AUCKLAND CITY SYMPOSIUM

Saturday, April 17 2021

School of Medicine
The University of Auckland
New Zealand

Programme and Abstracts

Table of Contents

Table of Contents

Welcome

Welcome to the Auckland City Symposium 2021; Anaesthesia and the Brain. It was such a let-down to have to cancel this highly successful standing event on a perennially challenging topic in 2020, that we have decided to revamp and revisit this in 2021. We are very grateful to our international and local speakers for accommodating the changes both in 2020 and in 2021. ACS 2021 is also our first hybrid event, offering both in-person and online options and it will be exciting to see how this format works as an educational option.

Our theme is Anaesthesia and the Brain. Protecting the brain from perioperative neurologic injury is a goal for all anaesthetists. Perioperative insults around the time of surgery can result in long-term neurological complications, especially in vulnerable populations such as the young and old. Our international faculty includes Professor Kristin Engelhard, Associate Professor Lis Evered and Professor Kate Leslie. Together with our local faculty, they will cover a wide range of topics from traumatic brain injury, the Perioperative Brain Health Initiative, paediatric neurotoxicity as well as updates on the most recent scientific evidence about depth of anaesthesia, management of acute stroke and novel uses of anaesthetic agents.

We are grateful to our industry partners for their generous support of this meeting. And thank you to all our delegates for your patience and for continuing to support the Auckland City Symposium.

We hope you all enjoy the day.

Carolyn Deng
Kathryn Hagen

International Faculty

Professor Kristin Engelhard

Professor, University Medical Center of Johannes Gutenberg University, Mainz, Germany

Professor Kristin Engelhard is vice-chair of the Department of Anaesthesia at the Johannes Gutenberg University in Mainz, Germany. She is a member and former leader of many national and international societies like the Neuroaenesthesia Research Group of the German Society of Anaesthesiology and Intensive Care Medicine (DGAI) and the Society of Neuroscience in Anesthesiology and Critical Care (SNACC). Her scientific interest lies within neuromonitoring and neuroprotection after brain injury. She is editor of the Oxford Textbook of Neuroscience and Anaesthesiology and co-editor/author of Miller`s Anesthesia textbook.

Associate Professor Lis Evered

Principal Research Fellow, University of Melbourne and Head of Research, St Vincent's Hospital

AP Lis Evered has a BSc, MBiostat and PhD in neuroscience. Lis is AP of Neuroscience at Weill Cornell Medicine, NY; Scientific Head of Research in the Department of Anaesthesia and Acute Pain Medicine, St Vincent's Hospital, Melbourne and AP, University of Melbourne. She is Associate Editor for BJA and Senior Editor for Anesthesia & Analgesia, and a reviewer for many peer-reviewed journals. She is the recipient of more than $8M in competitive funding. Lis is Chair of the International nomenclature consensus working party which has revised postoperative cognitive disorders from research to clinical guidelines and is currently finalising research guidelines. Her main area of research interest is identifying the impact of surgery and anaesthesia on the cognitive trajectory of older individuals.

Professor Kate Leslie

Professor, Royal Melbourne Hospital

Professor Kate Leslie is head of research in the Department of Anaesthesia and Pain Management, Royal Melbourne Hospital, and honorary professorial fellow at the University of Melbourne and Monash University, Australia. She is a member of the ANZCA Clinical Trials Network and is a chief investigator of the Balanced, RELIEF, PADDI, ROCKET and Chewy studies. She sits on the editorial boards of Anesthesia and Analgesia, Anesthesiology and the BJA, and is an editor of Miller's Anesthesia textbook.

New Zealand Faculty

Speakers

Dr Doug Campbell	Specialist Anaesthetist, Auckland City Hospital
Professor Jamie Sleigh	Specialist Anaesthetist, Waikato DHB
Dr Andy van der Poll	Anaesthetist and Intensivist Auckland City Hospital
Professor Fiona McBryde	Professor, University of Auckland
Dr Alan Barber	Neurologist, Auckland City Hospital
Dr Chen Chen	Anaesthetic Fellow, Auckland City Hospital
Ms Kylie Head	Mediator & Consultant, On the Table

Programme

ACS 2021 Scientific Programme

Saturday, 17th April 2021

0800 Registration Desk Open – Exhibitor Area, Atrium, School of Medicine	
0825-0830 Welcome and introduction	Kathryn Hagen & Carolyn Deng
SESSION 1 Head Injury Chair: Kathryn Hagen	
0830-0900 Early management of traumatic brain injury: from roadside to ICU	Andrew van der Poll
0900-0935 New and novel treatments for TBI	Kristin Engelhard
0935-1005 Protecting blood flow to the 'selfish' brain in health and disease	Fiona McBryde
1005-1035 Morning Break – Exhibitor Area, Atrium, School of Medicine	
SESSION 2 Updates on Anaesthesia and the Brain Chair: Karen Pedersen	
1035-1110 Are anaesthetic agents neurotoxic for the developing brain?	Kristin Engelhard
1110-1140 The future of neuroprotection in the adult brain	Doug Campbell
1140-1210 Patient centered outcomes in brain health	Kylie Head
1210-1310 Lunch Break – Exhibitor Area, Atrium, School of Medicine	
SESSION 3 Brain Health Initiative Chair: Doug Campbell	
1310-1335 The EEG response to anaesthesia is the exercise tolerance test for the brain	Jamie Sleigh
1335-1405 Balanced study: Depth of Anaesthesia and Delirium	Kate Leslie
1405-1435 Maintaining brain health: Overlapping pathophysiology for multiple disorders?	Lis Evered
1435-1450 Panel discussion	
1450-1520 Afternoon Break – Exhibitor Area, Atrium, School of Medicine	
SESSION 4 All About Stroke Chair: Carolyn Deng	
1520-1545 Interventional management of stroke	Alan Barber
1545-1610 Anaesthetic management of stroke	Chen Chen
1610-1635 Neurovision: Covert Stroke in Non-Cardiac Surgery	Doug Campbell
1635-1650 Panel Discussion	
1650-1700 Future meetings / Meeting concludes.	
1700-1830 Drinks & Canapés – Exhibitor Area, Atrium, School of Medicine	

Early management of traumatic brain injury: from roadside to ICU

Andrew Van der Poll
Anaesthetist and Intensivist Auckland City Hospital

There is a significant burden of disease.
•In Aus/NZ approx 1000 cases of severe TBI (GCS <8) per year with approximately 30% mortality and overall 50% are severely disabled or dead. Most recent USA figures are from 2013 and indicated almost 300,000 hospitalisations and 56,000 deaths. In 2010 TBI costs were estimated at $76.5 billion USD. This is a pathologv that strikes the young- it is the leading cause of disability in the under 40 age group and occurs because of

1. Motor vehicle accidents
2. Falls
3. Assaults

In this overview update talk we will cover

- Primary vs. Secondary brain injury and what we can do to prevent both
- Emergency roadside management and the targets for immediate resuscitation and care
- Management in the emergency department including updates on the research into the use of tranexamic acid as well as optimum blood pressure targets
- Further management within the secondary or tertiary hospital including
    - How to manage the head injured patient for non-neurosurgery
    - Indications for invasive intracranial pressure monitoring and what we use at Auckland City Hospital
    - Ongoing issues that arise after head injury and how to treat them, including “Brain Orientated Intensive Care”, the role of Decompressive Craniectomy, Barbiturate coma and most importantly, prognostication

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New and novel treatments for TBI

Kirstin Engelhard

Professor, University Medical Center of Johannes Gutenberg University, Mainz, Germany

Traumatic brain injury (TBI) is the leading cause of death in the young adult and leads to severe disability, with a substantial burden of physical, psychiatric, emotional, and cognitive disabilities, which disrupt the lives of the patients and their families. The incidence of mild traumatic brain injury (TBI) is higher in New Zealand compared to European countries or the United States. Furthermore, children and young adults (< 35 years) suffer more often from TBI. To change this the New Zealand Accident Compensation Corporation (ACC) has launched the Traumatic Brain Injury Strategy and Action Plan (2017-2021) which has the aim to reduce the incidence, severity and impact of TBI. Severe TBI is mainly caused by road traffic accidents, assaults, falls, and domestic abuse.

Pathophysiology

The extent of the primary injury cannot be influenced by therapy and, therefore, prevention e.g. by helmets or airbags is the only way to avoid the damage. The primary brain damage triggers a cascade of pathophysiologic changes, which then lead to the secondary brain damage. During the first post-traumatic days the aim of the treatment is to minimize the expansion of the secondary damage in order to rescue the potentially salvageable intact brain tissue. Pathologic processes as elevated ICP (due to edema, hemorrhage, obstruction of cerebrospinal fluid flow), diminution of arterial inflow, and consequent reduction in CPP with resulting tissue hypoxia, and loss of cerebrovascular autoregulation increases secondary brain damage. Additionally, inflammation, massive release of excitatory neurotransmitters, apoptotic cell death, high lactic acid concentrations due to anaerobic glycolysis, depleted ATP-stores, increased intracellular Ca^{2+} concentrations, inflammation, generation of free radicals and proteolysis are only some of the identified parameters contributing to secondary brain damage.

Unfortunately, despite intense research and characterization of all these contributing mechanisms, no drug has been identified which can improve outcome after TBI in randomized, prospective clinical trials. This is most likely related to the complex pathophysiology of brain damage, the heterogeneous patterns of damage, and various preexisting diseases of the patients. TBI also influences systemic parameters like interferences of the autonomic nervous system (sympathetic discharge), inflammatory responses, endocrine dysfunctions, electrolyte imbalance, cardiovascular and respiratory disturbances, and impairment of the coagulation cascade. These systemic effects have to be monitored and immediately treated as they also contribute to a deterioration of secondary brain damage.

Novel neuroprotective drugs

Despite decades of experimental and clinical studies and a thorough characterisation of the mechanisms that extend the secondary brain injury no neuroprotective “magic bullet” has been identified. There exist many promising new therapeutic approaches including stem cell therapy, nanoparticles, and investigations on mitochondrial dysfunction, microglial activation, cerebral microcirculation impairment, and the effect of gut dysbiosis following TBI. Digital analysis of large datasets (big data analyses) including genomics, blood biomarkers, advanced MRI combined with clinical data of e.g. physiological variables, frailty, and preexisting diseases might have the power to improve clinical decision making and outcome of TBI patients. Another approach is the combination and individualization of multiple potentially neuroprotective drugs with a special focus on the adequate timing of administration according to the pathophysiology.

Neuroprotective interventions

As the brain has no tolerance for hypoxia, TBI patients demand an immediate and knowledgeable treatment according to the current guidelines. In lack of a neuroprotective drug all therapeutic strategies focus on optimization of the delivery of oxygen and glucose to the brain cells. This includes maintaining adequate CPP, controlling ICP, and optimizing oxygenation. Therefore, in the critical care setting the management of TBI patients should follow established protocols with a close monitoring of parameters including CPP, ICP, and oxygenation status. Clinical standard assessments like continuous measurement of arterial blood pressure, heart rate, and pulse oximetry in combination with monitoring of skin turgor, mucous membrane hydration status, urine output, and GCS have to be performed.

Cerebral perfusion pressure
CPP results from the difference of mean arterial pressure (MAP) minus ICP and should be kept between 60-70 mmHg. Aggressive attempts to maintain CPP above 70 mmHg with fluids and vasopressors should be avoided as this treatment increases the risk of adult respiratory failure. Arterial hypotension with a systolic blood pressure below 90 mmHg is strongly related to poor outcome and has to be avoided in the preclinical and clinical management of TBI patients. There exists a smooth U-shaped relationship between systolic or mean arterial blood pressure and outcome, without any evidence of an abrupt threshold effect. Therefore, the recommendation in the guidelines to maintain systolic blood pressure above 100 mmHg for patients 50 to 69 years old or ≥ 110 mmHg for patients 15 to 49 or > 70 years old, possibly need to be specified for an optimal systolic blood pressure of 135 mmHg. To calculate the CCP the pressure transducer for MAP must be zeroed at the level of the midbrain.

Intracranial pressure
Elevate ICP above 22 mmHg is associated with increased mortality and should be treated. When ICP is elevated the following measures should be performed: optimisation of positioning, osmotherapy, deep sedation with barbiturates or propofol, and ventricular drains. Hyperventilation reduces cerebral blood volume (CBV) and ICP due to its vasoconstrictive effect, but at the same time hyperventilation leads to a mismatch between oxygen delivery and oxygen consumption. Therefore, hyperventilation is only a temporizing measure until other ICP-lowering measures are available. Decompressive craniectomy has been a strategy to lower ICP, but unfortunately, this intervention increases the number of patients surviving in vegetative state or with a severe brain damage but not of those with good outcome.

Oxygenation and ventilation
Patients with GCS ≤8 should be intubated and ventilated. An adequate oxygenation (PaO_2 > 80 mmHg) should be achieved, if necessary by using a positive end-expiratory pressure (PEEP) up to 15 mbar or kinetic therapy (prone position).

Sedation
For sedation of a TBI patient drugs with a short context-sensitive half-life like propofol should be used to facilitate the daily control of consciousness of the patient. Care should be taken to screen patients for the so-called propofol infusion syndrome which can occur when high-dose propofol is used over several days. Most of the barbiturates and benzodiazepines have a longer half-life and are, therefore, less suitable. Low-dose inhalational anesthetic can also be used, while in higher concentrations volatile anesthetics possess a direct vasodilatory effect, which increases CBV and, thereby, ICP. Ketamine was suggested to be contraindicated in TBI patients due to perceived risks of intracranial hypertension, but in intubated and ventilated patients ketamine has no adverse effect on ICP. At the same time Ketamine has several favorable effects like reduced need of supplementary vasopressors and narcotics, activation of bowel movement, and bronchodilatation. Narcotics, like sufentanil, fentanyl, and remifentanil have no negative effects on ICP as long as MAP is maintained. Muscle relaxants can be used in TBI patients, with a potential exception of succinylcholine, which possibly increases ICP. Nitrous oxide and etomidate should not be used in patients after severe TBI.

Additional interventions
Feeding of the TBI patients to attain basal caloric replacement between the fifth and the seventh day after trauma is recommended. Enteral feeding using a transgastric jejunal tube should be started as soon as possible. Early dilatatory tracheotomy facilitates the weaning of the patients and reduces mechanical ventilation days.
Up to 25% of patients with isolated TBI develop deep vein thrombosis (DVT) with the risk of pulmonary embolism. Low molecular weight heparin or low-dose unfractioned heparin should be used in combination with mechanical prophylaxis, despite the increased risk of expansion of intracranial hemorrhage.
The incidence of early posttraumatic seizures (during first week after TBI) can be reduced by phenytoin. As these early posttraumatic seizures do not influence outcome, this prevention is not obligatory. Late posttraumatic seizures are not susceptible to prophylactic interventions.

Protecting blood flow to the ‘selfish’ brain in health and disease

Fiona McBryde
Professor, University of Auckland

Protecting blood flow to the 'selfish' brain in health and disease

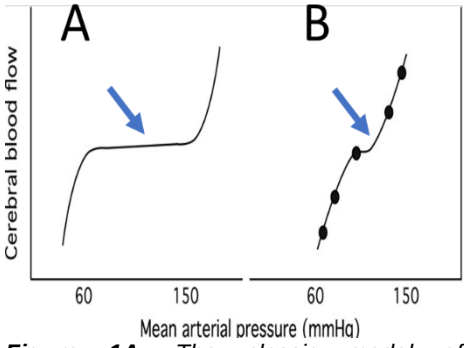

***Figure 1A**: The classic model of autoregulation based on Lassen's 1959[3] meta-analysis, pooling data between-subjects over a range of normal and abnormal resting blood pressure (BP). **1B:** Cerebral blood flow (CBF) as BP is varied within each (healthy) individual subject. Note the narrow plateau (arrowed).*

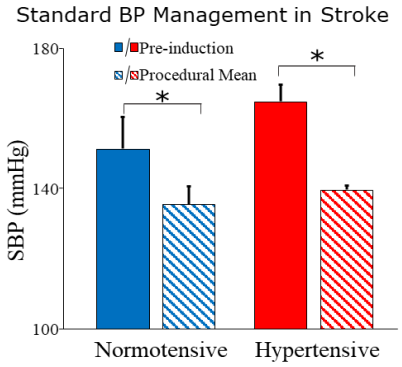

Figure 1: SBP before and during GA for endovascular thrombectomy in large-artery ischemic stroke patients, with (Hypertensive, n=16) or without (Normotensive, n=10) a prior history of hypertension. Note that the procedural mean is significantly lower than the pre-induction SBP in both groups, particularly for Hypertensive patients. *(Zhang, Billing, Campbell & McBryde, unpublished data, 2019).*

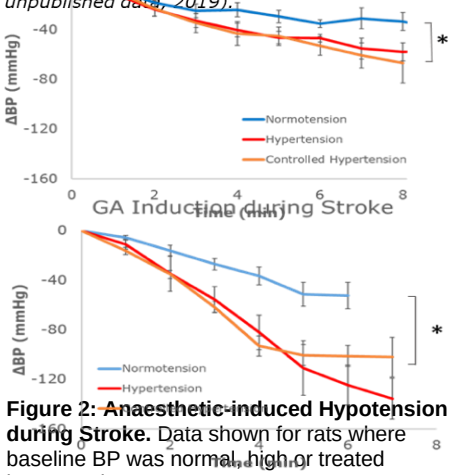

Figure 2: Anaesthetic-induced Hypotension during Stroke. Data shown for rats where baseline BP was normal, high or treated hypertension.

The brain is our most energy-expensive organ, with a constant and unrelenting demand for blood flow to meet its metabolic needs. Classic dogma holds that blood flow to the brain is largely protected by an in-built mechanism called "cerebral autoregulation". This states that as perfusion pressure falls, the blood vessels in the brain rapidly and automatically dilate (or constrict with an increase in pressure) to maintain cerebral blood flow. The textbook view is that this powerful mechanism operates to keep cerebral blood flow constant across a wide range of perfusion pressures, termed the 'autoregulatory plateau' (Fig 1A). An important but often overlooked point is that the classic curve shown in Fig 1A is NOT the response to varying pressure within individuals, but represents a mean steady-state measurement from multiple groups of subjects with pathologies resulting in abnormal resting blood pressure. We and others have shown that even in healthy individuals the dynamic relationship between cerebral blood flow and blood pressure has little or no plateau as previously thought (Fig 1B), meaning that moment-to-moment cerebral autoregulation in fact may be highly limited. How then does the brain protect itself from fluctuations in perfusion?

Our ‘selfish brain’ hypothesis predicts that reductions in cerebral perfusion trigger the brain to demand an increase in systemic blood pressure, thereby restoring the supply pressure and hence flow to the brain. This is consistent with observations in ischemic stroke patients, where the vast majority (>80%) show a pronounced increase in blood pressure. A key question is where should blood pressure be set in patients during interventional procedures such as endovascular thrombectomy?

The standard of care in New Zealand is for endovascular thrombectomy to be performed under general anaesthesia. Given the prevalence of hypertension as a risk factor for stroke, understanding how different BP targets affect subjects with varying levels of pre-stroke BP is of particular importance. A recent analysis of stroke patients undergoing endovascular thrombectomy at Auckland City Hospital, showed that standard BP management during GA resulted in BP below the pre-induction level even where there was no history of hypertension (ΔSBP: -15±4mmHg); this fall was much greater in patients with known hypertension (ΔSBP: -25±2mmHg). It is not yet known whether this reduction in BP may affect outcome.

In our preclinical rodent model of stroke, we have observed that inducing GA during occlusive stroke produces hypotension, which is greatly exacerbated in hypertensive rats, regardless of whether or not BP was treated and controlled prior to stroke (Figure 2). Taken together, these data suggest that the administration of anaesthesia during ischemic stroke may require particular care and more precise haemodynamic management than for other procedures.

Are anaesthetic agents neurotoxic for the developing brain?

Kirstin Engelhard

Professor, University Medical Center of Johannes Gutenberg University, Mainz, Germany

Animal Data

Various experimental studies in animals have shown that general anaesthetics are potentially toxic to the developing brain. By inducing apoptosis or interfering with neurogenesis anaesthetic exposure during a critical period of neuronal development can have significant impact on the neurocognitive functions later in life. In animal studies three main factors affect the toxicity of anaesthetics. The first is the timing of exposure, because the neurotoxicity of anaesthetics occur more often in the early stage of brain development. The second factor is the frequency and duration of anaesthetic exposure. Various in-vitro and in-vivo animal experiments have shown that frequent and long anaesthetic intervention are correlated with an increased neurotoxicity. Thirdly, numerous animal experiments reveal a clear dose-dependent element to toxicity. Increasing the dose of anaesthetics increases the number of apoptotic neurons, the degree of developmental impairment, cellular differentiation and synaptogenesis.

Retrospective Clinical Studies

Meanwhile it is well proven that anaesthetic agents do harm the developing brain of rodents and non-human primates. To answer the question whether these data can be transferred to paediatric patients many retrospective clinical studies have been performed. They investigate the interrelation between early exposure to anaesthesia during the first three to four years of life to learning and behaviour abnormalities in adulthood. Most of the studies have been performed in the USA, Australia, or Europe. The data for the studies were drawn from different birth cohorts or registries They explored e.g. the association between exposure to anaesthesia for inguinal hernia repair under the age of three and learning disabilities. Some of the studies from the USA and Australia detected a correlation between single or multiple exposure to anaesthesia and an increase in the incidence of learning disabilities. In contrast, several studies from Europe showed no such relationship. For instance, a Swedish study compared more than 33,000 children who were operated on within the first four years of their lives with those who have never been operated. The authors compared the academic performance in a standardised nationwide school test in the ninth grade in both groups, and were able to prove that gender or the maternal educational level influences the outcome ten times more than an operation. A twin research study is a possible method to exclude genetic difference. A retrospective analysis of the Dutch Twin Registry compared monozygotic twins of which one of the two children was exposed to general anaesthesia under the age of three years. They found no difference in the incidence of learning disabilities between the exposed and the unexposed twin. However, the incidence of learning disabilities was higher in pairs of twins in whom one underwent anaesthesia compared to the set of twins of where neither was exposed. The authors speculated that there might be a vulnerability about these twin pairs, rendering them more susceptible to conditions requiring anaesthesia such as diseases of the middle ear or herniotomy. Interpreting retrospective studies is generally difficult. One has to take into account that many of the data of the retrospective studies were generated in the late 1970´s and early 1980´s. At that time, the quality of paediatric anaesthesia was significantly different from the present day. Commonly used anaesthetics such as halothane were much more cardiodepressant than modern agents, hypotonic infusion therapy often led to hyponatraemia, and the lack of warming technologies led to severe hypothermia. Furthermore, the identification of disturbances was difficult, because measuring and monitoring respiratory, haemodynamic and metabolic changes was challenging or even impossible as capnometry, pulse oximetry and non-invasive arterial pressure monitoring did not enter clinical practice to complement the clinical expertise of anaesthetists until the 1990's.

Prospective Clinical Data

During the last years the results of three major prospective randomized multicentre clinical studies, the PANDA, MASK and GAS studies, have been published.

1) The PANDA study (Pediatric Anesthesia and Neurodevelopment Assessment). This was a multicentre study which examined the long-term effects of anaesthesia on cognitive function in children exposed to anaesthesia for inguinal hernia repair up to the age of 36 months. The neurodevelopmental and cognitive functions were tested at the age of eight and 15 years and were compared with the results of non-anaesthetised siblings. The study revealed that there was no correlation between anaesthetic intervention and IQ score.

2) The GAS study (General Anesthesia and Spinal): This study compared the effects of anaesthesia on neurodevelopmental outcome and apnoea in infants undergoing inguinal hernia repair up to the age of six months. They were randomly assigned to receive either general or spinal anaesthesia. The children then underwent developmental testing at the age of two years and neurodevelopmental and intelligence testing at the age of five. The authors concluded that slightly less than 1 h of general anaesthesia in early infancy does not alter neurodevelopmental outcome at age 5 years compared with awake-regional anaesthesia in a predominantly male study population.

3) The MASK study (Mayo Safety in Kids): This was a collaborative cohort study involving researchers from the Mayo Clinic and National Center for Toxicological Research in the USA. Children in Rochester, Minnesota, who received one or more anaesthetics before the age of three years were compared to with children no anaesthetic exposure. They use an extensive battery of neurocognitive tests, including the 'operant test battery', which is already evaluated in children and non-human primates. These authors also found that anesthesia exposure before age three years was not associated with deficits in the primary outcome of general intelligence.

In conclusion, after 20 years of concern and controversy it seems to be proven that anaesthetic agents do not harm the developing brain. The animal studies were alarming, but the human evidence overwhelmingly suggests that any effect of well-conducted paediatric anesthesia is insignificant or non-existent. Nevertheless, there are still many editorials, commentaries, and opinions stating that more studies are needed to characterise the potential mechanisms of anaesthetic neurotoxicity, develop alternatives to the current anaesthetic agents or even avoid any operation in children younger than four years. This is aggravated by the warning of the American Food and Drug Administration and the Australian Therapeutic Goods Administration that the use of anaesthetic agents in children younger than three years or in pregnant women may affect the development of children's brain.

At the same time the undue focus on the safety of anaesthetic drugs detracts the attention from other factors which possibly have a much higher impact on the outcome in paediatric anaesthesia. It is well known that the cardiovascular, central nervous and respiratory systems of the premature or newborn baby are extremely sensitive and vulnerable to haemodynamic and metabolic changes and derangements. We still do not even know which target values for blood pressure, blood glucose, oxygen or carbon dioxide partial pressure can be considered safe for anaesthetised children in this age group. Therefore, it might be rather the unexperienced anaesthetist than the anaesthetic agent who is a threat to developing brain. To investigate these potentially harmful factors during paediatric anaesthesia in large clinical trials (APRICOT and NECTARINE studies) a group of leading paediatric anaesthetists have formed the "Safe Anaesthesia for Every Tot" (SafeTots) initiative.

<u>Practical advice to clinicians</u>

There is no data to support the omission of anaesthesia or analgesia in newborn babies, infants or small children. As known from infants who received a circumcision without anaesthesia and analgesia, the pain and stress reaction induced by the surgery reduces the pain threshold in these children for several months. Animal studies revealed that painful stimuli in the absence of anaesthesia enhance pain perception, behaviour and learning disabilities and brain damage.

In practice, therefore, adequate anaesthesia and/or sufficient pain therapy during an indicated surgical procedure or painful examination are essential. In addition, transient or profound disturbance of physiologic parameters like hypotension, hypocapnia, hypoglycaemia or hypothermia, should be avoided as these changes might also affect neurodevelopment. To reduce the amount of anaesthetic agent required, a balanced anaesthetic technique including intraoperative multimodal pain therapy with local/regional anaesthetics, non-opioid analgesics and opioids is recommended.

In addition, anaesthetists, paediatricians and paediatric surgeons should define clear indications for surgical or non-surgical procedures under anaesthesia in the first years of life if postponement is inadvisable e.g. orchidopexy because of a non-descended testicle. Further, several surgical interventions

with multiple anaesthetics can be avoided if there is the possibility of performing more than one surgical procedure during the same anaesthetic.
Parents are usually well informed because of the rapid availability of information via the internet. The main problem with this source of information is the lack of reliability and validity. There should be an empathic and objective discussion with the parents to make sure they are properly informed, and two key points should be communicated:
· Anaesthesia is not an end in itself. It is necessary and indispensable for the indicated operation. To omit adequate sedation and/ or analgesia has damaging effects for the child.
· There are barely any indications that a competent and clinically well-performed anaesthesia with modern, short-acting anaesthetics has negative consequences such as cognitive developmental problems or learning disabilities.

The future of neuroprotection in the adult brain

Doug Campbell
Specialist Anaesthetist, Auckland City Hospital

Neuroprotection is the preservation of neuronal function and structure. Therapies have long been sought to prevent or slow the progression of disease in many central nervous system disorders including traumatic brain injury, stroke and spinal cord injury. Despite the differences in injury and disease process there are common mechanisms of injury including oxidative stress, excitotoxicity and neuro-inflammation. Neuroprotection has been demonstrated repeatedly in animal studies but, unfortunately, over a thousand human trials of drugs and techniques have been tested with very limited success to date. So, is the future for neuroprotection in the adult brain bleak?

These failures have improved our understanding of the problems. Many earlier animal studies suffered from poor experimental design. This has been improved by the introduction of the STAIR criteria to standardise and improve animal research into neuroprotection. However, even now most animal experiments are performed in young, male animals with no comorbidities that does not reflect clinical reality. Many clinical trials explored neuroprotection in time windows beyond 6 hours where there is very little evidence for neuroprotection. Furthermore, experimental paradigms where there is no reversible component to the injury are likely to be unproductive of permanent middle cerebral artery occlusion.

Many anaesthetists discuss neuroprotection in the context of traumatic brain injury, an injury with multiple mechanisms and associated injuries confounding our ability to find effective treatments. The era of percutaneous stroke intervention is delivering high volumes of patients with relatively homogenous, severe and partially reversible brain injury to our hospitals. This presentation will focus on discussions of potential therapies that are currently available, can be administered in the pre-hospital or hyperacute treatment window and will be familiar to anaesthetists with expertise in using the agents and techniques. These potential neuroprotective agents will be discussed solely in the context of acute ischaemic stroke as the current best brain injury model in clinical medicine.

All the major classes of anaesthetic agents have repeatedly been shown to be neuroprotective in animal models. Stroke models are our current best bet for discerning the different effects of propofol or inhalational agents. This should become an active area of anaesthesia research but their use will be restricted to the hospital setting because of their sedative properties.

NMDA antagonism is a major focus for neuroprotection research as glutamate release and subsequent excitotoxicity is a major component of the injury pathway. Ketamine is a major focus of research and could potentially be used in a pre-hospital setting. Xenon is neuroprotective and an active area of anaesthesia research. It is impractical to use xenon in the hyperacute setting because of limited supply, but argon, another noble gas is ubiquitous, cheap and has potential as a neuroprotectant.

Hypothermia remains an active area of interest as the only drug or technique with proven neuroprotectant properties in adults. Localised brain cooling by cold arterial injectates or by cooling skull caps is being investigated. Finally, nitric oxide, another inhaled gas has shown promise.

In summary, stroke is our current best injury model to identify neuroprotective strategies. Agents and techniques such as propofol, inhalational anaesthetics, ketamine, argon, hypothermia and nitric oxide are cheap, effective in animals, can be easily administered in the hyperacute or pre-hospital setting. Anaesthesia research in this area has the potential to find anaesthesia drugs with therapeutic effects.

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Patient centered outcomes in brain health

Kylie Head

Mediator & Consultant, On the Table

Abstract

Understanding the experience of patients and their whānau provides insight into health care as well as being a crucial step toward partnering with patients to drive improvement. This elevation in the conversation on the 'healthcare experience' resonates now more than ever. People are not just passive participants in a care transaction or simply recipients of care, rather they are partners in a care conversation, who must be acknowledged and cared for as people in a health care experience. Their voices, what matters to them and the expertise they bring, regardless of the side of the care equation they sit, must now be part of the overall solution.

What was revealed in this work is the experience that people in healthcare have matters to them in significant ways. And in recognition of this Stroke will be a major healthcare driver nationally, now and into the future.

Introduction

To live up to the mantra of 'nothing about me, without me' in health care, we need to understand the current experience of patients and their whānau as well as partner with them to drive improvement in health care. Listening to patient's experience and understanding is the first step. Taking the next step of engaging patients and whānau in improving care delivery is leading to a range of outcome benefits for healthcare organisations committed to a 'client focus'.

I've been working (part-time) as a Consumer Advisor for the National Stroke Network (NSN), Northern Regional Alliance (NRA) and Health Navigator since 2015, and the landscape has changed considerably in 5 years. When I started out patients advocating for their care was a relatively new concept. We have now moved from advocacy to patient and family activation.

All the terms used creates misunderstanding - "consumers", "patient- and family-centred care", "patient and family engagement", "patient experience", "people-centred care", and "lived experience" - many weren't really used 10 years ago. Now we have patient-whānau advisors working on quality improvement, and working in a very sophisticated capacity. We're making good progress in caring about the patient experience, and bringing in patient-whānau advisors and community members as partners.

We're doing a good job working with people as advisors, but we're still struggling to figure out how to partner with patients when they're actively sick.

Distinction between "people-centred care" and "co-designed care"

I respect and believe in patient and whānau-centred care. However, can we technically decide what this means without a 'patient' in the room? In theory, we can be patient-centred by thinking about how we deliver care from a patient-centred perspective.

On the other hand, you can't co-design without a person (with lived experience), whānau, or community members as a part of the process. There is a big distinction. The co-design language is new enough that people use the term to mean different things.

In the way the project group I worked with practiced co-design, whether it was at the bedside or when working with people on policy, a person (with lived experience), whānau, or community was part of the process. We used a model that has been successfully implemented nationally called Experience-Based Co-design. It's a very structured process where issues are identified by speaking with people, patients' whānau, and community members.

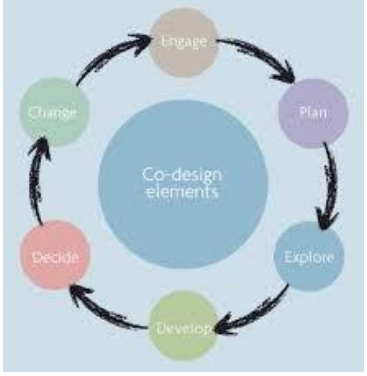

Figure 1. Healthcare Codesign
(https://www.healthcodesign.org.nz/)

Co-design means not coming in with assumptions. It means coming with an open mind and saying, "You tell us where we need to start. What is the most painful part of this process for you? What do we need to change?". This is in contrast to what we see with 'consumer'-advisory councils. For example, where the staff come to the council assuming that they know what bothers 'consumers' or what matters most to them: "We already know what the problem is. Now we want you to tell us what to do about it." When we start with our assumptions, we may or may not hit on the most important or meaningful issues for patients.

Taking this approach can result present requests.

That's the beauty of it. And there may be requests, but there are often simple solutions to those requests. The key priorities that patients and whānau typically describe tend to be things that aren't outside the box. Some real examples include things like hanging clocks in ED rooms, re-designing welcome booklets, and changing the time housekeepers empty trash containers on the unit. None of these are very costly or dramatic changes, but patients and whānau have said they mattered. It's also very likely that ideas like these wouldn't be identified or implemented without the co-design process.

Case Study:

Stroke Hyper Acute Process - First Phase

In June 2015, a proposal was endorsed by the Regional Governance Group, to develop a high level plan to develop a regional approach to hyperacute stroke care. There are two principle reasons for developing the regional hyperacute pathway:

1. The first was related to the international results of a series of clot retrieval studies and, as Auckland City Hospital (ACH) had participated in one of these studies, it already had some capacity to provide a service. The concern had been that ACH did not have capacity to provide a full 24-hour service and there was an imperative to develop a plan to manage what was expected to be an increasing referral demand. In addition to this, it was clear that volumes of referrals would be low and therefore effectiveness of a 24-hour service would be compromised unless thrombolysis rates increase (patients must have had thrombolysis to be eligible for clot retrieval).
2. Northern Region thrombolysis rates fell well below international best practice standards and with substantial improvement, not only patients who underwent clot retrieval would significantly benefit but also those who were treated with thrombolysis.

By improving our services to meet world's best standards, we believe that up to an additional 100 people each year could be free of significant disability after stroke. Improved hyperacute pathway processes could deliver two thirds of that gain.

Stroke Hyper Acute Process - Second Phase

Undertaken to capture patients' experience of care during the second phase of the Northern Region hyperacute stroke pathway and clot retrieval service implementation at Auckland City Hospital.

The Northern Region implemented the first phase of the regional afterhours hyper acute stroke pathway in August 2017 with patients living in the Waitakere Hospital catchment. This meant that anyone in West Auckland who had a stroke afterhours i.e. after 4pm on a week day, all weekend and public holidays, were diverted to Auckland City Hospital instead of Waitakere Hospital.

The second phase, which commenced on 3 September 2018, extended the afterhours hyperacute stroke service to all people living in the metro-Auckland area. The second phase concluded on the 31 August 2019 having seen a total of 133 diversions and a total of 230 percutaneous stroke interventions (or stroke clot retrieval) in the 12 months that it ran.

Scope

The scope of patient interviews was to get an understanding of how patients who had a stroke afterhours, experienced their care from the moment of symptom onset, their diversion or transfer to Auckland City Hospital for treatment, treatment/intervention at Auckland City Hospital, their transfer back to their local hospital, and the post intervention care at their local hospital.

The scope also extended to stroke patients who were transferred to Auckland City Hospital for percutaneous stroke intervention (PSI). These patients could be transferred at any time of the day as the PSI service operates 24/7 out of Auckland City Hospital.

Patient experience of their stroke rehabilitation was not in scope for these interviews although in many cases, interviewees did touch on aspects of their transition back to home, ongoing recovery and rehabilitation.

Findings from Patient Experience of Care Interviews

The experiences reported by patients or their carers identified 5 touchpoints in their care on the hyper acute stroke pathway. A touchpoint is any point of contact patients have with the service and is regarded as a key moment that shapes a person's overall experience.

Regional Hyperacute Stroke Pathway Touchpoints

The power of consumer narratives and stories, and the opportunity to include patients and their whānau as partners in decision making provided the opportunity to consider what truly mattered for consumers experiencing stroke.

Listening to patient narrative stories about their experiences provided insight into expectations of care, in a manner that healthcare professionals found interesting.

Ultimately, at the centre of really understanding patient values and preferences is establishing a relationship between clinicians, patients and patients' whānau grounded in strong communication and trust.

Many aspects go into making patient initiatives successful. Whether through training staff or including new technologies, each approach requires organisational commitment to work effectively.

Patient initiatives start with involving patients and their whānau to understand what makes them sick, what they need to stay healthy, and what they would like to do if their conditions get worse. It means motivating and empowering patients to work with clinicians - to be active participants in their care by asking questions, knowing their medications and medical history, bringing friends or relatives to appointments for support, and learning about care that may be unnecessary.

To be successful during an era of changing health reform, providers must give patients consistent messages about how to manage their care at all touch points. Providers have the opportunity to create two-way communications that are consistent and personalised to the needs of the individual and the specific point of interaction in their care. Patient stories which span an entire continuum of care are useful in identifying the many ways that patients interact and how issues outside of their care management, i.e. psychosocial, financial, impact how and when they choose to interact with the system.

Patient experience fits into the overall healthcare picture more today than it ever has. As population health management, accountable care and healthcare reform mature, the efficacy of those efforts will depend more and more on how well providers can integrate the design of patient experience and empowerment into the expanding care continuum.

Given the pressures and constraints that so many clinicians face, how can they build a trusting relationship with patients when they often have so little time with them?

We have to activate our patients. Activated patients who are able to take some responsibility for their health and health care can help take some of the burden off of those delivering the care. As we continue work at involving patients and families and improving patient experience, we also have to help those patients and families become more involved members of the health care team.

The ideal experience was grounded in caring, professional and helpful encounters. These positive experiences have significant consequences for healthcare

Recommendations for Improvement

The following recommendations have been made based on the information gathered from interviews.

People

Communication and trust

- All communication - verbal and non-verbal - must be done in a manner which is respectful, kind, compassionate, inclusive, professional and culturally sensitive.

Process

- Explanation should be given by ambulance staff to all patients and their families/ whānau as to why they are being transferred to Auckland City Hospital instead of their local hospital for treatment.
- All delayed transfers back to patients' home DHB should be communicated by Auckland City Hospital staff to patients and carers, and reasons for delay given. Communicate what is going well and also what isn't going so well.
- Review transfer pathways between Auckland City Hospital, St John and the different DHBs regularly.

- Inform patients and their families/whānau if the expectation is that they will be charged for the ambulance trip back to patients' home.
- Consider if patients in recovery should be transferred back to their local DHB in the early hours of the morning (between 12am and 7am).
- Reasons for delayed admission to the stroke unit should be investigated to understand the root cause and improved upon.
- Before discharge, patients should be briefed on the reason for their stroke and what might have caused it and what they can do to prevent its occurrence.

Place

- Monitor and reduce overcrowding in treatment and shared recovery areas as it can be disconcerting for patients.
- Monitor noise levels on the stroke wards to ensure that patients in recovery are not disturbed by other patients or their visitors.

Additional Observations

These are not strictly in the scope of the hyperacute stroke pathway interview but were raised by patients and/or their carers in the course of the interview.

- **Availability of more intensive community OT/ PT/ SLT**

Patients indicated they wanted to receive more intensive rehabilitation in the community

- **Rehabilitation to include supporting emotional needs**

Patient talked about their "rehabilitation" as activities centred around physical therapy received for restoring functions like – walking, talking, strength, speech. But there were other issues which were raised including:

  - Anxiety about recurrence of stroke
  - Wanting information around the effects of the disease on sex and intimacy post-stroke
  - Anxiety around driving

- **Rehabilitation closer to home**

Patients indicated a preference to be rehabilitated closer to home due to reasons including convenience for families to visit, parking challenges at the Auckland rehabilitation site.

- **Support for carers to be a part of patients' rehabilitation**

Some spouses acknowledged they were finding it difficult coping with the changes to their partners.

Closing thoughts from the regional stroke consumer lead

Healthcare organisations say they should be patient-centric or provide empathy and compassion, but what patients want are the tangible actions that exemplify those practices. While being listened to is a means of expressing empathy and compassion, patients don't want healthcare providers to say they are compassionate, they want healthcare providers to listen and act.

A consumer's experience is important to them; it is about being *acknowledged and engaged* in the healthcare process. How people are treated matters.

The need for *open and clear communication*, courteous and respectful treatment reinforces that to consumers, it is how they are engaged and treated in healthcare that matters.

The most important factor in having a good experience for consumers was the importance of being listened to. This reinforces how consumers see themselves as active partners in the healthcare process that they have something to say and to contribute and a voice that desires to be heard AND acted on.

The ideal experience was grounded in caring, professional and helpful encounters. These positive experiences have significant consequences for healthcare.

While efforts have been established to tackle the divisions of care delivery in healthcare, the consumer sees it all as one experience and so healthcare organisations need to consider how they address care in a continuous way.

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The EEG response to anaesthesia is the exercise tolerance test for the brain

Professor Jamie Sleigh
Specialist Anaesthetist, Waikato DHB

Over the last decade there has been an increasing body of work and interest in predicting and preventing postoperative disturbances in cognitive function. The most common occurs over hours to days and is usually termed postoperative delirium. The role of anesthesia in the aetiology of this condition is not fully understood as yet, but it is clear that certain intraoperative EEG patterns are strongly associated with the development of postoperative delirium. In particular a strong alpha (10Hz) oscillation is protective of delirium and a propensity for the burst suppression pattern – at low or moderate doses of hypnotic drugs – is associated with more postoperative delirium. Inclusion of these patterns of intraoperative EEG in multivariable predictions of delirium risk, tends to result in loss of the usual demographic risk factors e.g. age, preoperative cognition scores etc, from the predictive model. This has given rise to the idea that these patterns accurately reflect the "brain age" of the patient, and thus serve as biomarkers for patients with a 'fragile' or 'vulnerable' brain. Whilst it is unclear as to how much we can influence the outcome by using different drugs e.g. dexmedetomidine, or more carefully titrating the doses of routine hypnotic drugs to EEG patterns, the use of the intraoperative EEG to identify patients at high risk of postoperative cognitive disturbances would enable rational use of appropriate postoperative resources and management plans to ameliorate postoperative delirium consequences and severity.

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Balanced study: Depth of anaesthesia and delirium

Professor Kate Leslie

Professor, Royal Melbourne Hospital

In this talk I will speak about the magnitude of the problem of postoperative delirium, preventing delirium using the BIS monitor, and offer some advice and conclusions.

Postoperative delirium (POD) is part of a spectrum of disorders that have recently been described as "perioperative neurocognitive disorders (PND)".[1] POD is an acute onset fluctuating change in mental status characterized by a reduced awareness of the environment and disturbance of attention. It is common in the first seven days postoperatively, particularly in older adults. POD is associated with adverse outcome including increased risk of death in elderly patients. However there is inadequate research about how to prevent or treat it including:[2]

- Association of postoperative delirium with poor outcome not clearly defined
- Anti-inflammatory studies in animals not replicated in humans
- Effects of risk factor modification and lifestyle improvement not tested
- Use of anticholinesterases not explored perioperatively
- Choice of anaesthetic not proven to influence outcome
- Effect of depth of anaesthesia uncertain

As more than 300 million people have surgery every year and an increasing number of them are elderly and at risk of delirium, more action is required.

The pathophysiologic basis for delirium is uncertain. Anaesthesia and surgery are associated with immunosuppression, inflammation and hypotension. These may be greater with deep anaesthesia.[3] More than nine studies investigated the association between deep anaesthesia and delirium. Their methodologies have varied widely:

Paper	n	Surgery	Tests	Follow-up	Groups
Wong 2002[4]	68	TJA	Chart	3 days	Routine vs. BIS 50-60
Sieber 2010[5]	114	#NOF	CAM	2 days	BIS=50 vs. BIS>80
Jildenstal 2011[6]	450	Eyes	MMT	1 month	Routine vs. AEP
Ballard 2012[7]	74	Non-cardiac	MMSE	1 year	Routine vs. BIS
Chan 2013[8]	902	Non-cardiac	CAM	3 months	Routine vs. BIS
Radtke 2013[9]	1,155	Non-cardiac	DSM-IV	3 months	Routine vs. BIS
Whitlock 2016[10]	310	Cardio-thoracic	CAM	10 days	ETAC vs. BIS
Sieber 2018[11]	200	#NOF	CAM	5 days	OAA/S 0-2 vs. OAA/S 3-5
Wildes 2019[12]	1,232	Major surgery	CAM	5 days	Routine vs. BS avoidance

Most studies have reported more delirium in the deep group than the light group, sometimes at a statistically significant level. Only one has shown more delirium in the light group than the deep group (Wildes 2019):

Paper	n	Surgery	Groups	Delirium
Wong 2002	68	TJA	Routine vs. BIS 50-60	1 vs. 0
Sieber 2010	114	#NOF	BIS=50 vs. BIS>80	40% vs. 19%
Jildenstal 2011	450	Eyes	Routine vs. AEP	16 vs. 2
Ballard 2012	74	Non-cardiac	Routine vs. BIS	89% vs. 58%
Chan 2013	902	Non-cardiac	Routine vs. BIS	24% vs. 16%
Radtke 2013	1,155	Non-cardiac	Routine vs. BIS	21% vs. 17%
Witlock 2016	310	Cardio-thoracic	ETAC vs. BIS	28% vs. 19%
Sieber 2018	200	#NOF	BIS=50 vs. BIS>80	39% vs. 34%
Wildes 2019	1,232	Major surgery	Routine vs. BS avoidance	23% vs. 26%

We conducted a delirium sub-study of the Balanced Anaesthesia Study at 8 participating sites in Australia, China and the United States. The manuscript from this paper is currently under review at a journal. The results will be presented at the Auckland City Symposium.

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Maintaining brain health: Overlapping pathophysiology for multiple disorders?

Associate Professor Lis Evered

Principal Research Fellow, University of Melbourne and Head of Research, St Vincents Hospital

The first few slides of this presentation will cover my time in New York at Weill Cornell Medicine where I commenced on 1st January 2020. This will centre around the impact of the pandemic on an unprepared epicentre.

Perioperative Neurocognitive Disorders (PND) is a term which encompasses the preoperative and postoperative cognitive and functional impairment/decline temporally associated with anaesthesia and surgery, including frailty and any known or unknown vulnerabilities brought to the operating room by the patient. Vulnerabilities associated with older individuals including preoperative cognitive impairment (often subtle and undiagnosed) and frailty, which increase the risk of PND. Higher frailty scores preoperatively are associated with an increased risk of PND at 12 months postoperatively. Additionally, an assortment of co-morbidities is likely to burden older individuals presenting for elective surgery putting them at further risk. An important factor to consider is that cognitive impairment does not occur in isolation but is associated with functional disability over the longer-term, increased risk of dementia, institutionalisation and death.

I will focus briefly on postoperative delirium, one of the perioperative neurocognitive disorders. I will define the two types of delirium (hyperactive and hypoactive) and the current non-pharmacological recommendations to try to prevent an episode of POD and therefore prevent the long-term sequelae. In addition, I will present some recent work demonstrating long-term psychosocial effects of delirium following cardiac surgery following a recent qualitative analysis of interviews with patients 3 years post-surgery. These long-term themes include depression, trauma, loneliness and awareness of cognitive and functional decline.

Possible anaesthesia effects will be briefly covered revealing little evidence of a contribution to PND by anaesthetic agents. Our current neuroinflammatory / neuronal damage hypothesis will be presented and discussed in terms of PND and other inflammatory injury/disease that may overlap pathophysiological pathways, such as COVID-19 and acute traumatic brain injury. This work suggests surgery may play a more critical role than anaesthetic agents in the development of PND.

To date, with the assistance of biomarkers and with the elimination of other factors, the most supported hypothesis suggests that peripheral inflammation leading to neuroinflammation followed by downstream neuronal damage is the most likely pathophysiology underlying these cognitive and functional changes.

Multi-component interventions for the prevention of PND will be discussed in the context of routine care pathways as part of promoting ‘Brain ERAS’ to be part of every surgical care pathway. The PROTECT trial is a NHMRC funded non-pharmacological multi-component trial investigating strategies from preoperatively to 12 months postoperatively to prevent PND and will be briefly outlined. Resources for preventing delirium will be identified.

PND will be discussed in the context of COVID-19 ‘long-haulers’ and what we can learn from the overlap between the these and other disorders. The overlap between long-term symptoms will be addressed and suggests a possible common pathway. This may offer insights and opportunities for further research and preventive strategies. Biomarkers may hold the key to insight in this regard, and further biomarker research, particularly over the long-term is critical to preventing the long-term poor outcomes of both PND and inflammatory diseases such as COVID-19.

In summary, this presentation will discuss PND including delirium and associated poor outcomes; current evidence for mechanisms and possible overlap with other inflammatory disorders such as COVID-19 ‘long-haulers’ with overlapping risk factors.

Interventional management of stroke

Professor Alan Barber
Stroke lead, Auckland City Hospital, Professor of Clinical Neurology, University of Auckland.

Stroke is a leading cause of mortality and the most common cause of long-term adult disability worldwide. In New Zealand, stroke affects around 9,000 people per year with an estimated 50,000 people living with the effects of stroke in the community. The personal, social and economic costs of treatment and post-stroke care are substantial, with an annual cost of $NZ750 million. A large proportion of the overall cost results from the long-term disability that can follow stroke. One in five people with stroke will require long-term institutional care costing $NZ50,000 per year. Despite improved stroke prevention over the past decade, the population is increasing and ageing leading to growth in the overall stroke burden.

After many years of therapeutic nihilism there are treatments that reduce the extent of cerebral infarction and improve outcomes. These are intravenous thrombolysis with either alteplase, or in specific circumstances tenecteplase, and endovascular thrombectomy. Intravenous thrombolysis was first shown to be effective in a randomised controlled trial in 1996 but wasn't in widespread use until the mid- to late-2000s. One of the Ministry of Health's key performance indicators is that each district health board should be thrombolysing at least 12% of ischaemic stroke patients.

Endovascular thrombectomy is a more recent therapeutic advance. It has been provided at Auckland City Hospital since 2011 when patients were enrolled in the Australia/New Zealand EXTEND IA study, one of the pivotal endovascular thrombectomy studies published in 2015. Treatment was extended to patients living in Northland and the Midland Region in 2017. Christchurch and Wellington have also been providing thrombectomy to the Southern and Central Regions since this time.

Endovascular thrombectomy is a very effective therapy and is cost effective. Meta-analyses of randomised controlled trials have shown that in patients treated within 6 hours of stroke onset, there is clear evidence that endovascular thrombectomy improves functional outcome. When compared with what was considered standard therapy at the time of trials (intravenous thrombolysis), for every 100 patients treated with endovascular thrombectomy there are;

1. Forty more patients with functional improvement
2. Twenty three more who achieve functional independence
3. Sixteen fewer who require hospital level care
4. Four fewer deaths
5. No increased risk of symptomatic intracerebral haemorrhage.

These benefits extend to those treated 6-24 hours after stroke where treatment selection requires evidence of salvageable 'penumbral' brain tissue on CT or MRI perfusion imaging. When compared with standard therapy, for every 100 patients with evidence of penumbral brain tissue treated with thrombectomy 6-24 hours after symptom onset, there are;

1. Thirty two more who achieve functional independence.
2. Four fewer deaths
3. No increased risk of symptomatic intracerebral haemorrhage.

Regional networks of hub (treating) and spoke (referring) centres are required to provide this complex treatment. This is because endovascular thrombectomy requires close collaboration between ground and flight ambulance, emergency department, neurology, radiology and anaesthetic teams. Of the 225 patients treated in Auckland in 2020, only 15% were from the Auckland District Health Board (DHB) area with others transported from the two other Auckland DHBs by ambulance, and 30% flown by helicopter from Northland and the Midland regions encompassing a line north from Taranaki to Tairawhiti.

There are currently three New Zealand hub treating hospitals; Auckland (Northern and Midland Regions), Wellington (Central Region) and Christchurch (Southern Region). It is envisaged that the number of hub hospitals will increase with time, with Waikato Hospital likely being the next to start treating patients from the Midland region.

Australian Acute Stroke Management Guideline recommendations regarding endovascular thrombectomy (summarised).

1. For patients with ischaemic stroke caused by a large vessel occlusion in the internal carotid artery, proximal middle cerebral artery (M1 segment), or the basilar artery, endovascular thrombectomy should be undertaken when the procedure can be commenced within six hours of stroke onset (Strong recommendation).
2. For patients with ischaemic stroke caused by a large vessel occlusion in the internal carotid artery, proximal middle cerebral artery (M1 segment), or with tandem occlusion of both the cervical carotid and intracranial large arteries, thrombectomy may be considered when the procedure can be commenced between 6-24 hours after a patient was last known to be well, if clinical and CT or MRI perfusion mismatch indicates the presence of salvageable penumbral brain tissue (Strong recommendation).
3. For patients with ischaemic stroke caused by occlusion in the M2 segment of the middle cerebral artery, endovascular thrombectomy may be considered based on favourable individual patient, and CT/MRI angiography and perfusion imaging, factors (Consensus based recommendation).
4. Eligible stroke patients should receive intravenous thrombolysis while concurrently arranging endovascular thrombectomy, with neither treatment delaying the other (Strong recommendation).
5. Endovascular thrombectomy should be performed by experienced neurointerventionists with recognised training in the procedure.

Anaesthetic management of stroke

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Endovascular thrombectomy (EVT) is a proven technique for treatment of anterior circulation large vessel occlusion acute ischaemic stroke that reduces patient morbidity and mortality[1]. It is a time critical procedure and thus patient management requires a clear multidisciplinary approach between the neurology, interventional radiology and anaesthesia teams. The main goals of anaesthetic management are to reduce time to recanalisation of blocked cerebral vessel and to offer physiologic protection of the ischaemic penumbra prior to recanalisation.

The involvement of anaesthesia team occurs from activation of the stroke team, when a patient is confirmed on computerised tomography (CT) angiography to have a lesion amenable to endovascular treatment. The patient may have received thrombolysis, especially if they were transferred from another hospital in the region.

Anaesthetic challenges for EVT
Patient evaluation is often by the time sensitive nature of EVT. Clinical records from patients domiciled out of the treating district health board (DHB) are often not available. There is limited ability to assess the patient for medical comorbidities, fitness for anaesthesia, fasting status and airway concerns. This is partly as a result of symptomology from the stroke itself; patients can present with aphasia, dysphasia, dysarthria or decreased Glasgow Coma Score (GCS). Collateral history from the stroke team should be sought if there was no direct handover. Best interest consent is often required from the treatment team. Post-operative disposition to higher level care should be considered/arranged if patient is significantly comorbid or if there were neurologic concerns about extubation e.g. low GCS pre-operatively or combative patient.

Familiarity of the location and environment of the neuro-interventional suite is also an important factor to consider. It is often located away from the main operating rooms and therefore arrival of anaesthetic assistance may be delayed. The positioning of radiology equipment such as portable X-ray “C-arm” limits access to the patient. The airway, circuit, monitoring and intravenous lines must be meticulously secured and bundled away from moving equipment. Radiation exposure for the anaesthetist is also a consideration.

Controversies in anaesthetic management
There are now multiple papers comparing general anaesthesia (GA) to conscious sedation (CS) for EVT. Potential benefits of GA include definitive airway protection, patient immobility, control of ventilation, and no risk of conversion to GA during the case. Benefits of CS include shorter arrival to groin puncture time, less haemodynamic effects and direct mental state monitoring. Although initial observational studies suggested that patients had better outcomes after CS, subsequent randomised controlled trials (RCT) demonstrated that patients who had GA for EVT had at least the same or better functional neurological outcomes[2, 3]. A meta-analysis published in 2021 of four RCTs demonstrated that patients who received GA in specialised neuroanaesthesia centres had superior recanalisation rates (GA 86.2% vs CS 74.6%; OR 2.14, 95% CI 1.26-3.62; *P*=0.005) and better functional outcome at 3 months (GA 49.3% vs CS 36.6%; OR 1.71, 95% CI 1.13-2.59, *P*=0.01), with no significant difference in intracerebral haemorrhage or death[4].

The key physiological parameter affecting patient outcome during EVT appears to be blood pressure (BP). Physiological studies have demonstrated impaired cerebral autoregulation during acute ischaemic stroke, resulting in pressure-dependent perfusion to ischaemic penumbra. Evidently, lower BP is likely to result in decreased cerebral blood flow to the penumbra, leading to increased size of cerebral infarction[5]. While the exact BP target during EVT is still under investigation, both hypotension (systolic BP < 140mmHg)[6] and hypertension (BP > 220/120mmHg or > 185/110mmHg after thrombolytic therapy)[7] appear to worsen patient outcome due to increased infarct size and the risk of haemorrhagic transformation respectively. In all four RCTs comparing GA vs CS, BP management was strictly controlled with target systolic BP of >140mmHg in both intervention groups[2]. Previously published observational studies did not report or control for BP and the results favouring CS may have been confounded by more hypotension in the GA group[2-4]. Research examining the potential therapeutic benefit of augmenting BP (systolic BP to 170 ± 10 mmHg) compared to standard care (systolic BP 140 ± 10mmHg) prior to recanalisation is currently underway with the multicentre MASTERStroke

RCT (MAnagement of Systolic blood pressure during Thrombectomy by Endovascular Route for acute ischaemic STROKE)[8].

While both propofol and volatile anaesthetic agents reduce cerebral metabolic demand ($CMRO_2$), propofol is thought to maintain cerebral autoregulation thereby reducing cerebral blood flow (CBF), while the volatile agents increase CBF due to impaired cerebral autoregulation. There is controversy regarding which maintenance anaesthetic agent is superior for EVT and there are no large RCTs published in this domain. Local retrospective observational data from Auckland suggest that patients who had maintenance of anaesthesia with propofol had increased odds of functional independence at 3 months (odds ratio=2.65; 95% confidence interval, 1.14-6.22; P=0.03) and a nonsignificant trend towards reduced mortality (odds ratio=0.37; 95% CI, 0.12-1.10; P=0.07). This was despite the apparent low usage of propofol (59 patients, 19% in the cohort of 313 patients) compared with sevoflurane[9].

Maintaining normothermia and active treatment of hyperthermia (> 38°C) appears to confer better outcomes in patients undergoing endovascular thrombectomy, with studies thus far failing to show benefit of cooling stroke patients[6, 7, 10]. Published local data from Auckland analysing 458 patients between March 2011 to June 2019 found significant decrease in functional independence and increase in mortality with increasing body temperature both during and after EVT[10].

Currently there are few recommendations for intraoperative end-tidal/arterial CO_2 management during EVT. The 2019 American Heart Association/American Stroke Association guideline recommends moderate hyperventilation in cases of acute severe neurological decline due to increased intracranial pressure as a bridge to definitive therapy. There were no recommended targets for CO_2 during EVT[7]. A previous retrospective observational study published in 2014 by Takahashi et al. found that patients with low CO_2 appear to have worse outcomes after stroke intervention, and maintaining low normal CO_2 appears to cause least harm[11].

While hyperglycaemia is known to worsen brain ischaemia in experimental models, a review of 258 patients in Spain by Laredo et al. was not able to demonstrate an association between severe hypoperfusion on pre-procedure CT imaging and glucose levels. They did however demonstrate that elevated glucose pre-thrombectomy may be associated with an increased risk of parenchymal haematoma post EVT, which is a significant predictor of poor outcome[12]. On the other hand, treatment of hypoglycaemia is indicated to maintain blood glucose above 3.3mmol/L[7].

Post-operatively it is routine for the patients to be extubated for neurological assessment and admitted to the stroke ward for ongoing care. However, high dependency care or intensive care with ongoing ventilation may be required particularly if patient had pre-operative GCS < 8 or had been combative, or if the procedure was difficult and full recanalisation was not able to be achieved with ongoing concerns for haemodynamic and/or neurologic stability. Currently there have not been major trials studying post-procedural haemodynamic targets in patients who have achieved full, partial or failed recanalisation.

To summarise, during anaesthesia for EVT, there is good evidence to support the avoidance of hypotension (systolic BP < 140mmHg), and current evidence supports that patients having EVT under GA have at least equivalent if not better functional outcomes than patients having CS. However strong evidence for many key components of physiological management appears to be lacking. This includes the choice of anaesthetic maintenance agent, temperature, end-tidal/arterial CO_2 and blood glucose parameters. Further studies are required to provide definitive guidance.

Current Practice at Auckland DHB

In light of the evidence presented, the current guidelines for anaesthesia for EVT at Auckland DHB places emphasis on reducing the time to recanalisation with blood pressure management as key principles[13]. For this GA with endotracheal intubation is the preferred anaesthetic technique for most patients unless otherwise indicated (e.g. difficult airway, significant cardiorespiratory comorbidities precluding GA in a patient who is able to cooperate and follow instructions). Systolic BP targets are > 140mmHg and < 220mmHg, or < 180mmHg if the patient had been thrombolysed. Pre-induction invasive arterial BP monitoring is preferred given the need to avoid post-induction hypotension, and this may be inserted by the anaesthetist or by the interventional radiologist through awake placement of the femoral sheath. Choice of anaesthetic agents and drugs (opioid, muscle relaxant, vasopressors) is at the discretion of the attending anaesthetist and there are variations in practice. There are ongoing research and local quality improvement projects to improve patient outcomes after EVT at Auckland DHB.

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Neurovision: Covert stroke in non-cardiac surgery

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Overt stroke is an uncommon but devastating complication of surgery and anaesthesia. Incidence ranges from 0.3-2% depending on the age, comorbidities and surgery type. Postoperative stroke has much worse disability and mortality than in the non-surgical setting. Covert strokes are smaller strokes that have subtler signs and symptoms that may be masked in the postoperative period. Covert strokes may be more common than overt stroke. The incidence of covert stroke in noncardiac surgery and clinical implications were not known.

The NeuroVision trial aimed to explore this knowledge gap. A total of 1114 patients age over 65 years with an expected hospital stay of 3 days or more having surgery under general or regional anaesthesia were recruited. Baseline cognitive assessment, twice daily delirium assessments, MRI between day 3 and 10 and 1 month and 1 year were performed. The incidence of covert stroke was 7% with a doubling of the risk of delirium from 5% to 10% (P=0.02) and an increase of cognitive decline at 1 year from 29% to 42% (P=0.006). There was no difference between surgery or anaesthesia type. Most covert strokes were embolic in origin.

The POISE-2 trial has helped delineate our understanding of the risks of aspirin cessation or continuation on cardiac outcomes in the perioperative period. POISE-2 did not look at delirium, cognition or covert stroke as an outcome. We do not have good data to predict those at highest risk beyond traditional medical risk factors and high risk surgery eg carotid or cardiac or data on the relative risk and benefit of aspirin continuation or cessation in this setting. The cogPOISE study and NeuroVISION-2 study may provide further information.
Proposed strategies for advancing pre-operative stroke care and research include advanced risk prediction modelling, cerebrovascular reserve mapping and plaque-stenosis identification. Intra-operative care and research should be targeted at identifying critical cerebral flow thresholds. Postoperative care should include regular delirium and neurological assessment.
Covert stroke is common and consequential for delirium, cognitive impairment and subsequent disability. It should be discussed as one of the common, serious complications of surgery when weighing the benefits and risks of proceeding with surgical management. Currently, the optimum management regime for prevention, diagnosis or management in the perioperative setting is unclear.

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