Gazette Issue 06/17

The AUSTRALIAN AND NEW ZEALAND COLLEGE *of* PERFUSIONISTS GAZETTE

JUNE 2017

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The
AUSTRALIAN AND NEW ZEALAND COLLEGE *of* PERFUSIONISTS GAZETTE

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A MESSAGE FROM THE EDITOR

by Molly Oldeen, *CCP*

Welcome to the June 2017 edition of the ANZCP Gazette. We hope you enjoy the variety and quality of content. Be sure to read through the abstracts from the latest Intraoperative Management of Blood meeting that was held in Sydney this May.

I recently had the privilege of attending the combined meeting between the American Association for Thoracic Surgery (AATS) and the American Society of Extracorporeal Technology (AmSECT). It was held in Boston, Massachusetts at the Hynes Convention Center. Four days of various tracks were geared towards surgeons, perfusionists, or both. Attendees were able to both learn from and present alongside each other. There are clear advantages to collaboration between these two professions that are heavily dependent on each other for success.

In addition to attending the lectures and workshops, I was witness to the presentation of the John H. Gibbon award given to Professor Robert A. Baker. In this edition, Jane Ottens has written a nice summary of this honorable achievement, along with all of Rob's contributions to the field of perfusion and beyond over years.

These types of shared teamwork relationships should be happening throughout cardiac teams as a whole on a regular basis. Our academic training alone isn't always sufficient for the increased complexity of the field we work in. By collaborating, we can turn rooms full of clinical experts into expert teams.

Fostering teamwork can help assure mutually shared:

-Mental modes (i.e. we are here for the patient)

-Attitudes (i.e. collective efficacy and cohesiveness)

-Behaviors (i.e. closed loop and efficient communication, transparency)

The patients benefit the most from the relationships described above. With this investment, employees experience more job satisfaction, less stress and less turnover—to keep in mind when scheduling multidisciplinary practice meetings or department education series. I look forward to next years continued collaboration between AATS and AmSECT in San Diego, California.

Thank you again to all of the contributors! The editorial committee looks forward to choosing two $250 award winners for the best articles published within the Gazette to be presented in Melbourne at the ASM.

Kind Regards,

Molly Oldeen
The Gazette Editor

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A MESSAGE FROM THE PRESIDENT

by Mark Ambrose, *Acting President*

For those members of the College who do not know who I am, my name is Mark Ambrose and I am the Vice President of the ANZCP. Currently however I find myself in the position of Acting President of the ANZCP. Perfusion for me started as a trainee in 2002, at Sydney's Royal North Shore Hospital, where I still work today. I feel very fortunate to have the privilege of being the Acting President up to this year's Annual General Meeting. I am passionate about perfusion, and have high hopes that the ANZCP will increase the professionalism of our profession, and give value to its members.

I must make mention of the time and effort given by Jon Van Den Berg. On behalf of the membership and the executive, I would like to thank Jon for his contribution to the ANZCP as president. Thankfully Jon is continuing on with his work toward gaining registration for perfusion in New Zealand. We wish him much success in his future endeavours.

I would also like to recognize the wonderful achievement of Prof Robert Baker, Director of Research & Perfusion at Flinders University Medical Centre, Adelaide, who was awarded the John H Gibbon Jr. award by the American Society of Extracorporeal Technology. This is a fantastic achievement, well done. It is not only great for Rob, but also for our college and our profession as a whole.

The third Intraoperative Management of Blood (IMoB) conference was recently held in Sydney. This was an informative conference, focusing on intraoperative transfusion and haemostasis. This conference highlights the role of the perfusionist not only as the primary provider of extracorporeal circulation, but also as a resource for point of care testing, haemostasis, and intraoperative blood management. Congratulations to Darryl McMillan, Jane Ottens, and Christopher Morley.

Simulation is an area in which the ANZCP once led the perfusion world. It is my very strong belief that we all, as members of the College, can once again make full use of this powerful teaching tool in a number of ways. We are making moves in the right direction, and again thanks to Jane Ottens who recently coordinated a Perfusion workshop at a Cardiothoracic Trainee training day in Adelaide, utilising simulation. I understand that this workshop gave the surgical trainees a fresh perspective on the interaction of the heart lung machine with the patient, as well as the communication and teamwork required to conduct extra corporeal circulation. Thank you Jane and all the people from the ANZCP who gave of their time to make this a success. I am looking forward to the next Australasian Simulation and Perfusion Meeting (ASaP) in 2018.

Speaking again of meetings, this year seems to be flying by as we look toward the Annual Scientific Meeting in Melbourne. Hosted at the Langham from the 16th to the 18th of November, this is of course our main meeting of the year. Thank you Clarke Thys and Jim McMillan and co for being the main organisers and hosts. Killian O'Shaugnessy will be taking on the challenge of organizing the scientific programme with the assistance of Richard Newland and Rob Baker. The website for this conference is now available www.anzcpasm2017.com. I strongly encourage as many members to attend this meeting as possible, as either a speaker or attendee. It really is the attendees that make a conference, and we will have much to discuss as a College at this years AGM.

As we look to the future, our College and indeed profession, face not only a number of significant obstacles and challenges, but stands to gain some exciting ground. No one person is the College, and the College is no one person. We are all in this together, and together we need to adapt and change as an organization to best position our profession to capitalise on the gains. I strongly believe in perfusion as a profession, and the role of the perfusionist. I also believe in the people within our perfusion community, the past/existing people on whose shoulders we stand today. To steal somebody else's line, "Don't ask what the College can do for you, ask what you can do for the College". I look forward to meeting many of you in Melbourne this year, please come and have a chat with me. I also look forward to moving ahead with all of you as a College, improving our position professionally.

SIMULATION REPORT

by Jane Ottens

A Perfusion simulation Training day was run as part of the Cardiothoracic Training course 2017, held in Adelaide, in conjunction with the RACS Annual scientific congress Sunday 7th May to Thursday 11th May.

This was set up at Flinders Private Hospital theatres, using the ANZCP Orpheus simulator. We ran four sessions over the day, introducing all the trainees to perfusion.

The approximately 40 cardiac trainees, all had the opportunity to experience sitting at the heart lung machine ,understanding what we monitor, and having hands on experience with common issues such as low reservoir volume, venous air and occluded lines, along with the surgeon (me) asking them to hurry up or deal with the issue themselves. They also had exposure to gas failure and power failure, with all having a chance to hand crank an arterial pump.

They all got some insight into our issues and how we could all work together to provide better outcomes for our patients.

I have to thank Andrew Sanderson, the Orpheus driver for the day and the Flinders Medical Centre perfusion team of Farrar, Annette Mazzone, Richard Newland and Vijay Valiyapurayil for their help during the day.






A MESSAGE FROM ABCP CHAIRMAN

by L. Vincent Rajkumar

A warm hello to everyone…

It's a challenging year for the board. Few delegation and retention of the members ensued for the year 2017.

The Perfusion Course is running efficiently, there are 13 trainees at present. A total of seven candidates appeared for the Exam in February 2017 and all were successful in gaining the certification. Please do not forget to congratulate the following successful examinees on their achievement when you come across them:

- Jane Ennor
- Mitchell Bago
- Adam Roshan
- Cynthia Riddell
- Casey Edwards
- Majid Arammanesh
- Kirri Hogan

Mark Mennen decided to step down from the Chairman position after two years of exceptional service. We miss your unperturbed support Mark and wish you the very best for all your future endeavours. I urge you not to cut off completely as we request you to still extend your expertise when there is a need. Clarke Thuys agreed to continue as Course Coordinator which is a real bliss and we concede him as Board's helpline. He navigates and anchors without any fuss when we appear perplexed.

Jessica Ozdirik has taken on the Secretary position and still offers assistance on the Auto transfusion course. For 2017 Jessica is superintending the recertification process of Group 3 which is the largest one in the lot. It's a fair bit of work yet she quickly adapted to fit the bill. We have moved the last date of recertification document submission to 31st of May due to the transition of Secretary Position in the Board and the Treasurer position in the College. We would like to thank Kuljeet Farrar, our new treasurer, who assists Jessica in generating and handling the invoice for recertification. We also remember to acknowledge and thank Carla Zazulak, former Treasurer, for the prompt and fantastic support in the last couple of years. Kindly contact the Board if your recertification is due and have been missed or if you have changed your contact address.

We have Andrew Lahanas as Board executive who scrupulously contributes with zeal to uphold the integrity and the guidelines of the Board, also offers advice on the auto transfusion course. Sarah Varghese has undertaken the governance of auto transfusion course. The first term of the auto transfusion course had 16 enrolments and the number generally picks up in the mid-year.

The current board crew is fairly new and few who govern the educational and the legislative part of the College. The course content review is going on at a very slow pace. If you see yourself having a potential and passion to review the course content or willing to be a supervisor for reviewing and updating a particular module, kindly contact us without any hesitation. We need more support and will be delighted to embrace volunteers. Also encourage the students and the supervisors to provide feedback and offer assistance on reviving and refining the course content and delivery method. I would like to thank and appreciate Arthur Prevolous, Jon Van den Berg and Jane Ottens for their valuable time, contribution and continual support on the Board activities.

I request the practicing Perfusionists those who are settled in Australia and New Zealand, yet having overseas qualification to contact the Board. I encourage you to honour and pursue on accomplishing the certification process and being a part of the college to uphold the inherent professional integrity. We are striving to achieve accreditation and one of the primary reasons we are challenged is our trifling number. Kindly oblige and enrol yourself to be a vital part of the college sharing your talents and wealth of experience to bring in more value and prospect for the future.

AUSTRALASIAN BOARD OF CARDIOVASCULAR PERFUSION

AUTOTRANSFUSION COURSE 2018

The ABCP invites all healthcare workers interested in Autotransfusion to enrol in the 2018 Autotransfusion Course.
The purpose of the course is to provide current and future autotransfusionists with the background information necessary to provide a safe and effective service.

The course runs for 10 weeks and is conducted three times per year at a total cost of $250.00 (free to ANZCP members).

We are currently registering students for 2018 course commencement dates:

12 February (applications close 5 Feb)
21 May (applications close 14 May)
27 August (applications close 20 Aug)

For additional information and enrolment details, please contact the course co-ordinator, Sara Varghese at Sara.Varghese@act.gov.au

AROUND THE PUMP ROOM

by Brian Wright, *Fiona Stanley Perfusion Services*

Fiona Stanley Perfusion Services started in 2015 with the opening of a new purpose built unit 15 minutes south of Perth CBD. This resulted in the closure of two other Perth cardiac units and the staff from these services having the opportunity to be part of the new unit.

As politicians debate the current changes to visa and citizenship requirements and what it means to be a multicultural 'immigration nation' our team of perfusionists at Fiona Stanley Hospital are proud to say we are a truly multicultural department.

We are a team of 6 perfusionists with only 1 of us being Australian born. This apparent imbalance was not for want of trying to recruit local talent but no one from either the south or east of Australia seems to want to move to the 'wild west' We currently have 2 Indians, 1 Iraqi an English/Welsh girl and myself the incomprehensible Scotsman and of course the token Aussie to keep us right. Needless to say we all sat the test memorised the entire history of Australian cricket (very hard for a scot) and proudly hold our Australian Citizenship certificates. When asked if I consider myself Scottish or Australian I often answer Scotralian. Unless we're talking about football then 'oh sorry soccer' then I'm most definitely Scottish.

All of the team are either Australian or UK board certified and we have a stringent process of ongoing education and training in place including regular simulation using the Orpheus simulator

While our unit doesn't have a huge case load we undertake a broad and interesting range of techniques.

We perform approx. 500 bypass cases per year including aortic arch reconstructions, valve replacements, CABG etc. We are also the only Heart and Lung transplant unit in Western Australia and were the first unit in the southern hemisphere to use the Transmedics organ care retrieval system We still boast the longest successful transport and implant of a heart at nearly 10 hours using this system. We provide ECMO services within the hospital and also a retrieval service for patients requiring ECMO initiation and transfer to our unit.

The perfusion team are actively involved in both research and education and currently provide a 3 level ECMO training programme which consists of twice yearly foundation training, 6 immersive simulation sessions per annum and monthly wet labs. Our research programme is part of a larger network which has close links with Murdoch University and utilises their research facility on a regular basis.

However, what really makes Fiona Stanley Hospital a great place to work is the people and for me particularly the perfusion team. Having come from a large unit in Glasgow (Scotland) with 15 perfusionists and several thousand cases per year to what seemed at first an almost claustrophobically small unit, I've really come to appreciate the close knit team spirit that we have. I can honestly say they are a great bunch and without making you want to vomit in a bucket, I couldn't ask for a more genuine and hard working group of people to work alongside.

If you want to rectify the Aussie/ International imbalance then please feel free to apply next time we have a vacancy, however as things currently stand you may be waiting some time as thankfully It doesn't look as if anyone is moving any time soon.

NASRHP LETTER TO ANZCP MEMBERS

by Alison Horton *ANZCP Registration Committee and NASRHP*

Launch of the National Alliance of Self Regulating Health Professions (NASRHP) as an official body – driving quality, competent and ethical care from a certified health professional of self-regulated professions

The National Alliance of Self Regulating Health Professions (NASRHP) was originally an informal alliance which began in 2008 under the auspices of Allied Health Professions Australia, to support member organisations of self-regulating health professions, which includes the Australian and New Zealand College of Perfusionists. The recent transition to a formal body has been funded by the Federal Government via the Department of Health and Ageing.

NASRHP is now the national peak body for self-regulating allied health professions and sets benchmark standards for regulation and accreditation of practitioners within the professions. The 11 NASRHP standards have been closely modelled on the Australian Health Practitioner Regulation Agency (AHPRA) standards – those that apply to registered professions. Peak body organisations wishing to join NASRHP must demonstrate they meet the NASRHP standards.

How has NASRHP come about?

As you will know, the National Registration and Accreditation Scheme (NRAS) provides a national registration framework for health professions, which are required to be registered. The Australian Health Practitioner Regulation Agency (AHPRA) is the administering agency for NRAS.

Health professionals such as dentists, chiropractors, nurses, midwives, psychologists (to name a few) are 'registered' with AHPRA, and each year must demonstrate they meet AHPRA standards for their profession.

You may also know the NRAS is primarily aimed at keeping the public safe by "ensuring only health practitioners who are suitably trained and qualified to practice in a competent and ethical manner are registered".

However the Federal Government has seen no need to regulate many allied health professions, including perfusionists, which are viewed (rightly or wrongly) as low risk to the Australian public. Further, despite ANZCP's previous submissions, the Federal Government has confirmed that they have no intention of regulating these perceived low-risk or low number professions at any point in the near future.

Why do we need a national standards body?

Having an independent body ensures consistency in quality and supports self-regulating allied health professionals and satisfies national and jurisdictional regulatory requirements. Like AHPRA, NASRHP provides assurance to the public they are receiving quality, competent and ethical care from a certified health professional. NASRHP does not provide individual certification for practitioners – this remains the function of the practitioner's professional association – in the case of perfusion, this is the Australian and New Zealand College of Perfusionists.

The Australian and New Zealand College of Perfusionists aims to become a formal member of NASRHP during 2017/8. By becoming a member, ANZCP will have demonstrated that as a professional body it meets all NASRHP standards. This will assist in further increasing the credibility and recognition of the Certified Clinical Perfusionist (CCP) credential.

So, what does this mean for you right now?

Nothing changes directly for you as a CCP or in your day-to-day practice. The Australian and New Zealand College of Perfusionists continues to be your peak professional body and the Australasian Board of Clinical Perfusionists the administrator of your CCP credential. There will need to be one change in the future and that is you will have to meet your CPD basis requirements each year as opposed to a 3 yearly report that you have had in the past. This will take us a while to change so there is nothing you need to do or change right now. You do not need to have any direct contact with NASRHP itself.

What about in the future?

NASRHP standards are modelled closely on AHPRA standards, so if AHPRA makes an update to standards then NASRHP will also. In that case, NASRHP members including the Australian and New Zealand College of Perfusionists would need to also implement those changes.

In the near future, NASRHP will be seeking expressions of interest from certified practitioners who wish to join NASRHP's Pool of Assessors. NASRHP assessors will play an important role in the assessment of NASRHP membership and appeal applications from the professional peak bodies. I would encourage you to apply for a position if you feel you would like to be more involved in the ANZCP.

In a nutshell

- Having an independent national body builds public and government confidence in these professions as being of high quality, competent and credible.
- Nothing changes for you as a CCP in terms of your credential, practice provisions, or how you practice day-to-day.
- There will be a change in the future to yearly CPD reporting of recency of practice requirements. This will become an online reporting system making it easier for you to do each year.

For more information

For information about NASRHP and the NASRHP standards, please see the NASRHP website - **www.nasrhp.org.au**

If you have any specific queries regarding NASRHP please contact Alison Horton, ANZCP Registration Committee who represents the Australian and New Zealand College of Perfusionists on NASRHP.

WHICH WAY TO ENLIGHTENMENT?

by Anthony Black CCP

Perfusion is a dynamic, safety conscious profession, full of data sheets and reports, sterile environments and possibly even Mycobacteria. Does all this make us a better Perfusionist or keep us safe and warm within our bubble?

Having trained in Perfusion in the UK in the mid 90'S, I had a very well rounded training in all aspects of perfusion; adults, paeds, transplants, retrievals, ECMO, I could generate a list but you would stop reading, however I do feel as though I missed out on the most interesting and exciting years, and can only imagine the dedication of our forbearers, sterilising and setting up rotating discs, or managing the gas exchange in a bubble oxygenator.

There are circumstances when getting back to basics is a necessity and is what stands between a good patient outcome and not.

Ruby Hall Hospital 1st Heart TX

Having recently had the opportunity to help out with the charity "Healing Little Hearts" in conjunction with the "Grant Foundation" I accompanied my local surgeon from the Lady Cilento Children's Hospital, Brisbane; and along with anaesthetic and ITU colleagues headed off to the city of Pune, a 3 hour drive from Mumbai in India, to carry out a Paediatric Cardiac Surgery Camp.

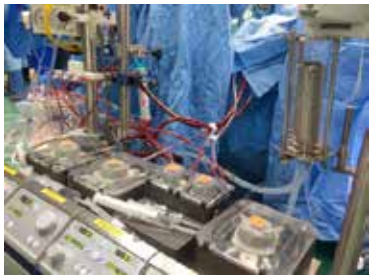
After a 14hr flight from Brisbane we had 2 nights of luxury in Mumbai , managed to fit in a bit of sightseeing in downtown "Bombay"(now known as Mumbai) appreciated the workers in the "Dobey" (outdoor laundry servicing 1.2 million articles of laundry daily, all of which are hand washed with not a repetitive strain injury in sight; although I cannot guarantee the absence of tropical disease in there, as well as a visit to "the Gateway of India"(think Indian Arc De Triomphe) before we had lunch in the Leopold café, sight of a major terrorist attack, where bullet holes are still evident in the walls.

We arrived in Pune early Sunday afternoon, and headed off to the Ruby Hall Hospital, an old Victorian hospital with an updated façade, but still requiring some major improvements within. I met with the local Perfusionist's to get an idea of the equipment I would be working with, every trip seems to bring up unique challenges that have to be understood even if they cannot be changed.

During this visit I had the pleasure in observing the very first heart transplant performed at the Ruby Hall Hospital, along with surgical team there were 20 other visitors, assistants and co-ordinators to ensure all went smoothly. The operation went very well with the patient discharged from ICU within a few days; the only issue I observed was in communication! Surgeon asked if heart had touched down yet, was given a positive answer and proceeded to explant the heart and lay it out on the table for everyone to marvel at the tissue merrily beating away with no blood supply, although the atmosphere did change somewhat when they discovered the donor heart was still mid-air and "someone" had given false information, I was having flash backs to my time in Newcastle when we couldn't land due to fog and had to be diverted to Edinburgh; no such problems this day, the heart duly arrived a few minutes later and the excitement continued until the heart began to beat in the recipients chest. In 2015 there were 146,133 deaths as a result of road crashes, and haven driven the 160km from Mumbai to Pune I don't believe this number will reduce any time soon!

So the first day of our Cardiac camp was eventful, I was woken at 4am with an ECMO call-out! This I realised was a call-out in Brisbane, and I hadn't turned off my "i-message", I

thought I was having a week away from the digital age, but it was still following me. Anyway I was now up bright and early anticipating a good day.



I arrived at the hospital around 7am, greeted by the trainee Perfusionist, she unfortunately had to spend the evening sleeping in the Perfusion room, (there is a camp bed on the floor in there), as the heart transplant hadn't finished until after 9pm and the hostel she lived closed the door at 9! Not really an issue I had had to contend with in the UK or Australia. She didn't seem too bothered about this, and accepted it as part of her day, don't know if I would have done the same?

We had decided to start the week off with just 1 case on the Monday. An older patient so as to get a feel of the systems and processes available. I was introduced to 30+ year old Sarns 8000 heart lung machine, well it's not really a heart lung machine more of a selection of pumps precariously balanced on a base with wheels, no battery back-up, level sensor or bubble detector, and as far as blood gas monitoring, there was a box bolted onto the machine which had the capability of attaching venous and arterial sensors, however as I was to find out, the sensors were too expensive to use for our patients, so someone had made use of the electrical plug and cut it off the power cable…. As such the only monitoring we had was crossing the arterial and venous lines to differentiate the colour, and therefore guesstimate what the sats where! There was 1 piece of monitoring available, an arterial line pressure measured via an old tygos gauge, although it hadn't been serviced since the days of the British Empire it seemed accurate, or at least gave us a number and did change when the pressure went up. it was a bit of a concern from a sterility aspect that there was no isolator between the blood and gauge but the locals seemed confident that the column of air in the line prevented contamination of blood, (they knew that an isolator should be used however it was an extra cost that had to be justified to the administrators!) Throughout the whole week I didn't actually see the blood come into contact with the gauge; local knowledge goes a long way!

They did have a timer on the pump, although it did take until the end of the week to change the AAA batteries needed to be able to read the numbers, and a bedside lamp was placed next to the reservoir to assist in watching the level in the reservoir.

So this was it first case ready to go, oxy primed, Cooley chamber primed (this was new to me having predominantly worked in paediatrics for the last 15 years) lines taken down. Time out complete, (sorry that was a joke), just about to open chest when, all power to the theatre went off!!

Everything would be ok, emergency lights would stay on, battery back-up would kick in, oh yes I forgot I was in India! As if in a well-practised drill everyone pulled out there smart phones flicked on their torch to light up the relevant area, whilst a theatre orderly went in search of the trip switch, which was conveniently located on the wall near the perfusion pump, power was returned to the theatre, a "bare" power cable on a syringe pump was identified (only after anaesthetist had received an electric shock to the hand) and changed and the procedure recommenced, I did insist on eyeballing the hand crank just in-case, although they did assure me that never happens.

The cases were completed by the local perfusionists under my guidance, and as the week progressed slight modifications were identified in the practice, which could be easily and cheaply implemented, in order to try and improve patient outcomes, increase the safety to the patient and reduce the possibility of human error.

The whole week was a great success and thoroughly enjoyable both for myself and local perfusionists with whom I worked, they work tirelessly under very difficult situations with equipment which could only be described as "old", yet still manage to produce successful outcomes.

We completed 9 cases during the week, with ages ranging between 1 month and 13 years, all of which were complex, and would have been expected to die within the year if not operated on. As is the nature of charity cases, these patients should have been operated on much earlier, although due to the cost of the procedure didn't have access to surgery.

If you ever get the chance, put your hand up, and step out of your comfort zone, amazing things can still happen!

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Healing Little Hearts
Give a child a new life

"The best way to find yourself is to lose yourself in the service of others."
~Mahatma Gandhi

OPEN HEART
INTERNATIONAL TANZANIA
NOVEMBER 2016

by Kuljeet Farrar

Tanzanian immigration is an adventure everyone should experience at least once in their lives, but only the once! After traveling twenty-three hours from Adelaide, nothing says "Welcome to Dar Es Salaam" more, than an imposing African woman in uniform, holding a rather large stick (I was sure she was about to prod me, before the look of sheer terror on my face made her think twice), asking if I already had a visa. Alas I did not have a pre-arranged visa and I curse that day!

We were constantly told, "look after your passport, don't let it out of your sight". So you can imagine my apprehension when after forty-five minutes, finally getting to the front of the queue, I presented my passport, which was then promptly taken away to a back room with no explanation and then told to pay my visa fee at the next window. Now what??

I waited and waited and waited, I was slightly bolstered by the fact that I was surrounded by countless other travelers, also milling around, with that very pained "f**k, where have they taken my passport" look. (This is when I became acquainted with "Tanzanian time". For those who have been on Open Heart International (OHI) trips before, you will be well acquainted with this time phenomena. It is also present in Fiji, Samoa, PNG, and Rwanda. There must be a rip in the space-time continuum above these countries.)

Periodically an Immigration officer would come out amongst us to deliver the now freshly stamped passports, reading out the names in his uniquely Tanzanian way. Finally after another forty-five minutes, a name was read out: "Cull-git Fa-ra-ra". No one came forward to claim the passport after thirty seconds, I then realized it was mine! (I have to say, I've never been more happy to see that Silver Fern radiating from the front cover of my passport.) Now to collect my bags and present them to Customs, this should be a breeze!

It's hard enough trying to explain to a lay person what exactly a Perfusionist is. But my job was made just a little bit harder when trying to explain to Tanzanian Customs, why I had two Cobe Venous Saturation modules, an affinity oxygenator holder plus several bags of cardioplegia in my luggage. In all seriousness I did want to say "I promise it's not bomb making materials" but I didn't really fancy the possibility of a strip search. Finally after almost two and a half hours after disembarking, I was met by the welcomed and very friendly face of John Dittmer.

John and I had been briefed beforehand in Australia of what to expect and what the expectations were of the local staff from OHI. This OHI trip was different due to the fact that the main hospital in Dar Es Salaam was already performing adult and paediatric cardiac surgery. Unfortunately, the morbidity and mortality of the adult cases was extremely high, but surprisingly the paediatric patients had an excellent survival rate. Our job was to identify and offer solutions as to what could be causing the low survival rate of the adult patients.

The next morning, John, myself and the rest of the team composing of biomed, nursing, cardiology, AV/PR, and logistics (surgery, anesthetics and ICU were to arrive in the country later) made our way to the Jakaya Kikwete Cardiac Institute (JKCI) based at Muhimbili National Hospital. Our eyes were dazzled by the vibrant colours, exotic fruit and friendly faces we encountered on our bus drive. Once there, we got down to the serious mission of unpacking and sorting out local stock levels.

John and I met also with the Chief Perfusionist Robert as well as the other four Perfusionists; Sophia, Fred, Sylvester and the trainee Peter. The first four had been perfusing for around five years each. They showed us around the JKCI that had been built by the Chinese government as a gift to the Tanzanian people. A lovely thought but unfortunately all the signs and labels in the theatre were in Mandarin and like the average Australian, the average Tanzanian did not speak a lot of Mandarin. But the Chinese in all their helpfulness had put the English translation underneath the Mandarin signs. "Do not enter when theatre is in use" became "be under operation"

Another helpful thing the Chinese had left were 2 HLM's that were manufactured in China, which suspiciously looked a lot like a Sarns Terumo HLM. Who knew that, you could not only get a Hermès

Birkin knock-off in the back streets of Shanghai, but around the corner you can get a complete HLM! Fortunately, when the Chinese left the gift of the 2 HLM's, they also left an extensive instruction manual, however, unfortunately it was all in Mandarin.

The main issue the local staff seemed to be having with the adult patients was adequate myocardial protection. They were currently using a 4:1 parts blood/crystalloid cardioplegia method. We measured the potassium of this solution (which they used for both initial and maintenance doses) and it came back at nine mmol/L. John did some calculations and worked out new initial and maintenance solutions that could be made up and maintained using local stock.

Unfortunately the only crystalloid available to the Perfusionist was Ringers lactate, which was used for the pump prime and

to make up the cardioplegia. Once we started using the new cardioplegia solution, the patients seem to have much improved myocardial preservation and would often spontaneously revert back with a rhythm after the cross clamp was removed.

Another thing that John and I immediately noticed was a definite lack of communication between the Perfusionist, Surgeon and Anaesthetist. I'm very used to working in a team environment, but there was a distinct lack of cohesion in theatre. Our OHI adult Surgeon and Anaesthetist also noticed this and we all spoke to our local counter-parts at length about the importance of communication for the best interest of the patient and the team.

Being there for such a short time, we didn't have the time to encourage major changes but we felt the changes we suggested were easy done with minimal cost to the JKCI. As always I come home from one of these trips feeling very humbled and lucky. If you've ever thought of participating in one of these trips, I strongly encourage it. We donate a small part of our time, skill and money and it can make a life-changing difference to someone else. Where else can you buy an Ostrich egg sized avocado for 25 cents!

DR CHARLES WHO??? AN ACCOUNT OF UNRECOGNISED BRILLIANCE

by Martin Gill

Within perfusion, cardiac surgery and health care in general there are many unsung heroes. The reasons for individual's lack of recognition within medical disciplines are many. These reasons could be a desire for anonymity, they could be due to an organisational superior taking credit for a junior's endeavours, they could be due to a general lack of interest from the wider community at that particular time, or the reason could be more sinister; perhaps due to a preconceived opinion that is not based on reason or actual experience.

Charles Drew was born in Washington D.C. on June 3, 1904. He was the son of Nora Rosella and Richard Thomas Drew, who between them had Negro, English, Scots, and Indian ancestry. As is often the case, it was the Negro component of that ancestry that defined the majority of Charles Drew's life.

Drew grew up in a middle class, interracial neighbourhood where he attended the segregated Paul Laurence Dunbar High School. Drew's early promise was as an athlete, winning a scholarship to attend Amherst College, Massachsetts. In 1926 Drew graduated from Amherst with a Bachelor's degree, and went on to become the Director of Athletics and Instructor of Biology and Chemistry at Morgan College, Baltimore.

With increased emphasis on academia Drew, in 1928, attended medical school at McGill University in Montreal. It was here Drew gained both Doctor of Medicine and Master of Surgery Degrees in 1933, as well as winning Canadian awards for hurdle, high and low jump.

Drew served and then completed his surgical residency at Royal Victoria Hospital and then Montreal General Hospital from 1933-1935. Drew then discovered that obtaining employment as a black surgeon was not straightforward in 1935 America. He became an Instructor of Pathology at the 'black' Howard University, Washington DC. Three years later drew became a surgical fellow at Columbia University's Presbyterian Hospital in New York. It was here that Dr Allen Whipple and Dr John Scudder introduced Drew to the new concepts of fluid and electrolyte therapy in the treatment of shock, as well as blood preservation in the blood bank at the Columbia-Presbyterian Medical Centre.

Whilst in New York, and with Dr Whipple's sponsorship, Drew passed the American Board of Surgery examinations. As part of the oral component of the examination process, Drew gave a renowned lecture on fluid balance and shock management. Drew eventually became the first African American to be appointed an examiner of the American Board of Surgery.

Between 1938 and 1941 Drew and his mentor at Presbyterian Hospital, Dr John Scudder, developed a blood bank comparable to those in major centres at that time. They also published a series of 12 articles under the heading of "Studies on Blood Preservation." Their studies relating to red cell potassium leakage after death resulted in the abandonment of the concept of cadaver blood transfusion. Further work, by Drew, addressing the changes to blood when shaken had implications for blood transport and storage during World War II.

Prior to America's entry into World War II, Drew was the medical supervisor of a program to provide liquid plasma to the injured soldiers of both France and Britain. This eventuality was a direct result of research by Scudder and Drew on the feasibility of shipping liquid plasma overseas. It is said that Drew's greatest strength in this plasma collection and processing project was in the coordination of people, products and ideas and translating them into practice. It must be remembered that prior to this time central laboratories and standardised procedures simply did not exist.

The Red Cross then appointed Drew to oversee a pilot project in New York to test out Drew's recommendations of a centralised operation with the aim of mass production of dried plasma, essentially becoming the first Red Cross mobile collection program. The introduction of a program whereby donors would give to unknown recipients without monetary gain was a new one, but also a successful one (see Figure 1).

Drew's New York pilot study for dried plasma went on to provide the blue print for the American Red Cross national blood plasma program. By this time, though, Drew had already returned to teaching at Howard University's Freedmen's Hospital, where he served until his death.

Many stories exist about Drew resigning from the Red Cross due to a refusal by the Red Cross to cease the practice of blood being segregated by race. This is unlikely to be true. Drew worked for the Red Cross from January till April 1941. At this time the guiding Subcommittee on Blood Procurement, National Research Council stated: "Donations should be accepted from both males and females and from members of all races". Later in 1941 the Red Cross did bow to the demands of the US military and began firstly refusing blood donations from black donors, and later segregating blood donations keeping black donations specifically for black recipients.

It is known that Drew detested the policy of blood segregation. A letter to his mentor, Dr Scudder in 1944, singled out the US military for being responsible for this occurrence. Ironically Dr Scudder had become a very vocal proponent of the blood segregation practice, giving many talks and writing many articles on the subject. Whilst at Howard, the now Professor Drew spoke out again about the practice and also went on to protest about the impediments of racial factors upon the delivery of health care. He then went on to illustrate that even though he was certified by the American Board of Surgery and was one of its examiners, his race made him ineligible for membership in the American College of Surgeons and the American Medical Association.

Drew's return to Freedmen's Hospital in 1941 marked the time he committed himself fully to teaching. Drew worked tirelessly to ensure his black students obtained internships and even

managed to get his top students speciality training at white medical institutions. Drew went on to train more than half of the black surgeons certified by the American Board of Surgery in the 1940's. Many people believe that it was in surgery and the training of surgeons where Drew's true greatness lay (see Figure 2).

Drew himself was a member of the American-Soviet Medical society for numerous years, in 1946 became a fellow of the International College of Surgeons, received Honorary Doctor of Science degrees from Virginia State College in 1945 and from Amherst College in 1947, in 1949 served as surgical consultant to the Surgeon General of the U.S. Army, in 1944 was awarded the Spingarn Medal by the National Association for the Advancement of Colored People for his work with plasma.

Dr Drew's life was sadly cut short in an automobile accident on a North Carolina highway in 1950. Many myths and legends have been told and retold over the years pertaining to the circumstances of Dr Drew's death.

It is understood that after a lengthy day at work Drew and three other doctors began the long journey to a conference in Alabama. Alternating drivers, the four physicians drove through the night. Around 7:30am Drew was behind the wheel when it is believed he fell asleep. The car, travelling at 70mph, veered to the left and rolled over, with Drew sustaining traumatic injuries to his head, thorax and left leg. All those in the vehicle were transported to Alamance General Hospital in Burlington, North Carolina. Drew was still alive upon arrival to the hospital 40 minutes after the accident, and was actually recognised by the attending surgeon.

A total of four surgeons are believed to have worked on Drew. One of the surgeons remembers observing Drew's fixed and dilated pupils, and signs of severe shock. Fluids were commenced and specialists from Duke University consulted for other treatment options. It was determined that Drew was unsuitable for transfer to Duke University due to the likelihood of him not surviving the 60 minute journey. Drew was pronounced dead at 10:10am in the emergency room.

Drew died from a closed head injury, severe soft-tissue injuries, and a crushed chest injury, which resulted in obstruction of the superior vena cava and avulsion of the hepatic veins. Drew's death certificate lists cause of his death as (1) brain injury, (2) internal haemorrhage lungs, and (3) multiple extremities injuries. Drew's hospital stay was confined to the emergency room and thus he was never formally 'admitted' to the hospital; an occurrence that over the years has fuelled many of the rumours, myths and legends surrounding his death.

One of Drew's colleagues present in the car with him, John Ford, did require hospital admission for 2 days. Due to the colour of his skin he was admitted to a room in the basement of the hospital specifically set aside for Afro-American patients.

Drew's body was taken back to North Carolina where his funeral took place on April 5th, 1950. Drew was survived by his wife and 4 children.

In the time following Drew's death he has received many awards and accolades. He was awarded a posthumous fellowship in the American College of surgeons. Many schools, academic institutions and medical facilities now carry his name. In 1981 the U.S. postal service even issued a stamp carrying his likeness (see Figure 3).

A memorial stone at the location of his automobile accident caries this inscription:

Charles Richard Drew
1904–1950

Black scientist and surgeon
Pioneer in the preservation of blood plasma
Medical director of the Blood-for-Britain Project, 1940
Director of the first American Red Cross Blood Bank, 1941
Teacher to a generation of American doctors, Freedmen's Hospital, Howard University, Washington, D.C.
Outstanding athlete, Amherst College and McGill University
Member of Omega Psi Phi Fraternity
Steadfast foe of racial injustice
Died in Alamance General Hospital 1 April, 1950, after an automobile accident at this site

"There must always be the continuing struggle to make the increasing knowledge of the world bear some fruit in increased understanding and in the production of human happiness."

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Figure 1. Charles Drew, left, with the first mobile blood bank collection unit, New York, 1941

Figure 2. Charles Drew teaching interns and residents at Freedmen's Hospital, 1947.

Figure 3.

WORKPLACE BULLYING; BAD THINGS HAPPEN WHEN GOOD PEOPLE DO NOTHING.

by Helen Scarrott

"There is absolutely, unequivocally, no way that you are going to win in this world without being noticed, accepted, admired, complimented, sought after and appreciated" states Dr Phil in his book "Life Code" [1]. Does this make sense? As children weren't we taught to be humble and team players? Didn't the tall poppy syndrome make us fear standing out in the crowd? Will this help us in today's work place? Does this attitude leave us vulnerable to bullying? As perfusionists, we work in an environment where we care for patients. However, are we caring for each other?

Try 'googling' bullying in the workplace. I just did and got 38 million results!!! Yet again the Townsville Hospital is in the news and described as having a culture of bullying and harassment [2]. This isn't just TTH where this is happening. Does this mean that there is an increase in this type of behaviour or is there a greater awareness? Alternatively are we all too thin skinned and/or have we not developed coping strategies for dealing with bullies?

Bullying differs from harassment [3]. The target (the person being bullied or harassed) is usually immediately aware of harassment. The focus is usually obvious (eg. gender, race) and has an offensive vocabulary. Bullying tends to be more insidious by comparison. It may be happening for weeks or months before it becomes obvious and tends to be an accumulation of incidents that taken in isolation may seem trivial, with the focus often on competence or popularity.

On the streets, the law deals with murderers and other perpetrators. But anywhere human relationships exist, such as work or home, we seem more likely to sit back and assume natural justice will prevail. Domestic violence is no longer tolerated in Australian society. When you think of the home as the woman's workplace, the comparison with paid work is obvious. Workplace bullying should be seen in the same light. [3]

We had a registrar who was a genuinely nice person. When she asked 'how is your day going' she really wanted to know. She was caring and hard working. She would bring in food for everyone quite often. I gave her a copy of the book "Nice girls don't get the corner office" to read (4). In that book it described how in a meeting of men and one woman, it was always the woman asked to organise coffee. Our registrar gave up cardiac as she was always being overlooked, undermined and undervalued. She has gone into another specialty where her character is valued. Should the 'nice ones' get abused, overlooked and driven out? Why did no one stand up and say that was not how she should be treated? No one bullies the bully.

We all know the bully, defined as "someone who knowingly abuses the rights of others to gain control of the situation and the individuals involved" deliberately and personally uses intimidation and manipulation to get their way. The key words are knowingly, deliberately and persistently. The target is an individual who often has the desirable qualities of competence, networking and emotional intelligence [1].

Sadly, the hospital setting seems to be a breeding ground for bullying and harassment. In 2001, Griffith University in Brisbane estimated Workplace Bullying cost the Australian economy between 6-13 billion dollars per annum!!! How much is the financial burden of lost productivity and replacement of staff on the overstretched health dollar? What about the time and money spent on investigations and mediation? That doesn't include the sometimes devastating effect it has on the target.

But what can we do to protect ourselves? What can we do to ensure we do not become the new focus of persecution and marginalisation if we stand up for our colleagues? We must have courage enough to stand up for what is right, for ourselves, our colleagues as well as the welfare of our patients.

From the outside, standing up to bullying sounds easy, but how we can manage bullying behaviour and protect ourselves? Here are some suggestions. [5,6]

- Mentally separate yourself from the bully. You do not deserve to be publically berated or humiliated for a mistake, no one does.
- Name the behaviour. Name either the blatant (overt) or subtle (covert) behaviour. Naming must be specific and observable. It must be a behaviour that is undeniable.
- During covert sabotage, acknowledging the behaviour can short-circuit it.
- During overt tirades, there is a sense of power and it gains momentum. Interrupting this can short-circuit the assault.
- Walk away from the overt bullying. By walking away the audience goes too. The bullying will stop when there is no audience.
- Openly criticising. Respond with I will be more receptive to feedback delivered calmly. If the bullying continues, walk away.
- Yelling – interrupt and say, I will talk to you when you are not yelling. If it continues, walk away.
- Solicit support from family and friends or consult a professional therapist. The less secretive you are, the less burden you must bear alone. Don't expect them to solve the problem but offer a safe place to discuss your feelings and keep you grounded.

- Support your conversations with facts and documentation. Keep a notebook and write down behaviours, dates, times and witnesses. Objectively sharing this information lets the bully know you won't be an easy target.
- Solicit witness statements.
- Present this information to your line manager or HR. If they don't address the problem lodge a formal complaint.

What do I think? Soliciting support seems to be the most effective way to deal with bullies. As the old saying goes, "A problem shared is a problem halved". Although management is committed to the safe work place, they appear to be fairly ineffectual by being slow to recognize, acknowledge and then act on bullying. We need to foster relationships with managers so that approaching them is not so daunting.

Queensland Health, as with all health institutions, has policies for dealing with bullying. Safework Australia's document [7] has set guidelines for workplace bullying. They state "that the risk of workplace bullying can be minimised as far as is reasonably practicable by creating and promoting a positive work environment where everyone is treated fairly and with respect. A combination of control measures aimed at the organisational level and at individual behaviours should be considered".

Effective leaders will send a clear message to workers that the organisation is serious about preventing workplace bullying and contribute to a positive workplace culture where unreasonable behaviour is not tolerated.

Managers can demonstrate commitment in various ways including by:

- Modelling respectful behaviours at all times.
- Developing and implementing a bullying policy which clearly identifies the expected behaviours and consequences of not complying.
- Dealing with unreasonable behaviour as soon as they become aware if it.
- Ensuring that reports of bullying are taken seriously and properly investigated.
- Consulting with workers.

It is easier to see bullying in others, but sometimes self-reflection is helpful. 'Beneath The Stitches Blogger' [6] suggests we ask ourselves this question- "Do I engage in any bullying behaviours?" and recommends we follow the steps below:

- Always address people by name – it shows respect, you have bothered to learn and remember their name.
- Give help when it is asked for, if able.
- Accept your fair share of the workload; help others with a more intensive workload.
- Don't gossip, if you have an issue talk to the person directly. It is better not to encourage rumours and gossip.
- If you see something speak up. By not speaking up you are accepting that behaviour.

Dr Phil recommends that:

- You need to get attention, not avoid it.
- You need to do things that put you on the radar.
- You do that by making noise, playing big, making an impression.
- Remember that only 7% of all communication is verbal.
- You must "stretch" and behave your way to success, even if it feels like "fake it until you make it".

I hope Dr Phil is right. Be assertive everyday; use it as practice for when we need to defend ourselves. Use being a strong part of the team as a way to protect ourselves from bullies. Peer support is essential in all aspect of our job. Within our own department of three, we need to and do support each other 100%. The ANZCP is a great networking establishment that allows regular contact with others in our small profession. Form relationships so we can easily call a colleague in a different centre and discuss what is happening to gain an outside perspective. We need to stand up to these bullies by showing a united front with all our colleges.

It is important to remember the old saying: "Bad things happen when good people do nothing"[3].

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As part of the AmSECT Experience, plan now to take advantage of the special Pediatric event this fall. Last held in 2013, this exclusive gathering focuses on the unique needs of pediatric perfusionists. **Check www.amsect.org for more details.**

PROFESSOR ROBERT A. BAKER.

DIRECTOR OF RESEARCH & PERFUSION AT FLINDERS MEDICAL CENTRE, ADELAIDE

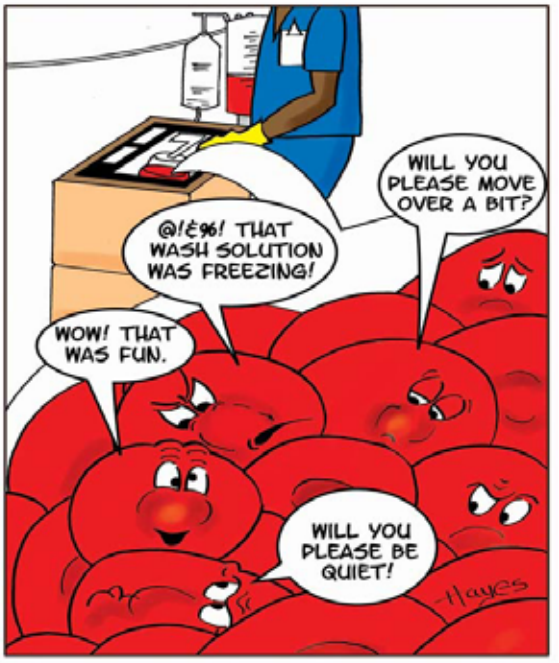
by Jane Ottens

Recipient of the 2017 AmSECT John H Gibbon Jr Award, May 2017, Boston USA.

American Society of Extracorporeal Technology's highest award, the John H Gibbon Jr Award is to honour a candidate making a significant contribution to the cardiopulmonary discipline interrelating with the field of extracorporeal circulation. It was first presented in 1974 to Dr Clarence H. Dennis. The illustrious list of recipients includes leaders in the field of cardiac surgery with names we all know from textbooks such as Doctors Lillihei, Debakey, De Wall, Buckberg and del Nido to name just a few.

Perfusion recipients are few and include Jeff Riley, Bob Groom and Mark Kurutz -leaders in the field of perfusion for many years. Rob, now appropriately joins that group for his contribution to perfusion. For the past 25 years Rob has been involved in both ANZCP and AmSECT with his wide area of interests both specific to perfusion but more broadly in relation to the outcomes of Cardiac Surgery. Recently one of his main areas of interest has revolved around registry, and he Chairs the Australian and New Zealand Collaborative Perfusion Registry, is on the Steering and Registry committees for the Australian and New Zealand Society of Cardiac and Thoracic Surgery database and is also involved the PERForm Perfusion Registry and the Massive Transfusion Registry. He is also involved in Guideline development work with the STS, SCA and AmSECT, and has contributed to the published Blood Management and Temperature guidelines, and the soon to be published Anticoagulation Guidelines.

On behalf of those who work with him, and the ANZCP, we would all like to congratulate Rob, but also thank him for his enormous contribution to our profession, within Australia and New Zealand, but also internationally.



THE 'WASH CYCLE' BLUES OF THE RBC.

NO? YES!
NO & CARDIOPULMONARY BYPASS

by Clarke Thuys, Stephen Horton, Martin Bennett, Simon Augustin, Stephen Bottrell, Bradley Schultz, Alison Horton, *The Royal Children's Hospital, Melbourne*

INTRODUCTION

CPB and ECLS are known to induce a systemic inflammatory response. This response is varied, highly complex and the specific cellular and molecular processes involved are not well understood. However these processes include complement activation, cytokine release, endothelial injury and apoptosis.

The endogenous nitric oxide (NO) pathway can play a protective role in the event of patients experiencing an inflammatory response, ischaemia/reperfusion injury and apoptosis.

This article reviews where NO comes from, and the mechanisms involved in these therapeutic effects. The NO setup for CPB and results of the prospective, randomised study conducted at RCH to investigate the clinical effects of administering NO as a component of the oxygenator sweep gas during CPB in children as published in Circulation14 are discussed.

Low cardiac output syndrome (LCOS) as a diagnosis within 48 hours of CPB was the primary outcome. LCOS was defined simply as evidence of poor tissue oxygen delivery, high dose inotropic or vasopressor support, or the need for ECMO. Secondary outcomes were use of inhaled NO, peritoneal dialysis, delayed sternal closure, blood loss, transfusion of blood products, the duration of mechanical ventilation, ICU length of stay and hospital length of stay.

Fewer patients in the NO group developed LCOS than in the control group. (15% vs 31%, p=0.007) Patients who developed LCOS required more dialysis, more use of inhaled NO, longer mechanical ventilation, longer ICU stay and longer hospital stay than those who did develop LCOS. The degree by which LCOS was prevented was dependent on age and complexity of surgery. There was no difference in bleeding or blood product transfusion between the two groups.

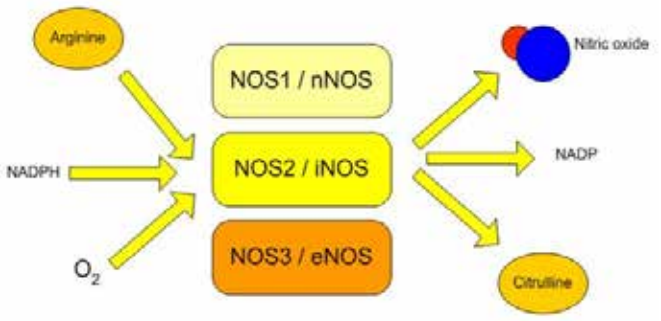
Given the potential pathophysiological benefits the use of synthetic gaseous NO administered via the oxygenator may improve outcome in some of our patients.

NITRIC OXIDE BACKGROUND

Why do we need nitric on bypass? Nitric oxide is synthetized by essentially all cardiac cell types is a ubiquitous cellular messenger that plays a key role in regulating cardiac function. Bypass is associated with the activation of different coagulation, pro-inflammatory, survival cascades and altered redox state. Nitric oxide has a role in many of these alterations. Despite significant refinements over the years, oxidative stress and inflammation remain major concerns when using bypass. The study by Cavalca1 shows that patients are already suffering from nitric oxide pathway impairment before bypass as a result of their altered pathology.

Where does Nitric Oxide come from? Broadly speaking there are 2 types of nitric oxide sources: endogenous and exogenous.

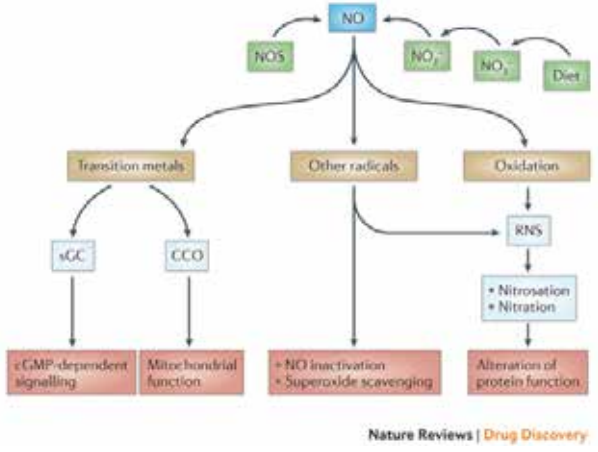
Endogenous NO in mammals is synthesized by a number of isoforms of nitric oxide synthase (NOS). Three distinct isoforms of NOS have been described: neuronal (NNOS, NOS-1), inducible (iNOS, NOS-2) and endothelial (eNOS, NOS-3). nNOS and eNOS are Ca++ dependent while iNOS is Ca++ independent.



From: University of Reading Nitric Oxide Research Group8: Production in Cells, Synthesis of Nitric Oxide.

It has been shown that NOS-independent reduction of dietary or endogenous sources of nitrate and nitrite are important contributors for the production of NO in mammalian tissues.

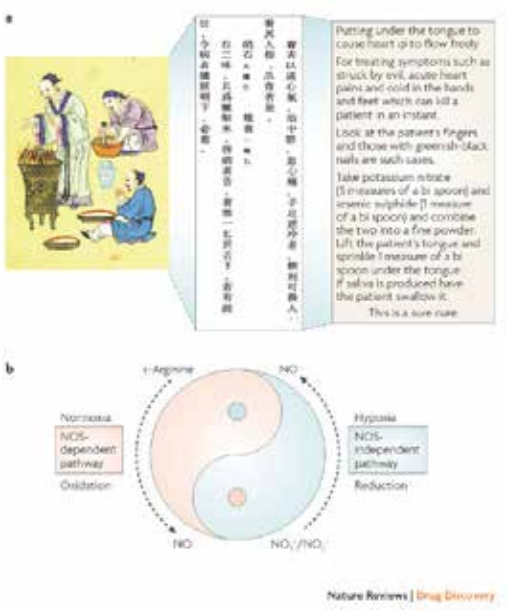
From: Lundberg O, Gladwin M, Weitzberg E.[2]



Human endothelial cells are capable of bio-activating organic nitrates to NO by an enzymatic, apparently thiol-sensitive pathway, in quantities sufficient to influence endothelial and platelet function. So there is a lot to said for good diet.[2]

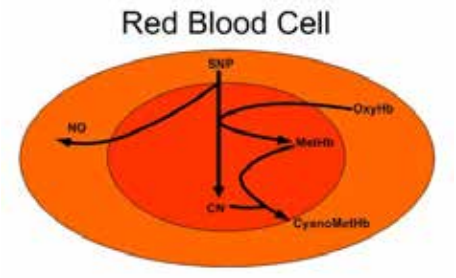
NO donors have unknowingly been used for centuries. The nitrate–nitrite–nitric oxide (NO) pathway has been harnessed therapeutically since the medieval times. This manuscript

illustrates the early appreciation of the effect of nitrate, readily available for meat-curing and gunpowder and reduced to nitrite in saliva, on cardiovascular conditions (angina and digital ischaemia).[3]



From: Lundberg O, Weitzberg E, Gladwin M.[3]

We know that sodium nitroprusside is a source of nitric oxide, but the metabolism of SNP is complicated. Initially within the red blood cell it reacts with oxyhaemoglobin to form NO, five cyanide ions and methaemoglobin. The methaemoglobin may then combine with cyanide to form cyanomethaemoglobin, which is thought to be non-toxic.



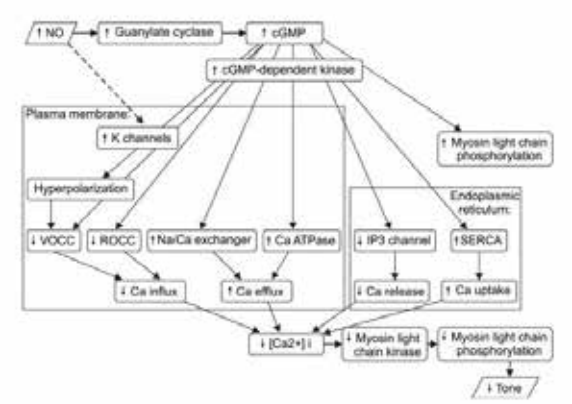
From: Pharmacology for Anaesthesia & Intensive Care. Peck & Hill.[4]

The Ikaria NO delivery system is easily utilised for CPB as it can be titrated into the sweep gas. The Ikaria system measures gas flow and automatically adjust the rate of nitric oxide flow to maintain a set concentration in parts per million.

THE ROLE OF NITRIC OXIDE

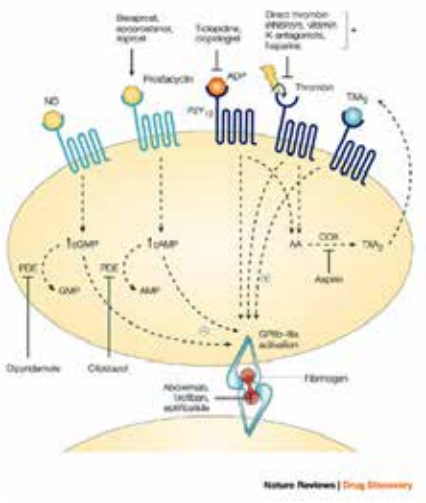
We now know where NO comes from, but what exactly does it do? We know about reduction in PVR, platelet deactivation and leukocyte deactivation, what is less well known in perfusion and cardiac surgery is its role in apoptosis, its role in reduced myocardial and renal injury, and as a neuroprotective agent.

To reduce PVR NO diffuses into the smooth muscle. Once inside the muscle cells, NO is iron and sulphur friendly, which enables it to easily combine with and activate the heme of guanylate cyclase, which raises the concentration level of intracellular cGMP. Intracellular cGMP induces vasodilation via many mechanisms.[5]



Nitric oxide and pulmonary hypertension.[5]

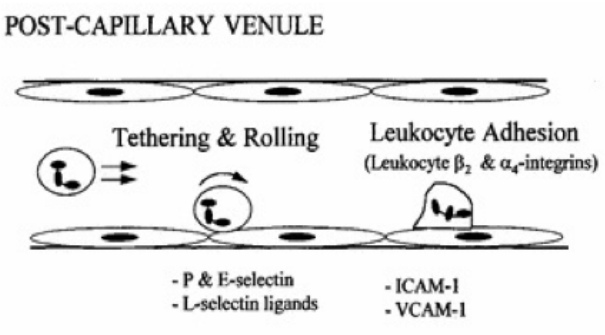
To inhibit platelets nitric oxide enters the platelet cytoplasm, where it stimulates soluble guanylate cyclase. This increases the cGMP levels and consequently inhibits platelet activation, adhesion and granule secretion.[6]



Jackson & Schoenwaelder.[6]

The anti-leukocyte effect of NO has several components. First, NO markedly attenuates leukocyte rolling along the endothelium by inhibiting the expression of P-selectin on the vascular endothelium. Second, NO inhibits the firm adherence of leukocytes to the endothelium. Downregulation of the cell adhesion molecules by NO occurs through inhibition of protein kinase C activation. Third, NO inhibits leukocyte action

by inhibiting the cytoassembly of NADPH oxidase, thereby attenuating the release of superoxide radicals by activated leukocytes, particularly granulocytes.[7]



Nitric Oxide and Leukocyte Adhesion: Experience with NO Inhibitors, NO Donors and iNOS-Deficient Mice [7]

Apoptosis is programmed cellular death. Inappropriate apoptosis (either too little or too much) is a factor in many conditions including neurodegenerative diseases and ischemic damage. This is the crux of the protective properties of NO. NO regulates apoptosis in large variety of cell types, although its effects are dependent on the amount of NO used and the type of cell. It has been shown to both induce and inhibit apoptosis. Nitric oxide inhibits apoptosis in a number of cell types including leukocytes, hepatocytes, trophoblasts and endothelial cells. Nitric oxide is able to affect apoptotic signalling at six points in the pathway:

1) Death receptors are cell surface receptors that transmit apoptotic signals. NO can regulate the expression of the death receptors in a cGMP dependent manner.

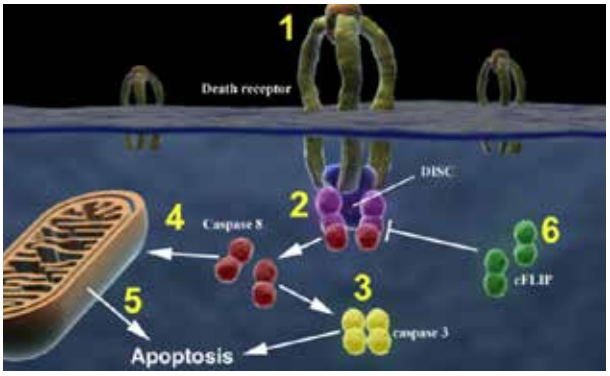
2) NO can alter the expression of proteins that help regulate the early signalling events in death receptor signalling. This reduces DISC (Death Inducing Signalling Complex) formation and is also mediated through the production of cGMP.

3) Caspases are a family of endoproteases that provide important links in cell regulatory networks controlling inflammation and cell death. The activity of the caspases can be directly affected by NO by inhibiting protein function. NO prevents apoptosis in hepatocytes by either directly or indirectly inhibiting caspase-3-like activation via a cGMP-dependent mechanism and by direct inhibition of caspase-3-like activity through protein S-nitrosylation.

4) Caspase-8 has a critical role in determining cellular outcome following death receptor activation. Caspase 8 activates Caspase 3 as well as acting directly on the mitochondria. The effect of NO on caspase activity and DISC formation leads to reduced amplification of apoptotic signalling through the mitochondria.

5) NO can also affect the expression of many members of both pro- and anti-apoptotic proteins.

6) DISC formation can also be affected through the recruitment of the anti-apoptotic protein cFLIP.

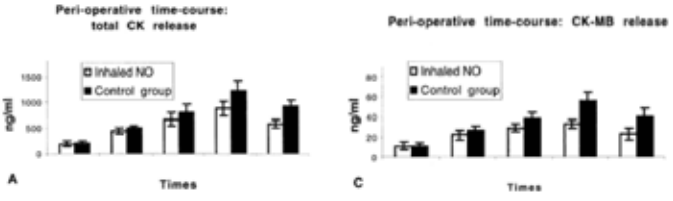


University of Reading: Nitric Oxide Research Group[8]: Role in Apoptosis

In myocardial injury oxygen derived free radicals are primarily involved in ischaemic-reperfusion injury. They exert maximal effect and affect the myocardium at the start of reperfusion, that is, immediately after removal of the aortic cross clamp. Reperfusion injury is multifaceted. It develops over around 2-24 hours and involves 2 specific but related events. The endothelial trigger occurs within a few minutes of re-establishing myocardial blood flow. While neutrophil amplification refers to the migration of neutrophils across the endothelium after leukocyte adhesion and a resultant dramatic increase in neutrophil adherence at about the 20 min mark post reperfusion.

The endothelial trigger and neutrophil amplification lead to the 'no-reflow phenomenon'. No Reflow refers to a state of myocardial tissue hypoperfusion in the presence of a patent epicardial coronary artery. These disturbances culminate in a profound degree of myocardial cell necrosis/apoptosis at 4-24 hour post reperfusion, with a concomitant reduction in myocardial contractility. This is the post-op dive usually seen a couple of hours after admission to ICU.

In a clinical study by Gianetti9 the nitric oxide was administered to each patient during the whole duration of intubation either the oxygenator when on bypass or the ventilator otherwise. The time points in these charts looking at CK and CK-MB release, both markers used to diagnose myocardial infarct, are pre surgery, and 4, 12, 24 and 48 hours post-surgery. You can see clear differences between the two groups 12, 24 and 48 hours post –op.



Supplemental nitric oxide and its effect on myocardial injury and function in patients undergoing cardiac surgery with extracorporeal circulation.[9]

A study by Checchia[10] demonstrated significantly lower troponin and B-type natriuretic peptide levels in patients receiving nitric oxide on bypass. Troponin and natriuretic peptide are markers of myocardial damage and congestive heart failure respectively.

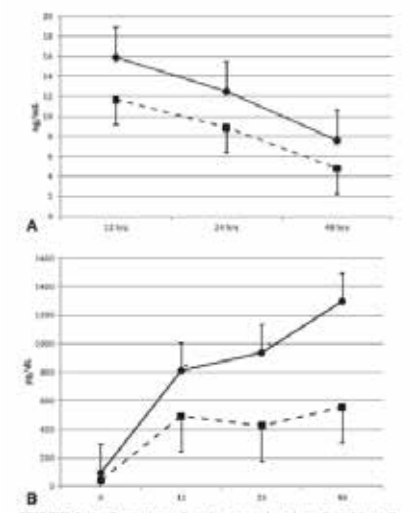


FIGURE 2. A, Comparison of cardiac troponin values. $P < .05$ at all points. B, Comparison of B-type natriuretic peptide measurements. *Solid line*, placebo; *hatched line*, gaseous nitric oxide group. *$P < .05$ at 12- and 24-hour points.

Nitric oxide delivery during cardiopulmonary bypass reduces postoperative mortality in children.[10]

Effects of NO on cardiac arrhythmias has been studied using animal and human subjects, particularly in chronic atrial fibrillation with promising results. Coronary endothelial dysfunction and NOS inhibitors reduce the coronary effluent NO levels and increase the incidence and severity of ventricular arrhythmias in rat models of ischaemia–reperfusion, whereas L-arginine and NO donors, even at subvasodilatory doses, reduce ischaemia/reperfusion-induced ventricular fibrillation (VF) in rats and dogs. Other studies have suggested that nitric oxide under certain influences may facilitate the occurrence of arrhythmias[11].

Cerebral Neuroprotection is linked to the role of nitric in the regulation of cell apoptosis via caspase-3, as well as maintaining cerebral blood flow, preventing neuronal injury, and inhibiting platelet and leukocyte adhesion. The study by Liu[12] showed NO donors exerted a neuroprotective effect against cerebral ischemia-reperfusion injury at different levels by influencing cellular oxidative status. Pre and postischemic administration of an NO donor attenuates the ischemia-induced increase of caspase-3 at 6 h of reperfusion and downregulates neuronal apoptosis.

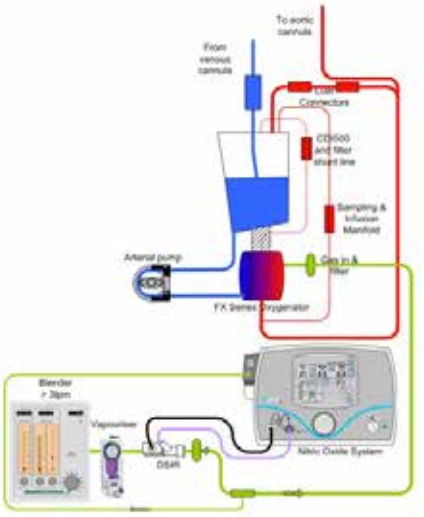
In a study, presented at the American Heart Association 2015 Scientific Sessions, Dr Lorenzo Berra[13] reported that the administration of nitric oxide during extended cardiopulmonary bypass for patients undergoing surgical replacement of multiple heart valves significantly reduced acute kidney injury from 63% to 50% (p=0.04). He stated that prolonged cardiopulmonary bypass produces high levels of plasma ferrous oxyhemoglobin, which depletes vascular nitric oxide. The administration of exogenous nitric oxide, on the other hand, causes the oxidation of ferrous oxyhemoglobin and would decrease the risk of kidney injury by reducing the plasma depletion of nitric oxide.

The RCH Study

The RCH conducted a randomised blinded study looking at ICU outcomes related to nitric oxide in the oxygenator sweep gas during bypass.[14]

- 198 patients randomised to NO or control.
- All patients < 1.5 lpm blood flow on bypass
- Changes to normal sweep gas flow
- NO 20 ppm
- ICU blinded

The nitric oxide was easily added to our bypass circuit, the only issue being that sweep gas flow had to be more than 3lpm. To facilitate this we titrated CO2 into the sweep gas and monitored it via the Terumo CDI500 aiming at normal levels for alpha stat blood gas management.



The primary outcome studied was LCOS (low cardiac output syndrome)

A diagnosis of LCOS, defined as any of the following at any time during the first 48 hours:

- lactate > 4 mmol/l with central venous saturation level < 60% (or SaO2-ScvO2 difference >35% with single ventricle physiology)
- ECMO support
- Vasoactive Inotrope Score (VIS) >10

The majority of LCOS patients (89%) were diagnosed on the basis of high inotrope requirement. No patient was diagnosed with LCOS on the basis of a high lactate and low ScvO2 alone.

The vasoactive inotrope score is an index of cumulative inotropes and dose rates.

Secondary outcomes were a mix of parameters that can also indicate slow recovery from bypass. They included use of inhaled NO, use of peritoneal dialysis, delayed sternal closure, blood loss, transfusion of blood products, the duration of mechanical ventilation, PICU length of stay and hospital length of stay.

Using nitric oxide the overall rate of LCOS was halved. The observed reduction in LCOS was age-dependant, with no effect evident in the oldest of the pre-defined age categories. This finding is not surprising, given that LCOS is most common in younger children. Younger children have an exaggerated inflammatory response to CPB, a microcirculation that is predisposed to capillary leak and limited cardiac reserve, making them particularly vulnerable in the post-operative period.

RACHS is Risk-Adjusted Congenital Heart Surgery, the higher the number the greater the risk of in house mortality. NO shows more benefit in sicker patients.

Incidence of LCOS within first 48 hours of ICU admission between treatment groups

	Nitric Oxide n=101	Control n=97	p-value
All Patients n=197	15/101 (15%)	30/97 (30%)	0.007
Age subgroups < 6 weeks n=57	6/30 (20%)	14/27 (52%)	0.012
6 weeks – 2 years n=76	2/33 (6%)	10/43 (24%)	0.026
>2 years n=65	7/36 (19%)	6/29 (21%)	0.901
RACHS classification 1-3 n=146	10 (14%)	19 (26%)	0.074
4-6 n=52	5/29 (17%)	11/23 (48%)	0.018

The clinical impact of LCOS is peritoneal dialysis rates increase, use of nitric oxide for reduction in PVR is higher, and duration of ventilation, ICU stay and hospital stay are prolonged with respect to non LCOS patients.

	LCOS n=45	No LCOS n=153
Peritoneal dialysis (%)	23 (51%)	24 (16%)
Inhaled NO (%)	13 (30)	2 (1%)
Duration of Ventilation (hours)	72.0 (26.0-163.0)	18.0 (0.0-44.0)
PICU stay (hours)	144.0 (70.0-235.0)	46.0 (24.0-96.0)
Hospital stay (days)	17.0 (10.0-23.0)	8.0 (5.0-15.0)

The cost impact of LCOS can be based on the difference in care required. PICU stay is approximately $4500 per day compared to a shared ward stay cost of $810 per day. The cost of peritoneal dialysis and ventilator support are both about $1500 per day. Nitric Oxide costs $150 per hour.

The bottom line of:

LCOS cost per 100 patients without NO

- 1 day PD + 5.8 days ICU + 5 days Ventilator + 2 days shared ward stay

  = ($1500 + $26100 + $7500 + 1620) x 30 patients

  = $1,101,600

LCOS cost per 100 patients with NO

= $ 550,800 (15 patients)

Cost of NO = 100 x $150 x 3 (Hours CPB)

= $450,000

Saving $100,800 per 100 patients

At its worst it is probably cost neutral, at best saving about $100,000 per 100 patients, not including the added throughput made possible by reducing overall length of stay. As age increases the financial benefit reduces. RCH has approx. 350 open heart procedures on patients under 2 years of age per year. This is a potential cost saving of $350,000 per annum.

As there appears to be benefit we will start with NO for every patient. However, any MAPCA or anatomical shunt may be affected by NO. In some cases there is increased return of blood to the operative field, making surgery difficult. If it is an issue we turn the nitric off and the problem usually resolves.

Currently we are organising a multicentre international trial to look further into the use of nitric oxide on bypass,

Conclusion

Ischaemia, reperfusion injury and neutrophil activation during bypass play a pivotal role in development of oxidative stress and the associated activation of pro-inflammatory and pro-apoptotic signalling pathways which can affect the function and recovery of multiple organs such as the myocardium, lungs, brain and kidneys. Administration of 20 parts per million of nitric oxide to the cardiopulmonary bypass oxygenator during paediatric cardiac surgery reduced the incidence of post-operative low cardiac output syndrome. This effect was age-dependent, with the greatest effect observed in younger children. We believe that this safe and relatively simple intervention may improve short-term outcomes for children under 2 years of age undergoing cardiac surgery and has the potential to reduce to cost per admission of these patients. There may also be benefit to adult patients.

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THE USE OF PERIOPERATIVE ECMO DURING THE REMOVAL AN INVASIVE NECK MASS IN A PATIENT WHERE INTUBATION WAS NOT POSSIBLE

by Mark Adams, CCP

Abstract

The utilisation of ECMO for the safe removal of invasive throat tumours is well documented. Here is a case of a 27 year old male with a history of progressive voice harshness and worsening stridor. It was found to be a large mediastinal tumour compressing the trachea. Without the ability to safely intubate, the patient was placed on VA ECMO and stabilised allowing convenient and safe removal. The patient was then weaned from ECMO incident free with a total support time of 135 minutes.

Introduction

Modern anaesthetic techniques in the general theatre environment make the inability to intubate a patient due to an invasive mass or contusion a rare occurrence. Combined with unfamiliarity and inexperience of cardiothoracic systems is a potentially fatal combination. So the challenge is to provide support during removal without potentially causing dangerous bleeding issues. Extracorporeal Membrane Oxygenation provides an excellent solution and has been well documented is this situation.

Discussion

A 27 year old male heavy smoker presented to the Royal Adelaide Hospital with a one month history of progressive voice harshness and a three week history of worsening stridor. Patient had a previous lung infection complicated with asthma treated with antibiotics and Ventolin. His partner reported some unintentional weight loss. Patient also noted some difficulty with swallowing but not with fluids. The patient was admitted with a suspected thyroid tumour.

On examination, patient was found to have a large right mediastinal neck mass with significant compression of the trachea. The large mass was extending to the inferior aspect of the right hemi thyroid. It appeared to be stuck to the trachea and well vasculated. The decision was made to progress to an operation. It was planned to be a neck dissection with transcervical access for mass removal. At this point, cardiac was called into general theatres with ECMO standby in anticipation of a difficult airway.

A rigid bronchoscopy was performed and there was an inability to pass beyond the inferior stenosed section of the trachea. On CT scan, the narrowest segment was approximately 4mm starting at the first trachea. The airway was found to be not suitable for fibre optic intubation and collar insertion ECMO was requested. After some discussion, the patient was moved to the Cardiothoracic Surgical Unit for femoral ECMO insertion as it was decided that a neck located ECMO cannula could possibly complicate the surgical site.

The patient was lightly sedated while self-ventilating. A cut down was performed to expose the right femoral artery and vein. After the administration of 10000IU heparin purse string sutures were place on both vessels and cannulated over guide wires.

ECMO was initiated uneventfully with MAQUET Bioline Coating 19fr Arterial HLS cannula, 25fr Venous HLS Cannula and a modified PLS set Plus (BE-PLS 2051). Flows of 4.0 -4.5 l/m were maintained at approx. 3800rpm. Once ECMO was stable, the patient was draped and prepared for the surgery to remove the neck mass. At this point, there was a noticeable drop in cerebral saturations. It was assumed that since the heart was still ejecting that there may be some issues with competitive flow. So the Anaesthetist decided to gently hand ventilate the patient with a mask if required. ACT were regularly performed and kept above 180seconds. A Fresenius CATS cell saver system was utilised but no product was returned to the patient.

The mass was successfully removed. It was found to be a neuroendocrine small cell carcinoma and was located close to the thyroid right pole progressing retrosternally. It was 49 by 67mm and up to 80mm thick. The patient was then weaned off ECMO without incident, and the vessels over sown. The leg was closed in layers. The total ECMO time was 135mins with no complications

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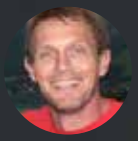

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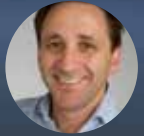

Associate Professor Vin Pellegrino
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CIRCULATING AND URINARY MIR-210 AND MIR-16 INCREASE DURING CARDIAC SURGERY UTILISING CARDIOPULMONARY BYPASS

by
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Presented at the 33rd Annual Scientific Meeting, Australia and New Zealand College of Perfusionists, Townsville, Australia.

Abstract

Aims: To measure and compare blood and urine miR-210 and miR-16 in patients undergoing cardiac surgery with cardiopulmonary bypass (CPB) and off-pump coronary artery bypass grafting surgery.

Methods: Frequent serial blood and urine samples were taken from patients undergoing cardiac surgery with CPB, (n=10) and undergoing off-pump cardiac surgery (n=5) before, during and after surgery. Circulating miR-210 and miR-16 levels were determined by relative quantification RT-PCR. Levels of plasma free haemoglobin, troponin, creatine kinase and creatinine were also measured.

Results: Perioperative serum miR-210 and miR-16 were elevated significantly compared to preoperative levels in patients undergoing cardiac surgery with CPB (CPB versus Pre Op and Rewarm versus Pre Op; $p<0.05$ for both). There were increases greater than 200% in miR-210 levels during rewarming and immediately postoperatively and a 3000% increase in miR-16 levels immediately postoperatively in urine normalised to urinary creatinine concentration. Serum levels of miR-16 were relatively constant during off-pump surgery. miR-210 levels increased significantly in off-pump patients perioperatively ($P<0.05$ Octopus on versus Pre Op), however the release was less marked when compared to cardiac surgery with CPB. A significant association was observed between both miR-16 and miR-210 and plasma free haemoglobin when CPB was used ($r=-0.549$, $p<0.0001$ and $r=-0.463$, $p<0.0001$ respectively).

Conclusions: Serum and urine concentrations of hypoxically regulated miR-210 and haemolysis associated miR-16 increased in cardiac surgery utilising CPB compared to off-pump surgery. These molecules may have utility in indicating severity of cardiac, red cell and renal injury during cardiac surgery.

Introduction

MicroRNAs (miRNAs) are small RNA molecules that can regulate hundreds of genes, and are involved in diverse biological processes including cellular differentiation, proliferation, angiogenesis and apoptosis [(1)]. miRNAs are resilient to degradation, stable in blood, urine and other bodily fluids and are emerging as novel biomarkers reflecting disease states [(2)].

An increasing number of studies have described changes in miRNA expression and explored their role in myocardial ischaemia in relation to coronary artery disease (CAD), myocardial infarction (MI) and ischaemia-reperfusion injury. Specific cardiac expressed circulating miRNAs, including miR-1 and miR-126, increase in patients with CAD and following MI [(3), (4), (5)].

Cardiac surgery provides a unique setting to observe levels of circulating miRNAs in response to a known ischaemic and operative insult. The use of cardiopulmonary bypass (CPB) has been associated with a variable degree of myocardial damage and other organ ischaemia. The extent to which operative, ischaemic and hypoxic stresses during cardiac surgery utilising cardiopulmonary bypass cause alterations in specific or total circulating miRNAs has not been fully explored.

Levels of miR-210 increase during hypoxia in many cells, including myocardial cells and play an important role in myocardial ischaemia-reperfusion injury [(6), (7)]. miR-210 represents major hypoxia-inducible miRs and is ubiquitously expressed with many functions including inhibition of cell proliferation and hypoxia induced cell cycle arrest [(8), (9), (7)]. Animal studies have shown miR-210 overexpression improves cardiac function and reduces injury after myocardial infarction, suggesting a potential therapeutic application for miR-210 in ischaemic heart disease [(10)].

miR-210 has also been investigated in patients with acute kidney injury (AKI), with levels increased amongst critically ill patients and miR-210 was an independent predictor of mortality, potentially reflecting release from renal cells in response to hypoxia (11). Impairment of renal perfusion and oxygenation during CPB may play a central role in acute kidney injury. Circulating biomarkers of renal hypoxia such as miR-210 could provide important insights into the aetiology and prediction of AKI following cardiac surgery.

Levels of miR-16 are often used in miRNA studies due to its consistent abundance in blood. The major release of miRNAs

including miR-16 from red blood cells during haemolysis has been reported [12]. During cardiac surgery, haemolysis is generally attributed to CPB and the CPB related stresses applied to red blood cells within the perfusion circuit. Venous, arterial and suction cannulae, roller pumps, oxygenators and the air to blood interface in the reservoir cause haemolysis and the intravascular rupture of RBC with plasmatic release of free haemoglobin with a haemolysis peak soon after CPB weaning [13]. Increasing evidence suggests that CPB-induced haemolysis may exacerbate kidney injury following cardiac surgery [14], [15].

The technique of "off-pump coronary artery bypass grafting surgery" (OPCABG) was developed in attempts to ameliorate the perioperative complications associated with CPB. The OPCABG technique allows the heart to continue beating and maintain systemic circulation, using a device to stabilize the heart during coronary grafting, thus offering more physiological pulsatile renal perfusion. However, large multicentre trials and meta-analyses suggest that while there may be benefit in short term outcomes, including reduced cerebrovascular and renal injury, longer term benefits have not been seen [12, 13].

In this study, we have examined in detail whether levels of hypoxically regulated miR-210 and levels of haemolysis susceptible miR-16 increase in the blood and urine of patients before, during and after cardiac surgery utilising both CPB and OPCABG. Investigation of the levels of miRNAs will determine the extent to which the stresses of cardiac surgery causes alterations in miRNAs and the extent to which cardiac surgery utilising both CPB and OPCABG differentially affect these circulating and urinary miRNAs.

Materials and Methods

Patient inclusion

Ten patients undergoing elective cardiac surgery with CPB were studied (4 patients isolated CABG, 4 isolated valves and 2 valve and CABG). Blood and urine samples were obtained at eleven time points including a pre-operative sample (prior to skin incision), 10 min following initiation of CPB, during the rewarming phase of CPB (Rewarm), post-operatively at skin closure and at 4, 6, 8, 10, 12, 18 and 24 h in ICU.

Five patients undergoing OPCABG were studied with ten blood and urine samples including pre-operative (prior to skin incision), 5 mins following application of the Octopus® for 1st graft, post-operatively at skin closure and at 4, 6, 8, 10, 12, 18 and 24 h in ICU.

The study was approved by the Southern Adelaide Clinical Human Research Ethics Committee (SAC HREC 202.13) and written informed consent was obtained.

On-pump procedure

Intravenous heparin (300 IU/kg) was administered immediately before cannulation for CPB and additional doses given to maintain an activated clotting time of 400 secs or greater. Cardiopulmonary bypass was instituted by cannulation of the distal ascending aorta and insertion of a single two-stage cannula into the right atrium for CABG and aortic valve replacement (AVR) procedures with bicaval venous cannulation utilised for mitral valve procedures. Roller pumps provided non-pulsatile flow rates of 1.8-2.4 L/min/m2, patient temperature was allowed to drift to maintenance temperature of 34°C and rewarmed to maximum of 36.5°C. Open reservoir membrane oxygenators, cardiotomy suction or cell salvage was used. Myocardial protection utilised blood-cardioplegia administered at ratio of 4:1 (blood:crystalloid) delivered at 34°C.

OPCABG procedure

Intravenous heparin (10000 IU) was administered prior to grafting. The tissue stabiliser (Octopus® Evolution AS TS2500, Medtronic, Minneapolis, MN) was placed to minimise movement in the arterial territory being grafted.

Collection of serum, plasma and urine

Arterial blood (10 mL) was collected from the radial arterial catheter. 5 mL was aliquoted into a silicone coated BD Vacutainer® blood collection tube (Becton, Dickinson and Company, Franklin Lakes, NJ) for serum separation and 4 mL Vacutainer® Plus blood collection K3EDTA tube (Becton, Dickinson and Company) for plasma separation. Blood was allowed to clot at room temperature for 30 min. Samples were centrifuged at 1200 x g for 10 min at room temperature. After transferring serum and plasma supernatants into a 15 mL tube, samples were centrifuged at 1800 x g for 10 min at room temperature to remove debris. Samples were aliquoted into 1.5ml microcentrifuge tubes and stored at -80°C until RNA preparation. Urine (10 mL) was collected from the sample port of the urinary catheter at each time point and stored at -80°C until RNA preparation.

RNA extraction

TRIzol LS Reagent (ThermoFisher Scientific, Waltham, MA) was used to obtain RNA from serum samples spiked with 0.02 pmol of cel-miR-54 RNA (miRBase database accession # MI000025). The pellet was re-suspended in 20 µL of RNase free water and stored at -80°C.

RNA was extracted from urine (1mL) using a Urine Exosome RNA Isolation Kit (Cat No 47200, Norgen Biotek Corp, Thorold, ON, Canada). Urine was spiked with 0.02 pmol of cel-miR-54 RNA. The pellet was re-suspended in 50 µL of RNase free water and stored at -80°C.

Following extraction of RNA, 10 µL of sample was pretreated with 0.5 µL of RNase Inhibitor (Cat No M0307L, New England BioLabs® Inc, Ipswich, MA) and 2 units of Heparinase I (Cat No H2519, Sigma-Aldrich®, St Louis, MO) (in 20 mM Tris-HCl (pH 7.5), 50 mM NaCl, 4mM CaCl2) for 1 h at 25°C to remove heparin (16). Preliminary experiments showed that heparinase does not affect PCR analyses in serum and urine from non-heparinised patients, while allowing unaffected PCR amplification in samples potentially contaminated with heparin.

miRNA real-time PCR

TaqMan miRNA assays (ThermoFisher Scientific) were used for expression profiling. A volume of 2.5 µL of RNase and heparinase treated serum RNA and 2.5 µL of RNase and heparinase treated urine RNA was used for each assay in the 7.5 µ L reaction containing 3.5 µL of master mix and 1.5 µL of reverse transcription (RT) primer.

Assay standard curves were prepared using cDNA generated from cell lines. RNA from the renal cell cancer line (RCC4) over expressing VHL + or control VHL - was used as positive controls for miR-16 and miR-210 respectively.

cDNA was synthesised from 2.5 µL total RNA heparinase-treated serum and urine using miRNA-specific primers according to the TaqMan Assay protocol (hsa-miR-210 Cat # 4427975, hsa-miR-16 Cat # 4440887, cel-miR-54 Cat # 4440887, ThermoFisher Scientific, Waltham, MA). 20ng µL of RCC4 VHL- RNA was used as a control for qRT-PCR investigating levels of miR-210, 20ng of RCC4 VHL+ RNA was used as a control for levels of miR-16 and 20ng of HEK 293 cell RNA spiked with cel-miR-54 was used as a control. Reverse transcription was undertaken at 30 min incubation at 42°C, 5 min incubation at 85°C and incubation at 4°C until ready for use in qRT-PCR.

Real time PCR was carried out according to the TaqMan microRNA assay protocol (Thermo Fisher Scientific) using triplicate reactions for each biological replicate including 1 µL of RT product, 0.5 µL miRNA-specific primer and probe assay mix, 5 µL 1X TaqMan universal PCR Master Mix No AmpErase UNG (Cat # 4324018, Applied Biosystems, Thermo Fisher Scientific, Waltham, MA) and 3.84 µL water. Thermal cycling was performed using a Rotorgene Q (Qiagen®, Foster City, CA) and with 10 min incubation at 95°C, 50 cycles of a 15 sec denaturing step at 95°C and a 60 sec annealing/extension step at 60°C. Relative expression levels were calculated from quantification cycle (Cq) values using Q-gene (17).

Isolation, measurement and quality control of small RNA from serum

The quantity and quality of the RNA extracted from 250 µL of serum was determined using an Agilent Bioanalyzer (Aligent Technologies, Santa Clara, CA) with a Small RNA Analysis Kit. RNA was extracted with the miRCURY RNA Isolation Kits for biofluids (Cat # 300112 Exiqon, Copenhagen, Denmark), and re-suspended in 50 µL of RNase free water. To concentrate RNA, samples underwent a further precipitation by adding 20 µg glycogen, 5 µL of 3M sodium acetate (pH 5.2) and 150 µ L of ethanol. After vortexing samples were incubated at -80°C overnight. The following day, samples were centrifuged at 16 000 x g for 30 min at 4°C. After removing supernatant, 200 µ L of 75% ethanol was added, vortexed and centrifuged at 16 000 xg for 10 min at 4°C. Following removal of the supernatant the pellet was resuspended in 2.5 µl of RNase free water and stored at -80°C.

Plasma-free Haemoglobin

Plasma free haemoglobin (fHb) level measurements were routinely carried out as part of a Serum Indices Test to detect haemolysis, bilirubin and lipaemia on a Roche/Hitachi Modular Analyser (Hitachi High-Technologies Corp. Tokyo, for Roche Diagnostics GmbH, Germany) in the SA Pathology Biochemistry Laboratory, Flinders Medical Centre. Levels of haemolysis were assessed by spectrophotometry with wavelengths scanning from 350 to 650 nm.

Troponin T, Creatine Kinase, Serum Creatinine

Routine creatine kinase and troponin T measurements were carried out preoperatively, 6 hr, 12 hr and 72 hr postoperatively. Serum creatinine levels were determined when clinically assessed.

Statistical Analysis

Expression of miR-16 and miR-210 for each individual was described using the mean of the 3 values as determined by qRT-PCR. In order to adjust for urinary creatinine, each individual value was expressed relative to levels of exogenous cel-miR-54 using quantification cycle (Cq) values from Q-gene. Repeated measures ANOVA was performed with group as a between-subjects factor and time as a within subjects factor. Between group differences at each time point were considered significantly different when the overall time X group effect based on 9 degrees of freedom was significant ($p<0.05$) in addition to a p-value <0.05 at each specific time point. Mann Whitney U test was used to determine differences in preoperative baseline values between the 2 groups. The Spearman correlation coefficient was used for assessing correlations between haemoglobin and miRNA expression. Statistical analyses and graph preparation were performed with GraphPad Prism 6 (GraphPad Software Inc, La Jolla, CA). Global and multiple comparisons adjusted P-values of <0.05 (95% CI) were considered significant. Descriptive data was presented using Box-plots with the whiskers defined using the 25th and 75th percentile ± 1.5 times the inter-quartile range (IQR). Outliers, numbers less than the 25th percentile or greater then the 75th percentile by more than 1.5 times the interquartile range are shown as separately plotted points.

Results

miR-210 and miR-16 in serum of patients undergoing cardiac surgery

Both miR-210 and miR-16 were readily detected in serum by qRT-PCR and baseline levels were similar in on-pump CABG patients and OPCABG patients (0.00087 ± 0.00078 vs 0.00031± 0.00026 for miR-210, p=0.12 and 0.0970±0.0847 vs 0.0695±0.0562, p=0.42). There was a 70% increase in the levels of miR-210 in the samples obtained shortly after the initiation of CPB (Figure 1A) ($p<0.05$), whilst miR-16 increased nearly 200% (Figure 1B) ($p<0.05$). These levels remained significantly elevated for both miRs during rewarming ($p<0.05$) and in the immediate post-operative period ($p<0.05$), returning to pre-operative levels at 4h (p=n.s).

miR-210 levels significantly increased in the sample at the time of placement of the Octopus® ($p<0.05$), and returned to baseline after 4 h (Figure 1C). Levels of miR-16 measured in serum of patients undergoing OPCABG were not significantly altered during or following surgery (Figure 1D).

Using repeated measures ANOVA, for miR-210, there was a significant overall effect of time ($p<0.001$), a significant overall difference between the two groups ($p = 0.017$), but no Group x time interaction effect ($p =0.118$). For miR-16, there was a significant overall effect of time ($p<0.001$), a significant overall difference between the 2 groups ($p = 0.022$) and a significant group x time interaction effect ($p =0.023$).

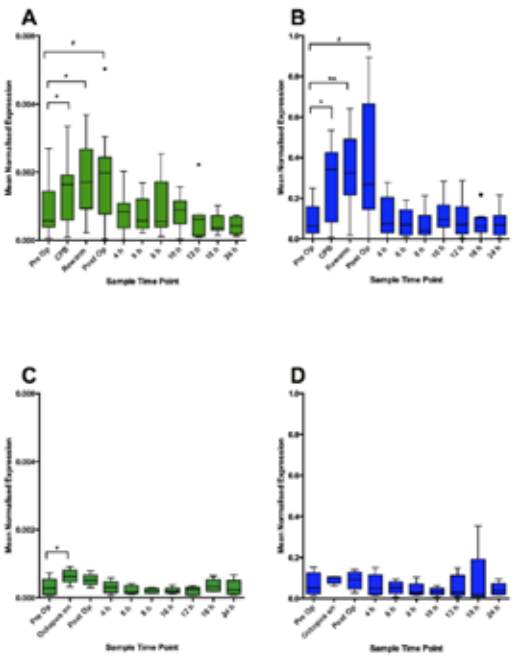

Figure 1 A) Serum miR-210 levels from on-pump patients before, during and after cardiac surgery (*P<0.05 vs. pre-op). B) Serum miR-16 levels from on-pump patients before, during and after cardiac surgery (*P<0.05 vs. pre-op.). C) Serum miR-210 levels from off-pump patients before, during and after OPCABG (*P<0.05 vs. pre-op. D) Serum miR-16 levels from off-pump patients before, during and after OPCABG.

miR-210 and miR-16 in urine of patients undergoing cardiac surgery

Both miR-210 and miR-16 were detectable in the urine of patients undergoing cardiac surgery, but mean concentrations were substantially less than in serum (100-fold less for miR-210 and 700-fold less for miR-16) in on-pump patients (Figure 2A, 3A). Both miR-210 and miR-16 were detectable in the urine of OPCABG patients with the mean concentrations less than in serum of on-pump patients (45-fold for miR-210 and 1000-fold less for miR-16) (Figure 2C, 3C). No changes in urine miR-210 or miR-16 levels were statistically significant. After adjusting for urinary concentration by normalizing with urinary creatinine concentration there was a large increase in miR-210 and miR-16 levels in the urine of on-pump patients. There was a greater than 200% increase in normalized miR-210 levels in both the rewarming phase of CPB and immediately postoperatively (Figure 2B). miR-16 levels increased by 3000% in the urine of on-pump patients in the immediate postoperative period (Figure 3B). There were no changes in patients undergoing OPCABG (Figure 2D, 3D).

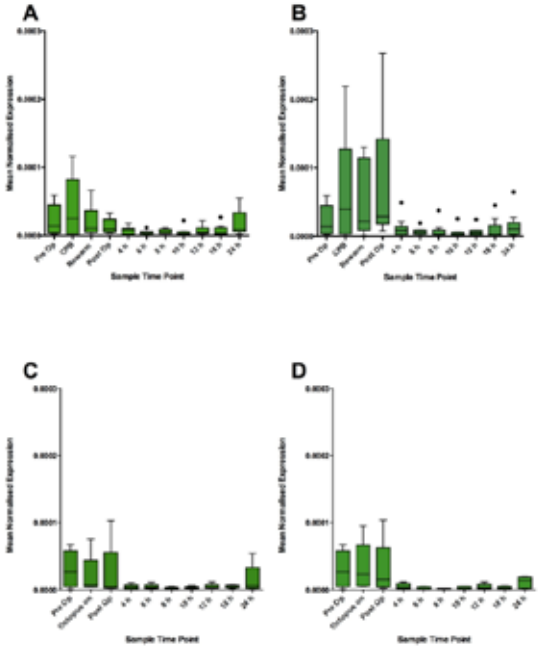

Figure 2 A) miR-210 levels in the urine of on-pump patients before, during and after cardiac surgery. B). miR-210 levels in the urine of on-pump patients before, during and after cardiac surgery when corrected for dilution of CPB using urine creatinine. C) miR-210 levels in the urine of off-pump patients before, during and after cardiac surgery. D). miR-210 levels in the urine of off-pump patients before, during and after cardiac surgery when corrected for dilution of fluid administered using urine creatinine.

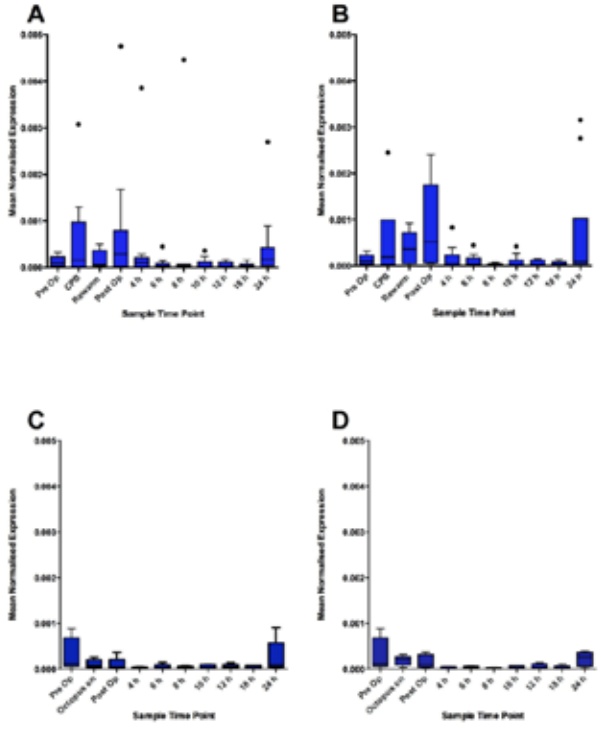

Figure 3 A). miR-16 levels in the urine of on-pump patients before, during and after cardiac surgery. B). miR-16 levels in the urine of on-pump patients before, during and after cardiac surgery when corrected for dilution of CPB using urine

creatinine. C) miR-16 levels in the urine of off-pump patients before, during and after cardiac surgery. D) miR-16 levels in the urine of off-pump patients before, during and after cardiac surgery when corrected for dilution of fluid administered using urine creatinine.

Plasma free haemoglobin levels

Mean plasma fHb levels showed no significant change in either on-pump or OPCABG surgeries (Figure 4). Plasma fHb did however demonstrate a significant positive correlation with levels of miR-16 and miR-210 (Figure 5A). These correlations were not observed in OPCABG patients (Figure 5B).

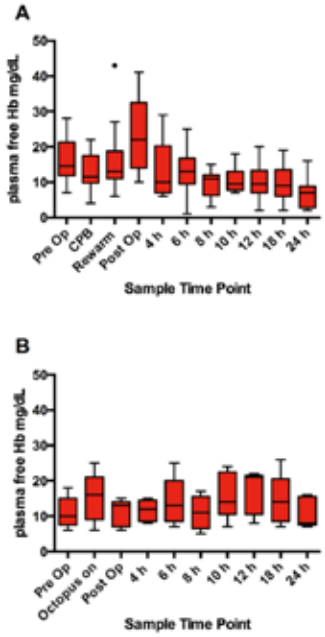

Figure 4. Mean plasma free haemoglobin (Hb) levels in A. Patients undergoing on-pump surgery, B. Patients undergoing off-pump surgery.

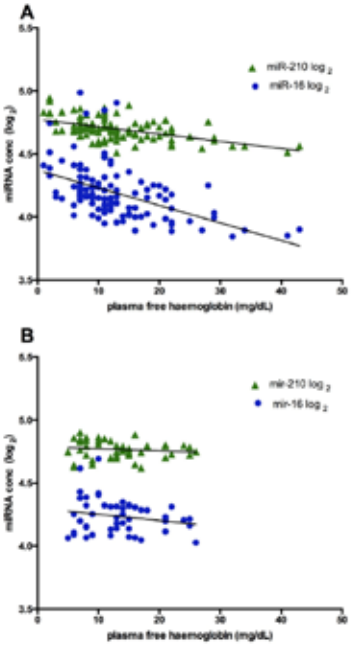

Figure 5. Correlation between plasma free Hb, miR-16 (log2) and miR-210 (log2) in A. Patients undergoing on-pump surgery. Quantitation cycle (Cq) values were transformed to log base 2. Cq values represent the number of cycles needed to reach a set threshold fluorescence signal level. A lower Cq value indicates a higher target miRNA expression in a sample. There is an inverse significant correlation between plasma free Hb and miR-16 levels ($r = -0.549$, P (two-tailed) <0.0001) and plasma free Hb and miR-210 levels ($r = -0.463$, P (two tailed) <0.0001). Line represents line of best fit. B. Patients undergoing off-pump surgery. No correlation between plasma free Hb and miR-16 levels ($r = -0.122$, P (two-tailed) = 0.398) or plasma free Hb and miR-210 levels ($r = -0.181$, P (two tailed) = 0.207).

Injury Markers- Troponin T, Creatine Kinase, Serum Creatinine

In order to examine for links between organ injury and miRNA release, correlations were sought between indicators of myocardial and renal injury and miRNA levels. There were no significant correlations between miR-16 or miR-210 levels with troponin T, creatine kinase or serum creatinine.

Comparison of small RNA levels: on-pump vs off-pump

The total concentration of small RNAs demonstrated a 10-fold mean increase during CPB, with a significant rise seen in the immediate postoperative period, returning to preoperative levels from 4 h postoperatively in on-pump patients (Figure 6A). A substantial increase in total small RNAs was also seen during application of the Octopus®, with levels returning to preoperative levels in the immediate postoperative period, although the rise was not statistically significant (Figure 6B).

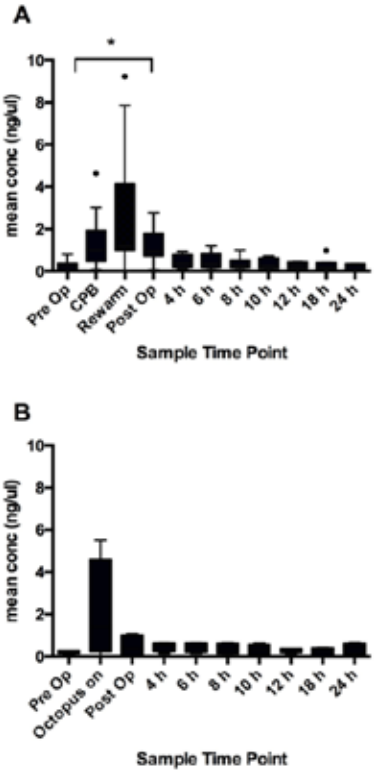

Figure 6. Quantification of small RNAs using Bioanalyzer. Mean serum concentration of miRNA (gated at nucleotide length 18-25 nucleotides. Average nucleotide length 22 nucleotides) in A.

Patients undergoing on-pump surgery (*P<0.05 Post op vs. pre-op). B. Patients undergoing off-pump surgery.

Discussion

Circulating miRNAs were successfully isolated and measured in patients undergoing cardiac surgery during and after CPB. Levels of miR-210, miR-16 and total circulating small RNAs markedly increased in the serum of patients upon initiation of CPB and continued to increase during the rewarming phase of CPB. Levels remained elevated into the immediate postoperative period, and returned to preoperative levels after 4h postoperatively. Levels of miR-210 and total small RNAs increased in the serum of patients during off-pump cardiac surgery but to a lesser degree than patients on CPB. No changes in circulating levels of miR-16 were observed during off-pump surgery.

The major release of miRNAs including miR-16 from red blood cells during haemolysis has been reported and our findings are consistent with haemolytic release of small RNAs during bypass (18). In paediatric patients, haemolysis induced by CPB has been associated with AKI (14). Patients with AKI, as defined by the Acute Kidney Injury Network (AKIN), displayed significantly higher plasma free haemoglobin levels during surgery compared to non-AKI patients (15). Although this study did not show any significant mean changes in plasma fHb during CPB, or in the immediate post CPB period, changes in plasma fHb correlated significantly with changes in detected miR-16 levels. The correlation between plasma fHb and miR-16 levels observed in the on-pump cohort most plausibly reflects exposure of the blood to the extracorporeal circuit with resultant haemolysis. Levels of plasma fHb were also associated with changes in miR-210 which may also be attributed to haemolysis (18). The absence of a significant mean increase in fHb during CPB may reflect the poor sensitivity of fHb to detect haemolysis at low levels of red cell damage in part due to quenching by haptoglobin and raises the possibility that miR-16 release might be a more sensitive measure of hemolysis or a reflection of sublethal damage to the red blood cell membrane not reflected in plasma free haemoglobin levels (19). In contrast, levels of miR-16 and plasma fHb remained relatively constant in the off-pump cohort.

Levels of miR-210 were also significantly increased during the period following application of the Octopus® during OPCABG. During off-pump surgery, it had been thought that the constant source of myocardial blood flow offered greater protection than conventional on-pump surgery. However, the surgical and mechanical trauma to the heart during OPCABG is sufficient to activate an inflammatory response in the myocardium with increases in lactate, creatine kinase MB (CKMB), troponin I and interleukin 6 during OPCABG (20) (21) (22), and the risks of ischaemia and renal injury are not eliminated (20). Hence the release of miR-210 observed during the grafting period of OPCABG surgery might be due to cardiac ischaemia or an effect of a decrease in cardiac output as the beating heart is manipulated, eliciting a hypoxic stress response from other organ systems. To help determine if the insult of surgery itself contributes to the increase observed in circulating miRNAs, samples were also taken following sternotomy, prior to the administration of heparin and initiation of CPB. Levels of miRNAs remained unchanged from pre-operative levels (results not shown).

The CPB period presents a potentially challenging situation for RNA determination because patients are systemically heparinised (300 IU/kg). Heparin has been shown to influence the results of qRT-PCR analysis interfering with amplification of PCR. Treatment of samples with heparinase degrades the heparin allowing successful PCR amplification and determination of miRNA levels (16). Furthermore, others have reported that the exposure to heparin does not have an effect on circulating levels of miRNAs (11).

Urine concentration of miR-210 and miR-16 (when corrected for the effects of urine dilution during CPB) were increased in patients during CPB, and in the immediate postoperative period, returning to preoperative levels after 4h postoperatively. The elevations of miRNA levels during CPB were particularly striking when urine concentration was accounted for by measurement of urinary creatinine (23). Levels of miR-16 detected in the urine of patients undergoing CPB increased almost 3000%. Levels of miR-210 showed a similar release in the urine of the OPCABG group, however when corrected for urinary creatinine concentration the release was less marked than the on-pump group. This may reflect inadequate perfusion of the kidney during the perioperative period leading to either elevated miR-210 production by the kidney in response to hypoxia, or increased circulating miR-210 being eliminated via the kidneys.

During the course of this work, Emanueli et al reported that the concentration of cardiac-enriched ischaemia-responsive miRNAs, including miR-210, increased in the plasma early after CABG surgery (6). We have confirmed this finding and extended it to demonstrate the release of miRNAs during the perioperative CPB period. In addition the profile in relation to OPCABG has not been elucidated previously.

Cardiac surgery is associated with variable degrees of myocardial damage related to the use of CPB and the ischemia-reperfusion insult of cardiologic arrest (6). In this study we focussed on miR-210 because of its established induction by hypoxia. In cardiomyocytes, miR-210 exerts cytoprotective effects during hypoxia (7). In a murine model of myocardial infarction, miR-210 was shown to improve angiogenesis, inhibit apoptosis and improve cardiac function (10). Hence the increase in miR-210 observed during CPB and into the immediate postoperative period could mediate cardioprotective effects and play a role in attenuating the insult of cardioplegic arrest.

These observations raise the question of whether such miRNA release or the associated haemolysis has beneficial or deleterious pathophysiological effects on the kidneys and whether they are predictive of adverse outcomes.

Study Limitations

In this preliminary study a detailed release profile of changes in miRNA release of two specific miRNAs during cardiac surgery was determined in a modest number of patients undergoing on-pump (10) and OPCABG (5) surgeries. We were unable to determine the cellular source of the observed miRNA release.

Conclusions

Levels of miR-210 and miR-16 are increased in the serum and urine of patients undergoing cardiac surgery with cardiopulmonary bypass displaying different release profiles to patients undergoing off-pump surgery. Haemolysis may partially account for such release. These results also indicate an independent accumulation of hypoxically induced miR-210 during on pump surgery with a lesser release observed during off-pump procedures. Levels of miR-210 and miR-16 were also increased in the urine of on-

pump patients compared to off-pump patients suggesting the release of specific miRNAs during surgery potentially related to the operative, ischaemic and hypoxic insult attributed to cardiopulmonary bypass.

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Conflict of Interest

There are no conflicts of interest to be declared.

Disclosures

None

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May 5th & 6th

2017

FRIDAY 5TH MAY

09:30 – 10:20 Registration: Refreshments sponsored by Liva Nova

10:25 – 10:30 Welcome

Session 1 10:30 – 12:00

- Red Cell Salvage Quality and Efficiency

Prof. Jonathan. H. Waters

- Quality of Wash blood "What are we looking for?"

Darryl McMillan

- Audience Survey, What are the current thoughts on the quality testing of returned wash blood.

Jane Ottens

Lunch - 12:00 – 13:00 Sponsored by Haemonectics

Session 2 13:00 – 14:30

- External Qc Testing for TEG and Rotem

Ms Roslyn Bonar Senior Scientist Hematology (RCPAQAP)

- Red Cell Salvage in Pediatric surgery

Mr Killian O'Shaughnessy

- Audience Survey- Current practices in Intra operative blood management

Afternoon tea 14:30 – 15:00 Sponsored by Liva Nova

Session 3 15:00 - 1700

- Red Cell Salvage in obstetrics surgery

Prof. Jonathan. H. Waters

- Treatment perspective – Introducing "strict" protocolised care

Dr. Bruce Cartwright

- Discussion

Trade Area Cocktail Hour 17:00 - 18:00

SATURDAY 6TH MAY

08:00 – 08:50 Registration/Refreshments sponsored by Medtronic

08:50 – 09:00 Welcome/House Keeping

Session 1 09:00 – 10:30

- Back to Basics: The Physiology and Pathophysiology of Blood Volume, Red Cell Mass, Plasma Volume and Hematocrit Regulation

Prof. James Isbister

- Red Cell Salvage's effects on hemostasis

Prof. Jonathan H. Waters

- Discussion

10:30 – 11:00 Morning tea

Session 2 11:00 – 12:30

- "Ironing our iron in the 21st Century"

Prof. James Isbister

- Shock

Dr. Kerry Gunn

- Discussion

12:30 – 13:30 Lunch Sponsored by Medtronic

Session 3 13:30-15:00

- Building systems to reduce blood use

Dr. Kerry Gunn

- Waste associated with the provision of blood

Jonathan. H. Waters

- Discussion

15:00 – 15:30 Afternoon tea

Session 4 15:30 – 17:00

- Current Transfusion practices in Cardiac Perfusion-Collaborative database.

Prof. Rob Baker

- Blood conservation in Cardiac Surgery

Mr. Darryl McMillan

- Discussion

17:00 Closed

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The following pages contain the abstracts from the 3rd Biennial Intraoperative Management of Blood Meeting Australian and New Zealand College of Perfusionists

RED CELL SALVAGE QUALITY AND EFFICIENCY

By Professor Jonathan H Waters

Professor, Departments of Anesthesiology and Bioengineering University of Pittsburgh
Chief of the Division of Anesthesiology at Magee-Womens Hospital of the University of Pittsburgh Medical Center
Vice Chair for Clinical Research for the Department of Anesthesiology
Medical Director for the Acute and Interventional Pain Program at UPMC
Program Director, Acute Pain and Regional Anesthesiology

The process of collecting shed blood and its readministration has been termed blood recovery, cell saving, autotransfusion and blood recovery. Others have suggested the longer phrase, intraoperative blood recovery and reinfusion. This lecture will address the intraoperative, washed blood recovery process. If this technique is used appropriately, several blood volumes of red cells can be recovered and readministered. In order to properly perform blood recovery and return a safe product to the patient, a sound understanding is needed of the components of the system and how they work. Mathematical modeling of blood recovery has revealed that small changes in red cell processing efficiency can make large differences in the maximum allowable blood loss that a patient can sustain prior to allogeneic transfusion therapy. These models suggest that a 70 kg patient with a starting hematocrit of 45% can sustain a blood loss of 9,600 ml if a transfusion trigger of 21% is used and blood recovery captures 60% of lost red blood cells. The sustainable blood loss rises to 13,750 ml if 70% red cell recovery is achieved. Multiple factors can influence the efficiency of the cell processing which will be addressed in the lecture.

In addition to optimizing red cells returned to the patient, attention to optimizing the quality of the product readministered is required. In order to optimize the blood quality being returned to patients, the American Association of Blood Banks (AABB) has issued perioperative standards to guide in the manufacture of a salvaged product. In addition, a guidance document is available which instructs the reader in how to comply with these standards. Implementation of these standards is mandatory if blood recovery is to be performed safely. Primary to these guidelines is the requirement for dedicated personnel to operate the equipment. Without this dedication, inadequate washing and concentration of the blood recovered blood can lead to complications such as disseminated intravascular coagulation, or acute renal failure.

This lecture will address the multiple factors which facilitate efficiency and quality of the cells which are returned to a patient.

Waters JH. Lee JS. Karafa MT. A mathematical model of cell salvage efficiency. Anesth Analg 2002;95:1312-7.

Standards for perioperative autologous blood collection and administration. American Association of Blood Banks. 6th Edition. 2015.

Waters JH, Dyga RM, Yazer MH. Guidelines for blood recovery and reinfusion in surgery and trauma. AABB Press. 2010

QUALITY OF RETURNED WASHED SALVAGED RED CELLS: WHAT ARE WE LOOKING FOR?

by Mr. Darryl McMillan

Cardiopulmonary Perfusion and Autotransfusion Unit, Department of Anaesthesia and Pain Management, Royal North Shore Hospital of Sydney, Australia

Cell salvage has been used since the early 80's and yet there are no clear guidelines as to the definition of the quality of the washed and concentrated red blood cells that are returned to the patient from the shed blood in the operative field.

By washing this shed blood, we are endeavoring to remove potentially harmful cellular and biochemical contaminants (including activated leucocytes, plasma free haemoglobin [Hb], and anticoagulant heparin), while reducing the need to expose the patient to the risk of homologous blood transfusions.

In 2014 the National Blood Authority gave recommendations for the quality of salvage blood, stating that: 'The institution should determine the frequency of blood sampling sent to the Pathology department for quality assurance testing'. The tests recommended include a full blood count and blood cultures; neither of which can be performed easily. Consequently, a search is being made for a more practical and rapid assessment of the quality of processed blood by exploring alternative markers.

Szpisjak et al. showed that potassium can be used as a surrogate marker of cell debris in cell salvaged blood. They found that a potassium less than 2 mmol/L correlates with a 78% elimination of leukocytes. Furthermore, a 1 liter wash for a 225 mL bowl reduced the heparin content by 99%. However, potassium was found to be a poor correlate of plasma free Hb. Nevertheless, Peng Dong et al., when examining calcium as a biochemical marker to evaluate the quality control of cell salvaged blood, found it to be significantly associated with amount of plasma free Hb.

Determining potassium and calcium levels may be a practical method to monitor the quality of the returned washed salvaged red cells, as most operating theatres have access to blood gas machines that can perform both measurements.

EXTERNAL QUALITY ASSURANCE FOR THE TEG AND ROTEM POINT OF CARE INSTRUMENTS

by Ms. Roslyn Bonar

The Royal College of Pathologists of Australasia (RCPAQAP) is an External Quality Assurance (EQA) program that has both national and international subscribers.

External Quality Assurance (EQA) for point of care (POC) instruments such as the thromboelastography (TEG) and rotational thromboelastometry (ROTEM) has proved challenging. Due to the nature of the testing, an alternative approach to EQA was developed. After a successful trial for both the TEG and ROTEM instruments, QA modules have been introduced in 2017 for the TEG and in 2016 for the ROTEM.

Surveys are performed twice a year with 2 lyophilised plasma samples per survey. Samples are custom made to represent different scenarios experienced by the operators. Worksheets are provided with detailed instruction on how to perform the testing.

Results are presented in the form of dot plots accompanied with a table containing the statistical analysis for each sample. Participants are able to compare their results against their peers for both the cartridge and reagent systems.

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RED CELL SALVAGE IN OBSTETRICS

by Professor Jonathan H Waters

Professor, Departments of Anesthesiology and Bioengineering University of Pittsburgh
Chief of the Division of Anesthesiology at Magee-Womens Hospital of the University of Pittsburgh Medical Center
Vice Chair for Clinical Research for the Department of Anesthesiology
Medical Director for the Acute and Interventional Pain Program at UPMC
Program Director, Acute Pain and Regional Anesthesiology

Peripartum hemorrhage is the leading cause of death in pregnancy worldwide. In the third world, risk of maternal death from post-partum hemorrhage is approximately 1/1000 deliveries.1 Risk of death during childbirth in the US has been rising steadily with 17.8 deaths per 100,000 live births in 2011. Of these deaths, 11.3% can be attributed to hemorrhage.[2]

The use of intraoperative blood salvage would naturally be attractive in this setting. Use of intraoperative blood salvage in obstetrics is classically contraindicated; however, little data are available to substantiate this contraindication. In fact, significant data exists which supports the use of cell salvage in obstetrics. This contraindication arises from a fear that shed blood can be contaminated with amniotic fluid, and readministration may lead to an iatrogenic amniotic fluid embolism. Fortunately, or unfortunately, the incidence of amniotic fluid embolism is so rare a study to demonstrate safety of intraoperative blood salvage use would require a study incorporating 1.7 million patients. Since this sample size is unrealistic, an evaluation of what we do know about intraoperative blood salvage use along with an evaluation of the alternative therapy, allogeneic transfusion, is warranted.

This talk will focus on the available safety data associated with applying salvage in the environment of obstetrical hemorrhage.

Abou Zahr C., Royston E. Global Mortality. Global Fact-book. Geneva: WHO, 1991.

[2] Pregnancy Mortality Surveillance System. Center for Disease Control and Prevention. http://www.cdc.gov/reproductivehealth/MaternalInfantHealth/PMSS.html (accessed February 16, 2015).

BACK TO BASICS: THE PHYSIOLOGY AND PATHOPHYSIOLOGY OF BLOOD VOLUME, RED CELL MASS, PLASMA VOLUME AND HEMATOCRIT REGULATION

by Professor James Isbister

Clinical Professor of Medicine, Sydney Medical School
Emeritus Consultant Physician, Royal North Shore Hospital
Conjoint Professor of Medicine, University of NSW
Adjunct Professor of Medicine, Monash University
james.isbister@sydney.edu.au

The maintenance of adequate microcirculatory function requires circulation of blood of the appropriate composition achieved by an effective cardiac output which in turn is dependent on an adequate intravascular blood volume and its return to the heart.

Factors regulating the relationships between red cell mass and plasma volume in determining the haematocrit are complex. Most scientific literature relating to total blood volume is found in relation to shock and resuscitation, the plasma volume literature relates to salt and water homeostasis and hypertension, and red cell mass literature relates to haematological disorders. The casual observer could be excused for thinking that red cell mass and plasma volume are independent variables.

The haematocrit of blood in macrocirculation is higher that calculated from measurement of the red cell mass and plasma volume (ie body haematocrit). In a normal stable state the relationship of the body haematocrit to the venous haematocrit is approximately 0.9. This F-cell ratio represents variations in the red cell mass distribution within the vascular space. Physiological haemodilution with a lower haematocrit occurs in the microcirculation.

Sudden requirements for increases or decreases in total intravascular blood volume can only be effectively achieved by alteration in plasma volume as the red cell mass cannot be acutely altered. This is in contrast to when chronic stresses are placed on the system, the red cell mass component of the intravascular blood volume can be appropriately regulated, by increased or decreased erythropoiesis under the influence of erythropoietin. It is thus evident that acute maintenance of an appropriate relationship between the volume of the intravascular compartment ("the vessel") and the absolute intravascular blood volume ("the fluid") results in acute changes in haematocrit as plasma volume adapts. In some circumstances, these changes in haematocrit of haemoconcentration or haemodilution may be appropriate to the stimulus, whereas in other circumstances, the changes in haematocrit are "unavoidable" by-products to permit a "higher priority" acute volume adaptation to occur. It is important that this is understood by clinicians as important clinical decision are may be made on changes in the haemoglobin level.

The ultimate determinants of total blood volume and haematocrit are complex and remain sub judice. This presentation will focus on the basic physiology of blood volume control. In the short-term, the maintenance of adequate cardiac filling and output is obvious. However, in the overall picture, the total blood volume is probably predominantly determined by the requirement for a reserve to respond to various stresses. In several physiological situations, there are substantial changes in total blood volume and in most cases haematocrit.

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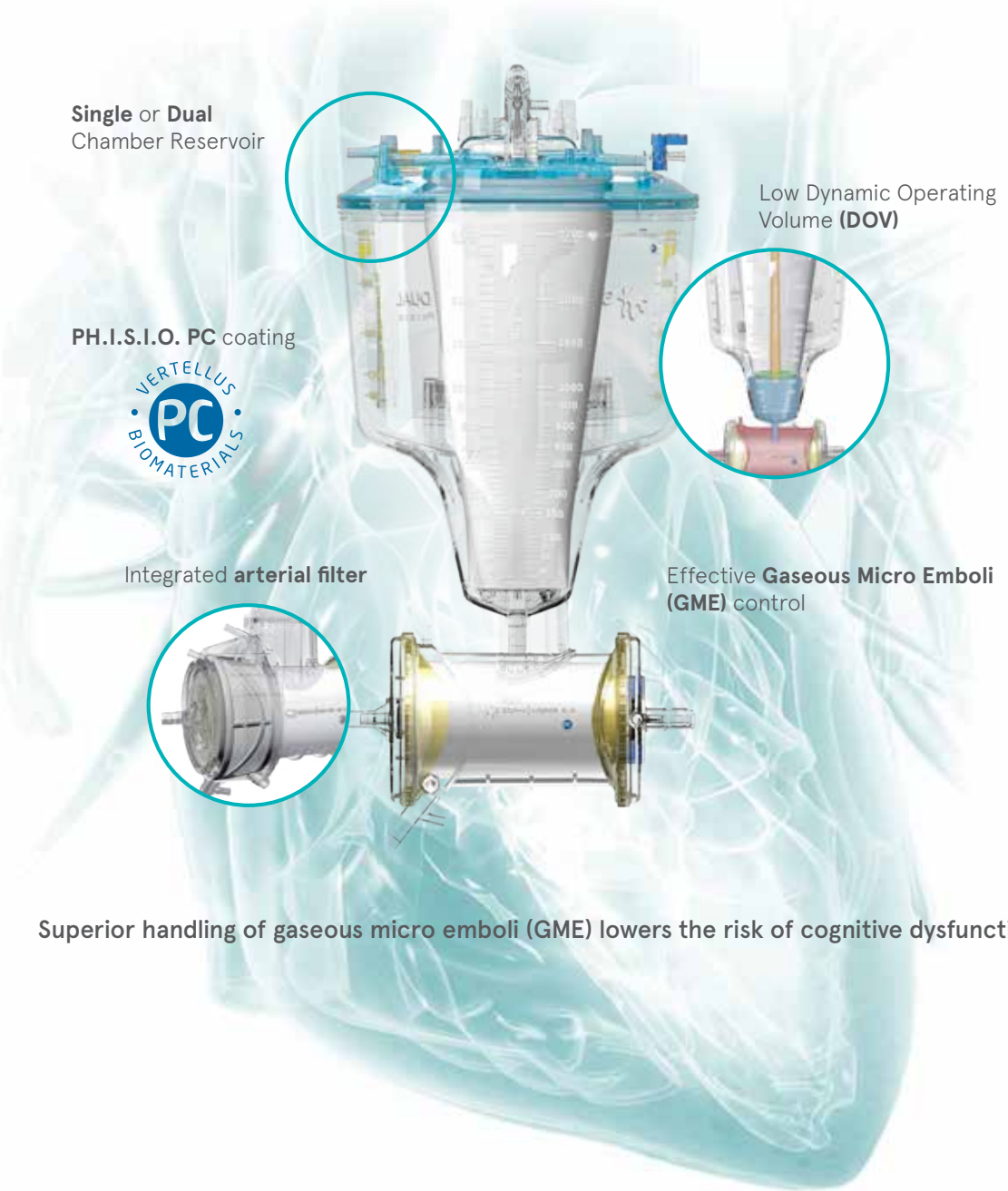

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RED CELL SALVAGE'S EFFECTS ON HAEMOSTASIS

by Professor Jonathan H Waters

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The development of a clinically significant coagulopathy after the infusion of salvaged blood has been a persistent concern. Although it occurred frequently in the past with the ad-ministration of large volumes of unwashed blood, coagulopathy is much less common now that modern cell-salvage instruments are in use. Dilutional coagulopathy may still be seen in patients receiving large amounts of washed RBCs. Yawn1 reported consistent decreases in platelet counts and coagulation-factor levels when more than 6 units of washed RBCs were given to patients undergoing complex aortic-aneurysm repairs.

Bull and Bull[2] described the salvaged-blood syndrome in which patients develop manifestations of increased capillary permeability (such as adult respiratory distress syndrome or anasarca), intravascular coagulation, or a combination of these complications. Their investigations found deposits of platelets and leukocytes in the centrifuge bowls and reinfusion bags of such patients. They postulated that platelets are activated during blood salvage, particularly if the blood has been diluted with significant amounts of saline before aspiration. During the concentration phase of processing, these platelets deposit on the walls of the centrifuge bowl, degranulate, and release procoagulant and leukoattractant substances. The activated leukocytes also adhere to the platelet deposits. With reinfusion, the procoagulant platelet phospholipid and activated phagocytes may initiate disseminated intravascular coagulation, adult respiratory distress syndrome, and anasarca.

In some cases of coagulopathy, additional factors such as acidosis, hypotension, hypothermia, and tissue trauma may contribute to the hemostatic abnormalities. Many of these patients will require supplemental transfusions of platelets and fresh-frozen plasma.

This lecture will focus on these causes of coagulation dysfunction as well as several others.

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2. Bull BS, Bull MH. The salvaged blood syndrome: a sequel to mechanochemical activation of platelets and leukocytes? Blood Cells 1990; 16:5-23.

"IRONING OUT IRON IN THE 21ST CENTURY"

by Professor James Isbister

Clinical Professor of Medicine, Sydney Medical School
Emeritus Consultant Physician, Royal North Shore Hospital
Conjoint Professor of Medicine, University of NSW
Adjunct Professor of Medicine, Monash University
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Patient Blood Management (PBM) is an evidence-based bundle of care to optimise medical and surgical patient outcomes by clinically managing and preserving a patient's blood. In the early days of PBM the main driver was the recognition that allogeneic blood transfusion is a risk factor for adverse clinical outcomes with no solid evidence-base for efficacy in many clinical settings.

The paradigm shift to a patient-focus returned clinicians from a donor blood product focus 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.

Anaemia, and specifically iron deficiency, are commonly not regarded as significant clinical problems and not taken seriously by many clinicians. In countries with developed health systems, it is uncommon to observe patients primarily dying from anaemia. However, from a more global perspective anaemia is a significant risk factor for morbidity and mortality in numerous clinical settings if not addressed appropriately. For many clinicians, anaemia receives a knee jerk response by "ordering" a blood transfusion, somebody else does the work, donor blood has been seen as free and promoted as safe and effective. Many surgeons in the past have regarded the availability of allogeneic blood transfusion as a substitute for poor perioperative blood management and poor surgical technique.

To some extend haematologists also have to bear some responsibility as to why the speciality has in recent years shown limited interest in anaemia and regard it as a problem for "others" to address. Until the early 1960's haematologists showed considerable interest in anaemia and its correct diagnosis and management. As there were few major advances in the understanding and management of iron metabolism and the nutritional anaemia interest waned. At the same time combination chemotherapy for fatal haematological malignancies was revolutionising the management of, and potentially curing, childhood acute leukaemia and Hodgkin's disease. As haematology became a major clinical specialty the focus and interest was naturally on haematological malignancy.

Iron deficiency anaemia has traditionally been classified with the nutritional anaemias and presents as a hypochromic microcytic anaemia. Ironically, iron deficiency in adults is rarely nutritional in origin and the majority of cases of iron deficiency are not anaemic. Commonly, cases of iron deficiency present in the normocytic normochromic phase with or without anaemia.

With the impressive developments in the understanding of iron metabolism over the last 50 years there has been a resurgence of interest in the investigation of patients' iron status. The introduction of ferritin measurements in the early 1970's and the better understanding of the pathophysiology of iron overload resulted in haemochromatosis "dominating" the clinical focus in making iron metabolism no longer "boring". The quite spectacular develops in the molecular genetics and biochemical understanding of iron metabolism and its regulation has stimulated a major reanalysis of the role of iron in the pathophysiology of numerous disease states.

This presentation will focus on current status of the understanding of iron metabolism in relationship to the practice of patient blood management.

SHOCK TRAUMA AND COAGULOPATHY

by Dr. Kerry Gunn

Department of Anaesthesia & Perioperative Medicine, Auckland City Hospital

Trauma has few benefits. But for the study of the response of the human's physiology to shock it provide a unique model to explain changes that have troubled clinicians for decades in understanding why patients continue to bleed when normally they do not.

If a patient has severe trauma defined by evidence of shock and ongoing, uncontrolled bleeding they have a 20% mortality, which increases to 40-50% if in addition they have a coagulopathy. They are 8 times more like to die in the next 24 hrs with a coagulopathy than not, and results from the PROPPR and PROMMTT [1] studies suggest that rapid resuscitation with fibrinogen rich blood products may reduce bleeding, improve short term survival, but not such that in hospital mortality is reduced.

The development of a coagulopathy has been recognised for many years since Cannon [2] recognised the delirious effect of resuscitation of patients with clear fluids in battlefield trauma. The dilutional coagulopathy does not explain the profound blockade in coagulation in shock. Evidence currently points to poorly perfused endothelium, stimulated by a hyper adrenergic sympathetic system exuding thrombomodulin and activated Protein C into the microcirculation.[3] This effects PAI-1 to promote fibrinolysis, inhibit FV and FVII to stimulate thrombin, and thus limit clot forming in the microcirculation. While this may preserve the organ if perfusion is re-established, the systemic effects of this are to induce non-surgical bleeding that increases mortality in the trauma patient.

Thus, and in tandem with this the previously intact glycocalyx is damaged.[4] When large crystalloid resuscitation fluids are used the protein and heparan matrix within the extra-endothelial layer loses its integrity. Fluid loss through the basement membranes increases, and he effectiveness of the circulation is impaired.[5]

Indicators of increased mortality using coagulation parameters show that they are the result of profound shock. Elevated Protein C levels, Syndactin–C levels (indicating glycocalyx destruction) and elevated adrenaline levels all are associated with abnormalities in coagulation parameters (INR, aPPT), and TEG abnormalities.[6] Similar changes in platelet aggregation occur.

The resulting clinical problems are a patient in shock with bleeding from non-surgical wounds, that continues to bleed after the trauma pathology is fixed. This leads to abdominal compartment syndrome, Multisystem organ failure and death.

Empiric responses to this have been a rapid recognition of patients at risk, rapid transport to a definitive site of bleeding control (operating room or interventional radiology), damage control surgery, which involved rapid surgery limited to stopping bleeding, then stopping, Damage control resuscitation which involves limiting crystalloid, empiric use of Tranexamic acid at a dose of 15mg/kg bolus plus an infusion over 1 hrs, blood given in either a 1:1:1 fixed ratio, or targeted to a TEG or ROTEM, and sometimes permissive hypotension. Patients with persistent acidosis and hypothermia are managed in the ICU until stabilised before definitive trauma surgery

Together these bundles of care have reduced mortality form massive haemorrhage in trauma substantially.[7]

The question is where these lessons can be applied in other surgical areas. While the principles are logically applied to any surgery that includes shock and uncontrolled bleeding, in normal high blood loss surgery evidence is lacking to aggressive resuscitation along these lines. A warm, not shocked patient with limited tissue trauma behaves differently and focused therapy is more logical. In Cardiothoracic surgery, the effect of drugs that are anticoagulant and antiplatelet need to be specifically reversed rather an empirically treated.

The concept of Goal directed therapy where abnormalities are corrected only in bleeding patients has the advantage of focussing therapy on laboratory abnormalities. The most validated of these is using a TEG or ROTEM. It further allows treatment with less exposure to allogenic blood products, and less system waste. [8,9] But it usually needs specialist skills and a dedicated person controlling the resuscitation.

The question in the future is if we need to add a person to the team. There has usually been an airway specialist, should we add a bleeding specialist?

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BUILDING SYSTEMS TO REDUCE BLOOD USE

by Dr. Kerry Gunn

Department of Anaesthesia & Perioperative Medicine, Auckland City Hospital

There have been a significant number of papers published in the last years questioning the benefits of liberal transfusion of red cells and coagulation products, but also indicating the place for a more systematic approach to their use. This talk will cover some of the more significant ones.

Limiting Red Cell Use

Many Western countries have seen a reduction in red cell use over the last five years. New Zealand and Australia are experiencing a sharp decline. In New Zealand, a 9.2% reduction of sales occurred from NZBS since 2010, and the reduction shows no signs of stopping.

It is uncertain what the cause of this is, but effective blood management programs, a move to less open and more minimally invasive surgical procedures, more rational management of bleeding, and the use of medicines instead of red cells are certainly a major factor in the reduced demand.

Patients bleeding from gastrointestinal haemorrhage were studied by Callcut who showed a restrictive policy of transfusion improved outcome in patients, with a mortality of 9% at 45 days if transfusion was commenced at a Hb< 90g/L vs a mortality of 5% if the Hb could drop to 70g/L before transfusion. It follows recent analyses of the NQIP surgical database indicating Hb should be supported with a haematocrit of 0.28 if bleeding more than 1,500ml in the case, but 0.24 if there is less than 500ml blood loss. In most cases, less transfusion is more.

Exceptions to this rule seem to be in Acute Coronary Syndrome. A recent small series by Carson's indicated increased mortality in patients with an acute MI having a Hb below 100g/L. This was underpowered to be definitive, but indicates caution in allowing severe anaemia to persist in a patient with chest pain. Similar trends are present in severe sepsis and acute stroke. These however are the exceptions to the general rule that a Hb >70g/L in non-bleeding patients should not need red cell transfusion.

On-going controversy persists with the effect of older blood on outcome. Currently a move to 14 day old blood being disposed of would mean too little red cells being available for patient care. However, if the restrictive transfusion policies continue, 20-25 day expiry may be possible.

Management of Anaemia

A new and exciting area of interest is anaemia. Preoperative anaemia increases perioperative mortality and complication rates. One third of patients present with anaemia if using the WHO criteria (Hb < 130g/L if male, and 120g/L if female). Half of that population has iron deficiency (ferritin < 40g/L) and respond to oral or IV iron. Evidence suggests, with treatment, 80% will raise their Hb by 20g/L in 8 weeks if taking oral iron, or 22 days if given a single dose of IV iron.

If this algorithm is applied to a population that has a perioperative blood loss of 1,000mls or greater this reduces the likelihood of red cell transfusion by 40% (from 52% to 12% in TKJR). This is a logical response to dealing with patients with preoperative anaemia. There may be an increased risk of infection with iron given intraoperatively but this is controversial.

To work effectively preoperative anaemia must be diagnosed and investigated at least one month before surgery is planned. This creates difficulties with most patients only having it noted immediately preoperatively or often never at all. New systems are being developed to assist anaesthetists to manage patients presenting for blood loss surgery to reduce the likelihood of transfusion.

Currently in NZ, iron polymaltose is the only preparation available for total dose, one time IV infusion. It traditionally has been given over a three-hour infusion. Faster rates seem safe. Iron carboxymaltose can be given as a single bolus, and may be available on the PML is costs can be justified.

Systems that use this data to empower clinicians to create better outcome with better targeted therapy with reduced waste are being rolled out over many countries, including impressive results in Australia.

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4. Safety and efficacy of intravenous iron therapy in reducing requirement for allogeneic blood transfusion. Litton and Xaio. BMJ 2013;347:f4822

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WASTE ASSOCIATED WITH THE PROVISION OF BLOOD

by Professor Jonathan H Waters

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Medical Director for the Acute and Interventional Pain Program at UPMC
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Wastage of all blood components, including red blood cells (RBCs), platelets (PLT), and plasma, is an important issue for hospitals worldwide. Waste is not limited to blood products and is present throughout the healthcare system. One study suggested that the overall cost of waste to the American healthcare system was perhaps as high as $910 billion dollars. Studies of systemic waste have examined the importance of workflows in the healthcare environment, and focused on minimizing operational sources of waste when issuing a variety of medications. In many of these studies relatively simple interventions resulted in marked reductions in waste.

Ideally wastage of blood products would never occur. Inevitably a low level of wastage is accepted due to the inherent need to have stock on hand at all times, and the often unpredictable demands on the inventory. Studies of the supply chain and blood usage benchmarking have demonstrated that while some wastage does occur in the blood bank, significant reductions may be achieved through targeted interventions.

Waste associated with the provision of blood comes in many other forms. This lecture will focus on the wide variety of waste that is associated with the provision of blood and blood products.

1. Berwick DM, Hackbarth AD. Eliminating waste in US health care. JAMA : the journal of the American Medical Association 2012;307: 1513-6.

BLOOD CONSERVATION AND PRACTICES IN CARDIAC SURGERY

by Darryl McMillan CCP

Cardiopulmonary Perfusion and Autotransfusion Unit, Department of Anesthesia and Pain Management, Royal North Shore Hospital, Sydney, Australia.

Cardiac surgery has one of the highest uses of blood products in the operating room. Cardiac surgical patients are often transfused due to preoperative anemia, anaesthesia associated haemodilution, and cardiopulmonary pump primes. However, we are constantly reminded in the literature that the benefits of blood transfusions have not been demonstrated, while the evidence for transfusion related harm continues to accumulate. Recently, it has also been shown that the double negative exposure to both red cell transfusions and anemia (Hct <25%) carries the highest morbidity risk. Therefore, managing preoperative anemia more aggressively and implementing measures to avoid operative anemia may circumvent negative exposure to red cell transfusions and improve patient outcomes.

Blood conservation strategies in cardiac surgery can be relatively inexpensive and should start with the preoperative assessment of the patient. Is the patient already anaemic and what is their risk of bleeding? The correct dosage of heparin and protamine is still unresolved. Since the 1950's, there has been many improvements in the cardiopulmonary bypass including the oxygenator and circuit surface coatings. Yet little has changed in what is considered a safe level of heparinisation. Furthermore, the appropriate heparin reversing protamine dosage is unclear. Protamine's role in post-operative bleeding is still being identified as it is an anticoagulant if given in excess. Retrograde autologous priming (RAP) of the pump circuit prior to the initiation of bypass replaces the priming solution with the patient's own blood. RAP is an effective, easy and cost effective way to decrease the haemodilution effect of the pump prime, and has been shown to reduce the rate of red cell transfusions. However, RAP requires a team approach as the surgeon, anaesthetist and perfusionist must work together for the best result.

Blood conservation does not need to use expensive technology but simply requires everyone working together as a team to achieve the same goals and be willing to change practices.

CURRENT TRANSFUSION PRACTICES: INSIGHTS FROM REGISTRY DATA

by Professor Robert A Baker

Director Cardiac Surgery Research and Perfusion,
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Adelaide South Australia

Topic objective:

1. Review and discuss registry data evaluating transfusion as it contributes to the evidence base for post-operative morbidity and mortality

Understanding clinical practice allows the clinician the opportunity to treat every patient appropriately; however what do we do when the science behind our clinical practice does not clearly define the optimal pathway forward. Many publications have evaluated the role of preoperative anaemia and the effect of blood transfusion on patient's morbidity and mortality. In the realm of cardiac surgery the question of causation rather than association is widely discussed with advocates suggesting preoperative anaemia and the patients co-morbidities provide the most significant driver of poor outcome (eg Kulier et al 2007[1]), while others are firmly entrenched with the view that red blood cell transfusion is central to poor outcome (eg Koch et al 2006[2]). Layered upon this is the question of when should transfusion occur.

The national Blood Authority as part of its Patient Blood Management[2] programme has clear recommendations for clinical practice in Australia, in the Perioperative period focusing on Cardiac Surgery these include:

In patients undergoing cardiac surgery, preoperative anaemia should be identified, evaluated and managed to minimise RBC transfusion, which may be associated with an increased risk of morbidity, mortality, ICU length of stay and hospital length of stay (Grade C). In surgical patients with, or at risk of, iron-deficiency anaemia, preoperative oral iron therapy is recommended (Grade B). In patients undergoing CABG either with or without CPB (OPCAB), clopidogrel therapy should be stopped, where possible, at least 5 days before surgery (Grade C). In adult patients undergoing surgery in which substantial blood loss (blood loss of a volume great enough to induce anaemia that would require therapy) is anticipated, the use of ANH should be considered (Grade C). In adult patients undergoing surgery in which substantial blood loss (blood loss of a volume great enough to induce anaemia that would require therapy) is anticipated, intraoperative cell salvage is recommended (Grade C). In adult patients undergoing cardiac surgery, the use of TEG should be considered (Grade C). In adult patients undergoing cardiac surgery, the use of intravenous tranexamic acid is recommended (Grade A).

How well clinicians adhere to recommendations and what practices actually transpire can be tracked from registry data, however registries are rarely established apriori to answer such questions. Whilst these recommendations are actionable, the level of evidence supporting them is not high.

Registry data provides the ability to compare transfusion practices between different centres and allows clinicians to ask the question "What are we doing differently that results in such variations in practice?" The PBM programme has practice recommendations in relation to transfusion:

RBC transfusion should not be dictated by a haemoglobin 'trigger' alone, but should be based on assessment of the patient's clinical status. In the absence of acute myocardial or cerebrovascular ischaemia, postoperative transfusion may be inappropriate for patients with a haemoglobin level of >80 g/L. Patients should not receive a transfusion when the haemoglobin level is ≥100 g/L. In postoperative patients with acute myocardial or cerebrovascular ischaemia and a haemoglobin level of 70–100 g/L, transfusion of a single unit of RBC, followed by reassessment of clinical efficacy, is appropriate.

The Society of Thoracic Surgeons and the Society of Cardiovascular Anesthesiologists has made recommendations in relation to transfusion specific for cardiac surgery[4]. The difficulty lies in understanding how well these recommendations are applied and this is able to be reflected in registry data.

The issues associated with understanding transfusion practices are recognised, registries are well positioned to inform clinicians of their practices.

1. Kulier A, Levin J, Moser R, Rumpold-Seitlinger G, Tudor IC, Snyder-Ramos SA, et al. Impact of preoperative anemia on outcome in patients undergoing coronary artery bypass graft surgery. Circulation. 2007;116:471-9.

2. Koch CG et al Transfusion in Coronary Artery Bypass Grafting is Associated with Reduced Long-Term Survival. Ann Thorac Surg 2006;81:1650-7.

3. National Blood Authority (NBA) (2012) Patient Blood Management Guidelines: Module 2 Perioperative https://www.blood.gov.au/system/files/documents/pbm-module-2.pdf accessed 6th April 2017

4. Ferraris VA, et al. 2011 update to the society of thoracic surgeons and the society of cardiovascular anesthesiologists blood conservation clinical practice guidelines. Annals of Thoracic Surgery: 2011, 91(3), 944-982.

IMOB *2017* PHOTOS

DR BRUCE CARTWRIGHT

DR JONATHAN WATERS

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CALENDAR *of* EVENTS

JUNE 2017

2-4

The 27th Annual Specialists in Education in Extracorporeal Membrane Oxygenation (SEECMO)

Children's Hospital of Colordo

Aurora, Colorado, USA

http://cmetracker.net/CHCOL/Login?FormName=RegLoginLive&Eventid=36148

14-17

17th European Congress on Extracorporeal Circulation Technology

Marseille, France

http://www.fecect.org

21-24

ASAIO 63rd Annual Conference

Hyatt Chicago

Chicago, Illinois, USA

JULY 2017

28 June - July 1

The New Orleans Conference Las Vegas Edition

The Four Seasons Hotel and Resort

Las Vegas, Nevada, USA

http://www.theneworleansconference.com/#

AUGUST 2017

17-19

Perfusion Down Under Winter Meeting

Novotel Barossa Valley

Barossa Valley, Australia

http://perfusiondownunder.com/

SEPTEMBER 2017

7-9

AmSECT Pediatric Perfusion Meeting

Miami EPIC Hotel

Miami, Florida, USA

http://www.amsect.org/p/cm/ld/fid=1445

7-9

Case Reports in the Sun XIII

PGA National Golf Resort and Spa

Palm Beach Gardens, Florida, USA

http://www.floridaperfusion.org/latest-news/case-reports-in-the-sun-x/

24-27

28th Annual ELSO Conference

Hilton Baltimore

Baltimore, Maryland, USA

http://www.cvent.com/d/x5qj6f

28-30

13th International Conference on Pediatric Mechanical Circulatory Support Systems Pediatric and Cardiopulmonary Perfusion

Pontificia Universita Urbaniana

Rome, Italy

http://www.pediatricmcsrome2017.it/

OCTOBER 2017

12-14

Asia-Pacific ELSO Conference 2017

Gold Coast Convention & Exhibition Centre

Broadbeach, Gold Coast, QLD

http://apelso.com/

18-21

AmSECT Quality and Outcomes

Portland, Oregon, USA

http://www.amsect.org/p/cm/ld/fid=1464

NOVEMBER 2017

16-18

34th Annual Scientific Meeting ANZCP

The Langham Hotel

Southbank, Melbourne

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17-19TH AUGUST 2017

PERFUSION DOWNUNDER

WinterMeeting
PERFUSION DOWNUNDER

Novotel - Barossa Valley