AQUA

Annual Queenstown Update in Anaesthesia

Programme and Abstracts, 2012

CONTENTS

Gold Sponsor

Silver Sponsors

Welcome to the fourth Annual Queenstown Update in Anaesthesia. AQUA 2012 continues with the theme of the previous meetings, which is to provide anaesthetists with an annual update on core topics relevant to current clinical practice. Add in an excellent social programme and the setting of Queenstown in the ski season, and the ingredients are in place for a great conference. AQUA is run in partnership with the NZSA and we are grateful for the enthusiasm and support of the NZSA executive.

AQUA 2012 features five international speakers who will add further depth and diversity to this year's scientific programme. The international faculty include; AP Nolan McDonnell, Dr Chris Thompson, Dr Laurence Weinberg, AP Frank van Haren and AP Peter Hebbard. Between them they will cover everything from obstetrics to the future directions of hardware, regional anaesthesia to goal directed therapy, patient safety and ICU care.

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

AQUA 2012 will again capitalise on the attractions Queenstown offers for the social programme. Although Coronet Peak has had a patchy season this year, we have arranged snow fall especially for the AQUA meeting. Night skiing will be held in conjunction with the AQUA BBQ on Friday night. The conference dinner will be held at Skyline Restaurant (not a buffet!). Sipping on a glass of bubbles, you will be whisked to the Skyline via the gondola enjoying spectacular views of Queenstown. Following a fantastic dinner accompanied by fine local wines, we will conclude with live test rugby featuring an All Blacks victory over the Wallabies.*

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

Once again we acknowledge the generous support of our sponsors, in particular GE who are the AQUA 2012 Gold sponsor. We have been well supported by industry this year and we would like to thank them all for being here. Please visit them in the exhibit area.

Enjoy.

Neil MacLennan
Martin Misur
Kerry Gunn
Karen Patching (KP)

* After last year's result, the Convening Committee do not guarantee the outcome of the rugby

SOCIAL PROGRAMME

Thursday, 16 August 2012

AQUA Welcome Drinks	5:00 p.m. – 7:00 p.m.

Friday, 17 August 2012

Ski bus to Coronet Peak departs Millennium Hotel	12:40 p.m.
MedRecruit BBQ at Coronet Peak base building commences	6:00 p.m.
Ski bus to Millennium Hotel departs Coronet Peak	9:00 p.m.

Saturday, 18 August 2012

Ski bus to Coronet Peak departs Millennium Hotel	12:30 p.m.
Ski bus to Millennium Hotel departs Coronet Peak	4:15 p.m.
Dinner	
Gondola from	6:00 p.m.
Dinner service from	7:00 p.m.

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

SCIENTIFIC PROGRAMME

Day 1 – Friday, 17 August 2012

0710-0755	*Breakfast*	*Exhibitor Area*
0755-0800	Welcome and Introduction – Andrew Warmington	*Galaxy Room*
Session 1:	***SUBSPECIALTY SYNOPSIS***	
0800-0840	An update in obstetric anaesthesia	Nolan McDonnell
0840-0905	Airway update	Jeanette Scott
0905-0930	Managing the obese – Lessons from bariatric surgery	Craig Birch
0930-0955	What's new in regional anaesthesia	Peter Hebbard
0955-1025	*Morning break*	*Exhibitor Area*
Session 2:	***BLACK BOXES***	*Galaxy Room*
1025-1105	The new generation anaesthetic machine	Chris Thompson
1105-1130	Goal directed therapy – Experience at a tertiary care centre	Laurence Weinberg
1130-1155	What's new in ventilation?	Chris Thompson
1155	*Close – Lunch packs available for you to pick up*	*Exhibitor Area*
1240	*Bus to Coronet Peak departs from Main Entrance, Millennium Hotel*	

Day 2 – Saturday, 18 August 2012

0715-0800	*Breakfast*	*Exhibitor Area*
Session 3:	***POTPOURRI***	*Galaxy Room*
0800-0830	ICU topics	Frank van Haren
0830-0855	Drug dosage – How many inches should you give?	Tim Short
0855-0920	What's the solution?	Laurence Weinberg
0920-0945	Enhanced recovery after surgery protocols	Matt Taylor
0945-1015	*Morning Break*	*Exhibitor Area*
Session 4:	***AND NOW FOR SOMETHING COMPLETELY DIFFERENT***	*Galaxy Room*
1015-1040	Learning from errors in obstetric anaesthesia – Designing systems to improve patient safety	Nolan McDonnell
1040-1110	A bug's life – So simple, and yet so fascinating too	Ben Harris
1110-1140	Mercenaries, misfits and medics – Helping out overseas	Wayne Morriss
1140	*Close – Lunch packs available for you to pick up*	*Exhibitor Area*
1230	*Bus to Coronet Peak departs from Main Entrance, Millennium Hotel*	

INTERNATIONAL FACULTY

Dr Nolan McDonnell

Department of Anaesthesia and Pain Medicine,
King Edward Memorial Hospital for Women, Perth

Dr Nolan McDonnell graduated from the University of Auckland in 1998 and undertook his training in anaesthesia at Palmerston North and Waikato Hospitals. In 2006 he and his family moved to Perth, Western Australia where he undertook a one year research and obstetric anaesthesia fellowship under the guidance of Prof Michael Paech. He now works as a Staff Specialist in the Department of Anaesthesia and Pain Medicine at King Edward Memorial Hospital for Women. This is Western Australia's only tertiary referral obstetric, gynaecology and gynae-oncology hospital and hence caters for a comparatively high risk population. During his time here he has continued his interest in research, undertaking a Masters of Clinical Research in post caesarean analgesia.

He is now a Clinical Associate Professor with the School of Women's and Infants' Health and the School of Medicine and Pharmacology, University of Western Australia and is widely published on a number of areas of relevance to obstetric anaesthesia. In addition to these roles he sits on the executive of both the Obstetric Anaesthesia and the Welfare of Anaesthetists Special Interest Groups, and he is involved in a number of local and international research studies as well as having a large clinical education commitment.

Dr Chris Thompson

Department of Anaesthesia
Royal Prince Alfred Hospital, Sydney

Dr Chris Thompson graduated from the University of Sydney in 1979. He was trained at Prince Henry / Prince of Wales Hospitals before becoming a Staff Specialist at RPAH. During his training he was fascinated by ventilation, and was with John Lawrence and others at Prince of Wales when they developed Australia's first CPAP / SIMV ICU ventilators from Bird machines. At the same time, his interest in electronics led to a high frequency ventilator driver, a simple ventilator alarm and a number of peripheral nerve stimulators. A lifelong interest in anaesthetic equipment ensued.

Chris currently represents the College on international standards committees and has been a design consultant to several international equipment manufacturers. For the last 15 years he has worked with Dräger in Germany on the development of their latest anaesthetic machines. His primary clinical interests are neurosurgery, cerebral protection, ventilation optimisation with modern machines and monitoring.

Dr Laurence Weinberg

Department of Anaesthesia,
Austin Hospital, Melbourne

Dr Laurence Weinberg graduated from The University of Witwatersrand, South Africa in 1997. He worked in teaching hospitals in the United Kingdom for the following six years being awarded his membership of the Royal College of Physicians of London in 2002. He completed his anaesthesia training at the Austin Hospital in Melbourne with clinical fellowships in cardiac anaesthesia and liver transplantation. In 2007, he was appointed as a staff specialist, and continues to serve the Department of Anaesthesia at Austin Hospital. He is also a Senior Fellow in the Department of Surgery, University of Melbourne, where he is completing a Doctorate of Medicine degree.

His primary clinical and academic interests are in the areas of liver transplantation, major hepatobiliary and cardiothoracic surgery. He has been the recipient of numerous research grants and has lectured both nationally and internationally. Currently he is the principle investigator for seven randomised clinical trials, three of which are multicentre studies.

A/Prof Frank van Haren

Intensive Care Unit, The Canberra Hospital, Canberra,
Australian National University Medical School

Dr Frank van Haren completed his dual specialist training as Physician and Intensive Care Specialist in Nijmegen, The Netherlands in 2000. He has worked as an Intensive Care Specialist in large teaching hospitals in the Netherlands and lectured for the Dutch National Intensive Care training programme on topics such as sepsis, nutritional therapy, and ethics.

From 2005 he worked in the Waikato Hospital, Hamilton, New Zealand, before moving to Canberra, Australia in 2011, where he has been appointed Associate Professor with the ANU Medical School, Senior Staff Specialist in the ICU of the Canberra Hospital, and Director of the ICU research programme.

In 2010 he was awarded a PhD in Medical Sciences by the University of Nijmegen for his work on gastrointestinal perfusion and microcirculation in sepsis. Other research interests include the effects of hypertonic fluid resuscitation in sepsis, immunomodulation in sepsis, nutritional therapy, and non-conventional inotropes and vasopressors. He has published and presented on a wide range of topics, and is reviewer for several international medical journals.

A/Prof Peter Hebbard

Clinical A/Prof University of Melbourne

Peter Hebbard is Clinical Associate Professor and member of the steering committee of the Diploma of Clinical Ultrasound at the University of Melbourne. He was the convener of the ASURA 2012 meeting in Sydney and has been an invited speaker on regional anaesthesia in the USA, Europe and Asia over the last two years. In 2008 he won the award for Innovation in Ultrasound guided regional anaesthesia at the ISURA meeting in Toronto for innovations in abdominal wall blockade. Current interests include novel regional anaesthesia, pain medicine and other techniques relating to ultrasound guidance.

NEW ZEALAND FACULTY

Dr Jeanette Scott
Middlemore Hospital
Auckland

Dr Craig Birch
Middlemore Hospital
Auckland

Dr Tim Short
Auckland City Hospital
Auckland

Dr Matt Taylor
Middlemore Hospital
Auckland

Dr Ben Harris
Southern Community Laboratories
Christchurch

Dr Wayne Morriss
Christchurch Hospital
Christchurch

ABSTRACTS

AN UPDATE IN OBSTETRIC ANAESTHESIA

Clin A/Prof Nolan McDonnell

Department of Anaesthesia and Pain Medicine, King Edward Memorial Hospital for Women, Western Australia
School of Women's and Infants' Health, University of Western Australia, Crawley, Western Australia
School of Medicine and Pharmacology, University of Western Australia, Crawley, Western Australia

This talk aims to highlight recent developments and research in obstetric anaesthesia that may have an impact on our day to day care of women in the birthing suite, the operating theatre and the post operative environment.

Maternal Mortality

There is generally good news on the subject of maternal mortality. Worldwide there has been a 34% decrease in the number of women dying in pregnancy and childbirth since 1990, with haemorrhage remaining the leading cause of death worldwide.[1] In developed countries, key reports have emerged from both the UK and NZ. The 2006-2008 UK CMACE report documents a decreased number of maternal deaths, mainly due to a fall in deaths related to thromboembolism after vaginal birth and from haemorrhage.[2] Of note is that sepsis became the leading direct cause of maternal death and the report highlighted the need for modified early warning charts and prompt recognition with aggressive, skilled management of these women in a multidisciplinary environment. Also, the CMACE report highlighted the important position of anaesthetists in the management of unwell women, with close to 50% of deaths receiving care from anaesthetists.

New Zealand is to be commended on the development of its own perinatal and maternal mortality reporting system, the Perinatal and Maternal Mortality Review Committee (PMMRC). The sixth report of the PMMRC was released in June 2012 and documented a total of 57 deaths over 5 years.[3] New Zealand's Maternal Mortality Ratio was reported as 17.8 per 100,000 maternities, a rate higher than the UK (11.4 per 100,000). Whilst this may be of concern, the case ascertainment in New Zealand is likely to be close to 100%, meaning that almost all maternal deaths are captured, so the results should be interpreted within this context. Similar to other worldwide reports, older mothers and those from disadvantaged social or ethnic settings had a higher risk of dying. Eighteen deaths were thought to be potentially avoidable. Suicide was the leading cause of maternal death over this five year period, being responsible for nearly a quarter of all maternal deaths. The H1N1 pandemic was responsible for four maternal deaths in New Zealand in 2009.

How does Australia compare? Well, superficially the most recent MMR in Australia is better than New Zealand and the UK, at 8.4 per 100,000 maternities.[4] However this data is now dated, being from between 2003-2005. Reporting in Australia is hindered by there being over 300 sites for maternity care, and many deaths may occur in ICUs without obstetric services on site. In addition, the numerous states and territories have different reporting obligations, making the true MMR difficult to deduce at present. The next report will likely be a 5-year summary published in late 2012 or early 2013.

Analgesia for Labour and Delivery

Neuraxial Analgesia

Delivery Method – Infusions, bolus and mandatory bolus techniques

Patient controlled or midwife administered bolus techniques appear to be superior to continuous infusion techniques, most likely due to the better spread of the solution in the epidural space. Patient controlled techniques (PCEA) have a number of advantages over midwife-administered top ups, particularly in regards to the decreased staff workload and improved maternal satisfaction. A background infusion is often used in conjunction with a patient administered bolus, the optimal combination of background and bolus dose volume and concentration is open to debate. To further improve on the PCEA technique, device manufacturers have been adding "mandatory

intermittent bolus" or "programmed intermittent bolus" modes to their infusion pumps. These deliver a bolus of a set volume at a set interval, generally in addition to the patient's own demands. These new modes have been associated with less overall local anaesthetic use and less motor block.[5,6]

Ultrasound Assisted Placement

Ultrasound assisted neuraxial techniques have now been well described. The potential benefits include more accurate identification of the level of insertion, with one study noting that in over 40% of cases the level of insertion was at least one space higher than initially thought (which is really only of major importance with combined spinal-epidural techniques).[7] From our own departmental experience and research ultrasound is useful in the morbidly obese parturient who does not have clearly defined anatomical landmarks, as well as in women with difficult insertions. In these situations the ultrasound can identify not only space but also the angle and potential depth of insertion. Routine use is difficult to recommend at present and like most techniques a learning curve is present and practice on less complicated cases is recommended.

Addition of Clonidine and Neostigmine

A number of options have been investigated to enhance neuraxial labour analgesia. These options may seek to prolong the initial duration of analgesia (and hence decrease supplementation needs), decrease motor blockade and decrease the incidence of hypotension. A number of additives to traditional neuraxial opioids and local anaesthetic have been examined with clonidine and neostigmine receiving recent attention. Clonidine has been extensively investigated and is a commonly used neuraxial adjunct.[8] Neostigmine has undergone less investigation but appears safe. Interestingly, neostigmine causes severe nausea and vomiting if administered intrathecally but this does not appear to be an issue with epidural administration.[9] In labour analgesia, Van de Velde et al administered neostigmine (500 mcg) and clonidine (75 mcg) epidurally after performing combined spinal-epidural analgesia in labour.[10] The duration of initial analgesia was extended from 95 to 144 minutes and overall local anaesthetic consumption was less. Interestingly, nearly a quarter of the women in the neostigmine / clonidine group delivered before additional analgesia was required. This may serve to be a useful technique in the future but caution is advised on a number of levels: the mixing and dilution of drugs at the bedside raises sterility and error issues and this technique has a number of ethical and medico-legal implications.[11]

Remifentanil

Not all women in labour will be able to have an epidural or will want an epidural. Remifentanil provides a viable option and has been shown to be better than pethidine.[12] Uptake has been considerable in some institutions where it has become the primary method of labour analgesia.[13] However it requires very close monitoring with 1:1 midwifery care and continuous SpO_2 monitoring[13] and significant respiratory complications have been reported.[14] Maternal satisfaction is high even though pain relief is not as effective as neuraxial analgesia.[15] Neonatal metabolism is rapid, even in premature neonates.[16] Bolus and infusion regimens vary, although a 40 mcg bolus with no background infusion has been recommended.[17]

Caesarean Delivery

General Anaesthesia

Mortality – Regional versus General Anaesthesia

General anaesthesia has traditionally been associated with an increased maternal mortality and the most recent CMACE report highlights continued deaths associated with the management of general anaesthesia.[2] Of concern, one woman died despite the presence of a functioning epidural catheter. Continued attempts at intubation were made despite oxygenation initially being achieved from an ILMA. Long acting muscle relaxants were then given, she died without a surgical airway being attempted.

Hawkins published one of the most widely quoted papers highlighting the discrepancy in deaths between general versus regional anaesthesia.[18] This data led to the common quote of a mortality difference 16-17 times higher for general anaesthesia over regional anaesthesia. However, this data is now relatively dated and looked at cases prior to 1990. In the intervening period there have been a number of advances in monitoring and equipment as

well as education and protocols for the management of the difficult airway. It is perhaps not that surprising that when Hawkins looked at more recent data (1997-2002), there was no significant difference in mortality between general and regional anaesthesia.[19] Of note, deaths secondary to regional anaesthesia had increased whilst those secondary to general anaesthesia had decreased.

Head-up Position

Placing the pregnant women in a 30-degree head-up tilt has been shown to increase the FRC. Although the mean increase is only approximately 190 ml this may be of benefit in a difficult airway.[20] Unfortunately an increasing BMI seems to decrease the benefits from the head-up position. Despite this, it seems difficult not to recommend the adoption of a 30-degree head-up position prior to general anaesthesia in pregnancy.

Neonatal Outcomes

Traditionally it has been suggested that the effects of general anaesthesia on the neonate would be transient and of little concern when skilled resuscitation staff are present. Data from Brisbane has suggested, when controlled for confounders, that general anaesthesia for fetal distress is associated with lower Apgar scores at 5 min and with at least twice the risk of a NICU admission.[21] This data is in keeping with data from Sydney published in 2009 which showed markedly increased early neonatal morbidity with general anaesthesia, with the greatest impact being on already compromised babies.[22] Whilst failure to provide general anaesthesia may lead to poorer outcomes, it does highlight the need for close communication between all team members in the setting of fetal distress.

Prevention of Hypotension

Research into the prevention of hypotension under spinal anaesthesia has been focused on three main directions – vasopressors, fluid pre- or co-load and the use of lower concentrations of local anaesthetics ("low dose" spinals).

Vasopressors

Phenylephrine is now firmly established in obstetric anaesthesia with well documented benefits for neonatal acid base status and maternal blood pressure control.[23] Metaraminol has not received as much attention and comparative studies with phenylephrine are lacking. Metaraminol has some potential advantages, particularly with respect to some effect at beta receptors, which may maintain cardiac output better. Of importance is that the majority of studies to date are in the elective caesarean situation with an uncompromised utero-placental unit. It is unclear whether the stress fetus may respond differently to these medications.

Fluids

The take home message is that fluids alone have an unpredictable efficacy and there is a high likelihood that vasopressors will be required.[24] Preload is partly effective when a colloid is used but not when a crystalloid is used. Co-loading with either solution can be effective but again is unreliable.

Low Dose Spinals

A significant amount of research has been directed at lowering the dose of intrathecal local anaesthetic. Whilst it is clear that this decreases maternal hypotension and nausea, the incidence of intraoperative pain is unacceptable.[25] The ED95 of hyperbaric bupivacaine is approximately 11 mg, but many studies define "low" dose as < 8mg. When used in this fashion, hypotension is reduced by approximately 20% but there is nearly a 4-fold increase in the requirement for intraoperative analgesic supplementation.[25] Hypotension can readily be managed with vasopressors but intraoperative pain will often require conversion to general anaesthesia. In the future it is difficult to see the place of low dose spinals except in selected situations.

Post-Caesarean Pain Relief

The combinations and permutations of options for post caesar pain relief continue to grow dramatically. What should be emphasised is that institutions should use what works best in their unique setting, rather than trying to

aspire to what may appear to be a new "gold standard".[9] Recent research has been concentrated around the TAP block and the incidence of chronic post-caesarean pain. The place of the TAP block has now been relatively well defined and appears to be useful when long acting neuraxial opioids are not used (eg GA caesarean).[26] There is little benefit to adding a TAP block to epidural or intrathecal morphine. Chronic post-caesar pain is an emerging issue, with recent studies documenting an incidence of between 10-20%.[27, 28] Whilst much lower than other high risk surgical populations, given the comparatively large number of caesareans being performed (over 90,000 per annum in Australia), the potential burden is high. A recent study has shown that gabapentin 600 mg pre-caesarean, when combined with intrathecal morphine 100 mcg, decreased pain post operatively.[29] Whilst the study was not powered to look at chronic pain, the incidence in the placebo group was 20% and there was a trend to a lower incidence in the gabapentin group. It is too early to make widespread recommendations in regards to gabapentin as the neonatal effects when administered pre-delivery have not been well assessed, but it may be a useful adjunct post-delivery in high risk women, with sedation the major side effect.

Complications

Haemorrhage

rVIIa, Tranexamic Acid and Fibrinogen Concentrate

The use of rVIIa has anecdotally appeared to decrease in obstetric haemorrhage, most likely secondary to the lack of positive outcome data and the closure of the ANZ Haemostasis Registry. The Registry recently published the data on rVIIa in PPH which showed a "response" rate of 76%,[30] but this is a poor outcome measure and the reported response rate is similar to that reported in the placebo arm of at least three RCTs investigating rVIIa. The World Health Organization (WHO) also suggests that there is not enough evidence to currently recommend its use.[31] Antifibrinolytics such as tranexamic acid have a robust evidence base for the reduction of transfusion requirements outside of the obstetric setting.[32] Evidence is currently lacking in obstetric haemorrhage although the WOMAN trial, a large international study, is underway to examine its potential role. Interestingly, despite the relative lack of evidence, the WHO do recommend tranexamic acid as a potential therapeutic agent in post partum haemorrhage. Commonly used doses are 1 g intravenously, followed by another 1 g if additional bleeding issues are present from between 30 minutes to 24 hours after administration.

Fibrinogen concentrate, presented as a powder for reconstitution, is a very attractive option in obstetric haemorrhage, especially in resource limited areas and given the correlation between fibrinogen levels and the degree of haemorrhage.[33] It is not widely available in Australia currently (although Perth did have a special supply during the CHOGM meeting). Case series are promising and clinical trials are currently underway.[34-36]

Massive Transfusion Protocols and Monitoring of Coagulation

It has been recommended that massive transfusion protocols be in place in all obstetric hospitals.[37] Protocols vary but most use a relatively aggressive ratio of red cells to coagulation factors. Fibrinogen levels appear to correlate well with increasing volumes / severity of haemorrhage.[33] It is important to note that baseline levels of fibrinogen in pregnancy are higher than normal and hence using standard laboratory cut offs may not reflect the overall degree of fibrinogen deficiency. Experience is growing with the use of TEG and ROTEM in obstetric haemorrhage and normal values for pregnancy have been defined. Our own experience with ROTEM would suggest that it is a valuable tool in major haemorrhage cases, allowing targeted coagulation factor replacement.

Interventional Radiology

Interventional radiology in the acute management of PPH has been shown to have excellent results.[38] More controversial is the use of interventional radiology in the elective situation, despite it being recommended in some guidelines.[39] A robust evidence base is not currently present, reported data varies from showing potential benefit to no benefit and there is a lack of common outcome measures and heterogeneity in the case selection. Data from a retrospective review from Auckland where catheters were used in 14 women documented that of the 11 where the balloons were inflated, 9 required a hysterectomy, no benefit from inflation was seen in 4 and the median blood loss was 4,600 ml.[40] In the same edition of the journal a case report of a women who suffered multiple vascular complications after the prophylactic insertion of balloon catheters.[41]

Post-dural Puncture Headache (PDPH) and its Management

Management options for PDPH that have been shown to be effective include ACTH, neuraxial opioids, epidural blood patch and the placement of intrathecal catheters. However, hydration, bed rest, caffeine and NSAIDs are not thought to have much of a role in management. Gabapentin may be a relatively novel agent in the management of PDPH.[42] A blood patch performed within 24 hours is less likely to be effective. The optimal volume of blood for an epidural blood patch is still not clear. The most recent multi-centre study examined 15, 20 and 30 ml with partial relief being found in 61%, 73% and 67% respectively.[43] Complete relief was surprisingly low at just 32% in the 20 ml group. Just under half of the patients in the 30 ml group did not receive the allocated volume secondary to back pain. The authors recommend aiming for 20 ml when performing an epidural blood patch. Another study examined the optic nerve sheath diameter as a measure of intra-cranial pressure with epidural blood patching and found that successful relief of PDPH was associated with evidence of a raised intracranial pressure, suggesting this as a possible mechanism of action of an epidural blood patch.[44]

Stroke

Anaesthetists should be fully aware of the differential diagnosis of headache in a pregnant or recently pregnant women, especially vascular causes and cerebral venous thrombosis.[45] The incidence of stroke in pregnancy has increased by at least 50% in recent times[46] with identified risk factors being hypertension, heart disease, obesity, diabetes and clotting disorders. Guidelines are now available for the management of cerebral venous thrombosis.[47]

VTE Prophylaxis

Thromboembolism has traditionally been the leading cause of direct maternal mortality in the UK triennial reports but in the most recent report there was an over 50% decrease in the number of deaths.[2] The decrease is generally put down to the adherence to risk assessment and management guidelines, with those of the RCOG being the most widely used throughout the UK.[48] Interestingly, the reduction in deaths was primarily in antenatal women and those after a vaginal delivery, with little change in the number of women dying after caesarean delivery. Australasian consensus guidelines have now been published.[49,50] The key take home messages are that pharmacological prophylaxis is recommended in most non elective caesareans and in elective caesareans with one additional risk factor. Important risk factors for the anaesthetist include post-partum haemorrhage, which dramatically increases the VTE risk. In addition, obesity appears to increase the risk with a higher risk with higher BMIs. Dosing guidelines for the obese obstetric patient are available but are based on expert opinion rather than evidence from clinical trials.[48]

References

1. Paxton A, Wardlaw T. Are We Making Progress in Maternal Mortality? New England Journal of Medicine. 2011; 364: 1990-3.
2. Cantwell R, Clutton-Brock T, Cooper G, et al. Saving Mothers' Lives: Reviewing maternal deaths to make motherhood safer: 2006-2008. The Eighth Report of the Confidential Enquiries into Maternal Deaths in the United Kingdom. BJOG : an international journal of obstetrics and gynaecology. 2011; 118 Suppl 1: 1-203.
3. PMMRC. 2012. Sixth Annual Report of the Perinatal and Maternal Mortality Review Committee. Reporting mortality 2010. Wellington: Health Quality & Safety Commission 2012.
4. Sullivan, EA, Hall, B, King, JF. Maternal deaths in Australia 2003–2005. AIHW National Perinatal Statistics Unit 2007;Maternal deaths series no. 3. Cat. no. PER 42.
5. Leo S, Ocampo CE, Lim Y, Sia AT. A randomized comparison of automated intermittent mandatory boluses with a basal infusion in combination with patient-controlled epidural analgesia for labor and delivery. International journal of obstetric anesthesia. 2010; 19: 357-64.
6. Capogna G, Camorcia M, Stirparo S, Farcomeni A. Programmed intermittent epidural bolus versus continuous epidural infusion for labor analgesia: the effects on maternal motor function and labor outcome. A randomized double-blind study in nulliparous women. Anesth Analg. 2011; 113: 826-31.
7. Lee AJ, Ranasinghe JS, Chehade JM, et al. Ultrasound assessment of the vertebral level of the intercristal line in pregnancy. Anesth Analg. 2011; 113: 559-64.

8. Paech MJ, Pavy TJ, Orlikowski CE, Evans SF. Patient-controlled epidural analgesia in labor: the addition of clonidine to bupivacaine-fentanyl. Reg Anesth Pain Med. 2000; 25: 34-40.
9. McDonnell NJ, Keating ML, Muchatuta NA, Pavy TJ, Paech MJ. Analgesia after caesarean delivery. Anaesthesia and intensive care. 2009; 37: 539-51.
10. Van de Velde M, Berends N, Kumar A, et al. Effects of epidural clonidine and neostigmine following intrathecal labour analgesia: a randomised, double-blind, placebo-controlled trial. International journal of obstetric anesthesia. 2009; 18: 207-14.
11. Paech M, Pan P. New recipes for neuraxial labor analgesia: simple fare or gourmet combos? International journal of obstetric anesthesia. 2009; 18: 201-3.
12. Schnabel A, Hahn N, Broscheit J, et al. Remifentanil for labour analgesia: a meta-analysis of randomised controlled trials. European journal of anaesthesiology. 2012; 29: 177-85.
13. Hill D. The use of remifentanil in obstetrics. Anesthesiology clinics. 2008; 26: 169-82, viii.
14. Bonner JC, McClymont W. Respiratory arrest in an obstetric patient using remifentanil patient-controlled analgesia*. Anaesthesia. 2012; 67: 538-40.
15. Ismail MT, Hassanin MZ. Neuraxial analgesia versus intravenous remifentanil for pain relief in early labor in nulliparous women. Arch Gynecol Obstet. 2012.
16. Welzing L, Ebenfeld S, Dlugay V, Wiesen MH, Roth B, Mueller C. Remifentanil degradation in umbilical cord blood of preterm infants. Anesthesiology. 2011; 114: 570-7.
17. Hinova A, Fernando R. Systemic remifentanil for labor analgesia. Anesth Analg. 2009; 109: 1925-9.
18. Hawkins JL, Koonin LM, Palmer SK, Gibbs CP. Anesthesia-related deaths during obstetric delivery in the United States, 1979-1990. Anesthesiology. 1997; 86: 277-84.
19. Hawkins JL, Chang J, Palmer SK, Gibbs CP, Callaghan WM. Anesthesia-related maternal mortality in the United States: 1979-2002. Obstetrics and gynecology. 2011; 117: 69-74.
20. Hignett R, Fernando R, McGlennan A, et al. A randomized crossover study to determine the effect of a 30 degrees head-up versus a supine position on the functional residual capacity of term parturients. Anesth Analg. 2011; 113: 1098-102.
21. Beckmann M, Calderbank S. Mode of anaesthetic for category 1 caesarean sections and neonatal outcomes. Aust N Z J Obstet Gynaecol. 2012.
22. Algert CS, Bowen JR, Giles WB, Knoblanche GE, Lain SJ, Roberts CL. Regional block versus general anaesthesia for caesarean section and neonatal outcomes: a population-based study. BMC Med. 2009; 7: 20.
23. Ngan Kee WD. Prevention of maternal hypotension after regional anaesthesia for caesarean section. Current opinion in anaesthesiology. 2010; 23: 304-9.
24. Mercier FJ. Fluid loading for cesarean delivery under spinal anesthesia: have we studied all the options? Anesth Analg. 2011; 113: 677-80.
25. Rucklidge MW, Paech MJ. Limiting the dose of local anaesthetic for caesarean section under spinal anaesthesia--has the limbo bar been set too low? Anaesthesia. 2012; 67: 347-51.
26. McDonnell NJ, Paech MJ. The transversus abdominis plane block and post-caesarean analgesia: are we any closer to defining its role? International journal of obstetric anesthesia. 2012; 21: 109-11.
27. Kainu JP, Sarvela J, Tiippana E, Halmesmaki E, Korttila KT. Persistent pain after caesarean section and vaginal birth: a cohort study. International journal of obstetric anesthesia. 2010; 19: 4-9.
28. Sng BL, Sia AT, Quek K, Woo D, Lim Y. Incidence and risk factors for chronic pain after caesarean section under spinal anaesthesia. Anaesthesia and intensive care. 2009; 37: 748-52.
29. Moore A, Costello J, Wieczorek P, Shah V, Taddio A, Carvalho JC. Gabapentin improves postcesarean delivery pain management: a randomized, placebo-controlled trial. Anesth Analg. 2011; 112: 167-73.
30. Phillips LE, McLintock C, Pollock W, et al. Recombinant activated factor VII in obstetric hemorrhage: experiences from the Australian and New Zealand Haemostasis Registry. Anesth Analg. 2009; 109: 1908-15.
31. World Health Organization, Department of Reproductive Health and Research. WHO guidelines for the management of postpartum haemorrhage and retained placenta 2009.
32. Ker K, Edwards P, Perel P, Shakur H, Roberts I. Effect of tranexamic acid on surgical bleeding: systematic review and cumulative meta-analysis. Bmj. 2012; 344: e3054.
33. de Lloyd L, Bovington R, Kaye A, et al. Standard haemostatic tests following major obstetric haemorrhage. International journal of obstetric anesthesia. 2011; 20: 135-41.
34. Glover NJ, Collis RE, Collins P. Fibrinogen concentrate use during major obstetric haemorrhage. Anaesthesia. 2010; 65: 1229-30.
35. Mercier FJ, Bonnet MP. Use of clotting factors and other prohemostatic drugs for obstetric hemorrhage. Current opinion in anaesthesiology. 2010; 23: 310-6.

36. Bell SF, Rayment R, Collins PW, Collis RE. The use of fibrinogen concentrate to correct hypofibrinogenaemia rapidly during obstetric haemorrhage. International journal of obstetric anesthesia. 2010; 19: 218-23.
37. Shields LE, Smalarz K, Reffigee L, Mugg S, Burdumy TJ, Propst M. Comprehensive maternal hemorrhage protocols improve patient safety and reduce utilization of blood products. American journal of obstetrics and gynecology. 2011; 205: 368 e1-8.
38. Royal College of Obstetricians and Gynaecologists. The role of emergency and elective interventional radiology in postpartum haemorrhage. Royal College of Obstetricians and Gynaecologists Good Practice Guideline No. 6; 2007.
39. Royal College of Obstetricians and Gynaecologists. Placenta praevia, placenta praevia accreta and vasa praevia: diagnosis and management. Green Top Guideline No. 27; January 2011.
40. Thon S, McLintic A, Wagner Y. Prophylactic endovascular placement of internal iliac occlusion balloon catheters in parturients with placenta accreta: a retrospective case series. International journal of obstetric anesthesia. 2011; 20: 64-70.
41. Bishop S, Butler K, Monaghan S, Chan K, Murphy G, Edozien L. Multiple complications following the use of prophylactic internal iliac artery balloon catheterisation in a patient with placenta percreta. International journal of obstetric anesthesia. 2011; 20: 70-3.
42. Wagner Y, Storr F, Cope S. Gabapentin in the treatment of post-dural puncture headache: a case series. Anaesthesia and intensive care. 2012; 40: 714-8.
43. Paech MJ, Doherty DA, Christmas T, Wong CA. The volume of blood for epidural blood patch in obstetrics: a randomized, blinded clinical trial. Anesth Analg. 2011; 113: 126-33.
44. Dubost C, Le Gouez A, Zetlaoui PJ, Benhamou D, Mercier FJ, Geeraerts T. Increase in optic nerve sheath diameter induced by epidural blood patch: a preliminary report. British journal of anaesthesia. 2011; 107: 627-30.
45. Klein AM, Loder E. Postpartum headache. International journal of obstetric anesthesia. 2010; 19: 422-30.
46. Kuklina EV, Tong X, Bansil P, George MG, Callaghan WM. Trends in pregnancy hospitalizations that included a stroke in the United States from 1994 to 2007: reasons for concern? Stroke. 2011; 42: 2564-70.
47. Saposnik G, Barinagarrementeria F, Brown RD, Jr., et al. Diagnosis and management of cerebral venous thrombosis: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2011; 42: 1158-92.
48. Nelson-Piercy C. Guideline No. 37. Reducing the risk of thrombosis and embolism during pregnancy and the puerperium. London: Royal College of Obstetricians and Gynaecologists, 2009.
49. McLintock C, Brighton T, Chunilal S, et al. Recommendations for the diagnosis and treatment of deep venous thrombosis and pulmonary embolism in pregnancy and the postpartum period. The Australian & New Zealand journal of obstetrics & gynaecology. 2012; 52: 14-22.
50. McLintock C, Brighton T, Chunilal S, et al. Recommendations for the prevention of pregnancy-associated venous thromboembolism. The Australian & New Zealand journal of obstetrics & gynaecology. 2012; 52: 3-13.

AIRWAY UPDATE

Dr Jeanette Scott

Middlemore Hospital
Auckland

This talk will discuss three topics that are proving to be influential in current airway management. Firstly, I will briefly mention one of the most important and prominent publications in recent years, the 4th National Audit of the Royal College of Anaesthetists on Major Complications of Airway Management (NAP4).[1] Secondly, I will discuss the difficulties of establishing the proper role of videolaryngoscopy (VL) in airway management and airway algorithms. And finally, I will discuss the now trendy use of nasal prongs to facilitate apnoeic oxygenation during intubation.

Lessons from NAP4

Given the size of NAP4 and its heavy presence in the literature, today I simply aim to either introduce or remind the audience of what this audit involved, and give three examples of how its findings influence current clinical practice and education.

During the year 2008-2009, the Royal College of Anaesthetists, in a joint project with the Difficult Airway Society (DAS), boldly attempted to capture every single major complication from airway management that occurred in the United Kingdom. By "major", we mean catastrophic – that is, death, brain damage, emergency surgical airway or intensive care unit (ICU) admission that resulted directly from airway management. All 309 NHS hospitals were involved, with appointed NAP4 representatives in 286 anaesthesia department to facilitate the capturing of cases. In addition to anaesthesia-related airway events, events that occurred in the emergency department (ED) or ICU were also included.

Once a case was identified, it was reported by means of an online questionnaire. The questionnaire had 164 questions; a mixture of multi-choice and free-text boxes. This arduous case-logging process is both the greatest strength and the greatest weakness of the NAP4 report – it provided an enormously detailed case series, however it is estimated that as many as 3 in 4 cases may have been missed.

Nevertheless, 286 cases were reported – 184 of which reached inclusion criteria. Each case was then reviewed twice by an expert committee, which included representatives from twelve relevant bodies of medical professionals, who looked for causes and themes.

To be able to calculate incidences current denominator data is required. To this end, in addition to the case series, a two-week UK-wide census was performed that aimed to record every single anaesthesia-led airway management event. The data was then extrapolated to estimate annual figures. No census was performed for ED or ICU environments.

In March 2011 the NAP4 committee published their findings in a 218 page report. The 184 cases of major airway complications led to 176 recommendations. One criticism frequently made is that in some situations the committee stepped beyond the evidence when making recommendations. However the committee makes no apologies for this, stating that the "authors and editors have taken as broad a view as possible in producing learning points in an attempt to maximise the value of the report" and as such "they represent a combination of literature interpretation, case review and expert opinion."

In my opinion, three important NAP4 findings include –

1) <u>The routine use of capnography in ICU and ED, and its ready availability in the recovery room, is the single change with the greatest potential to prevent disasters like those reported to NAP4</u>

In the ICU setting, failure to use capnography was implicated in 17 (82%) of events leading to death or brain damage. There were four cases in which an absence of capnography in the recovery room was said to be

contributory. Correct use and interpretation of capnography would have likely prevented half of the deaths in the ED (2 cases). There were seven deaths caused by unrecognised oesophageal intubation, two of which occurred in the operating room despite the presence of mandatory capnography. These two patients so rapidly deteriorated to cardiac arrest that the flat capnography trace was attributed to the absence of cardiac output. This serves as a reminder that when a patient is receiving CPR, the capnograph should be of low amplitude, not flat. A flat capnograph either means the ET is not in the oesophagus, or it is occluded (eg by a blood clot or mucus).

In the UK continuous capnography is now considered to be standard of care for every patient who is intubated, receiving moderate or deep sedation, or ACLS.[2,3] The European Board of Anaesthesiology now recommends that all patients with intubated tracheas should be monitored with continuous capnography, be they in operating theatres, ICUs or EDs, or outside hospital.[4]

2) Aspiration was the most common cause of airway-related deaths at the hands of anaesthetists

Aspiration accounted for 17% of primary anaesthetic airway problems and 50% of anaesthesia deaths. It is recommended that aspiration risk be routinely documented for every patient, and when ambiguity exists, a more serious aspiration risk should be presumed. The most common situation in which aspiration occurred was during the maintenance phase of laryngeal mask anaesthesia in patients with risk factors for aspiration. There were a number of cases where rapid sequence intubation (RSI) was clearly indicated but not used. NAP4 recommends that RSI should still be taught despite its limitations – in particular cricoid force can be abandoned if intubation is difficult. Second generation laryngeal masks that feature oesophageal drainage ports were rarely used (10% of all supra-glottic airways), however NAP4 proposes that these might be a safer choice if used in patients judged to have a small, but not zero, aspiration risk.

3) Anaesthetists were poor at performing emergency cricothyroidotomy

There were 58 emergency cricothyroidotomies performed in the operating room, and only 16% of them failed. However, of the 25 attempted by anaesthetists, 16 (64%) failed, 11 of which were thankfully rescued by surgical colleagues. There are two confounding factors worth considering – the first is that anaesthetists were probably forced to attempt cricothyroidotomy in the more serious upper airway obstructions, as, if time allowed, one imagines most anaesthetists would wait for a surgeon to arrive. Secondly, anaesthetists almost exclusively chose techniques involving a narrow-lumen cricothyroid cannulae (which was either ventilated down using a high pressure source, or was the first stage in a Seldinger technique to introduce a wider bore airway). In contrast surgeons preferred an open technique using a scalpel and / or blunt dissection to open the airway. All airway practitioners should be prepared to perform a surgical airway if required. The reasons for, and possible remedy of, the poor performance of emergency cricothyroidotomies by anaesthetists must be addressed.[5]

These three points, while important, do not do justice to the breadth and wisdom of the other lessons and recommendations within the NAP4 report. The original NAP4 document can be found in its entirety, free of charge online. Recommendations are grouped by topic, discussed with clear reasoning and are illustrated by chilling case examples. The NAP4 committee has also created educational podcasts that are available free from iTunes or the NAP4 website.

The Role of Videolaryngoscopy in Airway Management

Videolaryngoscopy (VL) has been met with enormous enthusiasm. As a rescue device after failed DL, there are now numerous papers citing greater success rates than what has historically been reported with persistent DL.[6] However, unlike DL, the weaknesses of VL are poorly understood. If VL is to become the routine first-choice laryngoscope, then it should have a proven higher success rate and lower complication rate than DL. If VL is to be selectively used, then we should be clear in what situations VL is helpful, and in which cases another device is likely to be more successful.

Evidence-based conclusions about VL are hampered by –

1. The absence of a standardised method of describing the level of difficulty of VL intubation (ie unlike DL, a Cormack-Lehane grade I or II view does not consistently mean an easy intubation)
2. The diversity of instruments on the market
3. The limited number of prospective studies involving patients with truly difficult airways, due to the ethical limitations of such studies and the rarity of such patients

The largest VL case series to date was published by Aziz and colleagues in Anesthesiology last year.[7] In their retrospective audit on the use of the Glidescope (GS) in two institutions over two years, they looked at 71,500 intubations, which included 2,044 GS intubations.

Aziz reported that in 576 cases of patients without predictors of difficult DL, the GS failed to achieve intubation 2% of the time. In 1,428 cases of patients with features of predicted difficult DL, the GS failed 4% of the time. When the features of the 60 failed GS patients were analysed, somewhat surprisingly the presence of reduced tissue mobility (such as patients with neck scars, radiation or masses) was the highest predictor of failure, followed by having a thick neck, a short TM distance and reduced cervical neck motion. What is clear from this study is that predictors of difficult DL may cross over to also be predictors of difficult VL. Furthermore, when VL failed, the most common form of rescue was DL (39%), followed by fibreoptic intubation (23%). In my view this study suggests that VL should still be considered as synergistic with, and not a replacement for, advanced DL and fibreoptic skills.

International difficult airway algorithms call for the use of an alternate device when DL has failed, but there is currently no specific mention of the role of VL.[8,9,10] The American Society of Anesthesiologists, Canadian Airway Focus Group, and the ANZCA Airway SIG are all currently working separately on rewriting guidelines for management of the difficult airway, and it will be interesting to see what conclusions will be made about VL. In the meantime I think that it is best to stick to what we already know. Firstly, in cases of failed intubation the provider must choose an alternate but familiar approach that addresses the reason, anatomic or otherwise, for failure of the primary approach. And secondly, prolonged and multiple attempts at intubation cause patient harm and should be abandoned in favour of other methods of oxygenation.

Apnoeic Oxygenation with Nasal Prongs

Finally, I would like to talk about a small and inexpensive trick that may improve safety during intubation – the use of high-flow oxygen via standard nasal prongs.

Quite simply, standard nasal prongs are placed on the patient and oxygen is delivered continuously during otherwise standard airway management. If the patient is conscious, they are set to 4-6 litres per minute. The patient is pre-oxygenated in the usual fashion via a face-mask that is placed over the nasal cannula. Once the induction drugs are given, or if the patient is already unconscious, the nasal oxygen is turned up to 10-15 litres per minute. If required, bag-mask ventilation is performed with oxygen supplied by both the face-mask and nasal prongs. If the patient is having an RSI, a good jaw thrust and continuous application of the face-mask should be used while fasciculations are waited for, in order to facilitate continuous mass diffusion of oxygen into the lungs. Intubation is then attempted with oxygen still flowing through the nasal cannula. Obviously, for this to work, a patent upper airway is needed, and in some cases nasal trumpets (nasal airways) may be used. The few small studies supporting this practice describe a delay in the time till oxygen desaturation by 1.5-3 minutes in normal[11] and obese[12,13] patients. Some practitioners now argue that the traditional practice of removing all oxygen sources during airway management cannot be justified with such a cheap and easy alternative.

References

1. Cook T, Woodall N, Frerk C. 4th National Audit of the Royal College of Anaesthetists and the Difficult Airway Society: Major complications of airway management 2011. http://www.rcoa.ac.uk/nap4
2. AAGBI. AAGBI Safety Statement. The use of capnography outside the operating theatre (updated). May 2011. http://www.aagbi.org/sites/default/files/Capnographyaagbi090711AJH%5B1%5D_1.pdf
3. Thomas AN, Harvey DJR, Hurst T. Standards for Capnography. London: Intensive Care Society, 2011. http://www.ics.ac.uk/event_documents/capnography_revision
4. European Section and Board of Anaesthesiology UEMS. EBA recommendations. June 2011. http://www.eba-uems.eu/recommend
5. Hung O, Scott J, Mullen T, Murphy M. Waiting to exhale! Anesth Analg. 2012 May;114(5):927-8
6. Crosby, ET. An evidence-based approach to airway management: is there a role for clinical practice guidelines? Anaesthesia, 2011. 66 Suppl 2: p. 112-8
7. Aziz M, Healy D, Kheterpal S, Fu R, Dillman D, Brambrink A. The Routine Clinical Practice Effectiveness of the Glidescope in Difficult Airway Management: An Analysis of 2,004 Glidescope Intubations, Complications, and Failures from Two Institutions Anesthesiology 2011; 114:34–41

8. American Society of Anesthesiologists Task Force on Management of the Difficult Airway: 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
9. Henderson JJ, Popat MT, Latto IP, Pearce AC. Difficult Airway Society guidelines for management of the unanticipated difficult intubation. Anaesthesia 2004; 59: 675-94
10. Crosby ET, et al. The unanticipated difficult airway with recommendations for management. Can J Anaesth, 1998. 45(8): p. 757-76
11. Taha SK, Siddik-Sayyid SM, El-Khatib MF, Dagher CM, Hakki MA, Baraka AS. Nasopharyngeal oxygen insufflation following preoxygenation using the four deep breath technique. Anaesthesia 2006;61:427-30
12. Ramachandran SK, Cosnowski A, Shanks A, Turner CR. Apneic oxygenation during prolonged laryngoscopy in obese patients: a randomized, controlled trial of nasal oxygen administration. Journal of Clinical Anesthesia 2010; 22: 164-168
13. Baraka AS, Taha SK, Siddik-Sayyid SM, et al. Supplementation of pre-oxygenation in morbidly obese patients using nasopharyngeal oxygen insufflation. Anaesthesia 2007;62:769-73

MANAGING THE OBESE – LESSONS FROM BARIATRIC SURGERY

Dr Craig Birch

Middlemore Hospital
Auckland

Obesity has reached epidemic proportions globally and it has become one of the biggest challenges facing healthcare today. The incidence of obesity has tripled in the developed world over the past 25 years. Well over one third of adults in developed countries are obese and the data regarding children is even more sobering.[1] New Zealand is unfortunately at the forefront of this epidemic, regularly ranking in the top 10 countries in the obesity world. The crisis now involves the developing world and the prevalence of severe and morbid obesity is increasing more rapidly than other degrees of obesity.[2]

Obesity is defined as a body mass index of more than 30 and severe (morbid) obesity as greater than 40. Body mass index is calculated as weight in kilogram / (height in metres squared): and expressed in the units kg/m^2.

Obesity impacts on nearly every organ system, resulting in a multitude of co-morbidities and issues that are important to anaesthetists in the perioperative period. These include airway management, obstructive sleep apnoea, obesity hypoventilation syndrome, postoperative respiratory failure, postoperative pneumonia, metabolic syndrome, diabetes mellitus, cardiac failure, ischaemic heart disease, hypertension, dyslipidaemia, hypercoagulability and venous thromboembolism and pharmacokinetics of anaesthetic agents.

Bariatric surgery is emerging as the only sustainable option for managing the obesity epidemic, with a favourable impact on all of the related co-morbidities.[2] There is increasing evidence that bariatric surgery provides a survival benefit over time, presumably due to the dramatic improvement in almost all co-morbidities associated with obesity. The cost effectiveness of the surgery to the health system is one of the reasons that our health department has embraced the surgery.

The incidence of obesity in the South Auckland population is alarmingly high, and our hospital has developed a bariatric surgery service to deal with the problem. We perform approximately 160 bariatric procedures annually. The service has published outcomes over the past three years.[3,4,5]

I aim to outline some of the perioperative issues related to caring for these patients, including relevant evidence from the literature.

Bariatric Surgery

In spite of all the challenges associated with anaesthetising obese patients, the fact remains that thousands of bariatric patients are undergoing surgery around the world each year, and it is relatively safe, especially from an anaesthetic perspective.[6] The fact that the surgery is so successful has resulted in a dramatic increase in the numbers of bariatric procedures worldwide.

The 30-day mortality ranges from 0.1% to 2%. Mortality rates depend on several factors – complexity of the surgery, patient co-morbidities, experience of the surgeon and the centre. Gastric banding has the lowest mortality rate (0.1%), but a high rate of long-term complications with the least favourable weight loss. Gastric sleeves have a mortality rate of 0.3-0.5%, but fewer late complications with a very favourable weight loss record, Gastric bypasses have a slightly higher mortality at 0.5%[6] but very good weight loss.

The perioperative complications are mostly surgical in nature, the most feared being an anastomotic leak (1-2.5%), and the other being a haemorrhage (1%). Pulmonary emboli occur in < 0.5% and postoperative respiratory dysfunction occurs in 0.6% of patients. Obesity is associated with atherosclerotic disease, left ventricular hypertrophy and cardiomyopathy however the incidence of perioperative coronary events and cardiac failure is very low.[6] The bariatric patients at very high risk of cardiac events are presumably screened out and advised against surgery. A recent review of 400 patients published by my hospital revealed one death, on day 19, which was of unknown cause and thought to be a cardiac arrhythmia.

Strategies used to minimise perioperative problems in bariatric surgery include –[5]

1. Thorough preoperative preparation including psychological preparation, exercise programs, nutrition with dietician involvement, and a three week anorectic supplementation preoperatively. No smoking and minimal alcohol in take
2. Laparoscopic surgery rather than open surgery
3. Intravenous glucocorticoids
4. Active warming
5. Multimodal analgesia thereby minimizing opiates
6. Head-up position post-op (30 degrees)
7. Most problems manifest in the first two hours post-op, ie in PACU. Pay close attention to patients during this time[7]
8. Postoperative O_2 is used until SpO_2 levels are back to normal
9. Early mobilisation (staff need help with large patients!)
10. Early physiotherapy
11. Early enteral fluid / feeding
12. Thromboprophylaxis with foot pumps, calf compression devices and low molecular weight heparin
13. Minimal use of drains / nasogastric tubes

A number of centres have published fast track or ERAS (enhanced recovery after surgery) programme where bariatric patients are being discharged close to 24 hours after the surgery. These programmes aim to optimise patient care during the perioperative period thereby minimising morbidity and also decreasing time in hospital. My institution has just completed a randomised controlled trial of ERAS for bariatric surgery and showed that using a comprehensive ERAS programme reduces hospital stay with no increase in morbidity. Patients randomised to the ERAS group went home on the day after their laparoscopic sleeve gastrectomy. The studies from the US and Europe concur that "expert" teams caring for these patients regularly is one of the key factors for maintaining patient safety.[7,8]

Obstructive Sleep Apnoea (OSA)

This is one of the most challenging of the perioperative issues facing anaesthetists. The obese population has a much higher than normal incidence of OSA (70-80% in bariatric patients).[9] Despite a better understanding of the pathophysiology of OSA, there are no validated management strategies for these patients. Most patients with OSA have not been formally diagnosed and while there are many theoretical benefits of preoperative sleep studies and subsequent CPAP there is no evidence that this improves perioperative outcomes, and it is not feasible to test all the patients.

There are several preoperative scoring tools to ascertain the severity of OSA and therefore guide referrals. An example is the STOP-BANG questionnaire formulated by Chung et al[10] which includes four questions with yes / no answers – Snoring, Tiredness during the daytime, Observed apnoea and high blood Pressure. Sensitivity is improved by incorporating questions regarding – BMI, Age, Neck circumference and Gender. A positive screen (more than three yes answers) indicates a high risk. This is validated for screening severe OSA, but not for ascertaining perioperative risk.

Other dilemmas include the postoperative setting and monitoring of these patients, when to send patients home, and whether perioperative risk is altered by anaesthetic technique (eg inhalational verse intravenous). The answers to these questions are not clear from the literature and we are guided by expert opinion and our own experience.[11]

Our threshold for referring the suspected OSA patients for polysomnography studies has risen with experience as we have found that the majority of patients with mild / moderate OSA get through their bariatric surgery with no complications. Weingarten et al set out to determine whether there is an association between perioperative complications and the severity of OSA in bariatric patients and they found no association.[12] Ahmad et al showed that in morbidly obese patients, in the first 24 hours after laparoscopic bariatric surgery, OSA did not increase the risk of hypoxaemia.[13]

Obesity Hypoventilation Syndrome (OHS)

It is imperative to ascertain which patients have a particularly high risk of perioperative problems, and these would certainly include the patients with OHS. This effects 0.15-0.3% of the general population but up to 8% of the bariatric surgery population.[13]

OHS is defined as a combination of obesity and chronic daytime hypoxaemia (P_{O_2} < 65mmHg) and hypoventilation(P_{CO_2} > 45mmHg) in patients without COPD or other causes for hypoventilation. These patients have worsened airway obstruction, a blunted respiratory drive, restrictive chest physiology, and they may develop pulmonary hypertension, right ventricular hypertrophy, and eventually right-sided failure. It is useful to screen patients by checking oxygen saturation, blood Hb levels (polycythaemia is a clue to chronic hypoxia) and perform blood gas analysis if these are concerning. Blood HCO_3 levels are useful for picking up chronic hypercapnia. Once the diagnosis is made, ECG, CXR, echo and sleep studies may be necessary to assess the degree of OHS. These patients should be managed with perioperative CPAP if warranted.

Patients with OHS experience higher morbidity and mortality than those who are obese with eucapnia. Surgical mortality rate in high risk OHS patients undergoing gastric bypass surgery is higher (2-8%) than the usual risk of 0.5-2% in other bariatric surgery patients.[14]

Monitoring

There is no evidence that obese patients or those with OSA require more invasive perioperative monitoring than other patients. The type of surgery and co-morbidities dictate the intensity of monitoring, and we very rarely use arterial lines for our bariatric patients undergoing laparoscopic sleeve gastrectomies.[15]

Pharmacokinetics of Obesity

With increasing obesity, fat mass accounts for an increased percentage of total body water and the lean body weight (LBW) / total body weight (TBW) ratio decreases. The majority of the cardiac output is still directed to the vessel rich or lean tissue groups. Therefore administration of a drug based on TBW in an obese patient may result in an overdose. Ideal body weight can be calculated according to BMI and height, but this has shortcomings as obese people often have a high LBW for their height, and so this may result in under dosing. LBW is the TBW minus the fat mass. This is ideal for calculating most anaesthetic drug doses, however it is difficult to accurately measure under normal clinical circumstances, and the equations proposed to calculate it are estimates based on gender and BMI and are therefore not particularly accurate.[16]

It is useful to have an understanding of the pharmacokinetic principles and then follow guidelines for the different groups of anaesthetic drugs –

- Induction doses of propofol should be based on approximate LBW rather than TBW, although in practice this is titrated to effect. Infusions of propofol should be based on TBW, as the drug is so lipophilic that it distributes rapidly from the plasma to peripheral tissues, including fat.[1,15]

- Dexmedetomidine is useful as an adjunct for anaesthesia and analgesia in morbidly obese patients. It reduces opiate requirements postoperatively.[16] The pharmacokinetic effects are yet to be described in morbid obesity, however we have found that giving a loading dose of 0.5mcg/kg over 10 minutes, followed by an infusion of between 0.2 and 0.4mcg/kg/hr is effective.

- Remifentanil should be dosed according to IBW or LBM. Fentanyl is the most commonly used drug and it has been suggested that the dose be based on LBM, one study suggested that a pharmacokinetic correction is made when using fentanyl, and for patients weighing 140kg to 200kg the pharmacokinetic mass should be 100 to 108kg.[17]

- Non-depolarising neuromuscular blockers are polar hydrophilic drugs and their volume of distribution is limited to the vessel rich organs, meaning that we should use IBW to calculate doses for these agents. Succinylcholine, on the other hand needs to be dosed based on TBW. This is thought to be due to the

excess of pseudocholinesterase in obese patients, and an increase in ECF. Studies have shown better intubating conditions after a dose based on TBW than IBW, or LBW.[16]

- Sevoflurane and desflurane are the obvious volatile agents to use because of their lower solubility in fat.
- The use of target–controlled intravenous infusion (TCI) systems in anaesthesia is based on normal weight subjects and some of the models (eg the Marsh TCI model for propofol) yield unacceptable concentrations for morbidly obese patients. One needs to adjust the TCIs based on the principles we discussed, and bare in mind that the TCI may not be accurate. We await models based on morbidly obese patients for accurate delivery of TCIs.[16]

Metabolic Syndrome and Diabetes

This is a distinct obesity related syndrome characterised by truncal obesity, insulin resistance, dyslipidaemia and hypertension. It is important for anaesthetists to recognise these patients, as there is an increased incidence of coronary artery disease, congestive heart failure, obstructive sleep apnoea, pulmonary dysfunction and deep venous thrombosis in these patients. We need to concentrate on all these associated risk factors and try to minimise patient's perioperative risk with relevant preoperative work-up and careful intra and postoperative management.[18]

Airway Management

It is well known that obesity is associated with difficulty in airway management, both in terms of bag mask ventilation, and intubation. The NAP4 audit of airway morbidity and mortality in the UK showed that obese patients were over represented with 45% of patients having obesity listed as one of the reasons for the complication.[19]

There are good explanations for why morbidly obese patients desaturate and become hypoxic. It is important to minimise the problems at induction and extubation with positioning, planning and preparation of equipment and staff.

Positioning the patients in a "ramped" head-up position has been shown to decrease the desaturation at induction, and difficulty with bag mask ventilation and intubation. The upper body and head should be elevated, with the sternal notch and the ear in the same horizontal plane.[20]

The study by Neligan et al[21] showed that in morbidly obese patients in the ramped position, there was no relationship between the presence and severity of OSA, BMI, neck circumference and difficulty of intubation or laryngoscopy grade. Videolaryngoscopes are a particularly useful alternative tool for difficult intubation in the morbidly obese population.[22]

Postoperative Respiratory Dysfunction

Postoperative pneumonia and respiratory failure are relatively common non-wound complications after bariatric surgery. In over 32,000 patients followed up by the American College of Surgeons National Surgical Quality Improvement Program, 0.6% of patients developed PP and PRF. These accounted for less than 20% of all complications at 30 days.[23] Factors associated with increased risk of developing these complications included CHF and stroke for PP, and coronary interventions and dyspnoea at rest for PRF.

There are a number of studies published in the last three years showing how intraoperative ventilation strategies can decrease the atelectasis associated with PP and PRF: including intraoperative PEEP levels of at least 8cm of water, and other alveolar recruitment manoeuvres during the surgery. Talab et al showed how intraoperative recruitment with a vital capacity manoeuvre followed 10cm of intraoperative PEEP was effective at preventing lung atelectasis as measured on post CT scans, improved oxygenation, shortened PACU stay and resulted in fewer post-op pulmonary complications.[24]

Conclusion

Obesity and the related co-morbidities pose numerous perioperative challenges for anaesthetists but experience with bariatric surgery has helped us deal with many of these issues effectively.

References

1. Passannante AN et al. Anesthetic Management of Patients with Obesity and Sleep Apnea. Anesthesiology Clinics of North America 2005; 23: 479-491
2. Yurcisin BM et al. Obesity and Bariatric Surgery Clin Chest Med 2009; 30: 539-553
3. Srinivasa et al. Early and mid-term Outcomes of Single-Stage Laparoscopic Sleeve Gastrectomy. OBES SURG 2010; 20:1484-1490
4. Lemanu DP et al. Single-stage laparoscopic sleeve gastrectomy: Safety and efficacy in the super obese. Journal of Surgical Research; 2012:1-6
5. Lemanu DP et al. Optimizing Perioperative Care in Bariatric Surgery Patients. OBES SURG (2012), in press
6. Poirier et al. Bariatric Surgery and Cardiovascular Risk Factors. Circulation 2011; 123:00
7. Bamghade OA et al. Fast track laparoscopic gastric bypass surgery: outcomes and lessons from a bariatric service in the UK. Obes Surg. 2012 Mar; 22(3):398-402
8. Bergland et al. Fast-track surgery for bariatric laparoscopic gastric bypass with focus on anaesthesia and perioperative care. Experience with 500 cases. Acta Anaesthesiol Scand 2008 Nov; 52(10)1394-9
9. Candiottti K et al. Obesity, Obstructive Sleep Apnoea, and diabetes mellitus: anaesthetic implications. BJA 2009: 103, 23-30
10. Chung F, Ward B et al. A systemic review of obstructive sleep apnoea and its implications for anesthesiologists. Anesth Analg 2008; 107: 1543-63
11. Bell RL et al. Postoperative Considerations for Patients with Obesity and Sleep Apnea. Anesth Clin North America; 2005;23: 493-500
12. Weingarten TN et al. Obstructive Sleep apnoea and perioperative complications in Bariatric patients BJA (2011) 106(1) 131-139
13. Ahmad S et al. Postoperative Hypoxemia in morbidly Obese Patients with and without Obstructive Sleep Apnoea undergoing Bariatric Surgery. Anesth Analg(107): 1; 138-143
14. Chau EHL, Lam D et al. Obesity Hypoventilation Syndrome. A Review of the Epidemiology, Pathophysiology and Perioperative Considerations. Anesthesiology 2012; 117: 188-205
15. Passannante AN et al. Anesthetic Management of Patients with and without sleep apnoea. Clin Chest Med 30 (2009) 569-579
16. Ingrande J et al. Dose adjustment of anaesthetics in the morbidly obese. BJA 2010; 105: 16-23
17. Shibutani K, Inchiosa MA et al. Pharmakokinetic mass of fentanyl for postoperative analgesia in lean and obese patients. BJA 2005; 95:377-83
18. Tung A. Anaesthetic Considerations with the metabolic syndrome. BJA 2010; 105: 24-33
19. 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. Br. J. Anaesth. (2011) 106(5): 617-631
20. Collins JS, Lemmens HJ et al. Laryngoscopy and morbid obesity: a comparison of the "sniff" and "ramped" positions Obes Surg. 2004 Oct;14(9):1171-5.
21. Neigan PJ et al. Obstructive sleep apnea is not a risk factor for difficult intubation in morbidly obese patients. Anesth Analg 2009 Oct; 109(4):1182-6
22. Marrel J, Blanc C et al. Videolaryngoscopy improves intubation condition in morbidly obese patients. Eur J Anaesthesiol. 2007 Dec;24(12):1045-9.
23. Gupta et al. Predictors of pulmonary complications after bariatric surgery. Surg Obes Relat Dis. 2011 May 13
24. Talab et al. Intraoperative ventilatory strategies for prevention of pulmonary atelectasis in obese patients undergoing laparoscopic bariatric surgery. Anesth Analg. 2009 Nov; 109(5):1511-6

WHAT'S NEW IN REGIONAL ANAESTHESIA?

Dr Peter Hebbard

Clinical Associate Professor
University of Melbourne

Local Anaesthetic Injection Location

Discussion around intra-neural vs extra-neural injection has been around for some years, the arguments boosted by the realisation that nerve stimulator techniques often lead to intra-neural needle placement. Bigeleisen, the most prominent proponent of deliberate intra-neural placement of local anaesthetic (LA) recently showed using Indian ink in cadavers that injections deliberately close to but not within nerves on ultrasound (US) may still spread to the perineural tissues between nerve fascicles.[1] Sonographic spread of LA around a nerve has been correlated with rapid and successful blocks. In addition, smaller nerves are blocked faster than large nerves. These findings suggest that transport or diffusion of LA into the nerve is often a limiting step.[2,3] The appearance of LA deposited into a cleavage plane around the nerve fascicles has been recognised for some time and it is unclear whether this represents intra-neural injection. Some have termed this site "sub-perineural" however a new histologic study has identified a separate connective tissue layer around nerves termed the para-neurium, which appears separate from the peri-neurium and may represent the ideal injection point as the sub-paraneural injection has a characteristic appearance.[4,5] Many studies have now confirmed that reliable nerve block is possible with very small doses of accurately placed LA, particularly if attention is placed to surrounding the nerve with LA.

Safety of Ultrasound Guided Regional Anaesthesia

Several large series containing prospective data have been published recently, looking specifically at the rate of complications of US guided regional anaesthesia (RA).[6,7] Ultrasound guidance does not seem to have altered the incidence of neurological complications, perhaps reflecting the multi-factorial nature of these events. Interscalene block for shoulder surgery continues to be over-represented in the incidence of post-operative neurological deficits. However this may be more related to surgical factors, as the incidence of deficits has been found to be the same independent of the use of RA.[8] With ultrasound we may be trying to place the LA very close to nerves, with the possibility of vascular disruption and LA toxicity. However, the incidence of LA systemic toxicity (LAST) was impressively small in the most recent database report, with only one seizure and no cardiac arrests in over 12,000 US guided blocks.[7]

New Blocks

Ultrasound has opened the gates to targeting previously difficult-to-block nerves, and reports continue to be produced on new blocks. These include the pudendal nerve,[9] obturator[10] and suprascapular[11] nerves as well as small sensory and motor nerves. Almost all the subcutaneous sensory nerves can be targeted with US. Clinically useful blocks include the lateral femoral cutaneous nerve, anterior femoral cutaneous nerve, infra-patellar nerve, supraclavicular nerves,[12] accessory nerve,[13] saphenous nerve, superficial peroneal nerve, deep peroneal nerve and medial cutaneous nerve of the forearm.[14] New potentially useful fascial plane blocks include the pectoral nerve block[15] and hamstrings block.

Education

The resurgence in interest in RA has been accompanied by its incorporation into the core curriculum of the new ANZCA training scheme. Techniques of learning are being investigated with distinction made between trainees and senior anaesthetists, in the suitability of training techniques.[16] The learning process of acquiring RA skills is being unravelled.[17] Specific errors such as advancing the needle without imaging the tip and unintentional probe movement are most common, along with failure to accurately image the distribution or mal-distribution of LA. A

cadaver study has shown approximately 30 ultrasound needling tasks are required to achieve adequate hand / needle co-ordination. The number of procedures to achieve competence may be much higher[18] and the conventional probe is preferable for learning compared to a hockey stick probe.[19]

Where Are We Going?

Anaesthetists have always had an interest in pain management, and increasing numbers are crossing over their US skills into chronic pain and musculoskeletal applications. At one level, the ability to pre-operatively map nerve location, find foreign bodies and identify pathology is being increasingly used by our surgical colleagues. Other practitioners are taking their US skills into new musculoskeletal and pain interventions as shown by the growth of meetings such as the International Symposium of Ultrasound in Regional Anaesthesia (ISURA) into Pain and Musculoskeletal imaging (MSK).

References

1. Orebaugh SL, McFadden K, Skorupan H, Bigeleisen PE. Subepineurial injection in ultrasound-guided interscalene needle tip placement. Reg Anesth Pain Med 2010 Sep-Oct; 35(5): 450-4
2. Prasad A, Perlas A, Ramlogan R, Brull R, Chan V. Ultrasound-guided popliteal block distal to sciatic nerve bifurcation shortens onset time: a prospective randomized double-blind study. Reg Anesth Pain Med 2010 May-Jun; 35(3): 267-71
3. Germain G, Lévesque S, Dion N, Nadeau MJ, Coté D, Nicole PC, Turgeon AF. Brief reports: a comparison of an injection cephalad or caudad to the division of the sciatic nerve for ultrasound-guided popliteal block: a prospective randomized study. Anesth Analg 2012 Jan; 114(1): 233-5
4. Franco CD. Connective tissues associated with peripheral nerves. Reg Anesth Pain Med 2012 Jul; 37(4): 363-5
5. Andersen HL, Andersen SL, Tranum-Jensen J. Injection inside the paraneural sheath of the sciatic nerve: direct comparison among ultrasound imaging, macroscopic anatomy, and histologic analysis. Reg Anesth Pain Med 2012 Jul; 37(4): 410-46
6. Barrington MJ, Watts SA, Gledhill SR, Thomas RD, Said SA, Snyder GL, Tay VS, Jamrozik K. Preliminary results of the Australasian Regional Anaesthesia Collaboration: a prospective audit of more than 7000 peripheral nerve and plexus blocks for neurologic and other complications. Reg Anesth Pain Med 2009 Nov-Dec; 34(6): 534-41
7. Sites BD, Taenzer AH, Herrick MD, Gilloon C, Antonakakis J, Richins J, Beach ML. Incidence of Local Anesthetic Systemic Toxicity and Postoperative Neurologic Symptoms Associated With 12,668 Ultrasound-Guided Nerve Blocks: An Analysis From a Prospective Clinical Registry. Reg Anesth Pain Med 2012
8. Sviggum HP, Jacob AK, Mantilla CB, Schroeder DR, Sperling JW, Hebl JR. Perioperative Nerve Injury After Total Shoulder Arthroplasty: Assessment of Risk After Regional Anesthesia. Reg Anesth Pain Med. 2012
9. Rofaeel A, Peng P, Louis I, Chan V. Feasibility of real-time ultrasound for pudendal nerve block in patients with chronic perineal pain. Reg Anesth Pain Med 2008 Mar-Apr; 33(2): 139-45
10. Soong J, Schafhalter-Zoppoth I, Gray AT. Sonographic imaging of the obturator nerve for regional block. Reg Anesth Pain Med 2007 Mar-Apr; 32(2): 146-51
11. Siegenthaler A, Moriggl B, Mlekusch S, Schliessbach J, Haug M, Curatolo M, Eichenberger U. Ultrasound-guided suprascapular nerve block, description of a novel supraclavicular approach. Reg Anesth Pain Med 2012 May-Jun; 37(3): 325-8
12. Maybin J, Townsley P, Bedforth N, Allan A. Ultrasound guided supraclavicular nerve blockade: first technical description and the relevance for shoulder surgery under regional anaesthesia. Anaesthesia. 2011 Nov; 66(11): 1053-5
13. Townsley P, Ravenscroft A, Bedforth N. Ultrasound-guided spinal accessory nerve blockade in the diagnosis and management of trapezius muscle-related myofascial pain. Anaesthesia 2011 May; 66(5): 386-9
14. Thallaj A, Marhofer P, Kettner SC, Al-Majed M, Al-Ahaideb A, Moriggl B. High-resolution ultrasound accurately identifies the medial antebrachial cutaneous nerve at the midarm level: a clinical anatomic study. Reg Anesth Pain Med 2011 Sep-Oct; 36(5): 499-501
15. Blanco R. The 'pecs block': a novel technique for providing analgesia after breast surgery. Anaesthesia 2011 Sep; 66(9): 847-8

16. Sites BD, Chan VW, Neal JM, Weller R, Grau T, Koscielniak-Nielsen ZJ, Ivani G; American Society of Regional Anesthesia and Pain Medicine; European Society Of Regional Anaesthesia and Pain Therapy Joint Committee. The American Society of Regional Anesthesia and Pain Medicine and the European Society Of Regional Anaesthesia and Pain Therapy Joint Committee recommendations for education and training in ultrasound-guided regional anesthesia. Reg Anesth Pain Med 2009 Jan-Feb; 34(1): 40-6
17. Sites BD, Spence BC, Gallagher JD, Wiley CW, Bertrand ML, Blike GT. Characterizing novice behavior associated with learning ultrasound-guided peripheral regional anesthesia. Reg Anesth Pain Med 2007 Mar-Apr; 32(2): 107-15
18. Barrington MJ, Wong DM, Slater B, Ivanusic JJ, Ovens M. Ultrasound-guided regional anesthesia: how much practice do novices require before achieving competency in ultrasound needle visualization using a cadaver model. Reg Anesth Pain Med 2012 May-Jun; 37(3): 334-9
19. Davies T, Townsley P, Jlala H, Dowling M, Bedforth N, Hardman JG, McCahon RA. Novice performance of ultrasound-guided needle advancement: standard 38-mm transducer vs 25-mm hockey stick transducer. Anaesthesia. 2012 Aug; 67(8): 855-861

THE NEW GENERATION ANAESTHETIC MACHINE

Dr Chris Thompson

Royal Prince Alfred Hospital
Sydney

Disclaimer – the author is a design and development consultant for Dräger. He has no other financial interest (shares etc) in Dräger or any other medical equipment company.

Background

All of a sudden we have these new machines, they're all different, we don't know what's inside them or how they work, they have lots more alarms, we feel uncomfortable using them, and we had no role in selecting them.

It was all a lot easier when everyone had a Boyles Machine with an Ulco ventilator, wasn't it?

Just 15 or 20 years ago, trainees were taught all about the machines of the day. We learned all about the gas path, from the VIE's, down the pipelines, to the machine and thence to the patient. We knew that there were indexed gas fittings for the hoses and that the cylinder connections were made safe by pin indexing and colour coding. We were taught about how the regulators and rotameters worked in great detail. The internal plumbing of a Boyle's machine was easy to understand. Whether or not gas was flowing into the circuit was immediately obvious, and it was easy to tell if a cylinder was empty. The breathing system was easily understood and all the connections were visible and easily checked. Switching from a bag to a ventilator was intuitively obvious; you just disconnected the bag, closed the APL valve, plugged the hose on to the ventilator, and turned it on. There was a Howison type oxygen supply failure alarm, a pressure based ventilator alarm, and an oxygen analyser. That was about it.

We understood what might go wrong – and how to fix it.

We felt comfortable with these machines because they were simple, their inner workings were understood with a high level of detail and clarity, and because they were all basically the same.

'Anti-hypoxia devices' were the beginning of the end. These mechanically complex and somewhat unreliable add-ons required second stage regulators and fiddly little components that were tedious to adjust to keep working properly. Most of us had a vague idea of how they worked but didn't appreciate their complexity or the additional hazards they brought to the machine. The bag / vent switch then added another level of complexity. Then international standards mandated monitoring of exhaled volume, and automatic sustained high pressure alarms even in man / spont mode. Bit by bit the complexity of the machine grew and grew, and mechanical solutions became more and more complex and expensive.

At the same time, digital electronic alternatives to mechanical devices became cheaper and more reliable. Calibrated rotameters became costlier than electronic gas mixers. Machines incorporating electronic mixers would prove to additionally provide a huge number of practical benefits over manually-adjusted rotameters, not all of which were anticipated. These included –

- The controls can't be accidentally bumped
- Output can be more precisely adjustable
- Desired values are able to be pre-set
- Current values can be 'held' and 're-activated'
- Confirmation of flow be displayed in novel ways
- Automatic self-testing of the machine for leak
- Quantification of leaks and inefficient gas supply usage
- Delivery of constant fresh gas flow despite variable line pressures (no need for second-stage regulators)
- Provision of automatic leak compensation
- Inclusion of 'smart' anti-hypoxia functionality
- Continuous self-check of the actual delivered gas flow

- Provision of alarms when problems arise
- Prediction of future circuit gas composition
- Provision of feedback control over gas flow and composition

The advent of cheaper computers and flat screens, robust and accurate sensors (many flowing on from industrial, aerospace and automotive applications), cheap programmable micro-controllers, robust two-channel computer control safety algorithms, and finally the development of graphically rich graphical user interfaces all made the shift to fully electronic machines inevitable.

At the same time, ICU ventilation capabilities increased, and machines that could better support spontaneous breathing and provide ICU ventilator performance in the operating room became more desirable. Some companies re-designed the breathing system to more closely emulate the performance of an ICU ventilator, because it is difficult to get low-resistance flow-responsive breathing systems with the conventional anaesthesia circuit. Just about all the suppliers revamped their ventilators to at least provide the same 'modes' as offered in ICU ventilators, even if they didn't quite have the same underlying performance.

Only in the last 10-15 years have all these factors converged. The result is a massive change in the technology underlying anaesthetic machines.

The new machines are increasingly becoming 'black boxes,' poorly understood at the most fundamental level. We no longer understand their internal workings. We don't know how the gas mixer works, and we sometimes feel uncertain about whether the gas really is flowing. How do we know if the numbers on the screen are actually what is happening? How do we know if the computer running the machine is working properly? When the machine makes all those alarm messages we've never seen before, what should we do?

If something goes wrong with the machine during the case, our professional responsibility is to know enough about it to promptly follow the appropriate corrective actions. We therefore have the unenviable task of learning about each of the new anaesthetic machines from the ground up.

Factors to Consider When Evaluating a New Anaesthetic Machine

When a new machine is released, it's different from when a new drug is released. New drugs are carefully studied by independent researchers, and the companies give us lots of information as well. As a result it's easy to figure out how and when to use a particular drug soon after its release. If it's not helpful, it's easy to not use it any more. But new anaesthetic machines are totally different. There is no reliable independent review or assessment service available to us. Once they're purchased, we are stuck with them for a long time. All too often, equipment purchases go to whatever machine is cheapest that meets the specification. But who writes the specification? What should be in it? Clearly it's difficult for people with limited experience of electronic machines to write a tender document that would get them the best machine. Some tenders are written by biomedical engineers or administrators. How does a tender get written that effectively values a clear, intuitive user interface, or a breathing system that better supports spontaneous breathing? Who should evaluate the machines? Should the clinicians or the administrators have the final say?

Some might argue that they all the same, like Boyle's machines. Are the differences between them clinically irrelevant?

That question can be best answered, perhaps, by identifying the differences, thinking about which differences really matter, and finally deciding how we would identify them.

I've personally been a close participant in the development of three new electronic anaesthetic machines, all by Dräger – the Primus, Zeus and Perseus – over a period of almost 20 years. I've assisted with safety concepts, algorithms, display technologies, alarms and so on. During this time I've reached a number of conclusions of particular relevance to people seeking replacements for old machines, and the purpose of this talk is to share them with you.

1. Choose a Manufacturer That is Likely to be Around in 10 Years Time

To design and manufacture a state of the art anaesthetic machine is very costly indeed. Development costs run into many hundreds of millions of dollars. Large companies benefit from economies of scale. It's sensible for them to invest large sums of money in the development of sophisticated, reliable and specialised electronic components only because they know they will sell a lot of them. Smaller manufacturers must use simpler components and often must compete for the 'low end' of the marketplace. They will face a lot of competition from Chinese manufacturers in the years to come, and it will be tough.

2. Up-front Cost Should Not Be the Main Issue

A typical anaesthetic machine would do between 10,000 and 20,000 anaesthetics in its lifetime. The acquisition cost per case is therefore small, much less than the disposables and drugs required for the case, let alone all the surgical and labour costs.

If a new machine prevented one significant adverse outcome in its lifetime it would pay for itself many times over.

The extra cost per case between the new machines on the market is very small.

Administrators always worry about purchase price, and it is important that they factor in savings related to reductions in adverse outcomes. Potential cost savings from reduced volatile agent or oxygen consumption, fewer ICU admissions, etc are typically much greater than the cost of the hardware.

Clinicians should not, in my opinion, worry greatly about cost. We are the patient's advocate. No-one else is. If a particular monitor, ultrasound machine, or anaesthetic machine seems to be markedly better from a clinical perspective than the others, the additional per-case extra cost of having that 'better' machine is trivial over the life of the machine. We should try our best to get the best equipment to care for our patients. This will only happen when clinicians are integral to purchasing decisions.

3. Basic Ergonomics

This is very important; after all, you'll be working with the machine for the next 10 years or so.

Where does the sucker go? What happens if we have to use the machine the other way around? Is there enough desk space to put the notes, the phone and your laptop? Is it easy to clean? Easy to move around? Are cable guards integrated into the wheels? Where does the breathing bag go, and all those cables? Can you reach all the controls while seated? Can the screens swivel to face you? Can you reach the ventilator controls easily on induction? Where do we fit an extra computer screen, or some additional monitoring?

All these issues are not 'new.' Some 'new' machines were so focused on design or technology that ergonomics was not their strong point. We should expect a return to more ergonomically appropriate designs as technology becomes less of a differentiator.

4. User Interfaces

Electronic machines have complex graphical user interfaces. Some are far more sophisticated than others.

The specifications of new machines generally list a range of features. Most machines have 'similar' feature lists but very different levels of technical, ventilator performance and user interface sophistication.

User interface 'superiority' cannot be readily evaluated by a standard tender process that just lists 'features.' A good user interface is intuitive, consistent, and easy to read at a distance, easy to understand, easy to operate, free from needless alarms, readily customised to suit different user expectations, and readily upgraded. It helps you get where you want to go quickly and efficiently and is a pleasure to use. It's important to define these factors in the tender documents, figure out an evaluation process, and ensure they receive a significant weighting in the decision making process.

Who should actually take part in a User Interface (UI) assessment process? Clinicians will always favour 'familiar' user interfaces over 'new' user interfaces – even if the old 'familiar' interface may prove to be very limiting down the track, it will often 'get the nod.' When evaluating machines, it can take months or even years to really get comfortable with an entirely different user interface. Sometimes the evaluation group should be a subgroup of the more visionary, lateral thinking and technologically savvy anaesthetists in a department. Remember that you are buying a machine that you'll still want to use 10 years down the track.

First up, before the trial, someone has to go through all the configuration options and set the machine up the way that is likely to work best in your department. Many electronic machines are highly customisable. Figure out in advance which alarms should be 'active' in each mode, and what the alarm set points should be. Touchscreen interfaces are often full of tricks and shortcuts. Find out what they are in advance. Make sure that each machine is set up in what you think is the simplest and most approachable way.

If after an introduction to the machine, there are any UI aspects you think might be improved, and so long as the tender doesn't prevent you doing so, seek out the manufacturer's feedback on each point. There may be a way to modify that behaviour, or in case there are shortcuts or work-arounds you don't know about, or if a software update is on the way to address that particular issue.

Having all the trial machines available in the department at the same time, to directly compare the user experience of one machine directly against the other, can be very effective. Get them all in the same room, and do the same things to each of them. Find out which responds most quickly and intuitively and is easiest to use. Rate things like –

- Start-up time
- The self-check 'experience' when there is a leak or a loose vaporiser or an empty cylinder
- What happens when the oxygen supply fails
- What happens when the electrical supply fails and when the internal backup supply fails
- If the screen organised logically
- Whether the display is legible at a distance
- Are the trends clear, legible and helpful?
- Are the waveforms of flow and pressure detailed and accurate?
- Does the machine display loops, and if so how well are they displayed?
- Is it obvious which numbers are 'monitored' values, and which are 'settings'
- Whether in one glance we can confirm that it is working 'normally'
- Can we easily tell which mode the machine is in?
- How easy it is to switch from man / spont to PS and back
- How sensible are the alarms, eg for a leak or a disconnection

Try doing the everyday tasks you'll do every day, like starting and stopping cases, switching ventilator modes, dealing with alarms, and find out which machine lets you do these things most efficiently. Think about things you might not do now, but may do in the future, such as ending every case or even inducing every patient with pressure support, using very low flows (and having to deal with leaks in low flow settings), using feedback control, looking at trends or loops, etc.

Have a robust discussion about how each machine manages a particular issue and decide which you think is best.

Make a list of all the factors that seem to differentiate between the machines, do a 'secret ballot' style formal UI assessment after a sufficient evaluation period, and ensure that the outcome of this evaluation is part of the tender process.

5. Ventilator Capabilities

Most modern machines provide 'new' ventilation modes such as pressure support (PS) and pressure control. Some also include volume-preset pressure control (auto-flow) type modes, where the machine attempts to deliver a fixed tidal volume but with a constant pressure during inspiration. Some may provide 'mandatory minute volume' or other ICU type modes.

Technically it is not particularly difficult to implement these modes on even a simple machine. There can be very significant differences in how effectively these ventilation modes are implemented from machine to machine,

particularly when it comes to supporting spontaneous respiration in such patients. So in a tender situation, a simple list of modes isn't enough to differentiate between machines that can actually perform very differently.

How then can we identify the differences in ventilator performance? It's actually very difficult. There are no particularly helpful papers in the literature.

Manufacturers with a core competency in ventilation are likely to get the ventilation part of a machine 'right,' but that is a generalisation that can't be applied to a tender process.

Pressure Support is probably the most useful new mode of ventilation. Well implemented, it markedly improves ventilation during spontaneous breathing anaesthesia. Adding PEEP should actually provide the benefits of CPAP during spontaneous breathing, and can minimise airway obstruction on induction due to negative intra-pharyngeal pressures.

Some of the things that need to be considered when evaluating a machine's ability to provide effective pressure support include –

- Ease and reliability of triggering, eg for very small children
- Peak inspiratory flow rate
- Time to achieve peak inspiratory flow rate after a trigger
- Time to drop airway pressure after end-inspiration is detected
- Inspiratory flow resistance
- Expiratory resistance
- Resistance compensation

A machine that performs a little bit better on each of these parameters can be much better overall at supporting spontaneous breathing.

When supporting spontaneous breathing, the machine must detect inspiratory effort and respond immediately by increasing circuit pressure to the desired support level. There should be no detectable 'blockage' or 'delay' in providing that increase in pressure. Delays in response can be seen as a fall in airway pressure at the start of inspiration. If a machine cannot deliver flow fast enough, the patient will feel difficulty breathing in, and not get the reduction in inspiratory effort that we are seeking.

At the end of inspiration, the machine must accurately detect the loss of inspiratory effort, and rapidly open a low-resistance exhalation pathway. Any delay in detecting the end of inspiration and any significant expiratory resistance will add considerably to work of breathing, and reduce the overall effectiveness of pressure support modes.

Generally most machines do a satisfactory job in pressure support modes for relaxed breathing in healthy patients. Not all machines, however, are able to effectively assist someone who is gasping for breath, or for infants at high respiratory rates, or patients in respiratory failure, for example.

Fortunately it is relatively easy to compare, in an A-B fashion, any two machines. Put a filter on each machine, block your nose, sit comfortably in a chair, and configure each machine with identical settings. First breathe normally in Man / Spont. Become familiar with the inspiratory and expiratory resistance of each machine. Note that expiratory resistance increases when the bag gets full, and is very uncomfortable if the APL valve is partly shut. Then swap to Pressure Support mode with, for example, 5 cmH_2O of pressure support and 2 cmH_2O of CPAP / PEEP. There should be an obvious improvement in the level of perceived resistance to both inspiration and expiration, although CPAP feels strange at first. Compare the two machines. The same settings on one may be much more effective, in practice, and not the other. Try rapid shallow gasping inspiratory efforts, and get a feel for how quickly the machine responds. A good machine will effortlessly and 'invisibly' support your breathing and make it easier.

Although it's not so easy to test, not all machines trigger reliably in small children. You can try making very shallow respiratory efforts. In clinical practice, some machines will easily manage 10 kg children on adult circuits, others won't.

Also find out what happens in Pressure Support mode when the patient becomes apnoeic for a time and then starts breathing again. This happens often in practice. Does the machine stay in the same mode, or switch to another mode? Do you have to do something, like respond to an alarm? Does the machine alert you in a sensible,

helpful way? If they start breathing again, does the machine get back to pressure support or does it remain stuck in some other mode?

Finally, is it easy to select a given mode, and switch to another? How are the default settings altered and stored. Is there a 'pause' mode? Are settings shared across the modes sensibly? A machine that requires several key presses to get from one mode to another can be frustrating to use. All these small details make a big difference to the usefulness of a 'feature' on a machine.

Our ability to effectively manage people with respiratory failure is made much easier if we have a machine with ICU quality ventilation abilities. A machine with excellent spontaneous breathing support makes supported ventilation techniques much easier than ever before. It sometimes takes quite a while before you realise how good a job you can do. Before I learned how to use effective pressure support and CPAP in spontaneously breathing obese patients, I would not uncommonly get a phone call from Recovery saying, "Your patient's saturations are only 89-90%, what do you want us to do." These days it just about never happens.

If you do A-B testing, it is usually fairly easy to rank the machines in terms of how effectively they support spontaneous breathing, and how easy it is to use the new modes in practice.

6. Feedback Control of Vapour Delivery

Feedback control of inspired oxygen and volatile agent concentration in the circuit, particularly at low or minimal flow during the maintenance phase of an anaesthetic, has a number of attractions. The main benefit is to reduce the annual cost of volatile agents. This benefits the hospital. The benefits to us and the patient include more stable agent and oxygen levels, less 'drift' over time that requires correction, and smarter anti-hypoxia systems that can target end-tidal oxygen, for example. However they add a number of hazards to contend with to gain those benefits, and the technical challenges in dealing with them are considerable.

The major manufacturers all have machines with feedback control capabilities in an attempt to make low flow or closed circuit anaesthesia safer and easier. First let's think about feedback control of volatile agent.

Administrators may be inclined to assist with the purchase of an expensive new machine on the basis that it will save a lot of money on volatile agents. If feedback control is effective and easy to use, such that a significant proportion of the department's staff will use it, then you should anticipate saving money on volatile agents.

Savings won't be effectively realised in rapid-turn-over type cases, because highish flows (and / or concentrations) are still required at the beginning of the case to 'load' the patient. Conversely, greater savings will accrue when longer cases are the norm.

Using sevoflurane throughout the case at minimal flow requires a low-hydroxide type CO_2 absorbent that makes little or no compound A. Fortunately these are now easy to obtain. I'm not sure it makes a lot of sense to switch back to isoflurane for feedback control, because it doesn't have such an ideal offset characteristic in long cases. Feedback controlling desflurane, particularly to end-tidal values, puts special demands on the controller algorithm to avoid abrupt and significant increases in inspired desflurane levels.

Feedback control of volatile agent adds greatly to the software complexity of the machine, and probably requires additional hardware (backup or 'second channel' agent and oxygen analysers) as well.

I personally think that it is essential to have two independent gas analysers in the machine, one to do the feedback control, and an independent analyser to measure the circuit concentration from a monitoring or checking perspective. If the same gas analyser is used for both monitoring and control, and that single transducer drifts or becomes faulty, then the anaesthetist will get no warning that the agent concentrations are in fact not being delivered correctly. The analyser doing the feedback control may operate from the sample line at the Y-piece, but if, for example, there is air entrainment via a leak in the sample line, then the control mechanism would start to overdose the patient, and the data presented to the operator would give them no clue as to this error. The only way that the operator would know about this problem would be if there was a second gas analyser internal to the circuit that confirmed that the mean agent concentration in the circuit was not consistent with the mean concentration at the Y-piece. If the two didn't match sufficiently, the machine should stop trying to do feedback control. Without an independent 'second channel' to monitor the controller, the risk of a dangerous failure is

unacceptably high. The additional agent analyser in the circuit adds to the cost of the system, and can itself drift or fail.

Alveolar deadspace causes end-tidal gas to look more like inspired gas, and this may cause an end-tidal feedback design to under-administer anaesthetic agent while increasing volatile concentration, though not to a huge extent in most cases. In steady state there would be little or no effect. Likewise, 'smart' anti-hypoxia devices that target end-tidal oxygen may over-estimate functional alveolar oxygen. If the anaesthetist knows that high levels of alveolar deadspace are likely, they can evaluate end-tidal anaesthetic agent or oxygen levels more critically than automated controllers.

If a blower is present in the circuit to provide continuous basal mixing of the vapour in the circuit, it is possible, though difficult, to get prompt and stable control of anaesthetic concentrations in the circuit, at near-zero flows, even when the patient's tidal volumes are small or if the patient becomes apnoeic. Without a blower, and if the agent concentration is controlled at the Y-piece, there can be delays from agent delivery into the circle and when those gases reach the analyser, and these can cause instability.

Conventional variable-bypass vaporisers – including the Aladdin cassette system – have a limit on the maximum output vapour concentration, and therefore cannot deliver vapour under true closed circuit conditions. To deliver vapour at zero flow, a direct vapour injection type vaporiser such as on the Zeus is required, and this is a complex and specialised vaporiser.

The vaporiser used in a feedback control system must be electronic, and therefore vapour delivery is impossible without electrical power. This is kind of obvious, and is an acceptable hazard unless there is a reasonable probability that the power may fail for a long time, such as an unintended extended power failure or in a disaster type situation. In these situations, mechanical vaporisers can be used on some machines with manual ventilation for extended periods of time without any electrical power. In reality, however, this is not a practical issue; not much surgery can be performed without electricity, so without electricity we might as well just close up and wake the patient up anyway.

Additionally, the gas blender must be electronic, because the machine may have to increase flow to wash agent in or out to keep the agent concentration where you want it, and then return it to a lower number under some kind of algorithm. Again, this is self-evident and not a particular drawback in a practical sense, particularly if the machine provides and emergency gas flow through a vaporiser that can be used with hand ventilation.

The user interface must clearly indicate whether or not you are in manual control mode or feedback control mode, and whether it is controlling inspired or expired values. Confusion over the mode of operation of the machine can be a safety issue. When using desflurane, the maximum inspired concentration the machine may generate in the inspiratory limb of the circuit when a step increase in the desired exhaled desflurane concentration is requested. You also need to know what happens to gas flows when a reduction or increase in agent concentration is requested, and how that may affect inspired oxygen or nitrous.

As for volatile agents, feedback control of inspired oxygen requires a second sensor independent of the gas sample line sensor used to monitor inspired / expired oxygen, and this sensor must be internal to the breathing system and independent of the gas sample line. Without that second sensor, the machine is intrinsically very unsafe, in my opinion.

Having the gas continuously circulated in the breathing system by a blower greatly simplifies feedback control of oxygen. It is particularly important in spontaneous breathing modes, should apnoea occur.

The need to tightly control oxygen around 30% more common in the past when nitrous oxide was intended to provide the majority of the anaesthetic. This technique is becoming archaic, and the most common carrier gas in our institution is now air. Controllers capable of tightly controlling inspired oxygen with nitrous as a carrier can be a challenge to design.

The optimal inspired oxygen value for anaesthetic maintenance in air is debatable. Eighty percent has been suggested to reduce infection rates, and 30-40% in air to minimise collapse. Hypoxia due to V/Q inequalities should be almost completely eliminated with inspired oxygen levels of 40% or more. Tight control of inspired oxygen during anaesthesia is probably only required for premature neonates and people particularly susceptible to hyperoxic damage, eg after chemotherapy.

To make a rapid change in oxygen concentration while at low flows, the machine must increase gas flow. If agent control is active at the same time under very low flow conditions, and if variable bypass vaporisers are used, the internal set point for the vaporiser will have to be turned down a great deal from the previously stable setting and the new value won't be obvious to the machine. The user needs to be aware of how the machine reacts to step changes in oxygen set point, and what effect that will have on vapour delivery.

It's interesting that the Dräger Perseus, the latest machine from Dräger, does not have feedback control of agent or oxygen, despite Dräger's extensive experience with feedback control in the Zeus and the fact that the machine has a blower and electronic gas delivery that 'should' make it easy. Not implementing feedback control in the Perseus has simplified the design of the machine, both from a software and hardware perspective, and with it the user experience. The vaporiser may be used with man / spont mode for prolonged periods without any electricity, and this is important in some markets. The Perseus can predict, based on measurement of uptake and pharmacokinetic modelling, what the likely future equilibration values will be for agent and oxygen, in near real-time as you adjust the settings. This can be a very useful guide to setting the vaporiser and the inspired oxygen at low flows. The intent is to facilitate the use of very low flows by minimising the number of adjustments required to get where you want to be, without the hazards and cost of feedback control.

It will be interesting to see whether the advantages of feedback control of agent using variable bypass vaporisers at very low flows proves to be a success in the marketplace. A lot will depend on reliability, field experience and user interface issues. Most likely a good controller would be very useful especially in longer cases, and would probably save money. There is limited experience with 'future prediction' technologies as in the Perseus, so we just don't know at this point whether it will be almost as useful as feedback control or not.

7. Integration with IV Pumps

Propofol, narcotic and relaxant infusions are commonplace in modern anaesthetics. The smooth integration of pumps into the anaesthetic workstation would be a big step forward. It hasn't happened mostly because of the simple fact that no anaesthetic machine manufacturer make their own pumps.

Often the user finds that there is nowhere to effectively put the pumps on the machine and not enough power points on the machine to provide power to them.

It makes sense to be able to control, record, and monitor a propofol infusion in a manner not dissimilar to a volatile agent. Some new machines do properly integrate data from pumps into their data architecture, and some permit control of the pump from within the user interface of the machine. I remain frustrated that better IV pump integration has not become a mainstream focus of the anaesthetic machine manufacturers.

The Dräger 'Smart Pilot' is one manufacturer's attempt to display drug interactions, both volatile and intravenous, in a clinically helpful manner. The system collects data from syringe pumps, the anaesthetic machine and the monitor automatically.

In the future we may be able to choose any point in a two-dimensional touch-screen space to set the desired 'anaesthetic / hynotic' and 'narcotic / analgesic' levels required for our patient, and have the machine dutifully do whatever is needed to get us to that point, using feedback control of vapour and TCI algorithms for the IV agents. At the same time the machine could calculate the predicted wake-up time, and fine-tune the pharmacodynamic estimate with data from the BIS or Entropy monitor.

But for now the level of IV pump integration is something that can be done better by most manufacturers.

8. Neonates and Paediatrics

The use of a T-Piece circuit with uncuffed tubes for premature babies and neonates remains standard practice.

Not all the new machines support ventilation through a T-piece, and if this is important to you, then make sure it is possible. All support an external 'common gas outlet' to which a lightweight T-piece may be connected for hand ventilation on induction, and all are capable of very precise pressure and volume control ventilation with quite light-weight circle circuits.

Increasingly, infants are being induced and managed on circle systems with lightweight hoses and being intubated with cuffed tubes. Volume control ventilation as for adults is now quite easy. Pressure support can be very effective with children. It's surprising to see a 10 kg child breathing effortlessly on an adult circle system while triggering pressure support without any difficulty!

Rise time when increasing volatile agent differs slightly amongst the new machines, depending on their circuit characteristics. GE machines have 'conventional' circle systems that behave like Boyles machines. In Man / Spont modes, most machines are much the same. The Dräger Primus has a piston ventilator in the inspiratory limb of the circuit. In Pressure Support and other control modes, the fresh gas has a greater time constant because it has to mix in the piston before getting to the patient. It makes very little practical difference in adults, but with small tidal volumes in children there can be a noticeable lag between dialling up the vaporiser setting and seeing it at the Y-piece. Dräger's newer Perseus machine has a low-deadspace blower instead of the piston which additionally provides a continuous basal flow around the circle, so it is now faster than any of the Boyle's type machines.

9. Oxygen Consumption

Machines with electrically-driven ventilators require much less oxygen than those with oxygen-driven ventilators. For example, when ventilating an adult to normocarbia at 500 ml/min FGF and a 50% inspired target, an electrically driven ventilator would only require about 0.4 L/min of oxygen, whereas a demand-valve oxygen-driven ventilator would typically consume 6-7 L/min and a venturi type oxygen-driven ventilator may use 15 or more. Oxygen is cheap, but if the mains oxygen supply fails, your cylinders will last a lot longer with an electrically-driven ventilator.

10. Computers and Micro Controllers

Increasingly, ventilators, monitors and anaesthetic machines are being programmed on computers using specialised versions of desktop operating systems. We all know that domestic computer operating systems can crash unexpectedly. So how do we make an anaesthetic machine 'safe' if we use a similar operating system?

The answer is not to use more reliable software. No software is perfect.

The usual design requirement is to assume that a thread or even the whole computer will crash and figure out how to manage that effectively.

A reliable design uses two separate computers and a bunch of micro-controllers. A micro-controller is the dedicated 'computer on a chip' that for instance operates the modern electronic watch, clock radio, or even your car. It has a set of instructions that it must loop through; if it 'freezes' and doesn't get to the end of the instruction within a certain time, it automatically resets itself and starts again from the top. These things are super-reliable. They can have any number of analogue inputs and outputs.

In a modern machine, there will be a dedicated micro-controller driving the ventilator, one on the gas bench, one on the power supply management board and another one decoding user interactions like when you click a button or press a knob. These micro-controllers are 'free-running' or autonomous to some extent. Usually they 'talk' over an internal network, getting instructions from the main computer, and reporting back to it all the time.

The use of micro controllers makes things much simpler for the main controlling computer. For example, the computer simply tells the ventilator micro controller to deliver a certain tidal volume in a certain mode, so that it does. The ventilator echoes the instructions back to the computer, to be sure it heard the message right, and also reports waveforms, measured values, and so on back to the computer for display on the screen. The ventilator micro controller can be highly optimised in ways that the computer cannot.

The key thing is that there is a verified two-way communication between the ventilator or gas bench controller and the main computer. The main computer focuses on how to draw the screen, how to manage trend data, integrating data, managing alarms, and the micro-controllers do the tedious repetitive stuff with a limited code base that is highly optimised and very unlikely to crash.

To guard against the main computer 'freezing' without any alarms being generated, a second CPU running a specialised monitoring application that 'supervises' the main computer is required. This provides the second channel of safety required by the standard. The 'supervisor' computer gets the same inputs as the main computer because it is connected to the same network, so it receives all the same user inputs and all the feedback from the micro controllers. Additionally it monitors the screen display driver memory and uses algorithms to ensure that the content of that display is what it should be by reverse-engineering the characters on the screen. It does all the calculations performed by the primary CPU and should reach the same conclusions. If the two reach different conclusions, the monitoring computer decides what to do. It may require the main computer to reset the ventilator or enter a 'failsafe' mode, but if the main computer is not responsive it may reset the main computer and take over the user interface until the main computer restarts.

Designing this level of safety into the computers within a machine is based on the Standards requirement that a single fault shall not result in an unacceptable risk. The designers of these machines must be certain that any failure is brought to the attention of the operator. They are required to test every possible fault and verify that the outcome is acceptable.

While it may seem like these machines are just a bit of software running on a computer, the reality is far more complex than this. To design, implement and verify all the software and safety requirements of the modern anaesthetic machine is a hugely difficult task. There may be new machines on the market from time to time, however I would be cautious about buying an anaesthetic machine from a new player until they had demonstrated the level of reliability required by our environment. It is reasonable to request in a tender that all machines provide a 'second channel' in computing terms to ensure that a freeze or crash of the main CPU will be immediately identified and appropriately reported to the operator.

11. Service and Reliability

Service contract pricing is a significant part of the overall cost of any machine with sensors and moving parts. Some companies have extensive experience of component failure rates and these are factored into their preventative maintenance schedules. I strongly recommend paying for preventative maintenance, just as it is important to maintain your car according to the recommended schedule.

Bear in mind that all software driven devices come to market with some bugs they missed, and some features or nice things that they just didn't have time to put into the release version. If you do purchase a Version 1 software machine, be prepared to discover some things that could be done better than you expected. Try to gauge the manufacturer's likelihood of responding to your feedback. Find out how often the manufacturer has provided software updates for their other products and whether they actively address problems with updates.

When evaluating new machines, find out the level of residual functionality should a sub-system fail. For instance –

- If the electronic mixer fails, is there an emergency oxygen system that delivers oxygen through the vaporiser into the circuit? Can the ventilator be used in this situation?
- If the ventilator fails, is hand ventilation still possible?
- If a valve in the ventilator fails, can the entire valve block be changed easily? In some machines, all the 'moving parts' are on one sterilisable block, and this can be replaced in a few minutes

Fault tolerance and field replacement of modules or parts by local biomedical engineers are important considerations in remote areas. Find out whether the internal construction is modular, and how easy or difficult it is to replace a module. If there is a part that can be readily replaced, it should be kept on site, particularly if it has moving parts.

Unexpected downtime is a significant issue for electronic machines, both administratively and clinically. Many machines just don't have a 'limp-home' mode, and cannot be used at all if some vital parts (eg the main CPU or the power supply) fail. Generally there is a need to have a back-up machine of some kind in case of an abrupt failure of a machine.

Preventative maintenance, especially in the early and late phases of a machines life-span, is important, and I would strongly recommend using the manufacturer's preventative maintenance contract. Make sure the contract has time-frames for support. Think about how the contract should address a failure to fix a failed machine after a specified time.

12. Integrated Data Collection and Anaesthetic Charting

Preparing an anaesthetic chart predominantly from automatically acquired data makes a lot of sense. The greatest challenge of any charting solution is to make it easy for the anaesthetist to quickly enter drugs and annotations that can be displayed in as good a manner as a paper record. Many print-outs lack the clarity of a carefully maintained hand-written record. The ideal solution would provide the benefits of automated collection with a high-quality, scalable, easily annotated visual display.

Most likely, high-resolution iPad type touchscreen tablets will make this possible within the next 10 years. Current software applications for 'paperless' anaesthetic records still seem a bit cumbersome to me at this point in time, but they are definitely becoming far more sophisticated.

Near-field wireless identification of patients, staff and equipment is likely to radically change how the anaesthetic record might be generated. It is feasible for a machine of the future to automatically identify the patient that has just been wheeled into the room, and prepare a report including their pre-op evaluation and all relevant lab tests, and then start adding any monitored data to their anaesthetic chart without any human interaction. When the anaesthetist enters the room, the machine may offer to automatically configure itself to their favourite settings.

13. Hospital-wide Integration

Automated logging of anaesthetic agent usage, start and finish times, parameters used and monitored data is not uncommon. Some suppliers provide tools to log data over a network and provide a range of reports.

Provision of integrated access to hospital information systems for pre-operative assessment, lab data, x-ray images, theatre management applications and the entire internet is increasingly helpful. Some systems permit this data to obscure monitored data, but I think most of us would agree that it should be presented on a separate monitor screen.

Remote viewing of anaesthetic machines and monitors, and remote diagnosis of error messages and problems, is now possible. These offer clear benefits when something seems wrong with the machine. If the operator has basic video-conferencing functionality in their smartphone, or included in the machine, it is possible to share real-time audio and video of what is happening around the patient to an on-line support person. These technologies can profoundly affect the level of support available to anaesthetists in relatively remote areas, especially if they are not familiar with a new machine.

Sometimes the networking infrastructure for these features can be provided by an 'open' hospital network, but some manufacturers require the use of dedicated networks and servers, which can be very expensive to implement. Increasingly we should anticipate wireless infrastructure solutions.

Machines of the future may record a lot more than is currently recorded. They may record every operator interaction with the machine. If they also record the occurrence of an alarm, it becomes possible to determine the attentiveness of the operator and the suitability of their responses. It is quite feasible for medical devices to incorporate mobile phone type cameras in their front panels, and to record whatever happens in front of the machine. These recordings could be effectively continuous, or just for periods of time when alarms are present. Such a black box recorder would be associated with significant privacy and medico-legal issues, but at the same time would be indispensable for post-hoc 'root cause' analysis after a problem case.

14. Training

It would be medico-legally difficult to defend an adverse outcome arising from the use of a new drug on the grounds of unfamiliarity with that drug. Before using a new drug on a patient, our professional responsibility is to know not just what the drug does most of the time, but to know about the adverse reactions it may cause and how to deal with them. The same responsibility probably extends to a new anaesthetic machine. In some European countries, the release of a new machine is associated with a formal training and accreditation process before a staff member may operate the machine. In Australia and New Zealand, it can be difficult to get any such training before arriving at a theatre to find that you have to use a machine you've never used before. If something went wrong that day and unfamiliarity with the machine contributed, what then?

This is a challenging problem. Generally the manufacturers will support training and education of anyone who asks, however the educators are typically not anaesthetists, and the focus is mostly on 'normal' usage rather than what may go wrong. There are very few clinicians who know enough to 'train the trainers' effectively.

We are very fortunate indeed that mostly the new machines are as very, very reliable, and relatively easy to use. It's amazing to think that we expect them to work 12 hours a day, five days a week, for 10 years or more – and generally they do! No car, toaster, washing machine or ordinary computer would.

Summary

Increasingly sophisticated anaesthetic delivery and monitoring systems are inevitable. They are great when everything works well. Understanding enough about those systems to know what to do when things go wrong is a significant challenge. Anaesthetists should ensure that they are a familiar with a new anaesthetic machine before they first use them. This poses a number of important questions about how that familiarity may be achieved.

GOAL DIRECTED THERAPY – EXPERIENCE AT A TERTIARY CARE CENTRE

Dr Laurence Weinberg

Department of Anaesthesia,
Austin Hospital, Melbourne

The purpose of haemodynamic monitoring is to assess circulatory performance to determine if cardiac output is consistent with maintaining tissue oxygen demand, and if not, to determine what components of the haemodynamic profile need to be adjusted to re-establish the consumption-demand balance and achieve the optimal cardiac output and mixed venous O_2 reserve.[1] Importantly, haemodynamic monitoring must be considered within the context of a proven medical therapy, success of which is dependent on the clinical condition, pathophysiological state, and ability to reverse the identified disease process. No device, no matter how accurate or insightful its data will improve patient outcomes unless its couple to a treatment which itself will improve outcome.[2,3]

Goal directed therapy, often defined as "setting a haemodynamic goal and fitting the patient to the goal," has been the subject of numerous reviews and commentaries. I prefer the concept "haemodymanic optimisation" whilst arguably similar in principle is subtly different and can be defined as "looking at the patient and fitting the goal to the patient who is having a specific operation."

The presentation will describe haemodynamic optimisation strategies that are being utilised at Austin Hospital, Melbourne, Australia, with a focus on our major hepatobiliary surgery. Currently our hospital provides anaesthesia services for ~50 liver transplants per year. Being one of the largest hepatobiliary units in Australia, each year we also provide services for ~50 major liver resections and over 50 major pancreatic resections including vascular reconstruction surgery.

In this context, haemodynamic optimisation strategies are discussed to assist colleagues or other institutions who may be developing haemodynamic optimisation strategies for similar surgical procedures for the first time, or who are interested in different perspectives on haemodynamic monitoring. This is not intended to be a dogmatic approach to the topic, but rather to suggest haemodynamic optimisation models to evaluate the effectiveness of care, improve clinician practices, and reduce the risk of major adverse events. In the absence of definitive guidelines for these operations, an approach to haemodynamic optimisation in our hospital is presented.

Key concepts discussed in the presentation include –

1. There is no such thing as normal cardiac output. Cardiac output is either adequate to meet the metabolic demands or inadequate to meet the metabolic demands
2. Hypotension is always pathological and should not be accepted or tolerated during any anaesthetic. Intraoperative hypotension is a strong and highly significant predictor for mortality[4]
3. Normotension however does *not* necessarily mean that the patient is "healthy"
4. Resuscitation of mean arterial pressure does not always restore microcirculation, ie pressure is *not* flow. Although the presence of arterial hypotension is clearly an ominous sign indicating a high severity of illness, arterial blood pressure alone is an insensitive indicator of tissue hypoperfusion[5,6]
5. CVP should not be used to make clinical decisions regarding fluid management. Central venous pressure does *not* predict fluid responsiveness[7,8]

There is consensus that advanced haemodynamic monitoring is better than no monitoring for high risk patients undergoing prolonged and complex surgery. Whilst haemodynamic goals are important, the best haemodynamic goal and how to achieve these goals is still unknown. Approaching consensus that haemodynamic protocols (reproducible care practices) are better than no protocols is important in improving patient outcomes.

There is also general consensus that all the commercially available haemodynamic devices (Swan-Ganz, FloTrac, LidCo, Pulsion, Deltex, etc) are clinically acceptable in most conditions. There are still some questions regarding their use in in highly unstable patients, after vasoactive drugs and about comparing trends. It must be recognised

that all these devices have independent values for stroke volume variation and trending stroke volume and cardiac output, and outcome studies are still necessary to show the true value.

We therefore are currently exploring different haemodynamic algorithms for patients undergoing major hepatobiliary surgery at our institution. It is important to appreciate that haemodynamic monitoring must be considered within the context of each operation, success of which is dependent on understanding the operation itself, providing the surgeon with optimal operating conditions, and tailoring the haemodynamic goals to the patient during each stage of the surgery. For example, the haemodynamic goals for a patients undergoing major hepatic resection are very different to the haemodynamic goals for patients undergoing pancreaticodudoenctomy (Whipple's Procedure). During each stage of the surgery, there are specific and tailored haemodynamic goals aimed to enhance patient safety whilst providing the surgeon with favourable operating conditions. The surgical and anaesthesia considerations for major liver resection are summarized in Figure 1.

These concepts will be detailed in the presentation.

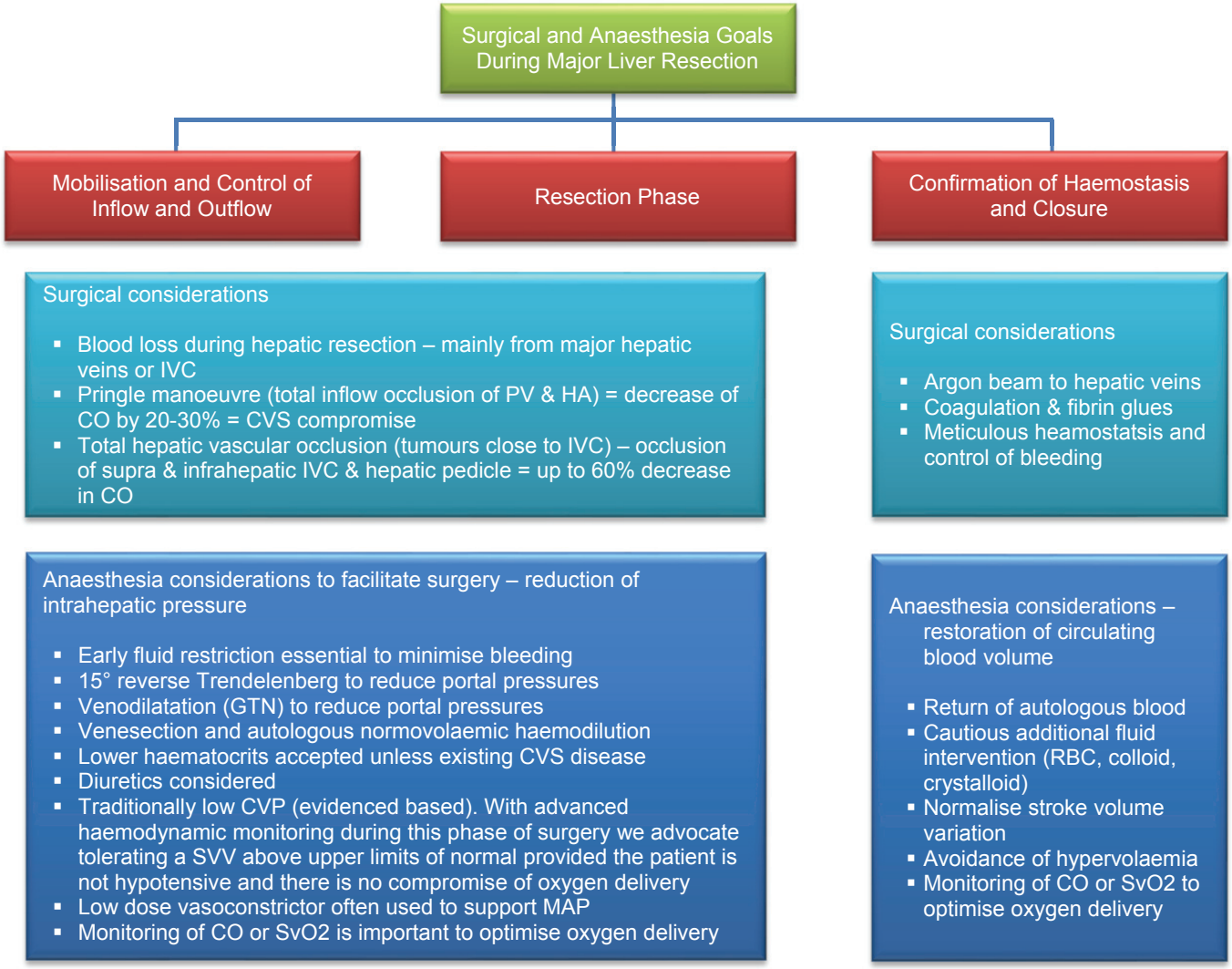
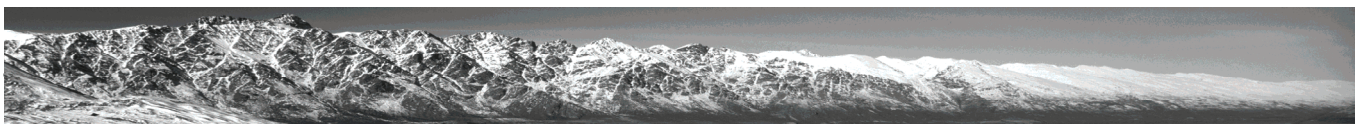


Figure 1. Important surgical and anaesthesia considerations during major liver resection

References

1. Bellomo and Pinsky. Invasive monitoring. In Timker J, Browne D, Sibbald W eds. Critical Care-Standards, Audit and Ethics. Arnold Publishing Co. London p82-104. 1996
2. Pinsky MR, Payen D. Functional hemodynamic monitoring. Crit Care 2005; 9: 566-72
3. Pinsky MR. Hemodynamic evaluation and monitoring in the ICU. Chest 2007;132: 2020-9



4. Sessler DI, Sigl JC, Kelley SD, Chamoun NG, Manberg PJ, 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
5. Bateman RM, Walley KR. Microvascular resuscitation as a therapeutic goal in severe sepsis. Crit Care 2005; 9 Suppl 4: S27-32
6. Bateman RM, Sharpe MD, Ellis CG. Bench-to-bedside review: microvascular dysfunction in sepsis - hemodynamics, oxygen transport, and nitric oxide. Crit Care 2003; 7: 359-73
7. Marik PE, Baram M, Vahid B. Does central venous pressure predict fluid responsiveness? A systematic review of the literature and the tale of seven mares. Chest 2008; 134: 172-8
8. Osman D, Ridel C, Ray P, Monnet X, Anguel N, Richard C, Teboul JL. Cardiac filling pressures are not appropriate to predict hemodynamic response to volume challenge. Crit Care Med 2007; 35: 64-8

WHAT'S NEW IN VENTILATION?

Dr Chris Thompson

Royal Prince Alfred Hospital
Sydney

Disclaimer – the author is an occasional design consultant to several equipment manufacturers, including Dräger, Datex-Ohmeda and Spacelabs.

Preamble

Anaesthesia equipment has always fascinated me. As a Registrar in the early 1980's I was lucky enough to work with John Lawrence as he developed the Prince of Wales CPAP / SIMV ICU ventilator – Bird Mk 7a pneumatics driving a bag-in-a-bottle with a CPAP system with two 5 litre reservoir bags! This stimulated what became a life-long interest in ventilation.

By the early 1990's, international standardisation, medico-legal concerns and the global marketplace ended the use of 'home-made' anaesthetic equipment, and my equipment development days were over. Or so I thought!

To my surprise, Ohmeda USA asked my advice with the design and software of a very advanced electronic anaesthetic machine, way ahead of anything else at the time. I spent a week in Madison evaluating how to best implement a full feedback control machine, from user interface considerations through to control algorithms. This was fascinating work. The end result, many years later, was the 7900 ventilator, still the basis for many current GE products.

Since then I've helped develop Dräger's advanced anaesthesia products, including later revisions of the Julian software, the Primus, Zeus and their future products.

With every manufacturer I've emphasised the need to keep the user interface simple, intuitive, quick and easy to use and – most importantly – reliable. I've encouraged uncluttered graphically-rich screens that utilise familiar icons and display algorithms, such as 'virtual rotameters' and the like, rather than a mass of numbers.

At the same time I've used the latest machines on a day to day basis, and as a result have had first-hand experience of new and innovative technologies such as electronic gas mixing, Pressure Support ventilation, feedback control of gases and volatile agents, breathing sound simulation, etc.

The focus of this talk is how to get the most out of the new ventilators on modern anaesthetic machines, how to compare one machine to another in performance terms, and what kind of user interface elements are most valuable and why.

Development of Anaesthetic Ventilators

Over the last 30 years, anaesthetic machines and ventilators have barely changed, despite significant advances in monitoring and IV therapy. In contrast, ICU ventilation has been through a kind of electronic renaissance over the same period.

Until recently, very few of these ICU features have been made available to the anaesthetist – mostly because anaesthetists told the manufacturers –

- We don't need any of these 'advances' to give a good GA
- Most of my patients are healthy anyway
- If they can't breathe on the bag, they need intubation
- Rotameters and bellows are essential
- Electronics and computers are unreliable and crash often

- It's safer for me to stick with something I know and understand

As a counter-argument to the above, those who have used ICU ventilation modes on anaesthetic machines might reply –

- Advanced ventilation modes really do make life easier, even with healthy patients
- LMAs and Pressure Support go very well together
- Quite a lot of our patients have worse lung function than we think – and we can now do something about it
- We don't actually need rotameters or bellows anymore
- Electronic machines are very reliable indeed – and far more precise and capable than anything we've used before
- Re-discovering the art of ventilation is possible only if we have the right tools for the job

Advanced Ventilation Capabilities

The following ventilation functionalities should – I think – be present on an advanced anaesthesia machine –

1. High-fidelity, clinically helpful displays
   - On-screen flow and pressure waveforms (+/- loops)
   - Intuitive indication of spontaneous vs controlled breaths
   - Clear indication of current 'mode' of ventilation
   - Clear and obvious indication of leak
   - Sensible and effective alarms
2. An effective Pressure Support / CPAP mode, requiring –
   - Reliable, sensitive detection of inspiration (triggering)
   - High flow capability once triggered (>120 L/min)
   - Rapid, controllable pressure rise to desired set point
   - Rapid drop to CPAP / PEEP pressure on termination
   - Low resistance to expiration
   - Sensible management of apnoea during Pressure Support
3. Integrated flow sensors for tidal volume waveforms, loops and Minute Volume monitoring
4. Real time breathing sound simulation based on actual gas flows –
   - Just like an oesophageal stethoscope
   - Think of it like a pulse oximeter for ventilation!
5. Time-synchronisation of control breaths to spontaneous efforts
   - An effective SIMV algorithm
6. Support for spontaneous efforts in-between control breaths
7. Volume-preset Pressure Control mode
   - Where the anaesthetist sets tidal volume, but machine delivers constant pressure
8. Circuit compliance compensation
   - So that the set tidal volume will actually be delivered
9. Fresh gas flow compensation or fresh gas decoupling
   - So that tidal volume is not affected by changes in fresh gas flow
10. ETT and / or breathing circuit resistance compensation
    - Reduces work of breathing for a given ETT / circuit to near-zero
    - Flow-proportional pressure increase on inspiration and
    - Flow-proportional PEEP / CPAP decrease in expiration
11. PEEP / CPAP optimisation
12. Lung recruitment
13. Completely automatic self-checking and calibration
    - With sensible, easily-understood error messages

Note –

1. A 'feature' that works really well on one machine can be almost useless on another – just because a machine says it has the feature doesn't mean it will work well in practice!
2. That the only way to reliably assess work of breathing is to breathe through a machine yourself, in a variety of modes, ideally in a direct A to B comparison

Pressure Support

Pressure Support is a patient-triggered ventilation mode intended to assist spontaneous respiration and reduce work of breathing. The machine must have sensitive flow sensors and the ability to rapidly start and stop high inspired gas flows of at least 120 L/min. Well-designed pressure support modes make it much easier for a patient to breathe through an anaesthetic circuit and provide a precise (and easily adjusted) amount of respiratory support.

Pressure Support means increasing airway pressure by a set amount as soon as the patient breathes in, and dropping airway pressure back to zero (or PEEP / CPAP pressure) as soon as they breathe out.

Any delay in bumping the pressure up and delivering gas flow quickly to the patient on inspiration is unhelpful, especially for tachypnoeic patients. Hence the machine must be able to sensitively and reliably detect the very start of an inspiratory effort (the 'inspiratory trigger'), and respond immediately with lots of gas flow in reserve to avoid 'flow starving' the patient. Not all machines are equal in this regard.

In general the most sensitive inspiratory triggers use inspiratory flow sensors, the sensitivity of which can be adjusted to avoid false triggering on the heartbeat. A good machine can trigger a 5 kg child on an adult circuit and do a better job in supporting their respiration at 40 breaths/min than you or I could with a T-piece!

The effectiveness of this mode also depends on prompt identification of the end of inspiration, a rapid fall in circuit pressure at that time, and minimal expiratory resistance. Again, these parameters vary significantly from machine to machine.

Effective Pressure Support can reduce the work of breathing in a circle circuit to zero or better than zero. It can almost completely eliminate inadequate ventilation, expiratory effort and see / saw respiration on LMA's, even in quite obese patients, and can be fast enough to support gasping or inadequate respiration even in near-moribund patients or small children. $EtCO_2$ values are invariably lower and work of breathing is reduced.

Pressure Support can be used for induction, maintenance and emergence. It is no longer necessary to squeeze the bag at any stage of the anaesthetic, keeping both hands free for other things. This is especially useful when both hands are needed to get a seal around the mask or when you need to support the patient's respiration at the end of the case, send for the next patient and write up the notes at the same time!

Most Pressure Support modes have some fall-back mode in case of apnoea. They will either revert to a control mode like Pressure Control – and stay there – or stay in Pressure Support and self-trigger at the fall-back rate, switching back to patient-triggering as soon as the patient starts to breathe again. The latter is preferable.

To provide the trigger sensitivity, high peak flows and low expiratory resistance required for optimal Pressure Support, 'hybrid' ICU / anaesthesia breathing systems have been developed. These incorporate ICU-type ventilator components within the circle system. Typically the inspiratory flow delivery device (bellows, piston, or blower) is located just proximal to the inspiratory valve of the circuit, and an ICU-type PEEP / CPAP valve is located just after the expiratory valve – just like an ICU ventilator. Circuits like these can provide much better Pressure Support than the traditional circle system layout.

Typical disadvantages of Pressure Support include –

- A significant learning curve. It takes a while to figure out which patients benefit most, how to assess the benefit in a given patient, how to determine the best settings to use, etc. It took me over two years before I became completely comfortable with using Pressure Support on induction of anaesthesia
- Traditional hand-bagging gives us visual feedback by watching the chest move and tactile feedback from squeezing the bag. In Pressure Support, the bag is not held, so that useful tactile information is no longer available. Instead, information about how stiff the lungs are and / or whether or not there is airway obstruction must be deduced from observation of the flow and pressure curves on a screen. If these

waveforms are available, there are very characteristic flow and pressure appearances for obstruction, 'stiff lungs,' etc. However it takes time to become really familiar with these. The utility of the Pressure Support mode in a given ventilator therefore depends not just on performance of the ventilator but also if the user interface provides simultaneous high-fidelity non-auto-scaling flow and pressure curves and whether or not breathing sounds are provided

- The machine be subject to false triggering (if the trigger threshold is too low)
- The machine may fail to trigger (if the threshold is set too high or if the patient becomes obstructed), or be so efficient as to induce cyclical episodes of hyperventilation-induced apnoea
- When the mask is taken off during Pressure Support the ventilator will immediately think that the patient has breathed in, and will dump anaesthetic gas into the room until it empties the bag. It's best to put the ventilator into a 'standby' mode (ie zero gas flow and no support) before adjusting the mask or taking it off the patient to intubate. The ventilator should be able to quickly and easily return from standby back to Pressure Support mode, and to restore all gas flows at the same time. When choosing a new anaesthetic machine, make sure that you can go in and out of standby quickly and easily, otherwise Pressure Support is not going to be much help on induction of anaesthesia

Well implemented Pressure Support provides an incredibly useful addition to our ventilation capabilities. Compared to un-assisted spontaneous respiration you should see obvious improvements in tidal volume, reduced respiratory effort, and improved monitoring. Additionally you should find it much more effective than hand bagging, better at generating CPAP, and very helpful when you want two hands to hold the mask. As an alternative to simple spontaneous respiration it should manage apnoea much better and provide useful alarms for disconnection, hypoventilation, etc.

Poorly implemented Pressure Support is of little benefit to the patient or the anaesthetist and the disadvantages start to outweigh the advantages. If you meet someone who says, "I tried Pressure Support, but it didn't seem much good!", ask what type of machine they were using, and how long they have been using it for.

Work of Breathing

Work = Force × Distance. Power / energy consumption is work done per unit time. For spontaneous respiratory effort, energy consumption translates to pressure exerted by the inspiratory muscles × flow generated per unit time.

Greater respiratory effort is needed to breathe through the circuit of an anaesthetic machine.

Inspiratory work can be completely overcome by Pressure Support. Resistance to gas flow through tubes is proportional to flow rate. 'Proportional assist' is a new ICU mode in which the amount of pressure support is increased dynamically according to inspiratory flow rate. Combined with pressure support this can make the breathing circuit appear more 'transparent' to the conscious patient, especially if they try to take a deeper breath than usual, however simple pressure support is sufficient for almost all anaesthetised patients.

Expiratory resistance adds to work of breathing because greater inspiratory effort is required to get a big enough breath to overcome that resistance. Pressure Support does not reduce expiratory work. Negative PEEP during expiration might seem helpful but isn't. A kind of reverse proportional assist in which PEEP / CPAP is dynamically reduced in proportion to exhaled flow would completely eliminate expiratory resistance but this is not available on any ventilator that I know of.

With good Pressure Support modes, the improvements in ventilation are obvious (just switch it on and off and compare tidal volumes and patient effort clinically, in good machines its chalk and cheese).

Comparing One Machine to Another

Jaber et al have verified that triggering in the latest anaesthesia machines is comparable to that of modern ICU ventilators.

There are no studies that compare overall work of breathing in Pressure Support mode from one anaesthetic machine to the next.

Pressure Control and Volume Preset Pressure Control (Autoflow) Modes

Control modes of ventilation (IPPV) can be implemented around either fixed inspired volumes (classic IPPV / Volume Control) or fixed inspiratory airway pressure (Pressure Control).

Volume control modes of ventilation are popular with anaesthetists because –

- Tidal volumes are maintained even if lung compliance varies during the case (and it does)
- Circuit integrity can be monitored using simple pressure alarms
- Barotrauma and very abnormal lung compliance are uncommon on routine lists
- 10 ml/kg x 10 is easy to remember

Yet the same classic volume control modes are rarely used in ICU. When using a control mode, intensivists almost always choose Pressure Control because –

- Airway pressures are higher even for breaths of the same volume
- Barotrauma and ventilator associated lung injury are reduced when pressure control is compared to volume control
- Analysis of the inspired flow curve in Pressure Support is helpful in fine-tuning inspiratory time
- Patient comfort is greater with synchronised pressure modes
- Inspired and expired flow monitoring can be used to generate equivalent alarms
- The square pressure wave gives maximal time for long time constant lung units to open (ie is more efficient)

Older anaesthetic machines with Ulco type ventilators had effective alarms in Volume Control modes, but there were no useful tidal volume alarms if the machine was set to pressure cycling. So, historically, a whole generation of anaesthetists were trained to use Volume Control mode for IPPV ventilation.

Now that modern anaesthesia ventilators include integrated flow sensors, they can provide effective minute volume based alarms in Pressure Control modes, making Pressure Control as safe to use as Volume Control.

However basic Pressure Control modes do not guarantee a predetermined tidal volume.

To address this and provide the best of both worlds, some newer ventilators can operate fundamentally in pressure mode but also dynamically adjust the driving pressure on a breath by breath basis to maintain a preset tidal volume. These "volume preset, pressure controlled" modes of ventilation go by several acronyms – PRVC, Autoflow, VC+, etc. Typically the first breath is a standard Volume Control breath, and the plateau pressure from this breath is used for the next Pressure Control breath. The machine then dynamically adjusts the pressure used for each subsequent breath to maintain, on average, the desired tidal volume.

In this mode an abrupt reduction in compliance will cause a fall in tidal volume, followed by an algorithmically-generated increase in driving pressure (as for classical Volume Control IPPV).

Pressure Control and Volume Preset Pressure Control (Autoflow) modes of ventilation are gradually being introduced on newer anaesthesia ventilators and provide a small but incremental improvement in ventilation efficiency. The lower peak airway pressures and improved ventilation efficiency of Volume Preset Pressure Control (Autoflow) modes are particularly helpful during laparoscopies.

Optimising Inspiratory and Expiratory Times

Few anaesthetists give much thought to what the best settings are for inspiratory and expiratory time. In most cases, the manufacturer default of an I:E ratio of 1:2 is used, or an arbitrary adjustment is made.

An I:E ratio of 1:2 approximates the normal value found in spontaneously breathing people. It is not necessarily the best value for ventilated anaesthetised patients.

Normal people unconsciously minimise respiratory work and are not concerned about atelectasis. It would be more work – and usually it would be unhelpful – for a conscious person to hold inspiration for any longer than say two seconds, and at the same time, a long time in relaxed expiration has no particular adverse effects.

On the other hand, anaesthetised supine patients are prone to atelectasis and collapse, and once ventilated by machine they do no work – the machine does it all for them. This means that the 'best' settings for the patient can now be those which optimise lung mechanics even if they require more effort to do so.

In a theoretically 'ideal' lung, all lung units have the same time constant. For such a lung, short inspiratory times would not result in regional V/Q inequalities, because all the alveoli would expand to an equal extent. But in a real lung, especially a middle aged anaesthetised patient, some lung units have longer time constants than others. If a number of slow lung units are not fully equilibrated at the end of inspiration, the distribution of ventilation will be unequal. The resulting V/Q inequalities will lead to an A-a gradient and potentially lung collapse and hypoxaemia.

In most cases the anaesthetised patient can be better ventilated by careful fine-tuning of inspiratory and expiratory times (and obviously the respiratory rate, all three being interconnected). This can only be done with a machine that has a Pressure Control mode and can show inspiratory and expiratory flow curves on-screen.

For most anaesthetised patients an I:E ratio of 1:1 at a rate of 12-14 is probably a much better starting point than the traditional values of 1:2 at a rate of 10.

Using Pressure Control mode, one can be sure that the inspiratory time is long enough when the inspiratory flow curve falls to nearly zero by the end of a inspiration. At this point in time, all lung units, regardless of time constant, will have reached equilibration. Holding the inspiratory time any longer provides no additional benefit in terms of ventilation and may have a negative impact on cardiac output. Any shorter and one risks atelectasis, A-a gradients and potential hypoxaemia.

The same considerations apply to expiratory time. Expiratory time should be long enough for expiration to finish, but no longer. Leaving the patient 'stalled' in expiration without further expiratory gas flow will encourage collapse. On the other hand, if expiration is not complete when the next breath starts, gas trapping and 'auto-peep' will occur, leading to hyperinflation and typically a negative impact on cardiac output.

Consider an obese patient in lithotomy with the following ventilator settings –

- Inspiratory time - 2s
- Expiratory time - 4s
- Rate – 10 breaths/minute
- I:E ratio – 1:2

If that patient fully exhales in one second (a typical value in this situation, because their weight helps push it out faster than normal), what happens to their lungs for the remaining three seconds of that expiratory time? Certainly it doesn't help their ventilation, because no more gas is leaving the lung. All that happens is that their lungs are progressively compressed under the weight of their abdomen. A shorter expiratory time – so that inspiration starts at the exact moment expiration has just finished – will permit a higher respiratory rate, lower tidal volumes, lower airway pressures while retaining overall minute ventilation and FRC.

Managing Lung Collapse – Recruitment Manoeuvres

Lung collapse occurs rapidly following induction of anaesthesia, especially when the following are present –

- No N_2 splinting (ie pre-oxygenation, use of nitrous)
- Periods of apnoea at zero PEEP (eg during intubation, induction apnoea)
- Obese or elderly patients
- Head down position
- Absence of appropriate levels of PEEP / CPAP
- Short inspiratory times
- Long expiratory times
- Underlying respiratory disease

It is not uncommon for as much as ½ to ⅓ of a patient's lung volume to become collapsed during induction where muscle relaxants are used to facilitate intubation. This usually goes unrecognised.

Until I started inducing patients with Pressure Support and CPAP I was completely unaware of just how much collapse occurs on induction – and how quickly it happens. What I saw was that the patient's lung compliance would fall abruptly after intubation – due to collapse. In fact, if I just took the mask off and did nothing for 30-40

seconds, lung compliance would fall a great deal. When I did the maths on how much stiffer the lung was, it became apparent that many patients 'lose' nearly a third of their functional lung volume on intubation!

One way to quantify collapse – with a modern ventilator – is to assess lung compliance before and after a recruitment manoeuvre. When I say lung compliance I mean lung plus chest wall or total compliance. The normal units are ml/cmH_2O. Healthy non-anaesthetised patients typically have lung compliance of about 100ml/cmH_2O, but typically under anaesthesia we are accustomed to values more like 50ml/cmH_2O.

Recruitment manoeuvres re-expand collapsed lung. Classically they involve holding airway pressure at between 30 and 45 cmH_2O for say thirty seconds. The magnitude of the improvement in lung compliance after recruitment quantifies the extent of any pre-existing lung collapse. Pressure Control mode can be used for both measurement of compliance and performing recruitment manoeuvres.

The approach I use is to turn the ventilator into Pressure Control mode and then –

- Optimise inspiratory and expiratory times as above
- Set / guess an initial PEEP value
- Select a differential pressure that gives a modest (say 50ml/kg) initial tidal volume
- Increase PEEP in 5 cmH_2O steps, holding each step for say 3-4 breaths
- Keep going up to a PEEP of 20-30, holding there for say 30 seconds
- Stepping the PEEP back down again to the PEEP value that gives the best tidal volume
- Noting the improvement in tidal volume, if any, at each step

This technique not only quantifies the improvement in compliance after recruitment – we can measure the increase in tidal volume for the same differential airway pressure – but it also determines the optimal mechanical PEEP value to use thereafter.

Strategies for maintaining open lungs (aka preventing or treating lung collapse) include regular recruitment manoeuvres, using and optimising PEEP, avoiding apnoea at all times, using Pressure Control mode for ventilation, optimising inspiratory and expiratory times as described above, and maximising circuit nitrogen levels. Should the patient be extubated supine in 100% oxygen they are likely to quickly re-collapse, so consideration should be given to extubating them sitting up, with PEEP on and not in 100% oxygen.

Optimising Ventilator Settings in Different Clinical Situations

Optimising means fine-tuning settings to best achieve a defined outcome.

For instance, to reduce physical trauma to the lung through shear stress, the lung should be recruited, PEEP optimised, a constant-pressure mode of ventilation used, and the ventilation pattern should tend to low-volume higher-frequency settings. Mild hypercapnoea may be acceptable.

If CO_2 elimination is important, rapid respiration with good tidal volumes, even if inspiration or expiration are incomplete or airway pressures are high, will easily solve the problem except if the patient has significant airflow resistance (eg severe asthma).

When airway resistance is a major issue, longer inspiratory and expiratory times and permissive hypercapnoea may be required. If oxygenation is an issue, it's important to clear atelectasis with recruitment, find an optimal PEEP (taking care not to negatively impact cardiac output too much), use a constant-pressure mode of ventilation, and try to ensure that all alveoli, including long-time constant (hypoxic) alveoli, are ventilated effectively. This means that both inspiration and expiration should be long enough in time so that the flow curves fall to zero before the next phase commences.

Phasic – in and out – gas flow during the respiratory cycle will have a net effect on the movement of pulmonary secretions in and out of the lungs. The drag of airflow on secretions is much less at low flows than high flows. Keeping flows low (ie constant) on inspiration while allowing high expiratory peak flows on expiration will cause elimination of sputum; doing the opposite just blows the spit back in.

From the above it can be seen that there really is an 'art' to ventilation. This has largely been lost on anaesthetists because our equipment has not given us the tools to 'see' what we are doing. With the newer machines, we can regain that sense of enthusiasm and control when dealing with patients with sick lungs.

A Short Note About Gas Consumption

In any gas-driven bellows-type ventilator gas consumption must at least equal the patient's minute volume. If a venturi is used (eg Ulco Campbell) at least 50% more is required. Ten litres/minute of drive gas for five hours a day, five days a week, 50 weeks a year equals 750,000 litres of oxygen per machine – in addition to fresh gas flow. In contrast, zero gas consumption for an electrically driven piston or turbine. This is partly why Dräger have moved away from bag and bottle type gas driven ventilators.

References

1. Jaber S, Tassaux D, Sebbane M, Pouzeratte Y, Battisti A, Capdevila X, Eledjam JJ, Jolliet P. Performance characteristics of five new anesthesia ventilators and four intensive care ventilators in pressure-support mode: a comparative bench study. Anesthesiology 2006; 105(5): 944-52
2. Bourgain JL, Billard V, Cros AM. Pressure support ventilation during fibreoptic intubation under propofol anaesthesia. British Journal of Anaesthesia 2007; 98(1): 136-40
3. Brimacombe J, Keller C, Hormann C. Pressure support ventilation versus continuous positive airway pressure with the laryngeal mask airway: a randomized crossover study of anesthetized adult patients. Anesthesiology 2000; 92(6): 1621-3
4. von Goedecke A, Brimacombe J, Hormann C, Jeske HC, Kleinsasser A, Keller C. Pressure support ventilation versus continuous positive airway pressure ventilation with the ProSeal laryngeal mask airway: a randomized crossover study of anesthetized pediatric patients. Anesthesia & Analgesia 2005; 100(2): 357-60
5. Banchereau F, Herve Y, Quinart A, Cros AM. Pressure support ventilation during inhalational induction with sevoflurane and remifentanil in adults. European Journal of Anaesthesiology 2005; 22(11): 826-30
6. Bosek V, Roy L, Smith RA. Pressure support improves efficiency of spontaneous breathing during inhalation anesthesia. Journal of Clinical Anesthesia 1996; 8(1): 9-12

ICU TOPICS

A/Prof Frank van Haren

The Canberra Hospital
Canberra, Australia

Introduction

In this era of evidence-based medicine, an enormous number of randomised controlled clinical trials (RCTs) are being done to improve the scientific foundation of daily intensive care patient management and ultimately to improve patient centred outcomes such as mortality. Data extracted from PubMed reveals a total of 168 published RCTs on sepsis, and 303 published RCTs on mechanical ventilation in 2011 alone. Unfortunately the majority of ICU studies demonstrate no beneficial effect of the intervention on the outcome, and interventions that do show a beneficial effect are often not implemented in clinical practice for a number of reasons.[1]

Recent Mostly-negative-but-possibly-practice-changing Trials

Disease Modifying Agents for Sepsis

The search for a disease-modifying agent that improves outcome in patients with sepsis has been on-going for many years. The list of initially promising agents that didn't live up to the expectations in clinical trials is becoming very long, and includes nitric oxide synthase inhibitors such as L-NMMA; anti-inflammatory agents such as corticosteroids and NSAIDs; anticoagulants such as anti-thrombin III and tissue pathway factor inhibitor.

The most promising and best-studied agent was recombinant human activated protein C, or drotrecogin alpha activated (DrotAA). Sufficient preclinical data and pathophysiological plausibility warranted a large RCT. In the PROWESS study (Prospective Recombinant Human Activated Protein C Worldwide Evaluation in Severe Sepsis), a phase 3 multicentre PRCT published in 2001, DrotAA administration was associated with a 6.1% absolute mortality reduction.[2] However, because of concerns with this trial (the trial was stopped early for efficacy; the study protocol was modified during the trial possibly influencing the outcome; the lack of confirmatory data from other PRCTs) the European Medicines Agency concluded in 2007 that sufficient doubt existed to warrant a new PRCT. In the multicentre randomised double-blind placebo-controlled PROWESS-SHOCK study, 1,697 patients with septic shock were randomised to receive DrotAA or placebo for 96 hours with 28-day mortality as primary outcome. The study failed to show a difference in overall mortality and in outcomes in any of the predefined subgroups.[3] On 25 October 2011 Eli Lilly and Co. announced a worldwide voluntary market withdrawal of Xigris, based on the results of PROWESS-SHOCK.

Fluid Management in Critically Ill Patients

Choice of Fluid

Resuscitation fluids administered to critically ill patients are not innocent bystanders, but may have an effect on morbidity and mortality. Some of these effects appear to be immune mediated and may depend on the type of fluid used as well as the timing of the fluids given.[4,5] Until recently, fluid choice and more specifically the crystalloid versus colloid debate in anaesthesia and intensive care has been to a great extent dependent on belief, dogma and local availability or practice and less so on sound evidence.[6]

The publication of the SAFE trial in 2004 (a comparison of 4% albumin and 0.9% saline for fluid resuscitation in the intensive care unit) confirmed that albumin resuscitation, although safe, does not have any significant benefit over saline resuscitation.[7] The SAFE study did suggest that patients with traumatic brain injury resuscitated with albumin had an increased mortality rate compared to saline. This was confirmed in a post hoc follow-up study of these patients.[8] Subgroup analysis of the SAFE study also showed a non-significant trend towards improved

outcome for patients with severe sepsis resuscitated with albumin. Following this interesting observation, three large-scale randomised studies were initiated to determine whether this potential benefit could be confirmed. The first of these studies, performed by the group of Dr Mira in France, has been presented at a major scientific meeting but not yet published. In this approximately 800 patients study, albumin resuscitation did not result in improved mortality or improvement of any of the secondary outcome measures. The second study by Dr Gattinoni et al in Italy is close to finishing recruitment of 1,800 patients, and the Canadian Critical Care Trials Group is conducting the third study.

With regards to modified starch solutions (hydroxyl-ethyl starch, HES), there has been much controversy regarding the safety and potential benefits of these solutions. Nonetheless, HES solutions are the most widely used colloids in the world, mainly because of widespread use in Europe. Studies investigating older HES preparations and hyperoncotic solutions found possible evidence of harm, but this was not confirmed in a large cross-sectional observational study.[9,10] In a recent meta-analysis of poor quality studies of a newer HES preparation (6% HES 130/0.4), and after exclusion of retracted fraudulent studies conducted by Dr Boldt, no harm or benefit of 6% HES 130/0.4 could be shown.[11] In a recent multicentre PRCT done by the Scandinavian Starch for Severe Sepsis / Septic Shock (6S) trial group, 804 patients with severe sepsis were randomly assigned to fluid resuscitation in ICU with either 6% HES 130/0.42 or Ringers' acetate.[12] The patients receiving HES had an increased risk of death at day 90 (51% vs 43%; RR 1.17; 95% CI 1.01-1.36; p=0.03), and were more likely to require renal-replacement therapy (22% vs 16%; RR 1.35; 95% CI 1.01-1.80; p=0.04).

Finally, in the recently completed Crystalloid Hydroxy-Ethyl Starch Trial (CHEST), 7,000 intensive care patients were randomised to receive fluid resuscitation with starch (6% hydroxyethyl starch 130/0.4) or saline (0.9% sodium chloride).[13] The results of this trial are still under embargo at the time this abstract was written, and will be publicly released September 2012.

New and promising developments currently under investigation include hypertonic resuscitation fluids[14,15] and balanced fluids.[16]

Timing of Fluid Resuscitation

It has become more clear over the last seven years or so, that overly aggressive fluid resuscitation is associated with worse outcome.[17,18] It has been suggested that adequate initial fluid resuscitation combined with conservative post-resuscitation fluid management is associated with improved outcomes and mortality.[19] However, most of these data are observational and could potentially have been confounded by patient severity unbalance, where sicker patients receive more fluid and have worse outcomes. Three large studies evaluating early goal directed therapy are currently underway and are expected to answer some of these pertinent questions.

Conclusion

In conclusion, the choice, amount and timing of fluid resuscitation have an impact on patients' morbidity and mortality. Based on the results of recent large clinical studies, there is no clear benefit in using albumin or hydroxyl-ethyl starch over crystalloid solutions in critically ill patients. Based on the best available evidence and awaiting on-going clinical trials, early adequate fluid resuscitation followed by conservative post-resuscitation fluid management is recommended.

Beta-adrenergic Agonists for Treatment of Acute Respiratory Distress Syndrome (ARDS)

Beta-adrenergic agonists have several potential beneficial effects that might enhance the resolution of acute lung injury (ALI) and ARDS including up-regulation of alveolar fluid resorption, anti-inflammatory effects and endothelial and epithelial protective effects. Beta-agonists have been shown to reduce pulmonary oedema in preclinical models of acute lung injury. Retrospective data suggested that inhaled salbutamol was associated with a shorter duration and lower severity of ALI.[20] In the phase II beta-agonist lung injury trial (BALTI) intravenous salbutamol significantly reduced extra-vascular lung water and plateau pressures in patients with ARDS.[21]

Based on these encouraging findings two large-scale trials were conducted. In the BALTI-2 study, the same investigators planned to randomise 1,134 patients with ARDS to receive intravenous salbutamol (15 mcg/kg IBW/h) or placebo. The study was stopped after 326 patients for safety concerns, with an increased 28-day mortality in the treatment arm (35% vs 23%, RR 1.47, 95% CI 1.03-2.08).[22] In addition, the salbutamol group had fewer ventilator-free and organ failure-free days, and more frequent tachycardia, new arrhythmias and lactic

acidosis. In the other large randomised study undertaken by the National Heart Blood and Lung Institute's ARDS Clinical Trials Network, the efficacy of inhaled salbutamol (5 mg every 4 hours) was compared to placebo in patients with ALI / ARDS.[23] The study enrolled 282 of a planned 1,000 patients and was stopped for futility after the first planned interim analysis. No significant differences were found with regards to ventilator-free days or mortality.

In conclusion, despite strong preclinical data and good biological rationale, neither inhaled nor intravenous beta-adrenergic agonists are beneficial in patients with ALI and ARDS. Intravenous delivery of salbutamol was poorly tolerated in critically ill patients with adverse cardiovascular effects and an increased 28-day mortality.

Nutrition in Critically Ill Patients

Early Parenteral Nutrition

The timing and optimal route of nutrition in critically ill patients remain unclear. The use of early parenteral nutrition (PN) supplement to reach caloric goals is recommended in European guidelines (within 48 hours after ICU admission) but not in North American guidelines (recommended initiation after day eight). To address this question, a randomised multi-centre study in 4,640 patients was conducted to compare early versus late initiation of PN as supplement to enteral nutrition to achieve the daily caloric goal intake.[24] Patients in the late initiation group had a small (6.3%) reduction in ICU length of stay, fewer ICU infections (22.8% vs 26.2%, p=0.008), a reduction in the proportion of patients requiring more than two days of ventilation, a reduction in the duration of renal replacement therapy, and a reduction in healthcare costs. No differences were observed with respect to mortality.

Omega-3 Fatty Acids in ARDS

Preclinical studies and several small clinical trials have suggested a potential benefit for omega-3 fatty acid supplementation in patients with ALI / ARDS. Nutrition enriched with omega-3 fatty acids can reduce inflammatory eicosanoid production by altering membrane phospholipid composition and can provide substrate for anti-inflammatory mediators such as resolvins and protectins.

Two recent studies addressed this issue. In a multicentre phase II clinical trial, 90 mechanically ventilated patients with ALI / ARDS were randomised to receive 6-hourly fish oil or placebo.[25] There was no difference in bronchoalveolar lavage fluid levels of Il-8, organ failure, ventilator-free days, ICU-free days or mortality. The other study "OMEGA" was conducted by the National Heart Blood and Lung Institute's ARDS Clinical Trials Network and planned to randomise 1,000 patients to receive an enteral supplement enriched in omega-3 fatty acids, gamma-linolenic acid and antioxidants versus an isocaloric control feed.[26] The trial was stopped for futility after 272 patients were enrolled. Patients receiving the omega-3 supplement had significantly fewer ventilator-free days, fewer organ failure free days and a trend towards higher 60-day mortality (26.6% vs 16.3%, p=0.054).

Conclusion

In conclusion, there is no benefit to initiating very early parenteral nutrition as a supplement to enteral nutrition to achieve caloric goals in critically ill patients. Enteral supplementation of omega-3 fatty acids is not beneficial in ALI / ARDS and could potentially be harmful.

Conclusions

The past year in Intensive Care Medicine has again seen the publication of a number of large PRCTs that may aid us in our daily management of critically ill patients. Unfortunately most of these trials are "negative," in that they only tell us what we should not do; the evidence outlining or providing guidance towards what we should do remains limited.

References

1. Ospina-Tascon GA, Buchele GL, Vincent JL. Multicenter, randomized, controlled trials evaluating mortality in intensive care: doomed to fail? Crit Care Med 2008; 36(4): 1311-22

2. Bernard GR, Vincent JL, Laterre PF, et al. Efficacy and safety of recombinant human activated protein C for severe sepsis. N Engl J Med 2001; 344(10): 699-709
3. Ranieri VM, Thompson BT, Barie PS, et al. Drotrecogin alfa (activated) in adults with septic shock. N Engl J Med 2012; 366(22): 2055-64
4. Rivers EP, Kruse JA, Jacobsen G, et al. The influence of early hemodynamic optimization on biomarker patterns of severe sepsis and septic shock. Crit Care Med 2007; 35(9): 2016-24
5. Dorresteijn MJ, van Eijk LT, Netea MG, et al. Iso-osmolar prehydration shifts the cytokine response towards a more anti-inflammatory balance in human endotoxemia. J Endotoxin Res 2005; 11(5): 287-93
6. Finfer S, Liu B, Taylor C, et al. Resuscitation fluid use in critically ill adults: an international cross-sectional study in 391 intensive care units. Crit Care 2010; 14(5): R185
7. Finfer S, Bellomo R, Boyce N, et al. A comparison of albumin and saline for fluid resuscitation in the intensive care unit. N Engl J Med 2004; 350(22): 2247-56
8. Myburgh J, Cooper DJ, Finfer S, et al. Saline or albumin for fluid resuscitation in patients with traumatic brain injury. N Engl J Med 2007; 357(9): 874-84
9. Brunkhorst FM, Engel C, Bloos F, et al. Intensive insulin therapy and pentastarch resuscitation in severe sepsis. N Engl J Med 2008; 358(2): 125-39
10. Vincent JL, Sakr Y, Sprung CL, et al. Sepsis in European intensive care units: results of the SOAP study. Crit Care Med 2006; 34(2): 344-53
11. Gattas DJ, Dan A, Myburgh J, et al. Fluid resuscitation with 6% hydroxyethyl starch (130/0.4) in acutely ill patients: an updated systematic review and meta-analysis. Anesth Analg 2012; 114(1): 159-69
12. Perner A, Haase N, Guttormsen AB, et al. Hydroxyethyl starch 130/0.42 versus Ringer's acetate in severe sepsis. N Engl J Med 2012; 367(2): 124-34
13. The Crystalloid versus Hydroxyethyl Starch Trial: protocol for a multi-centre randomised controlled trial of fluid resuscitation with 6% hydroxyethyl starch (130/0.4) compared to 0.9% sodium chloride (saline) in intensive care patients on mortality. Intensive Care Med 2011; 37(5): 816-23
14. van Haren FM, Sleigh J, Boerma EC, et al. Hypertonic fluid administration in patients with septic shock: a prospective randomized controlled pilot study. Shock 2012; 37(3): 268-75
15. van Haren FM, Sleigh J, Cursons R, et al. The effects of hypertonic fluid administration on the gene expression of inflammatory mediators in circulating leucocytes in patients with septic shock: a preliminary study. Ann Intensive Care 2011; 1(1): 44
16. Wilkes NJ, Woolf R, Mutch M, et al. The effects of balanced versus saline-based hetastarch and crystalloid solutions on acid-base and electrolyte status and gastric mucosal perfusion in elderly surgical patients. Anesth Analg 2001; 93(4): 811-16
17. Boyd JH, Forbes J, Nakada TA, et al. Fluid resuscitation in septic shock: a positive fluid balance and elevated central venous pressure are associated with increased mortality. Crit Care Med 2011; 39(2): 259-65
18. Payen D, de Pont AC, Sakr Y, et al. A positive fluid balance is associated with a worse outcome in patients with acute renal failure. Crit Care 2008; 12(3): R74
19. Murphy CV, Schramm GE, Doherty JA, et al. The importance of fluid management in acute lung injury secondary to septic shock. Chest 2009; 136(1): 102-9
20. Manocha S, Gordon AC, Salehifar E, et al. Inhaled beta-2 agonist salbutamol and acute lung injury: an association with improvement in acute lung injury. Crit Care 2006; 10(1): R12
21. Perkins GD, McAuley DF, Thickett DR, et al. The beta-agonist lung injury trial (BALTI): a randomized placebo-controlled clinical trial. Am J Respir Crit Care Med 2006; 173(3): 281-7
22. Gao Smith F, Perkins GD, Gates S, et al. Effect of intravenous beta-2 agonist treatment on clinical outcomes in acute respiratory distress syndrome (BALTI-2): a multicentre, randomised controlled trial. Lancet 2012; 379(9812): 229-35
23. Matthay MA, Brower RG, Carson S, et al. Randomized, placebo-controlled clinical trial of an aerosolized beta(2)-agonist for treatment of acute lung injury. Am J Respir Crit Care Med 2011; 184(5): 561-8
24. Casaer MP, Mesotten D, Hermans G, et al. Early versus late parenteral nutrition in critically ill adults. N Engl J Med 2011; 365(6): 506-17
25. Stapleton RD, Martin TR, Weiss NS, et al. A phase II randomized placebo-controlled trial of omega-3 fatty acids for the treatment of acute lung injury. Crit Care Med 2011; 39(7): 1655-62
26. Rice TW, Wheeler AP, Thompson BT, et al. Enteral omega-3 fatty acid, gamma-linolenic acid, and antioxidant supplementation in acute lung injury. Jama 2011; 306(14): 1574-81

DRUG DOSAGE – HOW MANY INCHES SHOULD YOU GIVE?

Dr Tim Short

Auckland City Hospital,
Auckland

Numerous recommendations have been made about how much drug we should give our patients. In particular, how should we adjust drug dose in otherwise healthy patients for obesity and age. The literature is very confused with numerous models for adjusting drug dose having been described. The confusion mainly results from a lack of clarity in conceptual thinking and a plethora of small studies describing limited populations.

The first truth is – *a model only describes the population that it was created from*. However even though all drug dose models are empirical, the second truth is that *a good model will allow extrapolation beyond the population it was created from*.

There are two basic questions I try and answer about some common anaesthetic drugs, how to scale –

1. Induction (bolus) doses
2. Infusion rates

Obesity

Numerous models have been suggested for scaling to body size –

Total body weight (TBW)		Wt
Body mass index (BMI)	Quetelet[1]	Wt / Ht^2
Lean tissue mass (LTM)	James[2]	$1.1 \times Wt - 128 \times (Wt / Ht)^2$
Body surface area (BSA)	Dubois[3]	$Wt^{0.425} \times Ht^{0.727} \times 0.007184$
Fat free mass (FFMd)	Deurenberg[4]	$40 - (1.2 \times BMI) + 0.23 \times Age - 16.2$
Fat free mass (FFM)	Janmahasatian[5]	$(9270 \times Wt) / (6680 + 216 \times BMI)$
Ideal body weight (IBW)	Devine[6]	$45.4 + 0.89 \times (Ht - 152.4) + 4.5$
Adjusted (IBWa)	la Colla[7]	$IBW + 0.4 \times Wt$
Allometric size model (ASM)	Cortinez[8]	$(Wt / 70)^{0.75}$

(Note – LTM, FFM and IBW are male formula only)

Some of these models are entirely empirical, usually derived from a description of drug dosage in obesity from a single study and some are based on research into body composition. For instance, Deurenberg studied changes in body composition with age, showing the reduction in body fat that occurs. Janmahasatian described changes in body composition with obesity in a group of middle-aged adults. James didn't study obese people and the equation shape is fundamentally flawed.

Graphing these equations for a 70kg, 170cm male who grows fatter makes them much easier to understand (Figure 1). Notice that between the extremes of total body weight, that increases linearly, and ideal body weight, that doesn't change, there is little to choose between the various curves. If all the equations were redrawn in the simple form $ABW = a + b(TBW)^c$ they would be easier to compare. Induction dose is largely distributed into the ECF and there is a correlation with cardiac output.[9] In obesity, CO and blood volume are increased only slightly and cardiac index is unchanged.[10,11] They follow closely to the FFM line on figure 1.

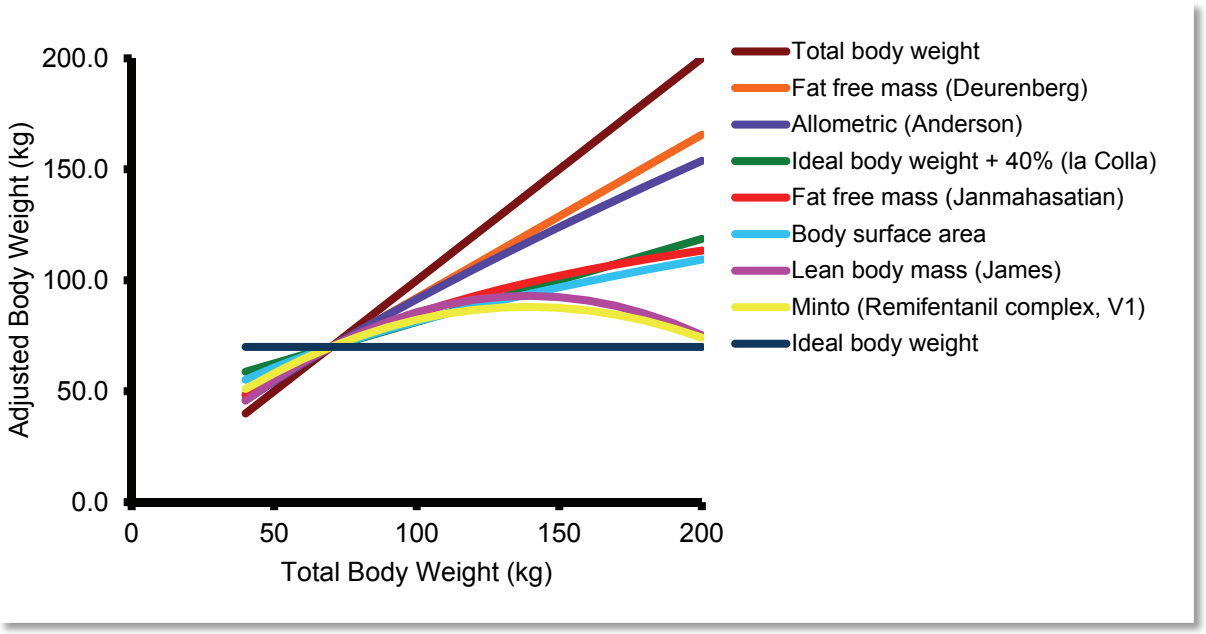


Figure 1. How each model describes a 70kg, 170cm adult male who grows fatter

Propofol

Propofol has been extensively studied. The drug is highly lipid soluble and so bolus dosage and infusion dosage behave differently. Analysis of a composite data set of thin, typical and obese patients [Short unpublished[12,13,14]] shows a dual picture. For bolus dosage, FFM and IBW performed similarly. For infusion adjustment ASM or TBW performed best, presumably because fat takes up propofol almost infinitely, although it is rather slow to get there and therefore does not influence induction dose. Measuring cardiac output confirmed FFM as an appropriate scalar for induction, although IBM was not assessed in this study.[15] The subject requires further analysis to come up with one model that predicts all patients. The Marsh model performed very poorly in obese subjects, presumably because none were included in the original study. None of the studies provided evidence that there were pharmacodynamic differences in obesity.

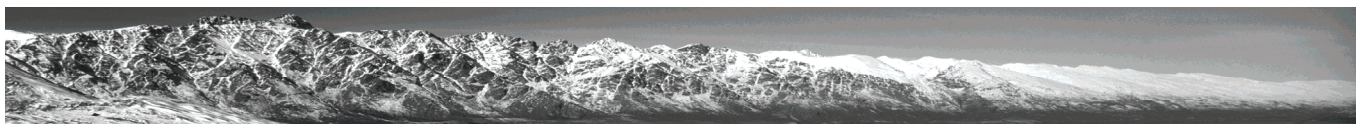
Opioids

Fentanyl should be dosed by either FFM, or linearly until TBW is 100 – 110 kg and then no further adjustment. It is noted that there may be greater respiratory depression for a given plasma concentration in the obese.[16]

Remifentanil has been studied in obesity [Egan 1998]. LTM was found to be an accurate predictor of blood concentration. The Minto model is based on an age stratified group of patients, and did not specifically look at the influence of obesity.[17] It scales the pharmacokinetics to LBM. This equation is flawed as explained in Figure 1. If FFM is substituted for LBM, then the model works better in obesity.[18] When the data are reanalysed using Egan's data taken in obese patients as well it performs better using FFM. The question is what to do with PK model controlled dosing in obese patients, such as is available in the Alaris pumps. Simulation analysis indicates that adjusting weight up to 100 – 110 kg with no further adjustment performs adequately.

Volatiles

Obesity can reduce functional residual capacity, expiratory reserve volume and total lung capacity. It has little effect on uptake and elimination of volatiles. Desflurane uptake in intubated, ventilated patients was virtually unaltered.[19] Isoflurane had a slightly larger ratio of F_i/F_{ET} consistent with its higher lipid solubility.[20] The results are consistent with the low perfusion of fat and small change in CI at rest in the obese. Sevoflurane has yielded similar results and demonstrated no change in either $T_{½}k_{eo}$ or pharmacodynamic effect as measured by BIS.[21]



Relaxants

All muscle relaxants are highly ionized molecules with low lipid solubility, they distribute in a volume equivalent to ECF. Clinical studies demonstrate duration of action is significantly longer if TBW dosing is used and IBW has been found to be the best descriptor of dose for atracurium, cisatracurium, vecuronium and rocuronium.[22-26] The dose of suxamethonium has been studied clinically. Intubating conditions were better when TBW dosing was used, however, examination of the graphs reveals that NMB was almost as profound with LTM or IBW dosing, but lasted 7 and 8 minutes, rather than 11.5 minutes with TBW dosing for recovery of 90% of twitch height.[27]

Other Drugs

There are no studies of ketamine or dexmedetomidine in obesity. Looking at their ionization and distribution volumes, a guess is that ketamine would adjust by IBW and dexmedetomidine by FFM.

Drug	Recommended Model in Obesity
Propofol	TBW or ASM
Remifentanil	FFM
Fentanyl	FFM
Relaxants	IBW
Volatiles	No adjustment
Ketamine	IBW my guess
Dexmedetomidine	FFM my guess

Table 1. Recommended models in obesity for common drugs. For intravenous drugs, recommendations are for infusions. For bolus dosing, FFM or IBW appears best for most drugs

Age

There are only a few high definition studies for many commonly used drugs.

Propofol

Schnider studied eight young, eight middle aged and eight elderly patients in a detailed PKPD study.[12,28] The analysis shows a similar decrease with age to remifentanil, using the EEG as a measure of effect. T_{peak} was increased from 1.5 minutes at age 20 to about 1.8 minutes, but was highly variable.

Opioids

Remifentanil has been studied in detail by Minto, who studied 20 young, 20 middle aged and 20 elderly patients.[17,29] There was a significant reduction in dose requirement with age for the same observed effect. T_{peak} was also doubled from 1.5 at age 20 years to 3 minutes by age 80 years.

Volatiles

These have been summarised by Nickalls and Mapleson.[30] There is a less severe reduction in MAC than for intravenous agents (figure 2). One small study measuring isoflurane concentrations in arterial blood found no change in end-tidal to arterial gradient for with age.

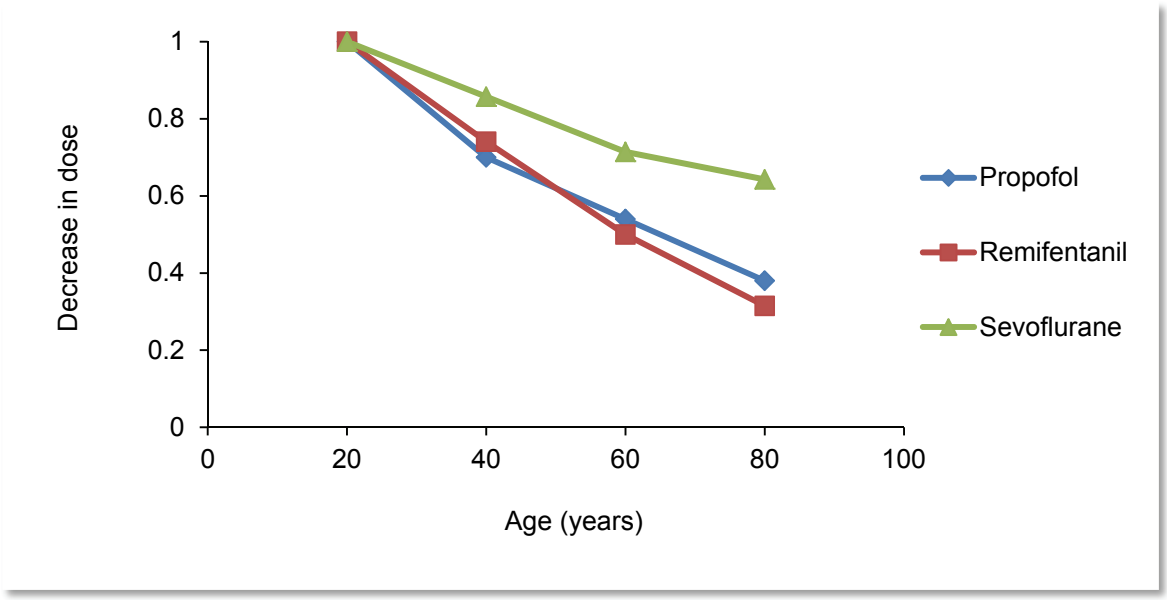


Figure 2. Reduction in dose requirements with age for three common anaesthetic drugs

Relaxants

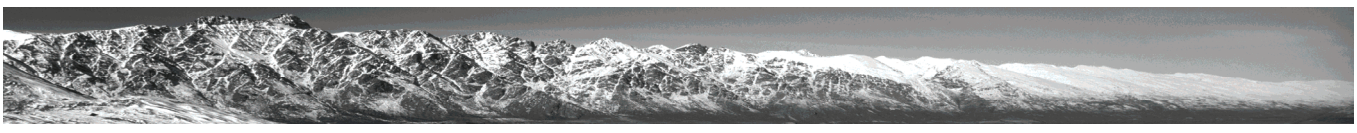
A comparison of patients aged 20 – 40 years with patients aged 60 – 75 years found onset time for rocuronium to be increased from 82 to 127 seconds and duration nearly doubled.[23] Another study found no change in onset time for unadjusted doses of atracurium, rocuronium and vecuronium but a significant increase in duration of effect.[31] Given the increase in duration of effect, it is likely there is a reduction in onset time that is compensated by the relative increase in dose with age. Only a high-resolution study can unravel this, but it is likely these drugs follow a similar path on figure 2 to the drugs graphed.

Conclusion

Obesity reduces drug requirements for intravenous drugs with the exception of propofol when infused, but the degree it is reduced by varies between drugs according to their disposition. Age has a large effect in reducing drug dose requirements, but there have been few high-resolution studies. There are no studies of anaesthetic drug interactions and age. The case for dose titration and direct measurement of effect in the elderly is very strong!

References

1. Quetelet LAJ, eds. Physique Sociale vol 2. Brussels, C. Muquardt,1869: 92
2. James W. Research on obesity. London: Her Majesty's Stationery Office; 1976
3. Du Bois D, Du Bois EF. Clinical calorimetry. Tenth paper. A formula to estimate the approximate surface area if height and weight be known. Arch Intern Med 1916; 17: 863
4. Deurenberg P, Deurenberg-Yap M, Foo LF, Schmidt G, Wang J. Differences in body composition between Singapore Chinese, Beijing Chinese and Dutch children. Eur J Clin Nutr. 2003; 57: 405-9
5. Janmahasatian S, Duffull SB, Ash S, et al. Quantification of lean bodyweight. Clin Pharmacokinet 2005; 44: 1051-1065
6. Devine D. Case study number 25 gentamicin therapy. Drug Intell Clin Pharm 1974; 8: 650-5
7. La Colla L, Albertin A, La Colla G, et al. No adjustment vs. adjustment formula as input weight for propofol target-controlled infusion in morbidly obese patients. Eur J Anaesthesiol 2009; 26: 362-369
8. Cortınez LI, Anderson BJ, Penna A, Olivares L, Munoz HR, Holford NHG, Struys MMRF, Sepulveda M. Influence of obesity on propofol pharmacokinetics: derivation of a pharmacokinetic model. Br J Anaesth 2010; 105: 448-56



9. Ingrande J, Brodsky JB, Lemmens HJM. Lean Body Weight Scalar for the Anesthetic Induction. Dose of Propofol in Morbidly Obese Subjects. Anesth Analg 2011; 113: 57-62
10. Stelfox, HT, Ahmed SB, Ribeiro RA, Gettings EM,Pomerantsev E, Schmidt U. Hemodynamic monitoring in obese patients: The impact of body mass index on cardiac output and stroke volume. Crit Care Med 2006; 1243-6
11. Wennesland R, Brown E, Hopper J, Hodges JL, Guttentag E, Scott KG, Tucker IN, Bradley B. Red cell, plasma and blood volumein healthy men measured by radiochromium(Cr1) cell tagging and haematocrit. Influence of age, somatotype and habits of physical activity on the variance after regression of volumes to height and weight combined. J Clin Invest 1959; 1065-77
12. Schnider TW, Minto CF, Gambus PL, Andresen C, Goodale DB, Shafer SL, Youngs EJ. The influence of method of administration and covariates on the pharmacokinetics of propofol in adult volunteers. Anesthesiology 1998 May; 88: 1170-82
13. Servin F, Farinotti R, Haberer JP, Desmonts JM. Propofol infusion for maintenance of anesthesia in morbidly obese patients receiving nitrous oxide. A clinical and pharmacokinetic study. Anesthesiology 1993; 78: 657-65
14. Egan TD, Huizinga B, Gupta SK, et al. Remifentanil pharmacokinetics in obese versus lean patients. Anesthesiology 1998; 89: 562-73
15. Ingrande J, Brodsky JB, Hendrikus JM, Lemmens JM. Lean body weight scalar for the anesthetic induction dose of propofol in morbidly obese subjects. Anesth Analg 2011; 113: 57-62
16. Shibutani K, Inchiosa MA, Sawada K, Bairamain M. Accuracy of phamracokinetic models for predicting plasma fentanyl concentrations in lean and obese surgical patients. Derivation of dosing weight ('pharmacokinetic mass'). Anesthesiology 2004; 101: 603-13
17. Minto CF, Schnider TW, Egan TD, Youngs E, Lemmens HJ, Gambus PL, Billard V, Hoke JF, Moore KH, Hermann DJ, Muir KT, Mandema JW, Shafer SL. Influence of age and gender on the pharmacokinetics and pharmacodynamics of remifentanil. I. Model development. Anesthesiology 1997 Jan; 86(1): 10-23
18. La Colla L, Albertin A, La Colla G, et al. Predictive performance of the 'Minto' remifentanil pharmacokinetic parameter set in morbidly obese patients ensuing from a new method for calculating lean body mass. Clin Pharmacokinet 2010; 49: 131-9
19. La Colla G, La Colla L, Turi S, Poli D, Albertin A, Pasculli N, Bergonzi PC, Gonfalini M, Ruggieri F. Effect of morbid obesity on kinetic of desflurane: wash-in wash-out curves and recovery times. Minerva Anestesiol. 2007; 73: 275-9
20. Lemmens HJ, Saidman LJ, Eger EI 2nd, Laster MJ. Obesity modestly affects inhaled anesthetic kinetics in humans. Anesth Analg 2008; 107: 1864-70
21. Cortínez LI, Gambús P, Trocóniz IF, Echevarría G, Muñoz HR. Obesity does not influence the onset and offset of sevoflurane effect as measured by the hysteresis between sevoflurane concentration and bispectral index. Anesth Analg 2011; 113: 70-6
22. Leykin Y, Pellis T, Lucca M, Lomangino G, Marzano B, Gullo A. The effects of cisatracurium on morbidly obese women. Anesth Analg 2004; 99: 1090-4
23. Adamus M, Hrabalek L, Wanek T, Gabrhelik T, Zapletalova J. Influence of age and gender on the pharmacodynamic parameters of rocuronium during total intravenous anesthesia. Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub. 2011; 155: 347-53
24. Schwartz AE, Matteo RS, Ornstein E, Halevy JD, Diaz J. Pharmacokinetics and pharmacodynamics of vecuronium in the obese surgical patient. Anesth Analg 1992; 74: 515-8
25. Meyhoff CS, Lund J, Jenstrup MT, et al. Should dosing of rocuronium in obese patients be based on ideal or corrected body weight? Anesth Analg 2009; 109: 787-92
26. van Kralingen S, van de Garde EM, Knibbe CA, Diepstraten J, Wiezer MJ, van Ramshorst B, van Dongen EP.Comparative evaluation of atracurium dosed on ideal body weight vs. total body weight in morbidly obese patients. Br J Clin Pharmacol 2011 Jan; 71(1): 34-40
27. Lemmens HJ, Brodsky JB. The dose of succinylcholine in morbid obesity. Anesth Analg 2006; 102: 438-42
28. Schnider TW, Minto CF, Shafer SL, Gambus PL, Andresen C, Goodale DB, Youngs EJ. The influence of age on propofol pharmacodynamics. Anesthesiology 1999; 90: 1502-16
29. Minto CF, Schnider TW, Shafer SL. Pharmacokinetics and pharmacodynamics of remifentanil. II. Model application. Anesthesiology 1997; 86: 24-33
30. Nickalls RW, Mapleson WW. Age-related iso-MAC charts for isoflurane, sevoflurane and desflurane in man. Br J Anaesth. 2003; 91: 170-4
31. de Almeida MC, Latorre F, Gervais HW, Kleeman PP. The effects of age on onset and recovery from atracurium, rocuronium and vecuronium blockade. Anaesthesist 1996; 45: 903-6

WHAT'S THE SOLUTION?

Dr Laurence Weinberg

Department of Anaesthesia,
Austin Hospital, Melbourne

Fluid management strategies are described in order to assist colleagues or other institutions who may be developing fluid management guidelines for the first time, or who are interested in different perspectives on perioperative fluid intervention. This is not intended to be a dogmatic approach to the topic, but rather to suggest a perioperative fluid replacement model to evaluate the effectiveness of care, improve clinician practices, and reduce the risk of fluid-related adverse events. In the absence of definitive guidelines, an approach to choice of fluid therapy based on patient factors, the type of surgery, and the type of fluid available is presented. Recently published recommendations for choice of fluid therapy and important clinical trials are examined.

When choosing an intravenous fluid for perioperative use, the following questions need be considered. Does the patient need fluid? How much fluid? What type of fluid- crystalloid or colloid? What type of crystalloid or colloid? When should it be given? What haemodynamic goal should we use to guide fluid delivery? The presentation focuses only on the specific choice of crystalloid or colloid, taking in to consideration the available evidence and each fluid unique physiochemical properties.

No fluid can improve patient-centred outcomes unless it is coupled to a treatment that improves outcome.[1] Thus, choice of fluid must be considered within the context of proven medical therapies, success of which is dependent on the clinical condition, pathophysiological state, and ability to reverse the identified disease process.

The composition and discriminate use of IV fluids should solely be dictated by the targeted fluid space. There appears to be no merit in differentiating between intraoperative, perioperative, postoperative and ICU settings.

Volume replacement should replace intravascular fluid losses and correct hypovolaemia to maintain haemodynamics and vital signs. Using a physiological solution that contains both colloid osmotic and oncotic components is reasonable. Fluid replacement, on the other hand, should compensate for an extracellular deficit as a result of cutaneous, enteral, or renal fluid loss. This is achieved with a physiological solution that contains all osmotically active components, ie an isotonic fluid. Electrolyte replacement or osmotherapy aims to restore a physiological total body fluid volume (intracellular fluid volume plus extra cellular fluid volume) when cutaneous, enteral, or renal fluid losses have altered the composition and / or volume of either or both fluid spaces (ICFV and / or ECFV).

Table 1 overviews the physiochemical properties of the available crystalloids

There is a growing body of evidence that normal saline (0.9%) should be avoided and replaced with balanced salt solutions. Normal saline results in hyperchloraemic acidosis and may have adverse effects on renal outcomes. Balanced electrolyte solutions containing K^+ can be used cautiously even in patients with AKI in preference to normal saline.[2] Currently there is not a single study showing normal saline to be superior to balanced solutions. Normal saline would not pass a single Phase 1 RCT, is unphysiological, and its use should be discouraged.[3] Even healthy human volunteers can take over two days to excrete a rapid infusion of 2 L of 0.9% saline.[4]

There is convincing animal data that demonstrates the adverse effects of chloride on the kidney. Hyperchloraemia has been shown to result in increases in inflammatory cytokines, increases renal afferent arteriole vasoconstriction[5] and adverse effects on renal oxygen consumption.[6] In human studies recently published research has also now demonstrated negative effects of chloride on renal blood flow in healthy volunteers.[7] In addition balanced solutions may reduce the incidence of major complications and kidney injury in patients undergoing major abdominal surgery when compared to normal saline.[8]

	Plasma	Plasmalyte	Hartmann's	Normal Saline
Sodium (mmol/L)	136 – 145	140	129	154
Potassium (mmol/L)	3.5 – 5.0	5.0	5.0	
Magnesium (mmol/L)	0.8 – 1.0	1.5		
Calcium (mmol/L)	2.2 – 2.6		2.5	
Chloride (mmol/L)	98 – 106	98	109	154
Acetate (mmol/L)		27		
Gluconate (mmol/L)		23		
Lactate (mmol/L)			29	
Osmolarity (mosmol/L)	290 – 310	295	274	308
pH	7.4	7.4	5.5 – 6.2	5.5 – 6.2
Strong ion difference (meq/L; effective)	42	49	29	0
Cost (AU$/L)		1.89	1.00	1.00

Table 1. Physiochemical properties of the commonly used crystalloids compared to plasma

The effective strong ion difference (SID) of each crystalloid will have significant effects on acid base homeostasis. Plasma has a normal SID of 42 meq/L. If a fluid with an effective SID lower than that of plasma is infused, this creates a metabolic acidosis by reducing plasma and extracellular SID. For example normal saline has an effective SID of zero. If large amounts of normal saline are infused, it will dilute the plasma SID forcing it towards that of normal saline. Hyperchloraemic acidosis is preventable if there is substitution of the organic anion Cl^- for HCO_3^-. The following anions are used as metabolisable bases in the available balanced crystalloids – lactate in Hartmann's solution, gluconate and acetate in Plasmalyte. The organic anions are strong anions and can be regarded as 'balanced,' provided they are metabolised rapidly after infusion. The effective strong ion differences of the crystalloids are summarised in Table 1.

Hartmann's solution contains 29 mmol/L of L-lactate (anion). Metabolism of L-lactate will generate HCO_3^- buffer. Importantly, lactic acid is an acid, but the lactate in Hartmann's is a base, metabolised by the liver at 100 mmol/hr (4 L/hr). The effective SID of Hartmann's solution is 29 meq/L. The lactate anion is the conjugate base of lactic acid and represents potential bicarbonate and not potential H^+. Hartmann's is useful for ECF and intravascular volume replacement. Severe acidaemia may result in inadequate hepatic metabolism therefore the production of HCO_3^- from the infused lactate may be impaired in this setting. If lactate cannot act as a HCO_3^- source, an iatrogenic hyperlactaemia may occur.

Problems with Hartmann's solution –

- Elevations in lactate if the lactate cannot be metabolised in the liver (eg liver disease / liver transplantation). This can create clinical problems if using lactate as a marker of resuscitation
- Diabetics – some evidence that lactate is converted to glucose causing hyperglycaemia[9]
- Hypotonic – 278 mosmol/L vs normal saline 300 mosmol/L (Na^+ of 129 mmol/l). Caution with cerebral oedema
- Oliguric hyperkalaemic renal failure
- Calcium – in US, AUS / NZ, cannot be co-administered with blood because it causes clotting in the IV line (not proven)
- O_2 consumption increases rapidly after lactate administration

Plasmalyte solution is another balanced solution that has substitution of organic anions for HCO_3^-. These organic anions are gluconate & acetate. Gluconate and acetate are metabolised by the liver, but also in extra-hepatic sources such as the muscle. Plasmalyte contains no lactate (will not cause iatrogenic hyperlactaemia) and no calcium (therefore can be used with blood without causing precipitation). Its effective SID is 49 meq/L, higher than plasma, and therefore corrects acidosis. It has a normal osmolality of 295 mosm/L, is isotonic, and has physiological amounts of Na^+, making it suitable in neurosurgery. Acetate is converted to bicarbonate in the liver and extra-hepatic tissue, is more rapidly converted than lactate, and has more alkalinising ability than lactate. It is worth noting that the supra-physiological concentrations of acetate buffer in some dialysate solutions may result in myocardial depression and hypoxia – therefore acetate has been abandoned as a buffer in dialysis in Australia. Gluconate is converted to bicarbonate in liver and extra-hepatic tissue and mostly (80%) eliminated unchanged in

urine. There is animal data showing that concentrations of 2.4 – 4.8 mmol/L are protection against post ischaemic myocardial dysfunction and oxidative injury.

Practice at Austin Hospital for crystalloid fluid intervention –

1. No role for normal saline
2. Hartmann's for the majority of cases
3. Plasmalyte for –
   - All major liver surgery including transplantation
   - Complex cardiac (aortic surgery, redo, double valve, etc)
   - Critically ill patients with existing metabolic acidosis

	Plasma	Gelofusine	Albumin (20%)	Albumin (4%)	Voluven	Volulyte
Sodium (mmol/L)	136 – 145	154	48-100	140	154	137
Potassium (mmol/L)	3.5 – 5.0					4
Magnesium (mmol/L)	0.8 – 1.0					
Calcium (mmol/L)	2.2 – 2.6					
Chloride (mmol/L)	98 – 106	120		128	154	110
Acetate (mmol/L)						34
Octanoate (mmol/L)			32	6.4		
Strong ion difference (meq/L; effective)	42	50	40	12	0	31
Cost (AU$/L)		~22			~17	~17

Table 2. Physiochemical properties of the commonly used colloids compared to plasma

A detailed overview of each of the colloids is beyond the scope of this presentation and excellently discussed in other review articles.[10] A brief summary of the physiochemical properties of each of the colloids is outlined in Table 2. Similarly to the crystalloids, an important component of the colloid is the chloride content and the effective SID. Except for Albumex 20, all colloids have a high chloride content and therefore the SIDs of the commercially available weak acid colloids are all greater than zero. There is thus a tendency for standard albumin and gelatin based colloids to cause a metabolic acidosis similar to saline. The new third generation tetrastarches contain no weak acids, and have an effective SID of zero. Their acid base effects are similar to saline. There is a growing move towards using balanced colloids, ie colloid solution in a balanced crystalloid carrier. Volulyte is one such solution.

References

1. Pinsky MR, Payen D. Functional hemodynamic monitoring. Crit Care 2005; 9: 566-72
2. Powell-Tuck J, Gosling P, Lobo DN, et al. British Consensus Guidelines on Intravenous Fluid Therapy for Adult Surgical Patients (GIFTASUP). London: NHS National Library of Health. http://www.ics.ac.uk/downloads/2008112340_GIFTASUP%20FINAL_31-10-08.pdf (accessed 11 January 2009)
3. Lobo DN. Intravenous 0.9% Saline and general surgical patients: A problem, not a solution. Annals of Surgery 2012; 255: 830-832
4. Drummer C, Gerzer R, Heer M, Molz B, Bie P, Schlossberger M, Stadaeger C, Röcker L, Strollo F, Heyduck B. Effects of an acute saline infusion on fluid and electrolyte metabolism in humans. Am J Physiol 1992; 262
5. Hansen PB, Jensen BL, Skott O. Chloride regulates afferent arteriolar contraction in response to depolarization. Hypertension 1998; 32:1066-70
6. Kellum JA, Song M, Almasri E. Hyperchloremic acidosis increases circulating inflammatory molecules in experimental sepsis. Chest 2006; 130: 962-7
7. Chowdhury AH, Cox EF, Francis ST, Lobo DN. A randomized, controlled, double-blind crossover study on the effects of 2-L infusions of 0.9% saline and plasma-lyte® 148 on renal blood flow velocity and renal cortical tissue perfusion in healthy volunteers. Ann Surg 2012; 256: 18-24

8. Shaw AD, Bagshaw SM, Goldstein SL, Scherer LA, Duan M, Schermer CR, Kellum JA. Major Complications, Mortality, and Resource Utilization After Open Abdominal Surgery: 0.9% Saline Compared to Plasma-Lyte. Annals of Surgery 2012. 255: 821-829
9. Thomas DJ, Alberti KG. Hyperglycaemic effects of Hartmann's solution during surgery in patients with maturity onset diabetes. Br J Anaesth 1978; 50: 185-188
10. Westphal M, James M et al. Hydroxyethyl starches. Anesthesiology 2009; 111: 187-202

ENHANCED RECOVERY AFTER SURGERY PROTOCOLS

Dr Matt Taylor

Middlemore Hospital
Auckland

What is ERAS?

Enhanced Recovery after Surgery (ERAS), also known as "fast track" surgery or "enhanced recovery protocol" (ERP) is a multimodal perioperative care pathway designed to decrease major morbidity,[1] and length of stay[2] together with promoting accelerated postoperative recovery and minimisation of postoperative fatigue.[3] It compiles a range of perioperative techniques aiming to promote early mobilisation and attenuate the surgical stress response. These include a variety of interventions from anaesthetic and surgical disciplines as well as physician, nursing and allied health.

Conception

Conceived by Dr Henrich Kelhet in the late 1990s, ERAS has amassed a considerable evidence base and undergone refinement over the past 15 years. His initial aim was to attenuate the body's response to surgical stress, typically characterised by an increase in cortisol, catecholamines, hormones promoting fluid retention, hypercoagulation and development of a catabolic state. It has been argued that this highly conserved response is unnecessary in the context of modern perioperative practice and may in fact be counterproductive in many of its aspects and is thought to contribute to postoperative morbidity.

Implementation of an ERAS protocol at Manukau Surgical Centre at CMDHB in 2005-2006 has seen a reduction in the average length of stay from a median of eight to four days (without an increase in readmission rate), a documented decrease in morbidity and cost savings (even when accounting for increased staff and setup costs). Dr Kehlet's group has refined this further, achieving ALOS of 2-3 days and even achieving <24 hour stay for colonic resections although three days appears to be a more realistic target.

Surgical Stress Response

The Surgical Stress Response refers to the hormonal and metabolic surgical changes seen after the body's exposure to a traumatic event. These serve to prepare the body in a "survival mode" during a period of starvation and healing.

The sympathetic nervous system is activated, and pituitary hormones are released,[4] resulting in the development of a hypermetabolic, catabolic state. Hyperglycaemia (from insulin resistance, gluconeogenesis and glycogenolysis) is seen, in addition to promotion of protein breakdown and lipolysis (mostly through cortisol). Sodium and water retention is enhanced due to release of vasopressin, and activation of the renin-angiotensin-aldosterone pathway. Increased cardiac demand, splanchnic vasoconstriction, immunosuppression and hypercoagulation are manifest.

A further, less discussed outcome of the surgical stress response is the development of postoperative fatigue, which impairs the patient's functional recovery over the days to weeks following discharge. These are aspects of postoperative outcome that are usually hidden from the anaesthetist and therefore not typically considered as part of anaesthetic outcome audit process, but never the less is impacted on by the ERAS protocol.[3]

Attenuation of the surgical stress response is mainly achieved by regional anaesthesia, minimally invasive surgery and pharmacological intervention (dexamethasone, beta blockers and anabolic agents) and these have a corresponding importance in ERAS protocols. Neural blockade with local anaesthesia reduces endocrine and metabolic activation and suppresses sympathetic stimulation, but has no effect on inflammatory responses.[5]

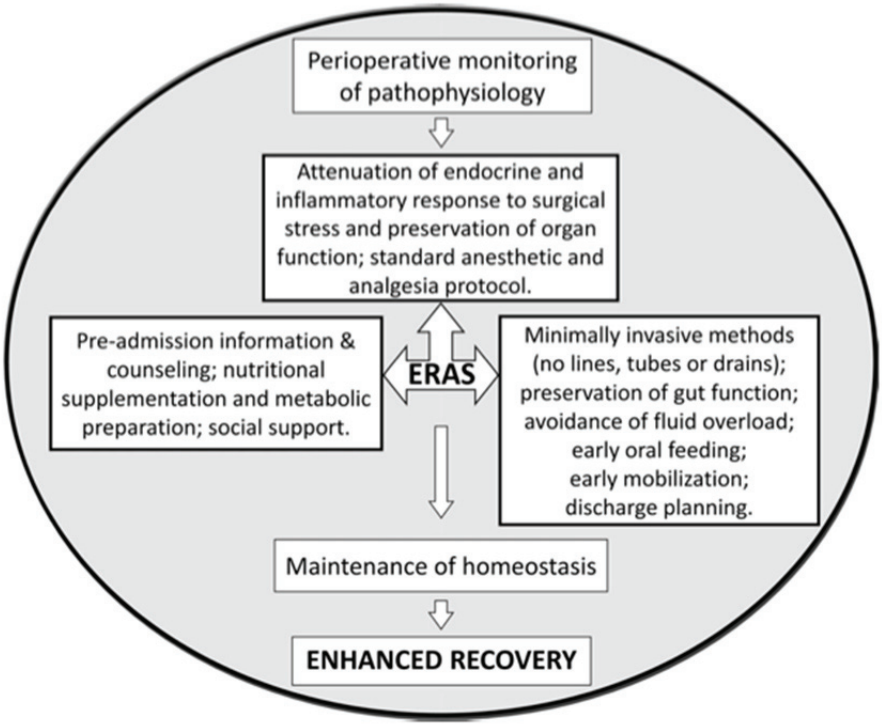
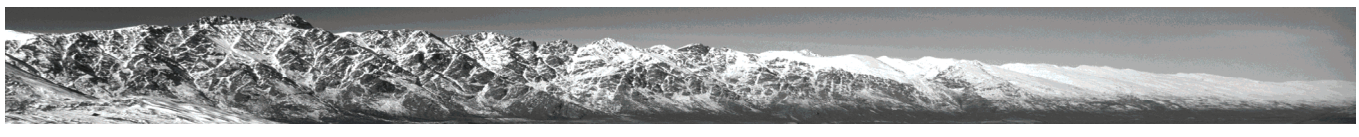


Figure 1. Philosophy of ERAS. (From Varadhan, 2010[6])

ERAS Protocols

ERAS protocols aim to achieve decreased morbidity and more rapid rehabilitation through an array of mechanisms.[7] The aims of these are, and mechanisms used to achieve this include –

1. Preoperative patient education
2. Surgical and anaesthetic techniques to reduce surgical stress response
   a. Epidural anaesthesia and analgesia
   b. Dexamethasone
   c. Thermal control
   d. Early nutrition
   e. Intraoperative fluid management
   f. Avoid overnight starvation / preoperative carbohydrate load
   g. Avoid bowel preparation (left sided cases get fleet enema)
   h. Minimally invasive surgical techniques
3. Aggressive postoperative rehabilitation
   a. Early mobilisation
   b. Short acting anaesthetic agents
      i. Avoid premedications (midazolam OK)
   c. Control of nausea, vomiting and ileus
      i. Antiemetic protocols
      ii. Opioid minimisation
4. Adherence to evidence based principles of perioperative care
   a. Avoidance of drains, nasogastrics
   b. Early withdrawal of urinary catheters



Colonic Resection Enhanced Recovery After Surgery (ERAS) Protocol for CMDHB

Timing	Intervention
Preadmission	Preoperative assessment in a dedicated outpatient session Programme information given, including specific daily milestones Social issues are identified and addressed Preoperative ward visit and orientation
Preop	Preoperative carbohydrate loading (PreOP). Four drinks day before surgery, and two drinks two hours before surgery Patients admitted to hospital on the morning of their surgery Left-sided operations receive a phosphate enema on arrival at the hospital Mechanical bowel preparation is avoided
Intraop	Thoracic epidural inserted and bupivacaine epidural infusion started Limited intraop intravenous fluids (1–2L crystalloids / colloids) Transverse incisions for right-sided open surgery if appropriate Prophylactic nasogastric tubes not used Intra-abdominal drains not used Calf stockings applied at the end of surgery
Recovery room	Vasopressor agents in preference to intravenous fluids to treat epidural-related hypotension Intravenous morphine / fentanyl PCA initiated
Day of surgery	Patients are mobilised to a chair Oral intake of fluids is started, aiming for > 800 ml of oral intake on the day of surgery Pre-emptive regular antiemetics (5-HT_3 antagonists as first line) Subcutanous low molecular weight heparin started for thrombo-prophylaxis
Day 1	Urinary catheter removed Full solid oral diet Resource supplement drinks (2–3 per day until discharge) Active mobilisation with nursing and physiotherapy input
Day 2	Epidural infusion is stopped, and epidural catheter removed Non opioid analgesia Oral opiates for break-through pain only
Day 3	Discharged home if fulfil following criteria – ▪ Tolerating full oral diet ▪ Passing flatus ▪ Adequate analgesia on oral medication ▪ Ambulating independently ▪ Satisfactory support at home
After discharge	Patient given a phone number for contacting the ward if required Nursing staff contact the patients three days after discharge for a phone interview Follow up outpatient clinic appointment within seven days of discharge

Elements With Anaesthetic Relevance

- Preoperative fasting and preoperative carbohydrate loading
  - Avoidance of overnight fasting, together with reduced use of bowel preparation (increases stress response and anastomotic leak rates) minimises patient hypovolaemia and electrolyte imbalance preoperatively. Carbohydrate loading prevents early formation of a catabolic state
- Premedication
  - Avoidance of premedication / use of short acting agents, designed to promote rapid postoperative mobilisation
- Prophylaxis against thromboembolism
- PONV prophylaxis
  - As part of PONV prophylaxis, dexamethasone is used to concurrently attenuate surgical stress response. The current evidence based dosage for this is 8mg[8] although lower doses may prove to be equally effective

- Preventing intraoperative hypothermia
  - Hypothermia may lead to augmented stress response during rewarming which increases cardiovascular demands and potential morbidity in at risk individuals, impairs coagulation and leucocyte function. Preservation of intraoperative and early postoperative normothermia has been shown to decrease surgical site infection, decrease intraoperative blood loss, postoperative cardiac morbidity and overall catabolism[9]

Elements Subject to Controversy

Urinary Drainage

In order to facilitate early mobilisation, for colonic resections, urinary catheters are removed on day one, prior to removal of the epidural (day 2). Despite initial concerns about the safety of this in context of retention, a CMDHB study showed no increase in retention compared with historical controls and in fact a significant decrease in renal complications due to a drop in urinary tract infections.[1]

Postoperative Analgesia / Prevention of Postoperative Ileus

The use of epidurals is one of the more controversial areas of ERAS management due to concerns about epidural morbidity, postoperative hypotension in fluid restricted patients and presumed reduction in benefit for laparoscopic cases. The current ERAS consensus evidence based guidelines advocates T7/8 epidural use as part of an ERAS protocol in order to attenuate surgical stress response and maintain gut motility through opioid minimisation and blockade of visceral sympathetic innervation.[9,10]

The place of laparoscopic surgery is currently not clear with several small trials showing improved analgesia and one showing improved bowel function, however a recent RCT[11] comparing 91 patients randomised into three arms of PCA, epidural or intrathecal morphine within an ERAS programme found epidural use worsened length of stay, bowel function and increased fluid administration. The use of transversus abdominus plane blocks is very unclear with little evidence for benefit beyond 12 hours in caesarian section. A recent Cochrane review combining TAP blocks and rectus sheath blocks for abdominal surgery found morphine sparing for the first 24 hours only, but with significant heterogeneity in the results.[12] Further studies on this topic are currently in progress.

Perioperative Fluid Management

Initial studies into fluid administration in colorectal surgery focused on limiting administration of crystalloid in an effort to promote postoperative bowel function. This was based on early work in Mecray in 1937 in animal studies. Lobo,[13] Brandstrup[14] and Nisanevich[15] all showed improved outcome with “restricted” intraoperative fluid administration. This became known as “fluid restriction” although on further review in subsequent years in the literature these studies are more correctly considered to be “neutral fluid balance” compared with “liberal” fluid.

The use of fluid regimes compared with traditional regimes was initially viewed with concern relating to increased morbidity. Historically, large volumes of crystalloid have been administered to compensate for preoperative starvation, mechanical bowel preparation, epidural preloading, abdominal evaporative losses and “3rd spacing.” It has become clear that these replacement fluids are not required in modern ERAS practice and in some case have been based on severely limited scientific rationale. ERAS patients have limited preoperative starvation, avoid bowel preparation and are supplied with preoperative carbohydrate fluids. As a result they are not volume deplete at commencement of surgery. Recent RCT evidence in obstetric anaesthetic literature has shown “preloading” for neuraxial anaesthesia does not prevent the requirement for vasopressor use. The need for replacement of abdominal evaporative losses by crystalloids or free water has been exaggerated previously and is insignificant.[16,17,18] Opinion on the “third space” described by Shires has shifted. Translocation of fluid into peritoneal and pleural spaces can occur but the magnitude of the third spacing detected and described cannot be replicated reliably and probably represents a measurement artifact.[16] Taken to excess, fluid restriction lower than neutral fluid balance is likely to result in adverse outcomes.

Goal directed therapy has emerged in a parallel line of research to be beneficial in colorectal surgery in terms of morbidity and length of stay outcomes. Its use has been advocated by the National Health Service UK National Institute of Clinical Excellence (NICE) and consideration of use by the ERAS group on a case by case basis. Its place within ERAS as a routine instrument of fluid management is currently unclear,[19] as is which “goal” to consider in differing populations such as colonic resections.

Non Colorectal ERAS Programmes

ERAS guidelines are emerging for areas outside of the original area of colorectal surgery. These differ form previous care pathways in their emphasis on suppression of surgical stress response and early mobilization. Whilst they are likely to translate to improvements in efficiency and patient outcomes, the evidence base in various areas need to be established. Similarly, the temptation to import all aspects of ERAS principles wholesale may not be appropriate in other surgical areas (eg-fluid restriction is unhelpful in daystay or laparoscopic cholecystectomy). Review of the pre-existing literature pertaining to the operative procedures planned and future research and audit is required in these areas.

References

1. Zargar-Shoshtari K, et al. Fast-track surgery may reduce complications following major colonic surgery. Dis Colon Rectum 2008; 51(11):1633-40
2. Kehlet H. Multimodal approach to control postoperative pathophysiology and rehabilitation. British Journal of Anaesthesia 1997; 78: 606-17
3. Zargar-Shoshtari K, et al. A Prospective Study on the Influence of a Fast-Track Program on Postoperative Fatigue and Functional Recovery After Major Colonic Surgery. J Surg Res 2009;154(2): 330-5
4. Desborough JP. The stress response to trauma and surgery. BJA 2000; 85(1):109-17
5. Holte K, Kehlet H. Epidural anaesthesia and analgesia: effects on surgical stress response and implications for postoperative nutrition. Clin Nitr 2002; 21: 199
6. Varadhan KK, et al. Enhanced Recovery After Surgery: The Future of Improving Surgical Care. Critical Care Clinics 2010; 26(3): 527-47
7. Kehlet H, Wilmore DW. Evidence-Based Surgical Care and the Evolution of Fast-Track Surgery. Ann Surg 2008; 248 (2):189-98
8. Zargar-Shoshtari K, et al. Randomized clinical trial of the effect of glucocorticoids on peritoneal inflammation and postoperative recovery after colectomy. BJS 2009; 96 (11): 1253-61
9. Lassen K, Soop M, et al. Consensus Review of Optimal Perioperative Care in Colorectal Surgery, ERAS group recommendations. Arch Surg 2009; 144(10): 961-9
10. Freise H, et al. BJA 2011; 107 (6): 859-68
11. Levy, BF et al. Randomized clinical trial of epidural, spinal or patient-controlled analgesia for patients undergoing laparoscopic colorectal surgery. BJS 2011; 98(8): 1068-78
12. Charlton SC, et al. Perioperative transversus abdominis plane (TAP) blocks for analgesia after abdominal surgery. Cochrane Database of Systematic Reviews 2010; 12: CD007705
13. Lobo DN, Bostock KA, Neal KR, et al. Effect of salt and water balance on recovery of gastrointestinal function after elective colonic resection: a randomised controlled trial. Lancet 2002; 359: 1812-8
14. Brandstrup B, et al. Effects of intravenous fluid restriction on postoperative complications: comparison of two perioperative fluid regimens: a randomized assessor-blinded multicenter trial. Annals of Surgery 2003; 238: 641-48
15. Nisanevich V, et al. Effect of intraoperative fluid management on outcome after intraabdominal surgery. Anesthesiology 2005; 103: 25-32
16. Brandstrup, B. Fluid therapy for the surgical patient. Best Prac Res Clin Anaesth 2006; 20(2): 265-83
17. Chappell, D. A Rational Approach to Perioperative Fluid Management. Anesthesiology 2008; 109: 723-40
18. Lamke, Acta Chir Scand 1977; 143: 279-84
19. Brandstrup B, et al. Which goal for fluid therapy during colorectal surgery is followed by the best outcome: near-maximal stroke volume or zero fluid balance? BJA 2012; advance access

LEARNING FROM ERRORS IN OBSTETRIC ANAESTHESIA – DESIGNING SYSTEMS TO IMPROVE PATIENT SAFETY

Clin A/Prof Nolan McDonnell

Department of Anaesthesia and Pain Medicine, King Edward Memorial Hospital for Women, Western Australia
School of Women's and Infants' Health, University of Western Australia, Crawley, Western Australia
School of Medicine and Pharmacology, University of Western Australia, Crawley, Western Australia

"To err is human, to forgive is divine." Alexander Pope wrote these now famous lines as part of a poem "An Essay on Criticism" in 1709. An error can be defined as *"the failure of planned actions to achieve their desired outcomes."* Central to the prevention and management of error in medicine and anaesthesia is to realise that we all make errors, not just occasionally, but every day. We are not perfect (as much as we may strive for perfection) and we function in systems that are often far from perfect and may actually increase our chances of making an error. Most of the time these errors are of little consequence but occasionally, what would otherwise be a minor error in another setting, may have devastating consequences for the patient, their family and the medical team. It is only when we appreciate that we are all susceptible to making errors that we can begin to look at ways in which we can prevent or mitigate the consequences of error in our own practice and start designing systems that are more "error proof."

James Reason, one of the pioneers of the "human factors approach" and who originally described the "Swiss Cheese" model of human error debunks a number of myths associated with human error. Appreciating these myths provides an insight into modern error management and what can be done to prevent errors from occurring. The first myth is that *errors are intrinsically bad*. It is not so much the error itself that is bad, but more the context in which the error occurs. For example, we may forget to switch something on at home when we are preparing dinner, an error that most of the time would be of little long-term consequence. This same error in the operating theatre could potentially have extremely serious consequences for the patient, eg if the volatile agent is not switched on after induction of anaesthesia or prophylactic antibiotics are not administered. Errors themselves are crucial to our learning to adapt to new or novel situations and in many ways errors and correct performance go hand in hand as we learn throughout life.

A second myth that is commonly portrayed is that *bad people make bad errors*. This myth is often deeply rooted in the perception that bad things happen to bad people and that the error is attributed to some characteristic of the person who made the error. In fact, the converse is often true. The best people are often performing the most difficult tasks and hence may be more liable to error. But, these same individuals can also adopt certain behaviors that are more likely to provoke errors, such as working when they are significantly fatigued. In "judging" an error, it is not so much the error itself which might be important but whether the behavior of the person involved made them more prone to making errors.

Another common myth related to errors is that *errors decline with practice* and that *they are random and highly variable*. Certainly, when an error is related to a lack of knowledge, then with more practice that error will decrease. This is one of the values of simulation training in medicine. But, much of the time spent in our working life will be spent with the mind functioning in more of an automatic mode, dealing with common situations, a procedure or a series of steps that are very well ingrained in memory. In these situations errors can result from slips or lapses and the chances of these types of errors increase the more we function in automatic mode. This is the price that is paid for freeing the conscious mind from needing to take "moment by moment" control of situations and allowing it to focus on other activities.

It is a commonly held belief that *highly trained people make fewer errors*. This myth has been debunked in a number of medical and aviation studies. What is apparent is that highly trained people are aware of their ability to make errors and they anticipate and practice the skills necessary to mitigate these errors – ie they are more effectively able to recognise an error and take immediate action to mitigate its consequences and recover from the error.

A further myth is that *a single error is normally sufficient to cause a bad outcome*. While on a superficial level this may seem true, in most systems there are a number of defensive layers built into that system to protect against

the error. Often it is the person on the front line, at the "sharp" end of medical care, that completes the error sequence (and is often blamed for the error), but the system itself contains "latent" failures and weak defensive processes such that allow the holes in the "Swiss cheese" model to line up and create the adverse outcome.

The final myth that James Reason describes is that in human error is *that it is easier to change people rather than situations*. Organisations often devote a considerable amount of their resources into targeting the individual behavior as a means to reduce error. This only targets one part of the error cascade. Human error is secondary to the complex interplay between humans and the situations that they are working in and the latent conditions that may exist within the systems. Human error is only avoidable up to a certain point, and it will still occur. That is the nature of being human. But, the situation within which that error occurs can be changed to trap or mitigate the error so that the consequences of the error are negated. This can influence all the people that interact with the system in the future.

So, given what we now know about the myths associated with human error and medicine, what can be done to either prevent error occurring or to limit the impact of such errors? Firstly, it is important to acknowledge that human error is part of being human and it will always occur, even in the most highly regarded and highly functioning individuals. We need to have the confidence to freely report errors. If managers and staff do not know about errors that occur then it becomes very difficult to design systems to limit the impact of such errors. Part of this is changing the attitudes of staff and patients to errors, we need to move away from the view that good doctors and good staff do not make mistakes, and realise that everyone is susceptible to errors and train people to detect errors and detect situations in which errors may occur more commonly. We need to avoid the naming, blaming and shaming reactions to errors when they occur – this inhibits the future reporting of errors and also places too much emphasis on the individual, rather than the systems contribution to the error.

Finally, the systems that the individuals are working in need to be designed in such a way so as to lessen the chance that an error may occur. In the example used in this talk, having two identical infusion pumps for magnesium sulphate and syntocinon made it inevitable that there would be an error in programming one of the pumps at some stage. Using different types of pumps for the different solutions can lessen the chance of this error occurring. In addition, the systems should be designed so that should an error occur, it can be rapidly detected before harm occurs or the effects of the error are minimised. With the magnesium example, by limiting the amount of magnesium that is contained in the infusion solution, even if a programming error occurs then the harm to the patient will be minimal. Lastly, staff should be trained in how to deal with potential errors in the systems they use. Completing the worked magnesium example, staff using this solution need to know what steps to take in the event of acute magnesium toxicity.

Additional Reading

Reason J. Seven myths about human error and its management. (Originally published in Italian as: Come limitare l'errore. KOS: Rivista di Nedicina, Cultura e Scienze Umane. 187:10-17, 2001.)

A BUG'S LIFE – SO SIMPLE, AND YET SO FASCINATING TOO

Dr Ben Harris

Medical Laboratory Scientist,
Southern Community Laboratories

The Basics are the Most Important – They are Easy!

Hand Hygiene

Hand hygiene is the single most important way to prevent the spread of bugs. Always think of your hands as contaminated until you have just washed or alcohol hand rub cleansed them. Cleanse before and after touching patients or their environs, after going to the toilet, before preparing food and refrain from touching your face, mouth or nose – there is a risk to you (viruses going into your mucosal surface) and a risk to patients (eg influenza virus shed a day before your symptoms begin). Commonly touched surfaces pose risks, eg knobs on anaesthetic apparatus, tubing, handled packages, bed rails, door handles, keypads, tap handles, pens, reception desks, waiting room chair arms and magazines, may all carry potentially harmful bugs. Normal skin carries 1,000 to 10,000 bacteria per square cm, 30% more under rings. Faeces contains a million million bacteria per gram, but 1,000 times more if virus is present (eg Norovirus). We only need to ingest 3-10 Norovirus to catch infection, or pass it on. It is a 'sticky' virus, harder to wash off than most, and partially resistant to alcohol rubs. Clostridium difficile is resistant to alcohol hand rubs (because of spores). Most other bugs are susceptible to alcohol disinfection. Contact precautions (gloves, gowns) should be used for direct or indirect contact of any biological fluids, or skin contact especially is the presence of known or likely MDRO (eg MRSA, ESBL, VRE).

Coughing

Coughing is the next most important way to spread bugs, especially colds, flu and other respiratory viruses. So cover your cough – cough into an elbow or tissue. Don't cough into and contaminate your clean hands with millions of viruses! Stay at home if you are ill. Wear a mask to keep cough droplets containing bugs in, or out, when appropriate. Treat masks as extremely infectious once worn – they are there to filter, and so concentrate any bugs in the air onto the mask surface. Use droplet precautions (mask, facemask, gown) for any respiratory virus, aerosol producing procedures, Bordetella, N. meningitides, or outbreak causing infections. Use airborne precautions (full PPE) for TB, measles, chicken pox, the immunecompromised.

Environment

Bugs generally require moisture, warmth and nutrients to survive and multiply. Most bugs die in the dry in a few hours to days – respiratory viruses generally within minutes, bacteria generally within hours to days. Walls floors and ceilings are not usually the risk (not wet) – it is us and our habits that are highest risk.

Good Bugs vs Bad Bugs?

Large numbers of almost any bugs can cause infections if in the wrong place. Think of the bugs in fresh pasteurised milk – harmless to drink and they probably help boost our immune system each time we drink (like when we breathe). However if the same bugs are present in large numbers as when the milk ages or 'goes off' they become nasty pathogens. Vulnerable people / patients are infected / affected by lower numbers of bugs. Low numbers of any bugs on compromised tissue or any foreign material (eg IV lines, prosthetic devices) rapidly form biofilms which are impenetrable to natural immunity and antibiotics, and become seats for infection.

We Need to Control the Numbers and Stop the Spread of Bugs

We live in a microbial world, not a sterile world. We have to learn to understand and manage the microbes or bugs / germs in and on us, and in our environment, to our best advantage. Hand washing does not sterilise our hands but if done thoroughly it removes any relatively newly arrived potential pathogens – the transient bacteria. The resident bacteria remain to help protect us (about 1,000 to 10,000 per cm^2) but like milk bottle bugs can cause problems if too many are in the wrong (wet) place, eg IV lines, wounds, dermatitis, food.

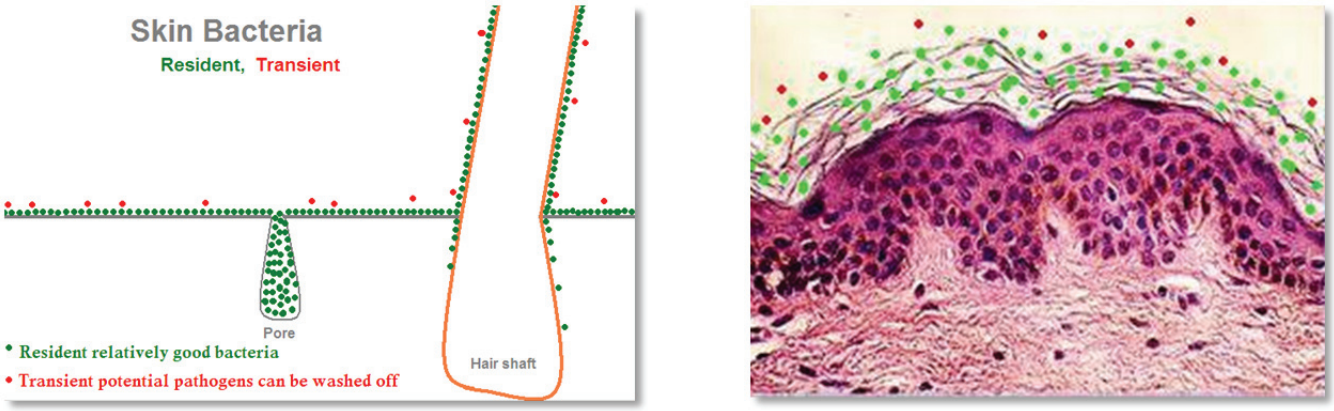


Figure 1. Normal skin cross section with 'bugs' added ●●●●

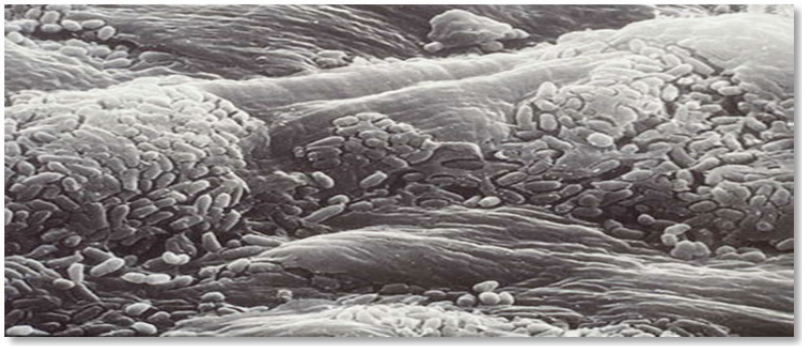


Figure 2. Skin surface with resident bacteria

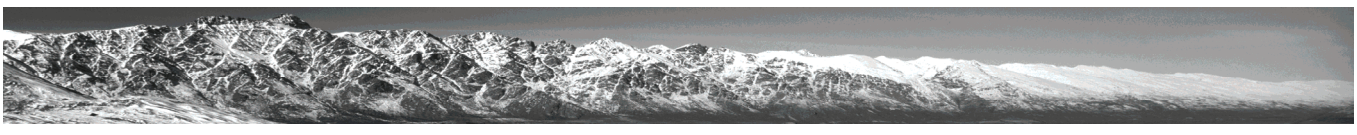
Infection prevention, and when this fails, infection control, are based on simple, common sense principles. These principles are based on science, written in procedures and protocols for us to implement. But a combination of our lack of knowledge or understanding combined with our habits of a lifetime overlaid by our more complex attitudes, behaviours and workplace culture can serve to make the implementation of these simple principles an on-going challenge.

We risk making our understanding of infection prevention more complex than we need to, perhaps similar to Churchill's comment on Russia –

"It is a riddle, wrapped in a mystery, inside an enigma; but perhaps there is a key. That key is Russian national interest"

And ours is good health by good hygiene practices, for all of our sakes.

It is likely at least 70% of cross infections are relatively easily preventable – yet even common colds, influenza, norovirus and food poisonings are on-going reminders of how much and far our individual and combined behaviours fall short. Good hand hygiene and respiratory etiquette are top of the list. We have improvements to make – and to do this we need to better understand ourselves and others before we can effect these changes.



We generally know what to do, but we collectively need to change our current ways or habits to make sure we do it, or do it better than we are now.

Bugs are fascinating (except perhaps their names!) – very simple in some ways and in their requirements to survive (moisture, nutrients, warmth), but remarkably adaptable and also complex. We know a great deal about them but there are endless opportunities to discover more – their rapid reproduction allows rapid evolution from small genetic changes, their multiple abilities to resist antibiotics, their ability to form impenetrable biofilms, swap genetic material and antibiotic resistance secrets with their own and other species, and their ability to communicate amongst themselves multi lingually by chemical language 'quorum sensing' for their common greater good, and their altruism, can all be easily overlooked.

How Do We Catch Bugs?

We and patients catch most bacterial infections from ourselves (endogenous), a facilitated emergence of the normal flora adjacent to where the integrity of our tissues has been compromised (surgery, wounds) and / or foreign body material is present (IV lines, dead tissue). Staphylococcus aureus from our skin, anaerobes (eg Clostridium difficile), coliforms and streptococci from our GI tract.

Another source are the hands / habits of medical staff, and commonly touched / contaminated surfaces. Our skin is like a lawn with grass on it (cf Staph epidermidis), but with some weeds present (Staph aureus). Tear the surface of lawn turf and the nearby weed fills the gap, similarly Staph aureus in wounds.

The central line associated bacteraemia (CLAB) programme is essentially to prevent the emergence of the above conditions – prevent emergence of low numbers of S aureus (recognised pathogen), or even high number of usually low pathogenicity Staph epidermidis – usually normal flora but in high numbers when facilitated by biofilms along a foreign body (IV line) can gain entry to the bloodstream.

Other bugs are not usually in or on us (exogenous) – viruses, many food poisoning bugs, some bacteria (eg TB or Cl difficile spores in the theatre / ward environment).

Antibiotic Resistant Organisms – How to Prevent Their Development and Spread

The WHO has identified emerging antibiotic resistance as one of the three major challenges facing mankind (along with food and water). Antibiotics are believed to give us on average an extra 10 years of life.

How do bacteria become resistant – we selectively breed them for resistance!

Every time we use an antibiotic (from this amazing, but limited resource) we kill the susceptible bacteria only (both the intended pathogens but also the unintended 'good' bacteria) but not those bacteria that are resistant – these then multiply, swap the genetic material conferring resistance, and keep emerging wherever and whenever we use antibiotics most, ie hospitals, ICUs, LTCFs. Overall the bacteria at these sites, both on our patients and us (we share the same bugs) become increasingly resistant. Hence the emergence of MRSA, ESBL. VRE, etc.

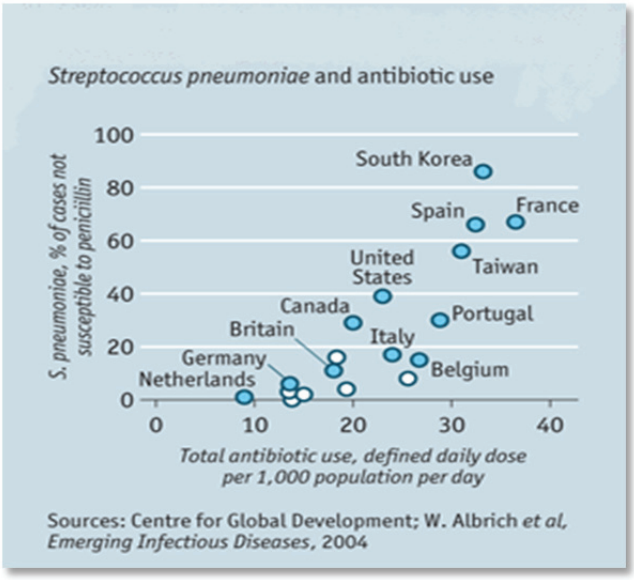
Routine practices including good hand sanitation with contact precautions are important strategies in preventing the spread of antibiotic-resistant organisms. However judicious antibiotic use is a critical measure to prevent their development in the first place. All antibiotic management should be evidence based and we should strongly resist the temptation to succumb to patient or clinician pressure to use them when they are not beneficial and are in fact detrimental to not only their but also the community's future health.

The sooner we use up this limited, valuable resource of antibiotics the sooner we will lose it.

'No action today, no cure tomorrow' – WHO antibiotic reduction catchphrase.

MRSA is primarily found in the nose and on the skin and can cause associated site infections. The primary reservoir of ESBL and VRE is the bowel and can cause UTI and wound infections.

A)



B)

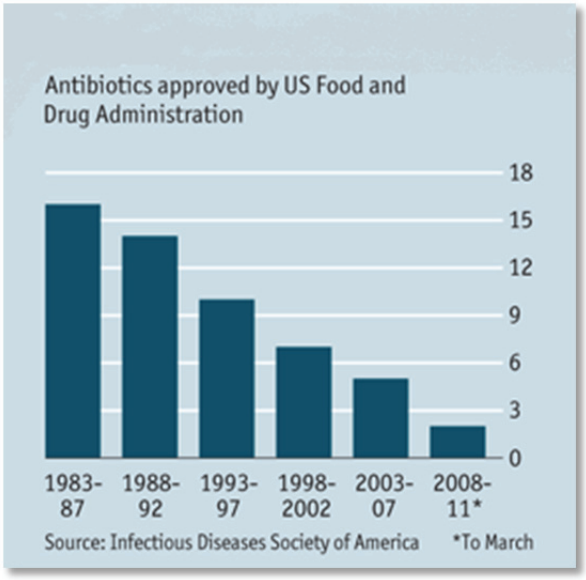


Figure 3. A) The link between antibiotic usage and resistance. B) New antibiotics being developed

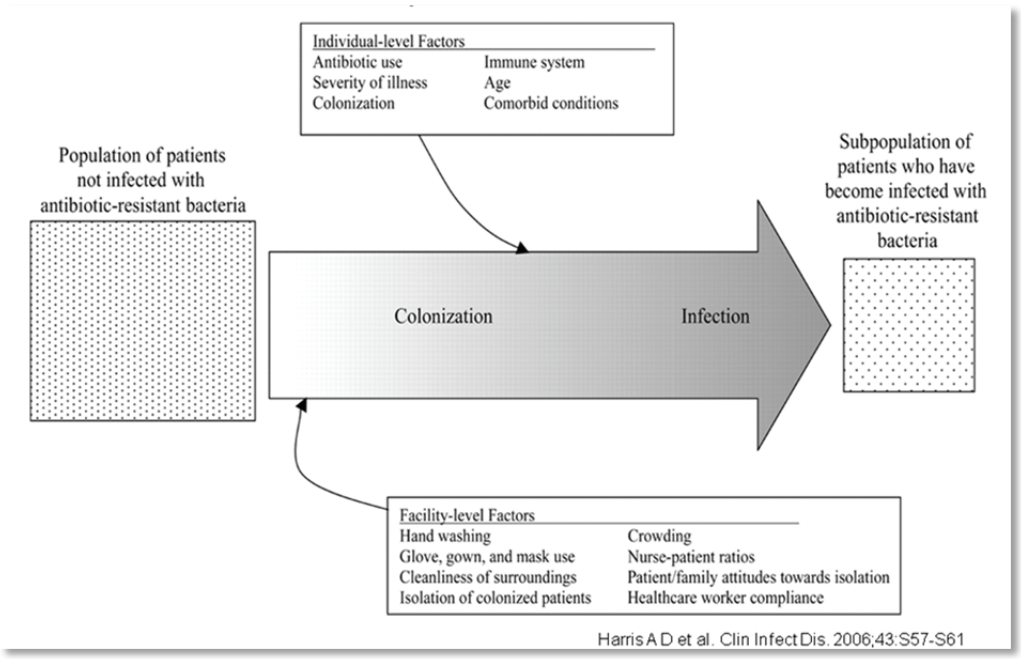
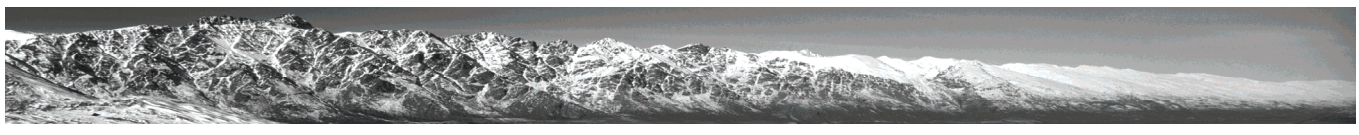


Figure 4. Factors that influence the acquisition of antibiotic-resistant bacterial infections

Total antibiotics used –

- 30% for human use
  - of which almost 100% is for treatment, 80% community, 20% hospitals (concentrated use)
- 70% for
  - Agriculture – caged poultry, pigs, plus Northern Hemisphere winter housed animals
    - 80% prophylaxis (added to routine feed), 20% for infection treatment
  - Horticulture – fruit sprays, kiwifruit PSA, etc
  - Aquaculture – fish farms, shrimp farms – routinely added to water

New Zealand is a low use antibiotic country, but still used 80 tons of antibiotics for non-medical use in the year 2000, the last year for which data is available. We import food from around the world, sell food (resistance) in supermarkets, travel widely and have many tourists – all sharing bugs and resistance liberally.



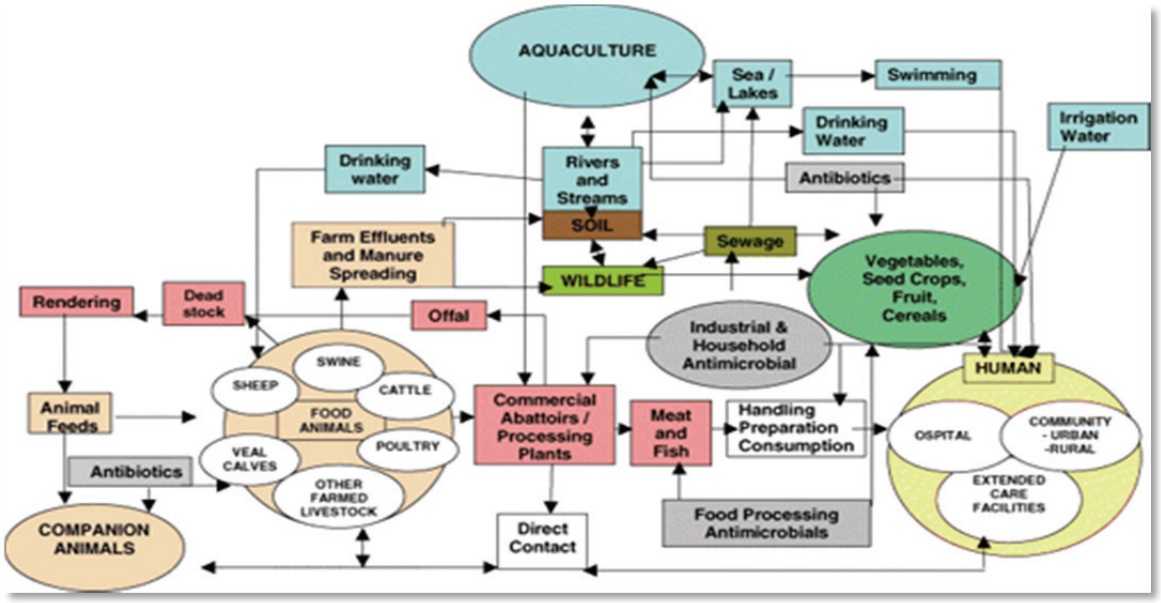
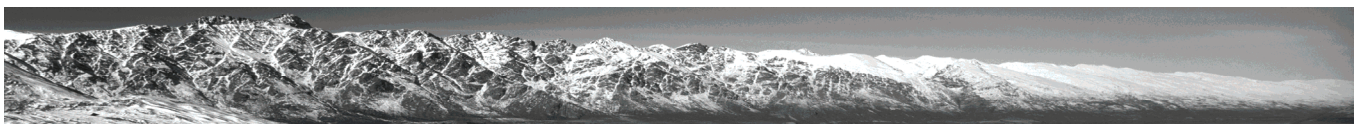


Figure 5. The spread of microbes

Infection Control References

- A-Z of health topics for the profession and patients – www.cdc.gov
- Profession resource – www.shea-online.org/guidelinesresources/guidelines
- Patient flyers for MRSA, VRE, Cl difficile, etc - www.shea-online.org/forpatients
- www.infectioncontroltoday.com



MERCENARIES, MISFITS AND MEDICS – HELPING OUT OVERSEAS

Dr Wayne Morriss

Christchurch Hospital
Christchurch

The title of this talk comes from the idea that overseas development workers are mercenaries, misfits or missionaries – the so-called "3 Ms." Medics don't always fit easily into this classification, but we often have the knowledge and skills that allow us to make a very significant contribution in resource-poor countries.

The "Overseas Problem"

It's easy to forget that about three-quarters of the world's population lives in poorer countries (low and middle income countries, LMIC). There is a huge disparity in per capita health spending between rich and poor countries, ranging from almost $7,000 in the United States to $8 in Sierra Leone (2005 figures).

When we look at surgical statistics, there are two main problems in developing countries – poor access to surgery and high morbidity and mortality rates.

An estimated 234 million surgical operations are performed globally each year.[1] Fifty-nine per cent of these operations are performed in rich countries, even though these countries make up only 16% of the world's population. At the other end of the scale, a tiny 3.5% of the world's operations are performed in the poorest countries. These countries account for 35% of the global population. We may be doing too many operations in high income countries, but it seems clear that many people are missing out on necessary surgery in low income countries.

Unsafe surgery is now recognised by the World Health Organisation as a major public health issue.[2] It is estimated that surgery causes over one million deaths and seven million disabling complications globally per year. In comparison, there are approximately half a million maternal deaths per year, 0.9 million deaths due to malaria and 2.0 million deaths due to HIV / AIDS.

In LMIC, unsafe anaesthesia is a major contributor to surgical death and disability. Statistics are usually unavailable, but one study in Togo, West Africa showed an avoidable anaesthetic mortality rate of 1 in 133.[3] In contrast, Australian figures for the 2003-2005 triennium showed an anaesthesia-related death rate of 1 in 55,000.

Walker and Wilson identified a number of issues associated with increased anaesthetic mortality – not enough medical anaesthetists, inadequate training and supervision of non-medical anaesthetists, limited monitoring, inadequate supplies of drugs, inadequate equipment, and lack of blood for transfusion.[4]

But What About The Pacific?

There are thirteen independent countries in the Pacific. While per capita income may be greater than many parts of Africa and Asia, health statistics are often very poor. Severe staff shortages are common and the region suffers from many problems related to communication and transport difficulties.

Papua New Guinea has a population of over six million people spread over an area 1.7 times that of New Zealand, but there are only 15 medical anaesthetists for the entire country, most based in Port Moresby.

In the latest global maternal mortality ratio statistics, the rate in Fiji is over 10 times the rate in New Zealand and Australia. The rate in PNG is about 40 times the rate in New Zealand and worse than many countries in sub-Saharan Africa.[5]

Is It All Too Hard?

The problem with presenting global statistics is that they are overwhelming and it's difficult to know where to start. But there are many ways we can help, and our efforts can be rewarded by real change.

Help can take many forms – clinical or teaching work, short term visits or long term stays, small or large projects, or supporting others to work overseas. To quote Peter Benenson, the founder of Amnesty International, it is "better to light a candle than curse the darkness."

Getting Started in Fiji

In 1999, I was offered a job in Fiji at the same time as a consultant anaesthetist job in Christchurch. I decided to take the Fiji job – a two year position as a Senior Lecturer in Anaesthesia and Physiology at the Fiji School of Medicine in Suva – and my family and I moved there in April 2000.

We expected to face many challenges during our time in Fiji, but we did not expect a coup! Five weeks after we arrived, on 19 May 2000, a small group of soldiers led by George Speight took over the parliament buildings and held members of the government hostage for 56 days. The seven months following the coup were very stressful, with a declaration of martial law, frequent episodes of civil unrest, and a rise in racism and religious intolerance.

The work was very challenging, both at the medical school and the Colonial War Memorial Hospital, Suva's main hospital. There were 17 coup-related deaths and hundreds of casualties. Limited resources were stretched even thinner, with severe drug shortages and loss of staff. One year after the coup, over thirty percent of doctors had left Fiji.

While not always comfortable, our time in Fiji was a hugely significant learning experience from a personal and professional point of view. Fiji shares many of the problems of other resource-poor counties and these problems were brought into sharp relief by the coup.

Education, Education, Education!

Inadequate training and not enough anaesthetists are common problems in LMIC. Good education is vital if we are to address these problems.

The World Federation of Societies of Anaesthesiologists (WFSA) plays a very important role in global anaesthetic education. It has a membership of over 120 anaesthetic societies, representing about 140 countries.[6] The WFSA itself has very limited resources but has an excellent track record of leveraging other funding. A recent analysis showed that, for every dollar spent by the WFSA, an extra $6.80 was contributed by volunteers and other organisations.

I have recently taken over as Chair of the WFSA Education Committee. We are a group of eight with members from Russia, Serbia, Kenya, Malaysia, the United States, Venezuela, Brazil and New Zealand. Over the next four years, we will be working hard to support numerous educational activities around the world, including training programmes in South America, Asia, Africa and the Middle East. Other committee activities include support for regional meetings and short courses in less affluent countries.

The most successful WFSA training programme is the Bangkok Anaesthetic Regional Training Centre (BARTC), based at Siriraj Hospital in Bangkok, Thailand. During the last 16 years, the programme has trained 60 anaesthetists from poorer countries in Asia. Many graduates of the programme have gone on to be the anaesthetic leaders and teachers in their own countries.

The Essential Pain Management (EPM) course was first run in Papua New Guinea in 2010, and it has now been run in Fiji, Solomon Islands, Vanuatu, Cook Islands, Micronesia, Tonga, Vietnam, Mongolia, Tanzania, Rwanda, Kenya and, most recently, Honduras.[7] It is an interactive one-day course that addresses local pain management barriers and uses a series of case discussions to illustrate the use of a simple framework for managing pain patients. Very importantly, there is also a half-day instructor course so that local instructors take over the teaching of the course within a few days. This encourages relevance and is vital for the local sustainability of the

programme. It's easy to underestimate the problems associated with providing even simple pain relief when resources are short. Pain is often not seen as a priority, there may be severe staff shortages, and essential drugs, such as oral morphine or even paracetamol, may not be available. EPM attempts to address these problems.

Short term surgical work can result in education at many levels. I recently went on a general surgery trip to a remote village called Mangelsen in western Nepal. The trip was very rewarding – I got to work with a great team, the surgery and anaesthesia were fascinating, and we were able to do some good for individual patients. For me, the trip was an example of "two-way education." I learned many useful things and I hope I was also able to impart some useful knowledge to the local health workers. One of the local nurses, Suresh, is the sole anaesthetic provider in the area and is often called on to do spinal anaesthetics for Caesarean sections. He was thirsty for knowledge and I hope that our time together will result in some real improvement in health care in this small corner of the world.

Getting Involved

There are many ways to get involved, some easier than others. The following are a few suggestions for getting started –

- Most people do not want to start by going on a six-month mission to a war zone with Medicin San Frontier. A gentler introduction would be to attend a meeting or conference in a LMIC and to meet some of the locals
- There are several courses that have been developed to give people the knowledge and skills to work in a resource-poor country. Consider attending the Real World Anaesthesia Course (held in Australia and New Zealand) or the Anaesthesia in Developing Countries course (run from the UK and held in Uganda)
- The NZSA Overseas Aid Committee and ASA Overseas Development and Education Committee both maintain databases of people interested in working overseas. Joining one of these databases can be a good way of making contacts and finding out about locum opportunities and longer term work
- There are a number of established programmes like the RACS Pacific Island Project and Interplast that organise surgical and educational trips to the Pacific and other regions. It may not be possible to join one of these teams without prior experience, but consider volunteering as an extra pair of hands

Conclusion

There is now greater awareness of the importance of safe anaesthesia in resource-poor countries. As anaesthetists, we have can help in many ways and our efforts can be rewarded with real change.

References

1. Weiser TG, et al. An estimation of the global volume of surgery: a modelling strategy based on available data. Lancet 2008; 372: 139-44
2. www.who.int/patientsafety/safesurgery/en/
3. Ouro Bang'na AF, et al. Deaths associated with anaesthesia in Togo, West Africa. Trop Doct 2005; 35: 220-2
4. Walker I, Wilson I. Anaesthesia in developing countries – a risk for patients. Lancet 2008; 371: 968-9
5. Hogan et al. Maternal mortality for 181 countries, 1980-2008: a systematic analysis of progress towards Millennium Development Goal 5. Lancet 2010; 375: 1609-23
6. www.anaesthesiologists.org/
7. www.anzca.edu.au/fpm/fellows/essential-pain-management

NOTES

SPONSORS

GOLD

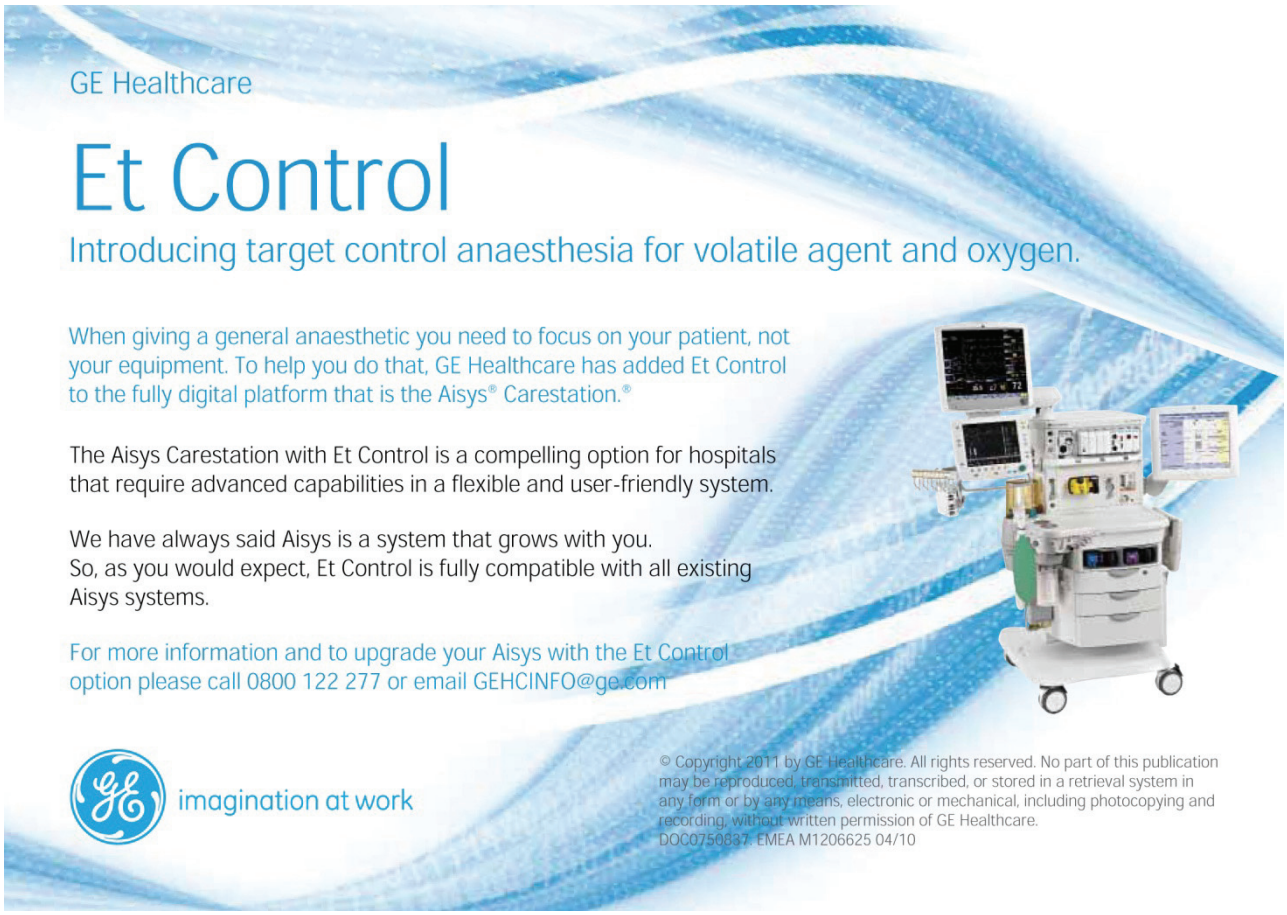
GE Healthcare

Preston Gash

Cell +64 21 476 080
Email preston.gash@ge.com

GE Healthcare provides transformational medical technologies and services that are shaping a new age of patient care. Our broad expertise in medical imaging and information technologies, medical diagnostics, patient monitoring systems, drug discovery, biopharmaceutical manufacturing technologies, performance improvement and performance solutions services help our customers to deliver better care to more people around the world at a lower cost. In addition, we partner with healthcare leaders, striving to leverage the global policy change necessary to implement a successful shift to sustainable healthcare systems.

Our "healthymagination" vision for the future invites the world to join us on our journey as we continuously develop innovations focused on reducing costs, increasing access and improving quality and efficiency around the world. Headquartered in the United Kingdom, GE Healthcare is a $16 billion unit of General Electric Company (NYSE: GE). Worldwide, GE Healthcare employs more than 46,000 people committed to serving healthcare professionals and their patients in more than 100 countries. For more information about GE Healthcare, visit our website at **www.gehealthcare.com**.



SILVER

Baxter

At Baxter, one of our primary goals is to advance the best in health care worldwide. We focus on assisting people with some of the most complex conditions – from haemophilia to cancer and immune disorders to kidney disease. Together, our caring hands help health care professionals enhance the care they provide their patients.

Making a Meaningful Difference in Patients' Lives.

www.baxter.co.nz

Grenville Ferreira
National Manager

Anaesthesia & Critical Care, NZ
Baxter Healthcare Ltd

DDI +64 9 574 2393
Cell +64 21 342 940
Email grenville_ferreira@baxter.com

SILVER

B|BRAUN
SHARING EXPERTISE

B. Braun is the largest privately owned medical device company globally. B. Braun has a growing product portfolio with extensive focus in the area of needle-free safety systems and Regional Anaesthesia products and most recently new and exciting developments in the area of Automated infusion Systems. At B. Braun we aim to preserve, acquire and expand knowledge for the Healthcare market and share it through dialogue with our partners. Our guiding principle "Sharing Expertise" rests on three pillars namely: Innovation, Efficiency and Sustainability.

www.bbraun.co.nz

Grant Roberts
National Sales Manager

B. Braun New Zealand
PO Box 62-027
Mt Wellington, Auckland

Cell +64 21 784 724
Email grant.roberts@bbraun.com

SILVER

Dräger

Dräger is an international leader in the fields of medical and safety technology.

The medical division product range includes anaesthesia workstations, ventilators for emergency and critical care, warming therapy for premature infants, patient monitoring, IT solutions, accessories and consumables, ceiling supply units and lights complete the portfolio.

www.draeger.com

Graham Singer

Cell +64 27 603 0599
Email graham.singer@draeger.com

SILVER

Sandy Scott
Critical Care Specialist NZ

Edwards Lifesciences
PO Box 30 4006
Hauraki Corner
Auckland

Cell +64 21 2744 810
Email sandy_scott@edwards.com

Edwards Lifesciences is the global leader in the science of heart valves and haemodynamic monitoring, with more than five decades of experience in partnering with clinicians to develop lifesaving innovations.

Edwards Lifesciences have continually been at the forefront of product development to assist clinicians to strive for better patient outcomes.

www.edwards.com

SILVER

Candice M Dobb
Key Account Manager NZ

Fresenius Kabi (NZ) Ltd
60 Pavilion Drive, Airport Oaks 2022
Auckland

Cell +64 21 494 002
Email candice.dobb@fresenius-kabi.com

Caring for life.

Fresenius Kabi Australia Pty Limited is one of Australia's and New Zealand fastest growing and innovative pharmaceutical companies, dedicated to improving the quality of life of patients in Australian and New Zealand hospitals by creating a new level of expertise and care.

Fresenius Kabi New Zealand specialises in these key areas -

- Infusion Technology
- Transfusion Technology
- IV Drugs
- Volume Therapy
- Parenteral Nutrition
- Gastroenterology

www.fresenius-kabi.co.nz

SILVER

John Reidy

Cell +64 274 764 009

The innovative GlideScope® Video Laryngoscope is designed to offer a consistently clear view of the airway, enabling quick intubation. Easy to learn, use and teach, GlideScope® covers all patient sizes from pre-term to morbidly obese and is available in both single use and reusable system configurations.

Visit us at **www.glidescope.com** or booth #4 at AQUA 2012 for more information.

SILVER

InterMed Medical Limited is a privately owned New Zealand distributor which proudly celebrated 30 years of sales into the New Zealand medical device market in 2010. InterMed represents well known agencies such as Arrow, Arizant International and Intersurgical. We look forward to seeing you during AQUA.

www.intermed.co.nz

Tori Walker
Anaesthesia Business Leader

InterMed Medical Limited
71 Apollo Drive, Albany 0132, Auckland

Tel +64 9 918 5351
Cell +64 21 376 771
Email Tori.Walker@intermed.co.nz

SILVER

SonoSite, Inc. is the innovator and world leader in bedside and point-of-care ultrasound and an industry leader in ultra high-frequency micro-ultrasound technology and impedance cardiography equipment.

Headquartered near Seattle, the company is represented by ten subsidiaries and a global distribution network in over 100 countries. SonoSite's small, lightweight systems are expanding the use of ultrasound across the clinical spectrum by cost-effectively bringing high-performance ultrasound to the point of patient care.

www.sonosite.com

Deb Stanley
Clinical Sales Specialist – NZ

SonoSite Australasia Pty Ltd

Cell +64 21 664 985
Email deb.stanley@sonosite.com

Tel: 0800 888 204 (toll free NZ)
1300 663 516 (toll free Aus)

MedRecruit BBQ at Coronet Peak

www.medrecruit.com

Mike Voyce
Marketing Manager

MedRecruit
P O Box 2064
Wakatipu 9349

Cell +64 21 314 719
Email mike@medrecruit.com

www.aqua.ac.nz