# AQUA 2018

## Annual Queenstown Update in Anaesthesia

Programme and Abstracts

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# Abstracts

AQUA
ANNUAL QUEENSTOWN UPDATE IN ANAESTHESIA

JAFA
Joint Anaesthesia Faculty Auckland

New Zealand Society of Anaesthetists

# Welcome to Queenstown

Dear Colleague,

Welcome to AQUA 2018 and our 10th birthday!

As always we have put together updates on a broad range of anaesthesia updates including paediatrics, airway management, regional anaesthesia and fluid management. This year, in order to expand interest and depth to the programme, we have invited a number of specialists from outside the specialty of anaesthesia. Dr Fiona Stewart will be providing an overview of recent developments in cardiology, Dr Teddy Wu will discuss the exciting field of interventional management of acute stroke, Colin McArthur will give an ICU update on topics relevant to anaesthetists and Professor James Isbister will discuss recent developments in transfusion medicine. We also have Ben Johnston, Chief Medical Officer of Air New Zealand presenting on managing aviation emergencies.

We have expanded our workshop offerings for 2018 with two satellite workshops (Regional Anaesthesia and TTE). In addition we have two workshops on Friday afternoon (Anaphylaxis and TEG/Major Haemorrhage).

The AQUA conference dinner on Friday night is at Walter Peak and features the TSS Earnslaw boat trip across Lake Wakatipu. The AQUA BBQ is on Saturday evening at Coronet Peak. We will be screening the second Bledisloe cup rugby match with kick-off at 7:35pm.

A big thanks to our sponsors for continuing their support of our meeting in 2018.

**Neil MacLennan**
**Kerry Gunn**

AQUA Conveners

# Social Programme

## THURSDAY, 23 AUGUST 2018

17:00 – 19:00

**Registration & Welcome Function**
**Exhibitor Area, Pounamu Room, The Heritage**

**Browns Fitting Service – Foyer outside the Pounamu Room, the Heritage**

## FRIDAY, 24 AUGUST 2018

18:00 onwards (you need to be at Steamer Wharf, 88 Beach Street, Queenstown, no later than 17:45)

**AQUA Conference Dinner**

**TSS Earnslaw & Walter Peak Gourmet BBQ Dinner**

## SATURDAY, 25 AUGUST 2018

18:00 onwards

**AQUA BBQ & Bledisloe Cup Rugby Function**

**Coronet Peak Base Building, Queenstown**

# Faculty

| | |
|---|---|
| Dr Shay McGuinness | Specialist Anaesthetist, Auckland City Hospital |
| Dr Teddy Wu | Consultant Neurologist, Christchurch Hospital |
| Dr Colin McArthur | Intensivist, Auckland City Hospital |
| Professor James Isbister | Emeritus Consultant Physician, Royal North Shore Hospital, Sydney and Clinical Professor of Medicine, University of Sydney |
| Dr Peter Cooke | Specialist Anaesthetist, Auckland City Hospital |
| Dr Jane Thomas | Paediatric Anaesthetist , Starship Children's Hospital / Pain Medicine Specialist, The Auckland Regional Pain Service |
| Dr Ben Johnston | Chief Medical Officer, Aviation and Occupational Health, Airline Operations and People Safety, Air New Zealand |
| Dr Gemma Malpas | Specialist Anaesthetist, Auckland City Hospital |
| Dr Kelly Byrne | Specialist Anaesthetist, Waikato Hospital |
| Dr Fiona Stewart | Consultant Cardiologist, Auckland City Hospital |
| Dr Dean Bunbury | Specialist Anaesthetist, Middlemore Hospital |
| Dr Jonathan Albrett | Specialist Anaesthetist, Taranaki Base Hospital |
| Dr Felicity Pugh | Specialist Anaesthetist, Auckland City Hospital |
| Dr Rob Fry | Specialist Anaesthetist, Auckland City Hospital |

## Anaphylaxis Workshop

| | |
|---|---|
| Dr Peter Cooke | Specialist Anaesthetist, Auckland City Hospital |
| Dr Setareh Ghahreman | Specialist Anaesthetist, Auckland City Hospital |

## TEG and Major Haemorrhage Workshop

| | |
|---|---|
| Dr Kerry Gunn | Specialist Anaesthetist, Auckland City Hospital |
| Chris Finlay | Technical Specialist, Point of Care Testing, Lab Plus |

# Scientific Programme

## Friday, 24 August 2018

**Session 1** — Icon Conference Room

| | | |
|---|---|---|
| 07:55 | Welcome and Introduction | Dr David Kibblewhite, President NZSA |
| 08:00 | RELIEF study; what does it mean for fluid management? | Dr Shay McGuinness |
| 08:30 | Interventional Management of Acute Stroke | Dr Teddy Wu |
| 09:00 | Intensive Care Medicine Update | Dr Colin McArthur |
| 09:30 | Transfusion Medicine Update | Professor James Isbister |
| 10:00 | Morning Break | Pounamu Room – Exhibitor Area |

**Session 2** — Icon Conference Room

| | | |
|---|---|---|
| 10:30 | Allergy and Anaesthetics | Dr Peter Cooke |
| 11:00 | Paediatric Pain Management | Dr Jane Thomas |
| 11:30 | Managing Aviation Emergencies | Dr Ben Johnston |
| 12:00 | Close – lunch packs and fresh fruit available for pick up | Mackenzies Restaurant |

**AQUA Workshops** *(please ensure you attend the workshop you registered for)*

| | | |
|---|---|---|
| 13:00 | ▪ Anaphylaxis Workshop (1) | Icon Conference Room |
| | ▪ TEG & Major Haemorrhage Workshop (1) | Pounamu Room – Exhibitor Area |
| 15:00 | ▪ Anaphylaxis Workshop (2) | Icon Conference Room |
| | ▪ TEG & Major Haemorrhage Workshop (2) | Pounamu Room – Exhibitor Area |

## Saturday, 25 August 2018

**Session 3**

Icon Conference Room

| | | |
|---|---|---|
| 08:00 | Update on Airway Management | Dr Gemma Malpas |
| 08:30 | Update on Regional Anaesthesia | Dr Kelly Byrne |
| 09:00 | Cardiology Update | Dr Fiona Stewart |
| 09:30 | Genetics for Anaesthetists | Dr Dean Bunbury |
| 10:00 | Morning Break | Pounamu Room – Exhibitor Area |

**Session 4** — Icon Conference Room

| | | |
|---|---|---|
| 10:30 | Crisis Management for Training for First Year Doctors | Dr Jonathan Albrett |
| 11:00 | Sustainability in the Operating Room | Dr Felicity Pugh |
| 11:30 | Towards Retirement: Managing those last years of practice | Dr Rob Fry |
| 12:00 | Close – lunch packs and fresh fruit available for pick up | Mackenzies Restaurant |

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.

# RELIEF study; what does it mean for fluid management?


## Dr Shay McGuinness

Cardiothoracic Intensive Care Unit, Auckland City Hospital, New Zealand


**Introduction**

Use of intravenous fluids during and after major surgery is ubiquitous and done for a variety of valid physiological reasons including to correct for preoperative fasting and other fluid deficits, anesthesia-induced vasodilation, hemorrhage, 'third space' losses, enhance tissue oxygen delivery and to maintain urine output. Traditional IV fluid regimens in abdominal surgery deliver up to 7 liters of fluid on the day of surgery which can lead to oedema and a 3- to 6-kg weight gain and may be associated with pulmonary, renal and wound complications. [1-4] Several small trials have shown that a more restrictive fluid regimen led to fewer complications and shorter hospital stay [5,6] and these have resulted in consensus statements that recommend a restrictive approach to fluid administration, particularly as a component of Enhanced Recovery after Surgery (ERAS) programs.[7-8]

To address the lack of high quality evidence in this area we conducted the Restricted versus Liberal Fluid Therapy for Major Abdominal Surgery (RELIEF) Trial[9] which compared a restrictive with a more traditional (liberal) fluid regimen for abdominal surgery. Our primary hypothesis was that a restrictive fluid regimen for adults undergoing major abdominal surgery leads to reduced complications and improved disability-free survival when compared with a liberal fluid regimen.

**Methods**

In a pragmatic, international, trial, we randomly assigned 3000 at-risk patients undergoing major abdominal surgery to a restrictive or liberal intravenous fluid regimen during and up to 24 hours after surgery. The primary outcome was disability-free survival through to 1 year after surgery. Secondary outcomes included 30-day acute kidney injury (AKI), a composite of septic complications, surgical site infection or death, and 90-day renal replacement therapy. Patients were eligible to be included if they were undergoing major abdominal surgery with an expected duration of at least 2 hours and a hospital length of stay of at least 3 days. At least one patient-level risk factor had to be present (including age>70yrs, diabetes, heart disease, renal impairment or obesity)

**Results**

3000 patients were randomised at 47 centres in 7 countries between May 2013 and September 2016 of these 2983 were included in the analysis and we had outcome data at 1 year available in 2901.

During and up to 24 hours after surgery, 1493 patients in the restrictive group received a median (IQR) 3.7 (2.9 to 4.9) litres compared with 6.1 (5.0 to 7.4) litres in 1490 patients in the liberal group (P<0.001). Disability-free survival at 1 year was 81.9% in the restrictive fluid group and 82.3% in the liberal fluid group (hazard ratio for death or disability 1.05, [95% CI, 0.88 to 1.24], P=0.61). The rate of AKI was 8.6% in the restrictive group and 5.0% in the liberal group (P<0.001). The rate of septic complications or death was 21.8% in the restrictive group and 19.8% in the liberal group (P=0.19). Rates of surgical site infection (16.5% versus 13.6%, P=0.024) and renal replacement therapy (0.9% versus 0.3%, P=0.048) were higher in the restrictive group.

**Discussion**

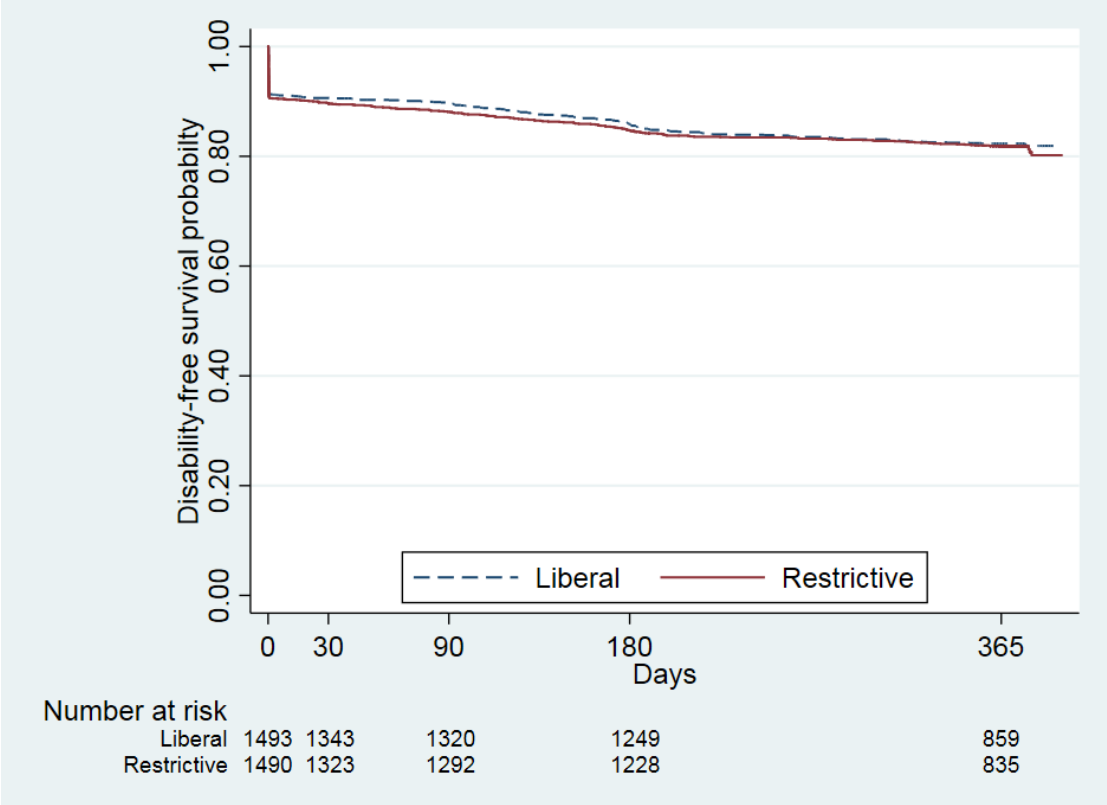
IV fluid regimens for abdominal surgery have been classified into restrictive (less than 1.74 litres per day), balanced (1.75 to 2.75 litres per day), and liberal (greater than 2.75 litres per day).[10] RELIEF study patients assigned to restrictive fluid therapy received a median 1.7 litres intraoperatively and a further 1.9 litres in the first 24 postoperative hours.[9] Patients in the liberal fluid group received 3.0 litres during surgery, and a further 3.0 litres in the first 24 postoperative hours. In previous studies intraoperative 'restrictive' fluid replacement varied from 1.0 to 2.7 liters compared with 2.8 to 5.4 liters in 'liberal' regimens.[11] Current recommendations suggest avoiding a greater than 2.5 kg weight gain – this was achieved in a majority of patients in our study, including those in the liberal fluid group. [12-14]

The RELIEF study findings should not be used to support excessive IV fluid administration. Rather, they show a modestly liberal fluid regimen is safer than a restrictive regimen. There is a belief that fluid-induced edema impairs wound healing. In contrast, we identified a higher rate of surgical site infection in the restrictive group, possibly because of wound and/or anastomotic hypoperfusion.

New Zealand patients included in the restrictive arm of the trial faired particularly badly if managed with a restrictive fluid approach, with a hazard ratio for death or disability of 5.59 (1.61-19.5, p=0.007). This is possibly a spurious result due to the low numbers of patients included (94 in total), although it is interested to reflect that the reason for low patient numbers from New Zealand was a lack of clinician (both anaesthetist and surgeon) equipoise with many stating that the liberal arm was "too wet".

**Conclusions**

In patients having major abdominal surgery, a restrictive fluid regimen did not improve disability-free survival through to 1 year after surgery. However, a restrictive fluid regimen increased the rates of AKI, renal replacement therapy use, and surgical site infection. Accordingly, our findings support the preferential use of a moderately liberal approach to perioperative fluid therapy.




Probability of freedom from death or persistent disability in the restrictive and liberal fluid groups through to 1 year after surgery.

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# Interventional Management of Acute Stroke

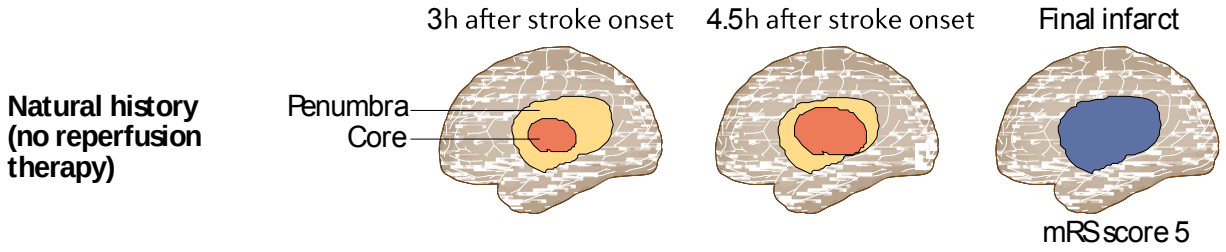

## Dr Teddy Wu

Department of Neurology, Christchurch Hospital, New Zealand


**Endovascular Clot Retrieval for Acute Ischaemic Stroke**

**Background**:
Acute ischaemic stroke results usually from occlusion of an intracranial vessel causing nutritional deficiency to the area of the brain supplied by the blood vessel. Within the region of ischaemic brain are regions of 'ischaemic penumbra', which is viable brain tissue at risk of infarction without timely reperfusion, in addition to regions of already infarcted brain tissue term the 'ischaemic core'. Without timely reperfusion there is progressive demise of the viable ischaemic penumbra resulting in enlargement of the ischaemic core leading to functional disability or death (figure). Reperfusion strategies target the salvage of the ischaemic penumbra.

*Figure. The concept of ischaemic penumbra and core with infarct progression in the absence of reperfusion. From J Baron 2018 (1).*




**Intravenous thrombolysis**
Intravenous thrombolysis with alteplase is effective at reducing disability compared to standard medical treatment when given within 4.5 hours of stroke symptom onset (2). However the effect diminishes exponentially with time with most benefit seen within the first 60 to 90 minutes of symptom onset time. However intravenous alteplase has limited efficacy in patients with proximal intracranial occlusion, with recanalization achieved in approximately 30% of patients with proximal middle cerebral artery occlusion and 10% or less in those with terminal internal carotid artery or basilar artery occlusion (3). Patients with large proximal intracranial occlusion generally have a high risk (~70-80%) of permanent severe disability or death in the absence of recanalization.

**Endovascular clot retrieval**
In late 2014 and 2015, the results of 5 multi-centre multi-national randomised controlled trials demonstrating efficacy of endovascular clot retrieval (ECR) in treatment of anterior circulation large vessel occlusion were published in quick succession in the New England Journal of Medicine (4-8). Endovascular clot retrieval resulted in successful removal of large intracranial clots in between 60 to 80% of patients and functional independence at 90 days was achieved in approximately 50% of patients in otherwise devastating stroke (3). In these studies most patients had to have the angiogram commenced within 6 hours of symptom onset thus limiting the therapeutic window.

The limitation of the 6-hour time window is obvious, particularly for patients who live remote to a primary intervention stroke centre. In many rural hospitals in Australia and New Zealand it may not be possible to have groin puncture started within 6 hours of symptom onset time following unavoidable delays in inter-hospital transfer. However experience from the Australia-New Zealand multicentre EXTEND-IA study, which was only trial to select patients for ECR on basis of favourable perfusion pattern, suggest that treatment for patients beyond 6 hours may be possible provided viable brain issue (penumbra) can be demonstrated (4). Furthermore, the individual patient meta-analysis also suggests the therapeutic window extends beyond the 6-hour time window (9).

Recently two randomised controlled trials in DEFUSE 3 and DAWN examined the effect of ECR on outcome in the so-called extended time window. Both these trials utilised advanced imaging in the selection of patients for treatment. In the DAWN trial ECR was performed in patients with a small ischaemic core with a severe clinical deficit and an intracranial arterial occlusion between 6 and 24 hours of last known well time. In DAWN 49% of treated patients achieved functional independence compared with 13% of patients receiving best medical treatment which is stroke unit care (10). The DEFUSE 3 trial utilised perfusion imaging similar to the criteria set out in EXTEND-IA and performed ECR in patients 6-16 hours from last known well time. DEFUSE 3 also demonstrated overwhelming efficacy in favour of thrombectomy patients with 45% functional independence rate compared with 17% in the medical treatment arm (11). There is a shift in the selection paradigm from a time-based approach to a 'tissue' based approach utilising advanced imaging or a radiological time clock. Furthermore, there is suggestion that treatment of patients beyond 24-hours may be possible (12) and may well be topic of future randomised trials.

**Areas of uncertainty**

**General anaesthesia versus conscious sedation**

An area of controversy is the choice of anaesthetic approach during ECR. Procedures may be performed more smoothly under general anaesthesia due to lack of patient agitation and may reduce procedural time. However associated delays in setting up general anaesthesia and the hypotension associated with anaesthetic induction may exacerbate ischaemic injury. To date 3 single centre randomised controlled trials (AnStroke, GOLIATH and SIESTA) (13-15) have been reported. All these trials comprised of relatively small sample size (AnStroke n=90, GOLIATH n=128, SIESTA n=150) each with different primary end points. In terms of 90-day functional outcomes only SIESTA reported a difference in rates of functional independence favouring the general anaesthesia group while no difference in independent outcome was reported in GOLIATH and AnStroke (13-15). More recently the individual patient meta-analysis from the HERMES collaboration (which consisted of patients from recently published ECR trial) comprised of 797 patients (236 had general anaesthesia) reported reduced odds of favourable functional outcome with general anaesthesia when compared with conscious sedation (16). However there was no standardisation of intra-operative haemodynamic control or anaesthetic agents used across these studies,- factors which may account for the conflicting results reported to date. Further multi-centre studies with standardised intraoperative anaesthetic management are needed to address this question.

**Treatment of patients without pre-existing functional disability**

The initial positive trials of ECR and the two 'extended' time window trials required patients to have strict pre-stroke functional independence with modified Rankin Scale score of 0-1. However, this would exclude patients who have mild to moderate baseline disability from previous stroke or other medical issues but otherwise are living at home independently. For example, a patient with mild chronic right leg weakness from a previous stroke, who had ceased driving since a stroke, who required a walking frame but was living at home independently would have been ineligible for inclusion into trials. In the recently published EXTEND-IA TNK study which demonstrated superiority of low dose tenecteplase (0.25mg/kg) than alteplase at achieve recanalization of large intracranial clot before clot retrieval had a broader inclusion criterion of baseline modified Rankin Scale score 0-3 (17). This study demonstrated 57% of treated patients had returned to their baseline functional capacity at 90 days. Selection criteria for treatment varies from centre to centre but the experience from the EXTEND-IA TNK study suggest that patients with pre-existing mild to moderate disability with an intracranial occlusion should be considered for clot retrieval.

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# Intensive Care Medicine Update


## Dr Colin McArthur

Department of Critical Care Medicine, Auckland City Hospital, New Zealand


Two important studies of transfusion practice have been influential. TRICS III (*NEJM* 2017, 377(22):2133-2144) randomised 5243 adults at moderate to high risk undergoing cardiac surgery to a restrictive (<75g/L) or liberal (<9.5 g/L in OR or ICU, <8.5 g/L in the ward) transfusion strategy. The composite primary outcome of death, myocardial infarction, stroke or new dialysis requirement to day 28 (censored at hospital discharge) occurred in 11.4% of the restrictive group and 12.5% of the liberal group (P<0.001 for non-inferiority). Red cell transfusion was less common in the restrictive group, and there were no differences in the individual components of the composite outcome (half of the events were new MI) or secondary outcomes between the groups. In the TRANSFUSE study (*NEJM* 2017; 377(19):1858-1867) 4994 patients undergoing first transfusion in intensive care were randomly assigned to receive either the freshest available compatible red cells or the standard issue of oldest compatible red cells for that and subsequent transfusions. In the freshest available group, mean storage duration was 11.8 days compared to 22.4 days in the standard group. Transfusion volume was a median of 2 units (IQR 1 – 4) in both groups. All-cause mortality after 90 days was 24.8% in the freshest available group and 24.1% in the standard group (P=0.57). New bloodstream infections were more common with the freshest-available transfusion (5.0% vs 3.6%). In sub-group analysis treatment effect varied by severity of illness (P=0.03 for heterogeneity) with significantly lower mortality transfusing older red cells in patients with higher than median APACHE III scores (OR 1.18, P=0.05). Following this and earlier studies confirming the safety of older red-cell transfusion (ABLE, RECESS and INFORM), the New Zealand Blood Service is currently consulting on a change to their Fresh Blood policy to not provide red cells with <14 days storage for adult patients undergoing cardiac surgery with cardiopulmonary bypass.

The use of steroids in sepsis, and septic shock in particular, has long been an area of controversy. Two recent trials have sought to provide clarity on the issue. In the "ADRENAL" study (*NEJM* 2018; 378(9):797-808), undertaken by the ANZICS CTG, 3800 patients with septic shock receiving mechanical ventilation were randomised to receive 200mg/day of hydrocortisone or blinded placebo for up to 7 days while in ICU. The primary outcome of all-cause mortality at 90 days was 27.9% in the hydrocortisone group and 28.8% in the placebo group (OR 0.90, 95% CI 0.82 – 1.10, P=0.50). However, there were significant differences in secondary outcomes: with hydrocortisone the initial period of mechanical ventilation was shorter (median 6 vs 7 days, P<0.001), duration of ICU stay was shorter (median 10 vs 12 days, P<0.001), fewer patients received a blood transfusion (37.0% vs 41.7%, P=0.004) and, in keeping with several prior studies, resolution of shock was quicker (median 3 vs 4 days, P<0.001). The second trial (*NEJM* 2018; 378:809-818) was conducted by the CRICS-TRIGGERSEP Network in Europe and randomised 1241 patients with moderate or severe septic shock to receive either hydrocortisone 200mg/day plus fludrocortisone 50µg/day or matching placebos. Mechanical ventilation was not required but was present at baseline in 92% of participants. Mortality at 90 days was also the primary outcome in this study and occurred in 43.0% of patients in the hydrocortisone + fludrocortisone group and 49.1% of patients in the placebo group (RR 0.88, 95% CI 0.78 – 0.99, P=0.03). Similar to ADRENAL, the hydrocortisone + fludrocortisone group had significantly more vasopressor-free days (mean 17 vs 15, P<0.001) and ventilator-free days (mean 11 vs 10, P=0.07) to day 28. Of the potential complications of steroid treatment, only hyperglycaemia was more common in the hydrocortisone + fludrocortisone group; no significant differences were found in the incidence of new infections, gastro-intestinal bleeding or neurologic sequelae. Although some have expressed concerns that there was an unmeasured hazard in the ADRENAL study to explain why mortality was no different when the secondary outcomes found benefit, the combination of these 2 studies has consolidated the use of low-dose hydrocortisone in patients with "significant" septic shock, especially if receiving mechanical ventilation. Outside of Europe, few intensivists consider that fludrocortisone is important, given the mixed glucocorticoid and mineralocorticoid effects of hydrocortisone.

The treatment of ICU patients with severe metabolic acidaemia with sodium bicarbonate has been suggested to improve the metabolic environment, in particular the expectation of improved cardiovascular responsiveness to vasoactive therapies. However, in current practice this has generally been considered inappropriate due to concerns about paradoxical intracellular acidification, conflicting results in physiological and observational studies and the

lack of evidence of impact on clinical outcomes. Treatment for such patients has focused on addressing the underlying disease processes and support with renal replacement therapy if required. However, the first major randomised trial to report effect of sodium bicarbonate on clinical outcomes in the critically ill has recently been published. The BICAR-ICU trial (*Lancet* 2018; 392:31-40) was an open-label randomised trial which assigned 389 ICU patients in 26 ICUs with severe metabolic acidaemia (pH<7.2, $P_aCO_2$ <45mmHg, bicarbonate <20mmol/L) to receive no sodium bicarbonate or 4.2% sodium bicarbonate to maintain pH>7.3, using aliquots of 125 – 250ml with a maximum of 1L in the first 24h. Randomisation was stratified by site, age, sepsis and significant acute kidney injury (AKIN grades 2 or 3). The primary composite outcome in all patients (death by day 28 or at least one organ failure at day 7) occurred in 71% of the control group and 66% of the sodium bicarbonate group (absolute difference -5.5%, 95% CI -15.2 to +4.2, P=0.24). However, in the acute kidney injury stratum (189 patients), there was a significant difference in 28 day mortality (63% vs 46%, P=0.017), organ failure at day 7 (82% vs 66%, P=0.014) and the composite of the two (82% vs 70%, P=0.04). Renal replacement therapy was required in significantly fewer patients in the bicarbonate group (52% vs 35%, P<0.001), and ICU stay was shorter (median 4 vs 7 days, P=0.06). Metabolic alkalosis, hypernatraemia and hypocalcaemia were more frequent in the sodium bicarbonate group, but with no life-threatening complications reported. This is the first trial to demonstrate significant clinical benefit of bicarbonate therapy in the critically ill and should be considered for patients with acidaemia in the presence of acute kidney injury.

# Transfusion Medicine Update


## Professor James P. Isbister

Royal North Shore Hospital, and Clinical Professor of Medicine, University of Sydney, Australia


**Patient Blood Management: How should it be defined, communicated and progressed?**

> *"A modem battle plan is like nothing so much as a score for a musical composition, where the various arms and units are the instruments, and the tasks they perform are their respective musical phases. Each individual unit must make its entry precisely at the proper moment and play its phase in the general harmony."*
>
> General Sir John Monash.

When making blood transfusion decisions there has been a tendency to ask the wrong question. Clinical practice guidelines, especially for blood component therapy, have been falling into the common trap of starting with an answer before the question (ie diagnosis) has been clearly defined. This is a similar error to that which is commonly made in marketing when a business does not clearly identify the sector in which it is operating, known as marketing myopia. The point is emphasized and illustrated in the classic Harvard Business Review article by Levitt in 1960. In the early history of railroads the tycoons considered they were in the business of making railroads, when in fact they were in the transport business. As a result they were not able to adapt appropriately when other means of transport became available.

By analogy, transfusion medicine is in the business of improving clinical outcomes, not primarily collecting donor blood for transfusion into patients. Patient's clinical outcomes are improved by evidence-based diagnosis and therapy of diseases in which blood component therapy may have a role to play with the risks understood and accepted.

The history of blood transfusion is dotted with resistance to the implementation of new therapies and changes in clinical practices despite their being based on sound evidence. In many cases it is not new evidence that should have changed practice, but rather a reconsideration of the basic sciences, pathophysiology and soundly based clinical decision making.

Patients think blood transfusion is special and beneficial, but have difficulty accepting small risks they can't control. Blood Donors believe their contribution is a gift to the community that will be used appropriately and safely. Clinicians think blood is ordinary, take blood transfusion for granted, benefit is assumed and risks regarded as minimal. Governments view blood as a commodity and transfusion medicine as an expensive support service which should be regulated and funded in a "McDonaldised" manner.

**Challenges to blood transfusion practices**

- Why is anaemia not regarded as an important diagnosable and manageable clinical problem?
- How is that >20% of elective hip and knee replacement patients on long waiting lists can come to surgery with untreated iron deficiency anaemia and receive red cell transfusions?
- Why can there be a variation of 0% to 90% red cell transfusion rates for comparable standard-risk hip and knee replacement cases in different institutions?
- Why do Jehovah's Witnesses, declining blood transfusion have better clinical outcomes for many elective surgical procedures compared with case-controlled benchmark patients?
- Why is allogeneic blood transfusion commonly regarded as one of the safest medical interventions?
- Why are many elective haemodynamically stable surgical patients exposed to a medical intervention (ie red cell transfusions) that probably has the greatest potential for harm, but has not been proven to improve clinical outcomes?

- Why do EBM experts demand randomised controlled trials to prove the "safety" of allogeneic red cell transfusion when there is virtually no evidence of "efficacy" of red cell transfusions in improving clinical outcomes for anaemic haemodynamically stable patients?
- Why do most surrogate endpoints for transfusion "efficacy" (eg Haemoglobin rise) not correlate with improved clinical outcomes?
- Why is the primary focus of transfusion medicine on the role and use of donated allogeneic blood (product focus) rather than appropriate management of the patient's own blood (patient focus)?
- Why does the precautionary principle dictate decisions on the supply side of transfusion medicine, but the opposite applies on the patient (demand) side?

**What is patient blood management?**

Patient Blood Management (PBM) is an evidence-based bundle of care to optimise medical and surgical patient outcomes by clinically managing and preserving a patient's blood. Patient blood management is not an 'intervention', not an alternative to transfusion, it is good scientifically-based clinical medicine. Blood transfusion is a major medical 'intervention' and its use should be based on good clinical medicine. PBM is a good news story for patients and bureaucrats. Advocating PBM can be hard work, requiring engagement of clinician's grey matter to be implemented and successful in the long-term. To the media PBM is probably viewed as; *"What's new, PBM is boring, why aren't all doctors practicing PBM as standard of care?"*

If the following are the core elements of PBM that need coordinating, managing and auditing, what is the story those passionate about PBM should be trumpeting?

- diagnosing and treating reversible preoperative anaemia if time permits
- tolerating mild anaemia
- taking a preoperative/pre-procedure history for potential bleeding
- advocating meticulous surgical haemostasis
- involving patients in decisions regarding their clinical care

PBM is not primarily about reducing blood transfusion, but improving patient care. A positive corollary is avoiding inappropriate blood transfusion, ensuring appropriate use and availability of altruistically donated blood and respecting what donors expect when they donate blood. Further down the corollary line is saving of the health dollar. There is a good news or bad news story here for the media depending on one's view. There is the risk of adversely impacting on the blood donor base that is already under challenging pressures. There is also the issue that the blood sector in general is coming under threat from the success of PBM, especially in countries where there are significant commercial interests. Various conspiracy theories can be proposed in this respect. We are fortunate in Australia that we have achieved a relatively seamless connection from patient care to the highest levels of State and Federal Government. There have been some concerns about some pushback in the US and Europe to PBM that appears due to successes of PBM impacting on commercial interests of the blood sector.

Anaemia is commonly not regarded as a significant clinical problem and not taken seriously by many clinicians. It is uncommon to observe patients primarily dying from anaemia, except in Africa and other developing malarial countries. However, from a more global perspective anaemia is a significant risk factor for morbidity and mortality in numerous clinical settings if not addressed appropriately. For many clinicians anaemia receives a "simple" knee jerk solution with blood transfusion. Somebody else does the work, donor blood has been regarded as free, promoted as safe and a valuable community service. Many surgeons in the past have regarded transfusion as being available as a substitute for poor perioperative haematological management and poor surgical technique. Early in my career I was "pressured' by surgical mentors to embark on a surgical career. I knew in my own mind this would not and should not be my career path. This decision was solidly imbedded in my thinking during my surgical and anaesthetic intern jobs. I was fortunate to observe several great surgeons passionately caring for their patients overall medical well-being, not to also mention their meticulous surgical technique and attention to surgical haemostasis.

I recently asked the health reporter of one of our leading broadsheet papers why there is so much interesting in negative stories about PBM and nobody wants to write the positive story. I told her that I have been involved in

haematology, transfusion medicine and PBM as a clinician and a blood donor for 50 years from my early medical student days when doing an elective research term in Papua New Guinea and have seen it all, the good and bad news stories. I pointed out that surely a health reporter should be interested in a story that results in improving patient care, improved patient safety, saving of health dollars and ensuring stewardship of the donor blood supply from altruistic blood donors.

I was not surprised at the answer I received. The reported admitted that she was more interested in writing a story about the "conflicts of interest" of various members of the PBM guideline development committees than what good initiatives all these assumed "unethical" clinically practicing health professionals, patient advocates, health administrators and community representatives are trying to achieve.

The story and core elements of PBM we are trying to get over to our colleagues, and the media I suspect, may appear complex and sometimes it is forgotten that the foundations of modern scientifically evidence-based medical management presuppose an understanding of the structure and function of the normal, pathophysiology of disease, diagnosis and indicators for severity of disease as well as understanding the natural history and consequences of untreated disease. These principles are implicit in the three pillars of PBM, and at risk of stating the obvious, it is worthwhile outlining **the logic and core elements of PBM from basic principles through to clinical practice.**

**PRINCIPLES** Essential characteristics of health care, the adherence to cannot be ignored

- Patient blood and haemopoiesis, whether normal or diseased should be managed appropriately in all clinical settings.
- Donor blood is a unique and costly resource held in trust that should only be used when there is evidence for potential benefit, potential harm will be minimized and there are no reasonable alternatives.
  **AXIOMS** That which is self-evident
- Evidence-based medical practice has its foundations in science, ethics and economics.
  **THEOREMS** Conclusions deduced from axioms
- PBM is standard of care with the aim of achieving the best clinical outcomes for individual patients.
  **COROLLARIES** Conclusions that inevitably follow-on from the theorem
- PBM results in avoiding or minimizing unnecessary allogeneic blood transfusions.
  **PRACTICE** The application, ongoing pursuit and monitoring of outcomes of clinical decisions
- Individualized patient management by multidisciplinary teams with multimodal interventions addressing the three pillars of PBM to:

1. Optimize haemopoiesis.
2. Minimize blood loss.
3. Tolerate haemopoietic deficiencies.

This might all be too much for some health professionals or regarded as boring. However, we must have a clear idea in our own minds as to how we define PBM and the language we use. We know PBM is a "simple" concept, but its communication and implementation can be complex.
The three-pillar matrix of PBM:

**1st Pillar:** Optimize erythropoiesis
**2nd Pillar:** Minimize blood loss & bleeding
**3rd Pillar:** Tolerate anaemia by harnessing & optimising physiological reserves

Clinical practice of the three-pillar matrix is determined by:

- medical or surgical context
- age and sex of the patient
- time frame for managing the primary clinical problem, ie. urgent, emergent or elective
- reversibility and treatability of the primary disease
- presence of comorbidities
- availability and costs of alternatives to blood transfusion
- specific patient preferences

**Definitions, evidence, questions, "no brainers" and challenges**

**Allogeneic Blood Transfusion**

A therapeutic intervention for which there is evidence of efficacy and safety in improving a patient's clinical outcome AND there is no alternative clinical management "available".

**Patient Blood Management**

Clinical management based in sound medical science that improves patient outcomes.

**Why is management of a patient's blood in the perioperative setting regarded differently from managing other systems of the body?**

- There is an excess focus on blood transfusion rather than diagnosis.
- Transfusion is usually a default and easy discretionary decision.
- The patient does not have a "blood advocate".
- Haematologists have a limited role/interest in perioperative medicine.
- Anaemia is not regarded as important.
- Surrogate endpoints are used for determining efficacy of transfusion.
- There is a false sense of donor blood safety.
- Donor blood has been promoted as "The safest pharmaceutical".
- Many clinicians don't acknowledge stewardship responsibilities towards altruistic blood donors.

**Relevant PBM questions**

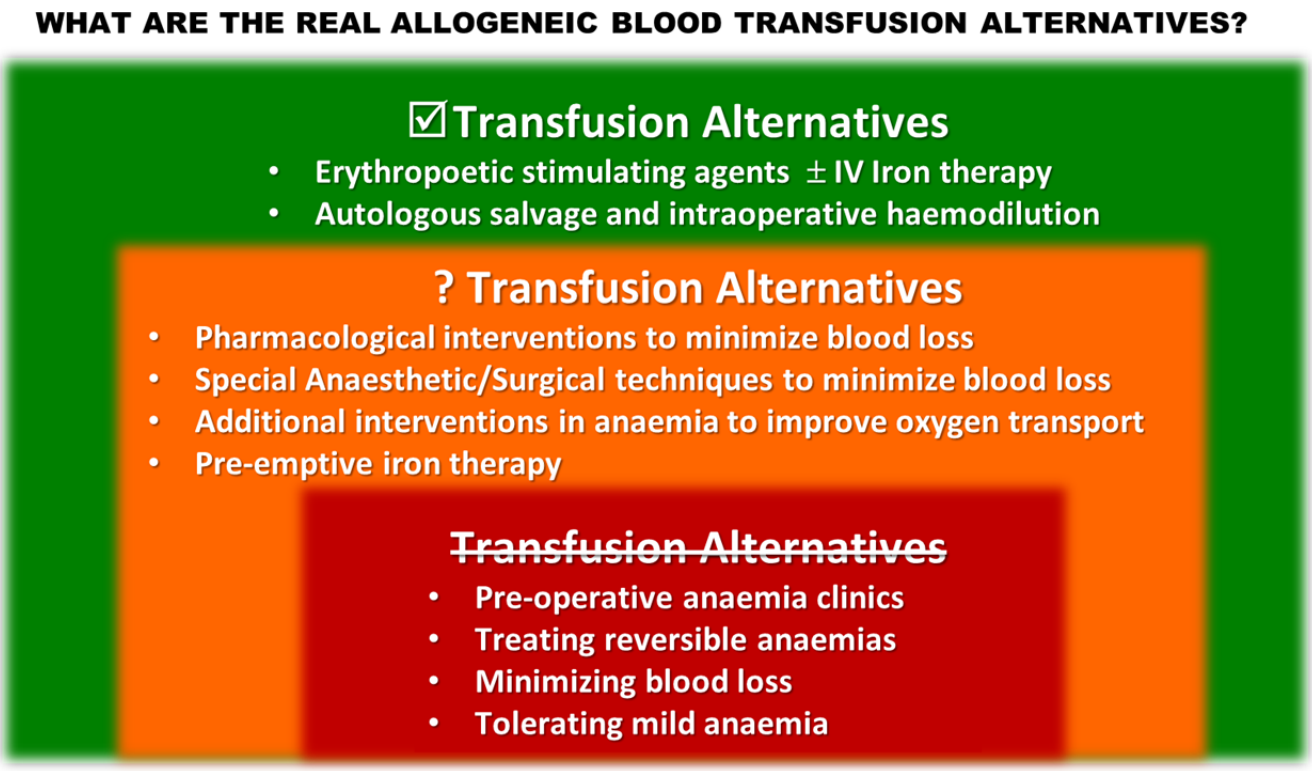
- Why manage the Haemopoietic System differently than other systems?
- Why do elective surgery on patients with reversible anaemia?
- Why not pre-empt and prevent excessive haemorrhage?
- Why not tolerate mild anaemia?
- Why administer potentially hazardous blood transfusions for which there is no good evidence for benefit or improved patient outcomes?
- How do you explain to an altruistic blood donor that their gift caused a serious complication in a patient in whom there was no evidence the patient would have benefited from the blood transfusion?
- What is the cost of not giving a red cell transfusion to a patient in whom it was not indicated?

**The no brainers**

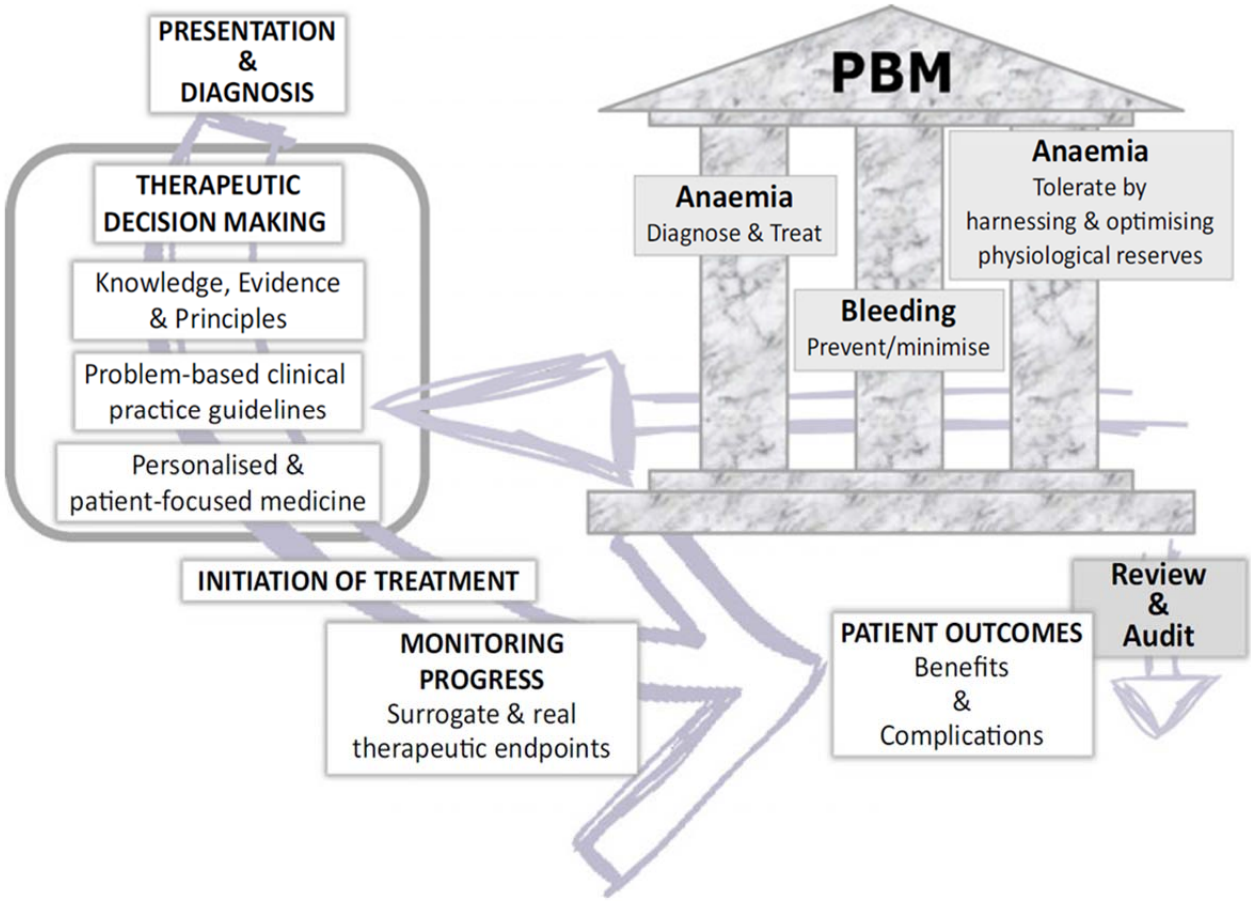
- Why manage the haemopoietic system differently than other systems?
- Why do elective surgery on patients with reversible anaemia?
- Why not pre-empt and prevent excessive haemorrhage?
- Why not tolerate mild anaemia?
- Why administer potentially hazardous blood transfusions in circumstances in which there is no sound evidence for benefit or improved patient outcomes?
- How do you explain to an altruistic blood donor that their gift caused a serious complication in a patient in whom there was no evidence the patient would have benefited from the blood transfusion?
- What is the cost of not giving a red cell transfusion to a patient in whom it was not indicated?
- Iron deficiency should and can be treated
- What is the best for the patient may not appear to be the cheapest, but may be the most cost-effective on the basis of full activity based costing
- Whenever possible the patient should be involved in decision making

**The PBM challenges**

- Awareness in relevant stakeholder groups
- Information to the public and patients at large
- Undergraduate and postgraduate education for nurses, physicians and other health professionals
- Patient empowerment and advocacy
- Incentives/disincentives for health care providers
- Perspectives/incentives for clinicians
- Monitoring of transfusion outcomes
- Transfusion/PBM benchmarking



**The application of the three pillars of patient blood management**
Isbister JP ISBT Science Series (2015) 10 (Suppl. 1), 286–294



Approaches to PBM in many clinical settings continue to evolve. It is elective surgery where the most evidence for achieving practice changes and the greatest benefits have been demonstrated in improving patient outcomes and reducing exposure to allogeneic blood transfusion. However, similar principles apply to patients with blood loss. Anaemia and iron deficiency are commonly not regarded as significant clinical problems and frequently not taken seriously by many clinicians. Iron deficiency anaemia (IDA) and iron depletion/deficiency (ID) in women with abnormal uterine bleeding (AUB) is common and well documented, but poorly addressed. Anaemia in the elderly is also a relatively poorly addressed. PBM in its broadest application includes the management of any quantitative or qualitative deficiencies in the haemopoietic system and the role of allogeneic blood components or plasma products as therapy on a sound evidence-base. Increasingly, there are recombinant plasma proteins available as true alternatives to blood donor-based plasma products.

*Our main business is not to see what lies dimly at a distance, but to do what lies clearly at hand.*
Thomas Carlyle (1795 – 1881)

**PBM of haemodynamically and haemostatically stable patients**
It is in the haemodynamically and haemostatically stable patients in which PBM has a great deal to offer in terms of minimizing or avoiding allogeneic blood components. This group is largely made up of the uncomplicated elective surgical patients and patients with chronic anaemia usually related to marrow suppression secondary to cancer chemotherapy and patients with myelodysplastic syndromes.

**PBM in critical haemorrhage and massive transfusion**
It is with the critical haemorrhage and massive transfusion category of patients that the focus is on a better understanding of underlying causes and pathophysiology. In this group the clinician is commonly faced with an urgent clinical problem in which any pre-insult or disease assessment of the three pillars is not possible, or at best assessed retrospectively from the patient's clinical history or the current clinical context. The focus is on rapid assessment of the underlying cause/s and the presenting 'status' of the three pillars of PBM, especially in relationship to the haemostatic system. This is where point of care testing and real time management of the three pillars is increasingly being shown to lead to minimising allogeneic blood transfusion and better clinical outcomes. In many of these circumstances minimising allogeneic blood transfusion results in better clinical outcomes in terms of less lung injury and multi-organ failure, reduced assisted ventilation, lower infection rates, fewer and shorter ICU admissions as well as shorter lengths of hospital stay. In other words, transfusions can be a two-edged sword in saving lives, but with them may come unintended adverse consequence that need to be minimised as far as possible. The two most challenging clinical settings in this respect are trauma and obstetric haemorrhage.

**PBM in congenital bleeding disorders, immunodeficiency, and immunotherapy management**
In the group with clearly categorized and specific haemopoietic deficiencies, including immune disorders, the underlying pathophysiology is understood and therapy usually has a sound evidence-base, enunciated in clinical practice guidelines. Additionally, the therapeutic blood products, human derived or recombinant, have proven efficacy and safety profiles and their manufacture is highly regulated and controlled.

**Patient empowerment and personalised medicine**
With greater empowerment of patients by involvement in determining their own clinical management there are complex issues surrounding consent as to what information about PBM and blood transfusion should be provided, how should it be communicated and documented to confirm that it has been validly achieved. In view of recent evidence implicating transfusion of labile blood components as an independent risk factor for adverse clinical outcomes, reconsideration of product information is warranted and wider dissemination of this information is important. Transfusions of allogeneic labile blood components are tissue transplants and have the widest and most heterogeneous potential hazards, probably greater than any other medical intervention, but this is not the message that is currently being communicated to clinicians, patients and the community. Evidence for benefit in improving clinical outcomes is increasingly a challenge and a reassurance patients and blood donors can reasonably expect.
Theodor Billroth (1829 – 1894), one of the fathers of modern surgery, had this advice to doctors as apposite today as it was over a century ago.

*"A person may have learned a good deal and still be a very bad doctor who earns no trust from patients. The way to deal with patients, win their confidence, listen to them (patients are more eager to talk than to listen) and help them; console them, get them to understand serious matters: none of this can be read in books. A student can learn it only through intimate contact with his teacher, whom he will unconsciously imitate ... The patient longs for the doctor's visit; his thoughts and feelings circle around that event. The doctor may do whatever is necessary with speed and precision, but he should never give the impression of being in a hurry, or of having other things on his mind."*

Links to references and further reading


1. Patient Blood Management Bundles to facilitate implementation
   https://www.researchgate.net/publication/315115941_Patient_Blood_Management_Bundles_to_facilitate_implementation
2. The three-pillar matrix of patient blood management
   https://onlinelibrary.wiley.com/doi/abs/10.1111/voxs.12135
3. Cornerstones of patient blood management in surgery
   https://onlinelibrary.wiley.com/doi/pdf/10.1111/tme.12476
4. *Building national programmes of Patient. Blood Management (PBM) in the EU.* A Guide for Health Authorities
   https://ec.europa.eu/health/sites/health/files/blood_tissues_organs/docs/2017_eupbm_authorities_en.pdf
5. Supporting Patient. Blood Management *(*PBM*) in the* EU*. A Practical Implementation Guide for Hospitals.*
   https://ec.europa.eu/health/sites/health/files/blood_tissues_organs/docs/2017_eupbm_hospitals_en.pdf
6. National and International Guidelines for Patient Blood Management in Obstetrics: A Qualitative Review
   https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5161642/
7. Patient Blood Management: the new standard
   https://onlinelibrary.wiley.com/doi/epdf/10.1111/trf.14095
8. Drivers for change: Western Australia Patient Blood Management Program
   http://www.haemoview.com.au/uploads/2/5/4/9/25498232/simon_towler_2013.pdf
9. Improved outcomes and reduced costs associated with a health-system–wide patient blood management program: a retrospectiveobservational study in four major adult tertiary-care hospitals
   https://onlinelibrary.wiley.com/doi/epdf/10.1111/trf.14006
10. Australian National Blood Authority: Best Practice
    https://www.blood.gov.au/best-practice
11. Australian National Blood Authority: Patient Blood Management Guidelines
    https://www.blood.gov.au/pbm-guidelines
12. At the end there are some infographic PBM resources**.**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**A bit of history**

In the early days of Patient Blood Management the main driver for change in transfusion medicine practices was the recognition that allogeneic blood transfusion may be an independent risk factor for adverse clinical outcomes, especially in contexts in which there was a questionable evidence-base for efficacy. Allogeneic blood transfusion had grandfathered its way into medical therapeutics and become culturally imbedded into clinical practice, with benefit being assumed and risks regarded as minimal. Blood transfusion had become the default decision in the context of clinical uncertainty. Of even more concern was perioperative red cell transfusions being administered in non-urgent clinical circumstances in which haematological deficiencies, usually anaemia, were correctable without blood transfusion.

It is only in recent years that there has been a concerted effort to establish a sounder evidence base for the benefits and hazards of allogeneic blood transfusion in the wide range of clinical settings in which it is, may be, or is not, appropriate therapy. Few would doubt the role of blood transfusion in the management of haemorrhagic shock, critical life-threatening anaemia and to enable the development of newer major medical and surgery therapies. The provision of blood component therapy for specific cellular or plasma deficiencies and the development of haematological supportive care for the management of haematological malignancies has become essential and generally on a good evidence base.

There has been a gradual awakening over the last 30 years throughout the blood sector, clinical practice, bureaucracies, governments, the community and the legal profession that, as Bob Dylan would have expressed, *"the times they are a changin."* There have been several drivers for change. The reassessment of the safety of transfusion in the context of questionable efficacy in improving clinical outcomes has been high on the agenda. Additionally, governments have become more focused on the blood sector leading to national reviews, economic evaluations and, in some circumstances, criminal proceedings against individuals. Lastly, altruistic blood donors can reasonably expect that their blood will be used to benefit the greatest number of patients with minimal chances of adverse impacts.

Patients being exposed to risk without evidence for benefit is a "bad news" story. The continuing resistance to acceptance of evidence questioning the efficacy and safety of transfusions in many circumstances should have resulted in an overwhelming case for adopting the precautionary principle to the use of allogeneic blood transfusion. This is especially the case in uncomplicated elective surgery and in haemodynamically stable patients with anaemia. Of even greater concern was the promotion by some European blood services that allogeneic blood was the 'safest pharmaceutical', implying commodification of an altruistically donated human resource.

The paradigm shift to a patient-focus returned clinicians to managing a patient's own blood. This was no different than the management of any other body system, normal or dysfunctional. A sound understanding of physiology and pathophysiology is a *sine qua non* in providing optimal patient care and ensuring the best clinical outcomes. This is a good news story, a no brainer, so what's new?

The following extracts from the British Medical Journal (1945) and the New England Journal of Medicine (1936) says it all, especially in the follow up letter by Major General Ogilvie after WWII.

BRITISH MEDICAL JOURNAL

LONDON SATURDAY MAY 5 1945

# SOME APPLICATIONS OF THE SURGICAL LESSONS OF WAR TO CIVIL PRACTICE

BY

W. H. OGILVIE, M.Ch., F.R.C.S.

*Major-General*

Now that the European phase of the second world war appears to be entering its final stages, it may be well to consider briefly what war has taught surgery and what it has taught surgeons, and how the lessons learned in the field can be applied to the teaching and practice of surgery in civil life.

The surgery of wounds in this war has passed through three phases. In the first, treatment by the closed plaster method was the rule; in the second, which was a period of long communications and poor supplies, wounds were excised and drained, the limb was immobilized in a padded plaster case or some form of plaster box splint, and closure by secondary suture or skin grafting was attempted about the third week, or as soon as the surface was covered with healthy granulations; in the third phase, which has been helped by the advent of penicillin, the wounds are excised by the forward groups and closed by delayed primary suture at the base between the fourth and the sixth day.

of purely surface injuries, war wounds can never be rendered entirely healthy and entirely sterile by surgical toilet. The limits of tissue damage cannot be decided with any accuracy, and bacteria, blasted in by the cushion of air that precedes the projectile or displaced along tissue planes by movements of the limb, may lie well outside the visible confines of the wound track. Where the bacteria are few and the remaining damaged tissues small in amount, the defences of the body will soon turn out the invaders unless they are hindered by tension. In a sutured wound the hyperaemia which should give protection is limited by the unyielding surroundings and finally replaced by ischaemia, and the outpouring of defensive fluids is brought to a standstill when the interstices of the wound are filled; the bacteria, on the other hand, find in the trapped discharges an ideal pabulum and in the anoxic tissues an easy prey.

The wounds of road and industrial accidents are, like those



# Correspondence

## Blood Transfusion

I am unrepentant in condemning the giving of blood during straightforward operations.

a smooth operation should lead to smooth convalescence without biochemical assistance.—I am, etc.,

London, N.W.8. W. H. OGILVIE.

The New England
# Journal of Medicine

1936

VOLUME 215 SEPTEMBER 3, 1936 NUMBER 10

## The Massachusetts Medical Society

## SECTION OF MEDICINE

**Lower Section Room, Municipal Auditorium, Springfield, Tuesday, June 9, 1936, 2 p. m.**

PRESIDING:
Dr. William D. Smith, Boston, Chairman.
Dr. Laurence B. Ellis, Boston, Secretary.

CHAIRMAN SMITH: Will the meeting please come to order.

The first duty of the Section is the selection of the Chairman and the Secretary for the coming year, and, in accordance with the usual custom, the Chair will appoint as the Nominating Committee to suggest names Dr. Dwight O'Hara, Chairman, Dr. George R. Minot and Dr. Chester M. Jones. They will report later and abide the pleasure of the Section.

I do not see Dr. Hamilton here. Apparently she is delayed, so we will pass on to the second paper. To those of us who have had our moments of indecision whether to transfuse or not to transfuse in some of our medical problems, Dr. Bock's paper should be of interest. His subject is "The Use and Abuse of Blood Transfusions."

## THE USE AND ABUSE OF BLOOD TRANSFUSIONS*

BY ARLIE V. BOCK, M.D.†

THE mass of literature on the subject of blood transfusions accumulated during the past twenty-five years is so great and most of it so readily available that one shows lack of temerity at least to attempt a discussion of the subject before this audience. The transfusion of blood may be a life-saving procedure under certain circumstances, it may be a necessary supportive measure under others, but it is too often undertaken when the doctor can think of nothing else to do after all other therapy has failed. My objective today is to discuss briefly the common surgical and medical conditions for which transfusion of blood is indicated, in which we can expect good physiological results, and to point out those conditions in which it is little more than a gesture, done, as it were, to satisfy the urge to do something.

### SURGICAL INDICATIONS

1. *Shock.* Many theories of the cause of primary and secondary shock have been offered by able investigators, most of them recently reviewed briefly by Blalock.[1] Because of the complexity of the events no theory yet proposed can be considered the final answer as to the etiology of shock. We know that if treatment of the condition is to be successful it must accomplish two things, restoration of diminished blood volume and elevation of low blood pressure. Blood volume may be reduced by gross hemorrhage or it may be reduced by blood lost in the periphery of the body, as suggested by Freeman,[2] or by extravasation of serum through damaged capillaries. If hemorrhage has occurred, transfusion of blood, together with such supportive measures as heat, is the immediate indication. No other therapy is so successful. In shock without much or any hemorrhage, 6 per cent gum acacia in normal saline may be just as effective as blood, and has the advantage of greater availability. Repeated transfusions of blood or infusions of acacia may be necessary but, are usually not, if no delay has occurred in the first instance. Acacia may be used as a supportive measure until transfusion can be arranged. Prolongation of the shock state results in tissue asphyxia, capillary damage, petechial hemorrhages, and rapid change in general to an irreversible state.

One of the common accompaniments of shock is dehydration, a state associated with loss of water, base, chloride and increase of nonprotein nitrogen. When such a state exists, transfusion alone is not adequate therapy but normal salt solution, often in large quantities, should be administered intravenously, or it may be given in eight-ounce quantities by rectum every half hour. When facilities permit, serum chloride

*Read at the Annual Meeting of the Massachusetts Medical Society, Section of Medicine, Springfield, June 9, 1936.

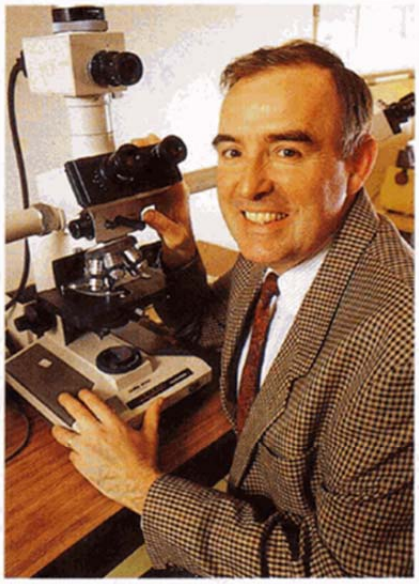
†Bock, Arlie V.—Physician, Massachusetts General Hospital. For record and address of author see "This Week's Issue," page 469.

THE BULLETIN, JULY 9, 1991

# Vein glory

*The AIDS epidemic has changed attitudes towards transfusions. GLENNYS BELL reports on the crisis facing the Australian Red Cross, which is fighting for its reputation and the confidence of donors – those who give blood as well as those who give money*

In a rare turnaround, the Australian Red Cross Society is coming under fire on its home front. The reputation of its blood transfusion service is being questioned in the courts where it is being sued by people who were infected with the AIDS virus. Behind the scenes, its operations and the way that it ensures the safety of the country's blood supply are to be evaluated by Australia's health ministers. Dr James Isbister, head of the department of haematology at Sydney's Royal North Shore Hospital, believes that standards in Australia are high but advocates reform of the blood transfusion service. He is one of the few experts in the field who has been vocal on the service's deficiencies. The last thing he wants to see is a loss of confidence as happened in the US. "It's in no one's interest," he says. "We don't want the same thing to happen here." But he is concerned because there is no co-ordinated approach to making blood transfusion services safe and effective.



Dr James Isbister: an advocate of reform


Isbister sees the service as a product of its past. It was born in crisis in the 1940's to help victims of war and conflict and depends on the altruism of donors to give blood for the benefit of others. It is the others, the patients, who may be forgotten by a centralised service. It is time, he believes, for a blood transfusion service which focuses on the people who receive blood as much as – if not more than — those who donate it.

I reference the above media article from 1991 published in the now defunct Australian Bulletin magazine. This was one of my early attempts at shifting the paradigm back to a patient's blood focus and not donor blood product focused. I was approached by the Bulletin when the AIDS crisis surrounding blood transfusion was regularly in the media. The main responses the article received related to "shooting the messenger". I should emphasise that this article was 27 years ago and a lot has changed since then. The last paragraph of the article is probably the first media reference to what was to become "Patient Blood Management" many years later. At a 2005 board meeting of the International Foundation for Blood Management I proposed the terms "Patient Blood Management" and "Donor Blood Management" as the more generic term "Blood Management" was resulting is some confusion. The term "Patient Blood Management" first appeared in the title of an article in the peer reviewed literature in 2008.

Anesthesiology 2008; 109:951–3 

# *Patient Blood Management*

## *The Pragmatic Solution for the Problems with Blood Transfusions*

**Donat R. Spahn, M.D., F.R.C.A.,* Holger Moch, M.D.,† Axel Hofmann, M.E.,‡ James P. Isbister, M.B., F.R.A.C.P.§**
*Institute of Anesthesiology, University Hospital Zürich, Zürich, Switzerland. donat.spahn@usz.ch. †Institute of Surgical Pathology, Department of Pathology, University Hospital Zurich, Zurich, Switzerland. ‡Medical Society for Blood Management, Laxenburg, Austria. §Department of Haematology, University of Sydney, Royal North Shore Hospital of Sydney, St. Leonards, New South Wales, Australia.

**Back to the future: What's old is new**

**https://archive.org/details/b28083593**

**The following was the year before Karl Landsteiner discovered the ABO blood groups**

# INTERCOLONIAL MEDICAL CONGRESS OF AUSTRALASIA.

## FIFTH SESSION.

**BRISBANE, QUEENSLAND.**

SEPTEMBER, 1899.

## SECTION OF MIDWIFERY AND GYNÆCOLOGY.

## THE SAVING OF BLOOD IN GYNÆCOLOGICAL OPERATIONS.

By Archibald Watson M.D. Paris, F.R.C.S. Eng.,
Professor of Anatomy and Physiology in the Adelaide University.

Mr. President,—

Dr. Byrne, the courteous secretary of your Section, has paid me the compliment of inviting me to make some remarks on the saving of blood in gynæcological operations within the pelvis, on the principle, I presume, that "spectators see most of the game." Often, however, I have only appeared on the scene after the game was quite over, and in my opinion this latter factor should make any comments from one outside of your ranks appear less trivial than they otherwise might.

INTERCOLONIAL MEDICAL CONGRESS OF AUSTRALASIA.

FIFTH SESSION.

BRISBANE, QUEENSLAND.

FIRST DAY—MONDAY, 18TH SEPTEMBER, 1899.

SECTION OF MIDWIFERY AND GYNÆCOLOGY.


INTERCOLONIAL MEDICAL CONGRESS
OF AUSTRALASIA.

TRANSACTIONS

OF THE

FIFTH SESSION

HELD IN

BRISBANE, QUEENSLAND,

SEPTEMBER, 1899.

Published under the direction of the Literary Committee.

By WILTON LOVE, M.B.

Brisbane:

BY AUTHORITY: E. GREGORY, GOVERNMENT PRINTER, WILLIAM STREET.

1900.



## POST-PARTUM HÆMORRHAGE: ITS TREATMENT—ANTICIPATORY AND ACTUAL.

By Ed. Luther, B.A., M.D. (B.Ch., Etc., Dub. Univ.),
Hon. Surgeon to the Lady Musgrave Hospital; and Hon. Physician, Wide Bay and Burnett Hospital, Maryborough, Queensland.

The first requisite against hæmorrhage from the post-partum uterus is the maintenance of its firm uniform contraction and tonic retraction (Lusk).

In my experience the chief cause of post-partum hæmorrhage amongst Queensland women is the want of this muscular tone, for amongst our women one meets with a large number suffering from inanition and anæmia, especially during the summer months. Whether this anæmia is caused by malaria, anchylostoma, or just the enervating heat, it matters not for the purpose of this paper. But, whenever I am engaged to attend at the confinement of a woman anæmic in appearance, I always anticipate having some post-partum hæmorrhage, for with the anæmia you will certainly have uterine inertia and flaccid abdominal muscles, that may have just sufficient stamina to expel the fœtus, but none left for the expulsion of the placenta, or for keeping the uterus contracted afterwards.

Now, what I wish to bring before you to-day is that by a preparatory treatment we can ward off this evil, dreaded by most accoucheurs.

My method of treatment is first to try and cure the anæmia and debility by tonics of iron and strychnine, and during the last fortnight or three weeks of gestation to place the patient on the following mixture:—Extractum ergotae, 22 gr.; liquor strychninæ, 1 dr.; acid sulphuric dilut, 1 dr.; glycerin, 1 oz.; aq. anisi, 8 oz.; a tablespoonful to be taken three times a day. I have now been adopting this treatment for the past four or five years in all cases that have a history of having had post-partum hæmorrhage at their former confinements, or where the patient seemed debilitated or a likely subject for hæmorrhage, and in every case with satisfactory results.

JAMA
The Journal of the American Medical Association

February 25, 1899, Vol XXXII, No. 8, Pages 395-450



## SIMPLIFIED METHODS OF BLOOD EXAMINATION. THEIR PRACTICAL APPLICABILITY TO GENERAL DIAGNOSIS.

BY ALFRED C. CROFTAN, M.D.
Late Assistant Professor of General Diagnosis, College of Physicians and Surgeons, Chicago.
PASADENA, CAL.

Laboratory aids to diagnosis, to be universally employed by the practicing physician, should be simple and rapid of execution, should require no costly or complicated paraphernalia, and should yield quicker and more positive results than purely clinical methods.

No period in the history of medicine has been free from attempts to find diagnostic clues in the examination of the blood; it was tried to interpret the rapidity of coagulation, the *crusta phlogistica* of eighteenth century physicians, the appearance of the blood as it flowed from the incised vein. With the development of microscopic technic and an insight into the truths of cellular pathology, valuable data relating to the morphology of the corpuscular elements of the blood in health and disease were discovered. During the last decade, especially through the efforts of German and American investigators—at their head Ehrlich of Berlin and Neusser of Vienna—a mass of purely empiric data on the appearance of the blood in a variety of morbid conditions has been gathered and the recurrence of a characteristic blood-picture in certain diseases verified; inversely, diagnostic clues have been gathered from the appearance of the blood.

# NO WOMAN NEED BECOME ANAEMIC

**Most women are anaemic at some time in their lives, though it is not nearly so common as it used to be. Gone are the wan, romantic maidens of Queen Victoria's time.**

THERE is no need now for any Australian to go without the essentials for normal blood formation, but many still do — some through ignorance, some because they prefer such things as alcohol, but most through carelessness.

The housewife often feeds her family, then settles down to a cup of tea and a slice of bread and jam, poor stuff for making good red blood!

Besides, anaemia is twice as common in women and adolescent girls as in men.

Anaemia really means a shortage in the red blood cells of the red pigment, haemoglobin, which carries oxygen all over the body. There may be insufficient red blood cells or insufficient red pigment in each.

The red cells are manufactured in the bone marrow from a variety of substances, some of which are made in other parts of the body, while others are taken in the diet.

### Difficulties

There can be difficulties in manufacture, and absence of certain essentials can stop production.

Even if the manufacturing side is perfect, many things can happen to the cells; certain germs, drugs, and chemicals may destroy blood, and a common problem is blood loss from ulcers in the stomach or duodenum, from piles, and even heavy "periods."

There are also times in our lives when there are greater demands on our blood, and it becomes extremely important that our bodies should be supplied with the right materials.

If I tell you something about these times and about the right materials, you will be better off than if I give you a long list of symptoms and say, "Go to the doctor if you get one."

After all, lots of very pale women have quite red lips, even without their lipstick, and are not anaemic at all.

**And quite a lot of women who feel a bit tired and short of wind, and secretly imagine that they "have a heart," are suffering from anaemia that could be easily treated by attention to their diet.**

You may be rather shocked to hear it, but liver injections are no good as a tonic.

Recent research work has established that liver and vitamin $B_{12}$ injections, which were once considered to have a general tonic effect, really have none.

Patients who were given injections of water did just as well as those who had liver injections or $B_{12}$, provided they all got a diet containing the essentials and believed they were getting the tonic.

The periods in life that make special demands on blood supplies include those of most active growth—infancy and adolescence.

The baby grows very fast from birth to two years, and anaemia is common, particularly in premature babies, twins, and children who have had a lot of infections, even just colds.

Milk, with its low iron content, is often their main article of diet, so you must be alert to the results of infections and the need for the dietary extras.

At puberty bodies demand extra iron and calcium and proteins for growth, and there's actually a big increase in the quantity of their blood.

All girls from puberty onwards lose blood regularly with their monthly periods; not a great deal of blood, and nothing that a normal girl can't cope with.

However, the blood has to be replaced, and the girls who are overworking or careless with their diet, and harassed young mothers may not always get the essentials.

Then there is pregnancy and lactation. Babies get all their body-building substances from their mothers.

During birth the mother loses more blood, and while she feeds the baby at the breast she passes sufficient iron into the milk to give her baby enough.

Artificially fed babies, on cow's milk for five months without extra food, often get anaemic because nature didn't arrange for a cow to add this extra iron to milk. Her calf goes out and chews grass at a very tender age.

It is estimated that the process of carrying, bearing, and feeding a child needs a third of the mother's iron and she has to supply it to the baby.

The baby of an anaemic mother is not anaemic at birth, because nature sees that it gets its requirements, but it soon becomes anaemic later on owing to a lack of reserves of iron.

What do we need for making blood? I've mentioned iron several times, but the research chemists have identified many other substances that the body itself cannot make but which are needed.

They include vitamin $B_{12}$, folic acid, unknown factors that occur in yeast and liver, certain fats, and metals such as iron, copper, and cobalt.

Don't think you should suck pennies, or have vitamin $B_{12}$ injections. Most of these substances are needed in very small quantities and a good diet of meat, eggs, liver, cheese, and wholemeal cereals contains them all.

In certain illnesses, when food is not being properly absorbed from the bowel, folic acid or vitamins, for instance, may have to be added.

If the anaemia has already developed, single essential substances may have to be replaced in bigger quantities than the diet can supply.

But that is the doctor's job; just now I am talking about prevention.

Other substances are needed for the body to be able to use the essential materials.

You need vitamin C and the vitamin B group, and certain proteins.

To supply vitamin B, Vegemite, Marmite, and wholemeal bread are easy to get; oranges and tomatoes provide vitamin C.

Blood-making isn't just a matter of chemistry. Hormones are very important; thyroid, ovaries, pituitary, and adrenal glands all work together in harmony, and a discord can cause anaemia.

Many middle-aged women have a mild thyroid deficiency and the strange anaemia of Victorian times, which used to affect young girls so severely that they died, was partly due to iron deficiency, but also to some ovarian disturbance at puberty.

### Not silly

It may sound silly, but it is true that happiness and emotional balance matter probably as much as balanced diet in preventing anaemia.

I must sound a funny doctor warning people against pills and potions, but I am talking about preserving health and preventing anaemia.

I prescribe iron pills for iron-deficiency anaemia, and injections for pernicious anaemia just the same as other doctors.

And I don't disapprove of those extra vitamins in hot milk at examination time or some extra iron and vitamins during pregnancy.

I'm pointing out that there are still many essential substances in the good food provided by nature that the scientists don't know about yet, so don't pin your faith on pills and tonics and refined foods.

## HELP FOR HOUSEWIVES by Clair Isbister,

*Australian doctor and housewife. This is our third extract from her book, "What is Your Problem, Mother?"*

July 23, 1958
PRICE 9d
THE AUSTRALIAN Women's Weekly

# A Generic Approach to Patient Blood Mangement

| Primary disease & PBM | EVIDENCE | ECONOMICS | ETHICS | RISKS |
|---|---|---|---|---|
| Epidemiology & Diagnosis Clinical/Laboratory | The nature and extent of the 1° condition and relevance of the blood and possible deficiencies that may have consequences for PBM Standard of care for managing the 1° condition. | Is knowledge of 1° condition and any haematological deficiencies complex or "simple" and the economics understood?<br>?Educational challenges | Responsibility of clinicians and bureaucrats to understand the issues surrounding the 1° condition and PBM<br>Equity of access to clinical care and pathology services | Adverse epidemiological data<br>Indicative of gaps<br>Geographical isolation<br>Limited laboratory services |
| Interpretation of pathology results | Are reliable pathology testing methodologies available and being reviewed and interpreted correctly? | Are diagnostic methods for 1° condition and PBM readily available and cost-effective? | Equity of access to specialist clinical expertise and interpretation of diagnostic pathology results when necessary | Lack of local expertise<br>Lack of specialist expertise<br>Incorrect interpretation of laboratory results |
| Clinical decision making and therapy<br>Monitoring & ongoing care<br>OUTCOME | Is there an integrated approach to management of the 1° condition and PBM? Are established and feasible guidelines available?<br>Are healthcare implementation methodologies and guidelines available and known to be effective?<br>1° condition and PBM can be managed resulting in optimal outcomes | Is standard of care of the 1° condition and PBM cost-effective for individuals and the health system<br>Blood transfusions are expensive and risky and only indicated if no alternative<br>Monitotring and ongoing management of 1° condition and PBM should be cost-effective. | Ensuring implementation of a standard of care for the 1° condition and PBM<br>Allogeneic blood transfusion is only used when indicated and stewardship of the donor blood supply is respected<br>Patient empowerment, informed consent and follow up is and integral component of clinical management | Clinical practice variation and failure to follow guidelines<br>Availability and therapy delays<br>Inappropriate blood transfusion<br>Timely and logistic availability of appropriate therapy<br>Failure to appropriately address patient engagement and informed consent. |
| OTHER DRIVERS<br>DATA → | Evidence improving patient outcomes<br>Variations in clinical practice and adherence to guidelines<br>Default management decisions | Contain/reduce health costs<br>The problem of excessive and inappropriate use of health resources | Appropriate Transfusion<br>Stewardship of donor blood<br>Patient informed consent and advocacy | Variable clinical contexts<br>Wastage of donor blood |

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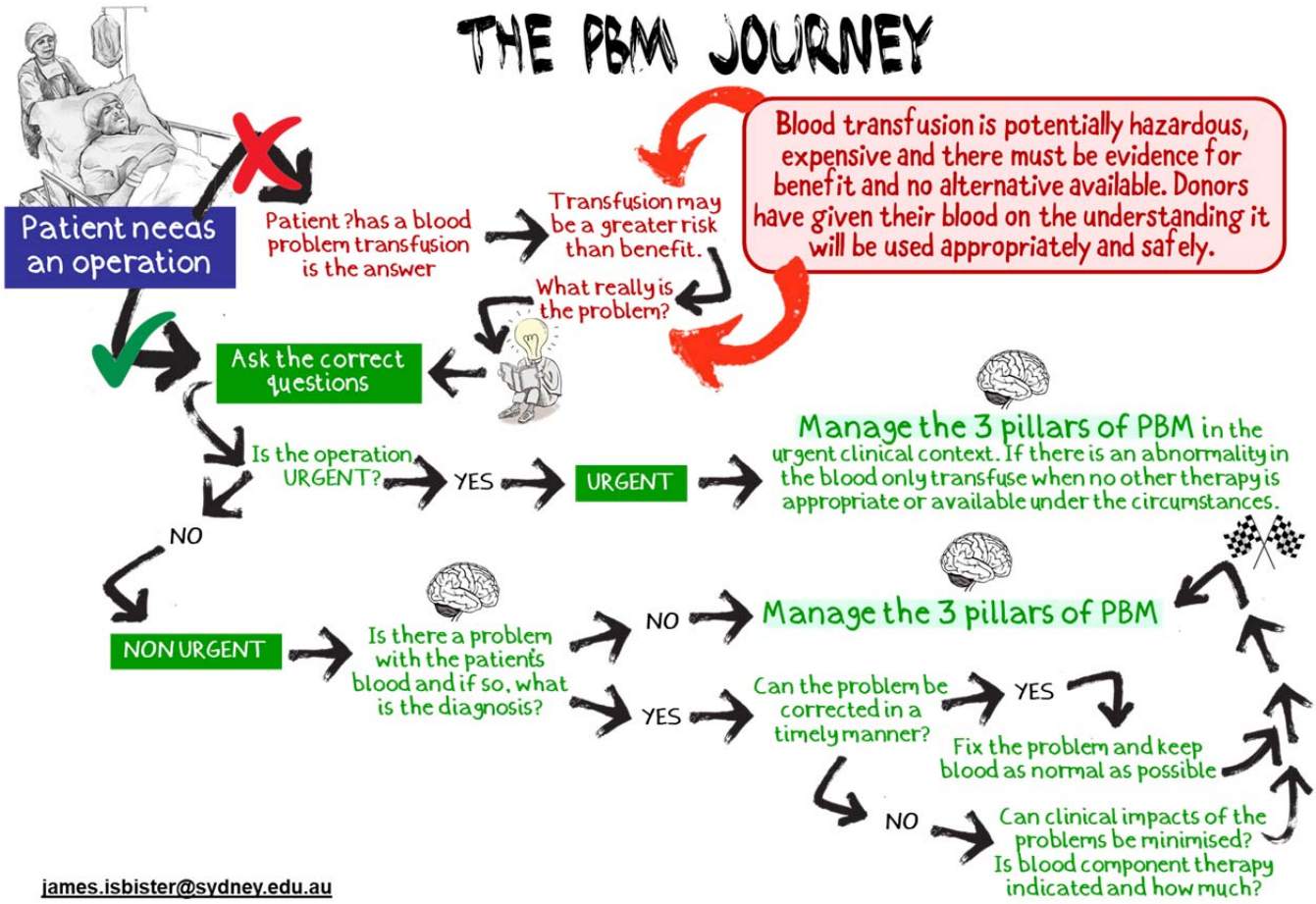
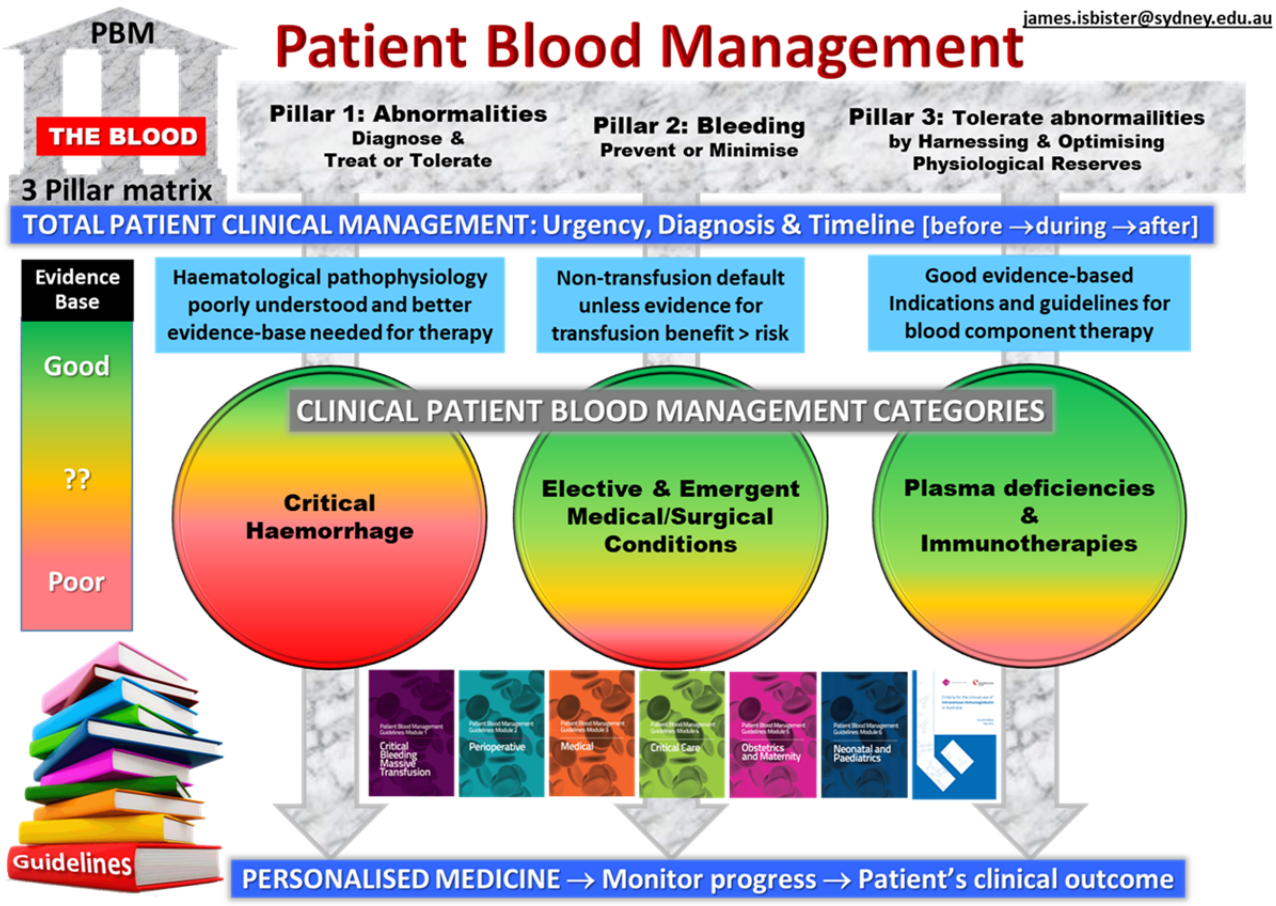
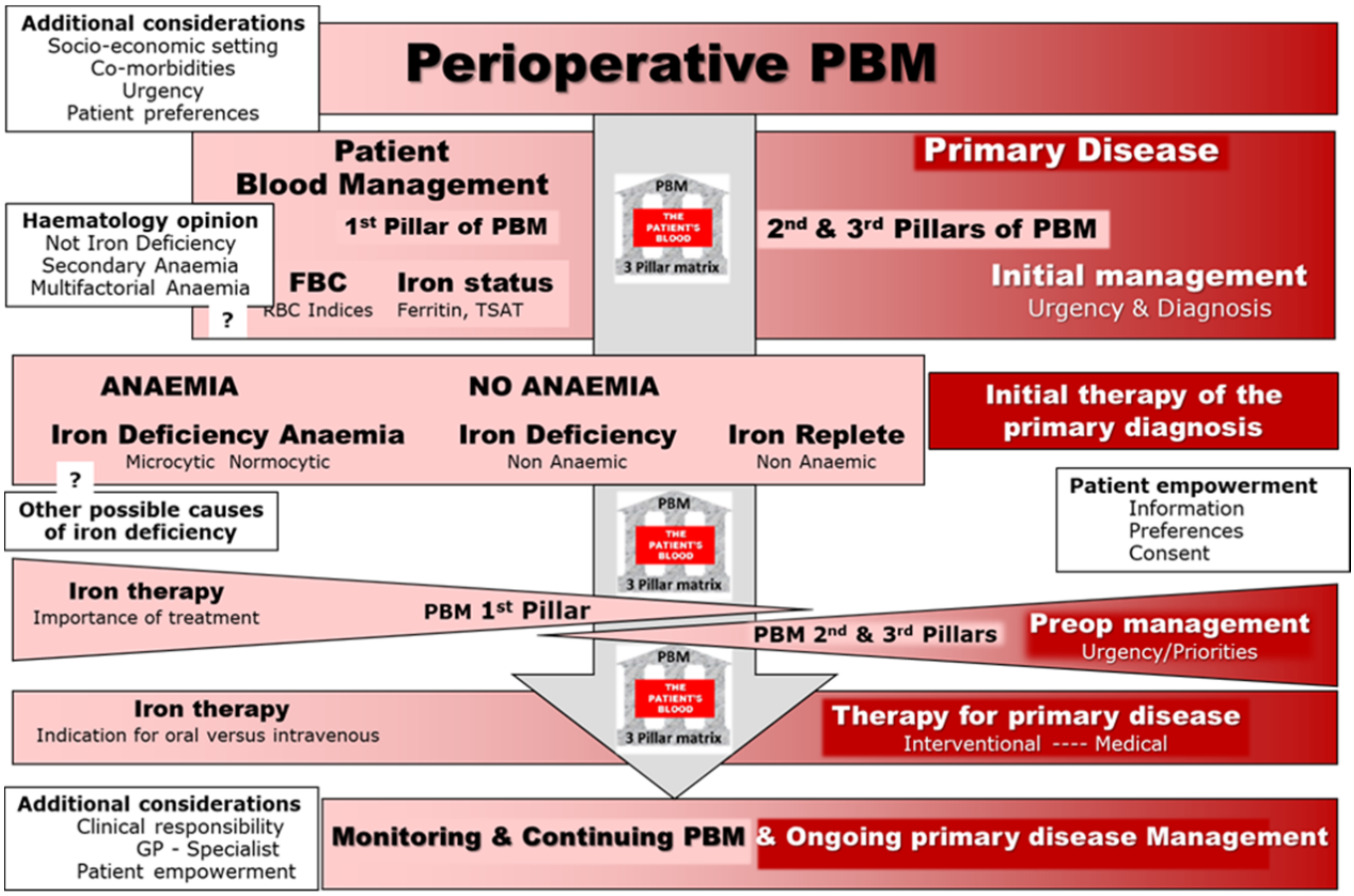

THE PBM JOURNEY
Patient needs an operation
Patient ?has a blood problem transfusion is the answer
Transfusion may be a greater risk than benefit.
What really is the problem?
Blood transfusion is potentially hazardous, expensive and there must be evidence for benefit and no alternative available. Donors have given their blood on the understanding it will be used appropriately and safely.
Ask the correct questions
Is the operation URGENT?
YES
URGENT
Manage the 3 pillars of PBM in the urgent clinical context. If there is an abnormality in the blood only transfuse when no other therapy is appropriate or available under the circumstances.
NO
NON URGENT
Is there a problem with the patients blood and if so, what is the diagnosis?
NO
Manage the 3 pillars of PBM
YES
Can the problem be corrected in a timely manner?
YES
Fix the problem and keep blood as normal as possible
NO
Can clinical impacts of the problems be minimised? Is blood component therapy indicated and how much?
james.isbister@sydney.edu.au


PBM
THE BLOOD
3 Pillar matrix
Patient Blood Management
james.isbister@sydney.edu.au
Pillar 1: Abnormalities
Diagnose &
Treat or Tolerate
Pillar 2: Bleeding
Prevent or Minimise
Pillar 3: Tolerate abnormailities
by Harnessing & Optimising
Physiological Reserves
TOTAL PATIENT CLINICAL MANAGEMENT: Urgency, Diagnosis & Timeline [before →during →after]
Evidence Base
Good
??
Poor
Haematological pathophysiology poorly understood and better evidence-base needed for therapy
Non-transfusion default unless evidence for transfusion benefit > risk
Good evidence-based Indications and guidelines for blood component therapy
CLINICAL PATIENT BLOOD MANAGEMENT CATEGORIES
Critical Haemorrhage
Elective & Emergent Medical/Surgical Conditions
Plasma deficiencies & Immunotherapies
Guidelines
Critical Bleeding Massive Transfusion
Perioperative
Medical
Critical Care
Obstetrics and Maternity
Neonatal and Paediatrics
PERSONALISED MEDICINE → Monitor progress → Patient's clinical outcome


Additional considerations
Socio-economic setting
Co-morbidities
Urgency
Patient preferences
Perioperative PBM
Patient Blood Management
1st Pillar of PBM
FBC
RBC Indices
Iron status
Ferritin, TSAT
Haematology opinion
Not Iron Deficiency
Secondary Anaemia
Multifactorial Anaemia
?
PBM
THE PATIENT'S BLOOD
3 Pillar matrix
Primary Disease
2nd & 3rd Pillars of PBM
Initial management
Urgency & Diagnosis
ANAEMIA
Iron Deficiency Anaemia
Microcytic Normocytic
NO ANAEMIA
Iron Deficiency
Non Anaemic
Iron Replete
Non Anaemic
Initial therapy of the primary diagnosis
?
Other possible causes of iron deficiency
Patient empowerment
Information
Preferences
Consent
Iron therapy
Importance of treatment
PBM 1st Pillar
PBM 2nd & 3rd Pillars
Preop management
Urgency/Priorities
Iron therapy
Indication for oral versus intravenous
Therapy for primary disease
Interventional ---- Medical
Additional considerations
Clinical responsibility
GP - Specialist
Patient empowerment
Monitoring & Continuing PBM & Ongoing primary disease Management

# Allergy and Anaesthetics


## Dr Peter Cooke

Department of Anaesthesia and Perioperative Medicine [Auckland Anaesthetic Allergy Clinic]
Auckland City Hospital, New Zealand


**Allergic Disease and Anaesthesia**

My talk reviews a range of allergic diseases and their anaesthetic implications.

Towards the end of the talk brief mention is made of two other topics.

- The recently released NAP 6 report
- The current controversy surrounding selection of the prophylactic antibiotic in patients with penicillin and or cephalosporin allergy

**Introduction**

The immune system is principally about defence of the body against invasion either from pathogens.

There are anatomical (intact skin for example) and physiological barriers. These defences are interlocked with other cellular and humoral elements that have been categorised as belonging either to the innate or the adaptive immune systems. Needless to say, these systems are interrelated.

The cellular elements in the innate system are derived from progenitor lymphoid tissue that includes macrophages, neutrophils, mast cells, natural killer cells and dendritic cells. Dendritic cells also become antigen presenting cells. These cell either phagocytise to destroy or produce chemicals such as cytokines that either destroy or help destroy invaders. Natural killer cells also control cells that have become altered such as cancer cells. The main humoral elements in the innate system are complement, mannose binding lectin, LPS binding protein, C reactive proteins and antimicrobial peptides. Lipopolysaccharide binding protein attaches itself to the bacterial cell wall and this facilitates destruction of the bacteria by monocytes. Macrophages and adipose tissues induce the release of C reactive protein (an acute phase protein) by the liver and this activates complement and binds to lysophosphatidylcholine expressed on dying cells and some bacteria. Mannose binding lectin recognises the carbohydrate pattern on many microbes, bacteria, viruses, protozoa and fungi and activates complement. Antimicrobial peptides have cell wall and cytoplasmic targets. Complement enhances or helps phagocytic cells, promotes the overall immune response and can also attack invaders directly.

The adaptive immune systems are comprised of cellular and humoral elements. The cellular elements T cells that confer specific cell mediated defense and the B cells produce antibodies that confer humoral specific defense.

The immune system can go wrong in at least three ways resulting in either an inability to protect, a misguided war on self or an overzealous reaction to foreign antigen. These malfunctions result in infection as a result of immunodeficiency, autoimmune disease or allergic disease respectively.

The most common allergic diseases are IgE mediated diseases. Plasma cells manufacture IgE to an allergen that is then distributed to mast cells and basophils. There is a strong genetic predisposition as well as environmental influences that result in allergic conditions. It is no surprise then that many of the diseases involve anatomical locations that in which mast cells are found.

For example in pollen induced rhinitis the pollen amount exceeds the mucosa's ability to remove it, and a small percentage of it gains entry to the sub mucosa. An interaction then occurs with a number of different types of antigen presenting cells that include macrophages, lymphocytes, monocytes and dendritic cells. The pollen antigen is then presented to circulating T cells that react with the antigen and transforms into an antigen specific T helper 2 cells. The T helper 2 cell then releases its signature cytokines that result in eosinophils production being increased

and mast cells being stimulated. The antigen specific T helper cell induces a class switching of B cells that now produce antibody to the pollen antigen that is redistributed to mast cells where it binds to high affinity receptors. The stage is now set for mast cell release by further antigen exposure. The mast cell releases preformed and newly generated substances and certain specific transcription processes take place that result in specific cytokine and chemokine releases. The symptoms of allergic rhinitis result.

T helper 2 cells are involved in IgE allergic disease as well as parasitic infections. T helper 1 cells are more involved with response to infection by bacterial pathogens. Some animal work suggest that general anesthesia may reduce Th-2 activity and recent study has shown that the incidence of allergic disease was reduced in a cohort of patients exposed to anaesthesia before the age of 1.

**Food allergy**
Reactions to food can be classified as either toxic or immune mediated or non-immune mediated.

The immune mediated ones can be broken down into the IgE and non-IgE mediated. Food protein induced entercolitis is the most common non-IgE related cell mediated allergy. Symptoms may be acute or chronic and mainly involve vomiting and dehydration. Some of these children may be seen referred as "an acute abdomen". IgE mediated food allergy is associated with dermatitis, asthma, allergic rhinitis. Milk, egg, soy, wheat usually start in childhood, and often resolve whereas peanut and tree nut allergy can develop at any point. Fish and shellfish allergy is more common in adulthood. Peanut and tree nut allergies account for 90% of fatalities from food allergy.

Obtaining and documenting the history of food allergy is important because patients need food and may otherwise inadvertently be offered the wrong food whilst in hospital. If the patient has had anaesthesia or sedatives, the patient's self-policing ability may be impaired. The occurrence of a food allergic episode prior to elective surgery might result in delay. Food allergy patients as mentioned above may have associated conditions such as asthma so these conditions need to be identified. There has been a case of cross contamination from anaesthetist to patient in a patient with peanut allergy who then developed a reaction. Many food allergy patients have an "Epipen". They are likely to bring it to hospital and we at least need to identify whether they have or not and work out whether they or the staff will administer adrenaline in case of a reaction.

There have been concerns about cross reactivity of certain anaesthetic drugs or substances cross reacting with food allergy patients. The most well-known and valid concern relates to kiwifruit, banana, avocado and chestnut allergy and all of these can be associated with latex allergy. Kiwifruit allergy patients are usually sensitive to birch pollen as well. There are 200 epitopes in Latex and similarities with the epitopes in the various fruits have been identified.

Anaesthetists have had concerns about egg, soy and peanut allergy being related to propofol allergy but the consensus is that the risk of allergy to propofol is no more that the populations risk in these patients. Seafood and shellfish allergy are not thought to be at risk of allergy to either radio-contrast media or povodine iodine but in patients with a definite and serious allergy to fish consideration should be given to protamine testing before its use.

Gelofusin is now not used so often but there is an association between red meal allergy and Gelofusin allergy with a number of cases. The patients were all positive for the specific IgE alpha gelatin.

**Atopic dermatitis**
The main features of this condition are pruritus and eczematous rashes. The dermatitis is chronic. It is associated with asthma, allergic rhinitis and food allergy. The condition is strongly hereditary but the traits are multifactorial. The barrier function of the skin is impaired and there is a general overexpression of T helper cell cytokines in this patient group. Patch testing is often positive for pollen and house dust mite. In addition there are hormonal, emotional, seasonal, dietary and climatic factors that contribute. There are diagnostic criteria where 3 of the major and 3 of the minor criteria are required in order to make the diagnosis. But In experienced hands diagnosis is straightforward. Recurrent conjunctivitis and keratoconus are two on the manifestations of atopia and the disease has a wide variety of expressions. The mainstay of treatment is topical corticosteroids. In severe cases systemic T-cell-suppressing therapies, such as azathioprine, methotrexate, mycophenolate mofetil and cyclosporine, are effective but limited by side effects. A new humanised monoclonal antibody, dupilumab, that targets interleukin-4

receptor alpha (IL-4R-alpha) is finding a place for severe cases with great improvements being described in very severe cases.

As anaesthetists we may have a role in ensuring that the skin condition and or related infection is under control prior to surgery. There is a risk of potential contamination of procedures such as an IV insertion from surrounding skin disease and a higher risk of staphylococcal infection. There is some evidence that atopic patients are more likely to have perioperative anaphylaxis in response to latex, propofol and ketamine exposure.

**Urticaria**

The medical condition urticaria is characterized by the development of wheals, angioedema or both. It is not urticaria caused by anaphylaxis, auto inflammatory syndromes, urticarial vasculitis or bradykinin mediated hereditary angioedema. The wheal in a patient with urticaria has 3 typical features: central swelling of variable size surrounded by erythema, itching and the fact that each individual wheal lasts a variable period of time, usually no longer than 24 hours. Angioedema in urticaria patients is a sudden swelling in the lower dermis, sub-cutis or mucous membranes, pain and a resolution that takes longer than the urticaria, typically up to 72 hours. Urticaria is regarded as acute if the rashes occur for less than six weeks and chronic if they continue to occur for a longer period. Urticaria can be classified as spontaneous or inducible and the inducible forms are induced by a number of different agents including cold, pressure, the sun, heat, water, and vibration. It is a mast cell driven disorder with release of histamine, platelet activating factor and cytokines.

Acute urticaria may have an underlying cause such as type 1 (IgE) food allergy, a response to NSAIDS or anaesthetic drugs. Morphine is a potent trigger of urticaria.

There are a number of similar looking but different disease conditions that need to be excluded and there may be underlying causes of chronic urticaria such as bacterial or fungal infection, H pylori infection. Underlying malignancy is not common. Diagnosis of the condition will require expert dermatology and immunology input, but not necessarily a raft of tests.

Treatment of chronic urticaria consists of avoidance of environmental triggers, in the case of inducible forms, reducing physical and emotional stress, and symptomatic pharmacological treatment. Second generation, non-sedating antihistamines such as loratadine and cetirizine are often used continuously for long periods, with little apparent risk of harm. Omalizumab which is a recombinant DNA-derived humanized IgG1k monoclonal antibody that specifically binds to free human (IgE) in the blood and interstitial fluid and to membrane-bound form of IgE (mIgE) on the surface of mIgE-expressing B lymphocytes. Cyclosporine reduces mast cell release but side effects limit its use. Montelukast a leukotriene inhibitor has been used in conjunction with antihistamines. Antihistamines may also be of value in preventing inducible urticaria prior to planned exposure to the inducing agent such as excercise of cold. Higher dose antihisamines have also been found to be helpful for some patients.

As anaesthetists we see urticaria in response to our medications. Antihistamines should be continued in cases of chronic urticaria and selection of medications with less propensity to produce urticaria may be helpful. Documentation of prior assessement by a dermatologist or allergologist would be valuable with a view to ensuring that treatment is optimised. Avoidance of physical factors that could induce urticaria in particular patients is important. This is particularly important in cases of urticaria that are associated with angioedema. Tranexamic acid and steroids are also helpful.

**Allergic Rhinosinusitis**

This is a common and costly condition, often associated with conjunctivitis and asthma with a prevalence approaching 40%. The nose acts as a barrier to inhaled particles so is an easy target for immunodysfunction.

Allergic rhinitis is a 2 type inflammatory disorder of the nasal mucosa and is IgE mediated. Dendritic cells located within the nasal mucosa capture the allergens and present them to T lymphocytes in the draining lymph nodes. This precipitates B cell switching, IgE to the allergen and distribtion to the mast cells. Eosinohils, CD4+T cells and basophils also are drawn to the submucosa in response to mast cell release. Important mediators are PGD2, LTC, leukotriene C4 and cytokines that include tumour necrosis factor alpha.

Symptoms of allergic rhinitis tend to build up over a period of prolonged exposure, a process known as priming. The major symptoms are nasal blockage, rhinorrhea, sneezing and itching, disturbed sleep, and reduced ability to learn and work. Many patients have identifiable allergies on skin prick or specific IgE testing, but in some the allergen is not identified.

Other causes of nasal congestion include preganancy, oral contraceptives, hypothyroidism and rhinitis medicamentosa. Unilateral nasal blockage may signal Wegeners granulomatosis, foreign body in the nose or deviated nasal septum.

Acute rhino-sinusitis is most frequently caused by infection with respiratory viruses, but some progress to bacterial infection. Chronic rhino-sinusitis, is most frequently an inflammation secondary to allergic rhinitis and the consequent blocking of the ostea to the sinuses with impairment of mucociliary clearance. The common symptoms of sinusitis are anosmia, facial pain and pressure.

There are a number of other variants including eosinophilic rhinosinisitis, allergic fungal sinusitis and aspirin-exacerbated disease. Nasal polyps form to a variable extent with these conditions.

Management includes avoidance of known allergens, saline douching, intranasal steroids and sodium cromoglycolate. The older steroids beclomethasone, budesonide have now been unsurpassed by new steroids, like fluticasone and mometasone, that are more potent but also more comprehensively metabolised by the liver minimising systemic effects. Oral antihistamines may be helpful. Antileukotienes are of 2 types, those that inhibit leukotriene formation and those that block the actions of leukotrienes. Nasal decongestants may be helpful - they work by reducing the swelling of the mucosa induced by vasodilation but they are safe for short term use. Systemic corticosteroids generally work well but their side effects prevent long term use. Some patients benefit from a desensitization programme.

Many anaesthetists are involved with the surgical management of these conditions and will have noted patients that these patients are frequently prescribed preoperative antibiotics and systemic steroids.

Because the nasal mucosa and sinuses have a rich capillary network, great emphasis is placed on vasoconstrictors intra-operatively and anaesthesia with a low to normal rather than higher blood pressure target. There is a literature on anaesthesia for nose and sinus surgery.

Nasal disease can contribute to OSA and patency of the nose. Should be part of every anaesthetic assessment?

**Asthma**

Asthma has been defined as a disorder of variable intensity, typified by sentinel symptoms, airway obstruction, inflammation and hyper-responsiveness. The primary symptoms of asthma are shortness of breath, wheeze, cough, increased sputum production and chest pain.

A recent report suggests that the term asthma should be viewed more as an overall descriptive term of the various pulmonary conditions, rather like arthritis is an overarching term that covers a variety of conditions.

This suggested approach incorporates many diseases such as emphysema, chronic bronchitis and allergic asthma under one umbrella and challenges us to think carefully about the symptoms, traits or characteristics that our individual asthma patient in front of us manifests. There are also many new bronchodilator medications, long and short acting, and many possibilities for combinations of these. Only some of our patients will benefit from bronchodilators or inhaled steroids. Patients may have other comorbidities for example asthma that impacts on respiratory function; others may have laryngeal dysfunction or remodeled restrictive bronchi with no reversibility. The Lancet commission paper is not directed at anesthetists but the thinking therein is likely to impact our practice in the future. It suggests new methods to identify patients at significant risk of severe attacks and many new diagnostic approaches.

Allergic asthma for example is characterized by eosinophilic disease that can be detected by airway sampling or in the blood. Whether or not there is bronchial smooth muscle reactivity and response to bronchodilator is also important.

How good are we at assessing our patient's asthma control prior to surgery? A useful online patient questionnaire is presented.

On average asthma patients present few problems intra-operatively but when problems arise they can be severe. Severe bronchospasm may be a manifestation of anaphylaxis, but it may also be a manifestation of asthma. Various treatments algorithms for severe bronchospasm are discussed.

**Mastocytosis**

This is a mast cell disorder associated with an increase in the number of mast cells and abnormality of c-kit trans-membrane mast cell wall protein in 50 to 90% of cases. This abnormality is a result of a mutation that can be identified. Mastocytosis is a heterogeneous group of disorders but can be categorized as either systemic or cutaneous depending on whether the excess mast cells are mainly in skin or internal organs. The cutaneous form presents often in childhood, the systemic in adulthood.

The majority of these patients are identified as a result of their skin disorder but others present with symptoms relating to mast cell release and occasionally with perioperative anaphylaxis. In mastocytosis there is a propensity for mast cell mediators to be released more readily usually without any requirement for IgE mediated triggered release. The mast cell tryptase is frequently chronically elevated. Release of mast cells can be triggered by stress, mechanical trauma such as a tourniquet, cold stress and drugs that are known to cause dose related mast cell release especially mivacurium and atracurium. Many other drugs have been implicated in reactions including morphine, pethidine, nefopam, NSAIDs and radio-contrast media, so a cautious approach is recommended.

**Severe cutaneous reactions**

These are reactions that anaesthetists need to be aware of in a patient's history in order to ensure that the culprit drugs are not re-administered. **DRESS syndrome** is short for Drug Reaction with Eosinophila with Systemic Symptoms. It occurs typically 6-8 weeks after exposure and the symptoms include fever, an itchy morbilliform rash, enlarged and sometimes painful lymph nodes and other symptoms due to variable internal organ involvement. Allopurinol, sulfasalazine and minocycline are the most frequent culprit drugs but it can occur after diclofenac, celecoxib or ibuprofen. It has a 10% mortality. It is a cell mediated type 4 (sub type 4b) reaction. It is T cell mediated but eosinophils play a large part in the reaction. There is a genetic predisposition and is more common in South East Asia.

**Stevens Johnson syndrome** and **toxic epidermal necrolysis** form a spectrum of disease with SJS being less severe. Early symptoms of SJS include fever and flu-like symptoms. A few days later the skin begins to blister and peel forming painful raw areas. Mucous membranes such as the mouth, are involved. Complications include dehydration, sepsis pneumonia and multiple organ failure. SJS and TEN most often begin between 4 and 28 days after culprit drug administration with a long list of possible drugs. The ones that anaesthetists might administer include: vancomycin, diclofenac, penicillins, valedecoxib, ibuprofen, sulphonamides and paracetamol. Other causes are viral or fungal infections. The reaction is a cell mediated, type 4 (sub type 4c), one that involves natural killer cell attack on self.

**Acute generalized exanthematous pustulosis** is a rare type 4 (sub type d) cell mediated skin reaction that in 90% of cases is related to medication administration. Skin eruptions occur five days after a medication is started. These eruptions are small white or red elevations of the skin that contain pus. The skin lesions usually resolve within 1–3 days of stopping the offending medication but more severe cases are associated with a more persistent disorder that may be complicated by secondary skin infections and involvement of the liver, lung and kidney. A range of drugs have been implicated but mainly antibiotics, including cephalosporins, and anti-inflammatory medication. AGEP is a type 4 d reaction, (T cell mediated) that then stimulates neutrophils to attack self-tissues.

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# Paediatric Pain Management


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Pain is a common symptom in both hospitalised children and children in the community. An estimated 40% of infants, children and adolescents experience pain at least once a week and 15-20% suffer from chronic pain (1). Pain is often under-recognised and undertreated in children due to inadequate pain assessment and a paucity of paediatric-focused pain services.

Studies in animals and neonates have demonstrated the adverse short and long-term consequences of poorly managed pain in early life (2). Recognition of the psychosocial impact of poorly managed pain in childhood and adolescence is also important. Psychosocial factors also influence pain reporting and a child's ability to cope with their pain (4). Parental factors also influence their child's pain experience and pain-related behaviour.

Pain management should follow a biopsychosocial model that appreciates the distress and suffering associated with pain. Pharmacological management must take into account paediatric pharmacokinetic and pharmacodynamic differences that determine analgesic efficacy. The use of opioids in children requires careful titration and appreciation of pharmacogenomic factors that influence opioid metabolism. The FDA have issued strong recommendations regarding the specific use of codeine and tramadol in children. However, all opioids are potentially dangerous when administered to children at greater risk of respiratory depression in the wrong setting e.g. children with OSA, underlying respiratory or neuromuscular disease (4). High-risk children having day-stay surgery should be admitted to hospital overnight for monitoring and to assess opioid response and sensitivity prior to discharge. Psychological support and specific interventions are useful to address pain-related anxiety, catastrophising, and fear-avoidance that may hinder postoperative recovery, mobilisation, and discharge from hospital.

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# Managing Aviation emergencies


## Dr Ben Johnston

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Anaesthetists are trained and experienced in managing a range of medical emergencies and are ideally suited to volunteer assistance during a medical emergency on board an airline flight. However an airline passenger cabin is an unfamiliar working environment for doctors; when you hear the crew page "Is there a doctor on board?" it is likely several questions will be running through your head. What kinds of problems can I expect to encounter in an airline cabin? What is my liability if things go wrong? What equipment and drugs are available to me? What is my role in decision making about aircraft diversions?

This presentation will answer these questions from the perspective of international airlines. The session will also briefly discuss prevention of in-flight medical events, with a focus on decision making around fitness to fly following short-stay surgical procedures.

# Updates on Airway Management


## Dr Gemma Malpas

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**Introduction**

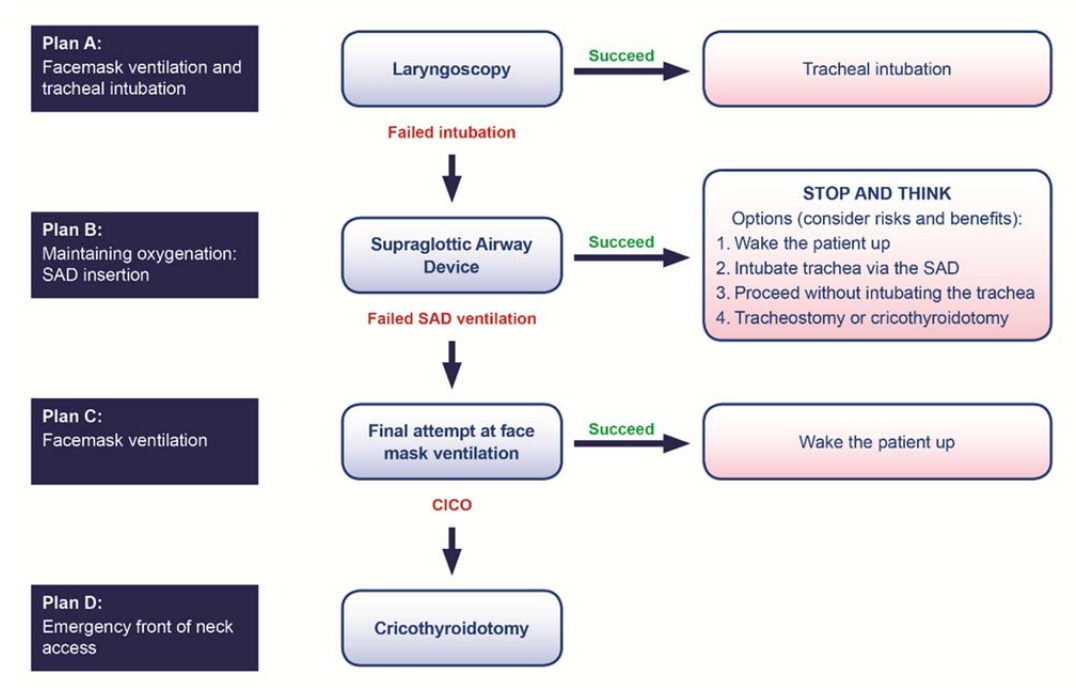
Despite events only occurring infrequently, complications in airway management remain an important contributor to morbidity and mortality during anaesthesia. Complications related to airway management have been well described and been subject to numerous reviews. The accurate data we have suggests that face-mask ventilation fails in 1 in 700 cases, difficulties inserting or ventilating via a supraglottic airway fails in 1 in 50 cases, and tracheal intubation fails in 1 in 1000 to 1 in 2000 cases. These difficulties result in an incidence of CICO of around 1 in 5,000 to 1 in 10,000 cases, with CICO having been reported as responsible for 25% of all anaesthesia- related deaths (1).

Complications related to airway management occur throughout the peri-operative journey. Closed claims databases (2) and the NAP4 (3) reported a similar distribution of airway events with 75% of all events occurring during induction of anaesthesia, and approximately 25% of events occurring during maintenance, extubation and post-anaesthesia recovery.

This presentation aims to cover recent updates in management, covering current airway guidelines as well as recent recommendations regarding airway management techniques

**Guidelines**

There are now many airway management guidelines in publication regarding the management of the difficult airway. The latest update of the DAS guidelines was published in 2015. These guidelines depict a plan A, B, C, D approach suggesting fluid movement through Facemask ventilation and tracheal intubation, maintaining oxygenation through SAD insertion, returning to Facemask ventilation, ultimately resulting in emergency front of neck access if all methods fail to achieve alveolar oxygen delivery. Branching off the flow chart are suggestions for management if any of the techniques result in successful oxygenation.

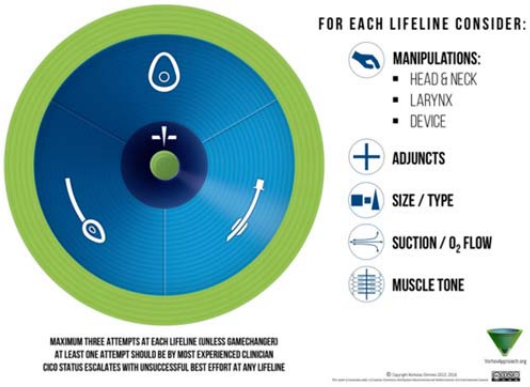



*The Difficult Airway Society Intubation guidelines. This flowchart forms part of the DAS guidelines for unanticipated difficult intubation in adults and should be used in conjunction with text.(4)*

The Vortex approach was designed and developed by Nick Chrimes in Melbourne as a cognitive aid to the management of encountered airway difficulties.

The vortex implementation tool is based on the premise that there are only 3 upper airway 'lifelines' (non-surgical techniques) by which alveolar oxygen delivery can be established and confirmed; facemask, supraglottic airway and an endotracheal tube. If a 'best effort' at each of these three lifelines is unsuccessful, then a can't intubate, can't oxygenate situation (CICO) situation exists and 'CICO rescue' or 'emergency front-of-neck-access' must be initiated.

The design of the vortex is such that with every unsuccessful technique, or 'loss of lifeline' CICO escalates and you move further down into the vortex. If, however, a technique results in successful alveolar oxygen delivery, you move out to the 'green zone' giving the opportunity to reassess the situation and consider the available options (such as wake-up, advanced techniques, more experienced help, FONA)(5).

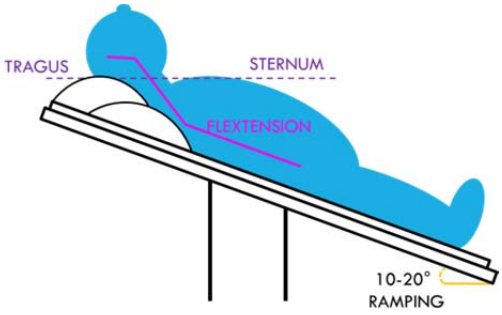



In addition to these guidelines, new guidelines for the management of tracheal intubation in critically ill adults have recently been published in the BJA. These guidelines describe a comprehensive strategy to optimise oxygenation, airway management and tracheal intubation in critically ill adults, in all hospital locations. They stress the role of the airway team, a shared mental model, planning, and communication throughout airway management.

There is currently an international, multidisciplinary group of airway specialist working together to produce a guideline that reflects, as much as possible, the consensus of existing published airway guidelines and can be applied to all episodes of airway care, across boundaries of geography, clinical discipline or context. They aim to present the final universal guidelines at the World Airway Management Meeting (WAMM) in Amsterdam in November 2019.

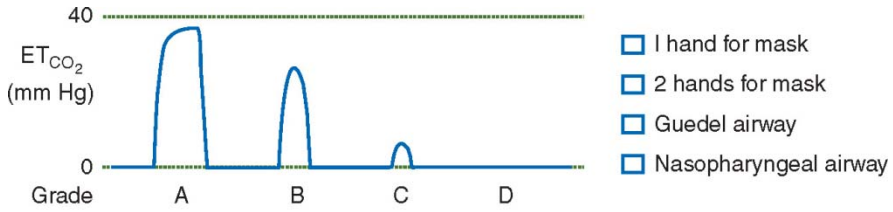
**Positioning**

Recently Tim Cook and colleagues have described a new term that describes anatomically the position required to improve the success of tracheal intubation. They have coined the term 'flextension' (6). This describes the position of the spine that aligns the oral, pharyngeal and laryngeal axis to create as 'straight' a line between the mouth and the glottic opening. This positioning describes flexion at the thoraco-cervical junction and extension at the atlanto-occipital junction. Ramping is useful for both physiological reasons (such as improving FRC, reducing aspiration risk) as well as improving upper airway patency and access in obese patients.




**Bag-Mask ventilation**

Lim et al have recently taken on the challenge of attempting to improve the communication around bag-mask ventilation. Through this they have proposed a new definition for difficult bag-mask ventilation. They propose that difficult mask ventilation is when 'the best attempt results in inadequate or absent exhaled CO2' (7). Using this definition, they propose a new grading scale for bag-mask ventilation




Grade A: plateau present
Grade B: no plateau, $ET_{CO_2} \geq 10$mm Hg
Grade C: no plateau, $ET_{CO_2} < 10$mm Hg
Grade D: no $ET_{CO_2}$

| Grade | Definition |
|---|---|
| A | Plateau present |
| B | No plateau, $ET_{CO_2} \geq 10$mmHg |
| C | No plateau, $ET_{CO_2} \leq 10$mmHg |
| D | No $ET_{CO_2}$ |

This new scale suggests grading the efficiency of bag-mask ventilation on the generation of the exhaled CO2 capnograph. Describing the capnograph allows for standardized and objective communication of mask ventilation outcomes.

The use of this scale does not preclude the subjective comments on mask ventilation such as 'easy' or 'difficult', but explains and justifies them with evidence from the monitor. The grades simply label the sequential phases of the capnograph. As ventilation improves, dead space, mixed and then pure alveolar gas produce the flat=line, upstroke, and plateau. The plateau phase (grade A) is already recognize as a marker of effective mask ventilation (7).

**Apnoeic Oxygenation**

There has been recent interest on the potential for high-flow nasal oxygen delivered by cannulae at up to 60l/min to prolong the safe apnoeic time. This has been shown to be effective during elective apnoeic oxygenation of patients with a normal BMI. However, its value in critically ill hypoxic patients or those undergoing RSI is less clear.

It is worth acknowledging that there are simple alternative methods to deliver oxygen during airway management. These include the use of nasal cannulae at a flow of ≥ 15 l/min once the patient is unconscious or buccal oxygen. The current evidence indicates that oxygenation techniques should be used for all patients in whom difficult airway management is anticipated, and arguably in all patients undergoing general anaesthesia. This is particularly relevant with high oxygen demand (the obese, critically ill, septic and pregnant), those who would be intolerant of hypoxaemia (pregnancy, neurological injury) and in those in whom airway management may be predicted to be prolonged.

**Video- Laryngoscopy**

Within the last 2 years several meta-analyses have been published comparing video-laryngoscopy to direct-laryngoscopy. Notably, Lewis et al. reviewed the literature of all published cases within the operating room (8), and Arulkumaran et al, outside the OR (9).

Lewis et al. concluded that Video-laryngoscopes may reduce the number of failed intubations, particularly among patients presenting with a difficult airway. However, following a sub-group analysis, the only device that did show an improved first-pass success rate was the CMAC video-laryngoscope with the Macintosh-type blade. Currently, no evidence indicates that the use of a video-laryngoscope reduces the number of intubation attempts, the incidence of hypoxia, or respiratory complications.

With a number of video-laryngoscopes currently available for use in clinical areas, communication and documentation of the devices used must be clear. Focus needs to be made on education on the use of each specific device as well as clear documentation when successful intubation is achieved.

**Awake intubation**

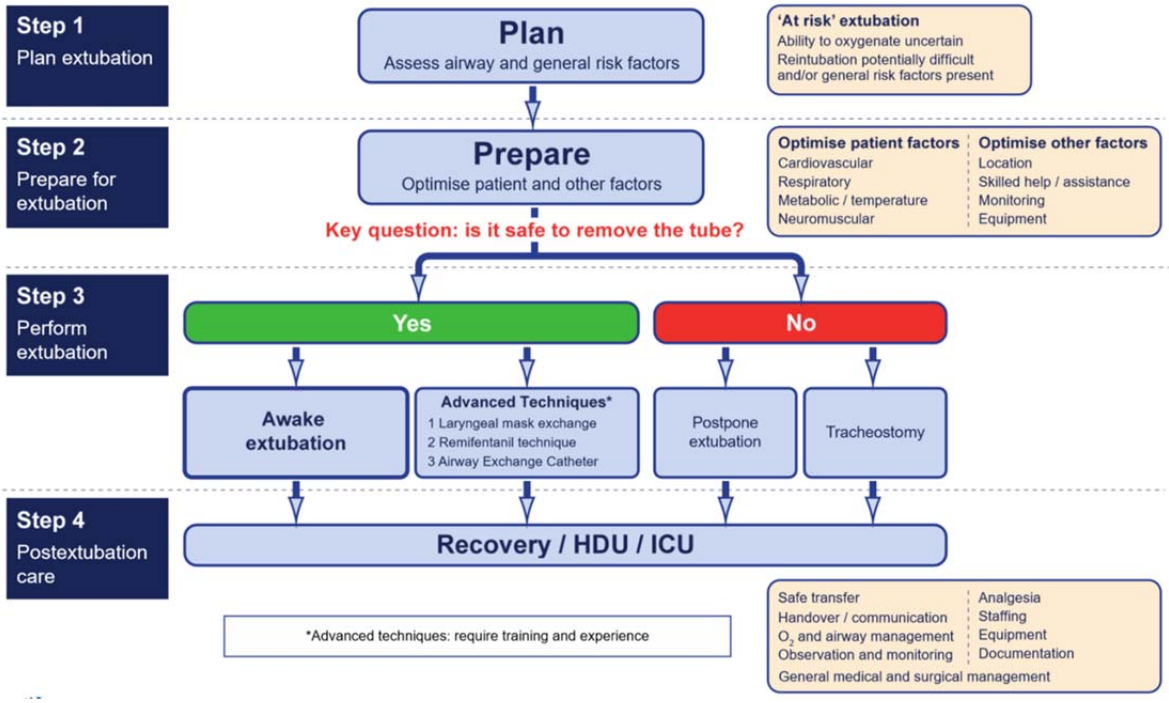
Alhomary et al. recently published a meta-analysis comparing the use of video-laryngoscopy to flexible bronchoscopy for awake intubations (10). There was significant heterogenicity between the studies, and significantly fewer studies published on the use of video-laryngoscopy, however, they did conclude that video-laryngoscopy appears to be safe for awake intubations, and can achieve overall and first-attempt success rates comparable to fiberoptic bronchoscopy.

It is important to be aware of the limitations of video-laryngoscopy for awake intubation.

- Video-laryngoscopes may not be the correct size or shape to match the patient's airway anatomy
- They aren't ideal in limited mouth opening
- They may be difficult or impossible to insert in patients with significant neck flexion deformities
- They have been associated with pharyngeal hyperreflexia

**Extubation**

The majority of complications that occur during extubation and emergence are minor, however, a small and significant number have resulted in serious morbidity or mortality. Up to 25% of all airway events have been related to the extubation and recovery period (3). The Difficult Airway Society developed guidelines for the safe management of tracheal extubation in adults in peri-operative practice. These guidelines discuss potential problems that may arising during extubation and recovery, and promote a strategic approach to extubation. Strategies to the extubation of the difficult airway are outline in a clear concise manner (11).




*The Difficult Airway Society Intubation guidelines. This flowchart forms part of the DAS guidelines for unanticipated difficult intubation in adults and should be used in conjunction with text(11)*

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# Update on Regional Anaesthesia


## Dr Kelly Byrne

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**The rise of the "good enough" block**

Regional anaesthesia is as vulnerable to fashions as any other part of anaesthesia or indeed medicine. The current trend has been for an expansion of fascial plane blocks. These techniques aim to place local anaesthetic in a fascial plane, which nerves run through, rather than having a specific, targeted nerve. Clearly this is unlikely to produce the same quality of analgesia or anaesthesia as when the target of the local is a major nerve plexus or a central location. In fact it is somewhat analogous to attempting to cut of the power supply to a house by blocking or severing individual power points, rather than blocking the mains power supply (which would be similar to an epidural).

However, there have been several reasons for the upsurge in interest in fascial plane blocks. Firstly, ultrasound has allowed us to clearly visualize fascial planes, often more easily and at a shallower depth than nerve targets. Secondly, despite its low incidence (estimated at 2-4 per 10,000 blocks (1)), nerve injury remains a significant concern for most regional anaesthetists. Fascial plane blocks often target sensory nerves only, where injury would be less clinically significant, and because the target is a plane, rather than a specific nerve, it is likely that the needle is significantly further away from any neural structures. Lastly, the structures that surround fascial planes, are almost by definition, usually muscles, and unlikely to be damaged from inadvertent misplacement of needle.

The current trend started with transversus abdominis plane (TAP) blocks, and these are probably the most investigated and most widely practiced fascial plan block. Despite, this history, the evidence for efficacy of these blocks is still unclear. A recent systematic review of the use of TAP catheters concluded "Because of the extremely heterogeneous nature of the studies, a specific consensus regarding their results, or the ability to construct a meta-analysis, is unviable "(2). The evidence favouring single shot TAP blocks over placebo is probably more convincing (3).

There are a number of reasons why there has been great variability in the results from studies investigating TAP blocks. While on the face of it, it seems like a very straightforward block to undertake, it is more difficult than people give it credit for, especially in the obese patient. This difficulty has probably diluted the ability of studies to show a difference between groups when a placebo-controlled trial takes place. Additionally, the comprehensiveness of the multi-modal analgesic regime that accompanies the TAP block, is highly like to influence the ability of the study to show a difference in groups in a placebo-controlled trial. This is a block, where if successful, the skin and muscle layers will be anaesthetized. For any surgery that is not superficial surgery, additional analgesia will be required. If all this additional analgesia is provided by a comprehensive multimodal regime, it will be difficult for the block to make a lot of difference to the patient, as the superficial component of the pain is likely to be a minority part of the overall pain.

These aspects of a placebo-controlled study should be considered when evaluating any fascial plane block study. Unsurprisingly then the evidence base for probably the second most widely practiced block, the Pectoral (PECS) block, is actually amongst the strongest of the all the fascial plane blocks (4,5). Because this block has predominantly been used in breast surgery, a form of superficial surgery, the analgesic benefit of the block has been relatively consistent. There has been at least one negative trial published (6), but there seemed to be significantly inequalities in the invasiveness of the surgeries between the groups, and there still seemed to be a benefit for the more invasive surgeries even though the study was not powered to show this.

The PECS block was first described by Blanco in 2011 (7,8). It is a relatively straightforward block, where local anaesthetic is deposited between pec major and pec minor (PECS I), and below pec minor (PECS II). Between 15-20ml of local is deposited between each plane, and this provides analgesia for most of the breast (the supero-medial quadrant is spared) as well as the axilla.

The two other blocks which cover the chest wall that have been recently described are the serratus anterior block (9) and the erector spinae plane block (10).

The serratus anterior block was also initially described by Blanco (9). It is essentially the same fascial plane as the PECS II block, but because of its more lateral position, the local anaesthetic deposited there tends to spread further caudad that for the PECS II block. Between 20-40ml of local anaesthetic is deposited above or below the serratus anterior muscle in the mid-axillary line. It provides analgesia from the mid-scapula to the nipple line by blocking the lateral cutaneous branch of the intercostal nerve and has been rapidly incorporated into rib fracture analgesia pathways. Its use has been described in a wide variety of surgeries from breast surgery (11) to paediatric coarctation repair (12).

The Erector spinae plane block (ESB) probably relies on the same fascial plane as the serratus anterior block but at its very posterior extension where it attaches to the transverse processes of the vertebral bodies. The aim of this block is to place the local anaesthetic under the erector spinae muscles of the back, which are made up of 3 different muscles that run just about the entire length of the trunk. While the exact mechanism of effect of the ESB is still debated in the literature, it probably allows local to track laterally and block the lateral cutaneous intercostal nerves as well as blocking the dorsal rami near the transverse process (13). There have been suggestions that the local also tracks back into the paravertebral space, but this is currently not proven (14).

While not as topical as the truncal blocks, the other essentially fascial plane block that has been prominent in the literature is the adductor canal block. This block is undertaken around the mid-thigh, and blocks the saphenous nerve, and probably at least one of the branches of the obturator nerve depending on exactly where in the adductor canal you target and what volume of local you use. Typically, 30ml of local will ensure that the entire adductor canal is covered. This block was developed to provide analgesia for total knee joint replacement without causing the quadriceps weakness that a femoral nerve block causes. When combined with a multimodal analgesic regime, it seems to provide similar analgesia to a femoral nerve block (15). Additionally, there have been recent studies that seem to show that it provides some additionally benefit in terms of analgesia and ambulation distance when added to an analgesic regime that includes high volume local infiltration (16;17). The adductor canal is also suitable for a peripheral nerve catheter and a recent study from Perth showed that a 5-day infusion post-operatively provided benefits in terms of pain scores and quality of recovery when compared to a 3-day infusion (18).

In summary, fascial plane blocks are currently enjoying wide spread popularity. They seem to have advantages in terms of ease of placement and safety. It is unreasonable to expect that they will provide the same analgesic benefits as more central blocks. Efficacy data on the individual blocks is evolving rapidly, and most will probably find their place in the analgesic armamentarium although some will be discarded along the way.

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# Cardiology Update


## Dr Fiona Stewart

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**What is new in Cardiology?**

As always there are plenty of new developments in Cardiology over the past few years. Some of the key areas by subspecialty are:

**Ischaemic Heart Disease:**
Treatment of acute coronary syndromes has become guideline driven with early angiography, dual antiplatelet therapy, aggressive statin management of lipids, β-blockers and ACE inhibition. In NZ, the development of the ANZACS-QI (All NZ ACUTE Coronary Syndrome – Quality Improvement) registry has enabled us to capture data on every patient admitted to a NZ hospital since late 2013, leading to major initiatives to improve equity of service whether by geographical location, patient ethnicity or gender. Through NHI tracking and pharmacy data bases information is also available on patient adherence to treatment in the longer term. There is increased interest in the group with ACS and non-obstructive coronary disease including Takotsubo syndrome, coronary spasm and microvascular disease.

Percutaneous intervention for chronic coronary disease is most effective for patients with haemodynamically significant disease with evidence of inducible ischaemia on functional testing or by Fractional Flow Reserve. Medical therapy is preferred for moderate coronary disease.

New generation stents are less thrombogenic. Surgery should be delayed by 1 month post PCI where possible.
Biomarkers – high sensitivity troponins and BNP have increased utility in the diagnosis and management of ACS and CHF.

PREDICT scores for prediction of cardiovascular risk are central to risk stratification in the community but this can be further improved with the addition of calcium scoring.

Statins remain the most effective preventative agents despite the groundswell of "fake news" around their safety. They do not impair cognition or cause dementia and are often not the cause of muscle aches. The risk of major cardiovascular events is reduced by 20% per year for every 1mmol/l reduction in LDL cholesterol and mortality by 10% per year. PCSK9 inhibitors are promising as highly effective new cholesterol lowering agents but their cost and administration by subcutaneous injection will limit their use.

**Valvular Heart Disease**
TAVR (transcatheter aortic valve replacement) is now recommended for intermediate as well as high risk patients. Heart Failure: BNP is increasingly useful for both the diagnosis and prognosis of patients with heart failure. Epleronone is now available as an angiotensin receptor blocker and an alternative to spironolactone giving the same benefit without the troublesome gynaecomastia. Neprilysin inhibitors are on the horizon but not yet available in NZ.

**Arrhythmias**
Monitoring systems for detecting arrhythmias have improved with the use of phone ap based devices (Kardia) and implanted monitors.

Leadless pacemakers and ICDs will reduce the long-term risks of these devices in younger patients.

AF: Rhythm control is being pursued more actively with PVI (pulmonary vein isolation) procedures using cryoablation in appropriate patients.

Rate control is important at rest and with exercise. B-blockers and diltiazem are the first line agents. Digoxin is rarely used and amiodarone is reserved for acute use or in selected high risk patients.

Anticoagulation based on a CHADS VASc score of 2 or more is recommended. Dabigatran or rivaroxaban are usually preferred to warfarin except with prosthetic valves. Bridging anticoagulation preop is only recommended with prosthetic valves and high-risk patients with a recent CVA.

For patients with a recent PCI and AF - triple therapy with aspirin, clopidogrel +NOAC for 1/12 followed by clopidogrel + NOAC until 1y then NOAC alone.

**Hypertension**

New guidelines in NZ and US are favouring treatment of BP to below 130/80. This carries an increased risk of syncope and renal impairment but is associated with fewer cardiac events. Treatment guidelines for preoperative BP control are less tight with concern about intraoperative hypotension causing myocardial ischaemia and renal impairment.

There is increasing interest in cardiac disease in the context of comorbidities. Cardio-oncology has become a recognised subspecialty with an increasing number of cardiac complications recognised from chemotherapy, hormonal and radiotherapy. Obesity is strongly associated with both diabetes and obstructive sleep apnoea. OSA is associated with a high risk of atrial fibrillation. There are newer diabetic medications some of which lower cardiac risk. Inflammation is an important risk for the development of coronary disease. HIV and autoimmune disorders such as rheumatoid arthritis therefore have an increased risk of CAD.

Cardiac genetics is a significant new frontier in cardiology. There are recognised genetic causes of arrhythmias (LQT, Brugada, Catecholaminergic VT), cardiomyopathies (DCM and HCM) and aortopathies. Work is progressing on identifying patients whose genotype may determine which treatment suits them best but has not yet become mainstream cardiology.

Despite all these advances a good history, examination, estimation of functional capacity and an ECG remain the cornerstones of good cardiac care.

# Genetics for Anaesthetists


## Dr Dean Bunbury

Department of Anaesthesia, Middlemore Hospital, Auckland, New Zealand


This talk aims to demystify many of the "omic" terms that may not have existed when AQUA attendees graduated from medical school. Using simple examples, complex and poorly defined terms such as proteomics, metabolomics, and epigenetics will be introduced and explained. This conversation will then touch on current issues and initiatives in genetics and genomics, linking their relevance to current and future clinical practice in anaesthesia. This thread will conclude with the possible relevance of "omics" to the promise of personalised medicine.

# Crisis Management for Training for First Year Doctors


## Dr Jonathan Albrett

Department of Anaesthesia, Taranaki Base Hospital, New Plymouth, New Zealand


**Sessions aims**

In this session I aim to describe how I developed my skills as an educator from the time I completed my part one exam through to today. I will outline what I think are the strengths of a clinical background in anaesthesia and/or intensive care as they pertain to teaching acute care medicine to PGY-1 and 2 doctors in New Zealand. I will outline a range of educational opportunities, that are available to all of us, that I found extremely helpful in my development as a clinical educator.

I would also like to outline the challenges that new graduate doctors face and how these differ from my own experience as an intern. Using anonymous survey results of interns I am able to assess their areas of need and how they change over the course of their first year. I will also highlight some of the resources you can use and challenges that you may face if you want to develop a similar course.

**My learning so far**

I think my development as an educator began after my completion of the primary ANZCA examination. I would give 100 to 200 power-point slide talks on Part One topics. While my intentions were good I now appreciate this was not the best use of time for either the candidate or myself. In formal teaching sessions for the next five years for both ANZCA and CICM examination candidates I employed a similar approach. Ever-increasing detailed presentations that I told candidates they didn't need to prepare for as I had what they needed!

My initial inspiration for committing to teaching my own methods started with an inspirational talk by Charles Gommersall, who suggested the best way to become an excellent teacher was to just get on with teaching and learn as you go! My first realisation that maybe training in education itself would also be of benefit was the ANZCA educators course facilitated by Maurice Hennessy. I strongly recommend this course for every anaesthetist who supervises trainees.

**The anaesthetist as clinical educator for first year doctors**

I think anaesthesia and ICU have become the major presence in the hospital senior work-force available 24 hours for deteriorating patients. Our hands-on approach and understanding of detection and treatment of a patient who deteriorates makes us ideal teachers for these practical skills. Crucial skills also include our understanding how we perform under pressure and methods to overcome human factors that can contribute to poor outcomes.

Formal training opportunities that are available to us include college workshops, short-courses, DHB, medical council and university workshops. Simulation is a particularly useful way of teaching for clinical practice. You could also consider papers, diplomas and degrees in clinical education. My personal view is these papers are extremely useful if you teaching regularly at the same time. Informal opportunities in education include watching each other teach, running teaching sessions together and surveying your learners on both their needs and your performance.

**Challenges the intern faces**

I think the intern year is one of the most exciting but stressful years of clinical practice. Potential areas of stress include;

- First job after substantial investment in terms of years, cost and stress
- Expectation versus reality of the job
- Workplace relationships
- Professional behaviours
- SMO burnout
- Student loans
- Oversupply of new graduates

- New environments
- Generational differences in attitudes to work
- Lack of "meaningful" work in some runs
- Career planning

Do not under-estimate how much they want to learn. Watching a first year doctor develop skills and confidence over the year and beyond is extremely rewarding and adds tremendous value to my own career satisfaction.

**Development of my acute care programme for house surgeons**

I returned to Taranaki as a consultant in 2010. I worked with some of the house surgeons. I was curious as to how they felt and concerned at how independent they are on the wards after-hours, especially when a patient deteriorates. Through anonymous surveys I came to appreciate just how under-prepared they felt for the job and where the gaps in their knowledge and skill were. I started a ten week programme for acute skills with the deteriorating patient. These sessions start with a few concepts and then move into case-based discussions that are designed to be immediately relevant and focused on the learner. I went on develop a monthly simulation programme. Feedback has consistently shown simulation to be the preferred teaching method after the ten week course.

I have also completed some audit work in the management of hypotension in ward patients with no treatment limitations who a first year doctor is typically asked to review. I encourage you to consider your approach to intra-operative hypotension and the marked contrast in detection and correction of hypotension your post-operative patient may experience on a ward.

**Advice for developing a teaching programme for interns**

Initially this can look overwhelming. I would encourage you to look at what your hospital provides in terms of PGY-1 and PGY-2 teaching. Your RMO unit is useful in this regard. Your prevocational educational supervisors, clinical directors and medical teachers will be able to show you what is currently available and resources you can utilise. Your own house surgeons will be the most valuable resource in terms of needs assessment. I use anonymous survey-monkey polls to establish what is needed. Your own registrars are also a useful resource.

Other groups are interested in the deteriorating patient. This includes the health round-table and the HQSC. The HQSC have developed the national EWS chart, encourage PAR teams and are auditing hospital performance. Your own DHB mortality review committee is very likely to review clinical cases that highlight delayed recognition and treatment of the deteriorating patient. Audit results and clinical cases are an excellent resource for highly relevant teaching material.

Challenges to developing a programme include;

- Time
- Funding
- Fear of failure
- Monitoring the changing needs of the intern group
- Securing the programme as an essential service

All of these challenges can be met and overcome with persistence and a genuine belief that this work is necessary, improves the intern experience and ultimately contributes to a higher standard of care for our community. I am making all of my resources available at year1doctor.com or please feel free to contact me at jonathan.albrett@tdhb.org.nz

**Conclusions**

I hope to have inspired at least one person here to develop their skills as an educator and possibly even develop or contribute to a similar teaching programme. I have been extremely fortunate that my programme has coincided with national health initiatives. I have also received some formal recognition for this work. The real rewards are in helping young doctors start their careers and watching them grow in competence and confidence.

# Sustainability in the Operating Room


## Dr Felicity Pugh

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We receive news almost daily about environmental tragedies such as flooding, forest fires, drought, air pollution, extremes of temperature, population migration and the effects of global warming. All these in turn affect population health and wellbeing. The WHO states that the environment is responsible for almost a quarter of all disease and mortality.

Healthcare produces significant volumes of waste, consumes large amounts of energy, polluting the atmosphere and producing greenhouse gases. The paradox is that in trying to improve healthcare we are at the simultaneously causing harm.

Operating rooms contribute substantially to this burden.

I will present an overview of the issues and propose ways that both anesthetists and operating theatre staff should be attempting to mitigate this, not just in the operating room but also in daily living.

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# Towards Retirement: Managing those last years of practice


## Dr Rob Fry

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**Introduction**

Ageing can be defined as a complex, variable, multifactorial process associated with numerous gradual declines in physiological function (that will inevitably lead to death), Chronological aging starts at birth and ends with death. In New Zealand almost 20% of anaesthetists are older than 60 (in comparison 15% of the USA workforce >65).

**Physiological Changes**

Physical functional capacity begins to decline from age 30 with a reduction of lean muscle mass and reduced reflex time however regular exercise can keep this almost unchanged between the ages of 45-65. Vision becomes impaired with conditions such as cataracts, glaucoma, age-related macular degeneration and accommodation, contrast sensitivity and visual acuity all decline. Hearing becomes progressively less sensitive, with losses being most significant at higher frequencies – important for alarm recognition and all chronic conditions or illness (e.g. musculoskeletal problems and ischaemic heart disease) increase with age. 89% of older anaesthetists who continue working however usually assess their health as Good to Excellent perhaps through self-selection. Cognitive decline, as measured by the MicroCog™ assessment tool, is often precipitous. Decline occurs with increasing age in the areas of cognitive function, inductive reasoning, verbal memory, and overall reasoning. Physicians generally score highest in cognitive functioning from ages 25-55 with little change between 55-69 and 70+. After age 60 domains such as processing speed (dealing with incoming information quickly and efficiently), working memory (short-term memory maintenance of new information) and episodic memory encoding (formation of new memories of specific events or episodes) decline. In one recent study however, the majority of practicing senior surgeons performed at or near the level of their younger peers on all cognitive tasks, as did almost half of the retired senior surgeons, one third of practicing surgeons in their 70s still matched younger surgeons in competence on a variety of learned tasks. Domains such as semantic memory (knowledge) and more "routine" behaviours" show little change; one possibility is that older adults use preserved knowledge and experience to form more efficient or effective strategies when performing tasks in which younger adults rely on processing ability i.e.: less experienced practitioners may rely on slower conscious deliberation or type two thinking. As physicians age therefore they are more likely to make errors from placing undue weight on first impressions but their ability to reach a diagnosis when minimal information is available improves with experience. Subjective cognitive loss equates with 4.5 x risk of progression to Mild Cognitive Impairment (MCI) within 7 years.

Dementia has a general incidence of up to 13% depending on the definition and a "cross over" with mild cognitive impairment of 10-20%. This appears to be the group that makes up a large proportion of the doctors referred to medical bodies and is responsible for up to 63% of all the causes of medical adverse events. Most, sadly, are determined to be preventable. Sleep quality reduces with age and sleep time becomes shorter. Tiredness can affect older doctors' performance and mood and cognitive performance of older shift workers may be more impaired during night work but they may be less aware of their degree of impairment than younger shift workers.
Simple "on-call" can be highly disruptive of sleep even when not called out but physicians with young children can also suffer chronic sleep deprivation with resultant impairment too.

**Assessment**

Unfortunately recognition of incompetence is very difficult and complex and the literature rather empty on this topic. There is extensive material on confirmatory testing once identified but there is a deafening silence regarding solutions. Most physicians have a limited capacity to self-assess their competence or ability and this is biased by multiple factors such as financial need, self-esteem etc. External assessment is essential but difficult especially for those in isolated private practice. Impairment risk increases from public practice through group private practice to solo private practice and retirement from public to private at career end "only a few years left to maximise my

income". Most older anaesthetists in good health continue to perform well but they have a responsibility to demonstrate insight into the potential impacts of ageing and be open to collegial advice regarding competence, to take the initiative, make plans for the future and discuss these issues within their department/private practice. There is very limited evidence that on-going education, 360 degree peer reviews, practice reviews, chart reviews are helpful. There is evidence for simulation, medical assessments and neuro-cognitive assessments but these are also very stressful for the practitioner. The older Anaesthetist needs to be cognisant of the 'human factors' involved, it is easier to support those who demonstrate humility listen to and take advice from colleagues, seek guidance at work if required and assess the work they do regularly.

**Towards Retirement**

General "job planning" changes might include daytime weekend work instead of overnight on-call, flexible working conditions with shorter working hours, less isolated working and less demanding or less stressful lists. Role changes might be appropriate for some such as pre-operative assessment clinic work, undergraduate or postgraduate education, clinical governance or other non-clinical roles. It's never too early to start planning, busy medical careers don't help but that should not be allowed to prevent planning, about 67% of anaesthetists "slow down" over a period of time to allow for transition. All new consultants should have a session with an appropriate "planner" for advice on practice insurance, life insurance, accounting, superannuation planning, GP, work life balance as the part 3 course recommends and possibly covered by their CPD allowances. Repeated every 10 years or so this can focus career options too. At around age 55 another session with a "life coach" and serious "life planning" is recommended. Ongoing sessions including a financial planning update, family situation, social structure, retirement occupation and hobbies is useful.

**Retirement**

Successful aging is multidimensional and encompasses physical health or freedom from disease, functional health or independence, psychological health or mental health and social health or active engagement although luck and genetics play their part. The decision to retire is influenced by personal health, financial status and pension arrangements, perceived status, family commitments job satisfaction and employer attitudes and norms plus working hours and the availability of work. With present day longevity this is a new "career" "The Third age" and may be up to 30% of one's life. In the US only 55% of older anaesthetists rate their financial status as good or excellent often as a result of poor planning.

**Financial Planning**

There are numerous on-line sites for this sort of information but some suggested goals include aiming to have ones annual salary saved by age 35, three times ones salary saved by age 45, five times saved by age 55 and eight times by age 65. Obviously mortgages, commencement of employment and student loans may prevent this exact progression. According to the "four percent rule" savings of $4 million are required by retirement to live on an annual pre-tax income of $160,000 every year throughout retirement or 25 x the desired gross annual "income". There are also many other ways of calculating what you can or should spend. Maximise drawdown to 75 or 80 then live on the government pension only, "Leave no inheritance" High spend at the start (ie "run out" - at "expected" calculated or programmed exit date), "Leave an inheritance" – fixed annual spend which remains unchanged throughout retirement (ie expect to "reduce expectations") or a "mix" – the general norm.

The seven big expenses from 55-65 versus 65-75+ are: Housing (34% of spending) – generally falls (20%), Transport (17-18%) – generally falls (10%), Food (12 - 24%) – slight fall (inflation) (7%), Pension Saving (13 -6%) – drops significantly (60%), Health Care (insurance & costs) (8 – 13%) – rises (20%), Entertainment (5 – 6%) rises slightly (10%) and Other (personal habits, care products, travel) (10-11%) - rises (15%). Overall expenses tend to reduce by up to 85%!

**Happiness in Retirement**

Have a purpose that gives you a reason to get up in the mornings and stay engaged. Downshift your lifestyle to be a more relaxed person with a simpler low stress life. Build up an 'inner circle' of friends who also have healthy habits and challenge you mentally and regularly meet and share good food. Be likable, 'Of the centenarians interviewed, there wasn't a grump in the bunch.' Put family first - signal that this is so with family rituals and importantly try new things.

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## SILVER

**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.

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## SILVER

Pfizer has operated in New Zealand since 1961, in this time Pfizer has contributed significantly to improving the health of Kiwis through supplying a wide-ranging health portfolio.

Our belief is the patient comes first. We continually invest in New Zealand and constantly seek to improve our quality and supply of medicines.

We are committed to New Zealand and have invested heavily in innovation and research to support the development of new medicines and given back to the community through new partnerships and philanthropic ventures.

To ensure our portfolio reflects patient need we work collaboratively with our customers such as Pharmac, healthcare professionals, patients and seek to add value to the wider healthcare sector by introducing products with safety enhancements or more efficient delivery that save time.

**Pip Acock**
Product Specialist - SI and Tenders
Pfizer Essential Health

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ferinject®
ferric carboxymaltose

NOW FUNDED
IN THE COMMUNITY[1]
(SPECIAL AUTHORITY CRITERIA APPLY)

# Iron Deficiency?

*Help them rediscover li*Fe

To find out more, see us at stand 3

**Reference 1.** PHARMAC funding criteria 2017.

FERINJECT® (ferric carboxymaltose) solution for intravenous (IV) use. **Presentation:** 2mL and 10mL vials containing 100mg and 500mg of iron, respectively. Ferinject® is a Prescription Medicine for the treatment of iron deficiency when oral iron preparations are ineffective or cannot be used. **Dosage:** The dosage must be calculated individually for each patient and must not be exceeded. IV injection: Maximum single dose of undiluted solution of 1,000mg (20mL) over 15 minutes per week. For doses 200-500mg, should be administered at a rate of 100mg iron/min. IV drip infusion: Maximum single dose of 1,000mg (20mL) in up to 250mL saline given over 15 minutes once per week. **Contraindications:** hypersensitivity to any of the ingredients; anaemia not attributed to iron deficiency; evidence of iron overload or disturbances in utilisation of iron. **Precautions:** parenteral iron preparations can cause hypersensitivity reactions, observe for hypersensitivity reactions for at least 30 minutes following injection; paravenous leakage can lead to skin discolouration and irritation; liver dysfunction; infections; pregnancy category B3. **Adverse effects:** Common: headache, dizziness, hypertension, flushing, nausea, injection/infusion site reactions, hypophosphataemia. Uncommon: hypersensitivity. Rare: anaphylactoid reactions. Please review full data sheet before prescribing, available at www.medsafe.govt.nz or call 0800 996 312 for further information. FERINJECT® is listed on the HML and is a fully funded prescription medicine – special authority criteria apply. Based on data sheet 6 June 2016. FERINJECT® is a registered trademark of Vifor Pharma Group used under license by Aspen Pharmacare. Aspen Pharmacare, C/O Pharmacy Retailing (NZ) Ltd, Auckland. www.aspenpharma.co.nz. TAPS PP2672-18JULY INSIGHT 8874

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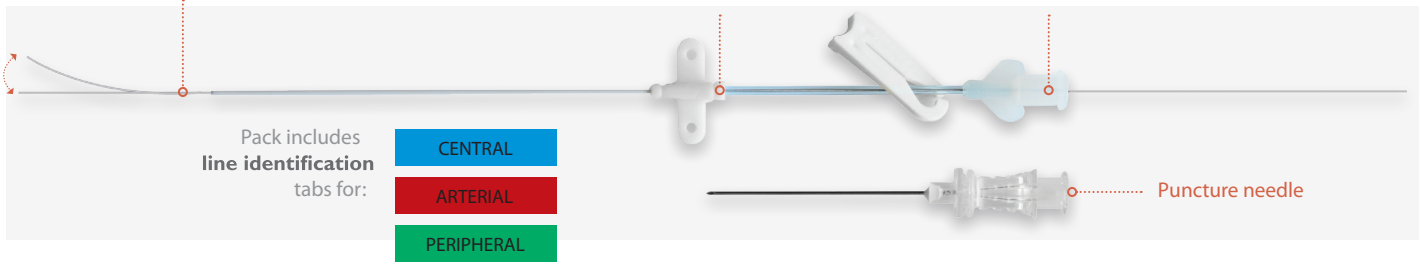


Pack includes **line identification** tabs for:

- CENTRAL
- ARTERIAL
- PERIPHERAL

**For further information:**
**Device Technologies Ltd**
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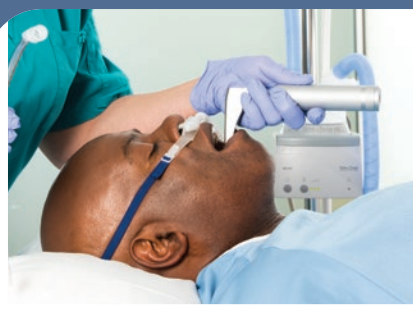
F&P Optiflow THRIVE™

# **Revolutionize** your practice of anesthesia today



**Prevents desaturation**[1,2,3] and extends the apneic window[1]

**Improves patient safety**[2,3]

**Optiflow THRIVE™ is a new airway management tool** that enables hands free preoxygenation and apneic ventilation using humidified nasal high flow.

**To evaluate Optiflow THRIVE™ or receive further information,** please contact Fisher & Paykel Healthcare at 021 274 3880


1. Patel A & Nouraei S. *Anaesthesia*. 2015
2. Miguel-Montanes R. et al. *Crit Care Med*. 2015
3. Badiger S. et al. *BJA*. 2015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# EXPERIENCE THE BRIDION EFFECT.

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

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

**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.** ® BRIDION is a registered trademark.  ANES-1208494-0001. First issued June 2017. DA1735MW essence MSD8467

MSD

bridion® sugammadex

# Precedex®

(dexmedetomidine hydrochloride injection)

Concentrated injection for (must be diluted prior to use) intravenous infusion

# In the ICU, only your patient matters.

## Patients on Precedex experience...

### Calm, cooperative, rousable sedation.[1,2]

### Sedation before, during and after extubation with significantly less opioids.[1-6]

### Respiratory and haemodynamic stability.[1,2,5,6]

1. Herr DL, *et al. Journal of Cardiothoracic and Vascular Anesthesia* 2003;17:576-584. 2. Hoy SM, *et al. Drugs*. 2011;71:1481-1501. 3. Arpino PA, *et al. J Clin Pharm Ther* 2008;33:25-30. 4. PRECEDEX Data Sheet 2017. 5. Martin E, *et al. J Intensive Care Med* 2003;18(1):29-41. 6. Venn RM, *et al. Crit Care* 2000;4:302-308.

Please review full Data Sheet before prescribing.

**INDICATIONS:** For the sedation of initially intubated patients in ICU (up to 24 hours). For the sedation of non-intubated patients prior to and/or during surgical and other procedures. **DOSAGE:** Individualize and titrate the dose. For IV infusion only. **ICU SEDATION:** Adult loading dose: 1.0 mcg/kg over 10 to 20 minutes. **MAINTENANCE INFUSION:** 0.2 to 1.0 mcg/kg/hr (recommend 0.4 mcg/kg/hr initially). Do not exceed 24 hours. Consider loading and maintenance dose reduction in the elderly and in patients with hepatic impairment. **PROCEDURAL SEDATION:** Adult loading dose: 1.0 mcg/kg over 10 to 20 minutes (including awake fibreoptic intubation). The loading dose may be omitted or reduced in the elderly and in patients with hepatic impairment; a loading dose reduction may also be suitable for less invasive procedures. **CONTRAINDICATIONS:** Dexmedetomidine hypersensitivity. **PRECAUTIONS:** Use only with suitable monitoring under supervision from an appropriately trained medical practitioner (ECG, blood pressure and oxygen saturation are recommended). Administration may reduce lacrimation. Bradycardia, sinus arrest (high vagal tone, rapid IV/bolus injection). Bradycardia and hypotension associated with infusion. Caution in patients with advanced heart block and/or severe ventricular dysfunction. Hypotension and/or bradycardia more pronounced in hypovolaemic patients and with diabetes mellitus, chronic hypertension, severe cardiac disease, elderly, pregnancy (Category B1), lactation. Caution in co-administration with other vasodilators or negative chronotropes. Consider midazolam or propofol dose reduction when used in combination with Precedex®. Transient hypertension during loading. Interactions: anaesthetics, sedatives, hypnotics, and opioids. **ADVERSE EFFECTS:** Hypotension, hypertension, bradycardia, dry mouth, nausea, somnolence. Each 2 mL vial of Precedex® concentrate contains 200 microgram of dexmedetomidine. Precedex® is an unfunded, prescription medicine in New Zealand – a prescription charge will apply. The full data sheet is available from www.medsafe.govt.nz or Pfizer New Zealand Limited (www.Pfizer.co.nz) or call 0800 763 363. Pfizer New Zealand Limited. Level 1, Building B, 8 Nugent St, Grafton, Auckland 1023. V4.5

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Preparation date September 2017
TAPS Approval NA 9242
GCMA Number PP-PDX-NZL-0018

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