recent attempts to answer questions relating to carbon dioxide (CO_2) homeostasis that are both of high practical relevance to the diving community and of potential interest to anaesthetists interested in the related physiology.

Prologue: Atom bombs

It was known in the 1930s that fluorination of anaesthetic drugs would produce less soluble (but nevertheless potent) agents that would be more stable and less metabolised. However, fluorine halogenation is technically difficult and fluorine chemistry was insufficiently advanced to produce such compounds. The impetus for advancement occurred during the Second World War for two reasons [1]. First, hydrogen fluoride could be used to accelerate the reactive steps in producing high octane aviation fuel. Second (and more importantly) uranium hexafluoride was found to be an obligatory reactant in the production of enriched uranium. The means of producing the former was developed as part of the secret Manhattan Project tasked to develop the atomic bomb. The fluorine chemistry component of this work was of great interest to (and partly funded by) drug companies. A number of potential anaesthetic agents were synthesised at the time, but none were suitable for use in humans. Nevertheless, this work indisputably accelerated development of Halothane which emerged in clinical use in 1951.

The Manhattan Project was successful in developing a nuclear weapon. The first detonation of an atomic bomb took place in the Nevada Desert on 16 July 1945, and the Hiroshima bomb was the second ever nuclear explosion (and regrettably the first use of a nuclear weapon on a human target), taking place only weeks later on 6 August. Nagasaki was the third nuclear explosion on 9 August.
The link with diving came after an interlude in nuclear testing following the war. Testing was resumed in 1946 with the fourth and fifth explosions both taking place at Bikini Atoll in the Pacific Ocean. These tests were designed to evaluate the effect of a nuclear blast on surface ships, and a large fleet of captured or surplus warships were anchored in the blast zone. In the end, only 19 of over 70 ships sank at Bikini.

These ships, lying in the sheltered enclosed waters of the Atoll, are now collectively considered one of the most iconic wreck diving sites on the planet, but getting to Bikini and diving the wrecks is logistically difficult and a true expedition-style undertaking. Of greatest interest among the wrecks is the aircraft carrier USS Saratoga. She is sitting intact and upright on the bottom at 52m depth. The flight deck is at 23m. We have explored the Saratoga extensively. It is a dangerous, silty, no-light environment inside the wreck which is becoming progressively unstable. Traversing the corridors must be undertaken with great care because there are numerous entrapment hazards, not least of which is the potential for silting and loss of visibility as a consequence of indiscriminate use of fins. One of our targets (understandably) has been the Sick Bay / Dental Surgery area which is down several decks and a long corridor traverse from the nearest entrance.

Circle circuits

Exploration of the Bikini wrecks is undertaken almost exclusively using underwater breathing apparatus based on circle circuits. Just as in an anaesthetic machine, these so-called "rebreather" devices recycle expired gas through a carbon dioxide absorbent and into a counter-lung from which it is subsequently re-inhaled. Oxygen levels in the circuit are typically maintained by a system incorporating galvanic fuel cells to measure the PO2 in the circulating gas. When the PO2 falls below a user-specified threshold, a microprocessor opens an electronic valve allowing more oxygen to flow into the circuit until the PO2 is restored. Gas is also added from a cylinder of nitrogen or helium-containing "diluent" gas during increases in depth to maintain the volume of the circuit (and to dilute the oxygen) as water pressure increases, but once at the target depth the only gas consumed is the exact amount of oxygen metabolised by the diver. The rebreather can therefore be seen as the ultimate low flow anaesthetic machine (without the anaesthetic), and one of their great advantages is parsimony with gas supply [2].
Rebreathers are complex devices with many failure points, and there are many accidents. Gas-related accidents are designated by "the three H's": hypoxia, hyperoxia, and hypercapnia. This presentation will focus on the hazard of hypercapnia. Hypercapnia in diving is undesirable because it can, of itself, cause unpleasant and debilitating symptoms of dyspnoea, headache, confusion, anxiety, and panic. In addition, hypercapnia is synergistic with nitrogen in producing narcosis, and it markedly increases the risk of cerebral oxygen toxicity during typical exposures to the elevated inspired PO2s typically chosen for deep dives; probably because of cerebral vasodilation with a consequent increase in the oxygen "dose" delivered to the brain.

The presence of a CO2 absorbent in a rebreather (commonly referred to in diving circles as a CO2 "scrubber") is an obvious failure point that, if faulty, expired, or incorrectly installed might precipitate hypercapnia due to CO2 rebreathing. Anaesthetists generally rely on their assistants to replace the CO2 absorbent in a timely manner, and medical absorbent material often reminds of the need for this by changing colour as it becomes consumed. In addition, the use of capnography provides a real time means to readily identify a CO2 absorbent failure. This would be indicated by failure of the capnography trace to return to zero during the inhalation phase of the respiratory cycle. Rebreather divers cannot see their CO2 absorbent so indicator material is no use, and although inhaled CO2 detection devices have been produced, their use is not yet widespread. Thus, the mainstay of prevention of CO2 rebreathing in rebreather diving is adherence to some simple rules and procedures. The rules include: checking the one way valve operation in the rebreather mouthpiece prior to assembly; packing the absorbent material into the canister meticulously; and installing the canister correctly; discarding the absorbent material after its recommended usage period (typically 3 – 4 hours); and "pre-breathing" the rebreather after assembly but before diving.

Pre-breathing a rebreather loop

The pre-breathe procedure has been of interest to us. Every rebreather diver is trained to sit quietly and breathe on the loop for 5 minutes prior to entering the water. This serves multiple purposes, but the one most heavily emphasised (and the reason for the recommended 5 minute duration) is based on the assumption that if there is a problem with CO2 rebreathing the prebreathe will result in symptoms of hypercapnia (such as shortness of breath) thus warning the diver not to proceed with the dive. This assumption had never been tested formally.

We conducted a study in which divers were randomised to perform a 5 minute prebreathe on a loop with a normal scrubber, no scrubber, or a scrubber with a fault allowing partial rebreathing of exhaled gas. The subjects were: blinded to the scrubber condition; instructed to treat the prebreathe as normal; and told to terminate the prebreathe before the 5 minutes was complete if they developed symptoms of hypercapnia (which they were reminded of). We monitored inspired and end tidal CO2, tidal volume, respiratory rate, and minute volume in all prebreathes. Despite the obvious expectation associated with participation, none of 20 subjects terminated when prebreathing with the normal scrubber. Only 2 of 20 subjects terminated when prebreathing with the partial fault despite the P_ICO2 being approximately 20mmHg. Fifteen of 20 subjects terminated when prebreathing with the absent scrubber, but remarkably 5 did not terminate, despite a $P_{ET}CO2$ in the 60s. We concluded that the 5 minute prebreathe is not a sensitive test for scrubber problems; particularly in the partial failure condition [3].

Hypercapnia caused by deranged control of ventilation

All FANZCA Part 1 examination candidates would be familiar with the simple relationship between some measure of CO2 state (alveolar CO2 or arterial CO2), CO2 production and alveolar ventilation, as described by the simple equation:
PACO2 = VCO2 / VA

Since VCO2 is effectively uncontrollable (unless physical activity is intentionally moderated) this equation exemplifies the critical role of control of ventilation in CO2 homeostasis. Under normal circumstances ventilation is automatically regulated by the respiratory controller, and not surprisingly the classical VE / VCO2 curve (in which ventilation is plotted against either CO2 production, arterial CO2 or end tidal CO2) appears as a straight upwardly sloping line; thus, as arterial CO2 increases, so does ventilation in an attempt to bring the arterial CO2 back to normal. In anaesthetic physiology teaching we have become familiar with the concepts that the curve can be shifted to the right and its slope reduced by drugs (particularly opiates) and hyperoxia, and shifted to the left by hypoxia.

Diving physiologists have become familiar with other fascinating phenomena in relation to this curve; in particular, the substantial inter-personal variability that can be masked by population curves, and the fact that this variability is maximally unmasked by increases in the work of breathing, particularly during exercise. Thus, in the face of an increase in work of breathing, it is as though the respiratory controller has a choice either to drive more work to maintain CO2 homeostasis, or to avoid the work and allow the arterial CO2 to drift upwards. Since the use of underwater breathing apparatus imposes an increase in the work of breathing (particularly at deep depths when the respired gas is dense) hypercapnia due to inadequate ventilation (sometimes referred to as "CO2 retention") is a well-recognised phenomena [4]. Importantly, this mechanism is distinct from hypercapnia caused by CO2 scrubber failure and consequent CO2 rebreathing although the two problems can interact; if a rebreather diver with a tendency to retain CO2 is breathing dense gas at depth and there is CO2 breaking through the scrubber, then dangerous hypercapnia will develop more quickly. We saw relevant examples in our prebreathe study described above. The divers were not exercising and at atmospheric pressure the gas was not abnormally dense, but the rebreather as configured for the experiment (including an anaesthetic antibacterial filter) did impose a higher work of breathing than normal. Among the subjects exposed to the absent scrubber condition some increased their ventilation substantially as their end tidal CO2 rose, whereas others did not increase ventilation at all even when their end tidal CO2 increased to greater than 60 mmHg.

Guidance on tolerable gas density

In view of the predilection for CO2 retention among some divers as work of breathing increases it is surprising that there are no published guidelines for gas density in planning deep dives. This is important because it is a variable that divers can manipulate by including more helium (a light, non-narcotic but expensive gas) in their breathing mix. Although the database was not compiled specifically for this purpose, we are about to publish an analysis of outcomes for a database of human test dives utilising various underwater breathing apparatus operated with gases of different density on a graded exercise protocol [5]. These tests, performed at a UK testing house for diving equipment under the supervision of Mr TG Anthony, resulted in "dive completion" or "dive failure" where one of the outcomes designated as "failure" was the development of an end tidal CO2 greater than 8.5 kPa (64 mmHg). There was a clear inflection of risk at gas densities above 6 g/L. Indeed, the proportion of dives failing because of hypercapnia rose from 7% at densities between 4.1 and 5 g /L, to 8.5% between 5.1 and 6 g/L, and to 41% between 6.1 and 7 g/L. On this basis we recommend that divers plan breathing gases to have a density that is ideally below 5.2 g/L and to treat 6.2 g/L as an absolute maximum.

CO2 retention during rest in shallow depths

This threat of CO2 retention during diving has been particularly troublesome for deep divers who spend hours decompressing from their dives whilst breathing high pressures of inspired oxygen (typically a PiO2 between 1.3 and 1.6 ATA). Although oxygen toxic seizures are rare at these PO2s, they do occasionally occur and the risk is thought to increase as the duration of exposure lengthens. Thus, the final hours of decompression are, in theory, the period of highest risk. This is a greatly feared

complication because a seizure underwater may result in drowning (not to mention the inevitable failure to complete prescribed decompression).

Since hypercapnia markedly increases the risk of cerebral oxygen toxicity, there has been interest in whether CO2 retention is likely during decompression. The final hours of decompression are typically spent resting in shallow depths, thus there is no exercise and the respired gas is not dense. These factors should lower the risk of CO2 retention, but the possibility had not been investigated in a real world study mainly because it is virtually impossible to establish real time end tidal CO2 monitoring in a typical diving environment.

This brings the narrative back to Bikini Atoll where we were diving the wrecks sunk by the 1946 nuclear weapon tests. We took advantage of the necessity for decompressions under a stable working platform in ideal surface conditions to conduct a simple field study in which we measured end tidal CO2 in divers immediately on surfacing after a very brief ascent from their final 3m decompression stop [6]. Data were gathered from 34 dives, and compared to resting end tidal CO2 levels from the same subjects at the surface two hours after the dive. In-water and post-dive resting end tidal CO2 readings did not differ, and were almost invariably less than a nominal normal value of 45 mmHg. One subject returned a surfacing end tidal CO2 of 48 mmHg. Thus, perhaps not surprisingly, we did not find a systematic tendency to CO2 retention in decompressing divers after their shallowest stop in ideal conditions. While this is reassuring, we cannot exclude the possibility of sporadic CO2 retention under these circumstances by predisposed individuals.

Epilogue: exploring the medical spaces on USS Saratoga

Over several expeditions to Bikini Atoll we have found our way into and explored the Sick Bay area on USS Saratoga. There is a very small operating room which is tight, obstructed by equipment and dangerous. I was unable to find any obvious anaesthesia equipment. The hospital ward area is more open and unobstructed, but there are few items of interest (unless sinks and urinals can be categorised as such). The dental surgery is by far the most fascinating. There are 3 largely intact work stations with drills, elaborate cascading spit bowl systems, instrument racks, and chairs with head clamps! It is an intriguing museum-piece exemplar of the approach to care at that time. Unfortunately, somewhat wasted on an anaesthetist.

References

1. Jones RM. Desflurane and Sevoflurane: inhalation anaesthetics for this decade? *BJA* 1990;65:527-36
2. Mitchell SJ, Doolette DJ. Recreational technical diving part 1. An introduction to technical diving. *Diving Hyperbaric Med* 2013;43:86-93.
3. Deng C, Pollock NW, Gant N, Hannam JA, Dooley A, Mesley P, Mitchell SJ. The five minute prebreathe in evaluating carbon dioxide absorption in a closed-circuit rebreather: a randomised single-blind study. *Diving Hyperbaric Med* 2015;45:16-24.
4. Doolette DJ, Mitchell SJ. Hyperbaric conditions. *Comprehensive Physiol* 2011;1:163-201.
5. Anthony TG, Mitchell SJ. Respiratory physiology of rebreather diving. In: Pollock NW (ed). Proceedings of the US National Oceanographic and Atmospheric Administration 2015 Workshop on Scientific Diving. Seattle, NOAA 2015: In press.
6. Mitchell SJ, Mesley P, Hannam JA. An observational field study of end tidal CO_2 in recreational rebreather divers on surfacing after decompression dives. *Aviat Space Environ Med* 2015;86:41-45.

Airway Update

Colin Marsland

Department of Anaesthesia, Wellington Hospital

Traditional ideas regarding the "difficult airway" are being challenged. This may be just as well since we are not very good at predicting it. What does "difficult mask ventilation" mean when shaving a beard makes it easy, or when insertion of a supraglottic airway can be performed awake or as a rescue technique? What does "difficult intubation" mean when an awkward Gd 3 direct view with a Macintosh blade becomes a Gd 1 VL view with a McGrath? What does "failed intubation" mean when a routine fibreoptic intubation is performed instead of an impossible laryngoscopy?

The development and refinement of new and existing airway equipment is influencing ideas about assessment, planning, performance, and documentation of airway management. The over-arching importance of oxygenation from induction to recovery is being re-emphasized as the glue that holds the multiple facets of airway management together.

Evolution of Equipment

The profound impact of the original LMA has been extended by 2nd generation supra-glottic airways. Starting with hands-free convenience for the anaesthetist, the indications for use now include rescuing of difficult airway situations, minimizing airway stimulation and protecting the airway from ENT bleeding. The second generation SGA's have extended the applications by providing access to the stomach and achieving a higher seal pressure, in particular making positive pressure ventilation safer and more effective. The proseal LMA has been particularly useful in adults and children but there is a development race to perfect the supraglottic triumvirate of high seal pressure, gastric access and conduit for intubation. The AirQ provides a good airway and conduit for intubation but gastric access is prototypical. The Auragain is a good device with effective gastric access. Intubation with normal sized tubes is readily achievable over a bronchoscope. The current version is slightly stiff at the tip which may make insertion difficult.

Video-laryngoscopes (VLs) with traditional or highly angulated blades are challenging the supremacy of direct laryngoscopy (DL) in routine practice. The longevity of nuanced DL is perhaps threatened by the simplicity and ease of VL. It is said that VLs can make difficult DL easy and easy DL difficult. With markedly angulated blades, difficulty visualising the glottis is replaced by difficulty locating the tip of the endotracheal tube, requiring specialised stylets. Airway bleeding or soiling can render VL techniques useless where DL techniques are invaluable.

The flexible bronchoscope remains the gold standard for management of most supraglottic airway challenges. Failure to use the technique when indicated was highlighted in NAP4. Barriers to appropriate use have perhaps increased due to the popularity of VL and attendant risk of inadequate training and exposure to bronchoscopic techniques. Barriers to using video-bronchoscopes have been reduced with the advent of the single use a-Scope from Ambu, the latest iteration of which is excellent for routine use. The unit cost of disposable bronchoscopes is similar to the processing and repair costs for re-usable scopes in our department.

The emergence of electronic record keeping in systems such as Safer Sleep make it possible to create comprehensive user friendly templates to record the detail of airway management, and could tie in with national databases and alert networks for complex patient airways.

Evolution of Drugs

Sugammadex allows exquisite control of the neuromuscular junction that has been blocked by rocuronium. Post-apnoea oxygenation is maintained longer with non-depolarising rocuronium than with depolarising succinylcholine and reversal with 16mg/kg sugammadex is faster than spontaneous offset with succinylcholine. This presumes that an adequate quantity of sugammadex is immediately

available, preferably in 5ml (100mg/ml) ampoules. The combination of rocuronium and sugammadex is attractive for higher risk rapid sequence intubations but is tempered by issues related to cost (sugammadex), allergy (rocuronium) and the impact of other induction drugs on the return of spontaneous ventilation.

Emphasis, Assessment and Strategy

Maintenance of oxygenation is assuming its rightful position as the over-arching concept driving airway management decisions from induction to recovery. NAP4 has had considerable influence with renewed emphasis on assessment and planning, front of neck access (CICO) and extubation issues. Airway algorithms have evolved from complex top-down decision and equipment flowcharts (ASA) to more simplified strategic plans (DAS) with the latest DAS iteration due out this year. The "Vortex" model is a useful visual concept that combines key airway decisions with dynamic oxygenation.

Placing oxygenation at the forefront of assessment and planning, helps shift emphasis toward patient-centric rather than tool-orientated airway management. Airway assessment then includes not only the key techniques (mask ventilation, supraglottic airway placement, laryngoscopy, front of neck access) but also systemic patient factors that influence the speed and consequences of hypoxemia and other complications. These include pre-oxygenation, apnoeic oxygenation, haemodynamic compromise (e.g ICU patient), ventilation-perfusion inequality (e.g obesity) and aspiration. A common thread in induction-related airway crises is running out of time due to failure of oxygenation. Benumof's desaturation curves may not apply in the critically ill or the inadequately pre-oxygenated patient with shunt. Desaturation to 85% may be as short as 23 seconds in critically ill patients compared to 8-9 minutes in healthy adults.

Running out of time may also reflect lack of comprehensive planning. There has been considerable emphasis on having a Plan B and C (DAS) but a comprehensive Plan A to prevent trouble happening might sometimes be lacking. Global assessment and planning should also include which rescue plans might be futile. (e.g inaccessible mouth or front of neck) A strategic framework that includes the finite possibilities for management of any patient's airway can be a useful tool for planning purposes and helps avoid the trap of blindly following an algorithm or of doing what normally works in normal patients.

This might include options for :

1. Consciousness: Awake (VL look, SGA, FOI, Tracheostomy, Extubation)
   Asleep; (Depth of anaesthesia, reversibility of relaxant)

2. Oxygenation: Spontaneous Breathing (Assisted, CPAP, Lung volume)
   Apnoeic (Pre-O2, Apneic -O2, CMV, Transtracheal)

3. Techniques: Positioning, Facemask/adjuncts, SGA, DL/VL/ETT, FOBI,
   Combinations of above.

Extubation / Emergence

There has been considerable emphasis on maintaining oxygenation before and after extubation since NAP4. The airway challenges facing the anaesthetist are often similar in magnitude to those at induction but made worse because:

1. It is more difficult to control emerging consciousness, reflex arcs, respiratory control and neuromuscular function, than it is to titrate sedation or render a patient unconscious and paralysed.

2. Airway management and respiratory function is influenced by surgery, analgesia, and the impact of residual anaesthesia and neuromuscular blockade.

3. Planning and preparation may be overlooked, complicating the response to a post-extubation crisis.

4. Control may have been delegated to PACU or ICU staff.

Basic clinical criteria for awake extubation include spontaneous ventilation, optimal lung volume, pre-oxygenation, airway toileting, demonstrated reversal of neuromuscular blockade, normo-thermia, haemodynamic stability, minimal volatile anaesthetic and a calm, comfortable, responsive patient.

The Cook Staged Extubation Kit includes a long soft wire and an airway exchange catheter both of which are surprisingly well tolerated by patients after extubation. It seems to be a sensible bridging technique between extubation and full patient autonomy when there is known airway difficulty, or when there is potential for difficulty to evolve because of oedema, infection etc. Hazards associated with the inappropriate use of long airway exchange catheters, particularly related to depth of insertion (direct trauma to the lung and barotrauma with high pressure oxygen) must be borne in mind.

Can't Intubate, Can't oxygenate

While the optimal techniques for front of neck access are being debated, the need for emergency oxygenation by this route in a CICO crisis is clearly established.

Needle cricothyrotomy

An attempt to locate a 14G cannula in the trachea is unlikely to cause direct harm, but it is essential to positively identify the tracheal lumen (free aspiration of air) and to quickly move on to a scalpel technique if identification is not or will not be possible e.g unclear anatomy or trauma/blood in airway.

The application of trans-tracheal oxygen from a high-pressure source can be either life-saving or life-threatening depending on the end-user. Clarity regarding safe techniques for emergency transtracheal oxygenation is developing rapidly. A single 1L breath of O2 in healthy post-apnoiec sheep with completely obstructed upper airways, can reinflate collapsed lungs and provide 5 minutes of normal oxygenation. The emphasis is rescue oxygenation followed by definitive management rather than normocarbia with its attendant risks of air trapping and barotrauma.

The Ventrain is a new device for transtracheal ventilation that can jet oxygen at high flows and low pressure as well as provide active expiration via a transtracheal catheter. It can achieve rescue oxygenation and normocarbia but the manual co-ordination required to use it safely may make it unsuitable for emergency use. The Rapid-O2 is a much simpler t-piece like device that works well for rescue oxygenation and provides palpable feedback to the user at higher airway pressures. Both these devices are flow controlled and when attached to a flowmeter open to 15L/min, will deliver 250ml O2 per second through a 14G cannula. The Manujet is pressure controlled. It is important to dial the pressure on the device back to 1bar and at this setting it will also deliver 250ml/sec O2 via a cannula but there is no pressure feedback. The manujet may be particularly useful if pressure needs to be titrated up to allow adequate forward flow of oxygen when the upper airway is relatively open.

CICO is fortunately a rare event but by definition it is highly stressful and there will be severe time pressure. An important human factor issue is the role of the arriving help and the asking of the key question that enables the emergency algorithm to unfold... "Is this a CICO?". We have attached CICO kits, (with cognitive aids based on the Heard algorithm techniques (needle cricothyrotomy, scalpel-bougie and scalpel-blunt dissection)) to our anaesthetic machines in all anaesthetising locations in Wellington Hospital. A rapid-O2 device is in each kit and the difficult airway cart also has a Ventrain (post-emergency sustained TTJV) and a Manujet (open airway). We are providing training that combines bench practice, simulation and wet labs (anaesthetised sheep) and at the time of writing approximately 30 senior staff have taken part.

References

1. Huitink JM, Bouwman RA. The myth of the difficult airway: airway management revisited. (Editorial) Anaesthesia 2015;70:241-257
2. Henderson JJ, Popat MT, Latto IP, Pearce AC, Difficult Airway Society. Difficult Airway Society Guidelines for management of the unanticipated difficult intubation. Anaesthesia 2004; 59: 675-694
3. Practice Guidelines for Management of the Difficult Airway. An updated report by the American Society of Anesthesiologists Task Force on management of the difficult airway. Anesthesiology 2003; 98: 1269-77
4. Cook TM, Woodall N, Frerk C. Major complications of airway management in the UK; results of the Fourth National Audit Project of the Royal College of Anaesthetists and the Difficult Airway Society. Part 1; Anaesthesia. B J Anaesth 2011;106:617-31
5. Kheterpal S, Healy D et al. Incidence, predictors and outcome of difficult mask ventilation combined with difficult laryngoscopy: a report from the multicenter perioperative outcomes group. Anesthesiology 2013 Dec;119 (6):1360-9
6. Weingart SD, Levitan RM. Preoxygenation and prevention of desaturation during emergency airway management. Ann Emerg Med 2012:59:165-175
7. Benumof JL, Dagg R, Benumof R. Critical hemoglobin desaturation will occur before return to an unparalyzed state following 1 mg/kg intravenous succinylcholine. Anesthesiology 1997;87:979-982.
8. Farmery AD, Roe PG. A model to describe the rate of oxyhaemoglobin desaturation during apnoea. Br J Anaesth. 1996;76:284-291.
9. Mort TC. Preoxygenation in critically ill patients requiring emergency tracheal intubation. Crit Care Med. 2005;33:2672-2675.
10. Bhatia PK, Bhandari SC, Tulsiani KL, et al. End-tidal oxygraphy and safe duration of apnoea in young adults and elderly patients. Anaesthesia. 1997;52:175-178
11. Altermatt FR, Munoz HR, Delfino AE, et al. Pre-oxygenation in the obese patient: effects of position on tolerance to apnoea. Br J Anaesth. 2005;95:706-709.
12. Collins JS, Lemmens HJ, Brodsky JB, et al. Laryngoscopy and morbid obesity: a comparison of the "sniff" and "ramped" positions. Obes Surg. 2004;14:1171-1175.
13. Ramachandran SK, Cosnowski A, Shanks A, et al. Apneic oxygenation during prolonged laryngoscopy in obese patients: a randomized, controlled trial of nasal oxygen administration. J Clin Anesth. 2010;22:164-168.
14. Taha SK, El-Khatib MF, Baraka AS, et al. Effect of suxamethonium vs rocuronium on onset of oxygen desaturation during apnoea following rapid sequence induction. Anaesthesia.2010;65:358-361.
15. Tang L, Li S, Huang S, et al. Desaturation following rapid sequence induction using succinylcholine versus rocuronium in overweight patients. Acta Anaesthesiol Scand. 2011;55:203-208.
16. Perry JJ, Lee JS, Sillberg VA, et al. Rocuronium versus succinylcholine for rapid sequence induction intubation. Cochrane Database Syst Rev. 2008;(2):CD002788.
17. Lee C, Jahr JS, Candiotti CA, et al. Reversal of profound neuromuscular block by sugammadex administered three minutes after rocuronium: a comparison with spontaneous recovery from succinylcholine. Anesthesiology 2009;110:1020–5
18. Difficult Airway Society Guidelines for the management of tracheal extubation. Anaesthesia 2012;67:318-34
19. Heard A, Green R, Eakins P. The formulation and introduction of a 'can't intubate, can't ventilate' algorithm into clinical practice. Anaesthesia 2009; 64: 601-608
20. Hamaekers A, Borg P, Enk D. Ventrain: an ejector ventilator for emergency use. British Journal of Anaesthesia 2012; 108: 1017-21
21. Berry M, Tzeng Y, Marsland C. Percutaneous transtracheal ventilation in an obstructed airway model in post-apnoeic sheep. Br J Anaesth 2014;113:1039-45
22. Paxian M, Preussler N, Reinz T et al. Transtracheal ventilation with a novel ejector- based device (Ventrain) in open, partly obstructed, or totally closed upper airways in pigs. British J Anaesth 2015;115:308-16
23. Noppens R. Ventilation through a 'straw': the final answer in a totally closed upper airway? (Editorial) British J Anaesth 2015;115:168-70

Update in Paediatric Anaesthesia

Indu Kapoor

Department of Anaesthesia, Wellington Hospital

This update covers topics relevant to the episodic as well as the specialist paediatric anaesthetist.

An update on maintaining skills and competency, including courses and networks in New Zealand, will also be discussed on the day.

Neurotoxicity and Paediatric Anaesthesia

Background

Repetitive painful stimulation in neonatal animals is associated with cell death and adverse neurologic outcomes, changes in behavioural and cognitive function and chronic pain syndromes. Similarly, untreated painful stimulation in early life in humans is associated with diminished cognition and motor function in later life.

However, in the last two decades all routinely used sedatives and anaesthetics have been found to be neurotoxic in a wide variety of animal species, including nonhuman primates.

Animal Data

The neurotoxic effects observed in animals include apoptotic neuronal cell death, diminished neuronal density, decreased neurogenesis and gliogenesis, alterations in dendritic architecture, diminution of neurotrophic factors, mitochondrial degeneration, cytoskeletal destabilization, abnormal reentry into the cell cycle, as well as learning and memory impairment (1).

Extrapolation of this animal data to humans has some inherent difficulties.

Animal studies did not control for physiological variables like blood pressure, oxygenation, acidosis, hypoglycemia; factors that can induce apoptosis in these anaesthetized animals. Also drug doses and duration of exposure in animal studies cannot be transferred to humans. Apoptosis secondary to anaesthetic exposure is only seen at certain stages of brain development and is not uniform. It is not known how these vulnerable development stages (immature neurons) in animals correlate with development stages of human brain. Animal studies, also, do not include continuous stimulation of the central nervous system by surgical stress and pain (2).

Human Evidence

Several epidemiologic human studies have observed an association between (more than one) anesthesia exposure in patients younger than 3 to 4 years and subsequent learning disabilities and language abnormalities (3), whereas others have not found this link (4).

It remains unresolved whether anaesthetic exposure (type, duration) or other factors, such as the underlying medical condition, surgery, inflammatory response, pain, physiologic abnormalities during surgery, or other unknown factors, are causative for the observed association/abnormalities (2).

Current Status

It remains very unclear if the animal studies have any clinical relevance; or indeed how, or if, clinical practice needs to be altered. Answering these questions is of great importance given the huge numbers of young children exposed to general anaesthetics Additional animal and clinical research is urgently needed to identify the phenomenon's underlying mechanisms, to assess human applicability, and to devise mitigating strategies (5)

Take home messages

- no changes in anaesthetic clinical practice are recommended, anaesthesia and surgery is only undertaken when absolutely necessary in children (APAGBI consensus statement July 2015)
- surgical procedures performed under anesthesia be avoided in children under 3 years of age unless the situation is urgent or potentially harmful if not attended to
- (www .smarttots.org/resources/consensus .html) (6)
- parents and care providers should be made aware of the potential risks that anaesthetics pose to the developing brain (6)
- surgeons, anaesthesiologists, and parents should consider carefully how urgently surgery is needed, particularly in children under 3 years of age (6)
- No consensus statement from SPANZA

1 Jevtovic-Todorovic V, Hartman RE, Izumi Y, et al. Early exposure to common anaesthetic agents causes widespread neurodegeneration in the developing rat brain and persistent learning deficits. J Neurosci 2003;23:876–82

2 Erica P. Lin, Sulpicio G. Soriano, Andreas W. Loepke. Anesthetic Neurotoxicity. Anesthesiology Clin 32 (2014) 133–155

3 Wilder RT, Flick RP, Sprung J, et al. Early exposure to anesthesia and learning disabilities in a population-based birth cohort. Anesthesiology 2009;110: 796–804

4 Sprung J, Flick RP, Wilder RT, et al. Anesthesia for cesarean delivery and learning disabilities in a population-based birth cohort. Anesthesiology 2009;111:302–10

5 Andrew J. Davidson, Karin Becke, Jurgen de Graaff, Gaia Giribaldi, et al. Anesthesia and the developing brain: a way forward for clinical research. Pediatric Anesthesia 25 (2015): 447–452

6 Bob A. Rappaport, Santhanam Suresh, Sharon Hertz, Alex S. Evers, Beverley A. Orser. Anesthetic Neurotoxicity — Clinical Implications of Animal Models. N Engl J Med 372;9: 796-797

Emergence Agitation

Background

Emergence delirium/agitation (ED/EA), discomfort, tantrums and pain are some of the early postoperative negative behaviour (e-PONB) observed after general anesthesia; more often in children (1).

ED is a very common occurrence in children; the reported incidence being as high as 70% after sevoflurane or desflurane anaesthesia (1). Children of pre-school age are most commonly affected. It is self-limiting but may cause distress to patients, parents and staff, and may result in physical harm to the child, particularly at the site of surgery, dressings and intravenous cannulae (2).

More than 10 points on the Pediatric Anesthesia Emergence Delirium (PAED) scale (3), that incorporates cognitive and agitation assessment, has been found to be the a valid and reliable method of diagnosing ED and is used in most studies studying ED in children.

Update

Paediatric Anesthesia Behaviour (PAB) score has been used to predict occurrence of postoperative ED in children (4).

Pathophysiology

At the termination of sevoflurane anaesthesia the electroencephalogram (EEG) in children with ED shows, coinciding with arousal with delirious behaviour, a variety of EEG patterns occuring during the

indeterminate state before the appearance of normal wake or sleep patterns. In children without ED, the indeterminate state transits to classifiable sleep or drowsy states, before peaceful awakening (5)

Diagnosis

It is difficult to differentiate pain at wakeup from ED in children. Somaini et al showed that even though it is difficult to differentiate between ED and pain using FLACC and PAED scores, 'No eye contact', 'No purposeful action', and 'No awareness of surroundings' criteria (PAED Score) significantly correlated with ED whereas 'Inconsolability' and 'Restlessness' are not reliable enough to identify pain or ED in the first 15 min after awakening (6)

Etiology

Sevoflurane, Isoflurane or Desflurane anaesthesia all have similar incidence of ED in children whereas Halothane and propofol TIVA or propofol for maintenance with sevoflurane anaesthesia have lower incidence of ED (7)

Adjuncts

Compared with no adjunct, effective adjuncts for reducing the risk of EA during sevoflurane anaesthesia include dexmedetomidine (7,8,9), clonidine (7,8,9) and opioids, particularly fentanyl (7,9). Even though these interventions were associated with delay in discharge from PACU, there was no delay in discharge from the hospital (7,8,10).

Evidence for a bolus of propofol, ketamine or midazolam at the end of anaesthesia for decreasing the incidence of ED was of only moderate strength in the Cochrane review by Costi et al (7). In the same review parental presence at wake up and midazolam oral premedication did not decrease the incidence of ED. Similarly, depth of anaesthesia, rapid awakening and type of surgery have not been shown to influence the incidence of ED (10). Melatonin premedication has not shown to decrease the incidence of ED post sevoflurane anaesthesia when compared to midazolam premedication (11)

In a recently published RCT, Costi et al showed that a short transition to propofol 3 mg/kg over 3 min at the end of sevoflurane anesthesia is a simple and effective means of reducing EA in children undergoing MRI scans, without any adverse effects except slight delay in discharge form PACU (12).

Treatment

If treatment of ED becomes necessary, a single bolus of propofol (0.5–1.0 mg/kg IV), fentanyl (1–2.5 mg/kg IV), or dexmedetomidine (0.5 mg/kg IV) (8) has been successful in decreasing the severity and duration of the episode (9)

Consequences

The odds ratio of having new-onset postoperative maladaptive behaviour is 1.43 for children with marked ED, as compared with children with no symptoms of ED (13)

Take Home message

ED is common in 2-5year olds, especially after inhalational anaesthesia with ether class of inhalational agents.

Propofol anaesthesia has a low incidence of ED but does not completely prevent it.

There is strong evidence for adjuncts like propofol (3mg/kg over 3 minutes), clonidine, dexmedetomidine or fentanyl (1mcg/kg), given at the end of the case, for decreasing incidence of ED. Evidence is not as strong for smaller bolus of propofol, midazolam or ketamine.

Speed of wakeup, parental presence, melatonin or midazolam pre-med and type of surgery has no

correlation/effect on the incidence of ED.

Control of pain is paramount even though ED is a distinct physiological entity compared to post-operative behaviour disturbance secondary to pain.

Occurrence of ED may have persistent postoperative maladaptive behaviour and should be managed actively, despite its self-limiting nature and parents should be informed of the same.

1 Stargatt R, Davidson AJ, Huang GH, et al. A cohort study of the incidence and risk factors for negative behavior changes in children after general anesthesia. Paediatr Anaesth 2006; 16: 846–59

2 Wong D D L, Bailey C R.Emergence delirium in children Anaesthesia 2015, 70, 375–392

3 Sikich N, Lerman J. Development and psychometric evaluation of the post anesthesia emergence delirium scale. Anesthesiology 2004; 100: 1138–45.

4 Beringer RM, Greenwood R, Kilpatrick N. Development and validation of the Pediatric Anesthesia Behavior score – an objective measure of behaviour during induction of anesthesia. Pediatric Anesthesia 2014; 24: 196–200.

5 Martin JC, Liley DT, Harvey AS, Kuhlmann L, Sleigh JW, Davidson AJ. Alterations in the functional connectivity of frontal lobe networks preceding delirium in children. Anesthesiology. 2014 Oct;121(4):740-52.

6 Marta Somaini, Emre Sahillioglu, Chiara Marzorati, Federica Lovisari, Thomas Engelhardt & Pablo M. Ingelmo Emergence delirium, pain or both? a challenge for clinicians Pediatric Anesthesia 25 (2015) 524–529

Fluids in Paediatric Anaesthesia

Background

For more than half a century, hypotonic fluids (sodium concentrations as low as 30 mmol/L) have been used for maintenance of hydration in children, mostly due to fear of hypernatremia with use of isotonic fluids (1).

Hypotonic fluids in children have since been shown to cause hyponatraemia; with some children having severe outcomes such as seizures, cerebral oedema, and death (2,3).

Current Status

Maintenance fluids in all hospitalised children -

Cochrane review of >1000 children (4) showed that isotonic fluids (in the first 24hrs of administration) were the best maintenance fluids for in-hospital children, isotonic fluids having substantially lower risk of hyponatraemia (17% versus 34%; RR 0.48; 95% CI 0.38 to 0.60, high quality evidence).

These findings were confirmed by the largest RCT (670 in-hospital children needing >6hrs of maintenance fluids, monitored for 72hrs with safety removal of children from study if with severe hyponatremia; using plasmalyte with 5%D as maintenance fluids) (5)

This trial confirmed that, in children who need maintenance fluid therapy, use of an isotonic fluid reduces the risk of hyponatraemia compared with use of a hypotonic fluid (0.45%NS with 5%D), with no increases in the risk of adverse outcomes.

It also showed a 7times (possible) increased risk of severe hyponatremia and seizures in the hypotonic fluid group **(5 children were removed due to safety concern with hyponatremia).**

The increased risk of hyponatremia with hypotonic fluids (compared to isotonic fluids) was seen in surgical (OR 0·32, 95% CI 0·12–0·82; p=0·02) and non-surgical patients (OR0·32,0·12 85;p=0·02) as well as patients in the intensive care unit (OR 0·15, 0·01–2·08; p=0·16).

Smaller studies from around the world (6,7) have shown similar results and confirmed the notion that isotonic maintenance fluid administration is safe in general pediatric patients and may result in fewer cases of hyponatremia.

Risk of Hypernatremia

No studies (RCTs or reviews) have shown hypernatremia with isotonic fluids (0.9%NS/RL/PL) for maintenance in the first 24 hrs of treatment (4,5, 6,7). There is no consensus on time for peak fall in sodium levels; varying from 6hrs (5) to 24hrs (4).
In one study (n=119, children with pneumonia)(7), in the isotonic group, there was significant increase in serum sodium between 24 and 48 hours (4.3, 95% CI: 0.1, 8.4 mEq/L; P=0.04). The authors recommended reduced volume isotonic fluids for maintenance after 24hrs with serum electrolyte measurements to guide treatment.

Maintenance fluids in critically ill children

Safety of isotonic fluids, as maintenance fluids, for children in intensive care unit has been proven by the results of the RCT by McNab et al (5)

Volume of intravenous fluid required for treatment will however vary according to disease states in critically ill children and will be directed by fluid status (hypervolemia, euvolemia, or hypovolemia), glucose and electrolyte levels (should be routinely monitored at regular intervals) in critically ill children (8).

Carcillo, in his review, recommended that both intravenous fluid composition and quantity of infusion should be adjusted accordingly on an evolving basis (8).

Questions

How much glucose and potassium, which isotonic fluid?

There is no conclusive evidence on addition of glucose and how much.

There is risk of hypoglycemia in critically ill children and in prolonged starvation postop with secondary starvation ketosis (8). All large studies in the last 5 years have used isotonic fluids with 5% glucose (5,6,7) or not studied hypoglycemia as primary outcome (4). However, perioperative administration of 5% glucose for prevention of hypoglycemia may result in stress- induced hyperglycemia. Postoperative hyperglycemia was observed in 94% of children receiving 5% glucose and in 37% of group with 3.33% glucose in 0.3%NaCl intraoperatively (9).

Fluid therapy with a Na^+-content close to the physiologic range with addition of 1.0-2.5% instead of 5.0% glucose has been advised (10). There are, however, no studies with fluids containing this concentration of glucose.

Similar lack of evidence exists with respect to potassium and risk of hypokalemia. In the RCT by McNab et al about 1/3 of the isotonic group had potassium added to the solution. Need for adding potassium to solutions on the wards raises substantial safety concern because of the risk of inadvertent drug error (5,10).

Different isotonic fluids might have different risk profiles due to differences in sodium load, the risk of drug and blood product incompatibilities due to added magnesium/potassium/bicarbonate/acetate, or higher chloride concentration leading to hyperchloremic acidosis.

Availability of commercial preparations and cost may be other limiting factors.

Take home message

Isotonic salt solutions with glucose, although more expensive, should be used for volume resuscitation, maintenance and perioperatively.

There is no role of hypotonic solutions in children needing maintenance fluids for more than 6 hours.

Intravenous fluid in children is therapy like other drugs and should be prescribed and altered regularly vis-à-vis volume and composition of fluid used.

Finally, there is a need for new balanced fluids with glucose to be developed for paediatrics and to be tested in well-designed trials

1. Holliday MA, Segar WE. The maintenance need for water in parenteral fluid therapy. Pediatrics 1957; 19: 823–32.
2. Halberthal M, Halperin ML, Bohn D. Acute hyponatraemia in children admitted to hospital: retrospective analysis of factors contributing to its development and resolution. *BMJ* 2001; 322: 780–82.
3. Hughes PD, McNicol D, Mutton PM, Flynn GJ, Tuck R, Yorke P. Postoperative hyponatraemic encephalopathy: water intoxication. Intravenous fluids for seriously ill children: time to reconsider. *Lancet* 2003; 362: 1320–23.
4. McNab S, Ware RS, Neville KA, Choong K, Coulthard MG, Duke T, Davidson A, Dorofaeff T. Isotonic versus hypotonic solutions for maintenance intravenous fluid administration in children. Cochrane Database Syst Rev. 2014 Dec 18;12:CD009457. doi:10.1002/14651858

Dexmedetomidine, the new wonder drug in paediatric anaesthesia?

Dexmedetomidine has been attributed to have sedative, anxiolytic, sympatholytic, and analgesic properties. It has been used for sedation during regional anesthesia, for radiology cases, for sedation in the intensive care unit and for premedication as well as for decreasing the incidence of emergence delirium and shivering in postanesthesia care units (1,2). Its alpha-2 selectivity, limited renal elimination, and relatively short half-life are desirable properties for paediatric anaesthesia. It has a buccal bioavailability of 82%.

Increasing doses of dexmedetomidine (1–3 mcg/kg) in children with no obstructive sleep apnea (OSA) is not associated with decrease in airway reflexes and does not appear to be associated with clinical signs of airway obstruction. The effect on airway of children with OSA is however not clear.

Based on animal studies, dexmedetomidine might be a useful adjunct to an opioid-based technique than current volatile anaesthetic techniques in neonates to decrease possible neurotoxic effects of anaesthetics on the developmental brain (3). However the dose and combinations need to be studied in more detail before it becomes standard practice.

Bradycardia, hypotension, cardiac conduction delay, and evoked potential changes all require greater scrutiny in the pediatric population and particularly with respect to different age groups (4). Similarly, systemic absorption and supra-spinal effects of dexmedetomidine in prolongation of effect of regional blocks needs to be explored further.

1. Costi D, Cyna AM, Ahmed S, Stephens K, Strickland P, Ellwood J, Larsson JN, Chooi C, Burgoyne LL, Middleton P. Effects of sevoflurane versus other general anaesthesia on emergence agitation in children. Cochrane Database Syst Rev. 2014 Sep 12;9:CD007084. doi: 10.1002/14651858.CD007084.pub2
2. Pickard, P. Davies, K. Birnie and R. Beringer Systematic review and meta-analysis of the effect of intraoperative a_2-adrenergic agonists on postoperative behaviour in children British Journal of Anaesthesia 112 (6): 982–90
3. Erica P. Lin, Sulpicio G. Soriano, Andreas W. Loepke. Anaesthetic Neurotoxicity. Anesthesiology Clin 32 (2014) 133–155
4. Joseph Cravero, Brian Anderson & Andrew Wolf . Whither dexmedetomidine? Pediatric Anesthesia 25 (2015) 868–870

Transthoracic Echocardiography (TTE) for Non-Cardiac Anaesthetists

Roman Kluger

Department of Anaesthesia, St. Vincent's Hospital

The title of this talk is appropriately TTE for the non-cardiac anaesthetist as it is really the non-cardiac anaesthetist who will use TTE in their practice more than the cardiac anaesthetist. You are more likely to have a patient who has not had appropriate investigations front up on your list. Your cases will usually have less invasive monitoring, possibly increasing the need for TTE.

There is a growing importance of ultrasound in peri-operative and acute care medicine, which strongly suggests that anaesthetists should be proficient users of ultrasound technology in areas beyond regional anaesthesia and central vascular access[1].

Use of echocardiography by non-cardiologists has grown exponentially over the past decade. This is especially so in the critical care specialties such as emergency medicine, intensive care and anaesthesia. There are a number of reasons for this:

1. More ready availability of portable, or at least easily moveable machines, with capability of high quality imaging.

2. Appreciation of the limitations of clinical examination and even invasive monitoring, while TTE is a powerful non-invasive tool able to rapidly provide important reliable information for the diagnosis of a variety of cardiovascular conditions.

3. Embracing the concept of a limited TTE. Unlike formal cardiology-based TTE, a limited TTE is often more appropriate in the peri-operative period to address a particular question and can usually be performed in under 10 minutes. Many studies have shown that clinicians can be trained to qualitatively determine left and right ventricular function and size, detect pericardial effusions, assess volume status, and identify clinically relevant valvular defects in a relatively short period of didactic and hands-on-training.

4. The increasing age of our patients and hence their cardiovascular co-morbidities.

A variety of acronyms / terms have been coined by different organisations to describe limited TTE examinations, e.g. HeartScan (Haemodynamic Echocardiography Assessment In Real Time), FADE (Fast Assessment Diagnostic Echocardiography), RUSH (rapid ultrasound in Shock), FATE (Focus Assessed Transthoracic Echo), Goal directed TTE, Focused TTE.

Learning and gaining experience in TTE

Anaesthetists are now trained in the use of ultrasound equipment for vascular access, regional nerve blocks and more. Hence they are already familiar with the physical principles behind ultrasound imaging and how to use the main features of the equipment.

Anaesthetists are trained (and examined) in cardiac and vascular physiology.

You are 3/4 of the way there!

There are many avenues for training in TTE for critical care specialists:

- Formal on-line university courses such as the Graduate Certificate in Clinical Ultrasound (GCertCU) from the University of Melbourne (www.heartweb.com.au) or Generalist Medical Echocardiography Course from the University of Otago http://www.otago.ac.nz/courses/papers/index.html?papercode=GENA717).

- Short duration hands-on training courses - these are regularly offered by many institutions or groups such as:
  Australian Institute of Ultrasound (http://www.aiu.edu.au/programs_and_courses/),
  Ultrasound Training Solutions (http://www.ultrasoundtraining.com.au/courses),
  Cardiac Skills Australia (http://www.cardiacskillsaustralia.com.au/workshops)
  iHeartScan courses (www.heartweb.com.au)
  Alfred Hospital ICU (Melbourne) , Nepean Hospital ICU (NSW) and more.

- Some hospital departments and course convenors have purchased high fidelity ultrasound simulators (suitable for transthoracic and transoesophageal ultrasound practice).

- Experienced Colleagues - the beauty of TTE is its safety and non-invasiveness. I have yet to meet a patient or male colleague who would be unwilling to be scanned. Hence the only limitation to gaining experience (perhaps after doing a short hands-on workshop) is machine availability and ideally (but not essentially) some local expertise.

- Arrange via your local cardiology department to spend some time with one of their sonographers (usually very skilled and excellent teachers).

- Some excellent websites. In particular I would very highly recommend the Toronto General Hospital Department of Anesthesia virtual TTE (http://pie.med.utoronto.ca/tte/TTE_content/standardViews.html) and TOE websites (http://pie.med.utoronto.ca/TEE/).

The College of Anaesthetists has a professional document: "Guidelines on Training and Practice of Perioperative Cardiac Ultrasound in Adults (PS 46)" relating to this area of anaesthetic practice (including transoesophageal echocardiography). Guidelines have also been published and training pathways proposed by emergency physicians and intensivists.

Indications for Limited TTE

Brian Cowie published our experience at St. Vincent's of a peri-operative TTE service for non-cardiac surgery run by anaesthetists[2]. We performed 170 studies in 3 years (2007-2010). 74% were performed in the pre-anaesthesia room, 13% in the operating room, 12% in the post-anaesthesia care room and 1% in ICU. Images of adequate quality to answer the clinical question were obtained in 167 out of 170 (98%) patients.

The indications were as follows:

Indication	Number (%)
Murmur detected / valve disease suspected	98 (58%)
Haemodynamic instability	37 (22%)
Ventricular function assessment	17 (10%)
Dyspnoea/hypoxia	6 (4%)
Poor functional capacity	3 (2%)
Other	9 (5%)

Some change in patient management resulting from the focused study occurred in 140 out of 170 (82%) patients (see table below)

Changes in management following peri-operative TTE	Number (proportion)
Invasive monitoring avoided	45 (24%)
Referred for formal cardiology TTE	34 (20%)
No management change	30 (18%)
Fluid bolus administered	25 (15%)
Invasive monitoring placed	22 (13%)
Change in anaesthesia technique	20 (12%)
Vasoactive drug administered	12 (7%)
Postoperative recovery location altered	12 (7%)
Procedure cancelled	7 (4%)
Fluid restriction prescribed	3 (2%)
Vasoactive drugs ceased	3 (2%)

Clinical examination is unreliable for assessment of the nature and severity of valvular pathology. This is especially important in the case of severe aortic stenosis which exposes patients to high peri-operative risks, has significant implications for peri-operative management, is increasingly common in our elderly population and is often relatively asymptomatic.

A prompt limited TTE is especially indicated for diagnosis of persistent hypotension in the face of apparently adequate fluid therapy / vasopressors. Unlike clinical examination and invasive monitoring, TTE will rapidly and reliably differentiate hypovolemia from poor left and right ventricular function and detect uncommon problems such as pericardial effusions or severe valvular disease.

Most information is obtained from parasternal, apical and subxiphoid views (see attached slides). Any one of these windows often provides enough images to make the clinical diagnosis, which is fortunate in case one window does not provide satisfactory imaging.

It is nearly always worth trying to perform a TTE before going to transoesophageal echocardiography (TOE) - even in ICU post cardiac surgery. TOE is invasive with Australian data suggesting a 1 in 1000 incidence of gastro-oesophageal injury and 1 in 5000 risk of death[3]. The caveat is that one should rapidly progress to a TOE if images are poor or any doubt remains regarding the diagnosis and the patient is deteriorating.

Doppler examination

Many limited TTE courses and protocols do not involve the use of spectral Doppler. I believe that Doppler should be part of a limited TTE study because:

- of the importance of assessing the severity of aortic stenosis by measuring velocities and gradients across the valve
- of the importance of estimation of right ventricular systolic pressure, as pulmonary hypertension is associated with increased peri-operative risks and is often minimally symptomatic
- it enables measurement of cardiac output
- learning it is readily achievable[4]

Limitations

Equipment availability - although this is becoming less of an issue. The higher end machines often provide better images (in challenging patients) and more reliable and sensitive Doppler. However, the small machines are improving and are nearly always sufficient to provide the diagnosis.

Patient positioning and access. The ideal position is for the patient to be turned half way to their left side, but this is often impossible - especially intra-operatively. However, in my experience, satisfactory imaging from at least one window is nearly always possible with the patient supine. The other problem intra-operatively may be access to the chest in the patient covered by drapes and Bair huggers.

Some patients have very poor acoustic windows and it is impossible to obtain useful images from any view. This is not always predictable from their body habitus.

Unlike TOE, the probe cannot be held in position continuously, hence TTE cannot act a continuous monitor.

Last, but definitely not least, there is always the risk of misdiagnosis from misinterpretation of the images or the inability to visualise an important pathology. The clinician must be aware of their limitations and when in any doubt, a second opinion from a more experienced colleague should be sought. A focused study or limited echocardiography does not replace a formal transthoracic echocardiogram in difficult cases or ones which will require on-going cardiac management.

References

1. Davinder Ramsingh, John Christian Fox, and William C. Wilson. Perioperative Point-of-Care Ultrasonography: An Emerging Technology to Be Embraced by Anesthesiologists. Anesthesia and Analgesia 2015; 120: 990-2.
2. B. Cowie. Three years' experience of focused cardiovascular ultrasound in the peri-operative period. Anaesthesia,2011; 66: 268–273
3. Piercy M, McNicol I, Dinh DT, Story DA, Smith JA. Major complications related to the use of transesophageal echocardiography in cardiac surgery. J Cardiothorac Vasc Anesth 2009; 23: 62-65.
4. Cowie BS, Kluger R. Evaluation of systolic murmurs using transthoracic echocardiography by anaesthetic trainees. Anaesthesia. 2011; 66: 785-90.

Intensive Care Update

Peter Hicks

Intensive Care Unit, Wellington Hospital

Ventilation

High flow nasal prongs (HFNP) providing 40-50 litres/min flow 21 -100% are a very useful addition to non-invasive support. They have been copied from paediatric/neonatal oxygen delivery systems. F&P have developed these in Auckland and marketed them worldwide with NZ being early adopters.

This was highlighted by a recent NEJM article showing the use of HFNP prevented invasive ventilation more than face mask CPAP did. This is a "surprising" result with some methodical issues. It may well be a compliance issue rather than a therapeutic advantage. HFNP are described as providing 3-5 cm CPAP.

Our impression is that they are very well tolerated with very little side effects and have the added advantage of humidification. There are commercially purchased systems for ward areas that provide up to 60% O2. They seem to provide a useful small level of assistance and don't seem to delay referral to intensive where it is appropriate.

The only down side is that they are so well tolerated that patients may not ask to have them removed which gives the impression they are dependant of them and so appear sicker than they are.

N Engl J Med 2015; 372:2185-219: High-Flow Oxygen through Nasal Cannula in Acute Hypoxemic Respiratory Failure

Excess Oxygen may not be good for your brain. Registry and observational data suggest a high PaO2 in the first 24 hours worsens patient outcomes after cardiac arrest. A high PaO2 in the first 24 hours for general ICU patients, is not associated with a poorer outcome. Feasibility studies are underway but the concept that excess oxygen may be harmful will force a change in our practise, particularly in ICU where the FiO2 is generally controlled by the nurses. We may start prescribing an upper SpO2 limit.

Intensive care medicine: 2015, Volume 41: 49 – 57, The association between hyperoxia and patient outcomes after cardiac arrest: analysis of a high-resolution database.

Temperature

Much effort has been put towards preventing and treating hyperthermia but Registry and observational data show differing effects of hyperthermia. For patients with infections hyperthermia may improve outcome compared to normothermia. For patients with neurologic diseases (stroke, bleed, trauma) a higher temperature is associated with a worse outcome but this is not the case if the neuro disease is an infection (encephalitis, meningitis).

Randomised studies of (not using) Paracetamol are underway as a way of assessing the effect. We are a long way from looking for strategies to make people hot but we may be less vigorous in cooling patients with temperatures < 39.0.
Intensive Care Med (2015) 41:823–832. Early temperature and mortality in critically ill patients with acute neurological diseases: trauma and stroke differ from infection.

Intensive Care Med (2012) 38:437–444. Early peak temperature and mortality in critically ill patients with or without infection

We know hypothermia worsens outcome for most patients but it may improve neurological outcome. So far cardiac arrest studies show no improvement and a recent studies in head injuries in children does not improve outcome. Strictly controlled normothermia seems to be the aim now. This is frustrating as hypothermia is an attractive treatment for managing raised intra cranial pressure.

Crit Care Med. 2015 Jul;43(7):1458-66. Hypothermia for Traumatic Brain Injury in Children-A Phase II Randomized Controlled Trial

Systems

After hours discharge from intensive care (after 1800 hours) is still associated with worse patient outcomes, independent of severity of illness and adjusting for other factors. This creates a dilemma for ICU when we get referrals in the evening and we have to balance the harm of not admitting one patient with the harm of an evening discharge. The not admitted patient is much harder to study so there is a research bias.

Intensive Care Medicine 2014 2005 – 2012. Mortality related to after-hours discharge from intensive care in Australia and New Zealand,

Medical Emergency Team (MET). 150 of 166 ICUs in Australia and New Zealand have a Medical Emergency Team (MET). Cardiac arrests with attempted resuscitation should be becoming a rarity. The Early Warning Score (EWS) looks to become a standard assessment on wards, on discharge from Emergency Departments and Intensive Cares, and may have a role pre hospital with GP triage and ambulance. Increasing numbers of MET calls in a hospital is associated with decreased cardiac arrest calls, and increased numbers of palliative care discussions and decisions. The effect on mortality is complicated because MET calls occur in patients who are dying, and for whom treatment is limited.

The Health Quality and Safety Commission have started a project to develop a national EWS. The response to the EWS will be different in different sized hospitals.

Future

Interventional angiographic clot retrieval after stroke improves outcomes. Two papers in the NEJM clearly show a benefit. I am uncertain about the need for anaesthesia for these procedures. Urgent intervention for acute cardiac/coronary events doesn't generally need anaesthesia. The number of eligible stroke patients are likely to be low and it will only be available in neuro centres.

N Engl J Med 2015;2285-95 Saver JL, Goyal M, Bonafe A, et al. Stent-retriever thrombectomy after intravenous t-PA vs. t-PA alone in stroke.

N Engl J Med 2015;2296-306 Jovin TG, Chamorro A, Cobo E, et al. Thrombectomy within 8 hours after symptom onset in ischemic stroke.

Citrate anticoagulation for continuous haemodiafiltration in ICU, is better than heparin at prolonging filter life, and does not cause systemic anticoagulation problems. It is becoming the preferred method. Citrate is infused pre-filter chelating the Ca+, and Calcium is infused post filter to correct it.

This has some implications for patients going to theatre from ICU where there was often an issue with the timing of stopping the heparin and/or dialysis. Now it will just be the logistics of getting the patient off the machine. The "new" problem will be citrate toxicity if the patient can't hepatically metabolise the citrate. This leads to low ionised Calcium and high (total) serum Calcium and is managed by stopping the citrate infusion.

Crit Care Med: 2015: 43(8): 1622 – 1629. A Randomized Controlled Trial of Regional Citrate Versus Regional Heparin Anticoagulation for Continuous Renal Replacement Therapy in Critically Ill Adults

Facts

Intensive care in NZ costs $3600/patient day, which compares well to Australia except we spend $47 NZD/person on intensive care and Australia spend $71 AUD/person. This is because they have more $$/person and a much bigger private insurance system which has 30% of the intensive care beds compared to NZ s 7% (Source: ANZICS CCR).

Long term outcomes: The following table shows the mortality outcomes for sequential time periods, after you are admitted to ICU. Pre-existing conditions have more long term influence than acute physiology. (Source: Wellington ICU)

	All	Age>=80	Have cancer
In Hospital	11.5%	20.6%	16.2%
First year after discharge	9.0%	15.1%	21.2%
Second year after discharge	3.7%	4.5%	13.6%

Reducing post-operative infections; the role of anaesthetists

Sally Roberts

Microbiology Department, Auckland City Hospital

Healthcare-associated infections (HAI) occur in about 10% of all patients receiving care. Surgical site infections (SSI) are the second most common HAI occurring in 2-5% of patients undergoing surgery.[1] SSI are associated with significant morbidity and mortality. Patients with SSI have longer lengths of stay in hospital and have 2-11 times higher risk of death compared to patients undergoing surgery who do not develop an SSI. The excess cost of SSI is not insignificant; the cost to patients, their family/whanau is typically not included in the estimate of cost but may result in a substantial burden on families. Patients exposed to surgery carry more than twice the overall HAI burden than those not exposed to surgery; almost half of the HAI were SSI.[2]

There are twelve "must do's" to reduce the risk of SSI:[3]

- Warm the patient
- Use of the surgical safety checklist to enhance communication and teamwork
- Prophylactic antibiotics in the correct dose at the correct time
- Appropriate skin preparation
- Continue to warm the patient
- High flow oxygen ≈ 80%
- Double gloves
- Monitor blood glucose; treat if >7.8mmol
- Consider wound protector for colorectal surgery
- Antimicrobial impregnated sutures for colorectal surgery
- Avoid blood transfusion
- Use good surgical technique

In 2009 a set of recommendations for a National Surgical Site Infection Surveillance programme were developed by a team lead by ADHB for the Ministry of Health Quality Improvement Committee. The recommendations were reviewed by the newly established Health Quality & Safety Commission in 2011 and a subsequent cost benefit analysis forecast that the benefits from the programme would build slowly until year 10 savings from SSI avoided was estimated at $4.4 million per annum.

In 2012 the Surgical Site Infection Improvement was established.[4,5] The programme is delivered jointly by Auckland and Canterbury District Health Boards and funded by the Commission. The initial focus was on hip and knee arthroplasties and this year the focus has shifted to include cardiac surgery. It is well established internationally that surveillance programmes, consisting of a comprehensive programme of data collection and sharing of information reduce SSI rates. The New Zealand programme is unique in that it tracks performance against adherence to internationally recognized best clinical practices known to prevent SSI. These measures, termed Quality and Safety Markers (QSM) are reported quarterly and targets have set. To date there has been significant improvement in the QSM. There have been a number of lessons learned along the way including the need for multidisciplinary teams engaged in the programme from the start.

References

1. Anderson DJ, Podgorny K, Berríos-Torres SI et al. Strategies to prevent surgical site infections in acute care hospitals: 2014 update. Infect Control Hosp Epidemiol 2014; 35 (6): 6-5-27
2. Sax H, Uçkay I, Balmelli C et al Overall burden of healthcare-associated infections among surgical patients. Ann Surg 2011; 253: 365-70.
3. Dellinger EP. SSI prevention: the must dos International Conference on Prevention and Infection Control, 16-19th June 2015, Geneva, Switzerland.
4. Health Quality & Safety Commission -www.hqsc.govt.nz
    a. Infection Prevention and Control Programme
    b. National Surgical Site Infection Improvement Programme
5. http://www.hqsc.govt.nz/our-programmes/infection-prevention-and-control/projects/surgical-site-infection-improvement/
6. Morris AJ, Panting AL, Roberts SA, Shuker C, Merry AF. A new surgical sire infection improvement programme for New Zealand: early progress. NZ Med J 15 May 2015. Vol 128: No 1414

Competing with Australia: How does our health system compare?

Kate Leslie

Department of Anaesthesia, The Royal Melbourne Hospital

The purpose of this talk is to provide an overview of the financing, payment, regulation and organisation of health care implemented in each country. This information may be useful for anaesthetists wanting to work in either country, to participate in trans-Tasman activities such as those of ANZCA, and or just to understand the current debates about health in Australia and New Zealand.

Australia and New Zealand have highly developed health care services for which governments of each country have overall responsibility. The health professionals of the two countries are highly educated, trained and regulated. Residents of the two countries enjoy exceptional health status and longevity on a global basis, with the exceptions of the Aboriginal and Torres Strait Islander people of Australia, the Māori of New Zealand and various disadvantaged immigrant groups. Australians utilise health services more and contribute higher out-of-pocket payments for health services and pharmaceuticals than New Zealanders. Various mutual recognition agreements provide for reciprocity with respect to the legal sale of goods, provision of services by medical practitioners and consumption of health services by citizens of the two countries.

Item	Australia	New Zealand
Governance structure	Federation of states	National
Number of governments with responsibility for health	Nine	One
Number of parliamentary houses	Two[1]	One
Financing		
Public hospital care (public patients)	Taxation revenue	Taxation revenue
Public hospital care (other patients)	Medicare[2], private insurance premiums, third party payers	ACC, other third party payers
Private hospital care	Medicare, private insurance premiums, out-of-pocket payments, third party payers	Private insurance payments, out-of-pocket payments, ACC, other third party payers
Private specialist consultations	Medicare, out-of-pocket payments	Private insurance payments, out-of-pocket payments
Primary care	Medicare, out-of-pocket payments	Taxation revenue
Payments to medical practitioners		
Public hospital care	Salary, fee-for-service, bonus	Salary
Private hospital care	Fee-for-service	Fee-for-service
Private specialist consultations	Fee-for-service	Fee-for-service
Primary care	Fee-for-service, pay-for-performance	Fee-for-service, capitation,
Payments to hospitals		
Public hospital care	Global budgets, case-mix adjusted funding	Global budgets, case-mix adjusted funding
Private hospital care	Per-day, per-item	Per-day, per-item
Regulation	National Regulation and Accreditation Scheme for 14 health professions; separate regulation and accreditation bodies for medicine	Single regulation and accreditation body for medicine
Organisation		
Public hospital care	Services organised by States and territories (Health Services)	Services organised nationally (District Health Boards)
Private hospital care	Privately organised	Privately organised
Private specialist consultations	Privately organised	Privately organised
Primary care	Services organised by the Commonwealth (mainly Primary Care Partnerships)	Services organised nationally (mainly Primary Health Organisations)

[1] Except for Queensland, Australian Capital Territory and Northern Territory which are unicameral
[2] Medicare is Australia's national insurance scheme, which is funded by general revenue and the Medicare levy

References

The Commonwealth Fund. 2014 International Profile of Health Care Systems. available at –

http://www.commonwealthfund.org/~/media/files/publications/fund-report/2015/jan/1802_mossialos_intl_profiles_2014_v7.pdf?la=en

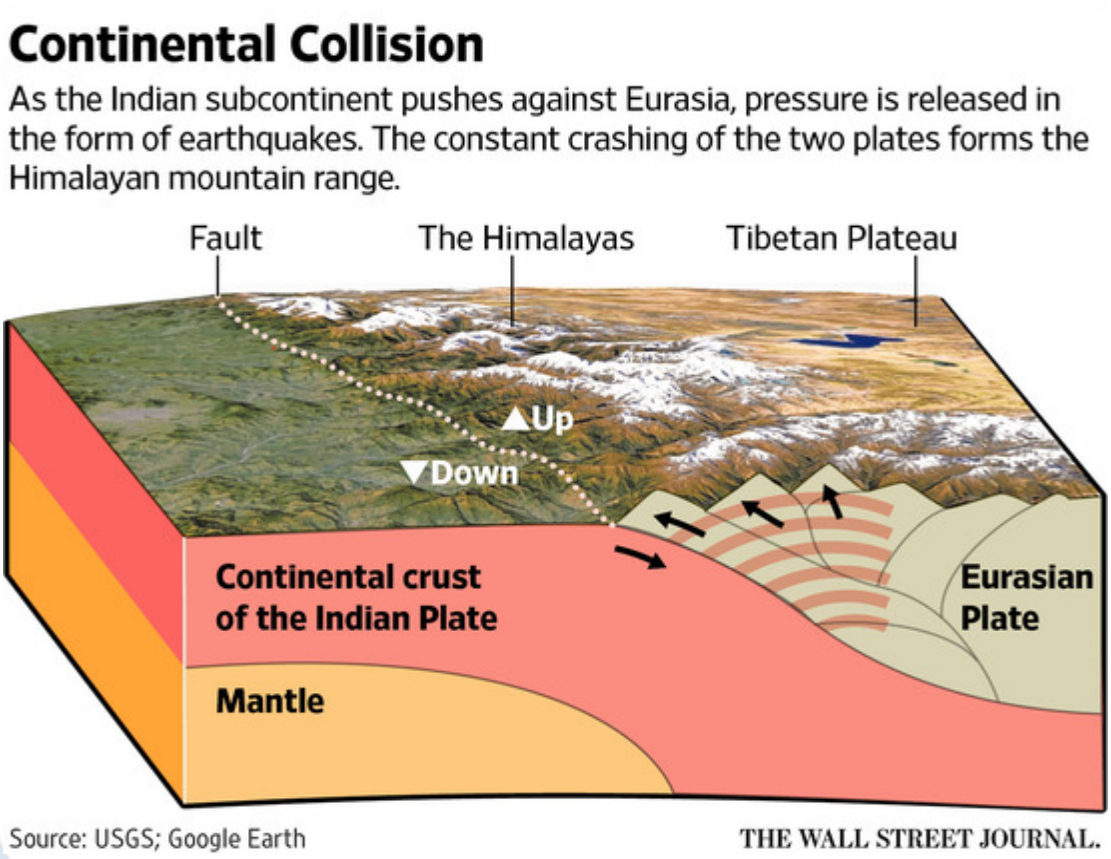
Nepal Earthquake Response

Maurice Lee

Department of Anaesthesia, North Shore Hospital

Nepal is a developing country that is mired in poverty and political instability. It only opened its borders to the international community in the 1950s. It is slightly smaller in size compared to the South Island of NZ, and has a population of about 28 million, 75% of whom are subsistence farmers.

The tectonic up-thrust of the Eurasian plate and the subduction of the Indian plate created the amazing mountainous landscape that defines Nepal. The Himalaya mountain range, which runs along the length of Nepal's northern East-West border with Tibet, is home to 8 of the 10 tallest mountains in the world. Along with the surface beauty and grandeur of the mountains, tension has built up deep inside the major Himalayan fault line spanning 5 countries, Pakistan, India, Nepal, China and Bhutan, a total length of 2300km.



In April and May 2015, the strain was released, and two large earthquakes struck mid-central Nepal, causing significant devastation in the affected districts.

This presentation will be on my experience during my deployment as part of a US-based disaster response team.

Sponsors

SILVER

Mark Ritchie
National Sales Manager
Admedus

T +61 8 9266 0100
M +61 438 035 017
E mritchie@admedus.com

Admedus is a diversified global healthcare group that is developing, investing in and delivering a range of exciting new technologies and leading edge medical devices that have the potential to make a real difference to the outcomes and quality of life of patients around the globe.

Admedus has been providing comprehensive healthcare solutions since 2003 with a focus on improving patient management and cost efficiency for both hospitals and private practitioners. Our infusion management solutions range from whole-of-hospital systems to smaller ambulatory pumps suitable for a broad scope of applications in the Australasian Healthcare market.

SILVER

Aspen Pharmacare offer a diverse range of tried and trusted brands in New **Zealand.** The product mix ranges across Ethical, Primary and Secondary care with key brands being Circadin®, Eltroxin®, Ferinject®, Redipred® and Simdax®.

For more information visit www.aspenpharma.co.nz

Jackie van Rooyen
Aspen Pharmacare

M +64 21 740 277
E jackie.vanrooyen@aspenpharma.co.nz

DDI +64 9 570 1080
F +64 9 915 9581
E aspen@aspenpharma.co.nz

P O Box 62027, Silvia Park Auckland 1644

SILVER

Preston Gash
Connected Healthcare Systems

M +64 21 639 665
E Preston@chsnz.co.nz

Connected Healthcare Systems, in association with Cass Medical, are proud to be the sole distributor for the Mindray patient monitoring and Life support systems including ultrasound in New Zealand. Internationally, Mindray is one of the leading global providers of medical devices and solutions.

Firmly committed to a mission of "sharing medical technologies with the world", they are dedicated to innovation in the field of Patient Monitoring & Life Support.

Cass Medical along with Connected Healthcare are a proud New Zealand company with a long history of providing a strong product line-up, customer focus and excellence in customer support.

Come and visit us on the stand to find out our solution for you.

SILVER

Chris Hughes
Product Specialist
Respiratory and Monitoring Solutions
Covidien New Zealand Ltd
A Medtronic Company

Level 3, Building 5,
666 Great South Road,
Penrose, Auckland 1051, New Zealand
Postal: Private Bag 94 315, Pakuranga,
Auckland 2140, New Zealand

T +64 9 967 4600
M +64 21 963 230
F +64 9 918 3742
E Christina.hughes@medtronic.com

At **Covidien**, we're passionate about making doctors, nurses, pharmacists and other medical professionals as effective as they can be. Through ongoing collaboration with these medical professionals and healthcare organizations, we identify clinical needs and translate them into proven products and procedures.

Our industry-leading brands – including Kendall, Mallinckrodt, Nellcor, Puritan-Bennett and Valleylab – are known and respected worldwide for uncompromising quality.

SILVER

Anthony Reid
Culpan Medical

M +64 21 544 036
E anthony@culpanmed.co.nz

Culpan Medical is a privately owned New Zealand business. Culpan Medical serves the needs of healthcare professionals and organisations with the timely supply of medical devices that offer patients the best prognosis. We are committed to the people we serve with a highly responsive and professional team. We are committed to important future developments in technology that enhance patient outcomes and provide viable, sustainable solutions. Our mission is to provide patients and medical professionals with the best healthcare solutions available worldwide.

SILVER

Device Technologies Ltd

47 Arrenway Drive
Albany
Auckland

T 0508 338 423
T +64 9 913 2000
W www.device.co.nz

Device Technologies is an Australasian-owned distributor of technologically-advanced medical equipment and consumables.

The company's wide range of products is supported by highly trained product specialists, clinical educators, service engineers and regulatory affairs personnel to provide superior outcomes for medical and surgical staff and their patients. Device Technologies maintains offices throughout New Zealand and Australia

Services

Customer service and support
Internationally-certified technical field service personnel
Regulatory affairs
Clinical education
Learning and development
Project planning
Inventory and logistics
Marketing services

SILVER

Kensi Naicker
Perioperative Leader, ANZ
GE Healthcare

T +61 041 530 1523
E anaesthesia.nz@ge.com

GE Healthcare provides transformational medical technologies and services to meet the demand for increased access, enhanced quality and more affordable healthcare around the world.

GE (NYSE: GE) works on things that matter - great people and technologies taking on tough challenges. From medical imaging, software & IT, patient monitoring and diagnostics to drug discovery, biopharmaceutical manufacturing technologies and performance improvement solutions, GE Healthcare helps medical professionals deliver great healthcare to their patients.

SILVER

Hospira

T + 64 4 384 7463

P O Box 9178, Marion Square
Wellington 6141, New Zealand

Hospira, Inc. is a global specialty pharmaceutical and medication delivery company dedicated to Advancing Wellness™ by developing, manufacturing and marketing products that help improve the productivity, safety and efficacy of patient care.

In February 2007, Hospira acquired Mayne Pharma Limited to become the world leader in specialty generic injectable pharmaceuticals. With 70 years of service to the hospital industry, Hospira's portfolio includes one of the industry's broadest lines of generic acute-care and oncology injectables, which help address the high cost of proprietary pharmaceuticals; and integrated solutions for medication management and infusion therapy.

Headquartered north of Chicago in Lake Forest, Ill., Hospira has approximately 15,000 employees worldwide. Hospira's news releases and other information can be found at www.hospira.com.

SILVER

Tori Walker
InterMed Medical

M +64 21 376 771
E tori@intermed.co.nz

InterMed Medical, one of New Zealand's largest privately owned Healthcare companies is pleased to present its key product portfolio at this year's AQUA conference.

InterMed represents a number of leading brands and offers multiple solutions for equipment, interventional and consumable supplies.

Now in our 35th year of trading in New Zealand, hospitals can be assured of high quality products, with proven technology from competent and well trained staff. Please take the time to visit our stand to discuss your Department's clinical needs.

SILVER

Deborah Stanley
Clinical Sales Specialist
FUJIFILM SonoSite Inc

M +64 21 664 985
T 0800 888 204
F +61 2 9939 1831
E deborah.stanley2@sonosite.com

FUJIFILM SonoSite, Inc., the world leader in bedside and point-of-care ultrasound, delivers solutions that meet imaging needs of the medical community. With its VisualSonics ultra high-frequency micro imaging technology, SonoSite continues to influence the future of medical imaging in both the clinical and preclinical markets.

For more information, please visit: www.sonosite.com.au

ADMEDUS

INNOVATIVE HEALTH SOLUTIONS

Superior Ambulatory Infusion Technology

The ambIT® offers a simple yet sophisticated way to control post-operative pain

The ambIT® facts and benefits

- Simple, programmable operation
- Basal Rate only, Basal Rate and Bolus, Bolus only
- Volumetric accuracy of ± 6%
- Visual and audible safety alerts
- Low-cost for high technology
- Single use or re-usuable options
- Full history recorded
- Flexibility to titrate dosage to meet patient needs

ADMEDUS

77386

www.admedus.com

Iron Therapy.

Intravenous iron should be considered in patients with confirmed Iron Deficiency Anaemia and one or more of the following:

- Demonstrated intolerance, non-compliance or lack of efficacy with oral iron
- Pregnancy and postpartum
- Intestinal malabsorption (e.g inflammatory bowel disease)
- Ongoing iron (ie, blood) losses that exceed absorptive capacity
- A clinical need for a rapid iron supply
- Chronic renal impairment receiving concomitant erythropoietin-stimulating agent therapy

Adapted from Pasricha, et al 2010[1].

To find out more visit
www.ferinject.co.nz

Password: **healthy**

ferinject®
ferric carboxymaltose

Mastering the art of iron therapy

Reference: 1. Pasricha, S, et al. Diagnosis and management of iron deficiency anaemia: a clinical update MJA • Vol 193:9 2010. FERINJECT® (ferric carboxymaltose) solution for intravenous (IV) use. **Indication:** Treatment of iron deficiency when oral iron preparations are ineffective or cannot be used. **Contraindications:** hypersensitivity to any of the ingredients; anaemia not attributed to iron deficiency; evidence of iron overload or disturbances in utilisation of iron. **Precautions:** parenteral iron preparations can cause hypersensitivity reactions, observe for hypersensitivity reactions for at least 30 minutes following injection; paravenous leakage can lead to skin discolouration and irritation. **Adverse effects:** Most common: headache; dizziness; hypertension; nausea; injection site reactions; hypophosphataemia. Uncommon; hypersensitivity. Rare; anaphylactoid reactions. **Dosage:** The dosage must be calculated individually for each patient and must not be exceeded. IV injection: Maximum single dose of undiluted solution of 1,000mg (20mL) over 15 minutes per week. For doses 200-500mg, should be administered at a rate of 100mg iron/min. IV drip infusion: Maximum single dose of 1,000mg (20mL) in 250mL saline given over 15 minutes once per week. **Presentation:** 2mL and 10mL vials containing 100mg and 500mg of iron, respectively. Prescription Medicine. Please review full data sheet before prescribing. Data sheet is available at www.medsafe.govt.nz. FERINJECT® is listed on the HML. FERINJECT® is a registered trademark of Vifor Pharma used under license by Aspen Pharmacare. Aspen Pharmacare. C/O Healthcare Logistics, Auckland, NZ. www.aspenpharma.co.nz. **Data Sheet-call 0800 996 312.** TAPS PP5755-15MY.

Vifor Pharma

aspen

Simple Smart Focused

mindray
healthcare within reach

Mindray's New High-end Touch Screen Ultrasound TE7

Mindray's new-generation**TE7 Touch Screen Ultrasound System** was officially released this March at the European Congress of Radiology in Vienna. TE7 combines an intuitive user interface with simplified workflow, an innovative ergonomic design, and focused exam presets. It is designed to provide superior-quality imaging to enable clinicians to make patient-care decisions more quickly.

TE7 takes only three seconds to boot up from standby and is equipped with other enhanced features such as **iNeedle**™ (enhanced visualization for needles), **iZoom**™ (full screen image), and Smart Track (continuous tracking of color flow and optimization of real-time scanning).

With its slim profile, retractable power cable, and built-in battery, TE7 is easy to transport and store. The equipment's seamless and non-porous touch screen with a 10-second screen-locking feature makes it easy to clean, even during exams. TE7 provides the ideal ultrasound solution for a wide range of applications such as anesthesia and pain management, critical care, emergency medicine, and musculoskeletal bedside examinations.

connected
healthcare systems

Special Conference Offer

To celebrate the launch of their new product range, **Connected Healthcare** has special introductory prices on all their devices.

Enquire now on any of our monitoring, anaesthesia or Ultrasound products and find yourself a bargain with our Spring prices

6C Jack Conway Ave, Manukau 2104
PO Box 97 301, Manukau 2241, Auckland
T: 0800 424 797 F: +64 9 929 3241
M: +64 21 639 665 E: admin@chsnz.co.nz
W: www.chsnz.co.nz

PIRAMAL SEVOFLURANE™

(Sevoflurane 100% v/v, Liquid for inhalation)
Prescription only medicine

Piramal
knowledge action care

INDICATIONS FOR USE

Piramal Sevoflurane may be used for induction and maintenance of general anaesthesia in adult and paediatric patients undergoing surgery.

MANUFACTURED IN USA

Representative labelling only

65 COUNTRIES

Use Piramal Sevoflurane™[1]

18 MILLION[2]

Procedures per year are conducted across the world using Piramal Sevoflurane™

1 in 4[3]

Sevoflurane bottles used in the USA are manufactured by Piramal

[1] Also known as Sojourn™ and Sevoflurane Piramal in overseas markets
[2] Based on internal estimates
[3] Based on IMS data

Review data sheet (available at www.medsafe.govt.nz) for prescribing information including dosage, contraindications, precautions, interactions and adverse effects. Piramal Sevoflurane™ is an unfunded medicine, a prescription charge will apply. Further information is available from Device Technologies NZ Ltd.

TAPS Approval No: PP6721 June 2015

FURTHER INFORMATION CONTACT
0508 DEVICE (338 423)
sales@device.co.nz | www.device.co.nz

Proudly distributed by DEVICE TECHNOLOGIES

Aisys Carestation Upgrade Programme

Your Aisys™ Carestation™ is designed to grow with you. The digital platform enables you to seamlessly integrate the latest innovative technologies available.

Upgrade today for a fraction of the cost of buying a new machine.

Extend your investment. Expand your capabilities. Enhance patient care.

For more information, contact us on 0800 434 325.

www.gehealthcare.co.nz

JB32926NZ

The 37°Company

Mistral-Air®

Forced Air Warming

It's all about 37°

Safe, Comfortable & Smart Warming

intermed

You. Me. Make a difference.

WHAT'S NEXT FROM SONOSITE?

BOOTH #9

COME AND VISIT US AT
BOOTH #9 AND FIND OUT MORE!

For more information contact us, phone **1300 663 516**
email **sarah.leverett@sonosite.com** or visit **www.sonosite.com.au**

SonoSite FUJIFILM

TGA approval pending

CIVCO MEDICAL SOLUTIONS

Multi-Modality Imaging

Providing Guidance for Regional Anesthesia.

infinitiPlus™
One Guide. Infinite Possibilities.

CULPAN MEDICAL

www.aqua.ac.nz