Gazette Issue 12/15

The AUSTRALIAN AND NEW ZEALAND COLLEGE of PERFUSIONISTS GAZETTE

DECEMBER 2015

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EDITOR

Molly Oldeen, *CCP*
Perfusion Department
Lady Cilento Children's Hospital
Level 7F, Clinical Directorate
501 Stanley Street
South Brisbane QLD 4101
Tel: 07 3068 3692

ASSOCIATE EDITORS

Carla Zazulak, *CCP*
Lady Cilento Children's Hospital
Brisbane QLD
Carla.zazulak@health.qld.gov.au

Jane Ottens, *CCP*
Ashford Hospital
Ashford SA
Email: jane.ottens@acha.org.au

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A MESSAGE FROM THE EDITOR

by Molly Oldeen, *CCP.*

This is probably the most exciting and successful issue to date for me since I volunteered as Editor. I really feel I have gotten a positive response from units to contribute articles for the purposes of facilitating communication across Australia and New Zealand in the field of perfusion.

Thank you to the College for another fantastic ASM in Manly. I genuinely enjoyed the presentations and the opportunity to catch up with perfusionists from around the world.

In addition to the clinical and research presentations, I appreciated the content surrounding teamwork and the operating room environment. Cardiac surgery involves some of the highest levels of complexity and requires the coordinated action of many. Previous searches of my own have found that there is a huge body of literature supporting the importance of communication, transparency, utilization of checklists, briefings and debriefings, and more, on the reduction of mistakes and incidents in the cardiac theatre. The relationship between teamwork and safety is critical. If you were unable to attend this meeting, I encourage you to read the abstracts that will be included in the first edition of the Gazette in the new year.

While this edition has some great scientific articles, it also bears the sad news of the loss of some of our fellow colleagues. They will be greatly missed and their contributions to our profession will not be forgotten.

Although some time has passed since our last IMOB meeting, we have included the abstracts, as well as a conference review, and some relevant photos. As a reminder, it has been decided that IMOB meeting with be held biennially.

Once again, I sincerely thank those who have provided content! Congratulations to the two Gazette Award winners (cash prizes), Casey Edwards and Helen Scarrott. I look forward to picking two more next year. I can only hope that this remains a trend and people in each unit continue to think of ways to share techniques and research with the rest of us.

Happy reading!

Kind regards,

Molly Oldeen
The Gazette Editor

ANZCP STRUCTURE

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Tim Willcox - PIRS Editor
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A MESSAGE FROM THE PRESIDENT

by Jon Van den Berg, *CCP.*

Before I begin I would like to thank the immediate past president Jane Ottens who has served in the role for the past four years. Much of the work done by the executive goes unseen to the membership. Jane has worked hard in tidying outstanding issues within the College and actively represented our profession in the MBS review. Jane will be acting as an advisory to the executive for the next twelve months.

This years Executive

President: Jon Van den Berg
Secretary: Matthew Sheminant
Treasurer: Carla Zazulak
Vice President: Mark Ambrose
Members Representative: Charles McDonald
Registrar: Tim Willcox
Board Representative: Mark Mennen

On behalf of the executive we extend a warm welcome to Charles McDonald and Mark Ambrose.

Introduction

In my view our organisation requires a solid mix of administrators, academics and politicians. (possibly in that order)

In self-assessment I fall to the latter group as a politician. I like to make things happen and see them through. My key strategy usually starts with analysing what the big steps are to achieve the goal. If they can be achieved the detail follows. Human resource often plays a major role, seeking advice, expertise and support. Working toward New Zealand registration involved contact with many individuals and organisations. Where possible this was done face to face. You are obliged to give me your full attention if I make the effort to come and see you, especially if I come by plane.

Moving Forward

Priority: Upgrade of the ANZCP website.

The ANZCP website is the window to our membership. The geographic spread of Australasian Perfusion units, from Dunedin to Perth dictates this. With respect to the webmasters we now require a professional upgrade. Site information needs to be current and attract regular viewing from the membership. Stakeholders to the site, the Executive, Board, Meeting Planners etc, need to be responsible for updating information. This project will be a key focus early in the new year.

Survey for Succession Planning

A steady 4-6 Perfusion positions are advertised regularly on the College website. We assume an ageing Perfusion population. However what is the true age demographic of the Perfusion workforce? how many trainees might be required to fill upcoming positions?

How many units meet the criteria as training centres? A survey of Australasian cardiac centres would provide valuable information to support training and ensure Clinical Perfusion can fill available and upcoming positions.

Increasing Membership

How many Clinical Perfusionists work in Australasia? What proportion belong to ANZCP? Membership could be enhanced through value-added features, increased access to reference sites or perhaps discounted liability insurance. Increasing ANZCP membership will strengthen the influence of Clinical Perfusion.

With best wishes for a low stress Christmas and a positive New Year.

Jon Van den Berg

President ANZCP
jon.vandenberg@waikatodhb.health.nz

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AUSTRALASIAN BOARD OF CARDIOVASCULAR PERFUSION

by Mark Mennen, *Chairman ABCP*

The ABCP's focus this year, as always, has been the continual improvement and maintenance of the Perfusion Diploma course, the Autotransfusion course and Certification.

Following a period of reduced trainee participation, the Diploma course is currently being undertaken by a number of local and overseas students. Now that the course management is established, we are now taking enrolment the third semester and this option was taken up by three candidates this year. Several local enrolments are likely next year plus two from Singapore. Our Diploma Course coordinator, Clarke Thuys, has recently completed a site visit to Hong Kong and was informed there we will likely receive one or two enrolments from Queen Mary Hospital and possibly a new Hospital as well which also meets the training criteria.

The Diploma course subject order is currently under review. It has been proposed the cell salvage and introduction to perfusion modules be brought forward. The suggestion is that trainees will be able to provide cell salvage independently at the end of term 2. Earlier perfusion based subjects should also make them able to get a better understanding of what they are doing sooner.

The Autotransfusion Course has settled on an enrolment number of around 10 per term after the initial high numbers. Hopefully a dedicated website will be up and running soon on the back of the ANZCP website to allow more streamline enrolment and payment. The course has proven to be a great success under the guidance of Chris Morley and Andrew Lahanas. Chris' term on the board ends this year and he will not be nominating for a another term so I would like to thank him for his years of great service to the board, particularly his instrumental role with the Autotransfusion course, IMOB and representation at the National Blood Authority. Thanks Chris.

Recertification continues to have a high return rate though a number of perfusionists have let certification to lapse over the years. We encourage lapsed individuals who are interested in recertifying to contact the board as we are always keen to encourage and facilitate recertification rather that obstruct.

Certification Exams will be held on the 24th and 25th of February, 2016. Interested candidates should contact Clarke Thuys early to allow ample time for a study package to be sent if required.

Thank you to all the Board members for their efforts this year. Vincent Rajkumar has been busy and ably taken on the secretary role and Sarah Varghese has provided excellent input as a nonelected member. Thanks.

PIRS REPORT

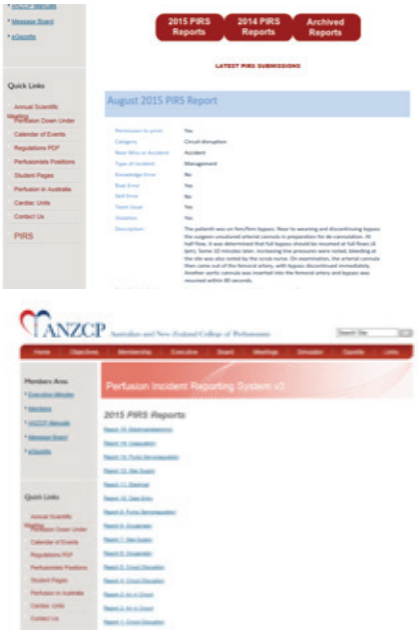
by Tim Willcox, *FANZCP - PIRS Ed*

The ANZCP PIRS has now instigated changes that arose from discussion at the last ANZCP ASM where a number of initiatives were proposed with the intention of providing more immediate feedback on incident reporting through the system.

We have established an email group within the PIRS web system we have called PIRSList. Known members of the ANZCP and key members from overseas perfusion societies were invited to join PIRSList in order to receive email updates of PIRS alerts and recently posted reports. Members are sent an email with a link to the relavant page where the new reports can be viewed. Rather than email individual reports as they were submitted (as first mooted) we have elected to email a link to the prevoius month's reports that have permission to publish. Scrolling down will reveal all reports for the month, and the 2015 PIRS Reports tab takes the reader to individial links to all reports for 2015 listed by category of incident. A pdf deindentified summary of the incident is sent to the reporter if requested on the PIRS submission form together an excel spreadsheet showing all the data that has been entered into the PIRS database for that incident. This includes the related human factors data that may not appear on the summary. This may facilitate local site incident data collection systems.

The next initiative is to create additional pages with resource material on perfusion saftey.

We have not promoted this heavily as there were delays to initiating these changes due to constraints with the website that have now been overcome. If you wish be added to the PIRSlist email the PIRS editor at PIRS@anzcp.org



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AROUND THE PUMP ROOM

Melanie Couyant, *The Royal Melbourne Hospital*

The Royal Melbourne Hospital is Victoria's oldest hospital, founded in 1848. It is now co-located with Melbourne Private, The Women's, Francis Perry hospitals, The Walter and Eliza Hall Institute of Medical Research (WEHI) and the spectacular Victorian Comprehensive Cancer Centre which is still under construction and due to be completed next year. Approximately 200,000 patients are treated at RMH per year, it has the busiest emergency department and the second largest trauma centre in the state.

Many of the original buildings have been replaced but there are still some old sections of the hospital dating back to the 1940's. There's a labyrinth of old service tunnels under the hospital and one 700 metres long that joins the Old Childrens Hospital. It was used to transport steam from the RMH boilers.

The theatre complex was built in the 1990s and has been undergoing recent renovation including theatres, change rooms, storage areas, tearooms and surrounding corridors. It is anticipated that the new Hybrid Operating Room will be opened by the end of the year. Simultaneously over the past couple of years we have been working amid construction of the VCCC and midair walkways to RMH.

In addition to this structural change, our team has weathered three changes in governance in as many years. Consequently the last few years have been a challenge to the team, however team work is what it's all about in our profession!

We are an eclectic group in age, experience and cultural background but we all seem to share a sense of humour and a strong work ethic.

The RMH Perfusion team comprises Mike Whitburn (who on occasion can be found playing his recorder in a quiet unlikely place in the hospital). Mike replaced long serving Peter Angelopoulos as Head Perfusionist who left in 2014 to take up the position at the Austin Hospital. Other full time members of the team include Greg Horne / part time home builder, the recently married and hard working Lisa Morgan (Hewitt), new member Smita Gavande. Also part timers, Alison Horton / regional netball coordinator , Patrick O'Neil / sports analyst, Melanie Couyant (I'm still there!) and Belinda Megee who is on Maternity Leave. Our trainee is Saukina (Sue) Hammoud who is well into her training now.

Our work comprises Adult Cardiac surgery; CAGs, Valve replacement and repairs, Minimally Invasive Valve Repairs, Reconstruction and Replacement of the Thoracic Aorta including the Arch, Adult Congenital Surgery and TAVI procedures and whatever else comes our way.

AROUND
THE PUMP ROOM

RCH Perfusion Team, *Royal Children's Hospital - Melbourne*

It's been awhile between reports from our group, so here is a little snap shot of the scene at The Royal Children's Hospital in Melbourne.

In November it will be 4 years since we moved into the new hospital, with the old RCH now a distant memory. In those 4 years, our team remains the same. Director Steve Horton, Clarke Thuys, Alison Horton, Martin Bennett, Simon Augustin, Brad Schultz and Steve Bottrell.

Clinically our case load is fairly constant. In 2014 we completed 507 bypass cases and are on track for a similar number this year. Despite a similar number of cases, as with many hospitals, the complexity of cases seems to be changing as demonstrated by the average bypass time changing from 113 minutes to 129 minutes between 2011 (452 bypasses) and 2014 (507 bypasses) with mean X-clamp times increasing from 61 to 68 minutes. Looking at the total number of hours when there is a perfusionist in house this time has increased from 3471 hours in 2008 to 3801 hours in 2014. So we are doing more cases and longer cases.

Outside of the cardiac theatre, much of our time is devoted to the ever growing field of ECLS. Last year we put 52 patients onto support, and somehow most of these seemed to occur in the middle of the night. The number of cases also requiring ECMO standby seems to be growing exponentially with the surroundings of the Catheter lab now becoming a bit too familiar. The long and drawn out tender process for our new ECMO pumps is almost complete. Our 15 year old RotaFlow system has served us well, but one by one these pumps are starting to fail. Hopefully we will be using new equipment by the end of the year.

Our long term VAD program continues to grow. Since 2005 when the program began, we have supported 36 patients with an overall survival of 72%.The Berlin Heart is still the device of choice in our younger patients but this year we were able to introduce the HeartWare for children greater than 30kg. This device has proven to be easy to manage and following the initial stabilisation period after implantation, has caused us little concern. To date we have successfully transplanted all 4 patients who were bridged on a HeartWare.

In terms of clinical practice, the most recent change is the introduction of Nitric Oxide into the oxygenator sweep gas at 20ppm. Following a randomised control trial which looked at the effects of Nitric on low cardiac output syndrome post bypass (in press), we have decided to now incorporate this into our standard practice. After consultation with Ikaria, we now have a INO Max DS-IR permanently positioned on the heart lung machine, easily available for every case.

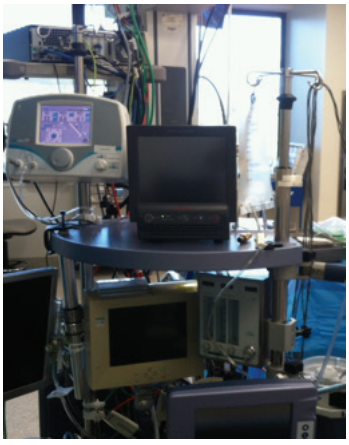

Photo - The Nitric delivery system now sits on top of the heart lung machine.

On the research front we are patiently awaiting the arrival of our prototype multichannel, wireless NIRS monitor. Following a donation from a foundation set up by Olympic Gold medallist Alisa Camplin in honour of her son Finnan who was a patient at the RCH, we set out in collaboration with Melbourne University to design the device. After many roadblocks the prototype should be ready for clinical trials before the end of the year.

Next time you're in Melbourne feel free to give us a call and drop in to say hello. If you can't make it to the hospital, make sure you visit Iconic Melbourne Pub, Young and Jacksons. Ask for a Naked Ale which is proudly brewed by Master Brewer Brad Schultz from Black Heart Brewery, Unofficial Brewery of the ANZCP.

THANK YOU TO JANE OTTENS

On behalf of our entire College, Associate Professor Rob Baker presented Jane Ottens with a small token our appreciation for all of her hard work over the last four years as President of the ANZCP.

Jane has done and continues to do an amazing job as a leader in the field of perfusion and remains an inspiration to us all to be involved in the advancement of our profession.

JAMES PICKEN RETIREMENT

After 34 years of perfusion, one of our fellow colleagues, James Picken, has retired. As you may be aware, his career commenced at the Royal Adelaide Hospital in 1973. Since then his work has taken him to Houston, Fiji, Vanuatu, China (through Operation Open Heart Outreach) and to Melbourne, establishing the first private hospital in Victoria to commence Cardiac Surgery. Also, the Austin Hospital was established in 1984 with his assistance. The years have been challenging, exciting and rewarding for James.

A Cell Saving service was commenced under his practice in Victoria in the early eighties, which serviced many private and public centers, metropolitan and regional, and then later expanded into Queensland. Again with great results. He is currently overseas having a well earned break. Bruno Marino has taken over his practice.

Thank you, James, for all of your hard work and commitment to the field of perfusion over the years!

TOWNSVILLE MOURNS TWO COLLEAGUES

Ray Greenwood

It is with much sadness that we inform you that Raymond (Ray) Greenwood passed away on Saturday the 5th of September. Ray had been in hospital receiving treatment for Acute Myeloid Leukaemia at the time of his death. As many of you would know Ray was the Senior Perfusionist at The Mater Misericordiae Hospital in Townsville. He began working at The Mater about 15 years ago. During this time we had the pleasure of getting to know Ray both professionally and personally. Ray was extremely dedicated and passionate about his work. On a personal level he was quietly spoken and private but always a true gentleman. It was easy to have a conversation with Ray and he always found the positive to any situation. We were fortunate to have several visits with him the week leading to his passing and during this time he continued to be up beat and optimistic. Ray is survived by his partner Sachiko, two daughters and four grandchildren. We will miss you terribly Ray.

Dr Mo Diqer

It is with much sadness that we inform you that Dr Al-Mutazz(Mo) Diqer passed away on July 20th 2015 after a short battle with lung cancer. Mo was a Cardiothoracic Surgeon who did his original training in Jordan before moving to St George Hospital in Sydney. In 1994 he moved to The Townsville hospital and then to The Mater Hospital. He recently decided it was time to move back to Jordan to be with his mother and sisters. In February he married Rowena, his long time partner, packed everything up and moved to Jordan. He was doing humanitarian work with refugees when he became unwell. He returned to Australia and died surrounded by his wife, children and closest friends. Mo was a colourful character. His funeral was full of funny Mo stories. He was dedicated to his patients and a committed family man. He loved red wine and fast cars and motorbikes. To work with Mo was an experience never to be forgotten.

Helen, Susan and Monica at The Townsville Hospital

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JIM GRANT
ANNOUNCMENT

GRANT, James (Jim) Edmund.

To those that met him – 'A GIANT of a man.' To those that knew him- 'A giant of a MAN.'3

Nearly four years ago Jim noticed one finger was not functioning as well as normal. It was the start of the devastating progression of Motor Neuron Disease, culminating in his recent death. Not once was he ever heard to complain about the situation. In typical Jim fashion, he accepted his lot, got on with living and enjoying being within the family activities and remarkably, kept upbeat! His family were amazing in their constant care and interaction. He was never left out of anything. In many ways this was a tribute to his nature and nurture as a husband and dad.

Jim died peacefully at home on July 26 2015, age 60, surrounded by loving family, wife Sue, children Fiona, Hamish, Robert, Annie, Katie, and Lucy, their partners Paddy, Ana, Gin, Alex, and Josh respectively, grandchildren Jayden, Phoenix, Olivia, James and Eva. A service to celebrate Jim's life was held at St Francis de Sales Church, Island Bay, on Thursday 30th July.

A perfusionist for more than thirty years, Jim served Wellington Public and Wakefield Private hospitals. He was known by everyone as a gentle giant, with a constant friendly demeanour and ready smile. He was also known as the 'number eight wire man', a reference to farming ingenuity. Jim took pride in the ability to make anything work, somehow. More than his perfusion prowess was his ability to get along with people and get the best out of them. His deep chuckle was infectious as was his enthusiasm. He loved his work and it showed in the everyday things he did. Nothing was ever too much trouble. He never declined a request for help. He was a perceptive man and knew when people were in need of sensible, caring advice and when to give it. Your secret stayed exactly that. Everyone liked Jim and will without doubt miss his 'large' friendly presence.

Jim, was overwhelmingly a family man. Apart from enjoying an evening playing poker with buddies, his life was dedicated to his wife Sue and the six kids and their kids! It was also evident they too loved him and held him in the highest regard.

We will miss the 'Big Man'. Rest in Peace friend.

Len Cooper CCP(Aust) 31/07/15

32ND ANNUAL SCIENTIFIC MEETING
AWARD WINNERS

Best Scientific Paper – Terumo Award

Charles McDonald – Hyrodynamic evalation of aortic cardiopulmonary bypass cannulae using particule image velocimetry

Encouragement Award – Medtronic

Rona Steel – Preserve Blood – Every Drop Counts

Sid Yarrow Award - Cellplex (LivaNova) – First time presenter/student

Emmerson Sgammotta - Cerebral protection management during DHCA: a review of current methods

ANZCP Meritorious Awards

Martin Gill – Plasma leakage through a polymethylpentene oxygenator

Mark Ambrose – Air Transmission Comparison of Two Adult Arterial Filters AF-100 vs. CB351: An In Vitro Study

ANZCP Best Trade Display

Medtronic

ANZCP Gazette Award

Helen Scarrott – "An in house audit of the use of cardiotomy suction during protamine administration at TTH" – Edition 12/14

Casey Edwards – Class of Perfusion 2015/2016 – Edition 05/15

Open HEART INTERNATIONAL

by John Dittmer
John Hunter Hospital

PNG to start an independent Cardiac Surgical Program in 2019

It is a real honour to be able to join a cardiac team on a trip with Open Heart International. We have the privilege of seeing little kids, so sick before their operation, transform into children playing and running around just a few days later; their little faces beaming as they blow bubbles into the air from a bubble pipe for their post op physiotherapy. Mothers, who were previously unable to look after their young families, can now live near normal lives thanks to replaced or repaired rheumatic valves. Big smiles light up the faces of those who have been ill their whole lives.

There is no way any of our OHI patients could afford an international trip for their life saving surgery. I remember that on one trip to Vanuatu the team was puzzled when a young child failed to show up for his surgery. It was a sobering moment when we learned the reason for his nonattendance; his family could not afford the bus fare for mother and child of about $3.50 each! How privileged we are to live in a land of abundance.

In June 2015, I was able to join a smaller than usual OHI cardiac team travelling to PNG. The purpose of this trip was to support the country's progress towards an independent cardiac surgery program. While we were there, the government announced that a schedule had been drawn up to start the program in four years time and that the finance had been approved. A meeting was called to discuss the proposal and to begin planning in earnest.

Flying into Papua New Guinea has always been quite an experience. Back in the early 1990's I looked out my aeroplane window to see the PNG coastline for the first time. I enjoyed seeing the ocean waves breaking onto the colourful coral reefs. Then came the coconut palms and tropical undergrowth with the occasional wisp of smoke rising from cooking fires in unseen villages below. Finally Port Moresby airport came into view. As the plane landed I noticed several large objects on the grass on the side of the runway only to suddenly realise that these objects were aeroplanes that had crashed and had been pushed to the side so that the next plane could land without having to dodge unfortunate obstacles….. Welcome to Port Moresby.

When the OHI team arrived at Port Moresby's International Airport in June this year it was noticeably different with the recent upgrades made for the 2015 Pacific Games. Around the city were multiple larger than life monuments of athletes performing their chosen sports, as well as many upgrades to the infrastructure.

At Port Moresby General Hospital the team spread out to their area of expertise. Surgeons and Cardiologists met to discuss and select the patients while other team members began to set up ready for surgery starting the next day. Among the 27 volunteers were a couple of OHI veterans, Dr Matt Crawford an anaesthetist who has volunteered with the PNG heart team on 22 occasions and Kim Stuart, a dedicated scrub nurse who has visited PNG 21 times and has travelled with OHI on almost 50 trips.

The Port Moresby Perfusion staff, Norman and Alex, have been working with both the OHI and Singapore volunteer heart teams for the past few years. They have been training in Adult Perfusion in Singapore and are looking for a place to train in Paediatric Perfusion.

This trip was a paediatric open-heart surgical visit and consequently Norman and Alex were keen to have hands on experience for as many cases as possible. We set up and tested the Medtronic Century (formerly Cobe) heart lung machine that is only a year or two old. It was then time to start the real business of Perfusion for our little patients.

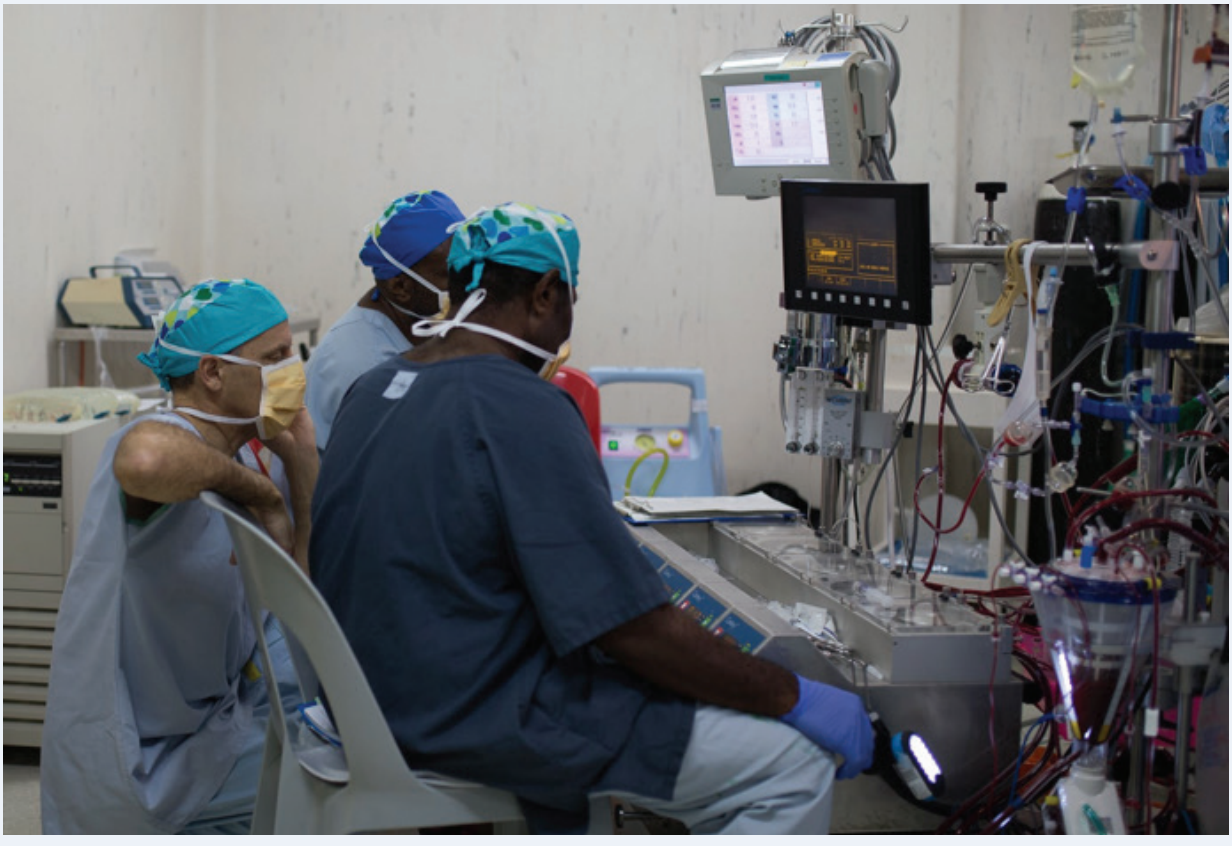
Using a heart lung machine that they had barely seen before was a challenge in itself for Norman and Alex. With so many different oxygenators and different sized tubing packs it was quite an education for them. However, they were keen, worked very hard and did amazingly well.

There was one learning incident that was picked up after going on bypass. It was noted that the arterial line pressure was unusually high and the SVO2 was well into the 90's. The solution? Remember to recalibrate the Arterial pump when changing pump boots from ¼" to 3/8"! Fortunately no harm was done as the oxygenator was rated to the higher flows.

During our trip, 15 bypasses were performed on children weighing 5Kg or more, and there was one closed case. All our patients did well and had positive outcomes.

An independent cardiac surgical program will be invaluable for the people of Papua New Guinea. I feel very fortunate to be part of a program that will provide such a service, and to witness the progress so far.

Setting up in a hurry as the patient is already in the room!
Photo by Adam Murakami



A training session with Norman and Alex
Photo by Adam Murakami

Open HEART INTERNATIONAL TONGA

by Martin Bennett
Royal Children's Hospital , Melbourne

In early September I had the pleasure of being part of Open Heart International's Tongan Mission trip. My first trip with OHI for more than ten years, I had some understanding of the organisation and what the trip may entail, but despite this, nothing can really prepare you for the week ahead.

The majority of the team gathered together for the first time on Saturday night at Fua'amotu Airport in Tonga. Many seemed to know each other from previous trips and there was a real air of excitement about what was to come in the next few days. On Sunday morning the full team assembled for some welcoming speeches and introductions but following this the work began.

I was joined by Bruce Treagus (St Vincents – Melbourne) a four time veteran of OHI's Tongan mission. A perfusionist for 41 years, Bruce was unfazed by anything and a great source of information and insight into what was about to confront us.

Our first task was to wade our way through about 100 boxes, to separate the perfusion items from the others and assemble a heart lung machine. A job that would normally be allocated a whole afternoon quickly became a stress test as the word circulated that the team would like to operate on the first case as soon as we were ready, or earlier if possible! The pump, a 20 year old Terumo System 1, required a fair bit work to get it into a configuration that would suit the theatre, the circuits and us. Following a couple of hiccups with verifying the concentration of the oxygen supply, the first patient was wheeled into the theatre at about 4 PM.

Somewhere amongst all this was a heart lung machine crying out to be assembled.

Monday came and in what turned out to be the biggest day of the week, we again had to hit the ground running. Working with circuitry and a cardioplegia delivery system you haven't seen before can certainly test a friendship. The circuit presumably had been designed to work on many different pump bases, unfortunately the System 1 appeared not to be one of them. A PDA ligation and four CPB cases later (including a very sick double valve) the theatre team was able to sit down for dinner by about 9 PM. After Monday I did have a feeling of "what have I gotten myself into", but as the week progressed so did the comfort level.

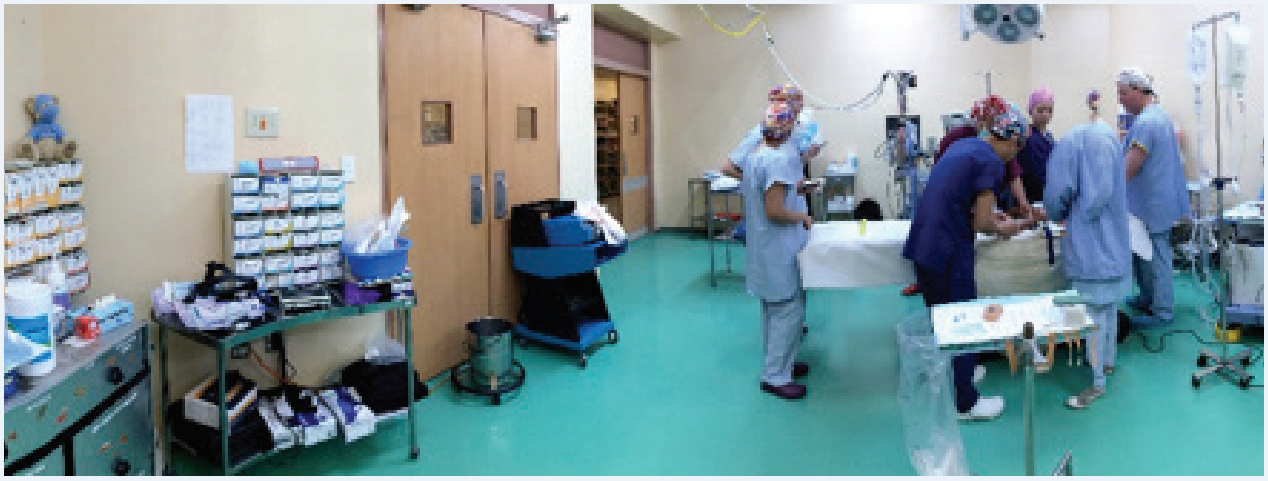
The pace of the week progressively slowed as circuity and systems became more familiar and with that we found a little more time to catch our breath between cases.

Although advertised as a paediatric week, the demographic of the patients was very different to what I was expecting. We operated on two small patients (11 and 4.5 Kg) but also on three patients 110, 123 and 125 kg! Overall the paediatric team was able to complete 14 CPB cases and 3 PDA ligations, to be finished by 1PM on Friday afternoon.

A country of only 100,000 resisdents, the main city of Tonga, Nuku'alofa, is not particularly beautiful but it is the local people, the other volunteers and the patients that make these trips so special. The gratitude of the local hospital staff and organisers is undeniable. Food plays a big part in Tongan culture and repeatedly their way of thanking us was with local produce for 6 meals a day including suckling pig for lunch one day. The other members of team are also inspirational. Some have done in excess of 40 trips while for others it was their first. Everyone though had the common

goal of giving something back to the community by using their skills to improve the health of the Tongan people. And finally the patients. A tour of the ward on the final day put it all into perspective. The smile on the face of the 2 year old we had operated on only 3 days earlier, as he bounced on the bed with his Dad close by, was able to say it all.

Open Heart International is a very well organised group and I would certainly encourage other members of the ANZ perfusion community to get involved with a mission trip. Like me, if it's been a while between trips, you will quickly be reminded that you get so much more out of these trips than what you give.



Conference Corner

MEETING OF THE FEDERATION OF ASIAN PERFUSION SOCIETIES (FAPS) 2015

by Helen Scarrot, Andrew Lahanas, Russell Miller, Clarke Thuys & Kaye Collins

The FAPS 2015 meeting was held in Kobe Japan. Kobe is a port city which has been rebuilt after the Great Hanshin earthquake of 1995. With a magnitude of 6.9, it was one of the worst ever recorded in Japan with over 6000 people losing their lives. Completely recovered, it is now one of Japan's most wonderful cities.

Terumo Australia arranged a tour of their Japan facilities for their overseas visitors attending the meeting. We were fortunate to visit the Terumo Medical Pranex, a training facility visited by 12,000 medical professionals annually, housing realistic simulation facilities including a catheter laboratory, Intensive care unit, operating theatre and a ward and a complete home care with patients in various stages of "recovery". Accompanying the perfusionists were Tina, Margaret and Kazu from Terumo, who were fantastic hosts during the event.

We next travelled to the TERUMO Ashitaka factory which is positioned near the famous Mt Fuji. We learnt about Dr Kitasato who founded Terumo in1922 making the first Terumo product, a thermometer, which gave rise to the name Terumo. Dr Kitasato continued to making advances in medicine, including the development of the Tetanus vaccine.

After a tranquil Japanese tea sitting beside the shrine we travelled to the Terumo manufacturing facility, where we were fortunate enough to witness the creation of their oxygenators from the construction of their individual hollow fibres to the complete assembly of all components to complete the final product which resembled a RX25 Oxygenator. In the afternoon we participated in the facility's earthquake and fire drill, along with 1500 Terumo workers dressed in white overalls. Our involvement in the drill was quite entertaining and only added to the experience since it is always a good idea to have a plan B when your factory is where 4 tectonic plates converge.

Of course, perfusionists from the Terumo tour met up with other Aussies and Kiwis attending the meeting at the bar of the Kobe Portopia Hotel. The Hotel was the accommodation centre for the huge conference facility and included about 300 delegates. It was an impressive venue, ideal for large events such as this. This conference is only run every second year

and the FAPS committee ensured they made the most of the event, cramming as many presentations into the two days available. Common themes included perfusion standards and guidelines, registration and education, paediatrics and ECMO, cardiopulmonary bypass techniques and blood management. There were numerous presentations in well researched areas from Asian centres, with a common theme of reducing donor blood administration.

A full house gathered for the breakout session on day two where there was plenty of interest in the topic of oxygenator engineering and design by Christoph Benk Ph D of Germany, who presented "the thought process in designing future oxygenators", with one delegate questioning the speaker about the customised requirements of the Asian population, who may require different sized oxygenators to that of the Caucasian. Clarke Thuys presented "is Plasma Lactate a Predictor of Outcome for Paediatric VA ECMO." Clarke's talk was well received. Perhaps we will have the pleasure of listening to him present at the next ANZCP conference.

Delegates from various countries presented their training and education systems, with Clarke stepping up and presented the ANZCP history and how our education of Perfusionists has evolved to include a recertification process. It appears the ANZCP is well ahead with our system of perfusion education, especially with the recertification process, Autotransfusion Course and the on-loan Orpheus simulator which is available to ANZCP members.

We all enjoyed the meeting and the hospitality of the Japanese people and went away from FAPS2015 feeling that we really are working towards the same objective as our Asian colleagues, especially with regards to circuit minimisation and avoidance of blood transfusions. We highly recommend FAPS for those who are considering attending an overseas conference and the next FAPS meeting will be held in Shanghai China in 2017.

INTRAOPERATIVE MANAGEMENT OF BLOOD (IMOB 2015)

by Darryl McMillan and Chris Morley

The Intraoperative Management of Blood meeting for 2015 was held at a new venue the Amora Hotel on the 27th and 28th March, Sydney. The conference attracted specialists from across the spectrum of surgical blood management disciplines. The attendees were a great mix of Anaesthetists, Autotransfusionists, Anaesthetic Theatre Nurses, Blood Bank personnel, Perfusionists and Transfusion nurses. A total of 57 registrants attended, down on the 73 from 2014 which was a little disappointing.

The conference once again focused on the growing reported problems associated with blood transfusion. There is little doubt that blood transfusions have saved many lives in cases of acute hypovolaemia and anaemia since its conception yet the studies continue to report transfusion related harm that may be associated with an increase in patient morbidity and mortality. Few would disagree that the evidence is growing and that there is a need to limit or to avoid transfusion by way of pre-operative preparation, assessment combined with conservation techniques in optimizing a patient for surgery. The meeting focussed on addressing these issues.

The program was as follows:

Friday

Session 1

- Team Management of Transfusion: Patient Blood Management - Dr. Bruce Spiess.
- Are we using blood management in CT patients? Pilot audit results for NSW sites- Ms. Sally Francis , Blood Watch - Clinical Excellence Commission.
- Quality control during peri-operative blood recovery, blood cell processing and autotransfusion. - Mr. Emerson Sgammotta - Perfusion Services.

Session 2

- Immunomodulation and Cell Salvage - Dr. Michelle Roets.
- Post-Operative Collection/Salvage of Red Blood Cells- Where are we today - Dr. Bruce Spiess.
- The 'world' we work in: who's who in blood and available resources. - Dr Amanda Thomson, Blood Watch - Clinical Excellence Commission.

Session 3

- Filtration - Darryl McMillan
- Audience survey –

Saturday

Session 1

- Bleeding in the presence of sepsis, liver failure - Dr. Bruce Spiess.
- Pre-existing medications that alter platelet - Dr. Bruce Cartwright.

Session 2

- The no brainer principles of Patient Blood Management" - Professor James Isbister.
- Practical Aspects of optimising Perioperative Patient Blood Management. - A/Professor Kerridge.

Session 3

- The Red Cell Transfusion was meant to Improve Oxygen Transport and Delivery: TRALI and TACO" Professor - James Isbister.
- Massive transfusion and Haemorrhage in surgery and trauma - Dr. Bruce Spiess.

Session 4

- Coagulation Changes during and after Cardiopulmonary Bypass (CPB): Views from the United States Dr. Bruce Spiess.
- Coagulopathy management in Spinal Surgery - Dr. Chris Sparks.

The feedback from those who attended was extremely positive with special note given to the high quality of the speakers and the relevance of the information in their presentations. Both Chris and I would like to thank each speaker very much for taking time from their busy work and family schedules to present on a Friday and Saturday and for sharing their expertise. In particular we would like to thank Bruce Spiess our international speaker.

I would also like to acknowledge and thank our corporate sector for their generous support and sponsorship of the meeting in particular, Haemonetics Australia for their support of Bruce Spiess return to IMOB.

CellPlex, Fresenius Kabi, Haemonetics, HaemoView Diagnostics, Medtronic and Terumo all had very impressive trade exhibitions relating to Autotransfusion devices and point of care coagulation monitors. The trade exhibits were a highlight for many of the delegates. Chris and I would like to take this opportunity to thank the College President, Jane Ottens, and the executive for their support of the IMOB meeting. We would like to give a very big thank you to the college treasurer Carla Zazulak who made our lives easy with dealing with the accounts. The meeting was once again underwritten by the ANZCP and run as a special interest meeting, with all funding coming from and returning to the college.

After a great deal of discussion it has been decided to organise future IMOB meetings biennially (every two years). Our reason for this is so that we can remain at the forefront of research and its application in surgical blood management. So please mark your calendars for IMOB's return in March/April 2017.

Dr. Bruce Spiess

Dr Bruce Cartwright

Dr. Chris Sparks

Kerry McMillan

Professor James Isbister

Associate Professor Ian Kerridge

Original Article

HOW 'ZEN AND THE ART OF MOTORCYCLE MAINTENANCE' RELATES TO PERFUSION: METAPHYSICS OF QUALITY.

by Clarke Thuys

While reading the October 2014 edition of Motorcycle Trader I came across a statement by Rob Blackburne in a book review entitled 'In defence of Zen and the Art'. He stated "While it is pretty sad to say that a particular book changed your life, I'm always happy to put my hand up to say that Robert Pirsig's Zen and the Art of Motorcycle Maintenance (ZMM) did it for me."

This sparked my interest enough to go and read the book. Now if you have read the book in question and have an interest in motorcycles you probably intuitively understood what Pirsig was getting at, but without that motorcycle background it is far less obvious. So for I thought I would attempt to put it into terms relevant to clinical perfusion.

Metaphysics is defined by the Collins English Dictionary as: the branch of philosophy that deals with first principles, especially of being and knowing. The Metaphysics of Quality (MOQ) is a theory of reality introduced in ZMM.

"Quality… you know what it is, yet you don't know what it is. But that's self-contradictory. But some things are better than others, that is they have more quality, but when you try to say what quality is, apart from the things that have it, it all goes poof!.... Obviously some things are better than others… but what's the "betterness"? What the hell is Quality? What is it?" (ZMM p184)

In ZMM Pirsig drew a distinction between what he termed the 'classic' (objective) and 'romantic' (subjective) aspects of Quality.

For example, some perfusion equipment can be classified as: 'a classic' by it satisfying certain specified (objective) quality criteria, such as the Jostra Quadrox D, epitomising a new class of hollow fibre (polymethyl pentene) oxygenators, or the Medtronic Biomedicus centrifugal pump being a 'classic' example of a centrifugal pump; or equipment can be subjectively 'romantic' such as the passion that drives someone to handcraft some piece of perfusion equipment or to modify a standard piece of equipment to improve it in their opinion, or perhaps better still is the 'snob' value of using a particular brand of equipment. This is more commonly seen in some markets where locally produced and marketed products are not perceived to be as good as internationally available goods.

After writing ZMM Pirsig developed a more complex set of ideas (drawn from Eastern religion and Western philosophy) about the concept of 'Quality' (for which Pirsig uses an upper case Q), which he called the 'Metaphysics of Quality'.

In his Metaphysics of Quality; or 'Value' as he also names it, Quality cannot be fully defined because it empirically precedes any intellectual constructions. It is the 'knife' or 'cutting-edge' of experience, known to us all, for example, knowing exactly how far you can push the flow of an oxygenator without risking reduced oxygen uptake, before you actually do it!

For Pirsig, Quality is the fundamental force in the universe stimulating everything, from atoms to combine to make molecules, to what causes animals to evolve and incorporate ever greater levels of Quality. According to the Metaphysics of Quality, everything (including mind, ideas and matter) is product and a result of Quality!

This means that Quality lies in the dynamic 'now' moment (Dynamic Quality, DQ) that we sense anything during the instantaneous present; with a short delay we then give this impression a 'static' form (Static Quality, SQ) by describing it as an emotion, a thing, a word, etc. These 'static forms', if they have enough good or bad quality associations, are given names and ideas about them are exchanged with other people, building the base of knowledge for a culture.

The ANZCP is one such Quality culture, with its focus on the provision of exchange of knowledge, regulation and education with respect to clinical perfusion and all it encompasses.

We are lucky – as clinical perfusionists we can experience the operation of extracorporeal life support systems (moments of 'dynamic Quality'), but understand the need to study ECLS systems in order to learn how to set up a CPB circuit, calculate flows based on patient data, or to calibrate the pump flow or occlusion for different circuits – the 'static forms' of Quality – tedious, but essential if you want the machine to operate as intended. We can then share this information with other perfusionists via the Annual Scientific Meeting or the Gazette, or even by visiting other units.

Quality therefore according to Pirsig is fundamentally a continuing dialogue between our personal (internal/subjective) values and beliefs, and certain publically accepted (external/objective) standards (ANZCP Regulations and Guidelines).

Note for example, Pirsig on motorcycle maintenance manuals:

"These were spectator manuals. It was built into the format of them. Implicit in every line is the idea that: "Here is the machine, isolated in time and space from everything else

in the universe. It has no relationship to you, you have no relationship to it. Other than to turn certain switches, maintain voltage levels, check error conditions…" And so on. That's it. Mechanics in their attitude towards the machine were really taking no different attitude from the manual's towards the machine… And it occurred to me that there no manual that deals with the real business of motorcycle maintenance, the most important aspect of all. Caring about what you are doing is considered either unimportant or taken for granted." (Pirsig, ZMM, p35, underlining is my emphasis)

Here Pirsig is criticising the application of the purely mechanistic approaches to Quality, because they lack human value. Such mechanistic approaches stand in stark contrast to the Zen notion of 'mastery' or to that of an artisan where the mastery of a technique is virtually indistinguishable from the person commanding it. Hence the 'Art" of Motorcycle Maintenance and the category of 'Zen Master'. At some time we have probably all said that there is an 'art' of Perfusion as well as the science of Perfusion.

Pirsig argues forcefully that if you apply the Metaphysics of Quality to even a routine situation or in solving an everyday problem (such as stripping the old circuit off the pump and setting up a new one) it is possible to get the same 'Zen high' from doing quite mundane tasks "The real as well as the 'dynamic quality moment' allows (that is by applying the boring old 'mechanistic' fitness-for-purpose principal, and doing exactly what the situation requires, not in doing or saying 'whatever you like' or by trying to impress others). This certainly sounds like Perfusion. In the Metaphysics of Quality, being excellent in what we do, and being 'fit-for-purpose' mean essentially the same thing.

According to Pirsig then, the internal (subjective) and external (objective) dimensions of anything, rather than being at opposite pole, fuse together into his more holistic concept of 'Quality'. Take the RCH pumps for example; they are Jostra HL30s, classics in that they were the first to allow pole mounting of all pump modules and look good; but they are reliable and most certainly 'fit-for-purpose'. They are Quality pumps (as are most makes of ECLS pumps).

In ZMM Pirsig further illustrates the holistic subjective/internal and objective/external dimensions of the Metaphysics of Quality through the use of the motorcycle as a metaphor. In the following quotation I have changed the word motorcycle for the words ECLS pump:

"The ECLS pump is a system. A real system…but the true system, the real system, is our present construction of systematic thought itself, rationality itself… That's all the ECLS pump is, system of concepts worked out in steel and plastic. There is no part in it, no shape in it, that is not out of somebody's mind… the ECLS pump is primarily a mental phenomenon." (Pirsig, ZMM, p102) And with the motorcycle metaphor:

"The real cycle you're working on is a cycle called yourself. The machine that appears to be "out there" and the person that appears to be "in there" are not two separate things. They grow towards Quality or fall away from Quality together. If things are not done properly both suffer. (Pirsig, ZMM, p325)

It could be argued that Pirsig over-extends the motorcycle metaphor as it being some sort of mechanical 'vehicle for thought', but the term 'Quality' has taken on a whole new meaning.

Now to finish MOQ with another quote from Pirsig:

What the "Metaphysics of Quality" does is take this separate category, "Quality", and show how it contains within itself both subjects and objects. The Metaphysics of Quality would show how things become enormously – fabulously more coherent—when you start with an assumption that Quality is the primary empirical reality of the world…."

(Pirsig, Lila: An Inquiry into Morals, 1991)

These ideas tell me that in order to really experience and enjoy the dynamic aspects of ECLS, it is also necessary to understand and properly maintain the ECLS System (patient included) – to achieve an appropriate balance or blend! When the two dimensions of what Pirsig calls 'Quality' , the internal' subjective and the external/objective, the 'dynamic' and the 'static' forms of Quality, become so fused because of their coherent interaction that they can no longer be easily distinguished from each other, then that brings Perfusion all together.

In a forum about Dynamic Quality on the Metaphysics of Quality website (MOQ.org.au) a discussion about how to get DQ into your life came up with a number of contributions. The best contribution comes from, I think, Warren Buffet, who is said to be one of the greatest investors of our time. One could call him a philosopher of investing. He and his partner, Charlie Munger have some good insights in to Life, as well. Buffet talked about his "intuition", the source of that "intuition", and those feelings that led him to his great successes. He said, to paraphrase, "that intuition without great and hard-won experience is guessing and, more than not, runs you upon the rocks. Intuition becomes the leap of insight that is of value (DQ?) only after great study and experience." Why? I think it touches upon the idea that genius, in one form, is the ability to rearrange what is known and with a leap of insight, create a new and better form, something not imagined before. One's mind has nothing to work with unless it has been filled with the wisdom and insights that have come before. Additionally, Charlie Munger added that, in this day of specialization, the more fields one accomplishes oneself in, perhaps the greater possibility of a synergy resulting in a new and better transmutation...a transformative insight moving us from static to DQ.

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This paper was adapted from an article in the Japanese Vintage Motorcycle Club Magazine Vol 134, 2014.

May we continue to strive to be masters of the art and science of perfusion.

Clarke Thuys

Original Article

THE EVOLUTION OF PERFUSION

by Cynthia Riddell, *CCP*

It was during a leisurely visit to the Museum of Technology and Transport (MOTAT) in Auckland, that I came across a very interesting piece of equipment, the Melrose heart-lung machine. As a trainee perfusionist, I was of course intrigued by it and also quite excited about my discovery. I proceeded to thoroughly examine the newly found apparatus, and this led me to reflect on the evolution of the bypass machine and cardiac surgery in general.

Professor Denis Melrose played a major part in the development of heart surgery and bypass techniques. He was working as a lecturer at the Medical School of Hammersmith Hospital in London, when he designed his heart-lung machine (1). Furthermore, the Melrose bypass machine was then to be used later on, in 1958, during the first successful open heart surgery operation in New Zealand, performed at Greenlane Hospital in Auckland by Sir Barratt-Boyes (2). Denis Melrose also pioneered the use of potassium-induced cardiac arrest and cold cardioplegia used for myocardial protection (3).

One of the striking feature of the machine is the seemingly oversized cylindrical rotating disc film oxygenator, which sits on an angle on top of the machine. The pumping circuit uses two single roller external compression pumps, one supplying the oxygenator (or artificial lung) and the other returning oxygenated blood to the patient. Another notable feature of the apparatus is the bulky cube-shaped arterial filter, which has a dual purpose as it acts as a bubble trap, as well as a coarse filter (4). Flowmeters and bourdon pressure gauges adorn the front control panel of the device.

The bypass machine used in Auckland Hospital (and therefore the one I am using as part of my perfusion training) is the Stockert s5, Sorin's 5th generation of heart-lung machines.

The oxygenator used is the Inspire hollow fiber membrane oxygenator, which also incorporates a hardshell venous/cardiotomy reservoir. Multiple double-headed modular roller pumps accommodate for complex surgeries and for the use of tubing of different diameter size. The display modules are numerical and controlled by touch-screen accessible advanced software. The control dials are smooth to operate and allow for accurate regulation of parameters. Numerous safety features and warning devices enable safer perfusion practice (5).

Cardiopulmonary bypass techniques and equipment have come a long way. Although the new generation of perfusionists are nowadays training with incredibly sophisticated equipment, this should not by no means be taken for granted. It took decades of hard work, dedication, and enduring brain power to develop and implement the advanced practices in the field of cardiovascular surgery that we work with today. This has enabled the provision of safer practices, as well as allowing a vast array of surgical procedures that were once thought to be impossible to be performed. Therefore, we ought to be grateful to these pioneers whose contributions impacted greatly on the lives of many and facilitated our job.

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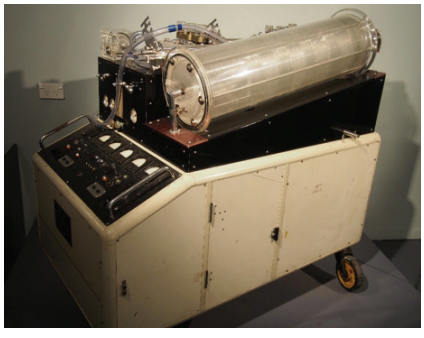

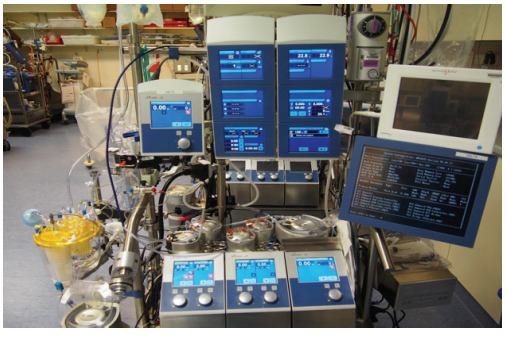

Photos: Top: Melrose heart-lung machine; Bottom: Stockert S5.

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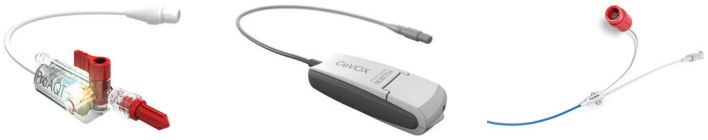

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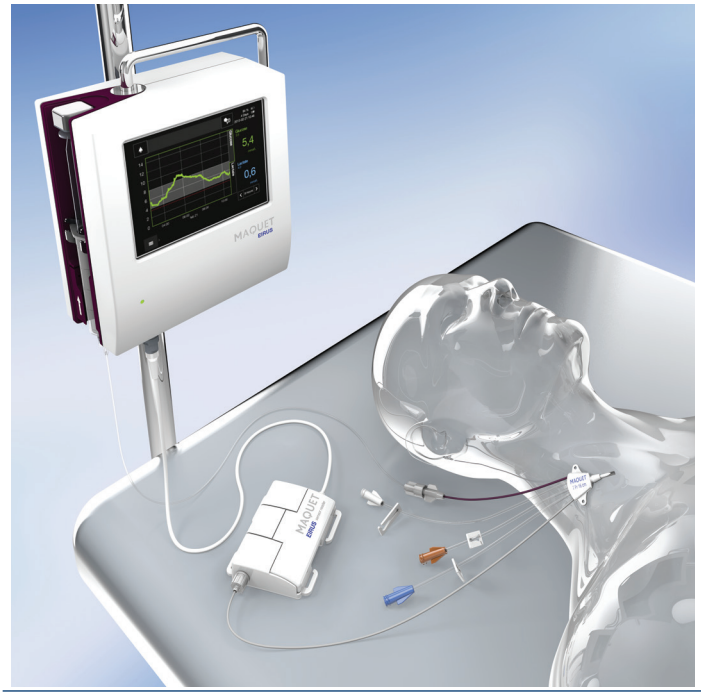

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Original Article

THE WAP
WESTMEAD AUTOLOGOUS PRIMING

R.Miraziz, *Westmead Hospital*

The Mini-RAP, VAP, Forward-VAP, CAP and Re-CAP are the usual Perfusion lingo used just before commencing bypass at Westmead Hospital.

These are autologous priming techniques (except the Re-CAP) that are implemented for the reduction of the priming volume prior to or while commencing bypass.

On average 800mls from a total of 1500 ml prime can be removed safely and quickly from our circuit without compromising patient's safety and delaying the institution of bypass and in some instances the surgeons.

All steps are easily aborted in the event of unforseen hemodynamic instability with a turn of a tap or a positioning of a clamp. Each step can be performed individually or multiple technique approach can be employed to achieve a maximum reduction in priming volume.

The Acronyms

Mini-RAP: Mini Small amount, **RAP** Retrograde Autologus Priming, the displacement of prime from the arterial line using patients own blood. In our set-up the **Min-RAP** is the displacement of prime up to the arterial filter.

VAP: Venous Autologus Priming. The displacement of prime from the venous line upto the reservoir using patients own blood.

Forward-VAP: The displacement of prime from the venous reservoir and the oxygenator prior to going onto bypass.

This technique takes less than 30 seconds and replaces the traditional RAP that is usually is performed over 10 minutes that usually requires inotropic support to maintain aortic root pressure.

CAP: (Cardioplegia Autologous Priming) the displacement of prime from the Cardioplegia circuit using the oxygenated blood from the reservoir

Re-CAP: the Cardioplegia chaser, the blood in the cardioplegia circuit is returned to the reservoir using a crystalloid chaser, keeping the cardioplegia circuit primed.

The Circuit

VAP: The perfusion circuit is bifurcated on the venous return line prior to the reservoir.

This bifurcation should have at least one, but preferably, two blood bags attached to it. A 1L and 2L bags configuration is preferred.

The benefits of 1L bag is:

- To remove excess priming fluid from the reservoir.
- To remove the initial priming fluid from venous drainage upon commencing bypass.
- To assist in venous retrograde priming post venous drainage or in the event of the accidental venous line disconnection and hence the drainage of the venous line.

The benefit of the 2L bag is for autologous blood collection upon commencing bypass, this is especially useful if the patient has high haemoglobin count or if DHCA is employed and the preservation of red cells or blood products is required.

Mini RAP and Forward VAP: the arterial line is bifurcated post arterial filter (ie main arterial and a recirculation line)

The recirculation line has a 2L blood collection bag attached to it and it is primarily used for draining the residual volume from the circuit post bypass.

CAP: the crystalloid priming line is connected onto the 3-way tap of the cardioplegia recirculation line.

Upon priming the cardioplegia circuit, the crystalloid volume is displaced into the empty priming bag rather than returned to the reservoir.

Re-CAP: the blood cardioplegia is chased back to the reservoir using a crystalloid once the Cardioplegia is no longer required (usually prior to coming off bypass)

The Sequence of events utilising all techniques

Mini RAP: Upon arterial cannulation the perfusionist checks the inline pressure while the anaesthetist administer small amount of metaraminol (if required) prior to the Mini-RAP step. The pump is turned off and the recirculation line is clamped post recirculation bag which is kept open to collect the displaced crystalloid.

The arterial line is then unclamped gently to allow for patient's blood to displace the crystalloid volume in the arterial line up to the arterial filter. (Approx. 200ml is removed and collected in the recirculation bag)

VAP: Upon venous cannulation and with the surgeon request to commence bypass, the VAP 1L bag is opened and the crystalloid volume is drained into this bag while being displaced by blood (Approx 300ml is removed).

Forward-VAP: with a partial clamp remaining on the venous return line, the venous line is unclamp and the main arterial pump is turned on allowing for the crystalloid volume from the reservoir, as it is being filled by venous return blood, to be directed to the recirculation bag until the blood has reached the arterial filter. Only if patient's volume permits i.e. if there is enough volume returning from the patient (400ml to 1L of crystalloid is removed)

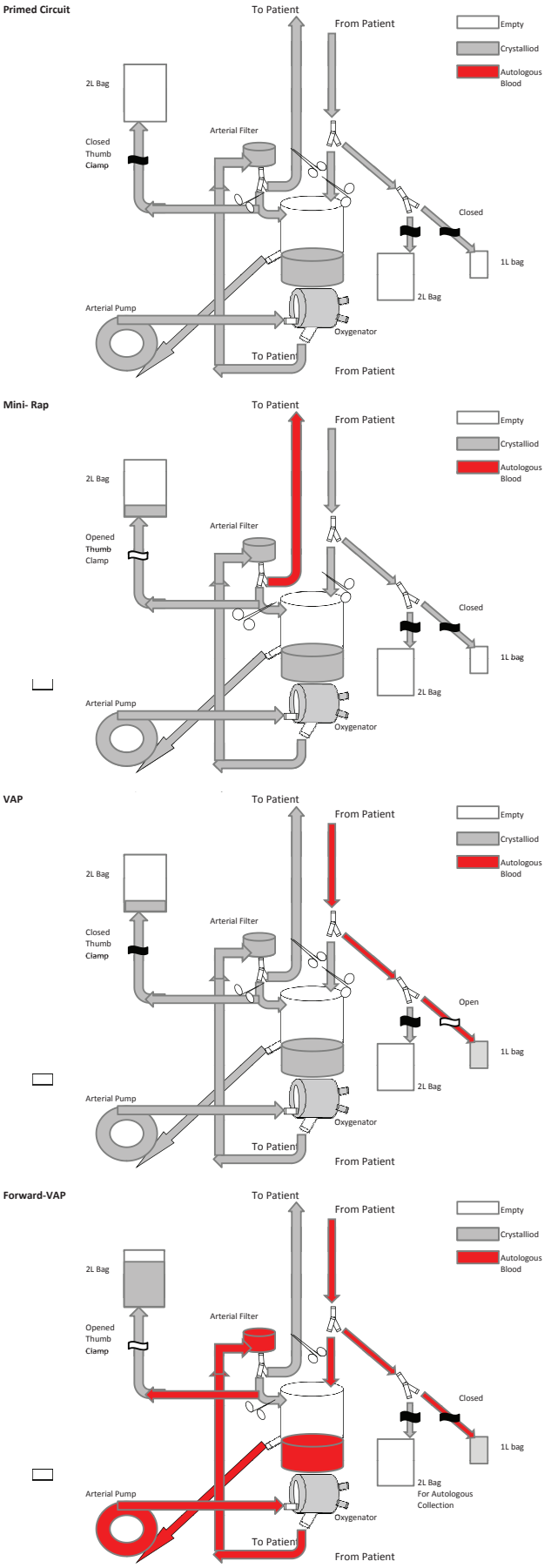
CAP: upon cardioplegia re-circulation, the cardioplegia prime volume is diverted to the prime bag. (Approx. 200ml collected).

Acknowledgment

We would like to thank Terumo Corporation for introducing Mr Phillip Scott, a Perfusionist from The Mayo Clinic, Rochester, Minnesota, whom I met and chatted during his short visit to Australia in 2014. We exchanged ideas and techniques as most Perfusionists do when they get together from different centres.

Phil introduced us to the Forward-VAP technique as an alternative to the traditional RAP, and without modifying our circuit the first Forward-VAP was achieved successfully on that day. Since then this technique along with the mentioned techniques, contributed and modified by the rest of the team, have resulted in a significant reduction in prime volume and allowed for overall haematocrit preservation with a significant reduction in transfusion rate.

The Mini-RAP, VAP, Forward-VAP, CAP and Re-CAP have revolutionised the priming technique at Westmead Hospital and contributed enormously to our blood conservation program which has seen a massive reduction in transfusion over a short time frame. These techniques have spread across multiple centres in Sydney.



Original Article

AIR TRANSMISSION COMPARISON OF TWO ADULT ARTERIAL FILTERS - AF-100 VS. CB351: AN IN-VITRO STUDY

Kieron C. Potger, *CCP, BSc, Dip Perf*; *MClinN*; Darryl McMillan, *CCP, Dip Perf;* Mark Ambrose *CCP, BNurs, Dip Perf*

Perfusion and Autotransfusion Unit, Dept. of Anaesthesia & Pain Management, Royal North Shore Hospital, Sydney, Australia
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Abstract

Arterial filters are commonly incorporated within the extracorporeal circuit to minimise cerebral injury by capturing particulate matter and microbubbles. We clinically use the Affinity CB351 arterial filter. The new Affinity AF-100 (BB851) arterial filter has less prime volume (100 mL vs. 212 mL) and a smaller screen pore size (30-µm vs. 38-µm). Our aim was to determine if the AF-100 was as safe as the CB351 in terms of relative microbubble transmission of introduced air. A recirculating in-vitro circuit primed with warmed blood was used to compare both arterial filters. Microbubbles were detected using a GAMPT BC100 Doppler in the arterial filter inflow and outflow line. While at 3 L/min and 5 L/min pump flow rates, measurements were taken one minute prior and for three minutes after bolusing 30 mL of air proximal to the venous reservoir. Both arterial filters transmitted microbubbles during air injection. Both of the arterial filters' proportion of bubble volume transmitted increased with higher flows. Both arterial filters transmitted a similar number of bubbles. However, the AF-100 transmits smaller sized bubbles and consequently less total bubble volume. The results of this in-vitro study show that the transmission of introduced air by the AF-100 arterial filter is not more than the CB351 arterial filter over a range of flow rates suggesting that it is as safe in terms of air handling.

Introduction

Arterial filters are commonly incorporated within the extracorporeal circuit (ECC) to minimise cerebral injury by capturing particulate matter and microbubbles with Class 1, Level A evidence justifying their use (1). Clinically we use the Affinity CB351 arterial filter (Medtronic Inc., Minneapolis, MN, USA) installed distal to the oxygenator outlet in our ECC.

The new Affinity AF-100 arterial filter (BB851; Medtronic Inc., Minneapolis, MN, USA) has a smaller prime volume (100 mL vs. 212 mL) and smaller screen pore size (30-µm vs. 38-µm). As part of our departmental consideration of a replacement arterial filter, we wanted to determine if the AF-100 was as safe as the CB351 in terms of microbubble transmission of introduced air. This was investigated by comparing the CB351 arterial filter with the AF-100 in terms of relative microbubble transmission during air introduction, in-vitro, over a range of flow rates. Specifically, we wanted to determine if these two filters transmit microbubbles when air is bolused into their respective inlets and, if they do, which of the oxygenator systems transmits more air?

Materials and Methods

Test circuit

A recirculating test circuit was constructed using heparin coated tubing (Carmeda®, Medtronic Inc., Minneapolis, MN, USA); all components were new (Figure 1). The patient was simulated by a hardshell venous reservoir (Affinity Fusion CVR, Medtronic Inc., Minneapolis, MN, USA). This 'patient' reservoir was filled to the 1000 mL level to facilitate the deairing of the recirculating prime. To provide a constant siphonage drainage pressure, the 'patient' reservoir fluid level was kept at 100 cm above the outlet of the circuit venous reservoir; this was accomplished by adjusting a variable clamp positioned on the 'venous line' connecting the 'patient' reservoir to the circuit venous reservoir. Emulating our institutions clinical ECC, the circuit venous reservoir was a collapsible bag (CBMVR 1600, Medtronic Inc., Minneapolis, MN, USA). This venous reservoir bag was tested in a fully compressed (minimal volume) position by closing the reservoir holder cage onto its back plate.

The prime that drained into the circuit venous reservoir was pumped out into the test arterial filter system by an appropriately calibrated roller pump that was set to be just occlusive (Sarns 8000, Terumo Corporation, Tokyo, Japan). The next component the prime entered was a membrane oxygenator (Affinity NT CB511, Medtronic Inc., Minneapolis, MN, USA). This oxygenator helped remove microbubbles that had been introduced into the circuit; a process that was facilitated by connecting the oxygenator's gas inlet port to suction (-200 mmHg) while sealing all its other gas openings. Circuit pressure was controlled by adjusting a variable clamp on the inlet to the deairing oxygenator. From the oxygenator, the prime was returned back to the 'patient' reservoir.

Test arterial filters

Two filter designs were assessed for their air handling performance: the 30-µm Affinity AF-100 [BB851], and the 38-µm Affinity CB351 (both Medtronic Inc., Minneapolis, MN, USA). See Table 1. Both filters included a filter bypass loop. The Affinity CB351 was heparin coated (Carmeda®, Medtronic Inc., Minneapolis, MN, USA); the Affinity AF-100 had a hydrophilic polymer coating (Balance®, Medtronic Inc., Minneapolis, MN, USA). Three copies of each arterial filter model were used. All six arterial filters were connected in parallel; appropriate clamping directed the blood flow only via the arterial filter being tested. The purge line on the arterial filter was kept closed during testing. Clinically, our institution only intermittently purges the arterial filter.

Prime

After denitrogenation by CO2 flushing for at least five minutes, the circuit was primed with normal saline (Baxter Healthcare, Australia). Then seven units (1800 mL) of expired donated FFP were added followed by seven units (1965 mL) of expired donated packed RBCs. The prime was haemofiltered (Sorin SH14 hemoconcentrator, Sorin Group Italia, Mirandola, Italy). Haemofiltering was performed to yield approximately 4700 mL of prime with a haematocrit of about 21%. A heater unit (Medtronic BioCal 370 Heart Pump, Minneapolis, MN, USA) attached to the in-situ oxygenator kept this prime at approximately 32°C.

Air challenge

The experiment was designed to challenge the arterial filters with microbubbles (≤500 µm diameter). The air injection site was in the venous line entering the venous reservoir. The air injection site was a Luer lock connector with injection port pierced by a 38 mm, 25-gauge hypodermic needle. Using a syringe pump as a holder, a 60 mL syringe with 60 mL of air was manually compressed down to 30 mL against a closed three-way tap directly attached to the syringe outlet (i.e. this 30 mL of air was held at two atm). With the syringe holder now keeping the syringe plunger depressed, opening the three-way tap allowed 30 mL of air to be rapidly discharged.

Microbubble detection

A GAMPT BC100 pulsed ultrasound Doppler system (GAMPT mbH, Merseburg, Germany) was used to detect microbubbles. The GAMPT Doppler was configured for microbubble detection in the 10–500 µm range. The instrument is unable to quantify the volume of bubbles larger than 500 µm; so, if any bubble of >500 µm diameter occurred, a default 65.45 nL with a count of one was recorded (i.e. one bubble of 500 µm diameter). In addition to a factory calibration, during measurement the instrument automatically self-calibrated so that parameters such as tubing material, blood concentration and flow speed have no influence (2).

A transluminal 3/8" ID microbubble detection probe was attached to the tubing approximately 50 cm distal to the outflow of the test arterial filter. The detection probe was connected to the bubble counter BC100; in turn connected to a laptop computer running BCView version 3.4.4 data acquisition software (GAMPT mbH, Merseburg, Germany). To maximise the reproducibility of all readings, the same probe was used. Ultrasonic gel was used to couple the probe to the tubing to exclude any air. A second microbubble detection probe was positioned 50 cm proximal to the test arterial filter (after the roller pump) to record inlet air entry. Immediately prior to bolusing, a sixty-second baseline measurement was recorded once the background bubble count had dropped to ≤4 counts/sec. A measurement was made for 180 seconds immediately upon bolusing. Thus a total of four sixty-second measurement intervals were created.

Test procedure

The test arterial filters were randomly subjected to two pump flows (3 L/min; 5 L/min). Both flow rates were chosen to represent typical adult pump flows. A variability of approximately 2.5% is given for flows (i.e. 5000 mL/min ± 125 mL/min) and 5% for reservoir levels (i.e. 1000 mL ± 50 mL).

Three copies of the two arterial filter designs at both flow rates yielded a total of 12 scenarios. These 12 scenarios (tests) were randomly sequenced and performed in one trial; three trials were run for a total of 36 tests. Arterial line pressures were kept at 120 mmHg at 3 L/min pump flow, and 180 mmHg at 5 L/min pump flow.

Statistical Analysis

Microbubble counts, volumes and sizes in tables are presented as medians and interquartile ranges. Microbubble size was derived from the median bubble size transmitted during each 60 second interval. A null hypothesis of no difference in median microbubble count, volume or size, transmitted during air bolus between the two arterial filters or within each arterial filter – at varying flows or times – was rejected if $p < .05$.

To compare the differences between the two arterial filters' microbubble transmission (count, volume and size) during air bolus at the two flow rates, non-parametric comparisons (using Mann-Whitney U tests for two groups) were computed. Non-parametric Wilcoxon Signed-Rank Tests were used to compare each of the arterial filter's [within] difference in microbubble transmission during air bolus (count, volume and size) at varying flow rates. To compare each of the arterial filter's [within] difference in microbubble transmission during baseline and air bolus (count and volume) and during air bolus only (size), the non-parametric Friedman Test was used. Post-hoc analysis with Wilcoxon Signed-Rank Tests was conducted with a Bonferroni correction applied.

The percentage of the bubble count of >500 µm in diameter is identified in the Tables. Statistical analyses were performed using StatView (StatView; Abacus Concepts, Berkeley, CA) and Microsoft Excel.

RESULTS

Part A) Microbubble transmission – count

1) Microbubble transmission [count] – between arterial filters.

Total microbubbles transmitted by the AF-100 during microbubble challenge was similar to the CB351 system at both pump flow rates of 3 L/min and 5 L/min. See Table 2 and Figure 2. And more specifically, similar during the first minute, second minute and third minute periods

There was no significant difference in total inflowing-upstream bubble counts between the two filters at 3 L/min, or at 5 L/min pump flows.

2) Microbubble transmission [count] – within each arterial filter at 3 L/min versus 5 L/min pump flows.

Consistently, the total microbubble count transmitted during microbubble challenge at 3 L/min pump flow was significantly greater than transmitted at 5 L/min in both arterial filters (both scenarios: $p = .008$). See Table 2.

The total inflowing –upstream microbubble count during microbubble challenge at 3 L/min pump flow was significantly higher than seen at a flow rate of 5 L/min for the AF-100 (4852 [IQR 844] vs. 4236 [IQR 2363]; $p < .05$), but not for the CB351 arterial filter (5027 [IQR 1160] vs. 4335 [IQR 975]).

3) Microbubble transmission [count] – within each arterial filter during baseline, first, second and third minutes.

Among/within both arterial filters, the number of transmitted microbubbles significantly altered over the four time periods (baseline, first, second and third minutes) at both pump flow rates (all four scenarios: $p < .0001$). Post hoc analysis identified significant differences between all time comparisons ($p < .05$). (Table 2; Figure 2).

Part B) Microbubble transmission – volume

1) Microbubble transmission [volume] – between arterial filters.

Total microbubble air volume transmitted by the AF-100 during microbubble challenge was significantly less than the CB351 system at both pump flow rates of 3 L/min ($p = .015$) and 5 L/min ($p = .03$), and more specifically during the first minute (3 L/min: $p = .015$; 5 L/min: $p = .03$), second minute (3 L/min: $p = .004$; 5 L/min: $p = .005$), and third minute (3 L/min: $p = .019$; 5 L/min: $p = .024$). Notably, a small but significant difference in transmitted air volume occurred during baseline between the AF-100 and CB351 system at 5 L/min ($p = .03$); however, baseline parameters were determined by count not volume. (Table 3; Figure 3).

2) Microbubble transmission [volume] – within each arterial filter at 3 L/min versus 5 L/min pump flows.

The total microbubble air volume transmitted during microbubble challenge at 5 L/min pump flow was significantly greater than at a pump flow rate of 3 L/min in the AF-100 arterial filter ($p = .008$). However, no difference in air volume transmitted was seen in the CB351 arterial filter between both 3 L/min and 5 L/min flow rates (Table 3).

The total inflowing (upstream) microbubble air volume during microbubble challenge at 5 L/min pump flow was significantly less than at a pump flow rate of 3 L/min in both the AF-100 and CB351 arterial filters ($p < .05$). See Table 4.

3) Microbubble transmission [volume] – within each arterial filter during baseline, first, second and third minutes.

Among both arterial filters, transmitted microbubble air volume significantly altered over the four time periods (baseline, first, second and third minutes) at both pump flow rates (all four scenarios: $p < .0001$). Post hoc analysis identified significant differences between all time comparisons ($p < .05$) (Table 3; Figure 3).

4) Microbubble transmission [volume]: trapped percent – between and within arterial filters.

At both pump flow rates, the AF-100 trapped a greater percentage of inflowing (upstream) air than the CB351 system (3 and 5 L/min: $p = .0003$). The trapped percent of introduced air significantly dropped (became less efficient) at the higher pump flow rate for both arterial filters (both scenarios: $p = .008$). See Table 4.

Part C) Microbubble transmission – size

1) Microbubble transmission [size] – between arterial filters.

Median microbubble size transmitted by the AF-100 was smaller than that of the CB351 system during each sequential minute at pump flow rates of 3 L/min (first & second minutes: $p = .0003$; third minute: $p = .009$) and at 5 L/min (first & second minutes: $p = .0003$; third minute: $p = .002$). (Table 5; Figure 4).

2) Microbubble transmission [size] – within each arterial filter during the first, second and third minutes.

Median microbubble size transmitted significantly dropped over the three time periods within the CB351 or AF-100 filter at 5 L/min (CB351: $p = .0003$; AF-100: $p = .001$), and at 3 L/min (CB351: $p = .0009$; AF-100: $p = .0006$). In both filters and at either 3 or 5 L/min, post hoc analysis identified a significant difference in size between first and second, first and third minutes, but not between second and third minutes. (Table 5; Figure 4).

Part D) Pressure gradient

1) Pressure gradient – between arterial filters.

The AF-100 pressure gradient at 5 L/min was slightly lower–though statistically significantly so–when compared with the CB351 ($p < .001$); however, no differences were seen at 3 L/min (Table 6).

Discussion

The results of this in-vitro study show that the transmission of introduced air by the Affinity AF-100 arterial filter is not more than the Affinity CB351 arterial filter over a range of pump flow rates, suggesting that it is as safe in terms of air handling.

More specifically, this study shows that during air bolusing into the ECC, both arterial filters transmit microbubbles. However, the Affinity AF-100 transmits smaller sized microbubbles and less air volume than the Affinity CB351.

Both arterial filters transmitted microbubbles that had been introduced into the ECC. This is not surprising as no commercial arterial filter has yet been designed to eliminate all arterial line air returning to the patient (3).

As noted by other investigators, the proportion of the bubble volume transmitted by both arterial filters increased with higher pump flows (4). Increasing the pump flow shortens the blood transit time (dwell time) within the arterial filter. At lower flows, the buoyancy of a bubble may overcome drag forces allowing the bubble to rise and be purged. However, as the velocity of the blood within the filter increases, the drag forces become more dominant thereby trapping bubbles onto the filter medium. Moreover, the higher arterial filter pressures associated with increased flows would compress bubbles (Boyle's law) thereby further deceasing bubble buoyancy. Additionally, by exceeding the bubble point pressure (BPP) of the wetted micropore, bubbles may be squeezed through the screen filter's pores. Microbubbles larger than the pore size would be retained by the filter medium until a higher pressure gradient across the filter – here associated with increased pump flows – exceeds the BPP that resists the bubbles' transit, thereby forcing these bubbles through the pores [see Appendix].

Of note is that the bubble count transmitted actually dropped with increasing pump flow. Upon closer inspection the inflowing (upstream) bubble count was significantly less at 5 L/min than at 3 L/min for the AF-100 alone, while the inflowing bubble volume was significantly less at 5 L/min than at 3 L/min for both filters. The higher flows have reduced the delivery of injected microbubbles to the arterial filter, perhaps by increasing the efficiency of the soft-shell reservoir to retain the air. Any future study should rectify this issue in the circuit design. However, increasing the pump flow did reduce the ability of the filter to trap bubbles.

The number of bubbles transmitted was similar for both arterial filters; however, the AF-100 transmitted bubbles that were smaller and consequently of less total volume of air than was passed through the CB351. Classical studies have established the positive relationship between carotid or middle cerebral artery Doppler detected embolic count and neuropsychological impairment on CPB (5–7). It would be reasonable to assume – despite not quantifying bubble sizes – that their higher counts were associated with increased embolic loads. Even though both filters in this study transmitted the same number of bubbles, as the AF-100 transmitted smaller bubbles and hence less air volume, there should be a reduced embolic load – which would be expected to be better tolerated by the patient.

The diameter of the filter pore is inversely related to BPP; a smaller pore size would increase the BPP – thereby not only would the smaller pore be more effective at trapping bubbles, a higher pressure gradient across the filter medium would be needed to force bubbles through the pores [see Appendix]. Our study confirmed the findings of others in demonstrating the benefits of smaller pore size in reducing bubble transmission. Back in 1983, Massimino and co-workers investigated the efficiency of seven – now presumably obsolete – screen arterial filters (pore size 20–40-µm) in removing particulates (polystyrene microspheres) and microbubbles. With particulates, efficiency was directly related to pore size (4). Regarding microbubbles, this principle also held true with the notable exception of a 33-µm filter performing worse than the 40-µm filters. This is not unanticipated as unlike particulates, bubbles can change size or shape, disintegrate or coalesce, dissolve or remerge. They noted that other factors in filter design such as fluid dynamics may also play a substantial role in filter efficiency (8). However, modern filter design would be expected to have addressed some of the dynamic blood handling issues making pore size more relevant now. Padayachee and colleagues clinically demonstrated the superiority of a 25-µm arterial filter over a 40-µm filter in reducing the gaseous microemboli (GME) count detected at the middle cerebral artery by Doppler ultrasound when using bubble oxygenators (9). Importantly, both filters showed marked reductions in GME when compared to the use of no arterial filter. A reduced arterial line embolic load [count] was also noted in Jabur's recent clinical study using a 20-µm filter versus a 40-µm arterial line filter (10). Not only did the 20-µm filter show improved filtration for bubbles less than 40 microns, but continued to show superior filtration for bubbles greater than 40 microns in size. An in-vitro comparison of ten arterial filters (eight 40-µm, two 20–21-µm) ranked the two smaller pore sized filters highest in terms of percent air volume removal (93.9–95.9%), whilst the 40-µm filters were noticeable less efficient in GME handling (56.3–81.3%) (11). Finally, Riley's in-vitro study ranking of ten arterial line filters for GME performance also concluded that the smaller the pore rating the higher the GME (count) separation ability rank. For example, arterial filters rated from 21–27-µm removed approximately 97–99% of 40–45 micron GME, while arterial filters rated from 37–43-µm were only 72–78% efficient (12). An exception was the 40-µm uniquely designed leukocyte reduction filter which performed similarly to a 20-µm arterial filter.

Although a smaller filter pore size increases the efficiency of the filter in capturing bubbles, the subsequently reduced pore-lumen area could theoretically increase blood shear stress resulting in increased blood trauma (13). Haemolysis, thrombocytopaenia and exacerbation of inflammation could be anticipated. Nonetheless, arterial filters are designed to slow the velocity of blood flowing through the large surface area media which, when combined with anticoagulation and haemodilution, may attenuate any blood trauma even with smaller pore sizes (12). However, this is an area for further investigation.

The consideration of a smaller sized arterial filter is derived from a desire to reduce the circuit prime volumes – and has been rationalized with Class 1, Level A evidence (1). By minimizing haemodilution, haemoglobin and clotting factor concentrations are preserved thereby reducing the need for homologous blood transfusions. Furthermore, the smaller device with a smaller 'foreign' surface area should theoretically reduce the body's inflammatory response.

Nevertheless, when it comes to gross air handling, a smaller volume arterial filter may be less capable of handling large quantities of air – especially if the air purge is closed. The filter design is partly dependent on dropping the blood flow velocity to allow more time for bubbles to separate from the blood and rise to the purge port (14); here, a larger volume promotes a greater drop in fluid velocity (15). In Yarham and Mulholland's in-vitro comparison of arterial filters, the smaller prime filters were less effective at handling a gross air bolus challenge. Interestingly, when comparing identical filters which differed only in pore size, the smaller pore size filter could handle a larger bolus of air (11). Although the AF-100 has a smaller prime volume than the CB351, having a smaller pore size may attenuate any difference in gross air handling between the two filters – though a further study would need to confirm this.

The smaller pore size of the AF-100 could theoretically have increased the filter's resistance to flow – but was not observed. Riley noted a lack of correlation between filter pore size and flow resistance suggesting "… that fluid rheology, filter housing geometry, filter media surface area, and … surface [coatings] …" had a greater role to play (12). The pressure drop across the filter is important – apart from traumatizing blood, it could contribute to exceeding the filter's BPP, facilitate bubbles coming out of solution post filter, or compress existing bubbles pre filter permitting their transmission. However, the pressure gradient across the filter media is actually quite low with differences between filters probably more dependent on factors other than pore size (16).

The surface coatings on the two Affinity filters tested are different and could theoretically influence their bubble handling capabilities. Biocompatible surface coatings make the filter material more hydrophilic thereby reducing the surface wetting angle between the fluid and the filter media. Consequently, not only does the arterial filter become easier to prime with a hydrophilic surface coating, but the BPP is increased, thereby increasing the filter's ability to remove incoming bubbles (13).

Future studies would ideally compare arterial filters with similar surface coatings to exclude this variable impacting on outcomes; however, different surface coatings would be expected to have a minimal effect.

It is not unexpected for bubbles smaller than the pore size to pass through the filter. However, both arterial filters allowed the transmission of bubbles larger than their rated pore size. This phenomenon has been observed by others (12) and may be due to several mechanisms. One mechanism already described occurs when microbubbles larger than the pore size are retained by the filter medium until the pressure gradient across the filter exceeds the BPP of the wetted micropore. These larger-than-pore-size bubbles distort through the pore only to expand in the lower pressure environment post filter (Boyle's law). Secondly, as a result of pressure drops across the filter medium, dissolved gases could re-emerge and coalesce post filter (Henry's law). A third reason is that larger bubbles disintegrate on the filter medium, pass through as smaller bubbles, then recoalesce post filter (17). A further explanation is that the GAMPT bubble counter tends to overestimate bubble size – particularly at higher flow rates (18). Thus the real bubble may be smaller than measured. As demonstrated, these mechanisms would be enhanced during the first moments after air bolusing, when the filter becomes temporarily overwhelmed, as bubbles larger than the pore size accumulate on the filter screen and partially obstruct fluid flow. These mechanisms would also be expected to be heightened at higher pump flows due to: higher flow velocities overwhelming buoyancy forces, higher pressures overwhelming the BPP, the compression of bubbles, and increasing gas solubility. However, we could not conclusively demonstrate this phenomenon with higher flows probably due to the study's unexpected reduced upstream bubble volume during the higher pump flow rate.

Our arterial filters were not purged during the air bolus challenge as this was our normal clinical practice. Medtronic states that the arterial filter can be operated in the open or closed setting but do cede that opening the purge will assist in the removal of any air entering the filter (19). Berman recommends using the purge to prevent excessive buildup of pressure on the inlet of the filter that could exceed the BPP (16). Not purging the arterial filter makes it only a bubble trap, which may result in it becoming a source of bubbles; effective filtration requires bubbles to be both captured and eliminated from the circulation (14). However, this gave us an insight into how the filters behave under the most demanding scenario.

It is challenging for the perfusionist-researcher to compare published arterial filter performance studies. Although there are guidelines on evaluating arterial filters, they are nonspecific and do not satisfy the requirements for perfusionist in evaluating and comparing arterial filters (20). Ideally, there should be a consensus on studies having a consistent standardised bench top evaluation criteria to appraise arterial filter performance including circuit design, prime composition and temperature, bubble challenge specifics, etc.

In summary, we investigated the relative microbubble handling capabilities of two arterial filters: the Affinity CB351 and the Affinity AF-100. These arterial filters' relative microbubble handling capabilities were examined under equal in-vitro conditions of air bolus, while varying pump flow rates. We conclude that, under the parameters of this in-vitro study, the Affinity AF-100 is as safe as the Affinity CB351 in terms of microbubble handling. Importantly, as both arterial filters transmitted microbubbles during air introduction – particularly at the higher pump flows, it is important to develop strategies to minimise microbubble entry into the ECC.

Appendix

$$BPP = \frac{(4\ \gamma \cos \phi)}{D}$$

BPP = bubble point pressure
γ = surface tension of the fluid
cos ø = wetting angle
D = diameter of the filter pore

Bubble retention within the wetted filter is accomplished by surface-active forces, which maintain fluid in the pores and prevents displacement of this fluid by bubbles. These forces are composed of adhesive forces (cos ø; wetting angle) holding fluid onto the filter medium, and cohesive forces (γ; surface tension) holding fluid within the pores. Together these forces constitute the bubble point pressure (BPP) – a measure of the resistance of the fluid to be displaced from the pore by a bubble (16).

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Table 1. Arterial filters specifications.

	Affinity AF-100 Arterial Filter BB851	Affinity Arterial Filter CB351
Min–Max blood flow rate	1–7 L/min	1–7 L/min
Static priming volume	100 mL	212 mL
Screen size	30-µm	38-µm
Coating	Balance	Carmeda®
Inlet/Outlet size	3/8" / 3/8"	3/8" / 3/8"
Surface area	290 cm^2	645 cm^2
Filter type & design	Polyester mesh with polypropylene support concentrically pleated together	Concentric pleated polyester mesh supported by a 'comb' structure

Table 2. Microbubble transmission [count]: median microbubbles count in the outflow of the two arterial filters during baseline and microbubble challenge with pump flows of 3 L/min or 5 L/min.

	3 L/min					5 L/min				
	Baseline	1st Minute	2nd Minute	3rd Minute	**Total**	Baseline	1st Minute	2nd Minute	3rd Minute	**Total**
CB351	7.0	4242	1056	252	**5550**	16	3987	726	211	**4896***
	(16.0)	(851.3)	(569.3)	(270.5)	**(1643)**	(11)	(640)	(383.5)	(223)	**(1227)**
AF-100	10.0	4295	1101	182	**5578**	16	4160	654	190	**5211***
	(19.5)	(392.5)	(556.5)	(233.8)	**(1127)**	(13)	(664.5)	(451.3)	(168.5)	**(1321.3)**

Data are medians and (interquartile range).

** p< .01, Same arterial filter 3 L/min vs. 5 L/min.*

Table 3. Microbubble transmission [volume]: median microbubbles volume (nL) in the outflow of the two arterial filters during baseline and microbubble challenge with pump flows of 3 L/min or 5 L/min.

	3 L/min					5 L/min				
	Baseline	1st Minute	2nd Minute	3rd Minute	**Total**	Baseline	1st Minute	2nd Minute	3rd Minute	**Total**
CB351	1.2	3603*	180.8†	45.4*	**3829***	3.5*	3783*	143.7†	46.9*	**3970***
	(1.6)	(2365)	(134.6)	(42.9)	**(2540)**	(2.1)	(3296)	(93.7)	(47.1)	**(3469)**
AF-100	0.5	1995	92	17.2	**2073**	1.1	2448	71	21.0	**2562‡**
	(1.40)	(768)	(58.6)	(15.7)	**(831)**	(1.1)	(1418)	(64.3)	(18.6)	**(1494)**

Data are medians and (interquartile range).

** p< .05, CB351 vs. AF-100.*
† p< .01, CB351 vs. AF-100.
‡ p< .05, Same arterial filter 3 L/min vs. 5 L/min.

Table 4. Microbubble transmission [volume]: median trapped percentage of microbubbles volume in the outflow of the two arterial filters during microbubble challenge and pump flows of 3 L/min or 5 L/min over 3 minutes.

	3 L/min			5 L/min		
	Inlet (nL)	Outlet (nL)	**Trap (%)**	Inlet (nL)	Outlet (nL)	**Trap (%)**
CB351	22840||	3829*	**83.8%†**	15889‡§	3970*	**76.2%†‡**
	(12175)	(2540)	**(2.4)**	(9068)	(3469)	**(5.4)**
AF-100	21979§	2073	**90.4%**	15401‡§	2562‡	**83.9%‡**
	(7219)	(831)	**(1.6)**	(8922)	(1494)	**(1.9)**

Data are medians and (interquartile range).

**p< .05, CB351 vs. AF-100.*
†p< .001, CB351 vs. AF-100.
‡ p< .05, Same arterial filter 3 L/min vs. 5 L/min.
§ 0.3%, bubbles >500 µm diameter.
|| 0.5%, bubbles >500 µm diameter.

Table 5. Microbubble transmission [size]: median microbubbles size (µm) in the outflow of the two arterial filters during microbubble challenge and pump flows of 3 L/min or 5 L/min.

	3 L/min			5 L/min		
	1st Minute	2nd Minute	3rd Minute	1st Minute	2nd Minute	3rd Minute
CB351	88.0*	51.0†	51.0†	89.0*	54.0†	52.0†
	(17.8)	(3.3)	(4.5)	(16.8)	(4.5)	(5.1)
AF-100	72.0	40.0	39.0	77.0	43.0	43.0
	(8.5)	(2.0)	(2.0)	(7.3)	(2.0)	(1.5)

Data are medians and (interquartile range).

** p< .01, CB351 vs. AF-100.*
† p< .001, CB351 vs. AF-100.

Table 6. Transarterial filter pressure gradient pump flows of 3 L/min and 5 L/min.

	3 L/min	5 L/min
CB351	55.0	125.0*
	(1.8)	(6.3)
AF-100	50.0	120.0
	(7)	(6.3)

Data are medians and (interquartile range).

** p< .001, CB351 vs. AF-100.*

Figure 1. Diagramme of in-vitro circuit.

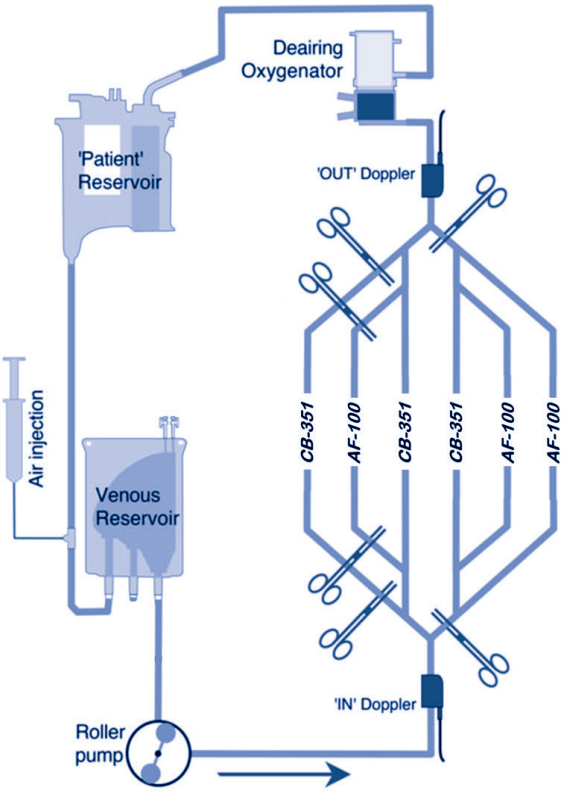


Figure 2. Boxplots of microbubble transmission (count) in the outflow of the two arterial filters during bolus air and pump flows of 3 L/min and 5 L/min measured during the first, second and third minutes.

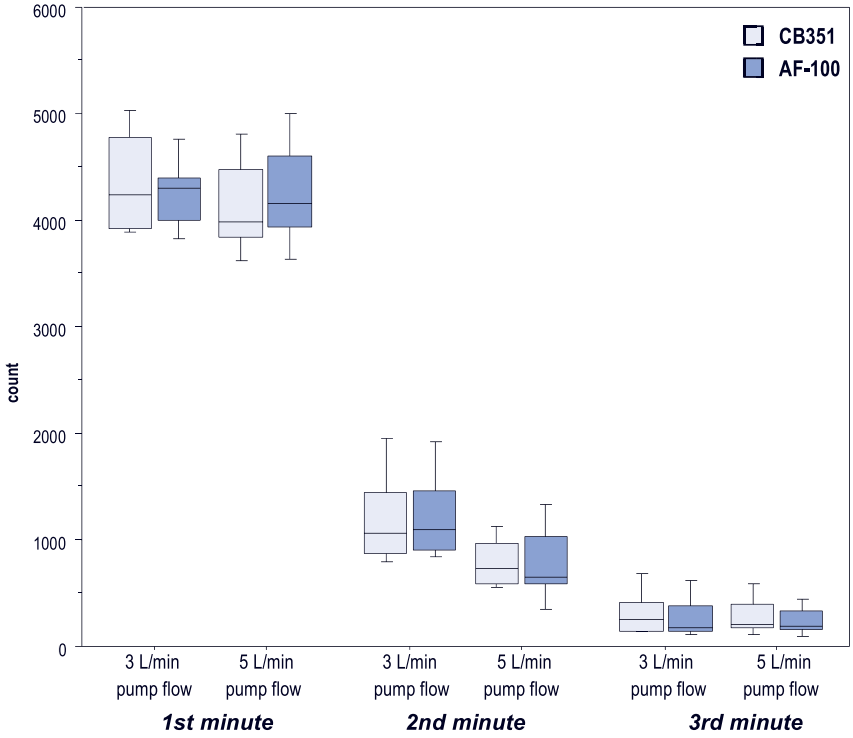


Figure 3. Boxplots of microbubble transmission (volume nL) in the outflow of the two arterial filters during bolus air and pump flows of 3 L/min and 5 L/min measured during the first, second and third minutes.

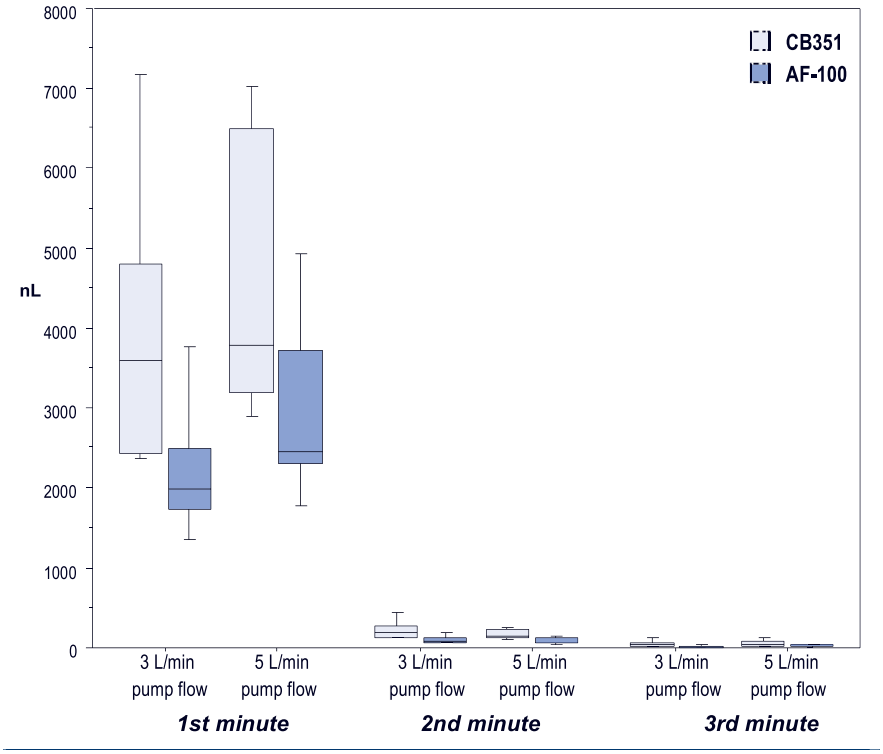
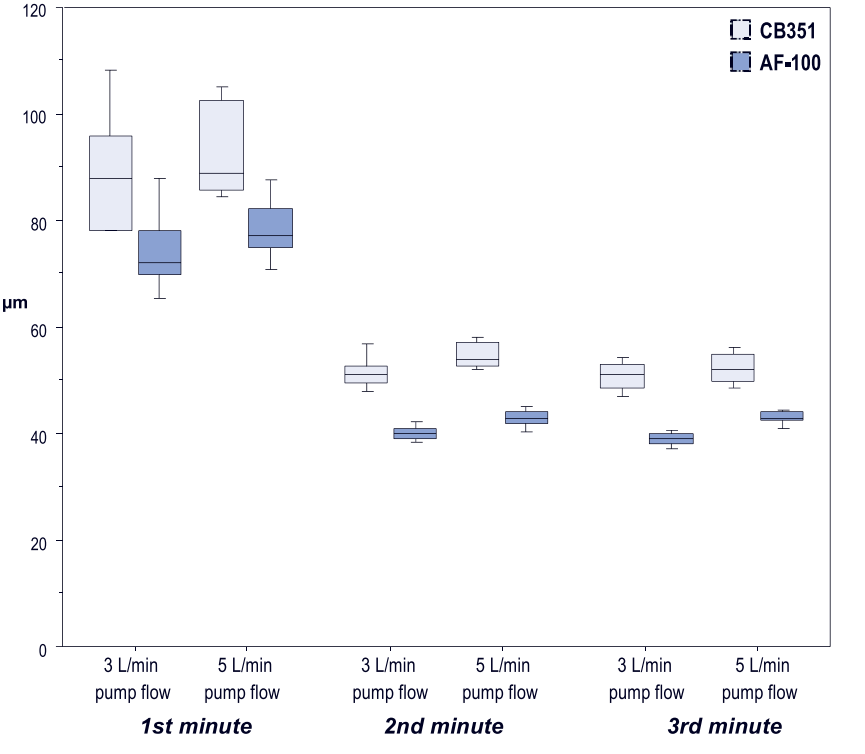


Figure 4. Boxplots of microbubble transmission (size µm) in the outflow of the two arterial filters during bolus air and pump flows of 3 L/min and 5 L/min measured during the first, second and third minutes.



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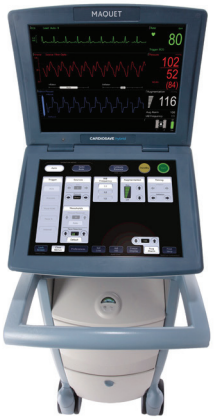

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Original Article

CLINICAL EVALUATION OF EMBOLI REMOVAL BY INTEGRATED VERSUS NON-INTEGRATED ARTERIAL FILTERS IN NEW GENERATION OXYGENATORS

Ghazwan N S Jabur, *MSc, CCP (Aust)* [§], Karishma Sidhu, *MSc*[¶],
Timothy W Willcox, *CCP (Aust)* [§¥], Simon J Mitchell, *PhD, FANZCA*[¥§],

[§]*Green Lane Clinical Perfusion, Auckland City Hospital, Auckland New Zealand*
[¶]*Cardiac Physiology, Auckland City Hospital, Auckland New Zealand*
[¥]*Department of Anesthesiology, Faculty of Medical and Health Sciences, University of Auckland, Auckland, New Zealand*

– Full version of this article can be found in the Perfusion journal –

Objective

To compare the emboli filtration efficiency of five integrated or non-integrated oxygenator-filter combinations in cardiopulmonary bypass circuits.

Methods

Fifty one adult patients underwent surgery using a circuit with an integrated filtration oxygenator or non-integrated oxygenator with a separate 20µm arterial line filter (Sorin Dideco Avant D903 + Pall AL20 (n=12), Sorin Inspire 6 M + Pall AL20 (n=10), Sorin Inspire 6M F (n=9), Terumo FX25 (n=10), Medtronic Fusion (n=10)). The Emboli Detection and Classification quantifier was used to count emboli upstream and downstream of the primary filter throughout cardiopulmonary bypass. The primary outcome measure was to compare the devices in respect of the median proportion of emboli removed.

Results

One device (Sorin Inspire 6 M + Pall AL20) exhibited a significantly greater median percentage reduction (96.77%, IQR=95.48 – 98.45) in total emboli counts compared to all other devices tested (p=0.0062 – 0.0002). In comparisons between other units, all removed a greater percentage of emboli than one device (Medtronic Fusion), but there were no other significant differences.

Conclusion

The new generation Sorin Inspire 6 M with a stand-alone 20µm arterial filter appeared most efficient at removing incoming emboli from the circuit. No firm conclusions can be drawn about the relative efficacy of emboli removal by units categorised by class (integrated vs non-integrated) however the stand-alone 20µm arterial filter presently sets a contemporary standard against which other configurations of equipment can be judged.

Keywords

Cardiopulmonary Bypass; Arterial Line Filter; Integrated Filtration Oxygenator; Gaseous Microemboli

Introduction

Exposure to microemboli during cardiopulmonary bypass (CPB) may contribute to post-operative cognitive dysfunction (POCD) following cardiac surgery.[1-4] Incorporation of arterial line filtration in the CPB circuit has resulted in reduced embolic load to the patient with associated reduced neurological deficit after cardiac surgery[1,5] and is recommended as a standard of care.6 Technological advances, including finer filtration of both inflow to the venous reservoir and outflow from the arterial line filter, have been shown to further reduce the embolic load from CPB circuits.[7-9]

A recent design development in new generation oxygenators has been the integration of the arterial filter into the body of the oxygenator to reduce prime volume and simplify the circuit. The Green Lane Cardiac Surgical Unit recently undertook a pre-tender clinical evaluation in circuits using several integrated and non-integrated oxygenators. As part of this evaluation the circuits were monitored for emboli during clinical use in order to identify any obvious differences in elution of emboli into the arterial line related to circuit design.

Materials and Methods

With institutional approval, an audit of emboli in the CPB circuit was undertaken as part of a routine pre-tender evaluation of the oxygenators listed in table 1. The device specifications are shown in table 2. The audit was conducted in 51 elective adult cardiac operations undertaken over the evaluation period when there was an operator available for the emboli detection and classification (EDAC) quantifier (Luna Innovations, Roanoke, VA). Nine to twelve procedures were monitored for each oxygenator-filter combination. Allocation of oxygenators to cases was conducted such that one perfusionist conducted at least four procedures for each device type, as is normal practice in such evaluations. The conduct of bypass was strictly according to routine institutional practice.

We subdivided the evaluated oxygenators into two groups: “Group A” were non-integrated oxygenator -arterial line filter (ALF) combinations and “Group B” were integrated oxygenator-arterial filter devices (table 1). All circuits incorporated the hard shell venous reservoir (HSVR) supplied as standard with the oxygenator being used. To comply with routine institutional practice the ‘Group B” circuits with integrated arterial filtration also included a separate

Pall AL20 ALF.

Circuit components were deployed on a Stöckert S3 heart-lung machine (Sorin Group GmbH, Munich, Germany) using SMARxT™ polyvinyl chloride tubing (COBE Cardiovascular, Arvada, CO) and silicone replacement pump raceway tubing (Natvar, City of Industry, Los Angeles, CA). A MYOtherm XP cardioplegia delivery system (Medtronic, Minneapolis, MN) was used in all cases. All circuits were flushed with CO_2 through the ALF for approximately 5 minutes at 1 L/min prior to priming with Plasma-Lyte 148 (Baxter International Corporation, NSW, Australia). Cephazolin (1g) and heparin (100 IU/kg) were added to the circuits after priming.

CPB was conducted according to usual institutional practice including: mean arterial pressure (MAP) ≥50 mmHg, PaO_2 20-30Kpa, venous oxygen saturations >70%, non-pulsatile flow (2.0-3.0 $L.m^{-2}.min^{-1}$), target haematocrit of >0.22 during CPB, DO_2i>270mlmin-$1m^2$, active cooling or passive drifting in temperature to mild hypothermia (34-32°C), α –stat acid/base management, and activated clotting time (ACT) >480 seconds. The ALF in the non-integrated oxygenator circuits was constantly purged during CPB via a connection to the venous inlet port. The integrated filtration oxygenators were purged during CPB according to manufacturer recommendations. The minimum volume sensor level was set to 250 - 300mL on all HSVRs.

The EDAC system was used to measure emboli in the circuits as follows: in both groups site one was immediately pre-oxygenator; in Group A (non-integrated oxygenators) site two was post oxygenator / pre stand-alone ALF, and site 3 was post stand-alone ALF. In Group B (integrated filtration oxygenators) site 2 was post integrated oxygenator-filter and site 3 (post stand-alone ALF) was monitored but not reported (figure 1).

Intraoperative emboli count was continuous from the initiation of CPB until the first termination of CPB. Data from each sensor site were recorded by the EDAC quantifier for emboli counts within 10µm size bands.

For each operation, the emboli count recorded at each of the EDAC sites was totalled, both for each size band and for all sizes combined, and these counts were divided by the duration of CPB to normalise the data for different durations on bypass. For simplicity, although these data effectively represent a "count per minute of CPB", we subsequently refer only to "counts". Pooled data for cases perfused with each oxygenator-filter combination were then used to calculate a median emboli count at each EDAC monitoring site.

The proportion of emboli removed by the oxygenator-filter combination for each procedure was calculated by subtracting the count downstream of the stand-alone filter (site 3 in Group A circuits) or integrated filter (site 2 in Group B circuits) from the count upstream of the oxygenator (site 1 in both circuits) and dividing the result by the count upstream of the oxygenator. Pooled data for cases perfused with each oxygenator-filter combination were then used to calculate the median proportion removed by that particular combination. The primary outcome measure was a comparison between oxygenator-filter combinations of the proportion of emboli (expressed as a percentage) removed. As a secondary outcome measure we performed the same comparisons with emboli stratified into 10µm size bands between 0 and 100µm.

Statistical Analysis

The median percentage of emboli removed by the oxygenator-filter units was compared for all possible combinations of evaluated devices using multiple Mann–Whitney U tests. Similarly, multiple Mann–Whitney U tests were used in the secondary analysis to compare the median percentages of emboli removed for all possible combinations of tested devices with emboli stratified into 10µm size bands. The Kruskal-Wallis test was used to determine any difference in duration of CPB between oxygenators. Hochberg's adjustment was applied to the *p*-values to adjust for multiple tests. The raw *p*-values and the adjusted significance of the raw *p*-values are reported. Statistical analyses were performed using SAS version 9.3 (SAS Institute, Cary, NC). All *p*-values resulted from two sided tests.

Results

The 51 audited procedures were a mixture of open and closed chamber operations (Table 3). There was no significant difference (p=0.35) in the median duration of CPB between the oxygenator types (median bypass duration ranged between 81.73 and 109.5 minutes).

The primary outcome measure is reported in Tables 3 and 4. Due to the observational nature of this audit (no attempt was made to manipulate incoming embolic load) and small sample size of oxygenator-filter combinations, natural variations in emboli counts immediately pre oxygenator were seen. Nevertheless, despite an incoming (pre-oxygenator) emboli count among the highest of all units, the Group A Sorin Inspire 6 M + 20µm ALF exhibited a significantly greater percentage reduction in median total emboli count compared to all other devices (Table 4). In comparisons between other units, all removed a greater percentage of incoming emboli than the Medtronic Fusion integrated unit, but there were no other significant differences (Table 4). When emboli less than 10µm were excluded from this analysis, the percentage reduction of emboli for the Group A Sorin Inspire 6 M + 20µm ALF remained significantly greater than all other devices except for the Group B Sorin Inspire 6F M. In the 10 - 100µm range there was no difference between any other device pairs (Table 4).

Table 5 shows the median percentage reduction of emboli (stratified according 10µm size bands) achieved by each oxygenator-filter combination, and the *p*-values for comparisons of all combinations of device within these size bands.

As there were EDAC sensors upstream and downstream of the Group A oxygenators in isolation we were able to compare the filtration capability of the oxygenator independent of the arterial filter. The percentage reduction in total emboli count from Site 1 to Site 2 was significantly greater for Sorin Inspire 6 M compared to the earlier generation Sorin Dideco Avant (84.48% and 39.21% respectively, p=0.0009).

Discussion

The introduction of new generation oxygenators with integrated arterial filtration has (in part) aimed to improve the safety of CPB by reducing the priming volume whilst efficiently filtering emboli.[10] To date all integrated arterial filters exceed a pore size of 20µm. This is of interest because in a recent study we demonstrated an advantage of a finer stand-alone 20µm screen

ALF [7], however current technology likely precludes 20 micron filtration in integrated devices. This clinical audit took advantage of a pre-purchase evaluation of recently introduced new generation oxygenators, some with integrated arterial filtration, to evaluate whether our previous results would remain applicable when a 20µm stand-alone filter was compared with integrated filters of larger screen size.

We found apparent differences in emboli filtration efficiency between devices. A new generation non–integrated oxygenator with a separate 20µm ALF (Sorin Inspire 6 M / Pall AL20) demonstrated superior percentage reduction of emboli compared to all others. It is notable in this regard that the Inspire 6 M oxygenator itself seemed very efficient in emboli removal when compared to the only other non-integrated oxygenator tested (Dideco Avant D903), and this may have contributed to the Inspire 6 M oxygenator – filter combination's overall efficiency. Since the ability to reliably measure emboli less than 10µm has been questioned[11] and since the clinical relevance of emboli less than 10µm on patient outcome can be debated we repeated the comparisons with the 0 – 10µm size band excluded. Exclusion of emboli less than 10µm from the analysis of total emboli removal did not change the findings in relation to comparison of the Inspire 6 M non-integrated oxygenator filter combination with other devices (Table 4) with the exception of the integrated Sorin Inspire 6F M. Some of the comparisons between other units which previously indicated a difference became insignificant (Table 4).

The obvious limitation in an observational study of this nature is that the embolic load presented to each unit could not be manipulated or standardized in any way. Different loads were presented to the various devices due to clinical variability beyond our control (Table 3). Although calculation of a "percentage removed" effectively indexes the efficacy of emboli removal to the incoming emboli count, we cannot exclude the real possibility that the performance of the units is affected by the magnitude of that incoming count or the size of the incoming emboli. Most obviously, it would seem plausible that a filter might become less efficient (that is, remove a smaller percentage of emboli) if a higher number of emboli are presented to it. In our study there were differences in numbers of incoming emboli (Table 3) which limit the conclusions that can be drawn.

The Group A non-integrated Sorin Inspire 6 M / Pall AL20 combination was exposed to an incoming emboli load that was among the highest, and that was comparable to similar loads imposed on the Group A non-integrated Dideco Avant – filter combination and the group B integrated Terumo FX25 unit (Table 3). Thus, our finding of high comparative emboli removal efficacy for the Inspire 6 M with the Pall AL20 ALF appears robust. The lack of significance in its comparison with the integrated Sorin Inspire 6F M for emboli removal at sizes greater than 10µm (Table 4) may be explained by the substantially smaller emboli inflow to the latter device.

The secondary outcome (comparison of percentage of emboli removed with emboli stratified into 10µm size bands) (Table 5) reveals few clear patterns, and as before, some comparisons are potentially confounded by differing incoming embolic loads (Table 3). Nevertheless, arguably the most interesting feature of these data was that comparison of the two non-integrated devices incorporating separate 20µm ALFs with the integrated devices (with the exception of the integrated Sorin Inspire 6F M) suggested that the non-integrated oxygen-filter combinations were superior in removing emboli in the larger size range (principally > 40µm) (Table 5). The rated screen size for the integrated filters (though the designs are not strictly a screen *per se*) are 25, 32 and 38µm for the Medtronic, Terumo and Sorin devices respectively. We have previously demonstrated superior removal of larger emboli by a smaller 20µm ALF (compared to a 40µm device)[7] and the results obtained here are probably a simple corroboration of that finding.

To our knowledge the current study is the first clinical study to investigate the difference in emboli filtration capabilities of these new generation adult integrated versus non-integrated filtration oxygenator combinations. Two other studies have reported a clinical comparison of integrated and non-integrated devices. A randomised controlled trial by Milano et al[12] investigated emboli filtration during either pulsatile or non-pulsatile flow in patients undergoing myocardial revascularisation using three different oxygenator filter combinations (integrated filtration Terumo Capiox FX25, Sorin Synthesis and the non-integrated Maquet Quadrox-i-Adult membrane oxygenator + 40µm ALF). They concluded that in these earlier generation devices, compared to those in the present study, pulsatile perfusion decreased emboli filtration with a significant difference seen in the non-integrated oxygenator. No important difference in clinical outcome and major neurologic events were observed in any circuit.

In the clinical setting the size and number of emboli introduced to the CPB circuit are variably influenced by vent suction, hemofiltration, vacuum assisted venous drainage, drug additions and air entrained to the venous line which have all previously been well documented.[11,14-18]

Although the best performing system in terms of emboli handling was a non-integrated system, we did not demonstrate a systematic disadvantage for integrated systems *per se*, and the potential for them to have other advantages must be acknowledged. For example, Gursu et al[19] compared clinical variables, inflammatory responses and transfusion needs in thirty-six patients who underwent coronary artery surgery randomised to CPB using either a Sorin Synthesis integrated filtration system or the Sorin Dideco Compactflo oxygenator plus a separate ALF. Emboli data were not reported in this comparison, however, they concluded that intraoperative haematocrit levels were higher and the need for postoperative red cell transfusion was decreased in the integrated ALF group.

Study Limitations

The principle limitation of this study is the non-standardisation of the incoming emboli load 'presented' to the various circuits. This has been discussed above. Another parameter that could not be standardised in the clinical setting was the CPB circuit flow rates which varied between 2.0-3.0 $L.m^{-2}.min^{-1}$ in accordance with normal practice. It is possible that higher flows (for example) could result in greater emboli production from known sources like cavitation at the pump head or through fractionation of larger emboli, but such phenomena should have been accounted for by measuring incoming emboli immediately upstream of the oxygenator. It is also possible that different flow rates could affect filter efficiency, though to our knowledge this is untested within the relatively narrow range of flows used here. A further obvious but essentially unavoidable limitation is the potential inaccuracies of counting and sizing of emboli by the EDAC quantifier. Although the EDAC quantifier is considered to be technologically advanced in its ability to measure and count emboli, in general it tends to underestimate the true diameter and undercount the

emboli passing in the circuit.[15] Given the questions around the ability of the EDAC to accurately discriminate emboli less than 10µm we reported our results both including and omitting the 0-10µm emboli counts. The inability of the EDAC to differentiate between solid and gaseous microemboli also leaves uncertainty as to the nature of the counted embolic load, though we suspect the vast majority are microbubbles. We did not explore the potential advantages and disadvantages of an integrated filtration system or 20µm stand-alone arterial filters in terms of blood cell damage or indices of inflammation.

The clinical significance of patient exposure to microemboli (and particularly microbubbles) is debated. It is beyond the scope of this paper to comprehensively discuss this subject but it has been recently reviewed. Mitchell and Merry (2015) presented an evidence-based case for pathogenicity of patient exposure to small arterial bubbles during cardiac surgery and CPB[20] and advocate for continued focus on prevention of emboli exposure in development of perfusion equipment and protocols. While there are numerous origins of emboli that may enter the patient's systemic circulation during cardiac surgery, transmission from the perfusion circuit continues to be reported.[14,15] It is therefore important that reduction of emboli during CPB is not overlooked and remains the subject of continued investigation. This is especially true in the context of redesign of CPB circuit components.

Conclusion

Among the units used, the new generation Sorin Inspire 6 M with a stand-alone 20µm ALF appeared most efficient at removing incoming emboli from the circuit. This may relate to the previously demonstrated efficacy of a stand-alone 20µm ALF, but our data also indicate that the Sorin Inspire oxygenator is independently effective in emboli removal. This may explain the relatively good performance of the integrated Sorin Inspire 6F M unit which incorporates the same oxygenator and has a filter screen rated at 38µm. No firm conclusions can be drawn about the relative efficacy of emboli removal by units categorised by class (integrated vs non-integrated) alone, however there is a reasonably clear signal in our data suggesting that modern integrated devices are not superior in this regard and that the stand-alone 20µm ALF presently sets a contemporary standard against which other configurations of equipment can be judged. On the basis of emboli removal we find there is no advantage to change to an integrated oxygenator-filter system at this time, but it is acknowledged that other arguments for use of these devices (not investigated here) may influence choice.

Acknowledgment

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Conflict of Interest

The Authors declare that there is no conflict of interest

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Table 1. Oxygenator group classification

GROUP A Non-Integrated Oxygenator with Stand Alone 20µm ALF	**GROUP B** Oxygenator with Integrated Arterial Filter
Sorin Dideco Avant D 903 * + Pall AL20 Arterial Line Filter ¶ n = 12	Sorin Inspire 6F M * n = 9
Sorin Inspire 6 M * + Pall AL20 Arterial Line Filter ¶ n = 10	Terumo Capiox FX25 § n = 10
	Medtronic Affinity Fusion ¥ n = 10

¶ Pall Corp. Portsmouth UK, * Sorin Group, Miranolda Italy, § Terumo Italia S.R.L, Rome, Italy, ¥ Medtronic Minneapolis, MN, USA.

Table 1. Oxygenator group classification

	Sorin Dideco Avant + Pall AL20 ALF	**Sorin Inspire 6 M + Pall AL20 ALF**	**Sorin Inspire 6F M**	**Terumo Capiox FX25**	**Medtronic Affinity Fusion**
Oxygenator Membrane Type*	Microporous Polypropylene Hollow Fibre	Microporous Polypropylene Hollow Fibre	Microporous Polypropylene Hollow Fibre	Microporous Polypropylene Hollow Fibre	Microporous Polypropylene Hollow Fibre
Max Blood Flow*	8 L/min	6 L/min	6 L/min	7 L/min	7 L/min
Static Priming Volume* (Oxy + Heat Exch ± Filter)	270mL??	191mL	292mL	260mL	260mL
Membrane Surface Area* (Oxy + Heat Exch ± Filter)	2.14m²	1.83m²	2.51m²	3.3m² ¥	2.5m²
Coating*	PC Phosphorylcoline (PHISIO)	PC Phosphorylcoline (PHISIO)	PC Phosphorylcoline (PHISIO)	Poly-2-methoxyethylacrylate (X-coating)	Balance-Biosurface (Trillium)
ALF Size*	20 micron (100mL / 0.55m²)	20 micron (100mL / 0.55m²)	38 micron	32 micron wrap around	25 micron – 6 stage

* Source; manufacturer instructions for use ¥ Source; Terumo Capiox FX Family of Oxygenators online brochure

Table 3. Comparison of emboli counts and percentage reduction between devices. Note: the median percentage of emboli removed is the median of the percentages of emboli removed in all cases perfused using each oxygenator – filter combination and cannot be calculated directly from the median pre-oxygenator and post-filter counts presented in this table.

Oxygenator	**Pre-Oxygenator Emboli Counts Median (IQR)**	**Post-Filter Emboli Counts Median (IQR)**	**Percentage Emboli Removed Median (IQR)**	**Open vs Closed Chamber Distribution**	
				Open	**Closed**
Sorin Inspire 6M + Pall AL20	1782.99 (1208.07 - 2218.5)	50.72 (24.81 - 84.54)	96.77 (95.48 – 98.45)	7	3
Dideco Avant D903 + Pall AL20	1640.47 (819.74 - 2852.01)	247.22 (133.7 - 564.3)	83.00 (77.44 – 88.18)	9	3
Medtronic Fusion	538.86 (379.04 - 2455.49)	380.60 (174.09 - 491.48)	61.75 (38.69 – 68)	4	6
Terumo FX25	2022.95 (1208.99 - 2173.13)	235.80 (166.48 - 331.58)	85.10 (70.45 – 92.02)	4	6
Sorin Inspire 6F M	396.21 (297.24 - 961.36)	106.67 (27.9 - 189.63)	88.38 (76.32 – 93.55)	4	5

Table 4. Comparisons by device of percentage reduction in total emboli count (all sizes) and in size range 10-100 micron.

Oxygenator 1	Oxygenator 2	Raw *p*-values	Adjusted Significance	10-100 micron Raw *p*-values	10-100 micron Adjusted Significance
Sorin Inspire 6 M + Pall AL20	Dideco Avant D903 + Pall AL20	0.0014	S	0.0004	S
Sorin Inspire 6 M + Pall AL20	Sorin Inspire 6F M	0.0062	S	0.0373	NS
Sorin Inspire 6 M + Pall AL20	Medtronic Fusion	0.0002	S	0.0002	S
Sorin Inspire 6 M + Pall AL20	Terumo FX25	0.0028	S	0.0004	S
Dideco Avant D903 + Pall AL20	Sorin Inspire 6F M	0.4555	NS	0.1265	NS
Dideco Avant D903 + Pall AL20	Medtronic Fusion	0.0033	S	0.0272	NS
Dideco Avant D903 + Pall AL20	Terumo FX25	0.7169	NS	0.9212	NS
Sorin Inspire 6F M	Medtronic Fusion	0.0101	S	0.016	NS
Sorin Inspire 6F M	Terumo FX25	0.5956	NS	0.1113	NS
Medtronic Fusion	Terumo FX25	0.0113	S	0.089	NS

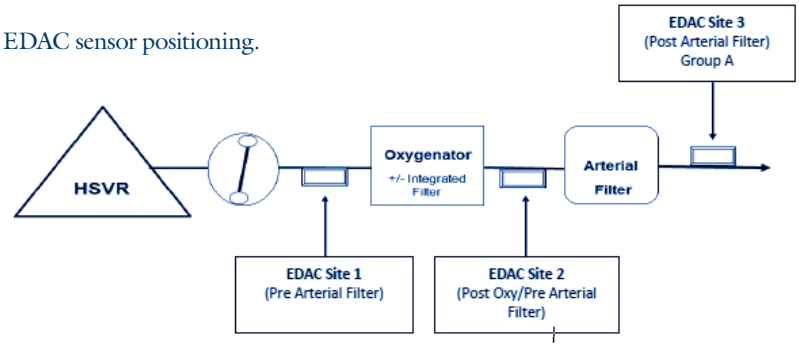
S: Significant difference NS: No significant difference
Adjusted significance: Significance after Hochberg Adjustment applied to Mann-Whitney U *p*-values

Table 5. Comparison of median percentage reduction, *p*-values and adjusted significance in total emboli count within 10 micron size bands

Comparison between		Emboli size bands -		"Median percentage reduction *p*-values (adjusted significance level)"							
Oxygenator 1	Oxygenator 2	0 - 10 µm	10 - 20 µm	20 - 30 µm	30 - 40 µm	40 - 50 µm	50 - 60 µm	60 - 70 µm	70 - 80 µm	80 - 90 µm	90 - 100 µm
Sorin Inspire 6 M + Pall AL20	Dideco Avant D903 + Pall AL20	94.5 vs 80.8 0.0027 (S)	97.7 vs 77.8 0.0002 (S)	99.3 vs 83.1 0.0003 (S)	99.3 vs 94.2 0.0051 (S)	99.2 vs 97.5 0.0443 (NS)	98.8 vs 98.6 0.5977 (NS)	98.7 vs 99.1 0.3734 (NS)	98.7 vs 99.3 0.3385 (NS)	97.5 vs 99.3 0.1052 (NS)	"98.7 vs 99.5 0.174 (NS)"
Sorin Inspire 6 M + Pall AL20	Sorin Inspire 6F M	94.5 vs 72.7 0.0029 (S)	97.7 vs 91.9 0.5500 (NS)	99.3 vs 96.0 0.0373 (NS)	99.3 vs 97.2 0.0373 (NS)	99.2 vs 95.1 0.1309 (NS)	98.8 vs 94.2 0.1779 (NS)	98.7 vs 96.7 0.0942 (NS)	98.7 vs 96.0 0.3053 (NS)	97.5 vs 95.7 0.6519 (NS)	98.7 vs 97.2 0.4857 (NS)
Sorin Inspire 6 M + Pall AL20	Medtronic Fusion	94.5 vs 57.8 0.0003 (S)	97.7 vs 56.0 0.0002 (S)	99.3 vs 55.3 0.0002 (S)	99.3 vs 65.4 0.0002 (S)	99.2 vs 78.4 0.0004 (S)	98.8 vs 85.3 0.0017 (S)	98.7 vs 88.8 0.0022 (S)	98.7 vs 93.6 0.0058 (S)	97.5 vs 94.6 0.0639 (NS)	98.7 vs 94.4 0.0639 (NS)
Sorin Inspire 6 M + Pall AL20	Terumo FX25	94.5 vs 83.2 0.0091 (NS)	97.7 vs 82.5 0.0010 (S)	99.3 vs 86.0 0.0004 (S)	99.3 vs 90.2 0.0013 (S)	99.2 vs 93.2 0.0022 (S)	98.8 vs 94.1 0.0046 (S)	98.7 vs 94.2 0.0091 (NS)	98.7 vs 93.8 0.0140 (NS)	97.5 vs 95.6 0.3068 (NS)	98.7 vs 94.7 0.1852 (NS)
Dideco Avant D903 + Pall AL20	Sorin Inspire 6F M	"80.8 vs 72.7 0.9151 (NS)"	77.8 vs 91.9 0.0700 (NS)	83.1 vs 96.0 0.0428 (NS)	94.2 vs 97.2 0.2410 (NS)	97.5 vs 95.1 0.5940 (NS)	98.6 vs 94.2 0.2410 (NS)	99.1 vs 96.7 0.0173 (NS)	99.3 vs 96.0 0.1260 (NS)	99.3 vs 95.7 0.1872 (NS)	99.5 vs 97.2 0.1075 (NS)
Dideco Avant D903 + Pall AL20	Medtronic Fusion	"80.8 vs 57.8 0.0229 (NS)"	77.8 vs 56.0 0.1062 (NS)	83.1 vs 55.3 0.0161 (NS)	94.2 vs 65.4 0.0004 (S)	97.5 vs 78.4 0.0003 (S)	98.6 vs 85.3 0.0002 (S)	99.1 vs 88.8 0.0003 (S)	99.3 vs 93.6 0.0005 (S)	99.3 vs 94.6 0.0022 (S)	99.5 vs 94.4 0.0014 (S)
Dideco Avant D903 + Pall AL20	Terumo FX25	80.8 vs 83.2 0.8175 (NS)	77.8 vs 82.5 0.2225 (NS)	83.1 vs 86.0 0.6682 (NS)	94.2 vs 90.2 0.1213 (NS)	97.5 vs 93.2 0.0092 (S)	98.6 vs 94.1 0.0027 (S)	99.1 vs 94.2 0.0014 (S)	99.3 vs 93.8 0.0009 (S)	99.3 vs 95.6 0.0191 (NS)	99.5 vs 94.7 0.0144 (NS)
Sorin Inspire 6F M	Medtronic Fusion	"72.7 vs 57.8 0.0550 (NS)"	91.9 vs 56.0 0.0247 (NS)	96.0 vs 55.3 0.0017 (S)	97.2 vs 65.4 0.0007 (S)	95.1 vs 78.4 0.0005 (S)	94.2 vs 85.3 0.0048 (S)	96.7 vs 88.8 0.0792 (NS)	96.0 vs 93.6 0.4875 (NS)	95.7 vs 94.6 0.6532 (NS)	97.2 vs 94.4 0.5954 (NS)
Sorin Inspire 6F M	Terumo FX25	72.7 vs 83.2 0.9393 (NS)	91.9 vs 82.5 0.1779 (NS)	96.0 vs 86.0 0.0373 (NS)	97.2 vs 90.2 0.0160 (NS)	95.1 vs 93.2 0.1309 (NS)	94.2 vs 94.1 0.3074 (NS)	96.7 vs 94.2 0.3477 (NS)	96.0 vs 93.8 0.8382 (NS)	95.7 vs 95.6 0.9024 (NS)	97.2 vs 94.7 0.9024 (NS)
Medtronic Fusion	Terumo FX25	57.8 vs 83.2 0.0376 (NS)	56.0 vs 82.5 0.0757 (NS)	55.3 vs 86.0 0.0257 (NS)	65.4 vs 90.2 0.0058 (S)	78.4 vs 93.2 0.0073 (NS)	85.3 vs 94.1 0.0211 (NS)	88.8 vs 94.2 0.0757 (NS)	93.6 vs 93.8 0.4727 (NS)	94.6 vs 95.6 0.6776 (NS)	94.4 vs 94.7 0.7337 (NS)

S: Significant difference NS: No significant difference
Adjusted significance: Significance after Hochberg Adjustment applied to Mann-Whitney U *p*-values

Figure 1. Circuit design showing EDAC sensor positioning.



EDAC: emboli detection and classification quantifier; HSVR: hard-shell venous reservoir

The following pages contain the abstracts from the 2nd Intraoperative Management of Blood meeting, Sydney.

THE NO BRAINER PRINCIPLES OF PATIENT BLOOD MANAGEMENT

Prof James Isbister
Consultant in Haematology and Transfusion Medicine
Clinical Professor of Medicine, Sydney Medical School
Royal North Shore Hospital of Sydney

"Muse before you Transfuse"

For the majority of patients undergoing uncomplicated elective surgery, and in most cases of cancer chemotherapy, it should be possible to minimize or avoid blood transfusion by practicing a standard of care that focuses on the management of a patient's own blood by optimizing and preserving haematopoietic reserves and tolerating deficiencies.

The term Patient Blood Management (PBM), in contrast to donor blood management (DBM), is now the widely used terminology to emphasise the patient-focus in contrast to product-focus of transfusion medicine. This paradigm shift to a patient focus has returned clinicians to managing a patient's own blood. This is 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.

Clearly, PBM "demands" (aka "No Brainer") the following and randomised clinical trials are unlikely nor appropriate, possible or ethical:-

- Administering a "therapy" for which there is no evidence for benefit can only expose a patient to potential hazards.
- Clinicians take on an extreme clinical responsibility when an intravenous cannula is inserted for the purpose of infusing any form of therapy, especial blood components.
- The first port of call for any substance infused intravenously is the lung.
- Patients with cardiac, respiratory or renal failure or compromised reserves are at risk of pulmonary oedema from the infusion of any fluid, especially blood components.
- Diagnosing and treating reversible anaemia, if time permits, is a high priority in all clinical settings, especially preoperatively.
- Taking a preoperative history for potential bleeding is haemostasis 101
- Advocating meticulous surgical haemostasis is surgical technique 101
- Involving patients in decisions regarding their clinical care, the benefits of treatment, its risks and possible alternatives is medical ethics 101

PBM is not an intervention or an alternative to allogeneic blood transfusion it is sound evidence-based clinical practice. PBM improves clinical outcomes and as a corollary minimizes or avoids unnecessary allogeneic blood transfusions, reduces risks and helps contain health costs. The use of some autologous transfusion techniques, erythropoiesis stimulating agents and anti-fibrinolytic agents may be regarded as transfusion alternatives, but also bring with them potential hazards and costs that need balancing in the same manner as the decision to transfuse.

Managing the triad of haematological risk factors for surgical patients, ie anaemia, haemorrhage and transfusion, is addressed by focusing on the three pillars of PBM. Maximizing total red cell mass, minimizing blood loss and tolerating of anaemia are of the essence. Most of the core principles of patient blood management comes from a sound understanding of physiological and pathophysiological knowledge of haemopoiesis, haemostasis and oxygen transport and the causative mechanisms of the well-understood hazards of blood transfusion.

The concept of personalized PBM is best epitomized by Sir William Osler in his aphorism: "It is much more important to know what sort of a patient has a disease than what sort of a disease a patient has."

"Light your lamp before it becomes dark"

Arabic proverb

TEAM MANAGEMENT OF TRANSFUSION: PATIENT BLOOD MANAGEMENT

Bruce D. Spiess, *MD, FAHA*
Richmond, VA
USA

Blood utilization for cardiovascular surgery is changing and the demand (usage) of allogeneic blood is decreasing. For the first time in over 100 years in the United States the total number of units transfused has dropped leading to some discomfort amongst certain blood banking centers/leadership. Cardiac surgery remains a major consumer of blood products but it has also led medicine in studying the adverse outcomes associated with transfusion. The reasons for the decrease in demand are certainly multi-factorial but the flood of literature describing risks of adverse outcome with allogeneic blood transfusion may to a great extent be fueling debate (one hopes) amongst professionals regarding when allogeneic blood is a positive therapeutic versus a risk for bad outcome. The transfusion decision is no longer dependent upon a single and simple surrogate hemoglobin transfusion trigger. A hemoglobin level is a surrogate because what we really want to know is the tissue oxygen delivery level in any given patient. Today we know that such delivery is far more complex than any one level of hemoglobin per liter of blood. One thing that is known is that 100gm/liter of blood is not an appropriate transfusion trigger. Neither is any one specific number. All patients are different and their oxygen demands changed minute to minute.

So the transfusion decision is important. Today we know that how many units are transfused is one of the most influential variables on outcome after CABG surgery. Indeed it appears to be the strongest correlate to early (in hospital) and long term (5 years) death after CABG. It is more important than the presence of diabetes, NY Heart Association classification, length of bypass, presence of CHF, etc. How can that be? One unit of blood has been associated with an 11-16% increase risk of major perioperative infection and the risks of transfusion related acute lung injury (TRALI) is running between 1-8% for all comers to heart surgery that are transfused. The risk of death in hospital with TRALI increases 13-15 fold if a patient gets TRALI. So the transfusion decision is serious and demands every member of the health care team's attention. All of us should be educated to not only the risks but alternatives and methods to conserve the patient's own blood (Patient Blood Management). Unfortunately we have a long way to go in these educational efforts. This lecture will show some of the newest data regarding adverse outcomes with transfusion, one study actually showing improvement with transfusion and present the need for collaborative teams. In the United States unfortunately the transfusion therapeutic option is presented to the patient in a very paternalistic misinformed request for patients to sign a consent. The lay public gets the lowest member of the team to present the consent form and it is most often done just prior to tart of surgery. Everything about that behavior is wrong. I have never once heard a surgeon or any other physician discuss risks of TRALI, infection, renal failure bleeding etc.

The transfusion decision should begin with the local MD, cardiologist or primary care physician. Pre-operative attention to anemia is tremendously important. Patients who are anemic do poorly in almost every type of operation. Anemia is certainly a marker for severity of chronic disease and its causes are complex. However, a number of papers have shown that with attention to detail anemia clinics can help to reduce transfusion utilization and can be cost effective. Algorithms are well published for how to approach these patients and most respond to intravenous (or oral at times) iron and multi-vitamins. The body has a complex reaction to chronic disease that decreases ferritin as well as stimulus/reaction to erythropoietin. The answer to pre-operative anemia is not universal use of erythropoietin. Perhaps of all the anemic patients only 10-15% of patients may benefit from red cell stimulating hormones. The cooperative team needs to discuss when it is appropriate to refuse surgery, delay or postpone anemic patients. The more we as a group embrace the fact that performing any elective operation in an anemic patient is a serious breach of proper care, then the more primary care physicians will work hard to resolve anemia. I believe we should refuse to do elective cases on anemic patients. In the United States, at least for heart surgery, the standard thinking has been if the patient is anemic not to postpone but to simply transfuse the patient. One group from Canada has gone so far as to suggest that since transfusion is very pro-inflammatory the patient should be brought in to the hospital early, transfused and then allowed to clear the inflammatory mediators before the secondary inflammation of cardiopulmonary bypass. To me this is misguided thinking.

In the operating room pre-bypass the anesthesia team can/should consider autologous normovolemic hemodilution (ANH). No perfect study exists showing that ANH is effective on its own in reducing allogeneic transfusions. However as part of a larger program for transfusion management it makes sense and plays into the team thinking about their use of blood. Aggressive use of ANH- taking several to 4 units of ANH blood is more effective than being conservative. Predictive algorithms and equations are available for the team to predict how much blood to withdraw based upon a target hemoglobin level on bypass.

Who should take the blood? It is probably less important whether perfusionists sequester units of blood or whether the anesthesia team performs this task. If a team can get to this discussion they are way ahead of other teams and it may be equally effective being done either way. In discussions with companies making equipment for cardiac surgery it seems that there is an unmet need and a machine to perform automated ANH might be widely utilized. The STS/SCA guidelines note that this can be used as part of an overall schema for transfusion reduction.

Anti-fibrinolytic drugs work. There is no doubt that these agents are effective in mildly decreasing blood loss. They have been less effective in reducing transfusion because transfusion is a human behavior not a measurable science driven event. Unfortunately we are left with the lysine analogues only in the United States and without getting into the depths of the aprotinin debacle it is instructive to note that the BART study has been refuted by Health Canada as well as the European

Regulatory Agency. Indeed when the data on mortality was reanalyzed with the 135 patients excluded by the BART investigators the signal for increased mortality with aprotinin was lost. It is most instructive to look back at this drug, the heated debate and note how readily physicians took sides on what should have been an unbiased dispassionate discussion. They did not take sides because they were impassioned about transfusion rather many took on a vigilante attitude to take down a drug company. New anti-inflammatory agents are being developed and aprotinin has been re-licensed in Canada and Europe. No single drug should be given to every patient and unfortunately today we do that in the United States with anti-fibrinolytic drugs. We certainly were headed that way with aprotinin.

Monitoring of cerebral oximetry, mixed venous gases, lactate and pH on bypass seem to function as surrogates for tissue oxygen demand. We should have a discussion in a pre-operative huddle or briefing regarding when we as a team will consider transfusion. I have never seen it done but it would make sense for the team to get together and to discuss at what temperature or with what parameters they would approach the use of allogeneic blood. This needs to happen.

Perfusionists use of small circuits, low priming volumes and retrograde autologous prime are all techniques that not only make sense but have been demonstrated to decrease transfusion utilization. Such decisions should not be made in a vacuum but at the least the surgeon and perfusionists should have an off line discussion of what type of circuit oxygenator and cannulas they will use with a focus upon decreasing the hemodilution necessary. Use of albumin in the circuit or being given by the anaesthesia team as a major component of fluid infusion is now showing improved survival and decreased red cell usage. Albumin is not just an oncotic fluid but a complex protein that is needed in the endothelial cell glycocalyx region and functions as a profound anti-oxidant. The surgeons and perfusionists should discuss and decide what type of coated tubing they wish to use for their patient with the idea that perhaps not all patients need the same perfusion tubing. We as yet do not have the perfect perfusion tubing, though data exist showing that at least inflammatory mediators can be decreased by the use of some types of tubing. Mediastinal suction versus cell saver use for mediastinal fluids is another point of discussion.

At the end of bypass the team should understand how they will process blood left in the bypass circuit- i.e. pump blood. Should it be all re-infused directly to the patient, sent to a cell saver spun, washed and re-infused, should modified ultrafiltration be utilized or a Hemobag used in conjunction with the modified ultrafiltration? There are strengths and weaknesses of each approach but he team at large needs to decide.

The approach to a bleeding patient is particularly important and the agreement upon coagulation monitoring technology with resulting algorithms for coagulopathy treatment is a team building important exercise. If such discussions are held the team has to be willing to stand by their agreements and to have patience for when a patient is bleeding to follow the science that they have embraced. There is no place in contemporary cardiac surgery for "shot gun" clinical impression use of coagulation precursors from the blood bank. This unfortunately is the most common technique utilized in the United States as an approach to the bleeding patient. It is driven by fear and ignorance which when combined amount to bad outcomes for patients. Fresh froze plasma (FFP) has been shown to have little effect either as a prophylactic agent for anticipated bleeding, is needed in large quantities to make a difference in blood serine protease levels and carries a high risk of TRALI. Today we have four agent prothrombin complex concentrates which have balanced activated serine proteases with anti-coagulants thereby seemingly creating more homeostasis. In our center FFP has been replaced by use of PCC. Fibrinogen concentrate is approved for use in the United States and Europe and should replace the use of cryoprecipitate. Platelet transfusions may be necessary but only when guided by low platelet count, platelet dysfunction by TEG, RoTEM, Mulitplate aggregometry or Verify Now, and only in patients that are bleeding.

Post-operative cell salvage will be dealt with in another lecture but suffice it to say we are throwing away a great deal of viable red cells and the team form the ICU should be part of a comprehensive teamwork approach to the cardiac patient. The teamwork approach to patient care even extends to the ward cardiology groups, housemen and to the home primary care physicians. It is disheartening to have a cardiac patient do well through a complex operation not receive any blood products and then to have them discharged only to be transfused by their local MD because that patient "seemed anemic".

Lastly and most importantly the team needs to have a rational approach to allogeneic blood. Patients for heart surgery at times will benefit from a transfusion. There needs to be a consensus amongst the care givers as to when that benefit will outweigh the risks and to have a balanced and educated approach. Zealots regarding not transfusing could be dangerous as are those who are too free and easy with blood transfusions. Blood transfusion is a behavior. It is a behavior for which we have little or no science to guide our approaches. It is a behavior driven by fear (fear of a bad outcome or in the United States fear of lawsuit) and one without effective monitoring. That combination leads to a great deal of dysfunctional behavior. We are now learning more and more about the risks of over transfusion but rarely do we hit the sweet spot of just giving enough, not too little and not too much!

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ARE WE USING BLOOD MANAGEMENT IN CT PATIENTS: PILOT AUDIT RESULTS FOR NSW SITES?

Sally Francis
NSW Bloodwatch programme leader

The aim of this pilot audit was to develop and test a methodology to measure the perioperative patient access to PBM care principles.

A collaborative working group developed 3 data sets to: measure health services capacity to provide care based on the principles described in the perioperative PBM guidelines; assess care delivery and compliance with perioperative PBM guidelines in 20 patient episodes; and collate patient factors and outcome factors to support local reporting and case review opportunities. Patients undergoing cardiothoracic surgery, irrespective of blood and blood product use, were the group selected for the pilot audit.

8 hospitals participated in the audit, with 157 perioperative patient journeys assessed. Hospitals were provided with an individual report that included a comparison with the other de-identified participants, as well as a collated report for NSW. This allowed hospitals to easily compare their care with like services, and therefore identify improvement and spread opportunities within the group.

From a NSW perspective, system improvement and leadership opportunities were identified, specifically where it was evident that particular care elements were provided to only a small number of patients.

The perioperative audit tools have now been refined and simplified, and a further data set for medical patients has been developed.

IMMUNOMODULATION AND INTRA-OPERATIVE CELL SALVAGE

Dr Michelle Roets
Staff Specialist Anaesthesia
Royal Brisbane and Women's Hospital

Transfusion related immunomodulation (TRIM) resulting from the transfusion of allogeneic blood may be associated with an increased risk of cancer recurrence and post-operative infection. During this presentation we attempt to summarise the literature on TRIM and identify the potential reduction of this risk and therefore improved outcomes by using intra-operative cell salvage blood instead. The area of TRIM is surrounded by a large amount of controversy in the literature. The existence of this phenomenon is however clear. The exact mechanism is still unconfirmed.

Independent of whether the mechanism for TRIM is specifically human leucocyte antigen dependent or related to non-specific bioactive soluble factors, the fact remain that if intra-operative cell salvage can be used instead of allogeneic red cell transfusion the risk if TRIM may be reduced.

How practical and useful would a large RCT on immunomodulation markers during the conduct of cell salvage be?

POST-OPERATIVE COLLECTION/SALVAGE OF RED BLOOD CELLS- WHERE ARE WE TODAY?

Bruce D. Spiess, *MD, FAHA*
Virginia Commonwealth University Medical Center
Richmond, VA USA

Chest tube drainage (often called chest tube bleeding) is highly variable after heart surgery. For routine first time CABG surgery it ranges between 400-1100cc in the first 24 hours and high rates of bleeding are often defined as greater than 200 cc per hour for the first 1-4 hours. Early re-exploration for bleeding sites appears to be beneficial in decreasing secondary complications. Massive bleeding has been defined by some authors as being transfusions of 5 units or greater. Whereas in trauma massive transfusion is regarded as greater than one blood volume lost/replaced within a 24 hour period. On the other hand, clotting of the chest tubes which can occur in almost 40% of chest tubes is a potential complication in that residual fluids can build up in the chest, lead to tamponade or create a nidus for future infection.

Capture of red cells being lost through the chest drains is not a new idea. H chaff at the Mayo Clinic first described doing this in 1978. From the very beginning of heart surgery it seemed that people were practicing blood conservation using techniques for sterile collection of the drainage and reinfusion. The practice seemed to hit its highest acceptance rate in the late 1970's and 1980's when anxiety regarding the HIV transmission of blood was at its height.

The contents of chest tube drainage are complex, not just whole blood and probably change dramatically over the first 24 hours. It is only in the most rapidly bleeding patients that the chest tube output has a close approximation to whole blood. The chest tubes, usually a minimum of two and possibly many more are place around the heart, in the mediastinum and often in one or both pleural cavities to drain fluid post operatively. As such these tubes will collect serous fluid, chylus fluid (if the thoracic duct has been damaged as well as blood, tissue fluids with inflammatory mediators, and fibrin break down products. Unless a patient is rapidly bleeding the drainage will have already undergone clotting and fibrinolysis. Therefor the fluid so collected will be filled with pro-inflammatory mediators as well as anticoagulant promoting proteases.

Transfusion in the ICU is highly prevalent. In the first McSPI study of transfusion behavior depending upon site 18-80% of patients were transfuse din the ICU after CABG. In 2006 the numbers had not really changed with 25-87% of patients receiving red cells in one of 65 sites studied. The use of FFP ranged from 3-95% of patients and 0-39% who received platelets in the ICU. So there is no standard for how to approach our patients after heart surgery and clearly a great deal of room for creative blood management.

Reports of high levels of fibrin split products I-6, I-8 TNF-alpha and CPK-M have all been found not only in chest tube drainage but in patients who received drainage unwashed as a method to conserve blood. In the 1980's and 90's a number of reports did show decreases in overall homologous blood transfusion when the chest tube drainage was re-infused after filtration. We know what the IL-6 and cytokine loads are in allogenic blood. They can be as high as 250 fold normal. So is the use of unwashed reinfusion chest tube drainage worse than the inflammatory load form banked blood? However in some of those series (often poorly controlled) there was more overall chest tube output in the group that received the re-infused drainage. Was it that drainage that increased the bleeding? Hard to say but if one conjectures that infusion of inflame med material and particularly fibrin split products might interfere with systemic coagulation function then it makes sense to wash the contents. Of the chest tube drainage.

One group had proposed the use of hard shell reservoirs form the CPB machine to collect chest tube drainage and then to use the cell saver system for washing. One study from the McSPI data base found that the use of shed mediastinal blood was associated with a higher rate of post-operative mediastinitis and infection, and they recommended not to use this fluid. However it was only in those patients who received only shed mediastinal blood and no other allogeneic blood that there was a small but significant difference in chest infection. Later work from Eastern Europe (Lithuania) used a prospective randomized model in which the first 4 hours of chest tube drainage was collected washed and re-infused. That group demonstrated no real differences in C-reactive protein (immediate), total chest tube output and overall leukocyte count. However the group that got the washed chest tube blood had a lower C - reactive protein level systemically at day five. And the total amount of transfusion was remarkably different 14.6% in those who received reinfusion v. 38.8% in non-re-infused patients. In the patients who received reinfusion of blood had a shorter (almost 50% decrease) length of hospital stay as compared to those that received allogeneic blood.

New auto transfusion devices using small Latham bowls have shown promise and utility in orthopedic surgery. In our University the application of this technology, combined with the use of lysine analogue plasminogen inhibitors and control for anemia pre-operative have dramatically reduced transfusion for total joints. In cardiac surgery similar devices have been developed by Haemonetics and work is underway to see how useful they may be.

In conclusion the use of post-operative scavenged chest tube drainage fluid has some controversy. The data seem equally distributed between supporting its usage and those detracting from usage. In some ways this is a fertile ground of research and advancement. Particularly the comparison of auto transfusion products to allogeneic blood and the inflammatory mediators. Health economics research could/should well be done in this arena as well.

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BLOOD FILTERS FOR TRANSFUSION OF SALVAGED RED CELLS: FALSE HOPE OR REDEMPTION

Darryl McMillan, *Perfusionist*
Royal North Shore and North Shore Private Hospitals

This presentation outlines the perceived benefits for using screen, microaggregate or leukocyte depletion filters when reinfusing intraoperative salvaged washed red cells.

Transfusion filters fit into three categories: screen filters, microaggregate depth filters and leukocyte depleting adsorption filters.

Screen filters have a pore size of 17–260 µm that traps the most clinically significant particles. Screen filters are commonly found in blood transfusion giving sets and are normally used with all blood components. The Australian Red Cross recommends using a standard blood-giving-set incorporating a screen filter (170–200 µm) that filters out large clots and aggregates, and ensures an effective transfusion flow rate. Screen filters may be satisfactory for the transfusion of washed salvaged blood from vascular and cardiac operative procedures.

Microaggregate depth filters have a micropore screen size of 20–40 µm; these filters are used for red blood cell transfusions as they trap degenerated platelets, WBCs and fibrin. The Australian Red Cross does not recommend the routine use of microaggregate filters, but they do acknowledge that clinicians may use them in massive rapid transfusions, despite reduced blood flow, or in hypotensive patients (1).

However, microaggregate depth filter may have a role in the re-transfusion of salvaged blood – particularly when used in orthopaedic surgery. Specific microaggregate filters for salvaged blood, such as the Lipiguard, are designed to filter out potentially harmful fat, leukocytes, anaphylatoxin C3a and microaggregates. Fat embolism syndrome is a phenomenon that may be associated with reinfusion of salvaged blood as fat particles may be present in the washed salvaged blood. It is unclear as to how much microaggregate is removed by these lipid reduction filters, but studies suggest the lipid removal rate is 40–80%.

The leukocyte depletion filter uses adhesion properties to remove 99–99.9% of WBCs and contaminates. It is recommended for use when re-transfusing salvage blood in maternity cases and cancer surgical patients. There is also evidence that the leucocyte depletion filter should be used routinely to remove fat particles, bacterial contamination and microaggregates in salvaged blood, but due to the low volume of blood transfused before a replacement filter is required and a slow blood flow rate, it unpopular for routine use.

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THE 'WORLD' WE WORK IN: WHO'S WHO IN BLOOD AND AVAILABLE RESOURCES

Dr Amanda Thomson
Transfusion Medicine Specialist, Blood Service

This session will provide a brief overview of the Australian blood sector and relevant national regulations, standards and guidelines together with a 'cooks tour' of available resources which can be used to support clinical practice.

BLEEDING IN THE PRESENCE OF SEPSIS AND LIVER FAILURE

Bruce D. Spiess, MD, FAHA
Virginia Commonwealth University Medical Center
Richmond, VA USA

Both of these coagulopathies are complex and multi-factorial. As the operating room team or critical care team caring for these patients there is no single magic drug or technique that will solve the coagulopathy. Bleeding can be the cause of death in both of these pathologies. Clearly the most effective way to treat them is to remove the underlying cause which for liver failure would seemingly be to perform a liver transplantation and for sepsis it is to correct the underlying cause of the sepsis. Both of them in their worst cases lead to diffuse intravascular coagulation which is not a single entity but a complex of consumptive processes leading to dramatic drops in platelet count, function and possible microvascular deposition of the platelets combined with consumption of serine proteases as well as fibrinogen.

Sepsis is defined as the presence of over whelming infection in the host such that systemic diffuse changes are encountered. This can be caused by a wide range of organisms including viral infection but is most often a number of bacterial infections. The days of rampant epidemic infectious diseases seemed over but today we are experiencing the insurgence of anti-biotic "super bugs" as well as the potential for worldwide pandemics of swine flu, influenza, Ebola, various pneumonias etc. Some common themes emerge as these agents overcome the resistance of an organism as a whole.

In its early stages sepsis is heralded as a pro-thrombotic disease with activation of endothelial cells and release of tissue factor. Endothelial cells have inducible tissue factor which is up regulated through NfKB and triggered by inflammatory cell activation (white cells and platelets) as well as by cytokines and particularly circulating lipopolysaccharides form gram negative bacterial cell membranes. Various histones and DNA all become activated with production of certain messengers for cellular dysfunction. Platelets are actually inflammatory cells capable of opsonizing bacteria and they participate in the early recognition phase of host invasion. As such when activated they respond with partial or total activation. As endothelium becomes more dysfunctional, loses its anti-thrombotic surface platelets begin to adhere to sites of endothelial invasion by bacteria/viruses. The endothelial cells release surface active compounds including thrombomodulin which triggers the reactive anti-thrombotic effect of protein and S. Anti-thrombin is consumed as the tissue factor creates thrombin on the surface of platelets and as fibrinogen is deposited at sites of microvascular hemorrhage and endothelial cell dysfunction. As endothelial cells diffusely become more and more dysfunctional the vasculature weeps, loses its integrity and loses its ability to control the micro-circulation leading to vasodilation and vasoconstriction. Capillary shut down but large vessel dilation leads to the characteristic hypotension and high cardiac output syndrome of sepsis. Really the coagulopathy of sepsis starts as a pro-thrombotic hypercoagulability that proceeds through consumption of the anti-coagulant or buffering proteins eventually to a massive consumption. By the time we as medical care teams are seeing the vasodilation and terminal events it may well be that the horse is quite literally out of the barn. Few therapies have been successful in turning back the coagulopathy of sepsis. Protein C/S drugs were tried and some small trials with AT-III have shown some success. We are involved in a large multi-centered study using high dose vitamin C which is a profound anti-oxidant. It seems that large upstream and early multi-pronged therapy with repletion of the buffering compounds and anti-oxidants along with perhaps PCCS could be effective. But research has always been focused on single intervention therapy- a research method doomed to failure. The goal with sepsis treatment is not only to remove the causative event but to decrease the number of patients that proceed from the early attack on endothelial cells to the multi-system organ failure that creates indulennt and slow death in our ICUs. We have been successful in creating equipment to keep people alive for days as their organs shut down but the challenge in sepsis is early recognition and perhaps creative multi-pronged early repletion of buffering compounds. What are those and how we can monitor for which ones are preferentially being depleted has yet to be fully appreciated.

TEG has been utilized in sepsis and a number of studies support that it can be an early warning system for impending sepsis. In

a study of 55 patients with SIRS compared to those without there was a discernable tendency to hypercoagulability shortened R time and changes in K and alpha angle. In late sepsis with consumptive coagulopathy the prolongation of R and the decrease in the MA as fibrinogen and platelets are consumed can be extremely dramatic. Eventually primary/secondary fibrinolysis prevails, feeds into the coagulopathy and leads to bleeding. It seems that clinicians play a reactive role by supply pro-coagulant precursors feeding the engine that has run away. Platelet transfusions cryo and FFP all do exactly that. One study using mild to moderate hypothermia showed a decrease in the consumptive coagulopathy. That makes some sense as the fevers of sepsis denature proteins and activate a number of serine proteases.

Liver failure is again complex and in its end strategies appears very much like sepsis. The liver is both a protein producing organ responsible for the production of most of the serine proteases as well as a detoxifying organ removing cytokines, and protein degradation products. Perhaps the worst coagulopathy encountered by operating room teams is during the an-hepatic phase of liver transplantation when there is no detoxifying, fibrinolysis is severe and D-dimers are not cleared. Platelets are severely effected by the presence of the fibrin split products and frankly nothing coagulates. In liver failure not only are the serine proteases not produced but neither are the buffering anti-thrombin, heparin cofactors , protein C and S etc. End stage liver failure causes splenic congestion and a shortening of the ife span of platelets. Platelet number is radically decreased often. Hepato-renal syndrome leading to renal failure may well add to the platelet dysfunction as clearance of urea and ammonia products can at least partially poison platelets. Until the patient s frankly uremic the effect of renal dysfunction is much like early sepsis in that the patients tend to be hyerpcoagulable.

The TEG and RoTEM have been invaluable tools in caring for patients during liver transplantation and in end stage hepatic failure. This lecture will demonstrate some of those methods to follow not only the course of dysfunction but the effect of therapy.

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PRE-EXISTING MEDICATIONS THAT ALTER PLATELET FUNCTION

Dr Bruce Cartwright

Cardiac Surgery continues to consume up to 20% of the blood product supply of most countries. Some improvements have occurred with the introduction of goal directed management of haemostasis by point of care testing based algorithms and through the targeted use of factor concentrates; however, this has been offset by the increasing age and co-morbidity of cardiac surgical patients together with the introduction of more aggressive antiplatelet regimes beyond the previous standard dual therapy of aspirin and clopidogrel.

This talk will explore:

1. The complimentary nature of platelet function testing performed point of care, in conjunction with viscoelastic tests of whole blood for assessing haemostasis.
2. The role of perioperative platelet function testing in cardiac surgery with a focus on balancing the need for a reduction in thrombotic events against the desire to avoid haemorrhagic complications of modern antiplatelet regimes, with an emphasis on the research that has started providing the information to stratify bleeding and thrombotic risk.
3. Future directions in antiplatelet therapy such as reversible and non competitive P2 drugs and the anti-PAR drugs in the pipeline.

PRACTICAL ASPECTS OF OPTIMISING PERIOPERATIVE PATIENT BLOOD MANAGEMENT

Ross Kerridge
Director, Perioperative Service, John Hunter Hospital, Newcastle
Conjoint Associate Professor, School of Medicine & Public Health, University of Newcastle

In common with many areas of clinical medicine, the challenge of translating the evidence about perioperative blood transfusion into actual change of clinical practice is just as important as defining the evidence. Blood transfusion is particularly complex due to multiple factors. Some current areas of research and new technology may have widespread implications. It is an area of multidisciplinary involvement. Practice variation and resource implications can be readily defined and audited. Finally, it is an emotional issue of interest to the general public, and thus a political minefield.

The excellent resources generated by the National Blood Authority in Australia are probably the best source of evidence in the world on Patient Blood Management. The recent European Guidelines for Management of Severe Perioperative Bleeding also provide a very useful and comprehensive overview of current evidence on this issue. They have been editorialised as "Everything we wanted to know about perioperative bleeding. Although the guidelines are very comprehensive, they are not "Everything".

Apart from scientific evidence, and perhaps more importantly, there must be effective clinical leadership in the practical challenges of translating the evidence into clinical practice. Knowledge and understanding of how to do this is part of "everything" we need to know about perioperative bleeding and blood transfusion.

What are some other aspects and practicalities of Patient Blood Management that perioperative clinicians need to 'want to know'?

"Back To Basics" There remains avoidable hazard to patients associated with poor transfusion practices in the 'basics' of knowledge of blood transfusion safety, clinical decision-making, blood sampling, and blood unit administration. 'Wrong Blood in Tube' incidents are a particular concern. (Double independent sampling is one strategy to engineer a solution). Clinical leadership by senior staff is a crucial component of preventing and changing these behaviours.

Transfusion Triggers Evidence now suggests that transfusion above a threshold as 'low' as Hb 70g/L is both lacking in benefit and is a cause of adverse outcomes (i.e. not just an association). 'Restrictive' transfusion is not just safe: - 'liberal' transfusion is dangerous. Nevertheless, this evidence is still not completely accepted by bedside clinicians, leading to ongoing practice variability. Perioperative clinicians need to know the evidence and advocate it to their colleagues.

Preoperative Anaemia Optimisation The potential benefits of improved preoperative optimisation have been well described, but implementation is restricted by mixed messages regarding intravenous iron therapies, logistics and practicalities. The development of better preoperative clinics provides a platform to translate this evidence into practice.

Patient Education & Counselling Perioperative clinicians must be able to address patient concerns about transfusion. Thus, they should know how to ensure patients are appropriately informed, addressing fears, and explaining the appropriate role of autologous or directed donation.

Cell Salvage The safety of Intraoperative Cell Salvage is now well established in most clinical settings (including obstetrics, wound soiling and malignancy), if the local practical consideration of logistics and training can be addressed.

Blood Collection and Reinfusion Use of systems for collection and reinfusion of spilled blood is supported by the evidence base, particularly for knee arthroplasty. Again, clinical practice varies from the evidence, perhaps due to 'aesthetic', rather than 'anaesthetic' considerations.

Waste Avoidance Clinical experience in transfusion-refusers emphasises the need to avoid blood wastage by careful and thoughtful clinical practice. Committed clinical leadership models these techniques to colleagues, nurses, and 'junior' medical staff.

Point of Care Testing & Thromboelstography The appropriate application of thromboelastography is controversial; opinions have traditionally differed between anaesthetists and haematologists. Recent technological developments may be the 'game-changer' in this issue. Whether lab-based or Point-of-Care, the results must be networked into the hospital information system to facilitate haematologists and operating theatre or ICU personnel viewing the display and communicating together in real time. This in itself may achieve the greatest therapeutic benefit

Massive Haemorrhage In Trauma and elsewhere, appreciation of the importance of techniques to minimise blood loss, and restore normal coagulation in massive haemorrhage, is necessary. Tranexamic Acid may be inappropriately being seen as a 'magic bullet' rather than as part of the suite of strategies for massive bleeding. Massive Transfusion Protocols should be used to optimise teamwork, not to facilitate access to blood resources.

'New' Blood Products Recent military experience has increased interest in 'frozen' blood products (especially platelets), particularly in remote situations. It is not clear if they will be clinically appropriate for more widespread civilian use.

The Age of Blood issue The recent European guidelines support use of blood stored up to 42 days, despite evidence of concern about storage lesions. Studies such as TRANSFUSE (Australia/New Zealand) may clarify this issue. If 'young' blood is shown to be much safer than 'old' blood, will the transfusion threshold issue need to be re-examined? Who should get the youngest blood? These results could have enormous ramifications on blood transfusion management, and become a major public policy issue.

Electronic Issue & Remote Release Most blood services in Australia now use 'electronic issue' or "computerised cross-

matching" (which, by the way, was first developed in Newcastle, Australia) There remains widespread misunderstanding of how this system works, limiting the full benefits of this system in minimising blood wastage.

Data Systems The development of linked clinical and blood service data systems can be used to identify areas for improving patient blood management. Recent work by the Western Australian Department of Health has demonstrated the potential for developing a 'whole of system' approach. Notably, this has reflected strong clinical leadership.

As in many areas of medicine, knowing the evidence is important, but it is not everything. Translating evidence into practice also requires teamwork between transfusing clinicians, haematologists, laboratory scientists, administrators and patients, and appropriate senior clinical leadership to change practice.

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THE RED CELL TRANSFUSION WAS MEANT TO IMPROVE OXYGEN TRANSPORT AND DELIVERY: TRALI AND TACO

Prof James Isbister

Consultant in Haematology and Transfusion Medicine
Clinical Professor of Medicine, Sydney Medical School
Royal North Shore Hospital of Sydney

TRALI and TACO may be rare complications, but they are very common for the patient who has it!

Transfusion related acute lung injury (TRALI) and Transfusion Associated Cardiovascular overload (TACO) are now receiving a great deal of attention as a potentially serious and potential fatal complications of blood transfusion and more common than originally thought. There is now greater awareness of the numerous possible adverse outcomes from allogeneic blood transfusion, probably greater than any other specific medical therapy. From early in this author's career allogeneic blood transfusion has always been a therapy fraught with balancing risks against assumed benefits. Of even greater concern, the author has had to observe the complacency of colleagues practicing blood transfusion in an evidence free zone in which they assumed transfusion is always safe and effective. Even if there is doubt concerning potential benefit many clinicians make a "default" and "culturally imbedded" decision to administer a transfusion. Thankfully, this mythological mindset is increasing becoming something of the past, although there are still many of the "old guard" who insist they know best. Hopefully, if they cannot be convinced, age will weary them and they will ride off into the sunset and no longer corrupt the minds of their students and mentees.

There are three condiciones sine quibus non (sine qua non), often forgotten or ignored with intravenous fluid therapy:-

1. Clinicians take on a critical clinical responsibility when an intravenous cannula is inserted for the purpose of infusing any form of therapy, especial blood components.
2. The first "port of call" for any substance infused intravenously is the lung.
3. Patients with cardiac, respiratory or renal failure or compromised reserves are at risk of pulmonary oedema from the infusion of any fluid, especially blood components.

Perhaps it is an over-emphasis, but it is sobering to consider the above in a medical legal context. Lawyers would refer to the "but-for" or "causa sine qua non" as a circumstance in

which a blood transfusion is the material cause of an adverse clinical outcome, in this context pulmonary oedema, without which the complication would not have occurred.

"But for" causation is in contrast to probable causation, in which pulmonary oedema may be, but not necessarily, causally linked to blood transfusion. In haemovigilance programs, such as SHOT in the UK, TRALI and TACO would be classified in the "but for" the blood transfusion the pulmonary oedema would not have occurred. Transfusion associated dyspnoea (TAD) suggests a high probability of the transfusion being responsible or a contributing for the respiratory failure.

TRALI

In the classic plasma neutrophil antibody-mediated form of the disease symptoms usually arise within hours of a blood transfusion. In contrast to most patients with other forms of acute lung injury, recovery usually occurs within 48 hours. The underlying pathophysiology of 'classic' TRALI is due to the presence of leukoagglutinins in donor plasma. When complement is activated, C5a promotes neutrophil aggregation and sequestration in the microcirculation of the lung, causing endothelial damage leading to an interstitial oedema and acute respiratory failure. This classic form of TRALI has been recognised for over five decades, but was thought to be a rare complication of allogeneic plasma transfusion. As leukoagglutinins occur typically in plasma from multiparous females there has been a move to using male donor only clinical fresh frozen plasma and relegating female plasma to fractionation into specific plasma products.

It is now accepted that there has been under-recognition and under-diagnosis of TRALI, partly due to a lack of clinical awareness, but also to a broader understanding of mechanisms by which blood transfusion may cause or be a contributory factor to lung injury. The term TRALI is now being expanded to include cases in which transfusion is identified as an independent risk factor predisposing patients to lung injury. There is recent experimental evidence supporting a "two hit" hypothesis in which patients may be "primed" by shock, sepsis or extracorporeal circulation making them more susceptible to acute lung injury from blood transfusion.

TACO

From the sine qua non comments above it should be clear why this author has always had difficulty understanding why circulatory overload from blood transfusion, especially red cell concentrates, is not acknowledged, diagnosed, treated appropriately and, more importantly, not prevented. There are many reports in the literature back in the 1930's and 1940's referring to what is clearly TACO. Ironically, a definitive article of the subject of transfusion fatalities from circulatory overload was published when the current author was 2 weeks old! Despite the over 60 years until haemovigilance programs started recording TACO as a serious hazard of blood transfusion little has been added to the clinical messages in a landmark article by Drummond.

"Due regard having been paid to deaths consequent upon agglutination and haemolysis either of donor's or of recipient's cells, there still remains in my opinion a more frequent cause of reactions and fatalities. I refer to overloading of the circulation in the chronically anaemic and diseased subject. In the Welsh Board of Health Regional Transfusion Service there is a mutual arrangement whereby all transfusions given in hospitals are notified on a standard record form. With the aid of these records it has been possible to track down and investigate some interesting transfusion reactions. It is noteworthy that in 2,300 transfusions notified in the last 2- years there has not been a single instance of death due to transfusion of cells of an incompatible blood group. There have been some cases of death from pulmonary oedema and cardiac failure due to circulatory overloading in chronically anaemic subjects."

Drummond, R. BMJ 11 September 1943

Review of cases of TACO in haemovigilance programs usually demonstrate pre-existing risk factors, poor clinical decision making, suboptimal transfusion practices and poor clinical awareness of TACO and its management and prevention.

The management of TACO once diagnosed, and the transfusion ceased, is standard of care for hydrostatic pulmonary oedema.

Regarding prevention, one only has to go back to Drummond's article of 1943 in which he summarises the causes of TACO under the following headings:

- Overloading after rapid IV Injections
- Risks in transfusing anaemia
- Too rapid transfusion in anaemia
- Cases of too large a transfusion

Each the above is considered in the context of the state of a patient's myocardial function, to which this author would add the state of the lung and pulmonary capillary endothelium.

Also of interest is Drummond quoting the famous British haematologist, Whitby who was one of the chief medical advisors to the military during WWII. "The reason why no cardiac embarrassment occurs in service casualties is, of course simple. First, the introduced fluid replaces lost fluid. Secondly, the myocardium is invariably normal and therefore functions efficiently. It is well known in clinical medicine that it is impossible to strain a normal heart." The "impossible" may be overdoing it a bit!

TRALI/TACO

In many cases of transfusion related pulmonary oedema there is an overlap between TRALI and TACO. This usually occurs in patients with co-morbidities that may predispose to pulmonary capillary leak and pulmonary oedema occurring at lower pulmonary pressures than would otherwise be the case.

TAD

Transfusion associated dyspnoea is characterised (ISBT definition) by respiratory distress within 24 hours of transfusion that does not meet the criteria of TRALI, TACO, or allergic reaction. Respiratory distress should not be explained by the patient's underlying condition. Not much more can be concluded about this possible hazard of blood transfusion until there is a better understanding and perhaps an as yet unrecognized hazard is yet to be defined.

COAGULATION CHANGES DURING AND AFTER CARDIOPULMONARY BYPASS (CPB): VIEWS FROM THE UNITED STATES

Bruce D. Spiess, MD, FAHA
Professor of Anesthesiology, emergency Medicine and Biology
Director VCURES Shock Institute
VCU Medical Center
Richmond, Virginia

Introduction: Coagulation function is a complex interactive, controlled, energy dependent and dynamic function of the blood and endothelium interacting. It is so complex that this author firmly believes it cannot be understood without bio-computational modeling. The change in blood from a freely flowing slurry fluid of cells, proteins, lipids and many other compounds to a tightly adherent cross-linked clot is of great importance for the individual organ/organism survival. Coagulation should be thought of us a sub-set of the larger inflammation healing process. Sometimes those of us who prefer to categorize processes have broken the act of clot production into protein coagulation and platelet thrombosis sets of reactions. This type of thinking is outdated yet still taught and leading to various misconceptions. Today a cell based mode of coagulation is embraced, wherein the biochemical serine protease cascade waterfall events of the various pathways happen on and are controlled by ligands expressed on the surface of various blood cell lines. Coagulation occurs by initiation, acceleration control and eventual re-canalization.

Of paramount importance is the endothelial cell (EC). It has been estimated that the ECs make up the largest organ of the body. The endothelium paves the walls of the vasculature, controls its surrounding tissues blood flow and creates both a barrier as well as a reactively permeable barrier for O2, CO2 other gases. The EC excretes NO. White cell movement, water, solutes, proteins etc. are highly regulated by each individual population of ECs. The plasma facing surface of the endothelium is lined by glycocalyxx (GC). GC is made up of proteo-glycan that possesses a surface charge that repels serine proteases. Imbedded/attached to it surface is heparan-glycosaminoglycan. Heparan binds circulating anti-thrombin, activates it and further creates an anti-thrombotic/anti-inflammatory surface. When the endothelium is disturbed it rapidly and reversibly sheds its glycocalyxx exposing either the naked EC membrane or basement membrane collagen expressing and attaching von Willebrand's factor. Furthermore if disturbed the EC shifts from production of anti-coagulant exported compounds, to an NfκB mediated production of pro-thrombotic signaling proteins including the exportation of tissue factor. Tissue factor (TF) is the potent activator of the extrinsic cascade. A common misconception is that TF is only present from extra-vascular tissue destruction. Indeed the EC itself contributes a great deal of TF and does so under the assault of CPB. Ischemia-reperfusion injury diffuse micro-embolism, oxidative stress, cytokines, compliment (C5-9), white cell degranulation, platelet adhesion and a vast number of other insults all can trigger the EC to shift form anti-coagulant (resting/normal) to a pro-inflammatory thrombotic phase.

Platelets have been the center of considerable study. Platelet EC interactions lead to chronic inflammation which in the end produces atherosclerosis. Each of us is embodied with a different genetic predilection for platelet "stickiness" as well as leukocyte-platelet-protein interactions. Studies of the genetic basis for coagulation are fascinating. Recent work has uncovered some alleles/mutations which are associated with higher risks for atherosclerosis, stroke, MI and early death. So also have some alleles been investigated and shown to be associated with increased risk of bleeding after CPB. What is very interesting is that the unfortunate souls amongst us who have multiple of these alleles have an additive/multiplicative propensity for more thrombosis or bleeding. It should be realized that although focus has been upon platelet biology all cell lines in the blood participate in both coagulation and inflammation. Erythrocytes have fibrinogen, GPIIb/IIIa ligands (as do white cells) and interestingly the red cells have to pre-process lipids handing them to platelets before platelets can make thromboxane. Therefore it makes little sense conceptually for us to analyze blood coagulation in isolation of either proteins or cells. That is especially true if we wish to assess risk for bleeding/thrombosis in the cardiac patient.

CPB Effects: The use of CPB creates a systemic insult that although practiced differently by each center performing the procedure has more similarities than variations. Individual patients can respond in a continuum from thrombosis- to little reaction- or all the way to massive bleeding. As mentioned above, there are a large number of genetic variations affecting the population. If one considers that there are over 2500 proteins circulating in the blood and at least several hundred known surface ligand proteins on platelets then it is not unexpected that variability is indeed normal. Each protein probably has at least one or more survivable polymorphism. Studies have looked at the genetic up/down regulation of genes in response to CPB. There exists probably 500-1000 gene known reactions to CPB, each having a feedback loop into other reactions. Therefore it is at present incomprehensible for us to predict how an individual will react to the CPB insult. These are patient characteristics for which we really have little control. Chronic disease affects both inflammation and coagulation profoundly.

CPB carries with it certain known insults: hemodilution, hypothermia, anti-coagulation, acidosis, surface activation, fibrinolysis, transfusion, tissue destruction, and pharmaceutical pre-intra-and post operative interventions.

Hemodilution: We can to some extent limit this. However there are limits of CBP circuit priming, oxygenator size and simple practicality. For example the circuit must stretch up from the CPB machine to the field and have enough tubing length so as not to pose, in itself, a threat to patient safety. Generally prime can be cut to 600-800 cc's by decreasing tubing diameter and by employing a techniques known as retrograde autologous priming of the circuit. In the end a small bodied woman (40kg) will undergo tremendously more hemodilution than a 130Kg male. A team response to these risks is extremely important but so it the pre-operative sensitivity to circulating volume and red cell mass. Red cell mass may seem not to be of great importance to coagulation but hematocrit rheology does influence the margination of platelets with anemic patients having a propensity to bleed. The pre-operative correction of anemia has been suggested to be one of the most important risk factors for intervention that can influence outcome.

Hypothermia: There has been a strong move towards doing warm/tepid CPB. Serine proteases dysfunction at temperatures below 34 degrees with clot being unable to be formed at temperatures below 30C. The wound edge of most CPB cases in the mediastinum is 24-27C so how can one expect normal coagulation. It is often that closing the chest alone will reestablish normal clot function. Deep hypothermia of course causes tremendous coagulopathy but the re-warming phase should re-normalize function. However platelets do marginate into the reticuloendothelial system with severe hypothermia and it may well take hours for the circulating platelet number to return to normal. The tendency for surgical care is to speed re-warming and to wean from CPB when the "core" temperature is above 35 C. It is very possible for many patients to simply not be re-warmed adequately with this philosophy. Platelets dysfunction, in the same way that serine proteases dysfunction below 34-35C. Once again how we practice simply does not make sense. On the other extreme temperatures above 38-39C are profound platelet and protein activators leading to denaturation of proteins above 40C. That can cause both severe hyper-coagulability- dysregulation and eventual diffuse intravascular coagulation. Aggressive re-warming with CPB temperatures above 37 c should be avoided.

Acidosis is not tolerated by either platelets or serine proteases. These natural functions are meant to work in an incredibly narrow range of pH. Levels of pH below 7.2 will yield an ever worsening coagulopathy. How often in the bleeding patient do we recognize and treat the acidosis as a cause of bleeding?

Anti-coagulation is a complex subject that has been debated by anesthesiologists, perfusionists and surgeons for years. To date we do not know the "best" dose of either heparin or protamine for any individual patient or population. There are centers performing acceptable CPB with dosages of heparin only 30% of that of others with a liberal philosophy. One thing is rather clear that protamine is far more effective at reversing heparin than historically we have believed. Perhaps most centers are using twice to 3 times the necessary protamine to reverse heparin.

Heparin has been the tried and true anticoagulant since the inception of CPB almost 60 years ago. That being said heparin is not ideal. Heparin is normally held in mast cells of the lung and gut, not in the vasculature and it although similar is different than heparan of the glycocalyxx. One has to wonder why it exists only in these tissues where the body has intimate contact with the "outside "world. Heparin when released form mast cells functions to open the interstitial spaces, and promotes the ability for leukocytes and macrophages to move through infected tissues. Therefore heparin's normal biologic function is to promote inflammation. Indeed to date we know of over 1000 cell signaling and regulatory effects of heparin. Therefore for us to focus upon it as a pharmaceutical anticoagulant alone is rather myopic. Heparin carries a poly-anionic charge of great density. Hageman factor, bradykinin and endothelial cells are reactively primed to have a profound inflammatory reaction to highly negatively charged species. Indeed the heparin contaminant- over-sulfated chondroiton sulfate (OSCS) that was in Baxter heparin between November 2007 and February 2008 killed many people and caused massive catastrophic vasoplegia hypotension as well as an epidemic of heparin induced thrombocytopenia (HITT). Heparin does not cause this routinely simply because AT is present in the circulation rapidly and irreversibly binding the heparin. AT is needed for heparin to exert its anticoagulant effect. But, heparin/AT complexes have effects upon compliment, bradykinin, serine proteases as well as thrombin. Unfortunately the AT/heparin complex can bind thrombin only in its free form. Free thrombin accounts for approximately 1% of thrombin production. Thrombin is produced by serine proteases on the surface of leukocytes and platelets where it is bound to various binding sites- GPIa and GPIIb/IIIa. Furthermore thrombin is bound to fibrinogen/fibrin where heparin/AT complexes cannot down regulate its function. CPB drops the circulating levels of AT by hemodilution and through consumption. The use of heparin in the pre-operative period can make this risk even higher. Levels of At during CPB and afterwards have been shown to be of key importance to bleeding and thrombosis (coagulation dysregulation). Few if any centers either routinely supply exogenous AT nor do most centers monitor its levels yet it is key to both the functioning of our standard anticoagulant. The use of non-heparin anticoagulants has been investigated and of interest the use of bivalirudin in some studies has shown less production of inflammation as well as less thrombosis. Bleeding has been a bit more but that is probably due to the fact that we have no antidote for this anticoagulant. It remains extremely useful in cardiology and some have adopted it for off pump CABG suggesting that it has a great deal to offer.

Heparin causes platelets to express a number of their ligand binding sites. Those binding sites in turn then are susceptible to shear forces as well as inflammatory mediators that render then inactive. When platelets are partially activated they expend energy and some go on to degranulate meaning they can no longer take part in a normal coagulation process. Therefore heparin sets up platelets to be dysfunctional after CPB. In the centers that have used heparin bound circuits it is only by decreasing their systemic use of heparin that they have decreased bleeding and inflammation. That should tell us something about heparin.

Heparin leads to full expression of platelet factor 4 (PF4) which is the natural way our bodies remove the heparan that gets loosened form EC. PF4 heparin complexes are highly antigenic (the body recognizes them as abnormal) and immediately begins production of antibodies to remove them. Patients on heparin for any length of time will create these antibodies. Over 50% of patients post CPB has circulating antibodies. Those antibodies when binding to intact platelets are the most pro-thrombotic stimulus known to medicine. They lead to HiTT which is a major problem and is under recognized. It is not uncommon to have patients on day 3-10 after surgery develop a MI, stroke or renal failure and the presence of these antibodies has been associated with an increased risk for such complications. So also has the presence of these antibodies been associated with increased bleeding. It is all a continuum.

Fibrinolysis is a near universal event for CPB. Again heparin has some causative relationship in that heparin stimulates the release of urokinase. TPA however is stimulated from endothelial cells by their exposure to thrombin. Remember thrombin is produced and not always bound by AT therefore the signaling properties of thrombin go on leading to release of thrombomodulin and TPA. Our response as anesthesiologists, perfusionists and surgeons has been to embrace the widespread use of some anti-fibrinolytics. The debate about what type of anti-fibrinolytic, the dose and the method of administration rages on. Further it simply seems to be irrational that either one dose or one medication should be given to all comers. Aprotinin, a bovine protein was used for about 15 years with remarkable success. I remain convinced that the drug itself is not either pro-thrombotic or dangerous. However the usage in all comers by us as physicians probably created some high risk events. Giving this drug to patients who were hypercoagulable, or those with HiTT antibodies almost certainly contributed to some, albeit rare, catastrophic thromboses. Let me state categorically that aprotinin does not cause renal failure and that it is not associated with increased mortality. Both of the studies that have led to its withdrawal from the market have been either disproven by weight of other studies or been examined and deemed to be improper conclusions (The BART Study). I do believe that a little knowledge of coagulation is very dangerous. Unfortunately that is what most have who are practicing CPB. It was the inappropriate use and lack of monitoring for hypercoagulability that led to adverse events with aprotinin.

Transfusion carries a wide range of pro-inflammatory mediators as well as bio-active lipids and cell particles. This subject deserves an

entire lecture by itself. But, there is great data showing that those patients receiving more red cell transfusions actually bleed more than those matched and not receiving transfusions. For example those matched patients (risk, age, sex, medication etc.) bleed more than those patients allowed to have routine transfusion practice. Once again what we do makes little sense. The use of FFP and platelet transfusions often are associated with worse bleeding rather than less. Judicious use and only in proven (TEG /RoTEM guided) coagulopathies makes sense. It has to be stated that historically 40-90% of patients received red cell transfusion during surgery. It is clear that when you do that you actually make the risk of adverse outcomes worse than by not transfusions. Anemia is a risk factor for adverse outcomes but the use of transfusion, rather than making it better makes it worse! If the majority of patients receive a transfusion during CPB is it the CPB or the transfusion that leads to bleeding and thrombosis?

Medications all have some effect upon coagulation. Almost everything we give has an effect upon platelets and neutrophils. For example cephalosporin antibiotics are profound anti-platelet agents (for a short term). They have been widely implicated in some cases of TTP, yet we give every surgery patient either these or another perhaps more obnoxious aminoglycoside antibiotic. There is tremendous focus upon clopidogrel, aspirin, dabigatran, and new anti-Xa agents. These agents all contribute to platelet dysfunction and definitely are related to increase bleeding. We have the technology for diagnosing how much effect these agents have in any individual's blood stream yet less than 15% of hospitals utilize the technology.

A rational approach to treating coagulopathies involves the use of presently available tests. The effectiveness of these tests depends upon the teamwork approach discussed previously with respect to patient blood management. If the team meets and can agree upon a rational algorithm for transfusion or use of pro-coagulant therapies then they will make more targeted and appropriate use of expensive drugs and biologics. This lecture will discuss the use of some of the agents and how so many of them are misused. For example FFP is widely utilized and has little effect upon either treatment of coagulopathy ad is probably useless in prophylactic prevention of bleeding. In our center we use the platelet count, fibrinogen concentration prothrombin time (INR)- far less useful- and the TEG. Other use the RoTEM. The lecture will present some of the more recent work on both systems, how one can use RoTEM to substitute for PT and aPTT and how the TEG can be used for platelet mapping. One of the most recent algorithms from Toronto, Canada has proven to be quite effective using the RoTEM. Another one using platelet mapping has proven highly effective and form Denver Colorado it has been shown that platelet mapping is useful not just for assessing the effects of P2Y12 drugs and aspirin but for the effects of CPB upon glycoprotein surface sensitivities. There are today over 2000 published papers on RoTEM and TEG yet in the United States far less than 50% of cardiac centers use one or the other. Laboratory medicine departments run the gamut from being supportive to being actively hostile to such monitoring. Next generation viscoelastic tests are being developed and a new TEG automated machine should be released in the United States within several months. Other tests being used in Europe with some success include the Mulitplate automated aggregometry for platelet dysfunction. The success of these tests is solely dependent upon the human systems put in place to run them and the speed with which blood samples can be delivered to the testing machine and how electronically networked the data is to the cardiac team. Use of the data in a bleeding patient can quickly discern between a surgical bleed and a coagulopathic patient. That yes, no decision tree has been utilized to decrease take backs to the operating rooms. Data do exist in some studies wherein the TEG and RoTEM have been shown to not correlate with chest tube output. These studies unfortunately do not understand the function of the machines nor the way that the coagulation data is presented. Chest tube output is probably not a linear relationship to severity of coagulopathy and certainly TEG data are not linear. The MA of the TEG is a 0-100 scale graphic representation of a clot force measurement that goes from 0 to infinity. The 0-100 measurement is not even logarithmic so trying to expect the data from TEG to correlate to chest tube output is mathematically impossible.

Summary: The coagulopathy of CPB is highly complex. When confronted with a bleeding patient post CPB anxiety and panic often drive the physician's responses. Truly only computer bio-computational modeling can understand coagulation/ inflammation. When the systems become dys-regulated it is very difficult to re-establish normalcy. It is for certain that emptying the blood bank of available products will not bring normality back to a runaway system. It makes sense that institutions should embrace education and coordinated approaches using multi-disciplinary team efforts. Embracing expanded and cutting edge coagulation management along with algorithm derived treatment modalities has and will be far more successful in creating improved outcome than will "clinical judgment".

Recommended Readings

1. Johansson PI, Solbeck S, Genet G, Stensballe J, Ostrowski SR. Coagulopathy and hemostatic monitoring in cardiac surgery: An update. Scand Cardiovas J 2012; 46: 194-202.
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COAGULOPATHY MANAGEMENT IN SPINE SURGERY

Dr Chris Sparks
VMO Royal North Shore Hospital and North Shore Private Hospital. (3,000 spines in 22 years)

Coagulopathy: generalized bleeding that cannot be controlled with Sutures or diathermy. Usually INR >1.5 and Platelets> 70,000 (to exclude simple thrombocytopaenia.)

What is different about Spine surgery?

Patient is prone. Very hard to do normal anaesthetic procedures after turning.
Very hard to stop operation.
Surgery might take 12 hours.
Big incision.
Need to strip periosteum to facilitate bone graft fusion.

Causes:
- Pre op medications.
- Intra Op Drugs
- Underlying illness e.g tumour infection
- Blood loss èDilution.
- Hypothermia Acidosis Hypotension
- Transfusion of Infected Blood.

Management:
- Stop or reverse causes
- Assess Coagulation defect. (Point of Care testing)
- Blood Products
- Good Surgeon
- Consider Stopping surgery

Blood Products: USA has been P Cells:FFP: Platelets 1:1:1
Europe Now P Cells: Cryo: Prothrombinex.

Activated Factor VII. Still controversial and expensive.
I have used it three times in 3,000 operations.

Reading List:

The Exclusive use of coagulation factor concentrates enables reversal of coagulopathy and decreases transfusion rates in patients with major blunt surgery.

Petra Innerhofer et al

Injury Int.J. Care Injured 44 (2013) 209-216

ESA guidelines: management of severe bleeding.

Eur J Anaesthesiology 2013; 30:270-282

Transfusion Related Acute Lung Injury: Current Concepts for the Clinician. Darrell J Triulzi

Anesth Analgesia Vol 108 No.3 March 2009 pp770-776

Coagulaopathy in Spinal Surgery. Andrew Milby et al

Essentials of Neurosurgical Anesthesia and Critical Care. Chapter 31. Publisher Springer.

Bleeding and Coagulopathies in Critical Care. Beverley Hunt.

NEJM 2014 V 370; 847-859

Patient Blood Management. Ronald D Miller.

Millers Anesthesia 8th Edition 2015. Chapter 61 pp1830-1867

The next IMOB Conference will be held in 2017

POINT OF CARE

Abbott

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In the complex, dynamic environment of the CVOR, there's no room for doubt—in your equipment or your team

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Reference:
1. Ojito JW et al. J Extra Corpor Technol 2012;44:15-20

CVOR: cardiovascular operating room

* Assumes eight arterial blood gas (ABG) tests being run in a four-hour operation and travel time to and from the blood analyser outside of the CVOR. Excludes delays due to human factors, or any equipment delays

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PDU COMMS

ISSUE 01 Nov 2015

ASSOCIATIONS INCORPORATION REFORM ACT 2012
Section 8

No. A0093172S

CERTIFICATE
OF INCORPORATION

This is to certify that
PERFUSION DOWNUNDER INCORPORATED

is on and from the 1 October 2015
incorporated under the Associations Incorporation Reform Act 2012

Given under my hand at MELBOURNE, this 1 October 2015

Deputy Registrar of Incorporated Association

VICTORIA

Victoria

Perfusion Downunder Incorporates

Perfusion Downunder has from 2005 been registered as Perfusion Downunder Pty Ltd., a company set up on behalf of PDU by Cellplex Pty Ltd. A number of coinciding circumstances has led to the restructure of PDU into an Association incorporated under the Victoria Associations Incorporation Act 2012 that was effective from 1 October 2015.

Incorporation of PDU importantly provides the vehicle for compliance with the Medical Technology Industry Code of Practice providing the appropriate structure for PDU Inc. in relation to the corporate sector.

The PDU Inc. management Committee is now responsible for the financial management of PDU and all other responsibilities of an Incorporated Association. The PDU Winter Meeting will continue with the 2016 Meeting at the Heritage in Queenstown on track for August 18—21. Berni Tackney has relocated to New Zealand and has been contracted by PDU Inc. as the conference coordinator.

this issue

PDU Inc. Committee

The PDU Inc. executive Committee.

President
Timothy Willcox FANZCP CCP

Vice President
Assoc. Prof Robert A Baker PhD CCP

Secretary /Treasurer
Michael McDonald FANZCP CCP

PDU Inc. Voting Members

Prof Alan Merry. MBChB FANZCA FFPMANZCA ONZM

Richard Newland BSc FANZCP CCP

Conference Coordinator

Bernardette Tackney

WAYNE PEARSON

19 Sept 1963—4 Sept 2015

Wayne Michael Pearson was a pivotal force in the success of Perfusion Downunder. He was an enabler. From the outset in 2004 when he came to me with Jill Futter-Crigan offering to sponsor a perfusion focused meeting through to the 11th meeting this year shortly before his untimely death Wayne has provided leadership *par excellence* for PDU. His gracious and generous support though Cellplex has cemented a legacy of regional perfusion innovation, collaboration and advancement. Throughout the last 12 years of PDU Wayne never sought the limelight, in fact to the contrary was quite retiring when it came to acknowledgements of his contribution. In 2014 Wayne delivered the Prof Merry Lecture and spoke of leadership in a talk entitled The end of the Beginning. He said *"A leader is defined as an individual who motivates and influences others towards the accomplishment of a pre-defined goal."* Indeed Wayne was a great leader. We don't all get to anticipate our departure as Wayne did. In his last weeks with us he was instrumental in setting up the pathway for PDU to become an incorporated Association in its own right . In his own words *"Every day, every minute, every moment in life represents the end of the past. More importantly, it heralds the beginning of the future."*

Tim Willcox - President PDU Inc.

PDU Faculty Reflections

Wayne was a really supportive person and has made a significant contribution both personally and through his company to the development of perfusion and cardiac surgery overall.

Assoc. Prof. David Scott - Melbourne

What a loss, Wayne was a rare combination of a great mind, a vision, a philanthropist, businessman but above all a gentleman and a bloody nice bloke and so true and genuine. If only this world had more Wayne Pearsons it would be a much better place.
Tim Jones. MD FRCS (CTh)
Birmingham United Kingdom

Wayne was a hugely supportive friend who epitomised ethical leadership in the healthcare industry and was an example of how life should be led.
Prof Alan Merry - Auckland NZ

A great loss
John W Hammon MD Winston-Salem, NC USA

So sorry to hear about Wayne. What a loss. So blessed to have known him. He was an amazing person. Thinking of my friends Downunder.

Associate Professor Donny Likosky, Ann Arbour, USA

He has been an extraordinary leader and wonderful supporter for evidence-based perfusion practices. And he was a great human being.

Professor Paul Myles - Melbourne, Australia

His legacy is safe. We were all enriched by Wayne's humanity and he won't be forgotten

Associate Professor Simon Mitchell - Auckland NZ

A real philanthropist and leader in medicine - he will be greatly missed.
Professor Clive Landis - Barbados

Wayne was a remarkable man. A man of good humour and kind words. I never heard him say anything that wasn't positive or graceful. Wayne exuded a warmth of personality that is a rare and valuable trait. We could all learn from him in that regard. He never seemed to be one for the limelight, but was always there behind the scenes making it all work. His support of PDU was legendary and one of the main reasons it was such as success.
David Sidebotham and Sara Allen - Intensivist anaesthetists Auckalnd NZ

This is sad and shocking news. I will miss Wayne. He was such a kind and friendly person. It was always great to see him at meetings. Always smiling and welcoming.

Bob Groom, Maine, USA

REBRANDING OF PDUC

ANZCPR

Australian New Zealand Collaborative Perfusion Registry

Quality Perfusion through Reporting

Why the name change?

The Perfusion Downunder Collaboration has had a name change, the collaboration will now been known as the 'Australian and New Zealand Collaborative Perfusion Registry'. The collaboration was initially developed through the Perfusion Downunder (PDU) Meeting, however the collaboration itself has always been a separate entity. Initially developed in 2007, the collaboration now has 9 sites throughout Australia and New Zealand contributing to the registry, with a number of other sites having expressed an interest in joining the collaboration. In our most recent data harvest, data from over 20,000 procedures was collected. These data have provided the collaboration with an enormous resource to help us understand, report and improve the practice of CPB. To date, the work of the collaboration has been completely un-funded.

To broaden the appeal of the collaboration and to build a broader alliance with the Australian and New Zealand perfusion community it was recognised that the collaboration should be given a new name and logo. It is also our intention that by creating a new identity, the collaboration can be more clearly recognised as distinct from the PDU Meeting

Recent developments

This year we improved the process of providing data reports to contributing centres enabling centres to better understand their own practice, and the ability to benchmark with other centres within the collaboration. We are also working on a website for the collaboration: www.anzcpr.org

Future Endeavours

The collaboration will endeavour to work more closely with the executive committees of both the ANZCP and PDU meetings to provide content for scientific sessions at both meetings. To date, the only source of funding for the collaboration has been receiving the meritorious award at the ANZCP meeting last year. We will be further investigating competitive research funding, but also seeking corporate sponsorship. This model should assist us to achieve our goals of improving sustainability, reporting, and importantly participation!

PDU Inc. 12th Winter Meeting

Queenstown NZ

18 to 21 August 2016

venue

The Heritage

https://www.perfusiondownunder.com/

Enquiries to btackney@perfusiondownunder.com

PDU Inc. is supported by a grant from LivaNova Melbourne Australia

PERFUSION DOWNUNDER INCORPORTATED

Incorporated under the Associations Incorporation Reforms Act 2012 on 1 October 2015

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AUTOTRANSFUSION SYSTEM
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saves time and simplifies the handling; suitable for all blood volumes.

Sensors

continuously monitor incoming and outgoing hematocrit values and red cell separation performance for better patient management.

Unique height adjustment of corpus

for user convenience.

Efficient data management

with bar code scanner and standard USB interface.

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CALENDAR *of* EVENTS

DECEMBER 2015

13-15
International Conference for Innovations in Cardiovascular Systems
Tel Aviv, Isreal
David Intercontinental Convention Center
http://2015.icimeeting.com

JANUARY 2016

10-13
Winter Park Perfusion Conference
Winter Park, Colorado
Vintage Hotel
http://www.hatravel.com/Page/WPPC2016MainPage

FEBRUARY 2016

4-7
37th Annual Seminar of the American Academy of Cardiovascular Perfusion
Savannah, Georgia
Hilton Savannah DeSoto Hotel
http://www.theaacp.com/annual-meeting/

24-27
CREF 2016
Long Beach, California
Hyatt Regency Long Beach
http://crefmeeting.com

24-28
Cardiology 2016
Orlando, Florida
Loews Royal Pacific Resort at Universal Orlando
http://www.chop.edu/cardiology2016

28-3
32nd Annual Children's National Symposium: ECMO and the Advanced Therapies for Respiratory Failure
Keystone, Colorado
Keystone Resort
http://www.cvent.com/events/32nd-annual-cnmc-symposium-ecmo-the-advanced-therapies-for-respiratory-failure/event-summary-db7f1938714146b2af3a95b9526d4bd4.aspx

MARCH 2016

15-19
AmSECT 54th Annual International Conference
Colorado Springs, Colorado
The Broadmoor Resort
http://amsect.societyhq.com/meetings/upcoming.iphtml

APRIL 2016

20-23
Sanibel Symposium
Fort Myers, Florida
Sanibel Harbor Resort and Spa
http://www.perfusion.com/symposium/

MAY 2016

18-22
12th International Conference Pediatric Mechanical Circulatory Support Systems and Pediatric Cardiopulmonary Perfusion
New York, NY
New York Presbyterian/Columbia University Medical Center
http://www.pennstatehershey.org/web/pedscpb/home/conferenceinfo

19-22
Mechanisms of Perfusion
Orlando, Florida
Omni Orlando Resort
http://mechanisms.com

19-21
11th Czech Annual Conference Society of Extracorporeal Circulation and Supporting Circulatory Systems
http://english.czesect.cz/

JUNE 2016

1-4
EuroELSO 5th International Congress
Glasgow, Scotland
Scottish Exhibition and Conference Centre
http://glasgow-euroelso2016.com

3-5
26th Annual SEECMO Conference
Cincinnati, Ohio

22-25
The New Orleans Conference
New Orleans, Louisiana
Ritz Carlton
http://www.theneworleansconference.com

AUGUST 2016

18-21
Perfusion Down Under Winter Meeting
Queenstown, New Zealand
The Heritage
https://www.perfusiondownunder.com

SEPTEMBER 2016

20-24
AmSECT Quality and Outcomes Meeting
Sheraton New Orleans
New Orleans, Louisiana

OCTOBER 2016

1-5
30th EACTS Annual Meeting
Barcelona, Spain
http://www.eacts.org/annual-meeting/30th-eacts-annual-meeting/

27-29
ANZCP 33rd ASM
Rydges Convention Centre
Townsville,Queensland

NOVEMBER 2016

24-25
Scandinavian Paediatric Perfusion Meeting
Lund, Sweden
Grand Hotel
http://www.rhppc.dk

Australian and New Zealand College of Perfusionists

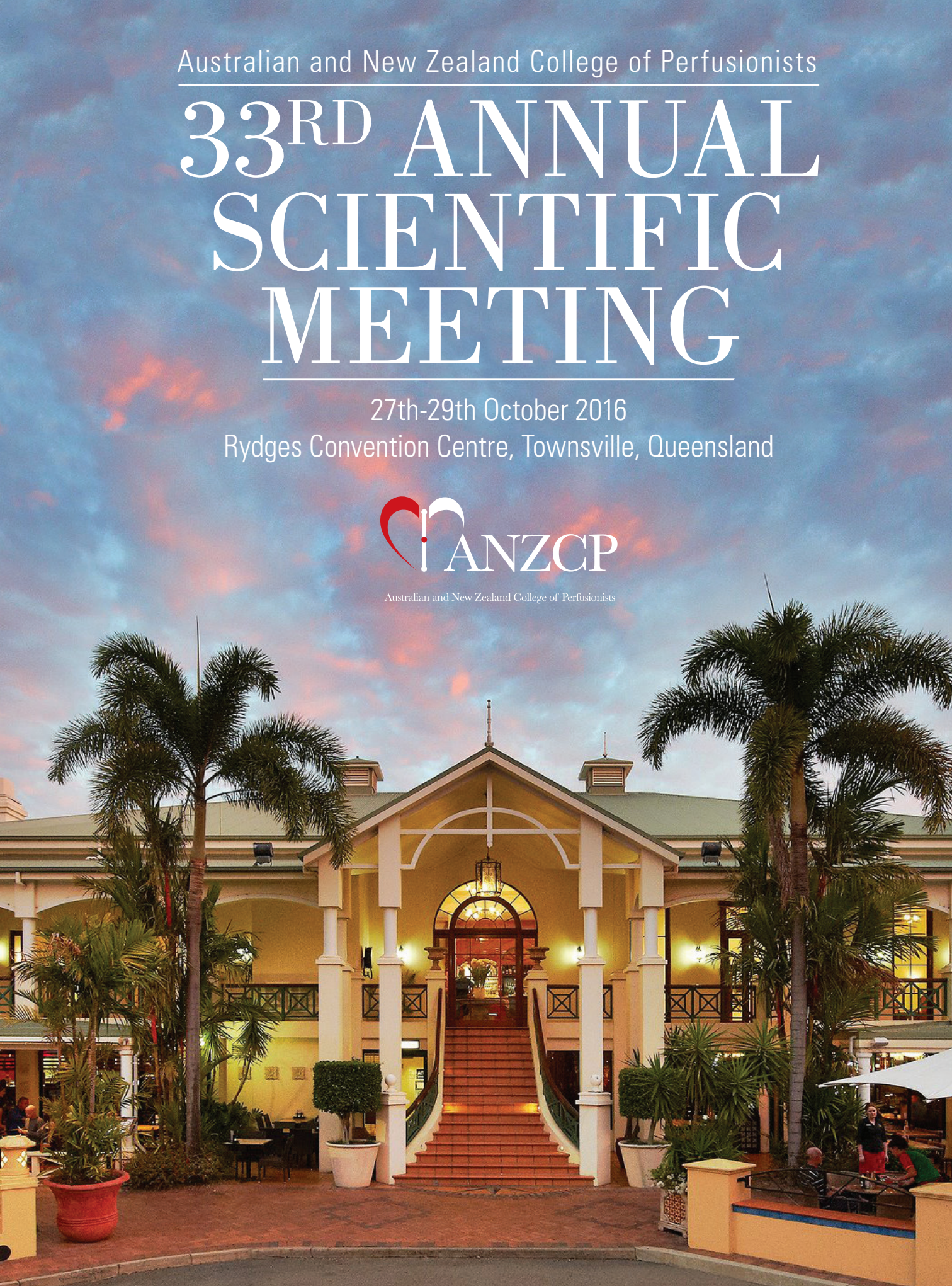
33RD ANNUAL SCIENTIFIC MEETING

27th-29th October 2016

Rydges Convention Centre, Townsville, Queensland

ANZCP

Australian and New Zealand College of Perfusionists



Australasian Simulation and Perfusion

Inaugural Australasian Simulation and Perfusion (ASaP) Meeting

6th – 7th May 2016

High and Low fidelity "hands-on" workshops

Perfusion Applications and Techniques

**Limited numbers 25-30*

Kolling Research & Education Building,
Royal North Shore Hospital Reserve Rd, St Leonards NSW 2065

Registration: $300 for the 2 day workshop

Organised by:

The Australian and New Zealand College of Perfusionists

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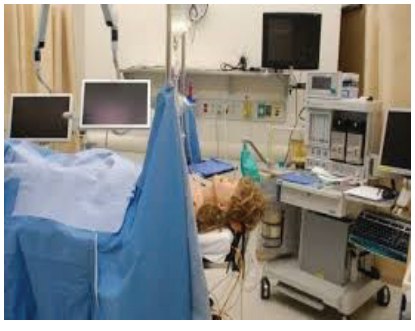

For further details contact:

Jane Ottens -
Jane.Ottens@acha.org.au

Darryl McMillan -
McMillan@med.usyd.edu.au

www.anzcp.org

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