Saturday, 24 March 2018

School of Medicine
The University of Auckland
New Zealand

Programme and Abstracts

Table of Contents

Welcome

Dear Colleague,

Welcome to the Auckland City Symposium for 2018: Our theme this year is 'Risky Business – How understanding risk affects decision-making in surgical patients'. As anaesthetists we are involved on a daily basis in assessing risk and making difficult treatment decisions in conjunction with other specialists and our patients. Patients are becoming older and more co-morbid and undergoing procedures that are more complex and physiologically challenging. Negotiating risk is part of the problem to be sure that the patient is making an appropriate decision.

To help us consider these issues and advance our knowledge in this important subject, we have invited speakers from various areas of expertise. Professor Hilary Grocott, Professor Kate Leslie and Professor Bernhard Riedel will be supported by local speakers to complete a programme which will include up to date thinking on many of the relevant issues. We will start by presenting the relevant background and science of risk and move on through the day discussing clinical and non-clinical concepts involved in the risk assessment, communication and the decision making process. Finally, we will present and discuss some high-risk clinical scenarios to test our new understanding of these complex issues.

I am grateful to our industry partners for their generous support of this meeting. I also wish to thank the organising committee and Karen Patching for their time and meticulous attention to detail.

I hope you all enjoy the day.

Dr Doug Campbell
ACS Convenor

International Faculty

Hilary Grocott MD, FRCPC, FASE
Editor-in-Chief, Canadian Journal of Anesthesia/Journal canadien d'anesthésie
Professor, Departments of Anesthesia & Perioperative Medicine and Surgery
University of Manitoba / St. Boniface Hospital
Winnipeg, Manitoba, Canada

Dr Grocott currently holds the position of tenured Professor in the Department of Anesthesia, Perioperative & Pain Medicine at the University of Manitoba in Winnipeg, Canada. He also serves as Editor-in-Chief for the *Canadian Journal of Anesthesia*.

He completed medical school in Canada at the University of Saskatchewan in 1990, an anesthesia residency at the University of Manitoba, and clinical fellowship and research training in Cardiothoracic Anesthesiology at Duke University in Durham, North Carolina.

He has published more than 300 peer-reviewed articles, abstracts and book chapters in a range of anesthesia and cardiac surgical topics. His research interests largely relate to the cerebral sequelae of cardiac surgery where he has focused on cerebral monitoring and adverse outcomes after cardiac surgery.

Kate Leslie AO FAHMS, MBBS, MD, M Epi, MhlthServMt, FANZCA, FAICD, Adjunct Professor
Royal Melbourne Hospital / University of Melbourne
Melbourne, Victoria, Australia

Professor Kate Leslie is a specialist anaesthetist and head of research in the Department of Anaesthesia and Pain Management, Royal Melbourne Hospital. She is an honorary professional fellow in the Anaesthesia Perioperative and Pain Medicine Unit, Melbourne Medical School, and Department of Pharmacology and Therapeutics, University of Melbourne, and the Department of Epidemiology and Preventive Medicine, Monash University. Kate was a councillor and president of ANZCA between 2002-12. She is a member and former chair of the ANZCA Clinical Trials Network executive, and one of the six editors of Miller's Anesthesia textbook (9th edition).

Kate was awarded the AMA Woman in Medicine Award for 2014, the ANZCA Robert Orton Medal in 2015, the Melbourne Health Chairman's Award in 2016, and was made an Officer of the Order of Australia (AO) and fellow of the Australian Academy of Health and Medical Sciences (FAHMS) in 2016. She received an Honorary Doctorate of Health and Medical Sciences from the University of Melbourne in 2017.

Kate is a committed to multicentre collaborative clinical trials in anaesthesia and perioperative medicine and growing the next generation of research leaders.

Bernhard Riedel MBChB, MMed, FCA, FANZCA, FASE, MBA, PhD
Peter MacCallum Cancer Centre / University of Melbourne
Melbourne, Victoria, Australia

Professor Bernhard Riedel is the current Director of the Department of Anaesthesia, Perioperative and Pain Medicine at the Peter MacCallum Cancer Centre and holds an honorary academic appointment at the University of Melbourne. Bernhard is an academic anaesthetist with a primary research interest that focuses on improving surgical outcomes, especially following cancer surgery.

Previous appointments include: Professor and Deputy Chair in Anesthesiology and Intensive Care Medicine at The University of Texas M.D. Anderson Cancer Centre and Professor in Cardiac Anaesthesia at Vanderbilt University (USA).

New Zealand Faculty

Dr Doug Campbell	Specialist Anaesthetist, Auckland City Hospital
Professor Rod Jackson	Professor of Epidemiology, University of Auckland
Mr Michael Puttick	General Surgeon, Auckland City Hospital
Professor Keith Petrie	Professor of Health Psychology, University of Auckland
Dr Anne O'Callaghan	Palliative Medicine Specialist, Auckland City Hospital / Senior Lecturer, University of Auckland
Mr Luke Boyle	Data Scientist, Orion Health

Programme

Saturday, 24 March 2018

0800	Welcome and introduction	Dr Doug Campbell

SESSION 1 - Chair: Prof. Simon Mitchell

0810	Shared Decision Making (SDM) in the Perioperative Setting	Prof. Hilary Grocott
0850	How should we measure risk?	Dr Doug Campbell
0920	CVD risk calculators: the New Zealand experience	Prof. Rod Jackson

0950	Morning Break	

SESSION 2 - Chair: Prof. Alan Merry

1020	Onco-Anaesthesia – Improving Long Term Cancer Outcomes	Prof. Bernhard Riedel
1100	The future of risk prediction	Mr Luke Boyle
1130	Futility: when not operating is the best option	Mr Michael Puttick

1200	Lunch Break	

SESSION 3 - Chair: Dr Catherine Sayer

1300	Patients' Perceptions of Risk	Prof. Keith Petrie
1330	Risks associated with procedural sedation	Prof. Kate Leslie
1400	Risks and the relief of suffering	Dr Anne O'Callaghan

1430	Afternoon Break	

SESSION 4 - Moderators: Dr Jay van der Westhuizen & Dr Neil MacLennan

1500	Case-based discussions	
1530	Panel discussion	
1630	Future Meetings	Dr Kerry Gunn
1640	Meeting concludes	

1640	Drinks and Canapés	

Shared Decision Making (SDM) in the Perioperative Setting

Professor Hilary P. Grocott

Professor, Departments of Anesthesiology, Perioperative & Pain Medicine and Surgery
University of Manitoba, Winnipeg, Manitoba, Canada

Although numerous definitions exist in the literature for shared decision making (SDM), (1-3) they all contain the fundamental tenets that relate to the process of using the best available evidence to support patients in making healthcare decisions based on their own values, preferences, and beliefs. It revolves around the concept that the patient is really the only expert on what is right for them. Indeed, in this model, the clinician serves as an expert on the various diagnostic and therapeutic options that need to be presented. When done successfully, SDM represents the pinnacle of patient-centered care by providing patients, as Coulter and Collins stated in 2011, “the care they need and no less, and the care they want and no more”. (4)

Decision making has seen an evolution over the past approximately 150 years from one based on paternalism, through to one of a more informed basis in the 1980s, to the current SDM model being emphasized today. Up until the 1970s, the paternalistic approach was commonplace, and in fact codified in the medical ethics writings from the American Medical Association. Indeed their first Code of Medical Ethics in 1847 stated that “the obedience of patients to the prescriptions of his physician should be prompt and implicit. The patient should never permit his own crude opinions as to their fitness to influence his attention to them”.(5)

Of fundamental importance in SDM is that it pertains directly to how risk is communicated to the patient, and how the patient interprets this risk within their own (perioperative) life. We know from past studies, that 1 in 10 of us will eventually have surgery sometime in our life with an increasing chance as we age. (6) As a result, many of us will need to make decisions that balance having the proposed surgery with the various risks and benefits. However, the difficulty is that often the risks of surgery are uncertain. As a result, effectively communicating risk to patients is even more uncertain, particularly as the understanding of it can be quite variable.(7)

We know that “risk” from surgical procedures has several components. It involves direct surgical complications themselves (e.g., wrong vessel cut), but the vast majority of risks revolve around other procedural or perioperative issues (i.e., the sequelae of the procedures, such as the consequences of ischemic-reperfusion injury). That is, these risks are not directly related to the procedure itself, but complications that can manifest because of the complex interaction between the surgical procedure and the patient’s morbid conditions. This contrasts with anesthetic risks, which are exceedingly small (8) and are usually limited to those risks (including mortality) that occur within 24 hours of surgery. Fundamental to SDM considerations is not just these surgical, procedural, and anesthetic risks, but also the risks of potential long-term loss of independence. Importantly, these are not always considered by the surgeon or the anesthesiologist, in part because these complex interactions are not known by all and seemingly too distant in the future to be fully appreciated. Also important to this consideration is that not all physicians contain access to all of the necessary data.

SDM Steps

There are a number of steps to the SDM process that have been well-defined in the literature.(2) The first one begins with an introduction to the concept that a decision actually needs to be made. That is, the patient needs to be “informed”. Secondly, one needs to “explain” the various options that exist to the patient. Thirdly, one needs to “identify the patient’s individual values and goals”. Following the actual next step to “make a decision”, one should also “evaluate” the decision in accordance with the patient’s wishes and the factual information known by the practitioner.

As part of the SDM process, one needs to respect the patient’s decisional preference.(9) However, understanding the patient’s desire as to what level to be involved with is often a challenge. There are a number of different types of decisional making processes, including active, collaborative, and passive. In the active form, the patient wants to be presented with the facts and make all the decisions themself, somewhat in isolation of the physician’s input. However, the vast majority (>50%) likely want this process to be a collaborative decision. There are certain patients, particularly in older age groups, who are more likely to take

an even more passive approach and are far more comfortable with this almost anachronistic paternalistic approach.

Communication is fundamental aspect to SDM. However, one of the difficulties with SDM is that physicians generally think they are better communicators than they actually are. Indeed, some of the pitfalls of communication in SDM revolve around insufficient time given for establishing the correct relationships and communicating the risks and benefits, the often common poor diagnostic and prognostic accuracy that is available to physicians, as well as the lack of confidence in one's own skill in patient communication techniques that physicians generally are not trained in. Statistical illiteracy and difficulty with numeracy are often shared by patients and physicians. In addition, many patients have a poor overall educational levels.

Thankfully there are a number of solutions to address these communication pitfalls, such as tools and guides to optimize the SDM process. Indeed, visual aid guides, pictographs and figures are often very useful with communication. Furthermore, it is important to use absolute risk over relative risk in order to avoid non-transparent framing risk. For example, if a 1 in 7,000 risk is changed to 2 in 7,000 risk, although is a 100% relative increase in the risk, is a relatively minimal change in the overall odds of having the adverse effect. Telling the patient that you are doubling the risk likely misrepresents the perceived severity of the risk increase to the patient.

As mentioned previously, another road block to the SDM process is that decision support materials may not be in the language of the patient, or at their level of education. It is estimated that most patients operate at only the 8th grade level (for English), whereas most materials are at a more advanced stage than this. In addition, many patients have difficulty understanding risk:benefit statistics and numbers – i.e., a deficiency in numeracy. Accordingly, it is much easier for patients to understand graphical formats versus numeric or verbal formats. In addition, difficulty with understanding qualitative statements is also problematic. For example, suggesting verbally that the patient is at "high risk" does not really give the patient an understanding of whether this is high relative to their understanding or relative to the physician's understanding of risk.

When considering why SDM should be incorporated into our patient-centered approach, it can often simply be seen as an ethical imperative. However, it has also been shown to reduce variability in treatment options (preferences), reduce decisional conflict and patient anxiety, increase patient knowledge and preparation, as well as decisional satisfaction and quality.(3) It is also import to understand that as SDM is still in its relative infancy compared to other the other decisional models, the needed research to address issues of cost, efficiency and patient outcomes is similarly early in its development.

Future SDM Research

Whether SDM actually is effective is a fruitful area for future research.(10) The benchmarks for success need to be carefully defined. Although there is some evidence that patient confidence and satisfaction increases, and that there is less decisional conflict, whether this leads to a reduction overall of patient anxiety is an important point to consider.

In summary, SDM is a fundamental tenet of patient-centered care. Ensuring that risk is communicated to patients, and interpreted within the nuances of the patient's own values and goals for the proposed procedure, and that a decision is made in which both parties share in the process, is the most desired outcome.

References

1. Ting HH, Brito JP, Montori VM. Shared decision making: science and action. Circ Cardiovasc Qual Outcomes 2014;7:323-7.
2. Lin GA, Fagerlin A. Shared decision making: state of the science. Circ Cardiovasc Qual Outcomes 2014;7:328-34.
3. Boss EF, Mehta N, Nagarajan N, Links A, Benke JR, Berger Z, Espinel A, Meier J, Lipstein EA. Shared Decision Making and Choice for Elective Surgical Care: A Systematic Review. Otolaryngol Head Neck Surg 2016;154:405-20.
4. Coulter A, Collins A. Making Shared Decision-Making A Reality. No decision about me, without me London: The King's Fund, 2011
5. American Medical Association. Code of ethics of the American Medical Association: Concord [N.H.] : Printed by Asa McFarland, 1850.

6. Weiser TG, Regenbogen SE, Thompson KD, Haynes AB, Lipsitz SR, Berry WR, Gawande AA. An estimation of the global volume of surgery: a modelling strategy based on available data. Lancet 2008;372:139-44.
7. Sutherland J, Harris I. Surgery-risks, benefits, and the value of shared decision-making. Anaesth Intensive Care 2016;44:338-9.
8. Bainbridge D, Martin J, Arango M, Cheng D, Evidence-based Peri-operative Clinical Outcomes Research Group. Perioperative and anaesthetic-related mortality in developed and developing countries: a systematic review and meta-analysis. Lancet 2012;380:1075-81.
9. Mulley AG, Trimble C, Elwyn G. Stop the silent misdiagnosis: patients' preferences matter. BMJ 2012;345:e6572.
10. Lipkin M. Shared decision making. JAMA internal medicine 2013;173:1204-5.

How should we measure risk?

Dr Doug Campbell

Anaesthesia Specialist, Auckland City Hospital

Risk is the potential of gaining or losing something. In the context of surgery, risk is the potential for having an adverse outcome or complication. However, the risk of adverse outcomes following surgery often follow a characteristic course with risk being highest immediately following the procedure and then reducing over time. This highlights an additional problem. When should we measure risk? The timing of measurement needs to be adequate to determine the cumulative incidence of risk. An additional problem is that most complications of surgery are exacerbations of existing medical disease eg myocardial infarction, pneumonia, or new manifestations of medical disease. There will be a background incidence of these diseases, so determining if the event is caused by surgery or merely associated with surgery can be problematic. Differentiating between perioperative and background events is difficult. These issues will be highlighted during the presentation using examples of perioperative mortality in low and high-risk groups. [1]

Mortality risk information is often presented to patients as it is meaningful and important outcome for healthcare providers. We often have reasonable data so it used as a basis for discussions around risk during shared decision making and informed consent. But mortality is an unlikely outcome, even in high-risk patients. Some risk calculators such as the American College of Surgeons National Surgical Quality Improvement Programme (ACS-NSQIP) risk calculator [2] provide risks for non-fatal outcomes. The range of outcomes make it difficult for the clinician to interpret the overall impact on the patient. Days Alive At Home (DAAH) has recently been validated in surgical patients [3]. It may be a useful metric for measuring a patient-centred non-fatal outcome that is easily interpretable by patients and clinicians alike.

Similarly, disability is measured in clinical trials of perioperative medicine with metrics such as WHODAS 2.0. However, even experienced researchers have difficulty interpreting the results. In ischaemic stroke trials, disability is measured using the modified Rankin Scale at 3 months [4]. This simple ordinal scale ranks patients' disability into 7 categories using a short 10 point questionnaire. Examples will be provided during the presentation to show how easily understandable these metrics are for describing non-fatal risk in surgical patients and provide a pathway for future description of risk in surgical patients.

References

1. Campbell D, Merry A, Frampton C, Short T. Estimation of the magnitude and duration of excess perioperative mortality with implications for surgical decision-making and assessment of health outcomes: a retrospective database analysis. BJA 2018. In press
2. https://riskcalculator.facs.org/RiskCalculator/
3. Myles PS, Shulman MA, Heritier S, Wallace S, McIlroy DR, McCluskey S, Sillar I. Validation of days at home as an outcome measure after surgery: a prospective cohort study in Australia. BMJ 2017. http://dx.doi.org/10.1136/bmjopen-2017-015828
4. Goyal M, Menon BJ, van Zwam WH, Dippel DWJ, Mitchell PJ, Dernchuk AM, el al, for the HERMES Collaboration. Endovascular thrombectomy after large-vessel ischaemic stroke: a meta-analysis of individual data from five randomized trials. Lancet 2016; 387: 1723-31

CVD risk calculators: the New Zealand experience

Professor Rod Jackson

CVD Epidemiologist, School of Population Health, University of Auckland

Over 40 years ago Framingham Heart Study investigators developed multivariable cardiovascular disease (CVD) risk prediction equations that identified high-risk patients much more accurately than traditional classifications based on blood pressure or blood cholesterol levels alone. As the benefits of CVD risk-reducing interventions are proportional to pre-treatment risk, treating patients assessed as high CVD risk using multivariable prediction equations is also more effective than treating patients based on high levels of single risk factors.

In the 1990s New Zealand developed the world's first national CVD risk factor management guidelines based on multivariable predicted risk and recommended using 1991 Framingham Heart Study prediction equations to inform treatment decisions. By 2003 all individual CVD risk factor-based guidelines had been combined in one national CVD risk management guideline, which recommended that blood pressure-lowering and lipid-lowering drugs be offered to people with a predicted 5-year CVD risk of 15% or higher, using a modified Framingham equation.

At the time, no local cohort studies were available to validate the Framingham equations. So in 2003, we developed PREDICT, a computerised decision support system that helped general practitioners implement the national guidelines while simultaneously generating a cohort study to investigate whether the Framingham equation was applicable to the ethnically and socioeconomically diverse 21st century New Zealand population.

Over the next 15 years we collected the CVD risk profiles of over 500,000 New Zealanders using the PREDICT tool. The study period coincided with the introduction of a Ministry of Health-funded national primary care CVD risk assessment target, which resulted in approximately 90% of eligible New Zealand adults completing a CVD risk assessment. We have linked the individual patient CVD risk profile data collected in PREDICT to national hospitalisations and deaths using the encrypted National Health Identifier (NHI) and have recently completed the development of new CVD risk predictions.

These new equations include several new significant predictors over and above the standard Framingham predictors, notably ethnicity and the NZ Deprivation Index. We have demonstrated that the previously used Framingham equation is now poorly calibrated in the contemporary New Zealand primary care population, overestimating risk by more than 50%. However, despite the addition of several important new predictors that were each present in 10-20% of the study population, the discrimination performance of the new equations, as measured by standard discrimination statistics is only modestly better than Framingham.

This study highlights the importance of assessing the calibration of internationally developed risk prediction equations in the local populations where they are applied. It has also highlighted the inherent weakness of standard equation discrimination statistics, which are global statistics, that are very insensitive to the addition of new predictors representing important high-risk sub-populations. New criteria will need to be developed to decide whether additional predictors should be added to existing equations.

In February 2018, the New Zealand Ministry of Health released updated CVD risk management guidelines, recommending that general practitioners now use the new PREDICT-derived CVD risk equations. The threshold for considering drug treatment has also been lowered to 5% over 5 years. This change was made because of the lower risk predicted by the new equations, the very low cost of most CVD preventive medications today and increasing evidence that blood pressure-lowering and lipid-lowering medications have minimal significant side effects. We are currently developing a family of CVD risk prediction equations relevant to multiple patient groups and we also plan to update equations on a regular basis. It has been proposed that these equations will be maintained in one central national risk engine that can be linked to all electronic patient management systems. Not only would this provide one national standard set of equations but if the data entered into the equations is stored, it can be used for updating equations and for auditing practice.

Onco-Anaesthesia – Improving Long Term Cancer Outcomes

Professor Bernhard Riedel

Peter MacCallum Cancer Centre and the University of Melbourne, Australia

Cancer is a leading cause of death worldwide. Surgery is the primary and most effective treatment for most solid tumours.[1, 2] As such, two-thirds of cancer patients (~10 million patients annually and 45 million by 2030) require cancer surgery; >80% of cancer patients require anaesthesia for curative or supportive therapy.[1, 2] Minimum residual disease from tumour cell dissemination is unavoidable given that cancer cells are often circulating in the blood stream at diagnosis. This may predispose patients to scattered micro-metastases. Consequently, despite surgical treatment, cancer recurrence occurs in many patients—usually as metastases in organs distant from the primary tumour. Metastases impose a significant health burden and are responsible for more than 90% of cancer deaths.[3] Several perioperative (surgical and anaesthetic) factors may accelerate progression of such minimal residual disease.

Strikingly, a number of recent retrospective clinical cohort studies provide evidence that choice of anaesthesia during cancer resection surgery is linked to cancer recurrence. In a substantial review[4] we highlighted that the perioperative period during cancer surgery is accompanied by stress, inflammation, supressed cell-mediated immunity and increased pro-angiogenic and growth factors (e.g. VEGF) aimed at promoting wound healing. Together, these factors also promote local and distant growth of malignant tissue. Additionally, anaesthetic agents are implicated in inflammatory processes and in immunomodulation. Specifically, inhalational anaesthesia (with volatile agents such as sevoflurane) impairs the primary host defence (especially Natural Killer [NK] cells, which resist residual cancer cells after tumour resection),[5] promotes pro-inflammatory effects on macrophages (with compelling evidence that macrophages contribute to metastasis formation[3]), and up-regulates anti-apoptotic, hypoxia inducible factor-1 alpha (HIF-1α), VEGF[6] and PI3K-Akt pathway signalling.[7] In contrast, propofol-TIVA and lidocaine anaesthesia enhance host defenses (NK cells) and have anti-inflammatory effects on macrophages,[8-10] and down regulates mTOR, p53, p38 MAPK and MMP signalling.[7] Amide local anaesthetics (e.g. lidocaine, the internationally preferred name for lignocaine), commonly used as an intravenous infusion for analgesia during general anaesthesia,[11] also exhibit immune preserving and anti-inflammatory properties.[9, 10]

Intravenous general anaesthetic agent (propofol) may reduce cancer recurrence

Alarmingly, a systematic review and meta-analysis[12] of retrospective observational clinical studies[13-15] suggests that traditional inhalational anaesthesia is associated with a decrease in both disease-free survival (DFS) and overall survival (OS) when compared with the alternative of total intravenous anaesthesia using propofol (propofol-TIVA). Our animal experiments support this: when cancer resections are performed with propofol-TIVA or with intravenous lidocaine infusion (*vs*. volatile anaesthesia alone) cancer progression is decreased.

A large, retrospective cohort study by Wigmore *et al.*[15] evaluated >7,000 patients treated at The Royal Marsden Cancer Hospital (London, UK); roughly half were given volatile anaesthesia, with the others given propofol-TIVA for cancer surgery. The hazard ratio for death over median 2.6 years (with propensity matched anaesthetic approach) was 0.68 for propofol-TIVA *vs*. volatile (95% CI: 0.60-0.78); P<0.001) and 16% *v*s. 23% for mortality observed 5 years after surgery, favouring propofol-TIVA anaesthesia. Wigmore observed the strongest signal within the subgroup of patients with gastrointestinal tract cancers (HR=1.68 volatile *vs*. TIVA, 95% CI: 1.33-2.12; P<0.001). Similar survival benefits are reported in a retrospective analysis of 2,840 Swedish patients with colorectal and breast cancer,[13] in a smaller study of Korean patients with breast cancer,[14] and more recently in a study of 897 propensity matched Chinese patients having gastrectomy for cancer surgery.[16]

Translational research by Buggy and colleagues has found a positive association with propofol-TIVA with regional anaesthesia on *ex-vivo* immune cell function in breast cancer patients (compared with patients who had received volatile anaesthesia) with preservation of NK immune cell function against breast cancer cells[17] and more breast cancer cell apoptosis.[18] A systematic review of the studies to date indicates that propofol-TIVA might in fact be the preferred anaesthetic choice in cancer surgery.[12] Similarly, our meta-analysis of these retrospective studies found that propofol-TIVA, when compared with volatile, associates with improved overall survival (HR=0.73, 95% CI: 0.62-0.86; P<0.01) and improved DFS (HR=0.70, 95% CI: 0.56-0.89; p<0.01). However, the overall evidence for these anaesthetic techniques is currently low quality and a randomised clinical trial is urgently needed.[4]

Lidocaine as an intravenous analgesic may reduce cancer recurrence

Intravenous infusions of lidocaine are increasingly used as an analgesic adjunctive therapy with general anaesthesia for opioid-sparing and anti-inflammatory effects e.g. reduced pain and ileus.[11, 19, 20] Currently ERAS guidelines have a strong recommendation for perioperative use of intravenous lidocaine to enhance postoperative recovery after elective colorectal surgery. When administered intravenously, lidocaine has a wide therapeutic margin and appears to be safe for extended durations up to 24 hours postoperatively.[11]

Given the large overlap between inflammatory signalling pathways and cancer,[4, 21, 22] it is not surprising that amide local anaesthetic drugs may affect cancer pathways. While the cancer biology of lidocaine is complex, lidocaine has been shown to inhibit invasiveness of non-small cell lung cancer cells through inhibition of Src protein tyrosine kinase (Src-dependent mechanisms),[9] and in colon cancer cells through inhibition of Src-independent mechanisms, by blocking voltage-gated sodium channels.[23] Lidocaine also inhibits activation of metalloproteinase-9 (MMP-9), an enzyme necessary for the degeneration of the extracellular matrix by malignant cells.[24] *In vitro*, lidocaine, at clinically relevant concentrations, preserved cytotoxicity of isolated human NK cells[10] and in clinical studies preserved lymphocyte response and T-helper (Th) Th1/Th2 balance after surgery.[25] This provides the intriguing potential to 'repurpose' lidocaine, a commonly used drug that is safe, affordable, and available worldwide,[26] for preserving perioperative immune function during cancer surgery and substantiates the need for urgent randomised controlled trials to test lidocaine's adjunctive effects in the cancer setting.

Propofol, lidocaine, and volatile anaesthesia are all commonly used in clinical practice, with equipoise in the evidence base and among practicing clinicians. Our survey of 1,000 Australian and New Zealand anaesthetists found that 50% of anaesthetists believe that anaesthetic technique does not affect cancer outcomes and >80% use volatile anaesthesia in preference to propofol-TIVA. Compelling prospective clinical evidence is urgently needed to guide clinical practice.

NSAIDS & B-Blockers:

An increasing number of reviews outline the rationale and early evidence for the adaptation of anaesthetic techniques and the strategic use of anti-adrenergic, anti-inflammatory, and/or antithrombotic therapies. These findings raise the possibility that perioperative modulation of neural signalling or inflammation might offset surgery-related immunosuppression and reduce the malignant potential of residual cancer cells. Many of these strategies are currently under evaluation in large-cohort trials and hold promise as affordable, readily available interventions that will improve the postoperative recurrence-free survival of patients with cancer.

In preclinical studies, propranolol has been shown to inhibit a variety of β-adrenoceptor-mediated processes including tumour cell invasion, angiogenesis, lymphangiogenesis, and epithelial-to-mesenchymal transition. A recent randomized double-blind clinical trial translated these preclinical findings into the clinical trial setting. Women were prescribed either the combination of propranolol (40 mg daily) plus the NSAID etodolac (800 mg daily) or placebo for 5 days before breast cancer surgery and for 5 days after surgery.[27] The investigators found that drug treatment, compared with placebo, partially mitigated the postoperative increase in inflammation as indicated by serum IL-6 levels (4.4-fold versus 5.7-fold increase, respectively; $P < 0.001$) and serum C-reactive protein levels (6.3-fold versus 8.3-fold increase, respectively; $P < 0.001$), both of which are markers of the severity of the surgical stress response. Propranolol plus etodolac, compared with placebo, also prevented the preoperative increase in inflammatory marker levels (IL-6, 11% versus 24%, $P < 0.0009$; C-reactive protein, 10% versus 41%, $P < 0.034$), suggesting that preoperative anxiety primes patients' stress responses before surgery. Notably, drug treatment also reduced the expression of several tumour-promoting genes including transcription factors involved in the promotion of metastasis, recruitment of myeloid cell types, and epithelial-to-mesenchymal transition. These findings demonstrate that brief inhibition of perioperative neural or pro-inflammatory signalling reduces the malignant potential of tumour cells at the time of surgery. Defining the relative contributions of β-blockers and NSAIDs to these effects will be important. Zhou et al.[28] examined the effect of propranolol (60 mg daily) on postoperative peripheral immune cell numbers. Treatment with propranolol was commenced on the day of mastectomy and was found to mitigate against postoperative elevation of circulating Treg cell numbers and suppression of a tumour-antigen-specific CD4+ T cell response. These findings raise the possibility that perioperative modulation of neural signalling or inflammation might offset surgery-related immunosuppression and reduce the malignant potential of residual cancer cells and may explain the protective signals observed with neuraxial anaesthesia; which when used in addition or as an alternative to general anaesthesia, reduces circulating catecholamine levels, inflammation, immunosuppression, and provides an alternative means of achieving sympathetic blockade during cancer surgery, inflammation, and immunosuppression. Overall, the conclusions of two meta-analyses of predominantly retrospective data published in the past 3 years show that the use of perioperative neuraxial anaesthesia is associated with a survival benefit.[29, 30] However, robust studies of neuraxial technique on cancer outcomes are awaited.

RIOT:

While we continue to work on basic and translational science projects to better understand the perioperative biology in the context of cancer care and await definitive clinical trials of preferential anaesthetic techniques for cancer surgery, our aim and efforts should also focus on optimizing the patient's preoperative condition (prehabilitation) to ensure the maximum benefits of surgery (neoadjuvant therapy when indicated, nutritional enhancement, physiological conditioning [strength and cardiovascular training], anaemia management, and behavioral therapy for stress reduction) to minimize postoperative complications and get the patient 'back on track' to complete their cancer journey (adjuvant therapies).

Within the perioperative care of cancer patients, the term "RIOT" is used to describe a surgical oncology quality metric for Return to Intended Oncologic Therapy. This simple formula divides the number of patients who initiated postoperative adjuvant therapy (can be surgical, medical, or radiotherapy) by the number intended to receive it based on stage of cancer to create the RIOT rate. Various adjuvant systemic chemotherapy trials give a glimpse of these data. Initial exploration of these metrics determined that failure to RIOT was associated with significantly worse oncologic outcomes.[31] These findings have received subsequent support by several groups studying outcomes in a number of different cancers.[32, 33] As such it is increasingly suggested that all surgeons who perform cancer surgery, be able to

quote/report their RIOT metrics. Further, RIOT may be a reliable surrogate endpoint for recurrence-free and overall survival, available early in the cancer care continuum.

In summary, effective perioperative care of the cancer patient is increasingly complex and our knowledge of the biologic impact of the adrenergic-inflammatory-immune (surgical) stress response and anaesthetic techniques on cancer progression pathways, and thus long-term outcomes, is rapidly expanding. As such, anaesthesia and perioperative care for cancer patients should not simply be the prevention of awareness and administration of analgesia but rather an opportunity to minimise the biological perturbation of the surgical stress response and to adjust anaesthetic techniques to minimize activation of cancer progression pathways. Importantly, we should also focus our perioperative strategies on reducing perioperative morbidity to ensure functional recovery after surgery that allows timely return to intended oncologic (adjuvant) therapies (RIOT).

It is this comprehensive approach to patient care that could potentially influence oncological outcomes by minimizing loco-regional recurrence and distant metastasis.

References

1. Alkire BC, Shrime MG, Dare AJ, Vincent JR, Meara JG. Global economic consequences of selected surgical diseases: a modelling study. *The Lancet Global health* 2015; **3 Suppl 2**: S21-7.
2. Sullivan R, Alatise OI, Anderson BO, et al. Global cancer surgery: delivering safe, affordable, and timely cancer surgery. *The Lancet Oncology* 2015; **16**(11): 1193-224.
3. Seyfried TN, Huysentruyt LC. On the origin of cancer metastasis. *Critical reviews in oncogenesis* 2013; **18**(1-2): 43-73.
4. Hiller JG, Perry NJ, Poulogiannis G, Riedel B, Sloan EK. Perioperative events influence cancer recurrence risk after surgery. *Nature reviews Clinical oncology* 2017.
5. Markovic SN, Knight PR, Murasko DM. Inhibition of interferon stimulation of natural killer cell activity in mice anesthetized with halothane or isoflurane. *Anesthesiology* 1993; **78**(4): 700-6.
6. Lu Y, Wang J, Yan J, et al. Sevoflurane attenuate hypoxia-induced VEGF level in tongue squamous cell carcinoma cell by upregulating the DNA methylation states of the promoter region. *Biomedicine & pharmacotherapy = Biomedecine & pharmacotherapie* 2015; **71**: 139-45.
7. Dubowitz JA, Sloan EK, Riedel BJ. Implicating anaesthesia and the perioperative period in cancer recurrence and metastasis. *Clinical & experimental metastasis* 2017.
8. Chen RM, Chen TG, Chen TL, et al. Anti-inflammatory and antioxidative effects of propofol on lipopolysaccharide-activated macrophages. *Annals of the New York Academy of Sciences* 2005; **1042**: 262-71.
9. Chamaraux-Tran TN, Piegeler T. The Amide Local Anesthetic Lidocaine in Cancer Surgery-Potential Antimetastatic Effects and Preservation of Immune Cell Function? A Narrative Review. *Frontiers in medicine* 2017; **4**: 235.
10. Ramirez MF, Tran P, Cata JP. The effect of clinically therapeutic plasma concentrations of lidocaine on natural killer cell cytotoxicity. *Regional anesthesia and pain medicine* 2015; **40**(1): 43-8.
11. Dunn LK, Durieux ME. Perioperative Use of Intravenous Lidocaine. *Anesthesiology* 2017; **126**(4): 729-37.
12. Soltanizadeh S, Degett TH, Gogenur I. Outcomes of cancer surgery after inhalational and intravenous anesthesia: A systematic review. *Journal of clinical anesthesia* 2017; **42**: 19-25.
13. Enlund M, Berglund A, Andreasson K, Cicek C, Enlund A, Bergkvist L. The choice of anaesthetic--sevoflurane or propofol--and outcome from cancer surgery: a retrospective analysis. *Upsala journal of medical sciences* 2014; **119**(3): 251-61.
14. Lee JH, Kang SH, Kim Y, Kim HA, Kim BS. Effects of propofol-based total intravenous anesthesia on recurrence and overall survival in patients after modified radical mastectomy: a retrospective study. *Korean journal of anesthesiology* 2016; **69**(2): 126-32.
15. Wigmore TJ, Mohammed K, Jhanji S. Long-term Survival for Patients Undergoing Volatile versus IV Anesthesia for Cancer Surgery: A Retrospective Analysis. *Anesthesiology* 2016; **124**(1): 69-79.
16. Zheng X, Wang Y, Dong L, et al. Effects of propofol-based total intravenous anesthesia on gastric cancer: a retrospective study. *OncoTargets and therapy* 2018; **11**: 1141-8.
17. Buckley A, McQuaid S, Johnson P, Buggy DJ. Effect of anaesthetic technique on the natural killer cell anti-tumour activity of serum from women undergoing breast cancer surgery: a pilot study. *British journal of anaesthesia* 2014; **113 Suppl 1**: i56-62.
18. Jaura AI, Flood G, Gallagher HC, Buggy DJ. Differential effects of serum from patients administered distinct anaesthetic techniques on apoptosis in breast cancer cells in vitro: a pilot study. *British journal of anaesthesia* 2014; **113 Suppl 1**: i63-7.
19. Eipe N, Gupta S, Penning J. Intravenous lidocaine for acute pain: an evidence-based clinical update. *BJA Education* 2016; **16**(9): 292-8.
20. Hollmann MW, Durieux ME. Local anesthetics and the inflammatory response: a new therapeutic indication? *Anesthesiology* 2000; **93**(3): 858-75.
21. Kim MP, Park SI, Kopetz S, Gallick GE. Src family kinases as mediators of endothelial permeability: effects on inflammation and metastasis. *Cell and tissue research* 2009; **335**(1): 249-59.
22. Kobayashi H, Boelte KC, Lin PC. Endothelial cell adhesion molecules and cancer progression. *Current medicinal chemistry* 2007; **14**(4): 377-86.
23. Baptista-Hon DT, Robertson FM, Robertson GB, et al. Potent inhibition by ropivacaine of metastatic colon cancer SW620 cell invasion and NaV1.5 channel function. *British journal of anaesthesia* 2014; **113 Suppl 1**: i39-i48.

24. Lenglet S, Mach F, Montecucco F. Matrix metalloproteinase-9: a deleterious link between hepatic ischemia-reperfusion and colorectal cancer. *World journal of gastroenterology* 2012; **18**(48): 7131-3.
25. Wang HL, Yan HD, Liu YY, et al. Intraoperative intravenous lidocaine exerts a protective effect on cell-mediated immunity in patients undergoing radical hysterectomy. *Molecular medicine reports* 2015; **12**(5): 7039-44.
26. Pantziarka P, Bouche G, Sullivan R, Ilbawi AM, Dare AJ, Meheus L. Perioperative therapies - Enhancing the impact of cancer surgery with repurposed drugs. *European journal of surgical oncology : the journal of the European Society of Surgical Oncology and the British Association of Surgical Oncology* 2017; **43**(11): 1985-8.
27. Shaashua L, Shabat-Simon M, Haldar R, et al. Perioperative COX-2 and beta-Adrenergic Blockade Improves Metastatic Biomarkers in Breast Cancer Patients in a Phase-II Randomized Trial. *Clinical cancer research : an official journal of the American Association for Cancer Research* 2017; **23**(16): 4651-61.
28. Zhou L, Li Y, Li X, et al. Propranolol Attenuates Surgical Stress-Induced Elevation of the Regulatory T Cell Response in Patients Undergoing Radical Mastectomy. *Journal of immunology* 2016; **196**(8): 3460-9.
29. Sun Y, Li T, Gan TJ. The Effects of Perioperative Regional Anesthesia and Analgesia on Cancer Recurrence and Survival After Oncology Surgery: A Systematic Review and Meta-Analysis. *Regional anesthesia and pain medicine* 2015; **40**(5): 589-98.
30. Weng M, Chen W, Hou W, Li L, Ding M, Miao C. The effect of neuraxial anesthesia on cancer recurrence and survival after cancer surgery: an updated meta-analysis. *Oncotarget* 2016; **7**(12): 15262-73.
31. Aloia TA, Zimmitti G, Conrad C, Gottumukalla V, Kopetz S, Vauthey JN. Return to intended oncologic treatment (RIOT): a novel metric for evaluating the quality of oncosurgical therapy for malignancy. *Journal of surgical oncology* 2014; **110**(2): 107-14.
32. Merkow RP, Bilimoria KY, Tomlinson JS, et al. Postoperative complications reduce adjuvant chemotherapy use in resectable pancreatic cancer. *Annals of surgery* 2014; **260**(2): 372-7.
33. Slankamenac K, Slankamenac M, Schlegel A, et al. Impact of postoperative complications on readmission and long-term survival in patients following surgery for colorectal cancer. *International journal of colorectal disease* 2017; **32**(6): 805-11.

The future of risk prediction

Mr Luke Boyle

Data Scientist, Orion Health

In many healthcare settings, a patients' care is influenced by an assessment of their risk for an outcome or condition. Prognostic risk models determine this risk and are part of contemporary clinical practice. In some settings, these models are more accurate than clinician estimate of risk [1,2]. These risk models have historically been built using traditional statistical methods such as linear regression and calculated using small local datasets.

Often, these models make explicit assumptions of the data, such as linear relationships of risk factors to overall outcomes, and predefine factors in the model before model construction. This approach ignores underlying complex relationships between risk factors and actual outcomes while also preventing new unexpected relationships being found between patient factors and risk. Risk prediction approaches that can investigate more nuanced relationships, effectively explore larger datasets and also provide more accurate assessments should be explored and compared to current methods.

Data availability and volume is increasing exponentially in the healthcare industry. This is being driven by the automatic digitization of data in hospitals and the huge volumes of data captured from daily routines through sensors and smartphones [3, 4]. Currently the focus is on data capture but increasingly the focus is moving to data usage. Investigations are centred on how these huge data sets can be harnessed to provide insights in real time. People across sectors and skill specialties understand that data collection alone is not enough and we need interdisciplinary teams working to unlock the potential of the data we capture. New Zealand is uniquely positioned to make good use of this transformation with government mandated data collection existing for years and unique identifiers making data linking simple [5].

Machine learning (ML) is a set of techniques that has gained prominence in recent years for its ability to handle big data and deliver new insights [6]. ML developed from work looking into automatic pattern recognition and these techniques can overcome many common limits of traditional statistics. ML allows a machine to learn patterns through reinforcement and building complex relationships which minimise error between predictions and observed outcomes [7].

In this talk I will discuss what ML offers risk prediction, illustrate examples of where these techniques already permeate your life and also discuss successful examples of ML in risk prediction and an anaesthetic setting. I will also provide background on how these techniques work and help you to better grasp what some of the current buzzwords such as 'Artificial Intelligence' (AI) and 'deep learning' actually mean. These techniques are invariably going to be applied in health care for risk prediction and decision support. I hope to outline the strengths and weakness of these approaches.

References

1. Moonesinghe SR, Mythen MG, Das P, Rowan KM, Grocott MPW. Risk stratification tools for predicting morbidity and mortality in adult patients undergoing major surgery. Anesthesiology 2013; 119: 959-81.
2. Wijeysundera DN. Predicting outcomes: Is there utility in risk scores? Can J Anesth 2016; 63: 148-58.
3. Dinov ID. Volume and Value of Big Healthcare Data. Med Stat Inform. 2016; 4: 3.
4. Raghupathi W, Raghupath V. Big data analytics in healthcare: promise and potential. Health Information Science and Systems 2014, 2:3.
5. https://www.health.govt.nz/nz-health-statistics/about-data-collection
6. Obermeyer Z, Emanuel EJ. Predicting the Future— Big Data, Machine Learning, and Clinical Medicine. The New England journal of medicine 2016; 375(13): 1216–9. https://doi.org/10.1056/NEJMp1606181 PMID: 27682033.
7. Dreiseitl S, Ohno-Machado L. Logistic regression and artificial neural network classification models: a methodology review. Journal of Biomedical Informatics 2002; 35(5–6): 352–9. PMID: 1296878.

Futility: when not operating is the best option

Mr Michael Puttick

General Surgeon, Auckland City Hospital

Futility: Noun, pointlessness or uselessness.

Futile: Adjective, incapable of producing any useful result; pointless.

When we focus on mortality, particularly 30-day mortality, as an outcome we have a very two-dimensional view of surgical success. For many elderly people major surgery can lead to an irreversible decline in function and a quality of life that is less than it would have been had they had no surgery at all.

It is clear that surgery is futile when the outcome with or without surgery is the same (usually death). However a more nuanced definition would be when surgery leads to a shorter or lower quality of disease free life than life without surgery but with the disease.

Data show that many patients who have an acute admission are in their final year of life and so the mindset of surgery and care should be palliative rather than curative

Surgeons will often request an Anaesthetic Review to help decision making but this can mean a number of things

1. Can this paint be medically optimised in order to make the surgery safer?
2. Just how risky is surgery and anaesthesia in order to achieve proper informed consent?
3. Should we even be performing surgery?

Unless these questions are made explicit then a patient can be optimised and even informed of high risk of anaesthesia, rather than a proper decision not to operate being made.

It is often easier to operate than not, and the conversations around not operating are difficult. When the outcomes we use, such as mortality, are simplistic we do not address the medium and long-term consequences of surgery. There is a myth that you will either survive surgery and return to normality or peacefully die in the operating room under anaesthesia; the reality is that neither outcomes are true. We quote mortality statistics in percentages and patients will legitimately ask why would I not take a 1% chance of surviving if it is there? We in turn ask ourselves who are we do deny them this?

The questions we need to ask are

- What do you want from life? and
- What do you most fear?

Many patients will fear loss of independence, pain or a stoma more than they fear death and if these questions can be used as a framework for discussion we are less likely to embark on surgery that could be regarded as futile and is certainly not wanted by the patient.

In this talk we will discuss elective surgery and emergency surgery and situations in which operating may not be the best thing and how conversations between surgeons, anaesthetists patients and their families could avoid futile surgery.

Further reading

1. Atul Gawande. Being Mortal: Medicine and What Matters in the End. Picador 2017
2. Paul Kalanithi. When Breath becomes Air. Random House 2016
3. Henry Marsh. Admissions: Life as a Brain Surgeon. St Martin's Press 2017

Patients' Perception of Risk

Professor Keith J. Petrie

Professor of Health Psychology, University of Auckland

The accurate perception of risk is critical for patients to make rational decisions about health care interventions. Health professionals attempt to aid this process by providing patients with information about the likelihood of future outcomes. However, there are large and important differences between the risks and benefits of treatment perceived by patients and those perceived by doctors. This talk discusses some of the factors that influence patients' (mis)perceptions of risk including low levels of knowledge about anatomy, confirmation bias, motivated denial, the "better than average" effect, numeracy difficulties and the way risk material is processed. These issues make the presentation and comprehension of risk information difficult for doctors and provide considerable opportunity for miscommunication in clinical conversations. Unfortunately, the talk provides no magic answers. Furthermore, there is considerable (greater than 90%) risk that those listening are likely to be left feeling despondent and miserable.

Risks associated with procedural sedation

Professor Kate Leslie AO FAHMS

Royal Melbourne Hospital, Australia

Introduction

Gastrointestinal endoscopy is performed in a range of settings and by a variety of health professionals. Specialist anaesthetists commonly administer sedation for endoscopy in Australia, but there is limited literature on the safety of this service model. Sedation practice in other comparable nations varies widely, with non-medical, non-specialist, specialist and hybrid models in place. In this talk I will use a recent study conducted in Victoria, Australia, to illustrate the risks associated with sedation. The aim of the study was to determine the risk profile of presenting patients and the incidence of significant unplanned events in patients having endoscopy at the nine public hospitals affiliated with the University of Melbourne that provide endoscopy services for adult patients.

Methods

The study included all adult elective and emergency patients who presented for upper or lower GI endoscopy (including enteroscopy and ERCP) at the nine University of Melbourne-affiliated hospitals that provide endoscopy services for adult patients. Data were collected during a 28-day period between March and August 2015. Sedation was administered by specialist anaesthetists or supervised ANZCA trainees. Outcome measures were incidence of significant unplanned events including airway obstruction, cardiovascular deterioration, abandoned procedure, unplanned intubation, advance life support and death within 30 days.

Results

2,182 procedures in 2,132 patients were included. Patients were aged 60 (range: 18-95) years and 42% were ASA physical status 3-5. The most common procedures were gastroscopy alone (33%), colonoscopy alone (41%) and combined gastroscopy and colonoscopy (18%). Patients were managed by a specialist anaesthetist without the participation of a trainee anaesthetist in 80% of cases. Oxygen saturation, blood pressure, ECG and capnography were monitored in 100%, 99%, 64% and 64% of patients respectively. Most (92%) patients were managed without an airway device. Propofol was used in 98% of cases at a median dose of 200 (IQR: 130-300) mg. Most (82%) patients were discharged home after the procedure with a median post-procedure admission time of 60 (IQR 33-82) minutes. Forty-seven patients (2.2%) had at least one subsequent procedure during the study period.

Emergency patients were older (63 $\pm$ 18 vs. 60 $\pm$ 16 years; P <0.0001) and had more co-morbidities than elective patients (Charlson co-morbidity score: 5 [IQR 3-7] vs. 3 [2-5]; P <0.0001). They were more likely to have gastroscopy alone (53% vs. 29%; P <0001), were more likely to have ECG monitoring (76% vs. 62%; P <0.0001), and were more likely to be managed with an airway device than elective patients (20% vs. 6%; P <0.0001). Emergency patients were more likely to receive neuromuscular blocking drugs (16% vs. 1.5%; P <0.0001) and intravenous fluids (68% vs. 47%; P <0.0001) than elective patients and they were more likely to have another endoscopy during the study period (7% vs. 1%; P <0.0001).

Significant hypotension was the most common significant unplanned event (11.8%). Seven patients (0.3%) required unplanned endotracheal intubation and two patients (0.1%) required advanced life support. The overall 30-day mortality rate was 1.2% (95% confidence interval: 0.8 to 1.8) with a median time to death of 11 (range: 0-28) days. Emergency patients suffered more intra-operative events (20.6% vs. 14.4%) and 30-day mortality (6.0% vs. 0.2%; P <0.0001) than elective patients.

Conclusion

This study demonstrated that many patients presenting for endoscopy at University of Melbourne-affiliated hospitals have high pre-procedure risk status. Intra-procedure significant unplanned events were common, especially in emergency patients. The current specialist anaesthetist-based service model provides the greatest flexibility with respect to sedation services for endoscopy at our hospitals. This study was noted for its high rate of significant unplanned events, which was attributed by one commentator to the patient population treated in our hospitals and the apparent preference for moderately-deep propofol-based sedation.

References

1. Leslie K, Allen ML, Hessian EC et al. Safety of sedation for gastrointestinal endoscopy in a group of university-affiliated hospitals: A prospective cohort study. Br J Anaesth. 2017; 118: 90-9.
2. Sneyd JR. Making sense of propofol sedation for endoscopy. Br J Anaesth. 2017; 118: 6-7.
3. ANZCA. Professional document: Guidelines on sedation and/or analgesia for diagnostic and interventional medical or surgical procedures (ps9) Melbourne: Australian and New Zealand College of Anaesthetists, 2014.
4. American Society of Anesthesiologists Task Force on Moderate Procedural Sedation and Analgesia Practice guidelines for moderate procedural sedation and analgesia 2018. 2018; 128: 437-79.

Risks and the relief of suffering

Dr Anne O’Callaghan

Palliative Medicine Specialist, Auckland City Hospital / Senior Lecturer, University of Auckland

Discussions of risk can be confusing to patients who are faced with difficult decisions at a time of heightened emotional intensity. Clinicians can relieve or exacerbate the suffering of patients and their families or whanau at this time. Strategies that are more likely to result in relief of suffering will be discussed. These include practical tips for changing theoretical risk into hope-enhancing interventions, whether surgical or not. There is a risk you will leave this presentation with some new skills and ideas.

Sponsors

BRONZE

Medtronic New Zealand Ltd

Level 3 – Building 5
Central Park Corporate Centre
666 Great South Road, Penrose
Auckland 1051, New Zealand

Private Bag 94-315, Pakuranga
Auckland 2140, New Zealand

Tel: 0800 377 807
Fax: 0800 633 876

Email: nzsales@medtronic.com

Website: www.medtronic.co.nz

As a global leader in medical terminology, services and solutions, Medtronic improves the lives and health of millions of people each year. We use our deep clinical, therapeutic, and economic expertise to address the complex challenges faced by healthcare systems today. Let's take healthcare Further, Together.

Learn more at www.Medtronic.co.nz

BRONZE

Merck Sharp & Dohme (New Zealand) Limited

Lynda McCree
Hospital Product Specialist

Level 3, 123 Carlton Gore Road, Newmarket
Auckland 1024, New Zealand

Email: lynda.mccree@merck.com

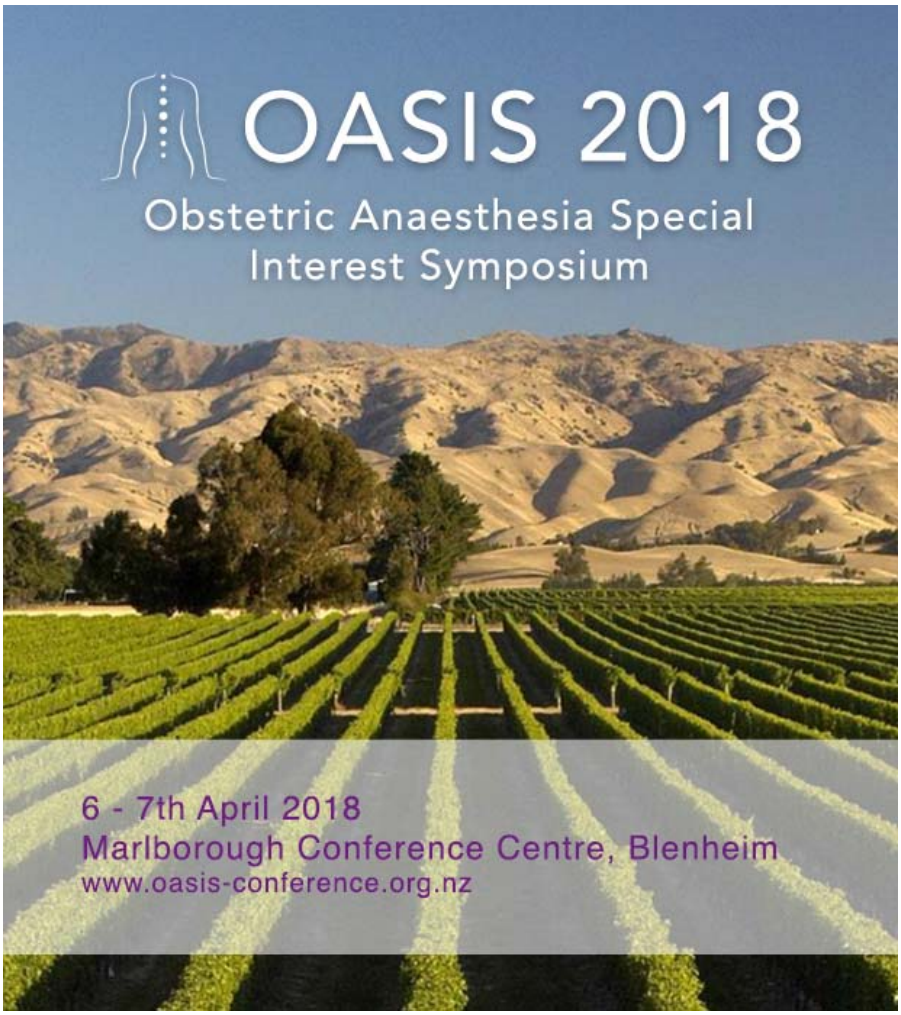
Mobile: +64 (0)27 585 7924

Website: www.msd.co.nz

Merck Sharp & Dohme (NZ) Ltd (MSD) was established in New Zealand in 1962 and is a wholly owned subsidiary of Merck & Co. Inc.

In New Zealand, MSD provides innovative medicines, vaccines and biologic therapies across a broad range of therapeutic areas. These include; oncology, anaesthesia, hepatitis C, cardiovascular, musculoskeletal, women's health, fertility, HIV/AIDS, antibacterials/antifungals and immunisations.

MSD is one of the few pharmaceutical companies still conducting clinical trials in New Zealand and invests approximately $NZ45 million in local clinical research. Current research programmes include oncology, infectious diseases, endocrinology, neurology, cardiology and vaccines. These trials provide free therapeutic access to hundreds of patients.


OASIS 2018
Obstetric Anaesthesia Special Interest Symposium
6 - 7th April 2018
Marlborough Conference Centre, Blenheim
www.oasis-conference.org.nz

BATS
BETTER ANAESTHESIA THROUGH SONOGRAPHY

Anatomy and Ultrasound for Regional Anaesthesia

This workshop combines gross anatomy from prosections with sonoanatomy in live models for the best regional anaesthesia learning experience.

FUJIFILM Value from Innovation SonoSite

ADMEDUS

B BRAUN SHARING EXPERTISE

To register please go to

www.bats.ac.nz

Contact: Karen Patching
Email: karenp@adhb.govt.nz *or*
Tel: 375 7085 / *Ext* 25700

JAFA Joint Anaesthesia Faculty Auckland

THE UNIVERSITY OF AUCKLAND FACULTY OF MEDICAL AND HEALTH SCIENCES

usabcd
UltraSound Airway Breathing Circulation Dolor

Focus Assessed Transthoracic Echo (FATE)

A one-day introductory echocardiography workshop

FUJIFILM Value from Innovation SonoSite

BATS BETTER ANAESTHESIA THROUGH SONOGRAPHY

JAFA Joint Anaesthesia Faculty Auckland

DSATC

Definitive Surgical & Anaesthetic Trauma Care Course

Masterclass for Anaesthetists, Intensivists, Surgeons, & OR Nurses

30 July – 1 August 2018

School of Medicine, Auckland, New Zealand

AQUA

ANNUAL QUEENSTOWN UPDATE IN ANAESTHESIA

SAVE THE DATE

23-25 August 2018

VENUE

The Heritage, Queenstown
New Zealand

TO REGISTER YOUR INTEREST PLEASE GO TO

www.aqua.ac.nz

The AQUA Conference 2018 can be claimed under the ANZCA CPD Knowledge and Skills category under the following activities:
Lectures 1 credit/hour. Small group discussions 2 credits/hour.

New Zealand Society of Anaesthetists

JAFA
Joint Anaesthesia Faculty Auckland


AUCKLAND CITY
SYMPOSIUM
Perioperative Medicine
Let's save the planet...
www.acs.ac.nz
SAVE THE DATE
Saturday, 30 March 2019
School of Medicine
The University of Auckland
Grafton, Auckland, New Zealand
JAFA
Joint Anaesthesia Faculty Auckland
THE UNIVERSITY OF AUCKLAND
FACULTY OF MEDICAL AND HEALTH SCIENCES

EVEN WHEN SILENT, YOUR PATIENT SPEAKS.
WE HELP YOU LISTEN.
CEREBRAL & END ORGAN COMPROMISES.

CAPNOSTREAM™ 35
Poratable Respiratory Monitor

INVOS™ Cerebral/Somatic Oximeter

Enables detection of regional oxygen desaturation (rSO_2), prompt intervention and improved patient outcomes.[4-6]

BIS™ Brain Monitoring System

CAPNOSTREAM™ 35 Poratable Respiratory Monitor

Continuous monitoring of $etCO_2$, respiration rate, and SpO_2.
In virtually any clinical setting.

CAPNOSTREAM™ 35
Poratable Respiratory Monitor

BIS™ Brain Monitoring System

Enables depth of anaesthesia monitoring and improved patient outcomes. Multiple studies have indicated reductions in post-operative delirium.[1-3]

REFERENCES

1. Chan et al-BIS Guided Anesthesia Decreases Delirium etc-J Neurosurg Anesthesiol 2013 v25 p33–42
2. Cochrane Review - Delirium 2016 CD005563_abstract. Interventions for preventing deliriumin hospitalised non-ICU patients (Review) Siddiqi N, Harrison JK, Clegg A, Teale EA, Young J, Taylor J, Simpkins SA
3. Aldecoa et al-ESA Evidence based and Cnsus-based Guideline on Post-op Delirium- Eur J Anaesthiol 2017 v34 p192-214
4. Murkin JM, Adams SJ, Novick RJ, et al. Monitoring brain oxygen saturation during coronary bypass surgery: a randomized, prospective study. *Anesth Analg.* 2007;104:51-8.
5. Slater JP, Guarino T, Stack J, et al. Cerebral oxygen desaturation predicts cognitive decline and longer hospital stay after cardiac surgery. *Ann Thorac Surg.* 2009;87(1):36-44; discussion:44-5.
6. Goldman S, Sutter F, Ferdinand F, Trace C. Optimizing intraoperative cerebral oxygen delivery using noninvasive cerebral oximetry decreases the incidence of stroke for cardiac surgical patients. *Heart Surg Forum.* 2004;7(5):E376-81.

Medtronic Australasia Pty Ltd
2 Alma Road
Macquarie Park, NSW 2113
Australia
Tel: +61 2 9857 9000
Fax: +61 2 9889 5167
Toll Free: 1800 668 670

Medtronic New Zealand Ltd
Level 3 - Building 5, Central Park Corporate Centre
666 Great South Road
Penrose, Auckland 1051
New Zealand
Fax: +64 9 918 3742
Toll Free: 0800 377 807

medtronic.com.au
medtronic.co.nz

Medtronic

EXPERIENCE THE BRIDION EFFECT.

PREDICTABLE.[1] COMPLETE.[2] RAPID.[2]

BRIDION provides complete reversal from neuromuscular blockade.[1,2]

#Reversal of neuromuscular blockade induced by rocuronium or vecuronium. **References: 1.** Blobner M, Eriksson LI, Scholz J, et al. Reversal of rocuronium-induced neuromuscular blockade with sugammadex compared with neostigmine during sevoflurane anaesthesia: results of a randomised, controlled trial. Eur J Anaesthesiol. 2010;27(10):874–881. doi:10.1097/EJA.0b013e32833d56b7. **2.** Jones RK, Caldwell JE, Brull SJ, et al. Reversal of profound rocuronium-induced blockade with sugammadex: a randomized comparison with neostigmine. Anesthesiology. 2008;109(5):816–824.

Bridion® (Sugammadex) is a Prescription Medicine, fully funded under Section H of the Pharmaceutical Schedule from 1 June 2013. Indications: Reversal of neuromuscular blockade induced by rocuronium or vecuronium. **Dosage & Administration:** Immediate reversal of intense block. 16.0 mg/kg IV, three minutes following administration of rocuronium (1.2 mg/kg) in adults, (including: elderly, obese patients, patients with mild and moderate renal impairment and patients with hepatic impairment). Routine reversal of profound block 4.0 mg/kg IV following rocuronium- or vecuronium induced block when recovery has reached 1-2 post-tetanic counts; in adults. Routine reversal of shallow block. 2.0 mg/kg IV following rocuronium- or vecuronium-induced block when recovery has occurred up to reappearance of T2; in adults; 2.0 mg/kg IV following rocuronium in children and adolescents (2-17 years). **Contraindications:** Hypersensitivity to sugammadex or to any of the excipients. **Precautions:** Repeated exposure in patients; respiratory function monitoring during recovery; use for reversal of neuromuscular blocking agents other than rocuronium or vecuronium; coagulopathy; severe renal impairment; severe hepatic impairment; marked bradycardia, use in ICU; hypersensitivity reactions (including anaphylactic reactions); pregnancy (Category B2); lactation; infants less than 2 years of age including neonates; prolonged neuromuscular blockade (sub-optimal doses) and delayed recovery. **Interactions:** Potential identified with toremifene, hormonal contraception. Could interfere with progesterone assay and some coagulation parameters. **Adverse Reactions:** Dysgeusia, prolonged neuromuscular blockade, anaesthetic complication (restoration of neuromuscular function), hypersensitivity reactions varying from isolated skin reactions to serious systemic reactions (i.e anaphylaxis), bronchospasm and pulmonary obstructive events. Severe hypersensitivity reactions can be fatal. Events associated with surgical procedures under general anaesthesia. Isolated cases of marked bradycardia and bradycardia with cardiac arrest. **Marketed by:** Merck Sharp & Dohme (NZ) Ltd, Newmarket, Auckland. Based on Medsafe approved Data Sheet, prepared 22 February 2016, available on www.medsafe.govt.nz.

MSD

bridion® sugammadex

www.acs.ac.nz